

The effect of starvation on the dynamics of consumer populations

Yeakel, Kempes, & Redner

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1 The behavioral ecology of most, if not all, organisms is influenced by the energetic state
2 of individuals. An individual's energetic state directly influences how it invests its stores in
3 an uncertain environment. Such behaviors are generally manifested as trade-offs, which often
4 concern investing in individual maintenance and growth (somatic effort) or allocating energy
5 towards reproduction (reproductive effort) (Martin, 1987; Kirk, 1997; Kempes et al., 2012).
6 The timing of these behaviors is often important and is under strong selective pressure, as
7 they tend to have large effects on the future fitness of the organism (Mangel and Clark, 1988).
8 To what extent, and when, organisms invest in somatic vs. reproductive expenditures may be
9 driven by habitat, seasonality, evolutionary history, inter- or intra-specific interactions, the
10 distribution of resources. Importantly, the influence of resource limitation on an organism's
11 ability to maintain its nutritional stores may lead to repeated delays or shifts in reproduction
12 over the course of an organism's life.

13 Maximizing fitness between growth and maintenance activities vs. reproductive efforts
14 in large part structures the life-history of species, and this can be achieved by alternative

15 behavioral strategies or via physiological switches, both of which are sensitive to resource
16 availability. Behavioral changes in somatic or reproductive investment occur in response to
17 limited resources (Morris, 1987). For example, reindeer invest less in calves born after harsh
18 winters (when the mother's energetic state is poor) than in calves born after moderate winters
19 (Tveraa et al., 2003), whereas many bird species invest differently in broods during periods
20 of resource scarcity (Daan et al., 1988; Jacot et al., 2009), sometimes delaying or foregoing
21 reproduction for a breeding season (Martin, 1987; Barboza and Jorde, 2002). Such breeding
22 behaviors is generally referred to as *capital breeding*. Freshwater and marine zooplankton
23 have been observed to avoid reproduction under nutritional stress (Threlkeld, 1976), with
24 those that do reproduce evincing lower survival rates (Kirk, 1997), while artificially induced
25 stress has been observed to decrease reproductive success in Atlantic cod (Morgan et al.,
26 1999). Organisms may also separate maintenance and growth from reproduction over space
27 and time. Many salmonids, birds, and some mammals return to migratory breeding sites
28 to reproduce after one or multiple seasons in alternative environments spent accumulating
29 body mass and nutritional reserves (Weber et al., 1998; Mduma et al., 1999; Moore et al.,
30 2014).

31 Physiological mechanisms also play an important role in regulating reproductive expen-
32 ditures during periods of high-risk. Diverse mammals (47 species in 10 families) exhibit
33 delayed implantation whereby females postpone fetal development (blastocyst implantation)
34 to time with accumulation of nutritional reserves (Mead, 1989; Sandell, 1990). Furthermore,
35 many mammals (including humans) suffer irregular menstrual cycling and higher abortion
36 rates during periods of nutritional stress (Bulik et al., 1999; Trites and Donnelly, 2003). In
37 the extreme case of unicellular organisms, nutrition is unavoidably linked to reproduction
38 because the nutritional state of the individual regulates all aspects of the cell cycle (Glazier,

39 2009). The existence of so many independently evolved mechanisms across such a diverse
40 suite of organisms points to the importance and universality of the fundamental tradeoff
41 between somatic and reproductive investment, however the dynamic implications of these
42 constraints are unknown.

43 The mechanisms by which different organisms avoid or delay reproduction during times
44 of nutritional stress has received tremendous empirical and theoretical attention owing to
45 the importance of these activities in shaping life-history (Cody, 1966; Martin, 1987; Man-
46 gel and Clark, 1988). Less well understood is how resource limitation and these behav-
47 ioral/physiological tradeoffs affect dynamics at the level of the population. Traditional
48 Lotka-Volterra models assume a dependence of consumer population growth rates on resource
49 density, thus *implicitly* incorporating the requirement of resource availability for reproduc-
50 tion (Murdoch et al., 2003). Although this implicit dependence connects resource limitation
51 to lower consumer growth rates, the following biological realities are not included: *i*) some
52 individuals experience nutritional stress at a given time and under a given set of external
53 conditions, while others do not; those that do have multiple pathways enabling reproductive
54 cessation; *ii*) the portion of the population that is not nutritionally stressed is expected to
55 reproduce at a near-constant rate and this is – averaged across species – determined strongly
56 by body size (Kempes et al., 2012); *iii*) the rates that individuals transition from nutrition-
57 ally poor to replete states and back have different metabolically-constrained timescales that
58 can lead to reproductive lags. Importantly, the exclusion of these biological details may have
59 important dynamic shortcomings, masking the effects of starvation on consumer population
60 dynamics.

61 Resource limitation and the subsequent effects of starvation may be alternatively ac-
62 counted for *explicitly*, such that reproduction is permitted only for individuals with sufficient

63 energetic reserves. Though straightforward conceptually, incorporating the energetic dynam-
64 ics of individuals (Kooijman, 2000) into a population-level framework (Kooijman, 2000; Sousa
65 et al., 2010) presents numerous mathematical obstacles (in particular a lack of smoothness,
66 or differentiability; Diekmann and Metz, 2010), and often suffers from over-fitting due to
67 an overabundance of parameters. These issue have limited the development of theoretical
68 models that may aid our understanding of the effects of such tradeoffs on population dynam-
69 ics. An alternative approach to individual-to-population frameworks is redirecting focus to
70 macroscale relationships guiding somatic vs. reproductive investment in a consumer-resource
71 system.

72 Here we explore how the energetic tradeoff between maintaining and building somatic
73 tissue vs. reproduction can influence the dynamics of populations, and how such dynamics
74 may be determined by allometric constraints. We begin by establishing a simple Nutritional
75 State-structured population Model (NSM), where consumer starvation, and cessation of
76 reproduction, is the consequence of resource limitation. Similarly, recovery from the starved
77 to a reproductive state increases with resource density. Importantly, the rate at which
78 consumers decline and recover from a starved state is wholly constrained by metabolism. By
79 relating different rate constants to allometric constraints, we uncover important relationships
80 between the timescales of physiological and reproductive processes, and show how organisms
81 of different body sizes and taxonomic affinities may be prone to alternative dynamics.

82 We show that rates of starvation and recovery tend to result in systems with stable,
83 non-cyclic fixed points and that the ratio between the rate of starvation and recovery may
84 – in part – contribute to lower size limitations. Moreover, larger consumer body size results
85 in rates that are less prone to cyclic dynamics than are rates for smaller organisms, pointing
86 to potential empirical verification of the NSM framework. Finally, we show that rates of

87 starvation and recovery appear to be constrained to a parameter range where both transient
 88 and equilibrial population dynamics result in the lowest risk of extinction for the consumer.
 89 This surprising result suggests that the risks associated with different fluxes of consumers
 90 in and out of a starved (non-reproductive) state may serve as an important selective driver
 91 over evolutionary time.

92 **Starvation dynamics**

93 We integrate the somatic/reproductive tradeoff into the dynamics of a consumer-resource
 94 system by dividing the consumer population into discrete energetic states, the occupation of
 95 each being contingent on the consumption of a single resource R . In the NSM there are only
 96 two energetic states for the consumer population: *i*) an energetically replete (full) state F ,
 97 where the consumer reproduces at a constant rate λ , and *ii*) an energetically deficient (hun-
 98 gry) state H , where reproduction is suppressed, and mortality occurs at rate μ . Consumers
 99 transition from state F to state H by starvation at rate σ and in proportion to the lack of
 100 resources $(1 - R)$. Conversely, consumers recover from state H to the full state F at rate ρ
 101 and in proportion to R . The resource has logistic growth with a linear growth rate α and
 102 a carrying capacity of unity. Resources are eliminated by the consumer in both states: by
 103 energetically deficient consumers at rate ρ , and by energetically replete consumers at rate β .

104 Accordingly, the system of equations is written

$$\dot{F} = \lambda F + \rho RH - \sigma(1 - R)F,$$

$$\dot{H} = \sigma(1 - R)F - \rho RH - \mu H,$$

$$\dot{R} = \alpha R(1 - R) - R(\rho H + \beta F).$$

105 There are three steady states for the NSM: two trivial fixed points at $(R^* = 0, H^* =$
106 $0, F^* = 0)$ and $(R^* = 1, H^* = 0, F^* = 0)$, and one non-trivial internal fixed point at

$$F^* = \frac{\alpha\lambda\mu(\mu + \rho)}{(\lambda\rho + \mu\sigma)(\lambda\rho + \mu\beta)},$$
$$H^* = \frac{\alpha\lambda^2(\mu + \rho)}{(\lambda\rho + \mu\sigma)(\lambda\rho + \mu\beta)},$$
$$R^* = \frac{\mu(\sigma - \lambda)}{\lambda\rho + \mu\sigma}.$$

107 Because there is only one internal fixed point, as long as it is stable the population trajectories
108 will serve as a global attractor for any set of positive initial conditions. In a multidimensional
109 system, linear stability is determined with respect to the Jacobian Matrix $\mathbf{J}|_*$ (where $|_*$
110 denotes evaluation at the internal steady state), where each element of the matrix is defined
111 by the partial derivative of each differential equation with respect to each variable.

112 If the parameters of $\mathbf{J}|_*$ are such that the real part of its leading eigenvalue is < 0 ,
113 then the system is stable to small pulse perturbations. In the case of the NSM, stability
114 is conditioned on the consumer's starvation rate σ relative to the its reproduction rate λ .

115 As σ becomes less than λ , the R^* crosses the origin and the fixed point becomes unstable.
116 This condition $\sigma = \lambda$ marks a transcritical (TC) bifurcation, thus marking a hard boundary
117 below which the system becomes unphysical due to the unregulated growth of the consumer
118 population. That the timescale of reproduction is larger than the timescale of starvation
119 is intuitive for macroscopic organisms, as the rate at which one loses tissue due to lack of
120 resources is generally much faster than reproduction, though we will show using allometric
121 principles that this relationship always holds.

122 In addition to the hard bound defined by the TC bifurcation, oscillating or cyclic dynam-

123 ics present an implicit constraint to persistence by increasing the risk of extinction. If cycles
124 are large, stochastic effects may result in either the consumer or resource population. In
125 continuous-time systems, a stable limit cycle arises when a pair of complex conjugate eigen-
126 values crosses the imaginary axis to attain positive real parts (Guckenheimer and Holmes,
127 1983). This condition is called a Hopf bifurcation, and is defined by $\text{Det}(\mathbf{S}) = 0$, where \mathbf{S} is
128 the Sylvester matrix, which is composed of the coefficients of the characteristic polynomial
129 describing the Jacobian (Gross and Feudel, 2004). In addition to the onset of stable cycles,
130 as a system with a stable fixed point nears the Hopf bifurcation, transient or decaying cycles
131 can grow in magnitude. Given that ecological systems exist in a state of constant perturba-
132 tion (Hastings, 2001), even the onset of transient cycles that decay over time can increase
133 the risk of extinction (Neubert and Caswell, 1997; Caswell and Neubert, 2005; Neubert and
134 Caswell, 2009), such that the distance of a system from the Hopf bifurcation, even in the
135 stable region, is relevant to persistence.

136 Allometric constraints

137 Our model explores a wide variety of possible dynamics yet we have not described the realistic
138 regimes occupied by organisms where the challenge is to constrain the covariation of rates.
139 Allometric scaling relationships highlight common constraints and average trends across large
140 ranges in body size and species diversity. Many of these relationships can be derived from a
141 small set of assumptions and below we describe a framework for the covariation of timescales
142 and rates across the range of mammals for each of the key parameters of our model. We are
143 able to define the regime of dynamics occupied by the entire class of mammals along with
144 the key differences between the largest and smallest mammals.

145 Nearly all of the rates described in the NSM are to some extent governed by consumer
146 metabolism, and thus can be estimated based on known allometric constraints. The scal-

147 ing relationship between an organism's metabolic rate B and its body size at reproductive
148 maturity M is well documented (West et al., 2002) and plays a central role in a variety of
149 scaling relationships. Organismal metabolic rate B is known to scale as $B = B_0 M^\eta$, where η
150 is the scaling exponent, generally assumed to be $3/4$ for metazoans, and varies in unicellular
151 species between $\eta \approx 1$ in eukaryotes and $\eta \approx 1.76$ in bacteria (?). Several efforts have shown
152 how a partitioning of this metabolic rate between growth and maintenance purposes can be
153 used to derive a general equation for the growth trajectories and growth rates of organisms
154 ranging from bacteria to metazoans (Kempes et al., 2012). More specifically, the interspecific
155 trends in growth rate can be approximated by $\lambda = \lambda_0 M^{\eta-1}$. This relationship is derived
156 from the simple balance

$$B_0 m^\eta = E_m \frac{dm}{dt} + B_m m \quad (-1)$$

157 [a and b notation — these parameters are easily measured bioenergetic parameters which
158 are often approximately invariant across organisms of vastly different size. Our notation
159 seeks to illustrate that the allometric model fundamentally depends on a small number of
160 free parameters.] where E_m is the energy needed to synthesize a unit of mass, B_m is the
161 metabolic rate to support an existing unit of mass, and m is the mass at any point in
162 development. It is useful to explicitly write this balance because it can also be modified
163 to understand the rates of both starvation and recovery from starvation. [Spell out the
164 connection to nutritional state more explicitly] [As we will see it is possible to derive both
165 sigma and rho from this balance]

166 For the rate of starvation, we make the simple assumption that an organism must meet
167 its maintenance requirements using digested mass as the sole energy source. This assumption

168 implies the simple metabolic balance

$$\frac{dm}{dt} E'_m = -B_m m \quad (-1)$$

169 where E'_m is the amount of energy stored in a unit of existing body mass which may differ
170 from E_m , the energy required to synthesis a unit of biomass. Give the adult mass, M , of an
171 organism this energy balance prescribes the mass trajectory of a starving organism:

$$m(t) = M e^{-B_m t / E'_m}. \quad (-1)$$

172 Considering that only certain tissues can be digested for energy, for example the brain cannot
173 be degraded to fuel metabolism, we define the rate for starvation and death by the timescales
174 required to reach specific fractions of normal adult mass. We define $m_{starve} = \epsilon M$ where it
175 could be the case that organisms have a systematic size-dependent requirement for essential
176 tissues, such as the minimal bone or brain mass. For example, considering the observation
177 that body fat in mammals scales with overall body size according to $M_f = f_0 M^\gamma$, and
178 assuming that once this mass is fully digested the organism begins to starve, would imply
179 that $\epsilon = 1 - f_0 M^\gamma / M$. Taken together the time scale for starvation is given by

$$t_\sigma = -\frac{E_m \log(\epsilon)}{B_m}. \quad (-1)$$

180 The starvation rate is $\sigma = 1/t_\sigma$, which implies that σ is independent of adult mass if ϵ is a
181 constant, and if ϵ does scale with mass, then σ will have a factor of $1/\log(1 - f_0 M^\gamma / M)$.
182 In either case σ does not have a simple scaling with λ which is important for the dynamics
183 that we later discuss.

184 The time to death should follow a similar relationship, but defined by a lower fraction
 185 of adult mass, $m_{death} = \epsilon' M$. Consider, for example, that an organism dies once it has
 186 digested all fat and muscle tissues, and that muscle tissue scales with body mass according
 187 to $M_{mm} = mm_0 M^\zeta$, then $\epsilon' = 1 - (f_0 M^\gamma + mm_0 M^\zeta) / M$. Muscle mass has been shown to
 188 be roughly proportional to body mass ? in mammals and thus ϵ' is effectively ϵ minus a
 189 constant. Thus

$$t_\mu = -\frac{E_m \log(\epsilon')}{B_m} \quad (-1)$$

190 and $\mu = 1/t_\mu$.

191 It should be noted that we have thus far used mammalian allometry to describe the
 192 size-based relationships for growth, starvation, and death. However, our presentation is
 193 general, and other functional forms for ϵ , for example, could be determined for other classes
 194 of organisms. Considering bacteria, we might expect that starvation or death is defined
 195 by the complete digestion of proteins, and in Table 1 we provide all parameter values for
 196 bacteria which we later use as a comparison in our analysis.

197 The rate of recovery $\rho = 1/t_\rho$ requires that an organism accrues tissue from the starving
 198 state to the full state. We again use the balance given in Equation to find the timescale to
 199 return to the mature mass from a given reduced starvation mass. The general solution to
 200 Equation is given by

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

201 with $a = B_0/E_m$ and $b = B_m/E_m$. We are then interested in the timescale, $t_\rho = t_2 - t_1$,
 202 which is the time it takes to go from $m(t_1) = \epsilon M$ to $m(t_2) = M$, which has the final form

203 of

$$t_\rho = \frac{\log \left(1 - \left(M \left(\frac{a}{b} \right)^{\frac{1}{\eta-1}} \right)^{1-\eta} \right) - \log \left(1 - \left(M \epsilon \left(\frac{a}{b} \right)^{\frac{1}{\eta-1}} \right)^{1-\alpha} \right)}{(\eta-1)b}. \quad (-1)$$

204 **The stabilizing effects of allometric constraints**

205 Although starvation leads to mortality risk for the individual, a moderate amount promotes
206 persistence of both consumer and resource populations. Analysis of the NSM shows that
207 (non-cyclic) stability of the fixed point generally requires a higher starvation rate σ rel-
208 ative to the recovery rate ρ . The intuition behind this is that transition to the hungry
209 (non-reproductive) state permits the resource to recover and transient dynamics to subside,
210 whereas a low σ overloads the system with energetically-replete (reproducing) individuals,
211 thus producing maintained oscillations between consumer and resource (Fig. 2). However if
212 σ is too large, mortality due to starvation depletes the consumer population, resulting in a
213 lower steady state density for the consumer and an opposingly higher steady state density
214 for the resource.

215 Whereas the rate of consumer growth defines a hard bound of biological feasibility (the
216 TC bifurcation), the rate of starvation determines the sensitivity of the consumer population
217 to changes in resource density. While higher rates of starvation result in lower steady state
218 population size – increasing the risk of stochastic extinction – lower rates of starvation result
219 in a system poised near either the TC or Hopf bifurcation (or both), which will lead to
220 elimination of the resource or the development of cyclic oscillations, respectively. Which
221 bifurcation is approached is wholly dependent on the rate of recovery: if it is high, then
222 cyclic dynamics will develop; if it is low, resource extinction becomes increasingly likely.

223 As the allometric derivations of NSM rate laws reveal, σ and ρ are not independent
224 parameters, such that the bifurcation space shown in Fig. 2 cannot be freely navigated if as-

suming biologically reasonable parameterizations. Given the parameterization for terrestrial endotherms shown in Table ?? with mass M as the only free parameter, rates of starvation and recovery are constrained to a fairly small window of potential values. We find that the allometric constraints given for terrestrial endothermic organisms confine dynamics to the steady state regime across all reasonable body size classes, which for mammals ranges from ca. 1 gram (the Etruscan shrew) to ca. 10^7 grams (represented by the Indricotheriinae, a subfamily of mammals living from the mid-Eocene to early Miocene). Moreover, for larger organismal mass, the distance increases between the allometric values for $\sigma(M)$ and $\rho(M)$ relative to the Hopf bifurcation, while uncertainty in allometric parameters (20% variation around the mean; Fig. ??) results in little difference in the position of the TC and Hopf bifurcations as well as consumer energetic rates. This result suggests 1) that small mammals are more prone to population oscillations – including both stable limit cycles as well as transient cycles – than large mammals, and 2) the decreasing distance to the Hopf bifurcation with lower body sizes is suggestive of a dynamic barrier to the mass of endothermic organisms.

Although the prediction of larger oscillations for animals with smaller body sizes generally holds, empirical observations of large animal population cycles are plagued by long generation times and the influence of top-down effects [REFS] making direct empirical observation problematic.

Higher rates of starvation results in a larger flux of the population to the hungry state, eliminating reproduction and increasing the likelihood of death, however it is the relative size of the rate of starvation to the rate of recovery that determines the long-term dynamics of the system. By examining the ratio σ/ρ , we can understand the competing effects of cyclic dynamics and population density on the probability of extinction. We computed the probability of extinction, which we defined as $H(t) + F(t) = 10$ at any instant across

249 all values of t , for 1000 replicates of the continuous-time system shown in Eq. XX with
250 random starting conditions within the interval $(R^*, H^*, F^*) \pm 10\%$ (check) for an organism
251 of $M = 100\text{g}$. By allowing the rate of starvation to vary, we assessed extinction risk across
252 a range of the ratio σ/ρ varying between XXX and XXX, thus examining a vertical cross-
253 section of Fig 2. As we expected, we found higher rates of extinction for both low and high
254 values of σ/ρ ; for low values the higher extinction risk is the consequence of transient cycles
255 with larger amplitudes as the system nears the Hopf bifurcation. For high values of σ/ρ ,
256 higher extinction risk is due to the steady decrease in the steady state consumer population
257 density. This interplay creates an ‘extinction refuge’, such that for a relatively small range
258 of σ/ρ extinction probabilities are minimized.

259 As has been described, the σ vs. ρ space cannot be freely traversed, such that not all
260 values of σ/ρ are biologically feasible. We observe that the allometrically constrained values
261 of σ/ρ (with $\pm 20\%$ variability around the expectation) fall squarely within the extinction
262 refugia, such that they are close enough to the Hopf bifurcation to avoid low steady state
263 densities, but far enough away to avoid large-amplitude transient cycles. The fact that allo-
264 metric values of σ and ρ fall within this relatively small window is suggestive of a selective
265 mechanism driving these rates within populations, and suggests that the NSM system pro-
266 vides insight to a general phenomena that may both drive – and constrain – natural animal
267 populations.

268 The ratio σ/ρ
269 both the location of TC and Hopf bifurcations over σ and ρ relative to the allometrically-
270 determined starvation and recovery rates of species across different body sizes
271 [First describe the dynamics of the model and HOW IT WORKS] [Then Allometry and
272 stability]

273 Analysis of the 2-stage consumer resource model shows that the equilibrial states of both
274 populations are highly sensitive to changes in starvation and recovery rates of the consumer.
275 The consumer and resource population densities vary inversely: when the consumer densities
276 are high, resource densities are low, and vice versa. High starvation and low recovery rates
277 result in low consumer densities and high resource densities. If starvation rates are low,
278 resources have a fixed point near zero for any value of the recovery rate. Full and hungry
279 consumer stages tend have fixed points that are tightly correlated, the extent to which is
280 driven by the similarity of consumer growth and mortality rates; if $\lambda = \mu$, then $F^* = H^*$.

281 A transcritical bifurcation exists at $\lambda = \sigma$, such that the condition $\sigma > \lambda$ is required
282 for biologically reasonable dynamics. The TC bifurcation occurs in this model because
283 we have assumed that the portion of the population that is not starved reproduces at a
284 constant rate. Because the process of starvation is incorporated explicitly, the consumer's
285 rate of reproduction is not dependent on the density of resources. In fact, the existence of
286 the TC bifurcation at $\lambda = \sigma$ reveals an important biological insight. Reproduction requires
287 maintenance and growth of biological tissues, both of which have strong scaling relationships
288 with body size. Recent work by Kempes et al. [REF] derived the timescale of reproduction
289 in terms of allometric considerations, where $t_\lambda \propto M^{1-\eta}$ (REF). Starvation is the loss of
290 energy required for maintenance, and we have shown it to have a timescale $t_\sigma \propto \log(M)$.
291 Accordingly, the timescale of reproduction is always larger than the timescale of starvation,
292 such that λ must be less than σ by definition. A third important parameter in our framework
293 is the rate of recovery. The recovery timescale t_ρ controls the rate at which individuals move
294 from the hungry class to the full class, and this requires not only tissue maintenance, but
295 growth, such that it is bounded on the short side by t_σ . Moreover, [why is recovery timescale
296 bounded on the high side], such that it is bounded on the long side by t_λ . Thus, incorporating

297 allometric considerations shows us that $\lambda < \rho < \sigma$ (alternatively $t_\lambda > t_\rho > t_\sigma$).

298 The 2-stage consumer resource model exhibits two qualitatively different behavioral
299 regimes. Because portions of the consumer population exist in either full or hungry states
300 simultaneously, the internal fixed point can either be a stable equilibrium, or exhibit sus-
301 tained oscillatory behavior, depending on the rates of starvation σ and recovery ρ . The
302 transition from stable non-oscillatory dynamics to oscillatory dynamics occurs at the Hopf
303 bifurcation condition where two complex conjugate eigenvalues cross the imaginary axis and
304 attain positive real parts. Although there is an analytical solution to the Hopf bifurcation
305 condition, it cannot be written efficiently.

306 When the starvation rate is low, oscillatory dynamics are more likely to occur for a given
307 value of the recovery rate. This can be understood intuitively: for low starvation rates,
308 resources are depressed by an infusion of full consumers, which subsequently starve thereby
309 allowing the resource to recover and continuing the cycle. When the starvation rate is high,
310 the response of consumer growth to resource abundance is muted, such that oscillations tend
311 to decay over time. Thus, higher starvation rates σ desensitizes changes in the consumer
312 population to changes in resource density, and lower rates of recovery ρ amplifies this effect.

313 Both full and hungry consumers remove resources at rates b and ρ , respectively. As the
314 rate of resource consumption by full consumers increases, the Hopf condition changes from
315 a convex to a concave function over σ , limiting the potential for oscillatory dynamics. These
316 rates are considered separately because full consumers need only to maintain their tissues,
317 whereas hungry consumers require both growth and maintenance, such that $t_\rho > t_b$, or $\rho < b$.
318 If this constraint is enacted, the likelihood of oscillatory dynamics is reduced for a given

319 If instead the consumer's growth was proportional to resource abundance, such that the
320 effects of starvation on reproduction were incorporated explicitly (where the 2-stage consumer

321 resource model collapses to the Lotka-Volterra consumer-resource model with logistic growth
322 of the resource), the TC bifurcation exists only for $\lambda = \mu$, such that the rate of mortality
323 cannot exceed the intrinsic birth rate.

324 whereas the traditional Lotka-Volterra dynamic assumes that the reproductive rate of the
325 consumer is scaled to resource density, such that the growth function would be $G(F, R) =$
326 λRF . Thus, the Lotka-Volterra dynamic *implicitly* accounts for starvation in reducing the
327 reproductive rate of the consumer. However, our 2-stage model *explicitly* accounts for starva-
328 tion as well as recovery, such that individuals who are not starved should adopt a reproductive
329 rate independent of resource density.

330 We have used scaling relationships between tissue turnover and growth to strictly con-
331 strain 5/6 population-level parameters in our 2-stage consumer resource model (including
332 the mortality rate t_μ , which we have shown is just a xxx of t_σ). This exercise accomplishes
333 two goals: 1) it allows us to constrain the plausible parameter space of the two-stage model,
334 and 2)

335 This allows us to derive many aspects of the system in terms of consumer body mass M
336 and the allometric scaling exponent η .

337 **Minimizing extinction risk**

338

Table 1: Parameter Values For Various Classes of Organisms

	Mammals	Unicellular Eukaryotes	Bacteria
η	3/4		1.70
E_m	10695 (J gram $^{-1}$)		10695 (J gram $^{-1}$)
E'_m	$\approx E_m$		$\approx E_m$
B_0	0.019 (W gram $^{-\alpha}$)		1.96×10^{17}
B_m	0.025 (W gram $^{-1}$)		0.025 (W gram $^{-1}$)
a	1.78×10^{-6}		1.83×10^{13}
b	2.29×10^{-6}		2.29×10^{-6}
$\eta - 1$	-0.21		0.73
λ_0	3.39×10^{-7} (s $^{-1}$ gram $^{1-\eta}$)		56493
γ	1.19		0.68
f_0	0.02		1.30×10^{-5}
ζ	1.01		
mm_0	0.32		

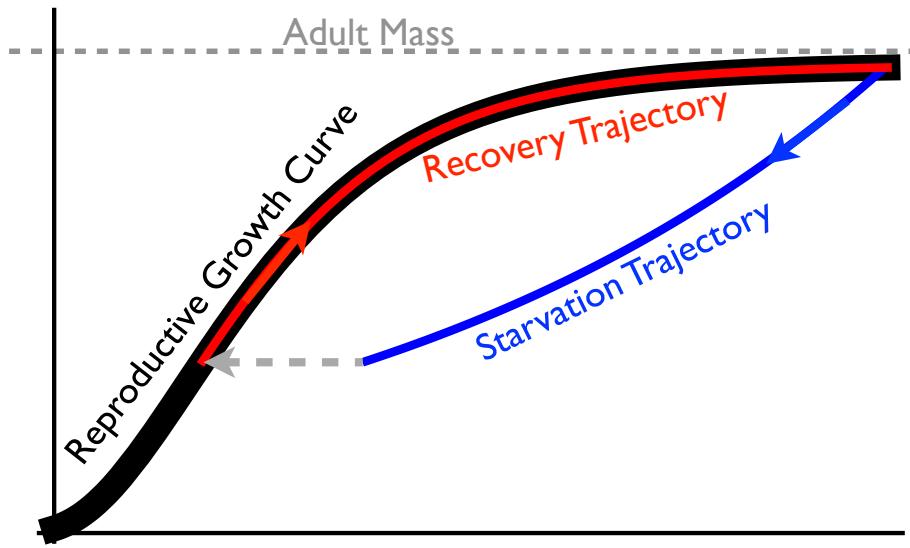


Figure 1

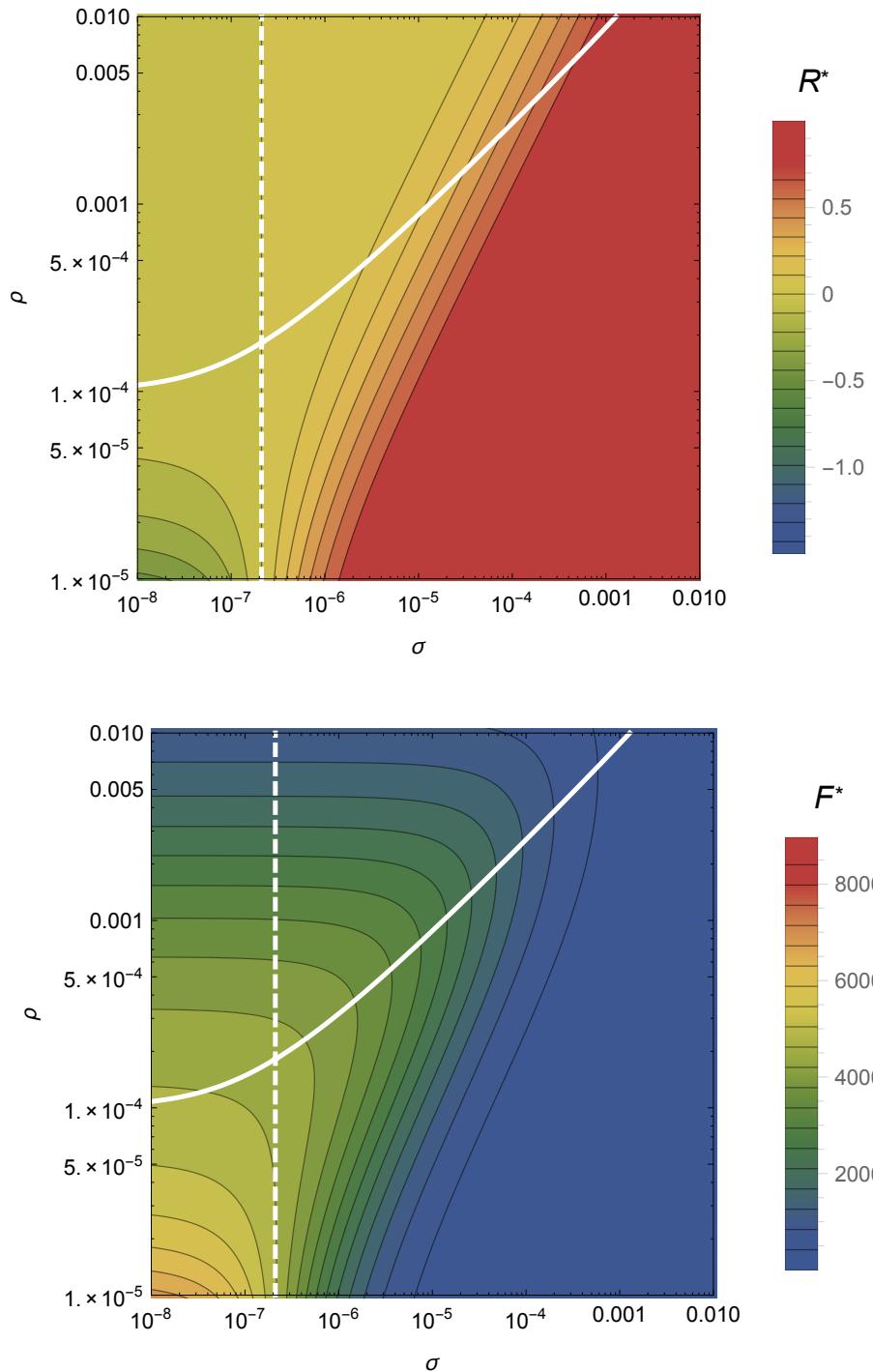


Figure 2

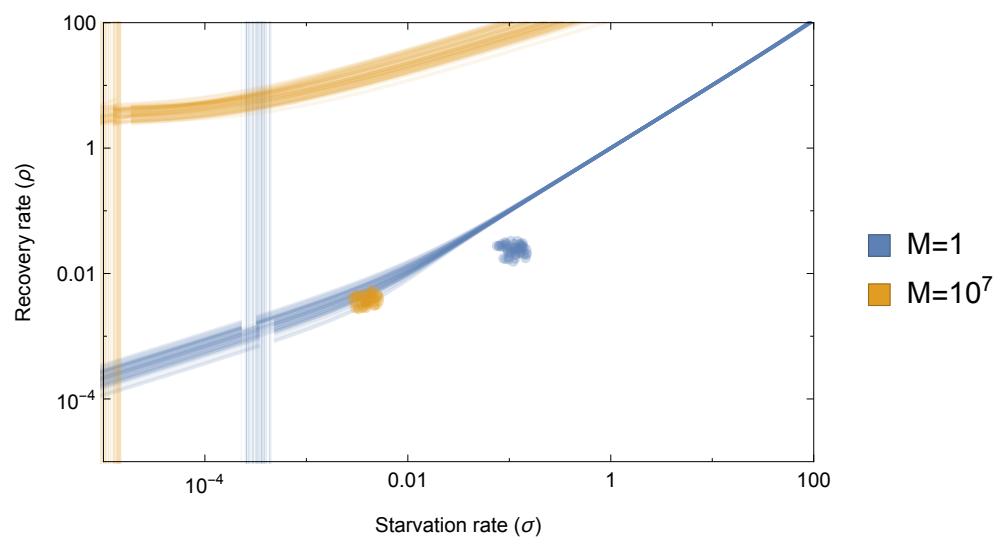


Figure 3

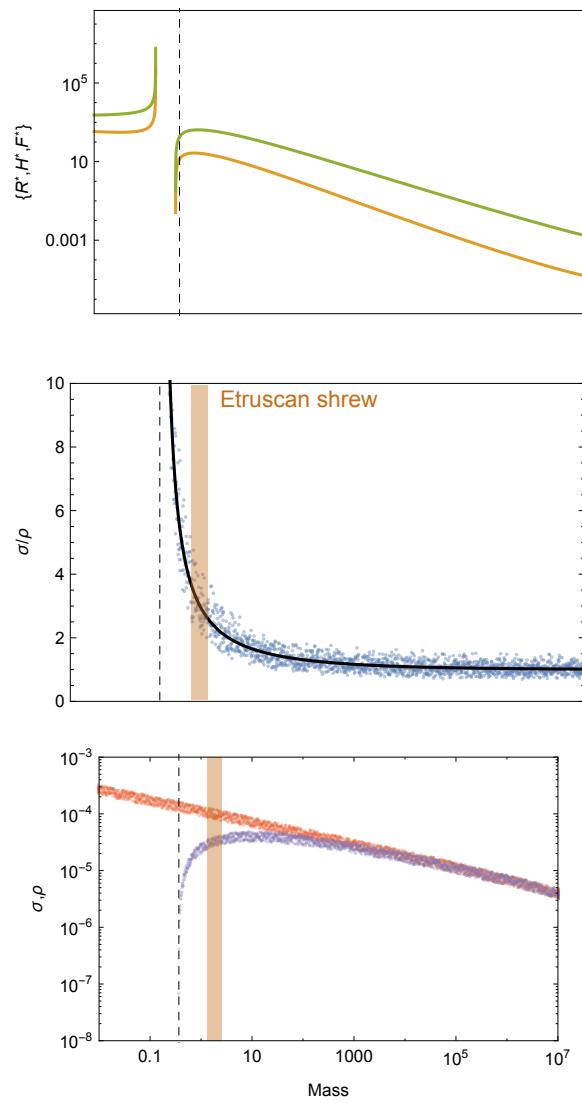


Figure 4

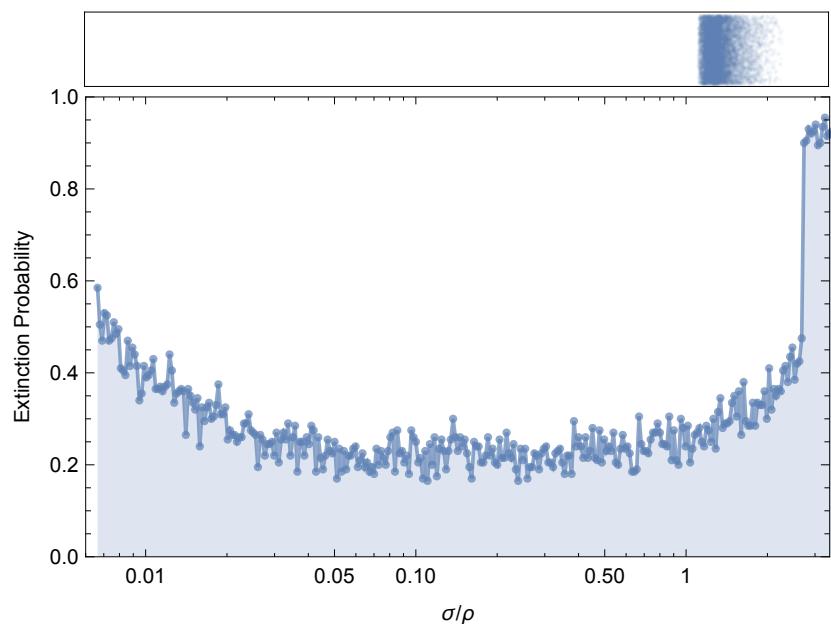


Figure 5

³³⁹ **References**

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