



Life histories and Cope's rule from an explicit resource–consumer model based on metabolic theory

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HIGHLIGHTS

- ▶ Consequences of metabolic theory and resource dynamics on life history traits.
- ▶ Individual-based model using allometric scaling of metabolic rates with body mass.
- ▶ Individuals using more faster growing resources have shorter lifespans.
- ▶ Adult body mass increases unlimited resembling Copes rule.
- ▶ Other factors than allometric scaling keep the evolution of body mass in limits.

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ABSTRACT

We explore the consequences of metabolic theory for life histories and life history evolution. We use a mathematical model for an iteroparous species and its resources, taking into account the allometric scaling of consumption, metabolism, and mortality with consumer body mass. Mortality is assumed to be density-dependent, and the dynamics of resources are modeled explicitly. By evaluating life history features in equilibrium populations, we find that in populations that use more or faster growing resources the individuals have a shorter lifespan and a higher mortality, and that individuals in populations with a larger adult body mass have a longer lifespan, a larger number of offspring per female, and a higher biomass density. When we allow the adult body mass to evolve, it increases in time without limits. When we allow the offspring body mass to evolve independently from adult body mass, it becomes smaller. However, when we take into account that larger individuals have larger offspring, both body masses evolve to larger values. These trends result from the allometric scaling of mortality and can be kept in limits by trade-offs other than those included in our model.

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1. Introduction

The life history of an organism is affected by trade-offs that are due to a limited energy budget. There are for instance trade-offs between growth in body mass and reproduction, between offspring number and offspring mass, between reproduction and survival and health, and between present and future reproduction (Stearns, 1992). It is generally believed that life histories are shaped by evolution such that they optimize the population growth rate. The central equation used to determine the

maximum growth rate r is the Euler–Lotka equation (Euler, 1760; Lotka, 1907)

$$\sum_X e^{-rX} l(X) m(X) = 1, \quad (1)$$

where X denotes the age class; $l(X)$ is the probability that a newborn individual survives at least to the beginning of age class X , and $m(X)$ is the average clutch size of individuals in age class X . For stationary populations that have reached their carrying capacity, the growth rate is $r=0$, and the average number of offspring that reach maturity is 1 per individual.

The main challenge in life history theory consists in obtaining realistic estimates of the functions $l(X)$ and $m(X)$, or, equivalently, of the elements of the so-called Leslie matrix (Leslie, 1945). Usually, they are postulated on the basis of plausible scenarios and empirical knowledge.

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Recently, it has been emphasized that metabolic theory has profound implications on the evolution of life histories (Brown and Sibly, 2006). Larger organisms have a lower rate of production per unit body mass, and they have a lower mortality (Ernest et al., 2003; Brown et al., 2004). Also, metabolic theory has been used to explore consequences for population-level processes, such as the intrinsic rate of exponential growth and carrying capacity (Savage et al., 2004), as well as population interactions, such as predation (Weitz and Levin, 2006). Recently, metabolic theory has been included in food web models (Rall et al., 2008; Berlow et al., 2009). However, studies investigating life history evolution in context of metabolic theory typically explore scaling relationships of life history traits such as those between age at maturity and body mass (Charnov and Gillooly, 2004). Here we take another approach by using individual based modeling of the dynamics of consumers and their resources, and incorporate metabolic theory to explore the power of the theory to predict life history attributes, such as adult body size and age at maturity. A similar approach has been used by Kawecki (1993), who considers consumers exploiting resources in a patch with the consumption rate and maintenance cost depending on body mass. The consumers may adopt different strategies for investing in growth and in offspring production. Which strategy has the highest fitness depends on the total amount of resources in the patch and on the other strategies present. This paper differs also in other respects from the classical life history literature, since it includes explicit dynamics and competition between different strategies, instead of performing an optimization calculation. As has been pointed out by Stearns (2000), approaches of this type to life history theory are very much needed.

In this paper, we derive for the first time life history traits from a full dynamical model for a consumer species and its resource, taking into account metabolic theory, which determines the scaling of consumption, metabolism and mortality with the body mass of the consumer. The consumer population size is limited by a density-dependent term, and consequently the population becomes stationary after some time. We evaluate various life history parameters in the stationary population. Furthermore, we perform an evolutionary simulation by introducing into the population individuals with a different body mass at maturity. We find that body mass evolves towards larger values. This means that Cope's rule (Hone and Benton, 2005) follows from metabolic theory alone, without need to take into account additional advantages of larger body size such as the ability to exploit new niches or to avoid predators more efficiently.

Our model is a minimal model insofar as it includes only the mentioned ingredients, i.e., consumer–resource dynamics and allometric scaling of consumption, metabolism, and mortality. These effects are due to different metabolic rates, i.e., due to the fact that the pace of life is slower for larger organisms. The model does not consider other factors that depend on body size or age and may therefore affect life histories, such as predation, aging, and body-size restrictions due to physical and environmental conditions, although we will discuss some of these effects. Our results therefore tell what would be the case if only the factors included in the model were at work. Surprisingly, already this minimal, in several respects incomplete or unrealistic, model reproduces various known features of life histories, while it is admittedly unrealistic in other respects.

2. Model

Our model is an individual-based model for iteroparous species. The dynamics of the body mass of each individual are simulated explicitly. The size of the habitat is defined as unit area. Hence, the biomass density of the population numerically equals

the total biomass of the population. After birth, individuals grow due to consuming resources, they lose mass due to respiration, and once a year they produce offspring if their body mass is above a threshold value. The probability of dying depends on body mass and density. Resources have a logistic growth, and they are reduced due to consumption. Our consumer–resource model is similar to the one used by Rall et al. (2008), which is based on the work of Yodzis and Innes (1992) and is updated with new allometric coefficients (Brown et al., 2004; Ernest et al., 2003). Since an individual dies as a whole and not partially, mortality is implemented as a probability for occasional death in our individual-based model. Since including males makes no significant difference apart from making the equation more complicated, we consider only females, like in other treatments of life history theory (Stearns, 1992).

The model is initiated with the resource biomass at the carrying capacity, $R = K_r$. The body mass of resources is taken as mass unit. Resources grow continuously according to the logistic function with a maximum growth rate G and lose biomass due to being eaten by consumer individuals

$$\dot{R} = G \left(1 - \frac{R}{K_r} \right) R - \sum_{i=\text{consumers}} \xi_i \varepsilon B_i F(R), \quad (2)$$

where the sum is taken over all consumer individuals. Consumer individuals eat resources according to a Holling type II functional response (Holling, 1959)

$$F(R) = \frac{R/K_r}{1 + R/K_r}, \quad (3)$$

where the attack rate and the handling time are absorbed into the parameters $\varepsilon \xi_i$ and K_r , so that our functional response is formally the same as the Monod equation (Liu, 2007). The body mass B_i of consumer i increases from the initial value $B_{\text{juvenile}} = 1$ due to feeding and respiration according to the equation

$$\dot{B}_i = -\xi_i B_i + \lambda \xi_i \varepsilon B_i F(R). \quad (4)$$

The ecological efficiency λ was chosen to be 0.4 (Turner, 1970), and the maximum consumption rate ε of the consumers relative to their metabolic rate is a parameter of the model. The loss term scales with body mass according to

$$\xi_i = a B_i^{-0.25}. \quad (5)$$

The allometric constant a is chosen to be 0.314, which is a typical value for consumers of resources with unit body mass (Rall et al., 2008). Larger individuals need less energy per body mass for metabolism. The parameters in the functional response are chosen such that R/K_r cannot be close to 0 in the stationary state of Eq. (2), which means that the resources are always exploited to some extent. Otherwise, the consumers would not be able to survive (if R/K_r was close to 0).

While we are aware of alternative approaches as to how attack rates and handling times should scale with consumer and resource body mass (Weitz and Levin, 2006), we chose here a form that is also used by other authors in the food web literature (Rall et al., 2008), and that has the attractive features that the maximum ingestion rate scales with consumer body mass in the same way as the metabolic rate and that the half-saturation density of the functional response does not depend on consumer body mass.

When an individual does not obtain enough food, its body mass may drop below the minimum juvenile mass B_{juvenile} , in which case the individual is removed from the system.

A year has the duration $\tau = 1$. In order to efficiently implement the mortality of individuals, each year is divided into 10 time intervals of equal length, at the end of which every individual dies with a probability $p \in [0, 1]$ depending on its body mass and the

total biomass of all consumers in the system

$$p = 1 - e^{-\chi_i}, \quad \text{with } \chi_i = \frac{1}{n_i} \sum_{\text{consumers}} B_i/B_i^{0.25}. \quad (6)$$

This is the time-discrete version of a density-dependent mortality, such as for instance implemented by Kertaschew et al. (2010). Larger individuals have a smaller probability of dying. A scaling of mortality with $B_i^{-0.25}$ means that the dominant causes of death affect the biomass of an age class in a similar way as metabolism does. This type of scaling is widely accepted (Brown et al., 2004; Charnov and Gillooly, 2004; Peters, 1983; Savage et al., 2003).

The parameter n is related to the maximum biomass imposed by the limited space of the system. Its inverse, $1/n$, specifies the strength of mortality. Dead individuals are removed from the system. Our model does not include a dependence of mortality on age. This agrees with the widespread idea that most individuals die of causes other than old age and that longevity is mainly determined by extrinsic mortality, so that aging is due to late-acting deleterious alleles that are not eliminated by natural selection (see for instance Mueller, 1987). Although empirical reality is more complex than this simple view (Williams et al., 2006), our minimal model is appropriate as long as it can be assumed that the majority of adults die due to extrinsic causes and not due to old age. In fact, we will argue in more detail in the discussion that including senescence into the model would not affect the trends observed in the simulation results and would therefore leave the main conclusions intact.

Once a year, reproduction takes place. We assume that a female is sexually mature once it reaches the minimum adult body mass B_{adult} . Females with a body mass B_{mother} larger than B_{adult} by at least twice B_{juvenile} produce

$$\frac{B_{\text{mother}} - B_{\text{adult}}}{2 \cdot B_{\text{juvenile}}} \quad (7)$$

offspring (rounded to an integer) with body mass $B_{\text{juvenile}} = 1$. Taking into account parental care such as nursing and feeding, we estimate the cost of each offspring to be twice B_{juvenile} , therefore the body mass of the mother due to offspring production is decreased by twice the offspring body mass, $2B_{\text{juvenile}} = 2$, for each child. The offspring body mass B_{juvenile} is the minimum juvenile body mass after parents have stopped investing into their offspring, i.e. when the juveniles begin to independently feed on resources.

In the following, we will explore the influence of the five parameters ε , G , K_r , n , and B_{adult} on life history traits such as age at maturity, mean generation time, expected life time, probability of surviving from birth to beginning of age class X , expected number of offspring for a female in age class X , and fecundity for a female in age class X . Furthermore, we will explore the evolutionary consequences of metabolic theory by studying the evolution of minimum adult and juvenile body mass, B_{adult} and B_{juvenile} , within this model.

3. Results

3.1. Influence of the parameter values on life history traits

After 200 years (200τ in the simulations) the consumer population has reached its equilibrium, i.e., it has obtained a stationary age distribution, and the population size and the total biomass remain approximately constant due to the density limiting factor n . In order to obtain good statistics, the life history traits were evaluated by averaging over two million years, $2 \times 10^6\tau$.

Fig. 1 shows an example data set for the fecundity of a female as a function of the age class X . Fecundity V_X is defined as the product of the expected number of offspring, m_X , and the

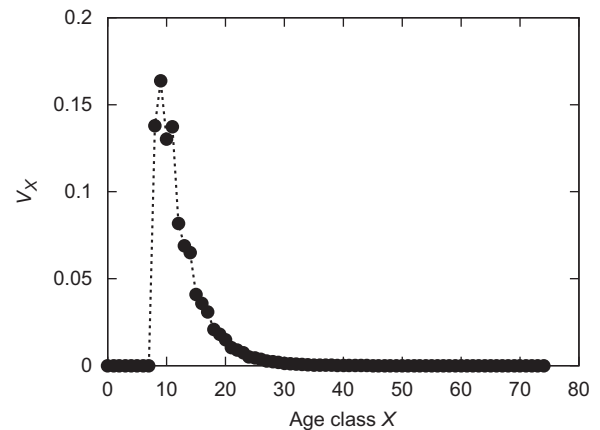


Fig. 1. Fecundity V_X for a female in age class X . The parameter applied are $\varepsilon = 15$, $n = 20\,000$, $G = 1$, $K_r = 40\,000$, and $B_{\text{adult}} = 20$.

probability of surviving from birth until reaching beginning of age class X , l_X . In our model only individuals with a body mass above B_{adult} can reproduce, therefore m_X remains zero until reaching maturity. Reproduction takes place once per year, and the clutch size is determined by the mother's body mass (Eq. (7)), which in turn results from resource consumption and metabolism (Eq. (4)). Therefore, m_X is independent of age after maturity. Similarly, mortality depends only on body mass and not on age in our simple model. Naturally, l_X decreases monotonously with age. It follows that fecundity is zero until reaching maturity and is thereafter identical to the product of l_X with m_X , with the latter parameter being approximately constant. In the following, we will explore in more detail the dependence of l_X on the model parameters.

The survival probability l_X is closely related to mortality, which depends on body mass but not on age (Eq. (6)). Since the body mass of an individual remains approximately B_{adult} after maturity, mortality is approximately constant for adult individuals in an equilibrium population. This means that the survival probability l_X decreases exponentially with age class X . This can be seen in Fig. 2, where the logarithm of l_X is perfectly linear once maturity has been reached. Since population dynamics are at equilibrium, the body mass of an individual grows within a population of approximately constant total biomass from birth until reaching maturity. Therefore, mortality decreases with age until reaching maturity (Eq. (6)), and so does the slope of $\ln l_X$ (Fig. 2).

Fig. 2 shows also that adult mortality decreases with decreasing ε , decreasing G , decreasing K_r , and increasing n . The decrease with decreasing ε or G or K_r is due to the fact that adults that consume less resources produce less offspring, which in turn means less competition from juveniles and therefore a longer lifespan of the adults. The decrease in adult mortality with increasing n has a similar reason: for larger n the total biomass in the stationary population is larger, implying that every individual obtains less food and produces fewer offspring.

Fig. 2(e) shows that the slope of $\ln l_X$ above the age at maturity is smaller for a population of larger adult body mass B_{adult} . This is to be expected since larger individuals have reduced mortality rates. However, before maturity the slope is smaller for a population of individuals of smaller adult body mass (see inset of Fig. 2(e)). This means that adults with a smaller body mass are less severe competitors for a juvenile. The reason for this is the larger total biomass of a population with larger B_{adult} , which results from the fact that larger individuals have a slower metabolism and can therefore maintain a larger biomass on the same resources.

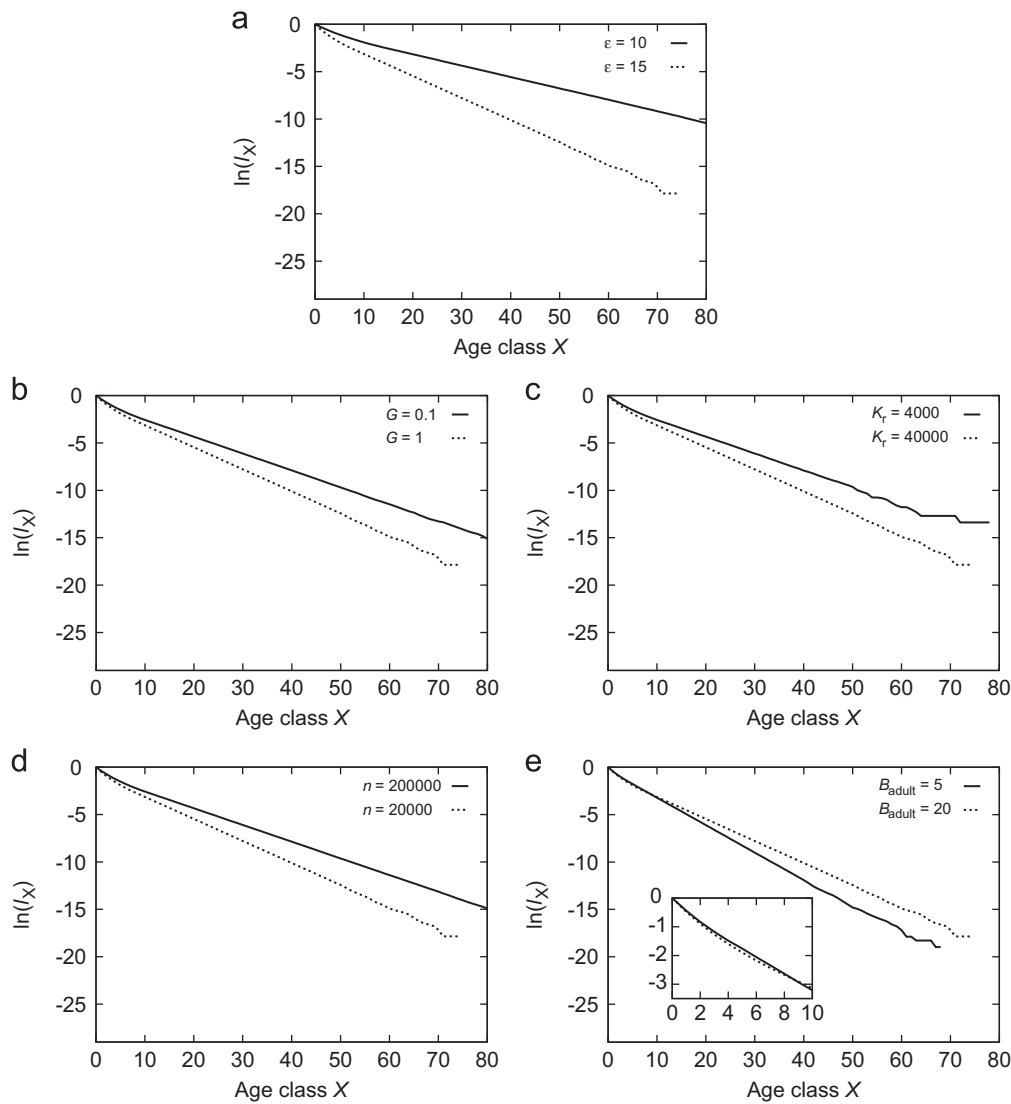


Fig. 2. Logarithm of the probability of surviving from birth to the beginning of age class X . The impacts of different variables on the survival probability are shown: (a) maximum consumption rate ε , (b) mass-specific maximum growth rate of the resource G , (c) carrying capacity of the resource K_r , (d) density limiting factor n , and (e) adult body mass B_{adult} . Parameters are chosen as in Fig. 1 if not labeled. The flat ends of some curves are due to bad statistics, because not many individuals with very large body size coexist.

We also examined the influence of the model variables on other life history traits, such as the age at maturity α , the generation time T , the expected lifespan LS , and the expected number of offspring for a female in age class X , m_X (Fig. 3). We define the age at maturity as the age at which an individual reaches a body mass of at least $B_{\text{adult}} + 2B_{\text{juvenile}}$, when it can give birth to its first offspring. The expected number of offspring, m_X , for a female in age class X , is zero until reaching maturity, and is independent of age when the maturity has been reached. We therefore dropped the index X from m in the figure legend.

We have pointed out above that for larger K_r or ε or G or a smaller n , an adult consumes more resources and that the total biomass and the mortality are larger. Therefore, the age at maturity, α , the generation time, T , and the lifespan, LS , decrease with increasing K_r or ε or G , and with decreasing n . The generation time is of course larger than the age at maturity (Fig. 3). The trend of LS follows that of α , meaning that populations that mature late live longer. For the same reason, the mean number of offspring per female per year, m , increases with increasing K_r or ε or G , and with decreasing n . However, since the population is stationary,

the expected total number of offspring that will reach maturity per female during her lifetime is identical to 1.

For sufficiently high resource growth rate, G , the curves become constant, because the resource biomass is constrained by its carrying capacity K_r (Eq. (2), Fig. 3). Changing the carrying capacity has a similar effect as changing G . This can be understood by rewriting Eq. (2) as a function of the ratio $\tilde{R} \equiv R/K_r \in [0, 1]$:

$$\dot{\tilde{R}} = G(1 - \tilde{R})\tilde{R} - \frac{1}{K_r} \sum_{i=\text{consumers}} a_i B_i^{3/4} \frac{\tilde{R}}{1 + \tilde{R}} \quad (8)$$

In this transformed equation, it is obvious that changing G and changing K_r have the same effect on the stationary value of \tilde{R} .

The increase of the generation time T and the age at maturity α with increasing B_{adult} is due to the fact that it takes longer to grow to a larger body mass at maturity. The increase of the mean annual number of offspring per female m with increasing B_{adult} results from the larger food intake of larger individuals, and the shorter lifespan is due to the larger juvenile mortality mentioned earlier. As l_X also increases with B_{adult} (Fig. 2), the fecundity $V_X = l_X m$ increases with body mass.

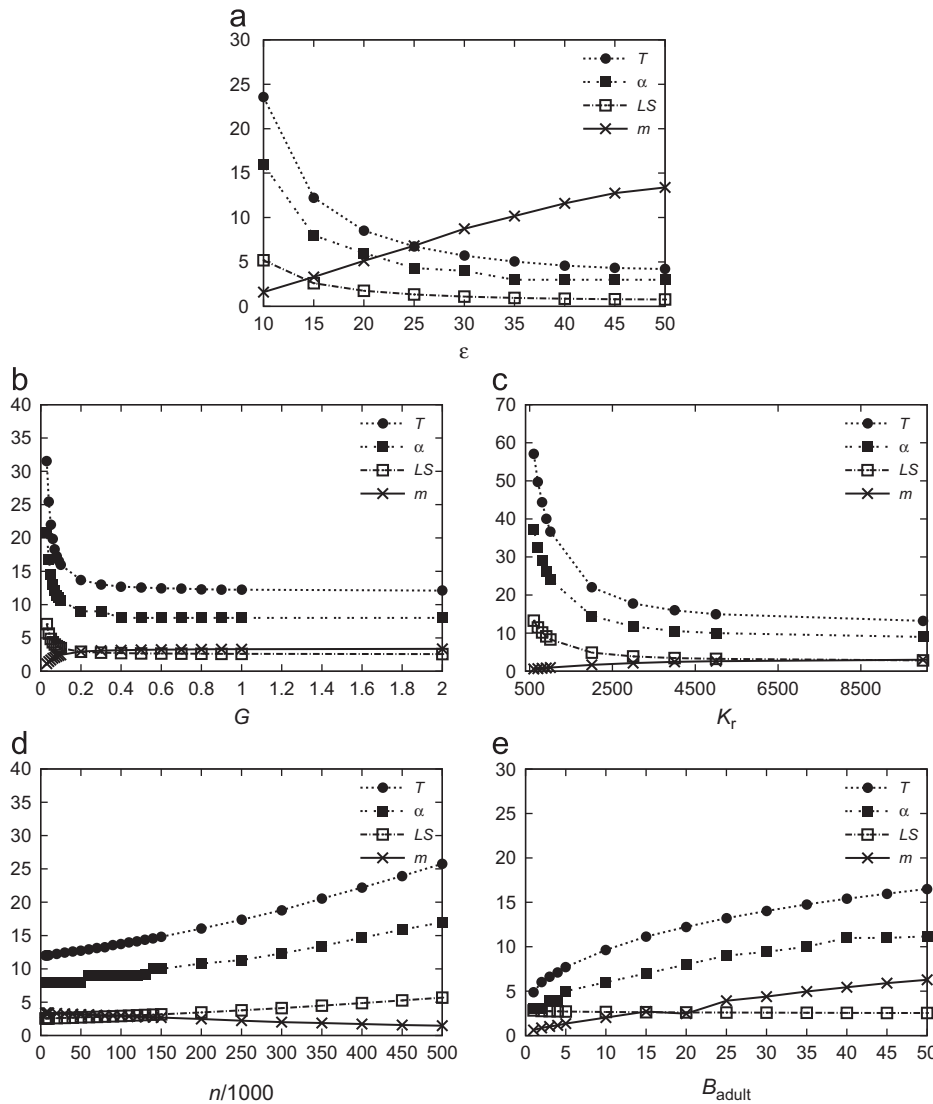


Fig. 3. Generation time T , age at maturity α , expected lifespan LS , and expected number of offspring for a female per year after maturity m . The influence of (a) maximum consumption rate ϵ , (b) mass-specific maximum growth rate of the resource G , (c) carrying capacity of the resource K_r , (d) density limiting factor n , and (e) adult body mass B_{adult} on T , α , LS , and m . Parameters are chosen as in Fig. 1 if not varied.

In order to understand which of all the features reported so far are due to the fact that mortality depends on density, we also investigated the case where mortality is independent of density, i.e.,

$$\chi_i^{(\text{density independent})} = \mu B_i^{-0.25}, \tag{9}$$

with a new parameter μ that plays a similar role as $1/n$ in Eq. (6). One can also imagine mortality to depend on the sum $\sum_i \xi B_i \propto \sum_i B_i^{0.75}$ instead of the sum $\sum_i B_i$, a situation that is intermediate between these two cases. When mortality is density independent, the survival probability l_x does not depend on G , ϵ , and K_r . It increases with decreasing μ and increasing B_{adult} . Since survival probability is independent of G , K_r and ϵ , the age structure of the population is also independent of these parameters, as is the amount of resources consumed per individual. Therefore, the generation time, lifespan, age at maturity, and mean number of offspring per female per year do not depend on these parameters either. The only change that occurs when these parameters are increased is an increase in the total population size. The fraction of available resource at equilibrium, R/K_r , is kept at the same level with increasing G and K_r , while this fraction significantly decreases with increasing ϵ . For this reason, the increase in population size with increasing ϵ lasts only up to a certain

value, then the population size decreases as ϵ increases further. When μ or B_{adult} is changed, the trends of T , α , LS , and m are the same as in the density-dependent case for changing $1/n$ or B_{adult} . The mechanisms causing these trends remain the same.

However, the change of the equilibrium resource biomass R with B_{adult} is different in the two cases. When mortality is density dependent, R increases with increasing B_{adult} , while it decreases in the density-independent case. In both cases, the total biomass increases with increasing B_{adult} . However, a density-dependent mortality counteracts the increase of the total biomass by killing more individuals when total biomass becomes larger. The result is that the increase in total biomass is weaker than $B_{adult}^{0.25}$ (Eq. (6)), which would be the increase required to obtain the same level of resource exploitation as in the case with density-independent mortality.

3.2. Evolution of body mass

All investigations reported so far were performed with a fixed adult body mass B_{adult} and a fixed minimum juvenile body mass $B_{juvenile}$. However, these two values are the result of evolutionary processes, and on the long run those body masses will dominate

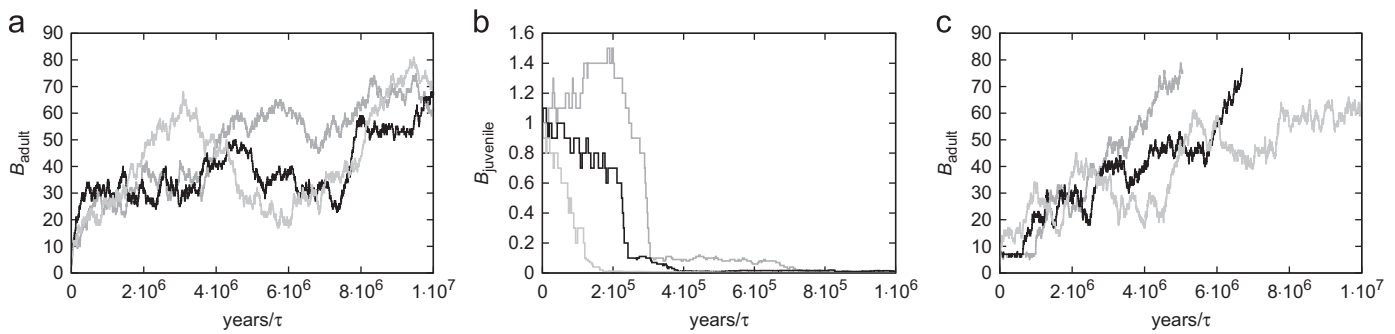


Fig. 4. Evolution of (a) adult body mass B_{adult} with fixed juvenile body mass $B_{\text{juvenile}} = 1$, of (b) juvenile body mass B_{juvenile} with fixed adult body mass $B_{\text{adult}} = 15$, and (c) of adult body mass B_{adult} with juvenile body mass B_{juvenile} scaling as adult body mass to the power 0.75. The values of those parameter that were not evolved are $\varepsilon = 15$, $n = 20\,000$, $G = 1$, $K_r = 80\,000$ (black, dark gray and gray curves are results from three independent simulations with different random number generator seeds).

that are the best “strategies” for dividing resources between growth and offspring production. We performed three types of computer simulations in order to study the evolution of these body masses. In the first simulation, we allowed the adult body mass to evolve, keeping the juvenile body mass fixed. In the second simulation, we allowed the juvenile body mass to evolve, keeping the adult body mass fixed. In the third simulation, we evolved both body masses together. This was done by letting a female with a body mass B_{mother} produce exactly one offspring per year, investing all of its body mass above B_{adult} into this offspring. This means that the body mass of the mother became B_{adult} after birth and the initial body mass of the offspring was $(B_{\text{mother}} - B_{\text{adult}})/2$. In this way, the juvenile body mass scales as the adult body mass to the power 0.75, because the amount of resources eaten scales in this way.

The evolution was done by first running the simulation for 200 years in the same way as before. After these 200 years, we assigned to half of the offspring born in this year a modified adult or juvenile body mass $B_{\text{mutant}} = B_{\text{original}} \pm \Delta$. In computer simulations for the evolution of adult body mass we set $\Delta = B_{\text{juvenile}} = 1$, and in computer simulations for the evolution of the juvenile body mass $\Delta = 0.1$ for $B_{\text{original}} > 0.1$ and $\Delta = 0.01$ for $0.01 < B_{\text{original}} \leq 0.1$, etc. We then let the simulations run until one of the two strategies present in the population had driven the other to extinction. Then we waited for another 200 years before introducing another mutant by modifying again the adult or minimum juvenile body mass of some individuals. Our computer simulation thus investigated the evolutionary stability of populations with a certain adult body mass with respect to invasion of a group of individuals with a slightly different adult body mass (Mylius and Dieckmann, 1995).

Fig. 4 shows three independent simulation runs for each of the three types of evolutionary simulations. In both cases studied (Fig. 4(a) and (c)) the evolution of adult body mass resembles a random walk with a bias towards larger B_{adult} . This means that an individual with a larger adult body mass has on average more children surviving to adulthood during its lifetime than an individual with a smaller body mass. This follows indeed from the allometric scaling of the terms contributing to population dynamics: an individual that matures 1 year later than the other members in its age class, starts producing children 1 year later. But then it has on average more or larger children per year and a lower mortality than other individuals of the same age, obtaining in total more children that survive to adulthood during its life time. This conclusion holds of course only as long as there are no disadvantages to larger body mass, since our model only includes the advantage of having a slower metabolism. Moreover, the evolution towards larger body mass depends crucially on the fact that mortality decreases as $B_{\text{adult}}^{-0.25}$ with increasing body mass, which means that mortality scales the same way as resource

consumption and metabolism. If mortality decreased slower with increasing body mass, the biomass loss due to mortality would eventually become larger than the biomass gain due to resource consumption. In this case, the evolutionary increase of B_{adult} would eventually stop. However, in our minimal model the only constraints on the adult body mass are due to the limited resources and the factor n that limits the total biomass of the population. As the adult body mass increases, the population size declines and the population thus becomes more susceptible to extinction (Stanley, 1973). Population extinctions happen in fact at the end of all the simulations shown in Fig. 4(a) and (c) (only two of the simulations end in the plotted time interval though).

The evolution of juvenile body mass shows the opposite trend. This means that it is always better to produce smaller offspring in our model, as long as correlations between parent and offspring body mass are ignored. In order to understand this, let us compare the production of an offspring of body mass B_{juvenile} with the production of two offspring of size $B_{\text{juvenile}}/2$. If each of the two small offspring have a chance larger than $1/2$ to survive until they reach the body mass B_{juvenile} , then an adult that produces the smaller offspring will have more surviving children during its lifetime than an adult that produces the larger offspring. Indeed, our model has the feature that the total biomass of all individuals that were born at the same time increases with time, until maturity is reached. Consequently, after the time required for an offspring born with body mass $B_{\text{juvenile}}/2$ to grow to the body mass B_{juvenile} , the expected total biomass of the two offspring is larger than B_{juvenile} . This means that the chance that a small offspring survives until reaching B_{juvenile} is larger than $1/2$. All this is a result of the mortality scaling in the same way as resource consumption and metabolism. Because all three determinants of growth scale in the same way with body mass, the rate of change of the total biomass of all individuals that were born at the same time is a constant. This constant cannot be negative, since the population would then die out. Therefore, it must be positive, with the result that it always pays off to produce smaller offspring. If mortality increased stronger with decreasing body mass, there would be a minimum offspring body mass. Similarly, if we included constraints in the relation between parent and offspring body mass, as was done in the simulations shown in Fig. 4(c), offspring body mass would increase with parent body mass. Indeed, such an allometric relation between parent and offspring body mass is well known (see for instance Peters, 1983, pp. 130–131), and its inclusion in the simulations is thus well justified.

4. Discussion

We have investigated the life history traits of a population feeding on resources which regrow by using a model that

considers explicitly the dynamics of both the consumers and the resources. The model takes into account the allometric scaling of resource consumption, metabolism, and mortality with the body mass of the consumer. Since no other factors are taken into account, this is a minimal model that investigates in the purest possible form the effect of allometric scaling on life history features. These life history features are evaluated in a stationary population. Despite the simplicity of the model, several life history characteristics known from natural systems emerge correctly from the model, for instance the correlation between age at maturity and longevity (Migliano et al., 2007; Pettay et al., 2005), the observation that resource scarcity or slow resource intake leads to a longer lifetime (Kirkwood and Shanley, 2005; McCay et al., 1935; Wolf, 2006), or the finding that larger clutch size is correlated with larger juvenile mortality (Martin, 1987). The reason behind these trends in our model is the following: populations that consume less resources have a slower growth and a smaller rate of offspring production. Due to the slower growth, maturity sets in later. Due to the smaller number of offspring, there is a decreased density-dependent mortality and therefore a larger life expectancy. In our model, these correlations occur in stationary homogeneous populations, where all individuals of the same body mass are equivalent.

A close look at our simulation data reveals that the product of adult mortality and generation time is essentially independent of the parameter values, and has a value around 2.6. This means that adult mortality is constant if related to the time scale of reproduction, and we found that the probability that the parents are still alive when their offspring reach maturity is approximately 15%. The scaling of mortality with generation time in our model is similar to the scaling of senescence rates with generation time found in nature (Jones et al., 2008) (Senescence rates are determined by ranking on the fast-slow life-history continuum). This means that the effect of senescence on our model would be identical to the effect of a somewhat increased mortality of adults. Whether mortality of adults is age-dependent or not is irrelevant in stationary populations, what matters is the average adult mortality and its dependence on the generation time. Therefore, the effect of including senescence in our model would be a small shift of all curves, but not a change of the observed trends.

When an individual with restricted resource intake is placed in a well fed and fast growing population in our model, its mortality is the same as that of the other individuals of the same body mass, because mortality is determined by population density and body mass alone. In contrast, in laboratory populations individuals that obtain less food live longer than well-fed individuals, due to the physiological changes occurring in these individuals (Kirkwood and Shanley, 2005; McCay et al., 1935; Wolf, 2006). We suggest that this response to food shortage may ultimately be shaped by evolutionary history, since earlier ageing does not confer a substantial disadvantage in stationary well-fed populations, but in resource restricted populations. Therefore, a mutant aging earlier might establish in well-fed populations but not in populations restricted in resource supply during evolution.

When adult body mass is allowed to evolve, it increases without limits in this simple model—until the population crashes because the number of individuals has become so small that random fluctuations can cause its extinction. This finding demonstrates that Cope's rule can result from allometric scaling alone, without taking into account other advantages of large body mass. In our model, the three conditions mentioned by Kozłowski and Wiegert (1987) as promoting an increased body mass are satisfied: larger individuals are better competitors due to a slower metabolism, they have a larger reproductive rate and a smaller mortality than smaller individuals.

An important condition for the evolution towards larger body mass is that the population is stationary. If the population was growing exponentially, individuals with a smaller adult body mass, which produce offspring earlier, could multiply faster. Another important condition for the evolution towards larger body mass is that there are no causes of mortality that deviate from allometric scaling as is the case in non-stationary populations as those, e.g., exposed to harsh environments or facing pathogens in spatially structured populations (Kirchner and Roy, 1999; Nylin and Gotthard, 1998; Southwood, 1988). Consequently, in order to produce pygmies the model for example would need to include mortality which is independent of body mass. Alternatively, smaller adults may be obtained by including catastrophic events killing a large portion of the population and allowing subsequent population expansion where smaller body masses confer an advantage due to earlier onset of reproduction (Connell and Slatyer, 1977; Huston, 1994).

We also found that the offspring body mass evolves towards smaller size in the allometric model when adult body mass is kept fixed and offspring body mass is allowed to evolve. In our model, the offspring body mass is the body mass that the offspring reached when parents stopped nourishing them. Furthermore, we have assumed that the investment into offspring is twice the body mass of the offspring, but the results would be similar had we used a factor different from 2. The result of our model suggests that offspring body mass cannot be explained based on allometric scaling of mortality and metabolism alone. However, if we included the constraint that offspring body mass scales with the parent body mass to the power 0.75 (which would be realistic for species without parental care after hatching, such as reptiles), evolution to larger body size of parents and offspring did occur. Including such a relation into the model is simply done by requiring that the number of offspring produced per year is independent of body size. However, the weaning mass of offspring is known to be proportional to the body mass of the adults in many species, in particular in mammals (Peters, 1983), and this can only be explained by taking into account the many factors that affect infant mortality and the parental investment required to produce offspring of a given body mass. When we implemented this feature in our evolutionary simulations, we did not obtain an evolution towards larger body mass, showing again that causes of infant mortality that are unrelated to energy considerations must be a strong determinant of juvenile body mass.

An important cause of juvenile mortality not considered in our model is predation. The effect of predation on juveniles does not scale with body mass. Smaller prey are consumed by a larger number of predators and with increasing body mass prey move towards size refuges from predation of an increasing number of predators. This is most pronounced in aquatic systems where prey swallowing is limited by the gape width of fish predators (Christensen, 1996; Persson et al., 1996). In fact, there is evidence that, e.g., predation on fish eggs of different mass follows a hump shape curve (Paradis et al., 1996). On the other hand, trade-offs concerning predation success also limit adult body mass. The evolution towards lower body mass of offspring in our model therefore lacks realism. However, this is conform with our goal to explore the evolution of life history characteristics based on the metabolic theory only thereby uncovering its predictive power but also its limits. Recently, it has been shown that for understanding predator-prey body mass ratios foraging theory needs to be incorporated considering that successful prey capture by predators changes with predator body mass (Brose et al., 2006, 2008). Rather than by a power law function, as predicted by the metabolic theory (Brown et al., 2004), energy flux in predator-prey interactions peaks at intermediate body mass of the predator as the escape efficiency of prey increases beyond a certain threshold of predator body mass (Vucic-Pestic et al., 2010). The

evolution towards higher body mass of adult body mass in our model apparently lacks realism as it is based solely on metabolic theory and ignores foraging theory. Again, it was not our intention to incorporate realism in our models but rather to explore the strengths and limits of metabolic theory in predicting life history characteristics. In the evolution of offspring body mass limitations are most obvious and call for the integration of foraging theory as in models on predator–prey body mass ratios in general.

To conclude, we presented a life history model solely taking into account resource use and metabolic scaling. Despite its simplicity, this model reproduces fundamental life history characteristics of species, such as number of offspring and longevity, which are in large consistent with nature. This was achieved by combining metabolic theory with consumer resource dynamics in stationary populations. The results reflect that resources do not only play a fundamental role in ecological processes, i.e., on short time scales, but also at shaping life histories, i.e., on evolutionary time scales. As the ingredients of the model are very fundamental and apply to any organisms consuming resources, consumer–resource dynamics combined with metabolic theory provide a null model for evaluating the role of other factors, such as predation, environmental stochasticity and spatial or temporal resource distribution, and for the evolution of life history characteristics in future studies.

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