

Life-history evolution under a production constraint

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The recently formulated metabolic theory of ecology has profound implications for the evolution of life histories. Metabolic rate constrains the scaling of production with body mass, so that larger organisms have lower rates of production on a mass-specific basis than smaller ones. Here, we explore the implications of this constraint for life-history evolution. We show that for a range of very simple life histories, Darwinian fitness is equal to birth rate minus death rate. So, natural selection maximizes birth and production rates and minimizes death rates. This implies that decreased body size will generally be favored because it increases production, so long as mortality is unaffected. Alternatively, increased body size will be favored only if it decreases mortality or enhances reproductive success sufficiently to override the preexisting production constraint. Adaptations that may favor evolution of larger size include niche shifts that decrease mortality by escaping predation or that increase fecundity by exploiting new abundant food sources. These principles can be generalized to better understand the intimate relationship between the genetic currency of evolution and the metabolic currency of ecology.

allometry | life-history theory | metabolic ecology

The recently formulated metabolic theory of ecology (1) predicts that many attributes of individuals, populations, communities, and ecosystems should be relatively straightforward consequences of the metabolic processes of the relevant organisms. Specifically, many rate processes should scale with body size and temperature in the same way as mass-specific metabolic rate:

$$R = R_0 M^{-1/4} e^{-E/kT}, \quad [1]$$

where R is the rate of some biological process, R_0 is a normalization constant, the $M^{-1/4}$ term gives the power-function dependence on body mass M , and the $e^{-E/kT}$ term or Boltzmann factor gives the exponential temperature dependence in terms of an “activation energy”, E , Boltzmann’s constant, k , and temperature, T , in Kelvin. This very general relationship holds both within and between species. It can be applied to the rate of production of biomass per unit mass, which is predicted to scale as $M^{-1/4}$ (e.g., refs. 2–4). If we ignore temperature or assume for the moment that it does not vary, the Boltzmann factor is a constant. Then, taking logarithms of Eq. 1 we have

$$\begin{aligned} \log(\text{mass-specific production rate}) \\ = \text{constant} - \frac{1}{4} \log(\text{body mass}). \end{aligned} \quad [2]$$

This prediction appears to be generally supported, although for some traits, the empirically measured scaling exponents appear to deviate from the predicted $-1/4$, sometimes being closer to $-1/3$ (e.g., ref. 5). For our argument here, however, the exact value of the exponent is not an issue; what is important is that it is negative. The consequence of the negative exponent is that in a log–log plot, mass-specific production rate is a declining straight-line function of body mass, as shown schematically by the solid black line in Fig. 2A. We take this to be a fundamental

causal constraint limiting life-history options as suggested by metabolic theory. The result is that smaller organisms have more resources, relative to body mass, to allocate to reproduction, so they produce new biomass, individuals, and genes at a faster rate. How does this fundamental feature of allometric production scaling affect life-history evolution?

To model analytically the effect of a production constraint on life history, we make the simplifying assumption that offspring are born at adult size. This “thought experiment” allows us to focus on the effects of body mass on production while avoiding complications arising from differences in growth and mortality between juveniles and adults. Unicells that reproduce by binary fission are examples of organisms in which all production is reproductive.

We first review the relevant parts of evolutionary life-history theory and show that life-history traits are selected to increase birth rates and to decrease death rates. This implies that reduced body size will generally be favored, because it increases production and birth rate if mortality is unaffected. Alternatively, increased body size may be favored, but only if it decreases mortality or enhances reproductive success sufficiently to override the preexisting production constraint.

Review of Evolutionary Life-History Theory

Evolutionary life-history theory is used to calculate how life histories evolve under the action of natural selection and to identify “optimal strategies” or predicted outcomes. Here, we use the theory to show formally that in the absence of age dependence, the optimal life history is the one with the highest value of birth rate minus death rate.

As with many other mathematical theories in biology, the foundations of the theory of life-history evolution are due to R. A. Fisher (6). The theory was developed principally in the 1970s and 1980s, with important contributions made in refs. 7–9, and now has wide application. Reviews include refs. 10–13. The present approach is based on refs. 7 and 14 and ref. 10.

The theory of life-history evolution is based on the postulate that genes affect the “vital rates” or *per capita* birth and death rates. In practice, vital rates also depend on the environments in which the organisms occur, and population density affects these environmental conditions. However, our treatment here assumes that environmental effects can be ignored by calculating vital rates averaged over all environments where the organisms occur (7, 14).

Given the postulate that genes affect vital rates, life-history theory calculates the rate at which particular genes with specified effects increase or decrease in the population (14). We use the terms gene and allele synonymously. Increase in frequency of one allele requires decrease in the frequencies of alternative allele(s). This is where the population-genetic concept of relative

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fitness, which calculates the relative rates of increase, can be helpful. For present purposes, however, it is not necessary to calculate relative fitness. Another approach is to use lifetime reproductive success as a fitness measure, but this lacks the generality of the method adopted here and can give misleading results (for further discussion, see the supporting information, which is published on the PNAS web site).

In the next section, we show that birth rate minus death rate is an adequate fitness measure for our purposes. This measure has been suggested previously (e.g., ref. 12), but here we show how it is derived from population genetics. Our derivation is based on a key result from life-history theory, that natural selection favors the gene that has the highest population growth rate. This is true so long as we assume, as we do here, that there is no overdominance, underdominance, or frequency-dependent selection (14).

Modeling Life-History Evolution Under Natural Selection

We now calculate the rate of increase of a gene that affects vital rates. Because these rates are specified, the life history of the individual bearing that gene is known, and the “population growth rate” of the gene can be calculated from this life history. This gives the allele-specific growth rate. We refer to this quantity as the *fitness* of the allele. Because we are calculating the growth rate of a population—here of alleles—there is an exact analogy with the method used in population dynamics to calculate the population growth rate of a population of individual organisms.

The simplest of all life-history models is a continuous time model in which vital rates do not change with age. Suppose organisms carrying the allele reproduce at instantaneous rate $2b$ per unit time and die at instantaneous rate d per unit time. (The model is valid for both sexual and asexual reproduction but is here applied to sexual reproduction, and hence to simplify notation we use $2b$ rather than b as instantaneous birth rate per unit time. In a sexually reproducing population, the chance of a given allele being copied into a given offspring is $1/2$, and so if organisms reproduce at rate $2b$, the allele is copied into offspring at rate b .) If the number of alleles in the population is N , then the number of copies gained through birth in a small time unit dt is $Nb dt$, and the number of copies lost through death is $Nd dt$. Thus, the net per copy growth rate of the population of alleles, F , is

$$F = \frac{Nb dt - Nd dt}{N dt} = b - d, \quad [3]$$

i.e., $F = b - d$.

To show the links with traditional life-history theory, we now derive Eq. 3 from a discrete time model, as follows. Suppose for simplicity that it takes some average time τ for carriers of the allele to acquire sufficient resources to produce n offspring, each of adult size at birth. The life history can be depicted schematically as shown in Fig. 1.

Again we assume that the death rate d is constant throughout life, so the chances of surviving the time interval τ , which we write as S , are $\exp(-d\tau)$. The fitness of the allele can now be calculated by using the Euler–Lotka equation (see, e.g., ref. 14), which for this life history takes the form

$$1 = \frac{1}{2} (S n \exp(-F\tau) + S^2 n \exp(-2F\tau) + S^3 n \exp(-3F\tau) + S^4 n \exp(-4F\tau) + \dots) \quad [4]$$

This is a geometric series that can be summed to give

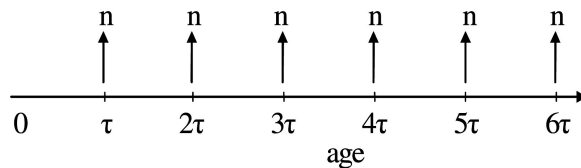


Fig. 1. Schematic depiction of the simple life history analyzed in the text. Age is shown on the x axis; thus, carriers of the allele reproduce at ages τ , 2τ , 3τ , \dots , producing n offspring each time they breed. Any given copy of the gene is copied into half the offspring on average. Although not discussed here, it is easy to see how this life history can be generalized, by introducing age dependence in fecundity and variation in the periods between reproductive events (7, 10, 14).

$$1 = \frac{\frac{1}{2} S n \exp(-F\tau)}{1 - S \exp(-F\tau)},$$

and rearranging

$$1 = \frac{1}{2} S n \exp(-F\tau) + S \exp(-F\tau) \quad [5]$$

and substituting $S = \exp(-d\tau)$, we obtain

$$1 = \left(\frac{1}{2} n + 1\right) \exp((-F - d)\tau) \quad [6]$$

and rearranging

$$F = \frac{1}{\tau} \log_e \left(\frac{1}{2} n + 1\right) - d. \quad [7]$$

Now, $\frac{1}{\tau} \log_e(\frac{1}{2} n + 1)$ is equal to the instantaneous birth rate, b . To see this, note that the “instantaneous birth rate” is an abstraction from the simpler continuous reproduction model considered above, in which population size grows exponentially at rate b if there are no deaths. So, for each initial allele, after time τ there are $\exp(b\tau)$ alleles. This is equal to $\frac{1}{2} n + 1$ because the initial allele is still present at time τ , but it is also then copied into half of the n offspring, as in Fig. 1. Taking logs we have that $b = \frac{1}{\tau} \log_e(\frac{1}{2} n + 1)$. This demonstrates the equivalence of Eqs. 3 and 7.

So, in this section we have shown that for the very simplest life-history models, fitness is given by birth rate minus death rate, i.e., $F = b - d$ (Eq. 3). Furthermore, since birth rate equals production rate in these models, it follows that alleles and the traits that they encode will be selected and increase in frequency in the population if they increase production or decrease death rate.

Ecological Compensation for Evolutionary Change

In the microevolutionary process, selectively favored alleles will increase in frequency in the population and, because $F > 0$, individuals carrying these alleles will have a population growth rate greater than zero. However, this situation cannot persist over the long term, because population size cannot increase indefinitely. Eventually, other factors must return F to zero. How does this come about?

In population dynamics, population increases from low density are eventually contained because the vital rates are ultimately density-dependent: death rates increase and/or birth rates decrease as the number of individuals increases. In many animals, this density dependence is the result of resource limitation, either directly, or indirectly through territoriality, dispersal, and spacing behavior (15, 16). Such adjustments in vital rates must also occur after microevolutionary changes, to return F to zero,

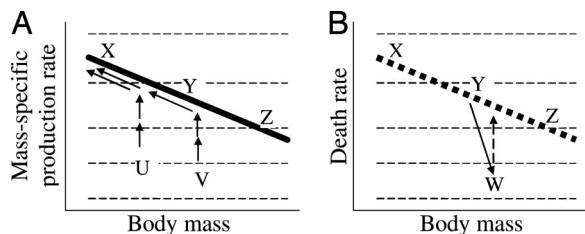


Fig. 2. Schematic depiction of the operation of natural selection on organisms subject to allometric constraints. In *A* the constraint is on production rate (thick line). When plotted on logarithmic axes, production rate scales as a straight line function of body mass. Fitness contours are horizontal dashed lines, with fitness increasing higher up the diagram. Organisms at positions U or V are expected to follow the trajectories indicated by the arrows. (*B*) To depict how natural selection can operate on death rate, the production constraint has been replaced by a mortality constraint (thick dotted line). Fitness contours are again horizontal dashed lines, but with fitness now increasing lower down the diagram.

and this has been termed the “principle of ecological compensation” (17).

Here, for simplicity, we restrict attention to just two vital rates, birth rate and death rate, and assume that neither varies with age. Since F is then equal to birth rate minus death rate, the principle of ecological compensation implies that evolutionary improvement in one of these quantities must eventually be compensated by adjustment of the other. Thus, after compensation, $F = b - d = 0$, and therefore $b = d$.

If Death Rate Is Constant then Small Animals Do Best

In this section, we apply the above principles of life-history evolution to organisms operating under an allometric production constraint. We begin with a very simple demonstration of the advantages of small body size. We first assume that ecological compensation does not occur, but then consider its effects.

Above, we showed that, other things being equal, fitness is maximized by increasing production rate and decreasing death rate. If death rate is constant, however, fitness depends only on production rate. This situation is depicted schematically in Fig. 2*A*, in which the horizontal dashed lines represent fitness contours, i.e., lines connecting points of equal fitness. Thus, fitness increases upwards, so there is selection for increased production rate, but this is subject to the allometric production constraint illustrated by the thick line in Fig. 2*A*. Since points on the same horizontal line have the same fitness, the strategy labeled X has higher fitness than Y, which in turn has higher fitness than Z. This means that natural selection will always favor smaller organisms over larger ones, other things (death rate) being equal. The reason is that smaller organisms have a higher production rate per unit body mass, so their rate of population growth is faster.

As a numerical illustration, compare the doubling rates of bacteria and elephants (18). Under ideal conditions, some bacteria reproduce by fission every 20 min. If this continued with no mortality for 36 h, there would be 2^{108} bacteria, enough to cover the entire surface of the earth in a layer a foot deep. By contrast, it takes years for a female elephant to produce two calves. Clearly the bacteria have a far higher production rate per unit body mass than the elephants. If bacteria and elephants had the same death rates and competed for the same resources, bacteria would very quickly win and take over the world.

Now consider the evolution of organisms arbitrarily started at positions U or V (Fig. 2*A*). Natural selection will favor mutations that increase production rate (vertical arrows), but eventually the production constraint is reached, and thereafter evolution is only possible by following the constraint up and to the left.

What would happen if the production constraint itself could evolve? It is easy to see from Fig. 2*A* that natural selection tends to move it upwards. So, if the smallest animals are unable to increase their production rates, i.e., the constraint is fixed at its left hand end, then the effect of selection would be to move the constraint toward the horizontal, reducing the, e.g., $1/4$ power scaling toward zero. That this has not happened is circumstantial evidence that the allometric scaling law is indeed a powerful constraint.

Fig. 2*A* suggests there is always selection for smaller body size, other things (death rates) being equal. In reality, however, death rates may also vary with body size. One reason is the process of ecological compensation discussed above. After compensation, death rates must equal birth rates and have the same scaling with body mass, e.g., as $M^{-1/4}$. This is shown by the thick dotted line in Fig. 2*B*. In Fig. 2*B*, the fitness contours are for organisms all having the same birth rate (in contrast to Fig. 2*A*, where all have the same death rate). The net result is that if birth and death rates vary with body mass as in Figs. 2*A* and *B*, respectively, then fitness is identical for organisms along the constraint line. So strategies X, Y, and Z in Fig. 2 can coexist if they have equal and zero fitness long-term, with birth rate matching death rate in each case.

Evolution of Larger Body Size

If smaller is better (higher fitness), why has natural selection resulted in the evolution of larger-sized organisms [e.g., Cope’s rule in mammals (19)]. Suppose a mutation for larger body size occurs in a community in which the species with the largest body size is at position Y in Fig. 2*B*. Before the mutation all organisms were subject to the production and death rate constraints shown by the thick lines in Fig. 2*A* and *B*. If the mutation were subject to these same constraints, then the population would not grow, since birth rate would still equal death rate even though their values were different from before. Although such a mutation might increase through genetic drift, it would not have a selective advantage. Clearly, however, there must be instances when larger individuals have higher fitness.

Indeed, selective advantages of mutations conferring larger body size are easy to imagine. For example, increased size may provide access to new niches and lifestyles with lower death rates and/or increased production rates. There are several possibilities:

- (i) A larger body might entail morphological and/or physiological innovations that circumvent preexisting constraints and make available new food resources. An example would be the evolution of novel digestive systems with large guts and microbial symbionts that allow rodents, lagomorphs, and ruminants to obtain energy and nutrition and attain high rates of production by feeding on vegetation that could not sustain their flesh-eating ancestors. Mammalian herbivores make a living by consuming relatively large quantities of relatively low-quality food. Larger size is generally advantageous, because the larger capacity of the gut and slower passage time allow microbes to break down otherwise indigestible plant materials and contribute importantly to host nutrition and productivity (e.g., refs. 20–22). Somewhat similarly, larger size in carnivorous animals might allow them to capture and consume larger prey, thereby increasing the variety and resource content of their food supply.
- (ii) A larger body might confer greater physiological homeostasis that would allow invasion of new habitats or resistance to abiotic environmental change. Increased body size of endothermic birds and mammals typically confers increased tolerance of cold stress and/or starvation. In this case, an increase in body size could reduce mortality so as to more than compensate for the reduced production due to the

allometric constraint. Examples are the rapid changes in body sizes of woodrats (*Neotoma* spp.) in response to changes in environmental temperature over the last 30,000 years. Body size can be estimated from the size of fecal pellets preserved in fossil middens, which can be carbon dated, and temperature can be estimated independently by plant species composition and other proxies. Local woodrat populations acted like “biological thermometers,” increasing or decreasing rapidly in body size during each episode of cooling or warming, respectively, in accord with Bergmann’s rule (23–25).

- (iii) A larger body might reduce predation pressure. For example, elephants experience much lower rates of predation than smaller coexisting species of herbivorous mammals. This mechanism is consistent with the rapid dwarfing that occurred repeatedly by convergent evolution when elephant populations were isolated on islands without large predators (26, 27). With large size no longer being so advantageous, the populations responded rapidly to selection to increase production rate and speed up the life history. These size changes, as much as two orders of magnitude in mass in a few thousand years, are among the highest known rates of morphological evolution.
- (iv) A larger body might confer an advantage in interference or contest competition. We suggest two possibilities. First, larger size may confer an ecological advantage, because it allows preemption of concentrated food resources or facilitates territorial defense. For, example Nee *et al.* (28) found that when congeneric bird species coexisted in the same habitat, the larger was usually more abundant, and they attributed this to dominance in interference competition. Second, large size may confer an advantage in competition for mates. Indeed, sexual size dimorphism, with males larger than females, is a frequent outcome of sexual selection (29, 30). It is easy to imagine how sexual selection for larger size in males could lead to evolution of larger size in females as a correlated trait. This has likely played a major role in certain lineages of mammals, where there have been evolutionary trends for the body size to increase [Cope’s rule (19)] and with increasing overall size for the ratio of male to female size also to increase [Rensch’s rule (29, 31)]. Eventually, species of very large size may go extinct, because their lower reproductive capacities and smaller populations increase their vulnerability to environmental change.
- (v) A larger body size might actually confer a higher mass-specific production rate on the very smallest representatives of certain taxa and lineages. Mass-specific metabolic rates also scale as $M^{-1/4}$. This means that small animals must consume relatively large quantities of high-quality food simply to maintain their biomass. They may have problems acquiring and assimilating additional resources to allocate to production. There is some evidence that very small animals may have production rates below the general $M^{-1/4}$ constraint line predicted by allometric metabolic theory and used in the present models (27, 32). Selection to increase production rates potentially offers a general explanation for Foster’s island rule, the tendency on isolated islands for very small organisms to evolve giant forms while their larger relatives evolve dwarf forms (33, 34).
- (vi) A larger body may not be advantageous *per se* but may be produced in response to environmental conditions and persist if not disadvantageous. Perhaps the best example is provided by the cases of Bergmann’s rule in ectotherms. In many taxa of terrestrial and aquatic vertebrates and invertebrates, populations in colder environments have larger body sizes, e.g., refs. 35–37. A likely explanation is that this pattern is produced initially as a direct phenotypic response

rather than a genetic adaptation to altered temperature. When individuals are reared in colder environments, ontogenetic development time is increased, and body size at maturity is larger (38). Such purely phenotypic changes may persist over ecological and evolutionary time so long as they are not disadvantageous. One reason why this might be so is simply that the vast majority of organisms are ectothermic, and their rates of metabolism and production all have approximately the same temperature dependence. Consequently, even though this mechanism causes a population of one species in a colder environment to have a lower rate of production, and correspondingly lower birth and death rates, it may not suffer any disadvantage if its coexisting competitors, predators, parasites, and diseases also have correspondingly lower vital rates. Of course, the larger body size in a colder environment may confer some advantage, in which case the phenotypic change would be acted upon and reinforced by natural selection.

In all of the above examples except the last, a mutant allele or new trait that has the effect of increasing body mass would enjoy a substantial advantage through lowered death rates (*ii/iii*) and/or increased production rates (*i/iv/v*). The outcome is that birth rate initially exceeds the death rate, and the allele and the population starts to increase. In the longer term, but still on an ecological time scale, density-dependent processes achieve a new balance between death rate and birth rate, and the allele and the population stop increasing, but the population retains a higher body mass and perhaps also permanently lowered mortality and increased production rates. The case where the new trait that increases body size affects the death rate is diagrammed in Fig. 2B; so here the death rate decreases initially (solid arrow) and then subsequently through the process of ecological compensation increases to equal the birth rate (dashed arrow). By their very nature, changes in body size and life history due to this process may be hard to observe. Times between advantageous mutations that affect body size may be long, and ecological compensation may be fast relative to evolutionary time. Nevertheless, the cases mentioned above are only a few of the many examples where investigators have shown linkages between the rates of production, the vital rates of life histories, and the ecological conditions that determine the direction and magnitude of life history and body size evolution.

Concluding Remarks

Our theoretical treatment here of the relationship between production rate, life history, and genetics is very simple. This is done to provide a mathematically explicit and rigorous analysis of the fundamental components and processes. We have deliberately made simplifying assumptions about the allometry of production, resource allocation to growth and reproduction, age structure and the schedule of the life history, and the genetic basis of the vital rates. For instance, effects that we identify as acting on female production and reproduction may in more realistic treatments act on juvenile mortality, growth rate, or age at first reproduction, or they may act primarily on male traits. More realistic treatments might model explicitly how metabolic resources allocated to maintenance and predator-defense could result in lower death rates. Despite these simplifications, the theory captures the fundamental principles, processes, and variables that link the vital rates and other features of the life history to the metabolic allometric constraints on production rate. The vast majority of biological processes, including the rates of growth and reproduction and the times of life-history stages, such as gestation, lactation, and first reproduction, scale with quarter powers of body mass as predicted by the metabolic theory of ecology (e.g., refs. 39 and 40). So the framework developed above should apply broadly to animals of diverse

taxonomic and functional groups. We are well aware of the simplifications we have imposed for the sake of clarity and brevity, but we are confident that the results will prove robust and general.

So, what do metabolic and life-history theory tell us about body size evolution? Putting the two theories together leads to several strong predictions. (i) Natural selection favors any allele, trait, or individual organism that increases production rate, which is equivalent to increasing the growth rate of the allele, trait, or type of organism within a population. (ii) Such increases of alleles, traits, and individuals will only be temporary, however, because density dependence and the principle of ecological compensation eventually will cause death rates to equal birth rates and prevent further growth. (iii) Everything else being equal, and provided there exist sufficient resources to allow high rates of production, natural selection favors smaller body size, because the allometric production constraint causes smaller organisms to have higher production rates and hence higher fitness. (iv) Evolution of larger size is possible only when some associated advantage increases birth rate or decreases death rate enough to override the preexisting allometric production constraint. (v) The constraint is most likely to be overridden and larger body size to evolve when a population increases its production or lowers its mortality by adopting a new niche or lifestyle.

So, what is maximized by natural selection? Putting life-history theory and metabolic theory together in this way offers insights into some of the most fundamental processes of organismal biology, evolution, and ecology. In particular, it clarifies the intimate relationship between the genetic currency of evolution and the metabolic currency of ecology. Natural selection maximizes fitness, which evolutionary biologists have traditionally measured in terms of the differential growth rates of alleles or differential rates of production of descendent offspring with genes for a particular trait. But the offspring that bear those genes are made of flesh and blood. Their production is fuelled by metabolism, and the rate of production obeys the constraints of allometry, temperature dependence, stoichiometry, and other processes that determine metabolic rate. In this article, we elaborate conventional life-history theory to clarify the relationship between production rate and the other vital rates. Then, we use the allometric scaling of production rate to analyze the evolution of body size. We show how this constrains the allometries of birth and death rates, and how these in turn constrain the evolution of smaller or larger size.

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