A General Model for the Scaling of Offspring Size and Adult Size

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abstract: Understanding evolutionary coordination among different life-history traits is a key challenge for ecology and evolution. Here we develop a general quantitative model predicting how offspring size should scale with adult size by combining a simple model for life-history evolution with a frequency-dependent survivorship model. The key innovation is that larger offspring are afforded three different advantages during ontogeny: higher survivorship per time, a shortened juvenile phase, and advantage during size-competitive growth. In this model, it turns out that size-asymmetric advantage during competition is the factor driving evolution toward larger offspring sizes. For simplified and limiting cases, the model is shown to produce the same predictions as the previously existing theory on which it is founded. The explicit treatment of different survival advantages has biologically important new effects, mainly through an interaction between total maternal investment in reproduction and the duration of competitive growth. This goes on to explain alternative allometries between log offspring size and log adult size, as observed in mammals (slope = 0.95) and plants (slope = 0.54). Further, it suggests how these differences relate quantitatively to specific biological processes during recruitment. In these ways, the model generalizes across previous theory and provides explanations for some differences between major taxa.

Keywords: allometry, life history, offspring size, seed size, Smith-Fretwell.

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Offspring face many hazards in reaching maturity. Increased provisioning from the mother (e.g., larger seeds for seed plants, eggs for invertebrates, or size at weaning for mammals) is known to increase offspring survival (Fox and Czesak 2000; Janzen et al. 2000; Moles and Westoby 2004*a*), but this advantage must come at the expense of the number of offspring that can be provisioned for a given amount of energy (Henery and Westoby 2001; Charnov and Ernest 2006). This trade-off between offspring number and survival drives the Smith-Fretwell model (SFM) for the optimization of offspring size (Smith and Fretwell 1974).

The SFM forms a foundation for theories of offspring provisioning across a variety of taxa (e.g., Fox and Czesak 2000; Coomes and Grubb 2003). Yet, a persistent difficulty is that the basic SFM does not account for the wide range of offspring sizes realized by taxa with similar ecologies and recruitment dynamics. For example, across coexisting plant species, seed size spans five to six orders of magnitude (Leishman et al. 2000). A development that seemed promising in the plant literature was to let seed and seedling size decide the outcome of competition to occupy vacant sites within vegetation. This generated frequency-dependent seedling survival, which resulted in a range of coexisting seed sizes within species (Geritz et al. 1988, 1999; Geritz 1995), but similar dynamics might also account for the wide range of seed sizes observed across species (Haig 1996; Rees and Westoby 1997). However, there now appear to be several problems with the cross-species extension. Empirical data indicate that the benefit in juvenile mortality rate for larger-seeded species is insufficient to offset the costs of producing larger seeds (Coomes and Grubb 2003; Moles and Westoby 2004*b*; Eriksson 2005). Also, species that as adults are larger or live longer and that consequently devote more total energy to reproduction can potentially offset the costs of producing larger seeds (Moles et al. 2004). These developments indicate that the SFM needs to be enhanced to account for variation in adult size and that survival needs to be considered across the entire period to maturity (Moles and Westoby 2006).

In mammals, offspring size spans five to six orders of

magnitude and is tightly coordinated with adult size (Blueweiss et al. 1978). Existing theory from Charnov (1993) suggests that this coordination arises through the influence of time to adulthood on the SFM: large adults have large offspring to offset the low survivorship that would otherwise be a consequence of longer juvenile periods. Charnov's (1993) model predicts a scaling slope of 1.0 between offspring size and adult size, making the ratio of offspring : adult size a life-history invariant. Recognizing that Charnov's model predicts coordination between offspring and adult size, it has been suggested that Charnov's treatment might also apply to plants (Moles et al. 2005*a*, 2005*b*). However, Charnov's model, as it stands, is unable to explain relationships that show scaling slopes different from 1.0. Although mammal data at the time Charnov (1993) published seemed consistent with a slope of 1.0, subsequent compilations show that the slope is slightly but significantly shallower (Purvis and Harvey 1995; Moles et al. 2005*b*). In plants, the scaling slope is shallower still (Moles et al. 2005*b*), with similar patterns recognized for other vertebrate taxa (Blueweiss et al. 1978; Lloyd 1987; Duarte and Alcaraz 1989). Thus, allometric, not isometric, relationships between offspring and adult size seem to be the rule rather than the exception. In addition, there are large differences in the intercepts of these relationships among taxa. For example, plants, fish, and reptiles have lower offspring mass for a given adult mass than do mammals (Blueweiss et al. 1978). Differences in either slope or intercept suggest fundamental differences among taxa in the allocation of energy for reproduction or in the efficiency of energy transfer among generations. Understanding these differences goes to the heart of evolutionary ecology.

Here we propose a general model for offspring size in relation to adult size, and we relate the predicted allometry to the ecology of recruitment. Elements are married together from different existing models. Parental fitness is maximized, offspring survival is frequency dependent, and predictions about trait coordination are couched as allometric scaling relations. The key innovations are explicit and separate treatments of three different mortality processes, including density-independent and density-dependent phases. The model proves capable of accounting for the scaling relationships between log offspring size and log adult size in both mammals and plants and shows how different possible slopes and intercepts relate quantitatively to specific biological processes during recruitment.

The Model

The Basic Reproduction Ratio as a Foundation for Life-History Evolution

As a starting point, we adopt Charnov's (1991, 1993) formulation for calculating the basic reproduction ratio R, the number of offspring reaching maturity from a single parent. The ratio R is expressed as the product of the length of reproductive period E (years), average annual fecundity during adulthood f (offspring adult^{-1} year^{-1}), and survival to adulthood for each offspring S_α (0–1). Annual fecundity f can be further partitioned into total mass devoted to offspring production A (kg adult $^{-1}$ year^{-1}) divided by the average mass per offspring W_0 (kg). The mass W_0 is calculated as the size at which offspring cease receiving resources from the mother rather than size at birth (i.e., size at weaning for mammals and seed size for plants), thereby accounting for total maternal investment in offspring. We will describe how S_α for a rare mutant individual with offspring size $W_{0,\mathrm{m}}$ depends explicitly on the size $W_{0,\mathrm{r}}$ of the prevalent resident strategy. Consequently, fitness for the mutant is expressed as a function of both mutant and resident strategy:

$$R(W_{0,\mathrm{m}}, W_{0,\mathrm{r}}) = \frac{AE}{W_{0,\mathrm{m}}} S_\alpha(W_{0,\mathrm{m}}, W_{0,\mathrm{r}}). \qquad (1)$$

Equation (1) is similar to Smith and Fretwell's (1974) formulation for parental fitness per reproductive episode (given by $(A/W_{0,\mathrm{m}})S_\alpha(W_{0,\mathrm{m}}, W_{0,\mathrm{r}})$), only expressed now as fitness integrated across the lifetime of the parent. Multiplication by E on the right-hand side of equation (1) shows how lifetime fitness can be considered the result of E separate reproductive episodes, where $S_\alpha(W_{0,\mathrm{m}}, W_{0,\mathrm{r}})$ is the probability of survival for a single mutant offspring during a single episode.

A further difference between equation (1) and the models of Charnov and of Smith and Fretwell is that mutant fitness is explicitly frequency dependent. There has been considerable debate in the evolutionary literature regarding the appropriateness of R as a measure of fitness (e.g., Stearns 1992; Brommer 2000); however, most concerns dissipate when density dependence and frequency dependence are included in the model (Brommer 2000). Following standard conventions in adaptive dynamics (Dieckmann and Law 1996; Dieckmann 1997; Geritz et al. 1998), we will assume timescale separation between demographic and evolutionary processes (mutation-limited evolution) so that residents are always demographically stable. Under these circumstances, evolutionary predictions obtained when using the basic reproduction ratio as a measure of fitness are equivalent to those obtained using the per capita rate of increase because the points of demographic and evolutionary stability are equivalent (Mylius and Diekmann 1995).

A Frequency-Dependent Survivorship Model

Here we develop a mechanistic model describing the survival from birth to maturity for offspring of size $W_{0,\mathrm{m}}$ growing in a cohort dominated by the competing resident strategy $W_{0,\mathrm{r}}$. As did Geritz et al. (1999), we assume an initial phase of frequency-independent survival, during which time mortality rates may be influenced by offspring size but are unaffected by the strategies of competitors, followed by a period of competitive ("self-") thinning, during which time mortality depends on the abundance and strategies of competitors. Self-thinning is mortality driven by the growth of crowded individuals (reviews in Harper 1977; Westoby 1984): as individuals increase in size, per capita rates of resource use also increase, so for a limited amount of available resources, additional growth increments must be accompanied by a decrease in abundance. Mortality typically falls on smaller individuals, while survivors increase in mass. Consequently, an individual's size relative to its competitor's becomes a key indicator of survivorship during the competitive phase.

The self-thinning process drives decreasing population density as body size increases within a developing cohort. This inverse relationship has two main consequences in the survival model. First, it provides a feedback that regulates the population density of adult individuals and, consequently, the average survival to maturity for the cohort. We do not explicitly track the population density of adult individuals; rather we assume that adult population density is always its maximum possible value, determined by available resources. Thus, for a population with adult life span E, a fraction E^{-1} of the resource supply becomes available to new recruits each year through the death of adults. The number of individuals reaching maturity from this cohort will then be limited, through resource supply, to replacing adult turnover. In this way, the average lifetime fitness of an individual with the resident strategy is fixed at $R(W_{0,\mathrm{r}}, W_{0,\mathrm{r}}) = 1$, so the fraction of resident offspring reaching maturity is

$$S_\alpha(W_{0,\mathrm{r}}, W_{0,\mathrm{r}}) = \frac{W_{0,\mathrm{r}}}{AE}. \tag{2}$$

Second, the inverse relationship determines both the rate of competitive thinning and the duration of the competitive phase, and as a result, the potential survival advantage to a mutant with an offspring size larger than that of the resident. This characteristic proves vital in generating different offspring sizes because it creates a feedback between parameters of the resident population (such as offspring and adult sizes and investment in reproduction) and model predictions.

We now incorporate these assumptions about the scaling of resource use in a model for offspring survival from independence to maturity (overview in fig. 1). For the resident population made up of adults having mass at maturity W_α and producing offspring with mass at independence $W_{0,\mathrm{r}}$, we distinguish three growth phases (establishment, density-independent, and competitive phases) during which log numbers surviving decline at rates p, q, and b, respectively, for each increment in log mass. Because the self-thinning process occurs in relation to mass gain rather than in relation to time, expressing survivorship in relation to mass gain is preferable to assuming first a survivorship per time and then, separately, a rate of mass gain per time.

Following an initial reduction in offspring numbers through dispersal by a factor $1 - s$, there may be an establishment phase during which offspring are supported by reserves obtained from the parent until their mass has increased D-fold. This phase incorporates the tendency for better-provisioned offspring to withstand various hazards during establishment (Moles and Westoby 2004*a*) but is present only in taxa that carry storage reserves from parents beyond independence (e.g., endosperm in plants). For the remaining taxa (e.g., mammals), $D = 1$. The mortality rate during establishment $-p$ is itself a function of offspring mass $p(W_0) = kW_0^{-1}$, where k is the offspring mass giving survival of D^{-1}. This function provides for survival through establishment to approach 0 when offspring mass is very small and to approach 1 when offspring mass is very large. During the second phase, mortality proceeds at a density-independent rate $-q$, set by external factors such as predation. These mortality factors are assumed to be independent of the initial size of the individual (and therefore similar for mutant and resident individuals). As individuals continue to grow, the total mass of the population and its resource use increase until a critical size $W_{T,\mathrm{r}}$, where total resource consumption matches supply; $W_{T,\mathrm{r}}$ thus marks the transition from density-independent growth to a phase of competitive thinning for resident cohorts. During the competitive phase, mortality proceeds at a rate $-b$. Values of 2/3 and 3/4 have been supported for b (Westoby 1984; Enquist et al. 1998). Combining these three phases of mortality (fig. 1), we find the proportion of the initial offspring density surviving to adulthood as

$$S_\alpha(W_{0,\mathrm{r}}, W_{0,\mathrm{r}}) = sD^{-p(W_{0,\mathrm{r}})}\left(\frac{W_{T,\mathrm{r}}}{DW_{0,\mathrm{r}}}\right)^{-q}\left(\frac{W_\alpha}{W_{T,\mathrm{r}}}\right)^{-b}. \tag{3}$$

Combining equations (2) and (3), we see how the mean mass at which the population begins size-competitive mortality adjusts in response to circumstances:

$$W_{T,\mathrm{r}} = \left(\frac{1}{sAE} W_{\alpha}^{b} W_{0,\mathrm{r}}^{1-q} D^{(k/W_{0,\mathrm{r}})-q}\right)^{1/(b-q)}. \quad (4)$$

Larger offspring size ($W_{0,\mathrm{r}}$), higher preestablishment or density-independent mortality (s or q), lower reproductive investment (A), or higher adult turnover (E^{-1}) causes an increase in W_T and, consequently, less competitive-induced mortality. Overall, this model is agreement with empirical data that demonstrate the self-thinning process within both animal and plant populations (Westoby 1984; Begon 1986; Lobón-Cerviá and Mortensen 2006; Coomes and Allen 2007), shifts in the position of the thinning boundary with site resource status (Westoby 1984; Deng et al. 2006), and also a two-phase thinning relationship, where an initial phase of growth is followed by a second phase with substantially steeper declines in density per-unit mass increment (Westoby 1984; Lobón-Cerviá and Mortensen 2006).

Consider now a rare mutant in the population with offspring size $W_{0,\mathrm{m}}$ larger than the resident strategy. The mutant (fig. 1, *gray line*) enjoys three benefits in survival over the residents (fig. 1, *black line*). First is the reduced mortality during the establishment phase. Second, if the size advantage at birth is maintained through ontogeny, then the mutant will be larger at the onset of competitive growth and consequently will experience a shorter duration of the competitive phase. Finally, because of its size advantage when entering the competitive phase, the mutant would also experience a survival advantage throughout the entire competitive phase. This will lead to a proportional reduction in general mortality rate during the competitive phase (b) by an amount $\alpha(x) = 2/(1 + x^{z})$, where $x = W_{T,\mathrm{m}}/W_{T,\mathrm{r}}$ is the relative size difference between the mutant and the resident at the onset of the competitive phase and z is the degree of competitive asymmetry. The survival function for the mutant is then given by

$$S_{\alpha}(W_{0,\mathrm{m}}, W_{0,\mathrm{r}}) = sD^{-(k/W_{0,\mathrm{m}})} \times \left(\frac{W_{T,\mathrm{m}}}{DW_{0,\mathrm{m}}}\right)^{-q} \left(\frac{W_{\alpha}}{W_{T,\mathrm{m}}}\right)^{-b\alpha(W_{T,\mathrm{m}}/W_{T,\mathrm{r}})} \quad (5)$$

Key to understanding the behavior of the mutant relative to that of the resident is the translation between time- and mass-based survival curves. Figure 1 is plotted in relation to mass because the point of onset for the competitive phase is determined more by the total mass of resident population (relative to resource supply) than by age. As individuals in the resident cohort grow larger, so does the total resource requirement for the cohort, pushing it closer to the self-thinning boundary. Because mutants are assumed to enter the population at a similar time point and possess growth rates similar to those of resident individuals, a mutant entering the population with a larger initial size should be larger than an average resident individual at any given time throughout ontogeny. Mutants are also assumed to be sufficiently rare that they do not influence resident dynamics, in which case the size at which mutants enter the competitive phase ($W_{T,\mathrm{m}}$) is determined by the size of residents at the onset of the competitive phase ($W_{T,\mathrm{r}}$) and the relative size advantage of the mutant over the residents at that time point. We assume that the relative size advantage at the onset of thinning can be related to the relative size advantage at birth via a function $\gamma(W_{0,\mathrm{m}}, W_{0,\mathrm{r}})$ that governs any increase or decrease in relative size advantage through ontogeny, so that

$$\frac{W_{T,\mathrm{m}}}{W_{T,\mathrm{r}}} = \gamma(W_{0,\mathrm{m}}, W_{0,\mathrm{r}}) \frac{W_{0,\mathrm{m}}}{W_{0,\mathrm{r}}}. \quad (6)$$

Substituting into equation (5) gives an expression for mutant survival,

$$S_{\alpha}(W_{0,\mathrm{m}}, W_{0,\mathrm{r}}) = sD^{-(k/W_{0,\mathrm{m}})} \left[\frac{W_{T,\mathrm{r}}}{DW_{0,\mathrm{r}}} \gamma(W_{0,\mathrm{m}}, W_{0,\mathrm{r}})\right]^{-q} \times \left[\frac{W_{\alpha} W_{0,\mathrm{r}}}{W_{T,\mathrm{r}} W_{0,\mathrm{m}} \gamma(W_{0,\mathrm{m}}, W_{0,\mathrm{r}})}\right]^{-b\alpha[\gamma(W_{0,\mathrm{m}}, W_{0,\mathrm{r}})(W_{0,\mathrm{m}}/W_{0,\mathrm{r}})]}. \quad (7)$$

In the first instance, we let $\gamma(W_{0,\mathrm{m}}, W_{0,\mathrm{r}}) = 1$; that is, we assume that the relative size advantage of a mutant at birth is maintained through ontogeny. Later, we relax this assumption and discuss the way this influences the model's predictions.

Evolutionary Predictions

For any combination of species and site characteristics, we can predict the long-term outcome of selection on offspring size using tools from game theory and adaptive dynamics (Dieckmann and Law 1996; Dieckmann 1997; Geritz et al. 1998). When we assume small mutations and asexual inheritance, the gradient of selection on an initially monomorphic resident population with strategy $W_{0,\mathrm{r}}$ is given by $G(W_{0,\mathrm{r}}) = (\partial/\partial W_{0,\mathrm{m}}) R(W_{0,\mathrm{m}}, W_{0,\mathrm{r}})|_{W_{0,\mathrm{m}} = W_{0,\mathrm{r}}}$ (Dieckmann and Law 1996; Geritz et al. 1998). Solving $G(W_{0,\mathrm{r}^*}) = 0$ for W_{0,r^*} gives the point where directional selection ceases. At this point, we are interested to know (1) whether W_{0,r^*} can be approached though a series of small mutations, each lying successively closer to W_{0,r^*}, in which case W_{0,r^*} can be said to be convergent stable (or an evolutionary attractor), and (2) whether W_{0,r^*} is located at a fitness maximum or a fitness minimum (i.e., whether

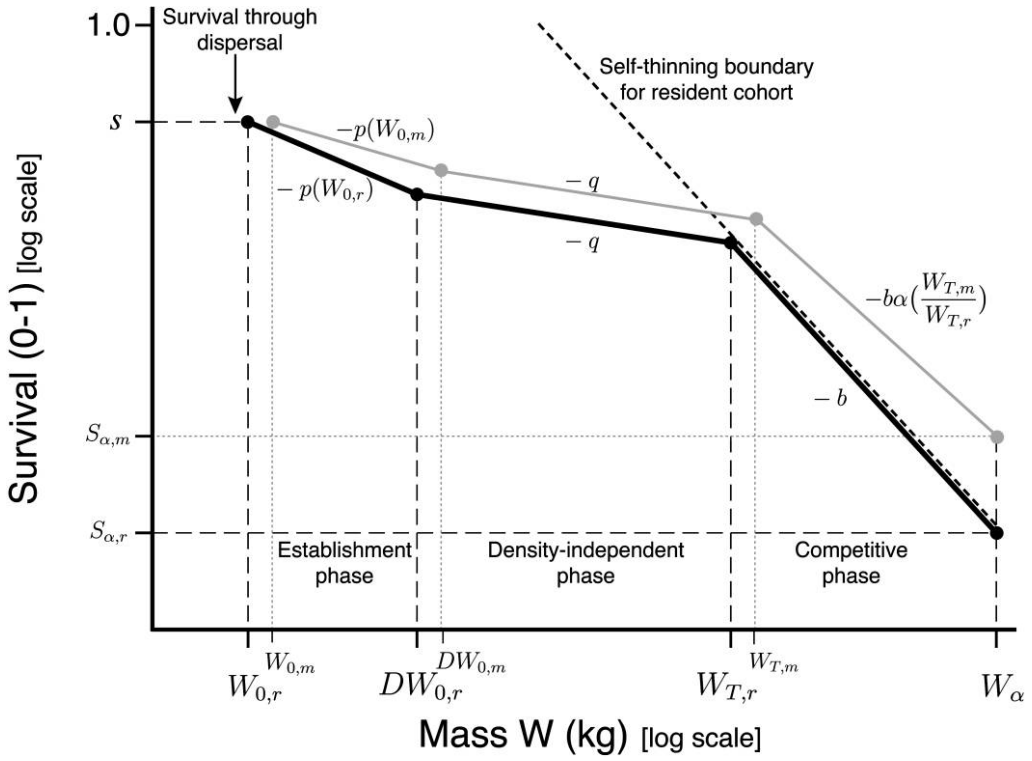

Figure 1: Survivorship of juveniles as they grow from mass at independence W_0 to mass on reaching adulthood W_α (both axes log scaled). Following an initial reduction of offspring numbers to s through preestablishment mortality, survivorship proceeds in relation to mass gain through three phases with slopes $-p$, $-q$, and $-b$. During the first phase (establishment), offspring are reliant on reserves obtained from the parent. Following establishment, survivorship proceeds at a density-independent rate $-q$ per increment of mean log mass. This phase continues until a self-thinning boundary is reached at mass W_T, after which survivorship proceeds along the boundary at $-b$, continuing until mass at adulthood W_α. Black lines indicate survivorship of the resident strategy $W_{0,r}$. Gray lines indicate the survivorship of the rare mutant strategy with larger mass at independence $W_{0,m}$ in a population dominated by the resident strategy.

it is immune to invasion by nearby strategies). If W_{0,r^*} is at a maximum, then it represents an evolutionarily stable strategy (ESS) and a possible endpoint of evolution. If W_{0,r^*} is at a minimum, the population may undergo evolutionary branching, leading to phenotypic diversification. Our model did not satisfy the criteria for evolutionary branching. Each combination of species and site characteristics was associated with a unique offspring size that was both convergent stable and resistant to invasion by all other strategies (i.e., a global ESS; app. A).

Data Sets and Analysis

To test the ability of the model to describe cross-species relationships between adult size and offspring size, we compiled data sets covering average dry mass of offspring and adults for a large number of plant and mammal species. Data on reproductive investment (A) and life span (E) are required as inputs to the model. Annual reproductive investment by a single mother A was calculated as the number of offspring reaching independence per year multiplied by average mass at independence per offspring (size at weaning in mammals and seed size in plants). Lifetime reproductive investment was calculated as A multiplied by E. As calculated, reproductive investment does not include accessory costs such as placenta (mammals) or dispersal structures (plants) or other costs related to securing a given reproductive investment (e.g., pollen, flowers, sperm, and courtship rituals).

Mammals

Mass data are from Ernest (2003). Annual reproductive investment by an individual over 1 year (kg individual^{-1} year^{-1}) was calculated as $A = y\Omega W_0 S_0$, where y is the litter frequency per year, Ω is the number of offspring per litter, and S_0 is preweaning survival. We were unable to obtain species-specific estimates of S_0, so a standard value of 0.5 was used, based on values of 0.45 for hares (O'Donoghue 1994) and 0.63 for 11 ungulate species (Gaillard et al.

2000). Fresh mass data were converted to dry mass by multiplying by $1 - 0.605$ (0.65 is the mean body water content measured in vitro for 11 mammals spread across the phylogeny; Wang et al. 1999). Average reproductive life span (years) was calculated from average adult mortality M, supplied by Purvis and Harvey (1995) as $E = 1/[1 - \exp(-M)]/12$. Although consistent with relationships, data from blue whales (*Balaenoptera musculus*) were excluded because they lay distant from the other data and would have had an unreasonably large influence on estimated regression parameters.

Plants

We supplemented previously compiled data on adult body size, seed size, and annual reproductive effort (Moles et al. 2004) with data from studies by Niklas and Enquist (2004) and Vile et al. (2006) and a few other studies (details available from the authors). All data are for individuals growing in field conditions. Annual seed production was estimated directly as kilograms of seed per individual per year (not including any accessory costs related to seed production). Where body size data were for aboveground mass only, we multiplied by 1.305 to account for missing root biomass, where 0.305 is the average root : shoot ratio across large data sets (Niklas 2004).

Statistical Analysis

Relationships between log-scaled variables were described using standardized major axis lines (Warton et al. 2006). These describe the best-fit scaling (or proportional) relationships between variables. Observed slopes were compared to hypothesized values b_H by testing for a significant correlation between fitted ($\log Y + b_H \times \log X$) and residual ($\log Y - b_H \times \log X$) data (Warton et al. 2006). All analyses were conducted using the SMATR library for R.

For mammals, the scaling constant in the relationship between W_α and annual reproductive investment was reduced by half in model runs to give the number of daughters produced per mother (Charnov 1991, 1993; Purvis and Harvey 1995). This adjustment was not required for plants, most of which are monoecious.

Other Parameters

The model requires several other parameters for which we have little data. Consequently, readers should beware that exact values predicted by the model may be modified when improved empirical estimates are available. However, we do not expect the main results to be altered because the qualitative behavior of the model is not influenced by choice of parameter values. The default parameters used were $q = 0.2$ for mortality rates in the density-independent phase, $b = 0.75$ for mortality rates in the competitive phase, $z = 3$ for the level of competitive asymmetry, $D = 2$ for the duration of the establishment phase (in plants), and $k = 10^{-8}$ kg for size of offspring having survival of 0.5 through the establishment phase.

Results and Discussion

The model predicts how offspring size should scale with other aspects of a species' life history, including adult size, reproductive life span, the amount of energy devoted to reproduction, and factors specific to sites or taxa, such as rates of density-independent and preestablishment mortality. There are general solutions for taxa with no establishment phase ($D = 1$), such as mammals, and taxa with an establishment phase ($D > 1$), such as plants (app. A). In both cases, offspring size is predicted to scale negatively with adult size to the power $-q/(1 - q)$ for a given reproductive investment and life span. The negative scaling with adult size is rarely seen in the real world because of cross correlations, but it demonstrates a real process: larger adult size has a cost through extending the length of the juvenile period and decreasing survival to maturity. In contrast, offspring size is predicted to scale positively with A, E, and s, all to the power $1/(1 - q)$. Offspring size is predicted to increase with the amount of mass entering the juvenile population (given by sA) because a higher reproductive investment means a potentially higher density of recruits, resulting in an earlier onset of the competitive phase (fig. 1) and, consequently, a larger advantage for a mutant offspring that enters the competitive phase at a size greater than that of the resident population. As the population evolves toward larger offspring sizes, the density of recruits decreases, lessening any advantage to a mutant that enters larger than the resident population in the competitive phase. This process continues until the competitive phase is sufficiently short that any remaining benefit to a larger mutant balances the costs of producing fewer but larger offspring. The positive scaling with reproductive life span also arises because of influence on the length of the competitive phase, this time by shifting the intercept of the self-thinning boundary; a longer life span means a lower adult turnover and thus fewer resources available to recruits.

We expect examples of negative scaling between W_{0,r^*} and W_α to be rare because, in general, A and E scale positively with adult size (Blueweiss et al. 1978; Charnov 1993) and the positive influence of W_α on W_{0,r^*} via AE more than compensates for the negative influence of lengthening time to maturity. Adult size thus influences W_{0,r^*} by extending the time to maturity, by increasing the number of offspring produced, and by decreasing the av-

Table 1: Standardized major axis relationships fitted across species mean data for different life-history attributes among mammals and plants (X, Y)

Y	X	n	r^2	Back-transformed intercept	Slope (95% CI)	b_H	P
Mammals:							
W_0	W_α	395	.969	.290	.947 (.931–.964)	1.0	<.001
A	W_α	302	.869	.413	.685 (.657–.713)	.75	<.001
E	W_α	53	.576	1.782	.301 (.250–.361)	.25	.0047
AE	W_α	40	.972	.895	.917 (.868–.969)	1.0	.002
Plants:							
W_0	W_α	322	.391	2.02×10^{-5}	.541 (.496–.589)	1.0	<.001
A	W_α	159	.539	.0348	.834 (.749–.928)	.75	.051

Note: n = number of species. Standardized major axis line was fitted to log-transformed data. P values are shown for comparisons of slopes against the hypothesized value b_H.

erage survival per recruitment event. Although A and E each have different effects on the model (E determines the rate of adult turnover, A the reproductive investment per year), they conveniently appear in equations (1), (2), and (4) and in the general solution as a product. This product corresponds to the lifetime reproductive investment for a single individual, so W_{0,r^*} scales with lifetime reproductive investment.

To clarify the overall relationship between offspring size and adult size, we let $AE = \sigma_1 W_\alpha^\beta$, where σ_1 and β are the scaling constant and the slope relating adult size to lifetime reproductive investment, respectively. Substituting into the general solution for cases where $D = 1$ yields the following prediction for the relationship between offspring size and adult size:

$$W_{0,r^*} = \sigma_2 W_\alpha^{(\beta-q)/(1-q)}, \tag{8}$$

where $\sigma_2 = (s\sigma_1)^{1/(1-q)} \exp\{2(b-q)(1-b)/[bz(q-1)]\}$. For cases where $D > 1$, a similar solution is obtained when adult sizes are considerably larger than k (the constant relating survival during establishment phase to mass), with the only modification being multiplication of the intercept in equation (8) by $D^{q/(1-q)}$. For small adult sizes approaching k, the predicted offspring size departs from the scaling represented in equation (8) (app. A) because of additional mortality in the establishment phase. However, overall, the inclusion of an establishment phase in the model has little bearing on the predicted relationships to adult size. Consequently, we refer mostly to the general solution for $D = 1$ because of its increased analytic tractability.

Predicting Scaling Exponents across Species and Differences among Taxa

The slope of the predicted relationship between W_{0,r^*} and W_α is determined jointly by β, the scaling slope relating adult size to lifetime reproductive investment, and q, the rate of density-independent mortality (eq. [8]). Other factors determine the intercept of the relationship, including preestablishment mortality, the rate of competitive thinning, the degree of competitive asymmetry, and the scaling constant σ_1 relating adult size to lifetime reproductive investment. The model thus offers a pathway for understanding factors that promote evolution of larger offspring size and for understanding how differences in the scaling of AE with W_α and other factors affecting the ecology of recruitment translate into differences in the scaling of offspring with adult size.

The positive relationship between A and adult body size arises because of increasing total energy and resource budgets with body size (Charnov 1993; Enquist et al. 1998). The reasons for the scaling of E with W_α are less clear, though empirical data support the relationship (Charnov 1993). Some theoretical treatments (Charnov 1993; Enquist et al. 1998, 1999) suggest slopes of 0.75 and 0.25 relating A and E to W_α. If these theoretical slopes are provisionally adopted (i.e., $\beta = 1.0$), then we predict isometric scaling between W_{0,r^*} and W_α, thus recovering Charnov's (1993) original prediction for an invariant offspring : adult size ratio.

Empirical data for mammals and plants do not show the isometric scaling between W_{0,r^*} and W_α that arises when $\beta = 1.0$ (fig. 3; table 1). In mammals, the slope of the relationship is 0.95, and in plants, it is 0.54. For these observed exponents to be recovered from the model, lifetime reproductive investment must scale allometrically ($\beta < 1.0$) with body size. When $\beta < 1.0$, increases in adult body size bring a proportionately smaller increase in lifetime reproductive investment, resulting in a relatively shorter competitive growth phase and consequently a less than proportional increase in offspring size. A value of $\beta = 0.95$ optimizes the fit between observed and predicted offspring size W_0 for mammals ($r^2 = 0.97$, $n = 395$). Available empirical data for AE and W_α (fig. 2, *right*) give an estimate for $\beta = 0.92$ (table 1), a value compatible

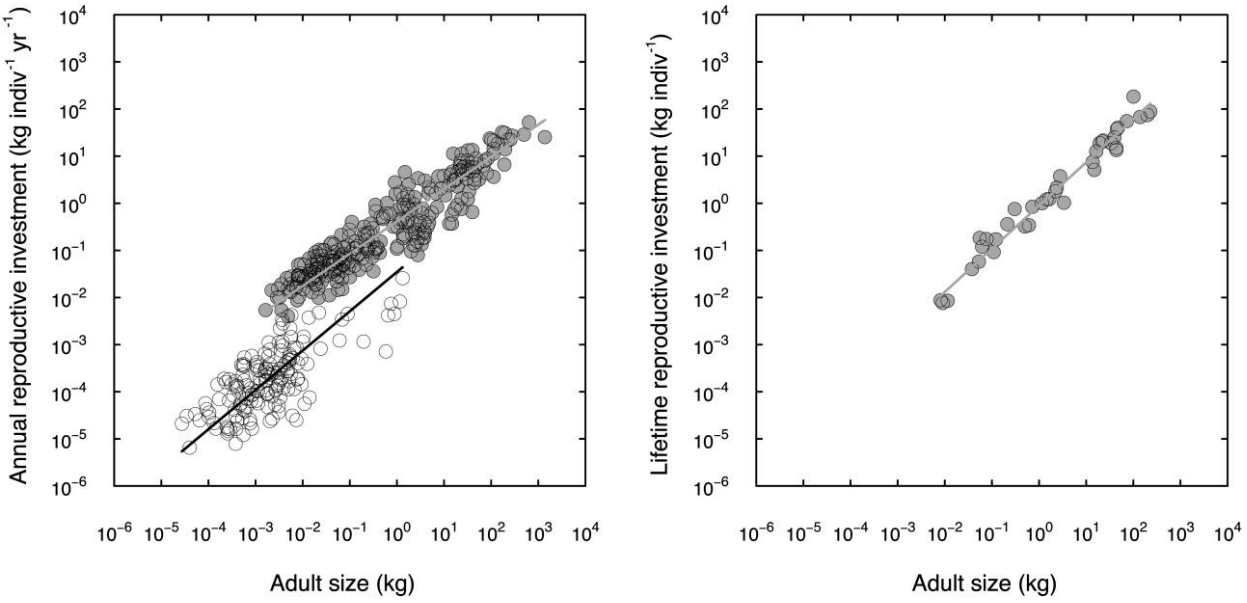

Figure 2: Empirical relationships between annual reproductive investment (*left*) and lifetime reproductive investment (*right*) versus adult body size for mammal (*gray*) and plant (*white*) species. Each point represents one species. Units are in dry mass. Lines represent fitted standardized major axis relationships. See details in table 1. Both axes are log scaled.

with $\beta = 0.95$ ($P = .102$), which gives the best fit and is significantly shallower than 1.0 (table 1). Here, we should make clear that we have little certainty in the adopted value for q ($=0.2$), the rate of density-independent mortality, and that this value affects the specific value of β that gives the best-fit relationship. However, this does not alter the main thrust of our argument: allometric scaling of offspring size with adult size requires β to be <1.0, irrespective of the amount of density-independent mortality, and this is what is observed empirically.

Other parameters affect the elevation of the relationship. Higher presettlement survivorship s or greater competitive asymmetry z both lead to increases in W_{0,r^*} by increasing the relative advantage to a mutant that is born larger than the residents. With higher survivorship, the advantage comes from a longer competitive phase. With higher competitive asymmetry, the advantage comes from increased survival per unit mass growth during the competitive phase. Unfortunately, reliable values for both s and z are unknown, though we expect presettlement survival to be higher for mammals than for plants. Assuming only a modest degree of competitive asymmetry for mammals with $z = 3$ results in a value of $s = 0.94$ to fit the empirical data (fig. 3).

In plants, the scaling slope between W_0 and W_α is shallower than that in mammals, offspring are smaller at a given adult size, and there is considerably more scatter in the relationship (fig. 3). The best-fit relationship between observed and predicted W_0 in plants is given by $\beta = 0.632$ and $s\sigma_1 = 1.7 \times 10^{-4}$ ($r^2 = 0.39$, $n = 322$). The latter term translates into a 3,000-fold difference between plants and mammals in the total mass of offspring from a parent of size 1 kg entering the juvenile population (post-establishment). The lower value for β means that the differences are even more pronounced at larger adult sizes.

If our model is to describe differences between mammals and plants in the scaling of adult size and offspring size, then we should observe differences in the scaling of effective reproductive investment (i.e., total mass making it through the establishment phase) between mammals and plants. Although data are limited, several differences between recruitment in plants and recruitment in mammals support this idea. First, plants seem to invest less in reproduction at a given adult size. Few data are available on lifetime reproductive investment for plants, but data on per-year reproductive investment indicate that plants produce 30–40 times less mass in offspring per year than a correspondingly sized mammal (fig. 2, *left*; table 1). This difference may result from a combination of factors, including higher metabolic activity of mammals at a given size (and note that inert tissue such as heartwood is included in the dry mass for plants), lower allocation of available energy to reproduction in plants, or increased accessory costs of offspring production in plants.

Additional factors experienced by plants but not by mammals may further reduce plant reproductive invest-

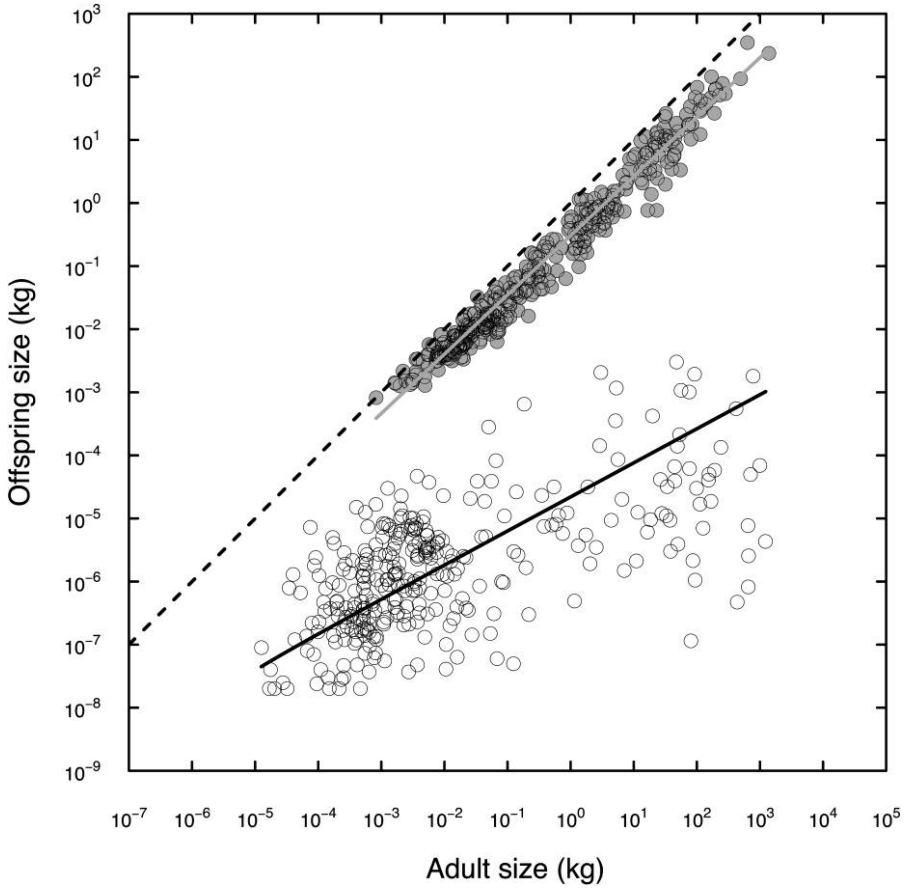

Figure 3: Empirical and predicted relationships between offspring size at independence (mass at weaning or seed mass) and adult body size for mammal (*gray*; $n = 395$) and plant (*white*; $n = 322$) species. Each point represents one species. Units are in dry mass. The dashed line indicates 1 : 1, which sets an upper limit to offspring size. Other lines indicate predictions from the model for different values of intercept $s\sigma$ and slope β relating adult body size to effective lifetime reproductive investment, showing how differences in the ecology of recruitment lead to different offspring sizes. With $D = 1$, the best-fit relationship for mammals was given by $s\sigma = 0.44$, $\beta = 0.952$ (*gray line*). With $D = 2$, the best-fit relationship for plants was given by $s\sigma = 1.7 \times 10^{-4}$, $\beta = 0.63$ (*black line*). These predictions explain 97% and 39% of variance in observed W_0, respectively. Other parameters are set at $q = 0.2$, $b = 0.75$, and $z = 3$. Both axes are log scaled.

ment. Because plants are sessile organisms, their opportunity for recruitment is often limited to times immediately following disturbance when competition from adults is relaxed. This dynamic, compared to that of mammals, fundamentally alters how the effective lifetime reproductive investment scales with adult size. Large mammals produce a series of litters across a number of years, with seemingly high efficiency in the transfer between mass allocated to reproduction and mass entering the population as juveniles in each year. In contrast, the inherent stochasticity of recruitment opportunity for plants means that this transfer efficiency will be substantially lower, at least when considered across the reproductive life span of the adult. The long time lag between offspring production and recruitment opportunity or the high spatial variability in dispersal destination ensures that most seeds do not survive to seedling stage. Indirect data (Moles and Westoby 2006) suggest average survival of <0.15. Further, these losses are likely to be greater for larger plants because opportunities for recruitment are rare in vegetation dominated by large, long-lived plants. This would lead to a greater proportional reduction in effective reproductive investment for larger organisms.

Combined, differences in allocation to reproduction and survival to establishment could explain the reduction in scaling intercept and slope between lifetime reproductive investment and adult size needed to recover the observed allometries between offspring size and adult size from the model. Differences in recruitment opportunity through disturbance intervals are also intrinsically site specific and

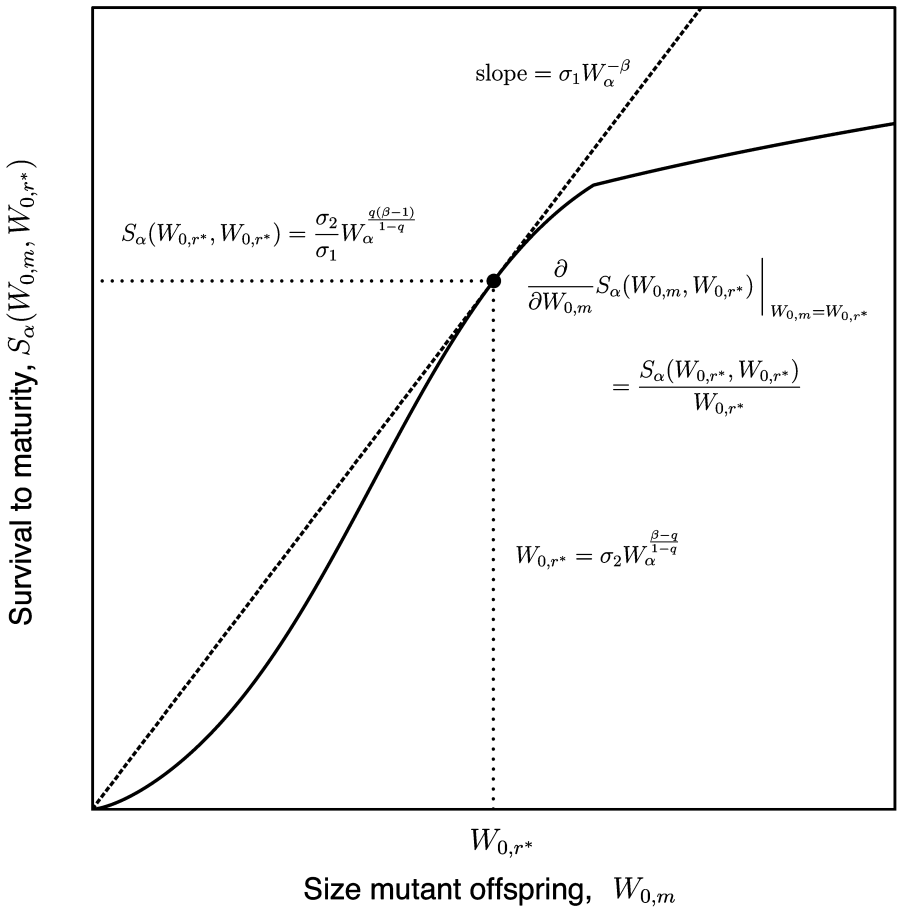

Figure 4: Survival to maturity (given by eq. [7]) for a rare individual with offspring size $W_{0,m}$ (*solid line*) in a population dominated by the resident strategy $W_{0,r}$, where $W_{0,r} = W_{0,r^*}$ is an evolutionarily stable strategy. Because parental fitness is proportional to S_α/W_0 (eq. [1]), the optimal allocation of resources from the parent's perspective occurs where the gradient of a line through the origin intersecting the survival curve (*dashed line*) is maximized (*circle*; Smith and Fretwell 1974). The equations show how equilibrium values for offspring size, survival to maturity, and marginal return in offspring fitness vary with other parameters.

are therefore capable of producing the increased scatter about the general relationship in plants compared to mammals. These results lead us to suggest that other organisms with even lower presettlement survivorship (e.g., $s = 10^{-3}$ to 10^{-9}) and with unpredictable recruitment opportunities, such as sessile marine organisms or pelagic fish, should show similar or wider scatter around the general relationship between W_0 and W_α (e.g., Duarte and Alcaraz 1989).

Predictions about Survival

The original SFM did not allow for predicted offspring size to respond to adult resource status or to other characteristics of the site and species, such as reproductive life span. Our model overcomes this limitation by making mutant survival (eq. [7]) dependent on population characteristics so that fitness is no longer a linear function of offspring number (Venable 1992). A direct result is that fitness is now frequency dependent, so the usual paradigm attributed to the SFM of an optimal seed size determined by external factors no longer applies. However, our results retain the essence of Smith and Fretwell's (1974) approach. Equation (7) produces a classic logistic curve expressing offspring survival in relation to offspring size (fig. 4). A logistic (or diminishing) curve is assumed by most previous models (Smith and Fretwell 1974; Lloyd 1987; Charnov and Downhower 1995), but here it arises via ecological interactions and particulars of the population rather than being postulated as a plausible form for S_α. At the ESS, Smith and Fretwell's (1974) criterion for the optimal allocation of parental resources (Lloyd 1987),

$(\partial/\partial W_{0,\mathrm{m}})S_\alpha(W_{0,\mathrm{m}}, W_{0,\mathrm{r}^*})|_{W_{0,\mathrm{m}}=W_{0,\mathrm{r}^*}} = [S_\alpha(W_{0,\mathrm{r}^*}, W_{0,\mathrm{r}^*})]/W_{0,\mathrm{r}^*}$, is also satisfied (fig. 4). One important consequence is that though parental fitness is maximized, offspring fitness is not. The positive slope of the survival curve at the optimum ensures that offspring would benefit from additional resource allocation. This condition provides the basis for understanding conflict between mother and offspring (Haig and Westoby 1988). Substituting from equation (2), our model also predicts that the slope $(\partial/\partial W_{0,\mathrm{m}})S_\alpha(W_{0,\mathrm{m}}, W_{0,\mathrm{r}^*})|_{W_{0,\mathrm{m}}=W_{0,\mathrm{r}^*}}$ scales as $\sigma_1 W_\alpha^{-\beta}$ (fig. 5). In other words, the marginal return in survival from additional resources decreases with body size. Consequently, we predict the magnitude of the conflict between mother and offspring to be greater in smaller organisms when resources are allocated optimally for the mother.

The model makes several other predictions about the scaling of $W_{T,\mathrm{r}}$ (size at the onset of competitive mortality) and survival with adult size. Combining the general solution in equation (8) for the ESS offspring size with equation (4) for size at onset of thinning $W_{T,\mathrm{r}}$, we see that $W_{T,\mathrm{r}}$ scales linearly with W_α for populations at evolutionary equilibrium, with intercept $\sigma_3 = (\exp\{2(b-q)(1-b)/[bz(q-1)]\})^{(1-q)/(b-q)}$. This means that the duration of the competitive growth phase (measured in log growth increments) and survival through the competitive phase, $S_{\mathrm{comp}} = (W_\alpha/W_{T,\mathrm{r}})^{-b} = \sigma_3^b$, will be independent of β and invariant across taxa for similar values of b, z, and q. However, overall survival from independence to maturity is predicted to scale allometrically with adult body size as $S_\alpha(W_{0,\mathrm{r}^*}, W_{0,\mathrm{r}^*}) = (\sigma_2/\sigma_1)W_\alpha^{[q(\beta-1)/(1-q)]}$. Thus, when $\beta < 1$, larger organisms are predicted to have lower survival per offspring at evolutionary equilibrium, with the decrease arising through higher density-independent mortality rather than through density dependence.

Importance of Size-Asymmetric Competition

Larger offspring size can confer several different survival benefits. Nevertheless, our model indicates that the sole mechanism driving evolution toward larger offspring size is the benefit in survival realized by an individual that enters the self-thinning phase at a size larger than that of its competitors. This can be shown by assuming that advantages in relative size at birth are not carried through to advantages in relative size at the onset of thinning. Biologically, this corresponds to the situation where the sole effects of offspring size are increasing establishment success and shortening time to maturity. In this case, mutant fitness is given by $R(W_{0,\mathrm{m}}, W_{0,\mathrm{r}}) = D^{k[(1/W_{0,\mathrm{r}})-(1/W_{0,\mathrm{m}})]}(W_{0,\mathrm{m}}/W_{0,\mathrm{r}})^{q-1}$, and the ESS is $W_{0,\mathrm{r}^*} = k\log D/(1-q)$. If, instead, we assume that the mutant maintains a size advantage but this does not lead to reduced mortality through the competitive phase (i.e., $z = 0$), then a similar result is obtained, $W_{0,\mathrm{r}^*} = k\log D/(1-b)$, only the mutant advantage is seen in shortening the competitive phase rather than the density-independent phase. When $D > 1$, these predictions are analogous to those of the original SFM: a single optimum offspring size is predicted independent of adult size and resource status. However, when $D = 1$, there is no solution; offspring size is predicted to evolve toward ever-smaller sizes because the disadvantages of lengthening the juvenile phase are insufficient by themselves to offset the benefits of producing more offspring.

Factors such as smaller resident size, higher adult resource status, or higher density-independent survival promote earlier onset of self-thinning as a result of greater density of surviving offspring and thus serve to increase the selection gradient toward larger offspring size. Greater competitive asymmetry also leads to higher equilibrium offspring size for a given body size (eq. [8]; fig. 5) but with diminishing returns. As z increases, $W_{T,\mathrm{r}^*} \to W_\alpha$, so the increased competitive advantage is countered by a decrease in the duration of the competitive phase. For large z, the intercept σ_2 of the predicted scaling relationship between offspring and adult size approaches $(s\sigma_1)^{1/(1-q)}$ (fig. 5). With decreasing competitive asymmetry, the predicted offspring size decreases in the case of $D = 1$ (fig. 5), and in the case of $D > 1$, the population becomes unviable once adult size drops below a critical value (i.e., $R(W_{0,\mathrm{r}}, W_{0,\mathrm{r}}) < 1$ for all $W_{0,\mathrm{r}}$).

Size advantage during competition has long been known to influence mortality in plant communities (reviews in Harper 1977; Westoby 1984). Research on mammals, however, has been focused largely on extrinsic causes of mortality, to the extent that Charnov (1993) built his theory on the assumption that mortality during later stages of development was entirely due to extrinsic factors. More empirical evidence about the influence of size-asymmetric competition on mortality from groups other than plants would be valuable. Our model gives rise to some predictions about what field data might be expected to show.

In the model, even when size-asymmetric competition plays an important evolutionary role in causing larger adults to produce larger offspring, only low to moderate levels of density-dependent mortality are predicted in populations at evolutionary equilibrium. This occurs because when competitive mortality is strong, natural selection drives offspring size upward, and this, in turn, decreases offspring densities so that the competitive phase does not begin until later during ontogeny. For the parameters used in figure 3, size at the onset of the competitive phase is predicted to be 72% of W_α, while survival through the competitive phase is ~0.78. To test this prediction, field studies might need to manipulate density experimentally because observations of proximate causes of death, such

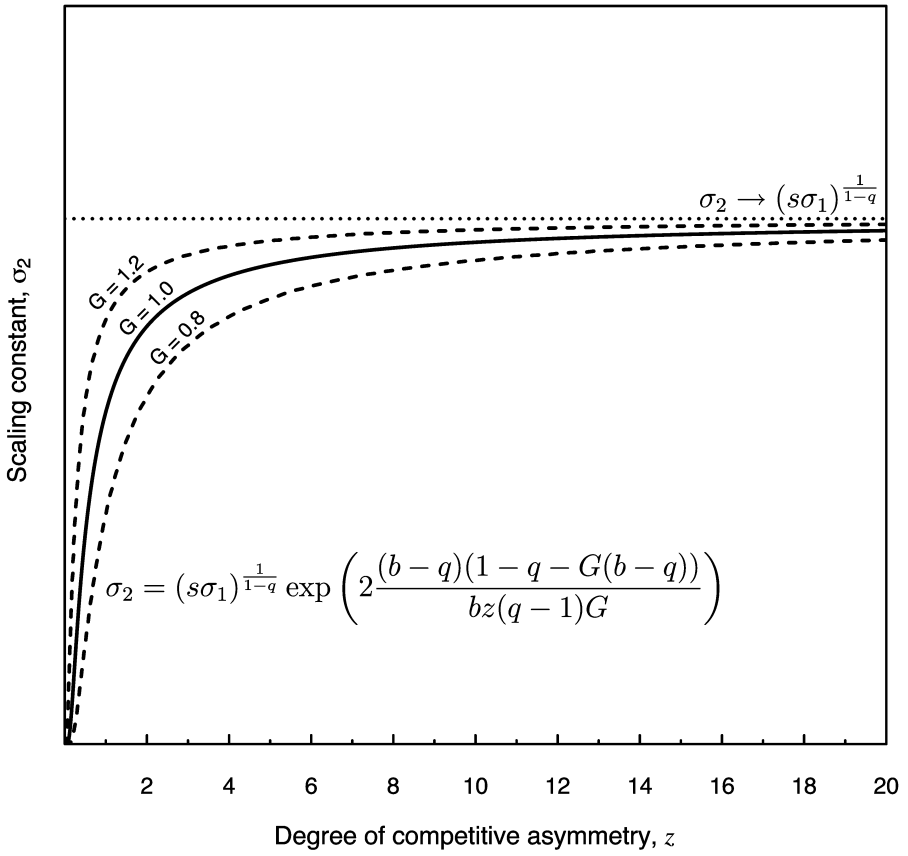

Figure 5: Relationship between the degree of competitive asymmetry (z) and intercept (σ_2) of the predicted scaling relationship between offspring and adult size for different values of G and when $D = 1$. The equation shows how σ_2 depends on G and on other population parameters; G indicates the proportional change from birth to onset of the competitive phase in the size ratio between mutant and resident individuals (see text for details), with $G = 1$ giving the solution when mutant advantage is maintained through ontogeny (eq. [8]). The dotted line shows the asymptotic behavior of σ_2 as z becomes large.

as predation or starvation, do not show whether mortality was density related.

The Influence of Variable Mutant Advantage on Model Predictions

Until now, we have assumed that the relative size advantage of the mutant over the residents is maintained from birth throughout ontogeny or at least until the onset of the competitive stage. This is equivalent to assuming $\gamma(W_{0,\mathrm{m}}, W_{0,\mathrm{r}}) = 1$ in equations (6) and (7). After the onset of the competitive phase, it seems reasonable to assume that any size advantage is maintained or even amplified because of the size-asymmetric advantage experienced by the mutant in acquiring resources during the competitive phase. However, there are several reasons we might expect the relative size advantage experienced by the mutant at the onset of competitive stage to differ from that experienced at birth. Here we explore some possible variations on the assumption of $\gamma(W_{0,\mathrm{m}}, W_{0,\mathrm{r}}) = 1$ and ask whether they have any bearing on model predictions.

We first investigated the situation where the mutant advantage at the start of the competitive phase varies by some fixed proportion of initial advantage; that is,

$$\frac{W_{T,\mathrm{m}}}{W_{T,\mathrm{r}}} - 1 = G\left(\frac{W_{0,\mathrm{m}}}{W_{0,\mathrm{r}}} - 1\right).$$

If $G > 1$, then mutant advantage increases through ontogeny. This could happen if, for example, size-asymmetric competition played a strong role in determining the amount of resources captured and thus growth rate early during ontogeny but did not influence mortality until later during ontogeny (defined as the competitive phase). Alternatively, the mutant's relative size advantage might decrease ($G < 1$) because of ontogenetic trends in relative

growth rate (RGR). With no differences in growth rate, maintaining relative size advantage is possible only with a constant, size-independent RGR. But for most organisms, RGR is known to decrease with increasing body size (West et al. 2001) so that mutants and residents both converge on some asymptotic size.

Incorporating either a fixed reduction or an increase of the mutant's size advantage through ontogeny into the model causes a shift in the intercept (but not the slope) of the predicted relationship between adult size and offspring size. Increasing mutant size advantage ($G > 1$) causes an increase in offspring size at a given adult size, while decreasing mutant size advantage ($G < 1$) causes a decrease in offspring size (fig. 5). While the direction of this result is intuitively obvious, the influence of G on σ_2 is nonlinear, in the same manner as the nonlinear relationship between the degree of competitive asymmetry and the scaling constant σ_2. Also, as the degree of competitive asymmetry increases, the results from the modified model converge on the same limit as the original model with $G = 1$ (fig. 5). These results are interesting because they show how size-asymmetric competition late during ontogeny can still drive evolutionary increases in offspring size, even if the initial size advantage is diminished over time.

Another possibility to consider is that the proportional reduction in the mutant's size advantage varies in a systematic way with adult size, potentially leading to changes in the slope of predicted adult size–offspring size relationships. To investigate this, we modeled $\gamma(W_{0,\mathrm{m}}, W_{0,\mathrm{r}})$ as a function of offspring size, adult size, and size at the onset of thinning by incorporating a general model (West et al. 2001) for size-related changes in RGR (for details, see app. B). This extension led to the following interesting result. When $\beta = 1$, we predict a constant reduction in the mutant's relative size advantage from birth to the onset of thinning across species, leading to a change in the intercept of the predicted relationship between offspring size and adult size but no change in slope (as above). However, when $\beta < 1$, we predict a change in both the slope and the intercept of the relationship, with the amount of change in the slope dependent also on β (fig. 6). This result arises because of the nonlinear scaling of $W_{T,\mathrm{r}}$ with W_α when $\beta < 1$, meaning that the relative duration of the competitive phase varies among small and large species. This, in turn, influences $\gamma(W_{0,\mathrm{m}}, W_{0,\mathrm{r}})$ and, consequently, the size advantage to the mutant in a systematic way. As before, the changes are most pronounced at low levels of competitive asymmetry. As z increases, the results converge on those of the basic model, in both intercept and slope (fig. 6).

Overall, we conclude that assumptions about the relative size advantage of the mutant can lead to important differences in predicted relationships between offspring size and adult size. However, these changes do not alter the primary conclusions of this article, that allometric scaling of offspring size and adult size is possible only with allometric scaling of lifetime reproductive investment with adult size and that differences in the scaling of reproductive investment among taxa can lead to differences in scaling of offspring size to adult size. Further empirical and theoretical work is clearly needed to fully dissect the interactions between assumptions about size advantage in growth, mortality, relative growth rate, and their influence on model predictions and to determine appropriate parameter values.

Relationship to Several Other Models of Offspring Size

Our model extends frameworks originally laid out by Smith and Fretwell (1974) and Charnov (1993). Charnov's simplifying assumptions had the effect of requiring offspring size to scale isometrically with adult size. Our extensions make allometric scaling possible, as actually observed. Other deficiencies of Charnov's model were also noted by Rees and Venable (2007). They pointed out, following Kiflawi (2006), that Charnov's model predicts positive scaling of offspring and adult sizes only when growth or mortality rates of seedlings during the earliest stages of ontogeny are correlated with adult size. However, because empirical evidence for such a correlation is weak (reviewed by Rees and Venable [2007]), it seems unlikely that this mechanism can explain the strong coordination between adult size and offspring size observed across a variety of taxa (e.g., Blueweiss et al. 1978; Damuth 1987; Duarte and Alcaraz 1989; Garcia-Barros 2000; Moles et al. 2005*b*).

Modeling by Rees and Venable (2007) adopted some assumptions in common with Charnov's work: that density dependence operates only during early life and that an individual's survival through ontogeny is independent of the seed size and abundance of competitors. In the model presented here, density dependence operating late during ontogeny together with size-asymmetric advantage during the competitive phase proved to be essential mechanisms for allometric scaling to evolve between adult size and offspring size at independence. Rees and Venable's modeling did not incorporate those assumptions, so it is consistent with our results that they were not able to produce a general explanation for the allometric scaling.

Our model shares many similarities with those developed by Geritz and coauthors (Geritz et al. 1988, 1998, 1999; Geritz 1995) for investigating evolution of seed size in "safe sites." Safe sites are patches just large enough to hold a single adult individual. Within a safe site, the outcome of competition is decided by size-asymmetric interactions. Geritz's (1995; Geritz et al. 1999) models, like

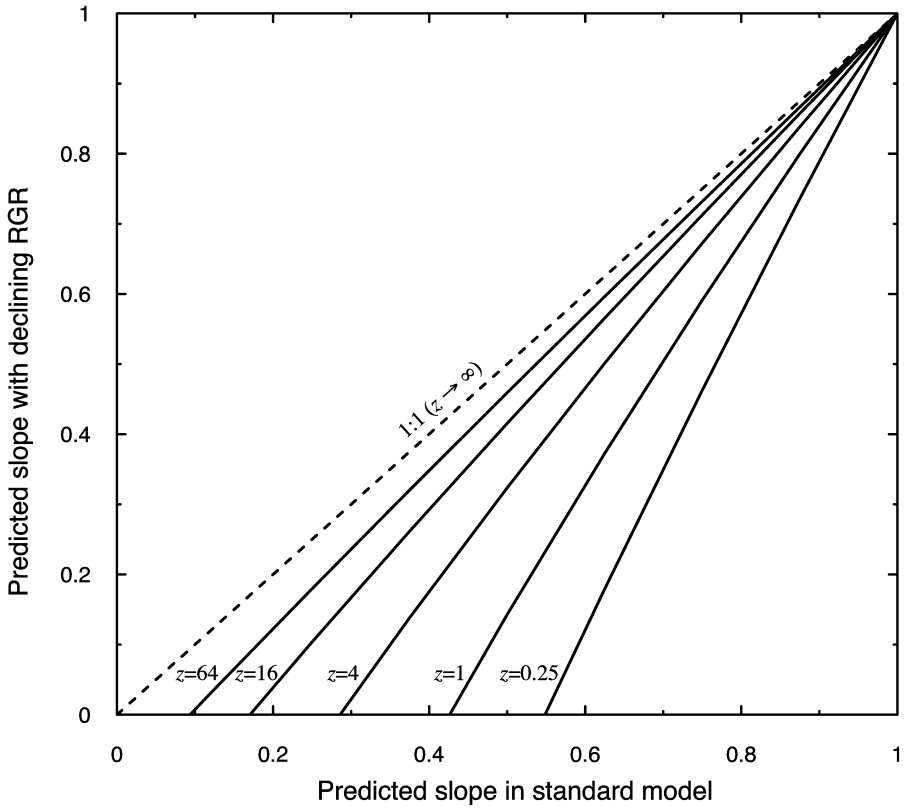

Figure 6: Relationship between the predicted slope of offspring : adult size scaling relationships in the standard model, given by $(\beta - 1)/(1 - q)$, to predictions from the modified model, which allows for ontogenetic trends in relative growth rate, at different levels of competitive asymmetry. The predictions from the two models match when lifetime reproductive investment scales isometrically with adult size ($\beta = 1$) or when competitive asymmetry is high ($z \to \infty$).

ours, predict evolution toward larger offspring size as result of competition and, in the absence of any size asymmetry, a single optimum offspring size that balances the benefits of larger reserve mass during establishment against the costs of producing fewer, larger offspring.

The Geritz models did not consider differences relating to adult size. They could easily be extended, though, by making the density of sites per ground area a function of adult size, as well as the resources available for reproduction within each site. Under these conditions, we expect results from our different models to be largely compatible. However, predictions would differ in one important way. Because of nonlinear relationships between the number of seeds produced and the proportion of patches that can be occupied, Geritz's (1995; Geritz et al. 1999) models predict the emergence of a range of coexisting seed mass strategies within species, while our model predicts a single strategy that is evolutionarily stable. The reason for this difference can be traced to assumptions regarding density dependence. Geritz et al. assume that the population density of adults may vary between generations and, consequently, that the available resources may not be fully utilized, while we assume that adults are always at their maximum density. In our model, density dependence operates only by adjusting the amount of competitive- and noncompetitive-induced mortality required among recruits (i.e., the position of $W_{T,r}$) so that recruitment matches adult turnover. These differences in turn reflect different assumptions regarding the spatial interactions among individuals (these are treated explicitly in Geritz's models but not in our model). We see these two frameworks not as alternatives but as complementary, highlighting some of the different outcomes that arise when different aspects of density dependence are incorporated into offspring size models. Our approach seems more suited for understanding broad-scaling relationships across species (whether the prediction is for a single strategy or a range of strategies) because it does not depend on the arbitrary distinction of a safe site, it is not restricted to sessile organisms such as plants, and it connects mortality during competition to ontogenetic

trends in resource use in a scale-invariant manner. On the other hand, Geritz's models seem superior for understanding how spatial interactions during recruitment may lead to a range of offspring sizes within species through adaptive processes. It remains to be seen whether inclusion of spatial dynamics in our model would also lead to additional shifts in either the slope or the intercept of expected cross-species relationships and whether these shifts would account for significant differences among taxa.

Implications for Understanding Offspring Size Variation

The results from our model point to several challenges for future work. Most pressing is critically evaluating the idea that size differences at birth can translate into size-asymmetric survival advantages later in life. Because most empirical work has focused on quantifying survival during early life only, little is known about the long-term influence of initial differences in offspring size. Also, several of the parameters in the model are largely unknown. With relevant parameters known for a variety of taxa, a more ambitious test of the model would become possible. Broadly, we hope that the current work provides a foundation for understanding similarities and differences in life-history strategies across taxa. The processes included in the model, such as the scaling of reproductive investment, life span, and population density with body size, as well as the predicted scaling between offspring and adult size, have been observed empirically across a variety of other taxa, including birds, fish, reptiles, and invertebrates (Blueweiss et al. 1978; Damuth 1987; Duarte and Alcaraz 1989; Garcia-Barros 2000; Lobón-Cerviá and Mortensen 2006), suggesting that dynamics similar to those proposed here may underpin these correlations.

For plants, our model represents a shift in thinking about variation in seed mass. Here we have shown that a unique seed size is predicted for each combination of adult size and longevity as a result of frequency-dependent interactions among competing individuals within species. Interactions among species do not enter the model explicitly, though competition from neighboring species can be thought of as influencing the general availability of resources for each species. Because of coordination between seed mass and adult mass, processes sustaining the wide range of seed size strategies at a single site are intertwined with processes sustaining the wide range of adult sizes. Thus, the challenge remains to develop models that incorporate frequency dependence in such a way as to account for coexisting mixtures of different strategies spread along an axis of coordinated life-history variation, from small to big. Here, size-asymmetric competition for light undoubtedly plays an important role in driving the coexistence of species differing in adult stature (Falster and Westoby 2003), as well as for driving the evolution of larger offspring sizes.

Acknowledgments

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APPENDIX A

General Solution for Evolutionarily Stable Strategy and Test for Evolutionary and Convergent Stability

*General Solution for Taxa with No Reserve Mass (*D = *1)*

When $D = 1$, the general solution for the evolutionarily stable offspring size is given by

$$W_{0,\mathrm{r}^*} = \exp\left[2\frac{(b-q)(1-b)}{bz(q-1)}\right]\left(\frac{sAE}{W_\alpha^q}\right)^{1/(1-q)}. \tag{A1}$$

To determine whether W_{0,r^*} is evolutionarily stable, we calculated the second derivative of $R(W_{0,\mathrm{m}}, W_{0,\mathrm{r}})$ with respect to mutant strategy and evaluated it when $W_{0,\mathrm{m}} = W_{0,\mathrm{r}} = W_{0,\mathrm{r}^*}$ (Geritz et al. 1998):

$$\frac{\partial^2}{\partial W_{0,\mathrm{m}}^2} R(W_{0,\mathrm{m}}, W_{0,\mathrm{r}})\bigg|_{W_{0,\mathrm{m}}=W_{0,\mathrm{r}}=W_{0,\mathrm{r}^*}} = \frac{sAE}{4W_{0,\mathrm{r}^*}^3}\left(\frac{W_{T,\mathrm{r}^*}}{W_{0,\mathrm{r}^*}}\right)^{-q}\left(\frac{W_\alpha}{W_{T,\mathrm{r}^*}}\right)^{-b}$$

$$\times \left\{\left[bz\ln\left(\frac{W_\alpha}{W_{T,\mathrm{r}^*}}\right) + 2b - 6\right]\left[bz\ln\left(\frac{W_\alpha}{W_{T,\mathrm{r}^*}}\right) + 2b\right] + 4(2 - bz)\right\}.$$

Because the first terms are all positive, the sign of the second derivative is given by the term in braces. We then make use of the following observation: from equations (4) and (A1), we find that when $W_{0,\mathrm{r}} = W_{0,\mathrm{r}^*}$, $W_{T,\mathrm{r}^*} = (\exp\{2(b - q)(1 - b)/[bz(q - 1)]\})^{(1-q)/(b-q)}W_\alpha$, so $\ln(W_\alpha/W_{T,\mathrm{r}^*}) = 2[(1 - b)/bz]$. Substituting $\ln(W_\alpha/W_{T,\mathrm{r}^*})$ above, we obtain

$$\frac{\partial^2}{\partial W_{0,\mathrm{m}}^2} R(W_{0,\mathrm{m}}, W_{0,\mathrm{r}})\bigg|_{W_{0,\mathrm{m}}=W_{0,\mathrm{r}}=W_{0,\mathrm{r}^*}} = \frac{sAE}{4W_{0,\mathrm{r}^*}^3}\left(\frac{W_{T,\mathrm{r}^*}}{W_{0,\mathrm{r}^*}}\right)^{-q}\left(\frac{W_\alpha}{W_{T,\mathrm{r}^*}}\right)^{-b} \times (-4bz) < 0.$$

Consequently, we can conclude that W_{0,r^*} is an evolutionarily stable strategy, meaning that it cannot be invaded by phenotypes different from W_{0,r^*} once it has become established as the resident strategy.

To determine whether W_{0,r^*} is convergent stable, we evaluated the difference in the second derivative of $R(W_{0,\mathrm{m}}, W_{0,\mathrm{r}})$ with respect to resident and mutant strategies to see whether this term is >0 (Geritz et al. 1998). Convergent stability implies that W_{0,r^*} can be approached through a series of small mutations. Taking the derivatives, we obtain

$$\frac{\partial^2}{\partial W_{0,\mathrm{r}}^2} R(W_{0,\mathrm{m}}, W_{0,\mathrm{r}}) - \frac{\partial^2}{\partial W_{0,\mathrm{m}}^2} R(W_{0,\mathrm{m}}, W_{0,\mathrm{r}})\bigg|_{W_{0,\mathrm{m}}=W_{0,\mathrm{r}}=W_{0,\mathrm{r}^*}}$$

$$= \frac{sAE}{W_{0,\mathrm{r}^*}^3(b - q)}\left(\frac{W_{T,\mathrm{r}^*}}{W_{0,\mathrm{r}^*}}\right)^{-q}\left(\frac{W_\alpha}{W_{T,\mathrm{r}^*}}\right)^{-b}$$

$$\times \left\{(q - b)\left[2 - 2b - zb\ln\left(\frac{W_\alpha}{W_{T,\mathrm{r}^*}}\right)\right] + zb(1 - q)\right\}.$$

Again, the sign of this term will be given by the sign of the term in braces. Substituting for $\ln(W_\alpha/W_{T,\mathrm{r}^*})$ as before, we obtain

$$\frac{\partial^2}{\partial W_{0,\mathrm{r}}^2} R(W_{0,\mathrm{m}}, W_{0,\mathrm{r}}) - \frac{\partial^2}{\partial W_{0,\mathrm{m}}^2} R(W_{0,\mathrm{m}}, W_{0,\mathrm{r}})\bigg|_{W_{0,\mathrm{m}}=W_{0,\mathrm{r}}=W_{0,\mathrm{r}^*}}$$

$$= \frac{sAE}{W_{0,\mathrm{r}^*}^3(b - q)}\left(\frac{W_{T,\mathrm{r}^*}}{W_{0,\mathrm{r}^*}}\right)^{-q}\left(\frac{W_\alpha}{W_{T,\mathrm{r}^*}}\right)^{-b} \times [zb(1 - q)] > 0.$$

Combined, the tests for evolutionary stability and convergent stability imply that W_{0,r^*} is a continuously stable strategy (Geritz et al. 1998).

General Solution for Taxa with Reserve Mass (D > 1)

For $D > 1$, we obtained the following solution for equilibrium offspring size:

$$W_{0,\mathrm{r}^*} = XD^{q/(1-q)}\exp(L), \tag{A2}$$

where $L = \mathrm{Lambertw}\{[k(2q - 2b + bz)\log D]/[bz(q - 1)D^{q/(1-q)}X]\}$ and X is the solution for W_{0,r^*} when $D = 1$, given in equation (A1). The Lambertw function gives the inverse of $f(y) = ye^y$ (i.e., $y = \mathrm{Lambertw}(f(y))$) and must be estimated through numerical methods. Except for very small adult sizes, $L \sim 0$ so $\exp(L)$ is approximately 1, meaning

that the $\exp(L)$ term in equation (A2) has little bearing on predicted offspring size. For $D > 1$, W_{0,r^*} can be shown to be both evolutionarily stable and convergent stable through numerical procedures.

APPENDIX B

Influence of Declining Relative Growth Rate on Model Predictions

To determine the potential influence of size-related declines in the relative growth rate (RGR) through ontogeny on the evolutionarily stable offspring size and its scaling with adult size, we incorporated the general model for ontogenetic growth (West et al. 2001), which describes mass-based growth as $\dot{W} = aW^{0.75} - cW$, into the general solution for offspring size via influences on $\gamma(w_{0,m}, w_{0,r})$. The exact choice of model is of less importance than its general features (declining RGR). Assuming constant uptake rates for mutants and residents during the precompetitive phase, the size W at a given time and the time t to reach a given size are each functions of initial size,

$$W(t, W_0) = \left(\frac{a}{c}\right)^4\left[1 - \left(1 - \frac{c}{a}W_0^{0.25}\right)e^{-(c/4)t}\right]^4, \tag{B1}$$

$$t(W, W_0) = \frac{-4}{c}\ln\left(\frac{a - cW^{0.25}}{a - cW_0^{0.25}}\right). \tag{B2}$$

Age at onset of the competitive phase (which will be the same for mutants and residents) can then be obtained by substituting $W_{T,r}$ (from eq. [4]) and $W_{0,r}$ into equation (B2). Following from this, $e^{(c/4)t}$ in equation (B1) can be written as $[(a/c) - W_{0,r}^{0.25}]/[(a/c) - W_{T,r}^{0.25}]$. The size ratio between mutant and resident at the onset of the competitive phase is then given by

$$\frac{W_{T,m}}{W_{T,r}} = \frac{W[t(W_{T,r}, W_{0,r}), W_{0,m}]}{W[t(W_{T,r}, W_{0,r}), W_{0,r}]} = \left[\frac{W_{T,r}^{-0.25}(W_{0,m}^{0.25} - W_{0,r}^{0.25}) + 1 - (c/a)W_{0,m}^{0.25}}{1 - (c/a)W_{0,r}^{0.25}}\right]^4 \tag{B3}$$

Beyond parameters already included in the model, the only extra term needed to model changes in relative size under this scheme is the ratio a/c. Species-specific estimates for these ratios were obtained by relating it to adult size W_α as follows. The maximum allowable size under the general growth model above is given by $(a/c)^4$ (West et al. 2001); however, at this point, there would no longer be any mass produced that could be allocated to reproduction. So that all species have positive reproductive investment, we assume that size at maturity is some fraction g of maximum possible size, $W_\alpha = g(a/c)^4 \Rightarrow a/c = (W_\alpha/g)^{0.25}$. Substituting into equation (B3), the mutant's relative size advantage at the onset of thinning can be shown to have the form $(W_{T,m}/W_{T,r}) = \gamma(W_{0,m}, W_{0,r})(W_{0,m}/W_{0,r})$, where

$$\gamma(W_{0,m}, W_{0,r}) = \left[\frac{(W_\alpha/W_{T,r})^{0.25}[1 - (W_{0,r}/W_{0,m})^{0.25}] + (W_\alpha/W_{0,m})^{0.25} - g^{0.25}}{(W_\alpha/W_{0,r})^{0.25} - g^{0.25}}\right].$$

Thus, with a single additional parameter g, we were able to derive a function for $\gamma(W_{0,m}, W_{0,r})$ (used in eqq. [6] and [7] of the main text) that can be incorporated into the general model for studying effects of declining RGR on evolutionary outcomes. Because the modified model cannot be solved analytically, evolutionary outcomes were generated through numerical procedures.

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