

Trade-offs between growth and mortality and the maintenance of individual variation in growth

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ABSTRACT

Trade-offs between growth and mortality can occur for a variety of reasons. These include foraging and predation risk, growth and mature function, growth and somatic development, growth and immune function, or growth and resistance to physiological stressors. We use a simple life-history model to show how individual trade-offs between growth and mortality can lead to the maintenance of individual variation in growth rates through nearly equal fitness (solution of the Euler-Lotka equation) for individuals growing at different rates. We explore these consequences for the relationships between other life-history variables.

Keywords: fitness, growth, individual variation, maturity, mortality, trade-offs.

INTRODUCTION

Many species exhibit pronounced individual differences in growth rates, often in response to variation in resources (Hentschel, 1999). However, even when animals are provided with food under controlled conditions, variation in growth trajectories may persist. This phenomenon has been observed, for example, in turtles (Davenport and Scott, 1993), fish (Gallego and Heath, 1997; Wang *et al.*, 1998), water striders (Klingenberg, 1996) and lizards (Andrews, 1982; Stamps *et al.*, 1998). These individual differences in growth rate may be genotypically autocorrelated across ontogeny – for example, animals that grow more rapidly at one age also grow more rapidly at other ages (Kirkpatrick and Lofsvold, 1992; Vaughn *et al.*, 1999) – but not always (for alternatives, see Stead *et al.*, 1996; Johnsson *et al.*, 1997).

We are thus led to ask why there is so much individual variation in growth rates in different members of the same species living sympatrically? All else being equal, one would expect a positive relationship between growth rates and fitness. For example, juveniles that grow faster reach size or age at maturity more quickly than slower-growing juveniles, while adults that grow more quickly typically have higher fecundity or mating success than slower-growing adults. On this view, individual differences in growth rates are often interpreted in terms of variation in individual quality (e.g. variation among individuals in terms

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of the ability to acquire or compete for resources required for growth). If growth rates are positively related to fitness, it is difficult to explain the maintenance of pronounced individual differences in growth rates when animals live in environments that should permit maximal growth rates for every individual, unless environmental influences on phenotype are extremely large.

Here, we show how trade-offs between growth and mortality for individuals in the same population can generate a range of growth rates which all yield equivalent fitness. The classic trade-off arises in the case of foraging under predation risk, in which animals may grow at submaximal, but fitness-optimizing, rates due to predation risk. However, other trade-offs may lead to the same result. We investigate two interrelated questions. First, why might individuals not grow at the maximum rate possible. Second, are individual differences in growth rates widespread in nature because a range of growth rates produces equivalent fitness?

PROCESSES THAT GENERATE TRADE-OFFS BETWEEN GROWTH AND MORTALITY ACROSS INDIVIDUALS

Trade-offs between growth and mortality are generally discussed in the context of variation in growth rates among rather than within populations (recently reviewed in Arendt, 1997). Perhaps the most familiar example concerns the trade-off between foraging activities and an elevated risk of predation (Anholt and Werner, 1995, 1998). The general result is that animals with high growth rates experience higher risk of mortality than animals with low growth rates; this often leads to an intermediate level of foraging effort that is a balance between the risk of predation and the risk of starvation.

Foraging and predation risk

The proximate bases of this trade-off have been studied in a number of animals. For example, juvenile Atlantic salmon forage at night, thus having reduced growth rates but highly reduced mortality rates (Metcalf and Fraser, 1997). Juvenile rainbow trout show a significant negative relationship between aerobic swimming performance and food consumption (Gregory and Wood, 1999). Treatment of juvenile brown trout *Salmo trutta* with growth hormone increases growth rates but reduces anti-predatory responses (Johnsson *et al.*, 1996). Similar results have been obtained in transgenic salmon bred to contain and transmit a growth hormone transgene; relative to control fish, transgenic salmon grow faster, consume food at higher rates, are more active and spend more time foraging in potentially dangerous locations (e.g. in proximity to a predator; Abrahams and Sutterlin, 1999). Salmonid growth hormone gene has the same effect on catfish, which grow faster but have poorer anti-predatory behaviour (Dunham *et al.*, 1999). A variation on this theme is that sawfly larvae ingest toxins that protect them from predators, but these same toxins reduce their growth rates (Bjorkman and Larsson, 1991).

Growth rate and mature function

There may also be a trade-off between instantaneous growth rate and capacity to perform mature function. Cells and tissues that are dividing or increasing in size cannot simultaneously perform mature function at peak efficiency (Ricklefs, 1979; Ricklefs *et al.*,

1994, 1998; Arendt, 2000). As a result, at the same body size, individuals with a high intrinsic growth rate will perform a variety of functions less efficiently than slow-growing animals (Morgan *et al.*, 2000). For instance, at the same length, fast-growing animals swim less efficiently than slow-growing animals (Kolok and Oris, 1995; Gregory and Wood, 1998, 1999). Transgenic coho salmon grow more than twice as fast by length as control fish, but have critical swimming speeds half those of control fish of the same length (Farrell *et al.*, 1997).

Compromises between growth rate and somatic development

High growth rates may lead to the formation of bodies that are more vulnerable to a variety of sources of mortality. In birds, there is a developmental conflict between rates of bone growth and bone strength or rates of bone deformity (Carrier and Leon, 1990; Emmerson, 1997; Leterrier and Constantin, 1999), while in pumpkinseed fish, fast-growing individuals delay ossification relative to slower-growing conspecifics (Arendt and Wilson, 2000). Similarly, fast-growing salmon are more likely to develop coronary arterial lesions over all ages than are slower-growing individuals (Saunders *et al.*, 1992). Other trade-offs have been reported between growth rate and developmental stability (Leamy and Atchley, 1985).

Growth rate and immune function

We expect a trade-off between growth rate and immunological competence, because of the substantial nutritional and energetic costs associated with immunological stress and the maintenance of an efficient immune system (Lochmiller and Deerenberg, 2000); even minute exposures to pathogens (e.g. exposure to microbes that normally live in the gut) evoke immune responses that significantly reduce growth rates. Furthermore, many of the cells in the immune system are highly nutrient-demanding; for example, macrophages turn over adenosine triphosphate at rates comparable to those of maximally functional heart muscle. In laboratory studies, immune-growth trade-offs are most obvious when animals are not maintained on *ad libitum*, nutritionally rich diets.

A general concern in the poultry industry is that strains of chickens and turkeys artificially selected for high growth rates are more susceptible to a variety of pathogens than strains selected for other traits, such as high rates of egg production (Nestor *et al.*, 1996; Reddy, 1996; Bayyari *et al.*, 1997; Praharaj *et al.*, 1999). The same inverse relationship between growth and susceptibility to pathogens may be true for other domestic animals, such as cattle (Frisch and Vercoe, 1984).

Growth rate and resistance to physiological stressors

A field study of *Amphibolurus* lizards showed that, within a given age cohort, slow-growing animals survived at higher rates than fast-growing animals, especially during periods of drought (Bradshaw, 1971). Physiological studies have indicated that slow-growing individuals are much better at resisting the elevated plasma sodium concentrations and other physiological consequences of water deprivation than fast-growing individuals (Bradshaw, 1970). Similarly, mice that have been selected for resistance to dietary toxins grow more slowly on a toxin-free diet than mice selected for susceptibility to dietary toxins (Hoenboken and Blodgett, 1997).

EMPIRICAL EVIDENCE FOR GROWTH–MORTALITY TRADE-OFFS FOR INDIVIDUALS LIVING SYMPATRICALLY

To date, studies of growth–mortality trade-offs have focused on different populations, or on individuals experiencing different environmental conditions, so we generally lack evidence concerning growth–mortality trade-offs for individuals living together. However, Gotthard (2000) generated variation in larval growth rates in the butterfly *Pararge aegeria* by manipulating photoperiods early in development, and then demonstrated that predation rates were higher for fast-growing larvae than for slow-growing larvae living on the same host plants.

Indirect evidence of growth–mortality trade-offs comes from fisheries biologists, who have developed techniques to estimate life-history parameters for different individuals captured together. These techniques involve counting growth rings on scales or otoliths to back-compute age at capture, and using width of annual growth-zones to compute size at previous ages and hence growth rates prior to capture (Ricker, 1979; Das, 1994). Early applications of this technique revealed that, when fish of different ages were captured in the same samples, back-calculated sizes at any given age were often larger for younger individuals than for older individuals (Lee, 1912). In other words, older individuals in the sample appeared to have grown more slowly when young than was the case for younger individuals in the same sample. Several hypotheses to explain Lee's phenomenon have been advanced. Current evidence suggests that this pattern is due to an inverse correlation between growth and mortality rates across individuals, such that slow-growing individuals make up more of the older age groups than do fast-growing individuals (see reviews in Lee, 1912; Das, 1994; Shuter *et al.*, 1998). This has been found for heavily fished populations, such as cod, in which fast-growing individuals are removed at higher rates than slow-growing individuals (Kristiansen and Svasand, 1998), but similar results have also been obtained for species like long rough dab, in which direct fishing is non-existent and incidental take mortality is extremely low (Fossen *et al.*, 1999). Thus, even in the absence of human intervention, fast-growing individuals have lower survival rates than slow-growing individuals living sympatrically.

FITNESS CONSEQUENCES OF GROWTH–MORTALITY TRADE-OFFS

We now examine the consequences of growth–mortality trade-offs by extending the model of Stamps *et al.* (1998) with respect to growth rates for individuals living sympatrically. We assume that growth in skeletal length is described by a two-stage Von Bertalanffy equation. Before maturity, individuals grow according to

$$\frac{dL}{dt} = \phi(q - aL(t)) \quad (1)$$

where $L(t)$ is length at age t and ϕ , q and a are parameters. Parameter a measures the cost per unit length of growth and parameter q is an index of maximum growth rate; asymptotic size is q/a . Parameter ϕ ($= 1$ in Stamps *et al.*, 1998) is an index of overall growth rate; we call it the relative growth rate or growth parameter. We treat q and a as fixed. We allow ϕ to vary across individuals; whether it is viewed as genetic or phenotypic variation is immaterial for the main argument of this paper.

After maturity individuals continue to grow (Beverton, 1992), but at a reduced rate, with the amount of reduction determined by the size at maturity:

$$\frac{dL}{dt} = \phi \left(q - \left(a + \frac{b}{L_m} \right) L \right) \quad (2)$$

where $L_m = L(t_m)$ is the length at maturity, t_m is the age at maturity and b measures the additional growth cost associated with maturity. For purposes of illustration, we use parameter values approximating life histories of brown trout *Salmo trutta* or Arctic Charr *Salvelinus alpinus* (Mangel, 1996). For simplicity, we denote length by $L(t|\phi, t_m)$.

We use the Euler-Lotka equation to provide a measure of fitness assuming annual reproduction; more advanced methods (Houston and McNamara, 1999; Clark and Mangel, 2000) add complexity of analysis without affecting the qualitative points.

We let $s(t|\phi, t_m)$ and $f(t|\phi, t_m)$ denote the survival to age t and fecundity at age t , respectively, of an individual with growth multiplier ϕ and age at maturity t_m . Given these, the Euler-Lotka equation is

$$\sum_t e^{-rt} s(t|\phi, t_m) f(t|\phi, t_m) = 1 \quad (3)$$

We assume that fecundity is given by the allometric relationship

$$f(t|\phi, t_m) = s_e L(t|\phi, t_m)^B \quad (4)$$

where s_e is the probability that an offspring survives to reproductive adulthood and B is an allometric parameter, measured across individuals of the same species, relating length and fecundity.

For simplicity, we assume size-independent mortality so that

$$s(t|\phi, t_m) = s(t-1|\phi, t_m)(1 - \phi M) \quad (5)$$

where M is the per period mortality. The model is easily adapted for size-dependent mortality, as happens in most aquatic environments, but doing that adds a layer of complexity to the presentation and analysis. Also for simplicity, we have assumed a linear relationship between the growth parameter and the intensity of mortality. It is possible that the relationship is non-linear. For example, Turner (1997) found that a two-fold increase in growth rate in a pulmonate snail led to an eight-fold increase in daily mortality rate, while Gotthard (2000) found that a four-fold increase in relative growth rate in butterfly larvae was associated with a 30% increase in daily predation rate.

Base case parameters are shown in Table 1. For each value of ϕ , there is a corresponding optimal age at maturity that maximizes the measure of fitness r . Over a ten-fold range of growth parameter, age at maturity varies between 1 and 5 years (Fig. 1a) and fitness shows a peak as a function of growth parameter. However, the peak of the fitness curve is relatively flat (Fig. 1b), suggesting that a variety of different growth parameters produce virtually the same fitness (see also Mangel and Clark, 1988, p. 285).

These results are based on several simplifying assumptions, such as that individuals can precisely and accurately measure the mortality rates. However, if individuals vary slightly in their estimates of environmental variables such as mortality rates (Bouskila and Blumstein, 1992), then the consequence will be a blurring of the line shown in Fig. 1b. For

Table 1. Parameters used in the model

q	maximum growth rate	50
a	cost of growth	0.2
b	additional cost of growth due to maturity	5
l_0	initial size	4
m	mortality rate	0.1
s_e	offspring survival to adulthood	0.0002
B	allometric parameter relating length and fecundity	1.5–2.8

example, in Fig. 1c, each value of growth parameter has associated with it 15 different values of M and s_e , allowing them to vary within 5% of the base case. The result is a band of virtually equal fitness that spans a two-fold variation in growth rate.

At the individual level, the model predicts that animals with different values of ϕ will mature at different sizes, but grow to approximately the same asymptotic size (Fig. 2). Hence, variance in growth rates weakens the strong positive correlation between size at maturity and asymptotic size predicted by Stamps *et al.* (1998), under the assumption that all juveniles grow at the same rate.

We conducted tests of the model to variation in the base case parameters to verify that the flatness of the fitness surface versus growth parameter curve near the peak is general.

DISCUSSION

Theoretical analyses of relationships between growth rates and fitness traditionally focus on identifying the optimum growth rate for the members of a population living together. While optimum growth rates are also predicted in this study, we have focused on the shape of the fitness surface, rather than on the location of its highest point. Our results suggest that, if there are strong trade-offs between growth and mortality rates, the fitness surface may be exceedingly flat, such that a wide range of growth rates yields virtually the same fitness. Furthermore, if we make the assumption that individuals lack perfect knowledge about mortality rates in their environment, and instead rely on proximate behavioural mechanisms that yield slightly different estimates of mortality rates for different individuals, then the result is a band of equal fitness over a wide range of growth rates.

Most previous explanations for the maintenance of individual differences have been based on frequency-dependent selection (Roff, 1998) or a balance between mutation and selection (Santiago, 1998). In contrast, we make no assumptions about frequency-dependence and we assume that growth rates are under very strong selection. However, we also assume that growth rates are under strong selection from processes that both favour and discourage high growth rates. Because these processes oppose one another, they produce a set of circumstances in which a wide range of growth rates yields virtually the same fitness. Whether a value $r = 0.110$ ‘really differs’ from $r = 0.111$ is a question that can be considered at many levels; this was one of the disagreements between Fisher and Wright (Provine, 1986). Theoreticians and empiricists may also have differing views about the significance of the shapes of the fitness surfaces that are generated as a result of growth–mortality trade-offs. However, in light of the simplicity of the model and the fact that

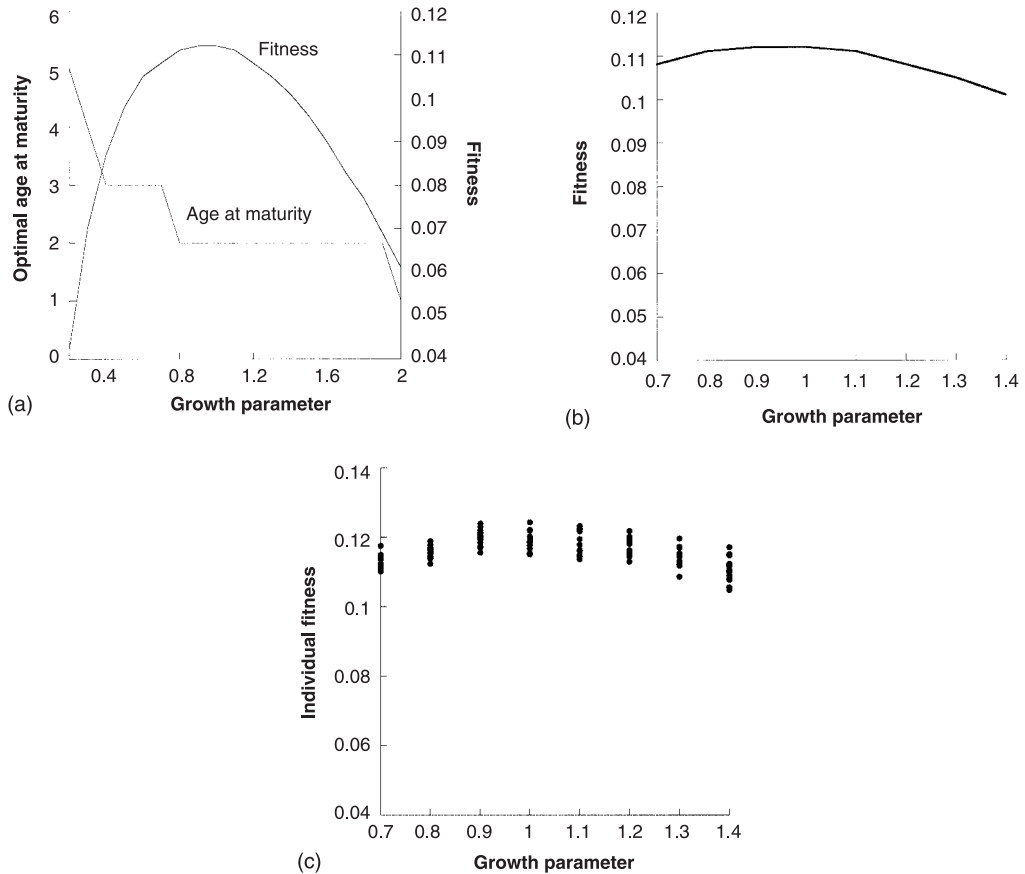


Fig. 1. (a) The age at maturity and fitness (solution of the Euler-Lotka equation) over a 10-fold range in ϕ . (b) Near the peak, the fitness surface is very flat, allowing the possible persistence of different growth parameters for long periods of time. (c) When there is individual variation in mortality or offspring survival, the curve is replaced by a band of fitness values.

environments vary in both time and space, the persistence of multiple growth rates over ecological time is plausible (Mangel, 1991).

Relationships between other life-history parameters may be affected by individual differences in growth rates. For instance, Stamps *et al.* (1998) predicted a clear and inverse relationship between linear size at maturity and linear asymptotic size, based on the assumption that all individuals grow at the same rate before maturity. Adding individual variation in growth rate to that model blurs the predicted relationship between size at maturity and asymptotic size. Since many taxa with asymptotic growth after maturity also exhibit inter-individual variation in juvenile growth rates, it might be advisable to control for variation in growth rates when studying relationships between size or age at maturity and asymptotic size.

Many studies of juveniles have observed relationships between social interactions and growth, such that dominant individuals grow more rapidly than subordinates. Many

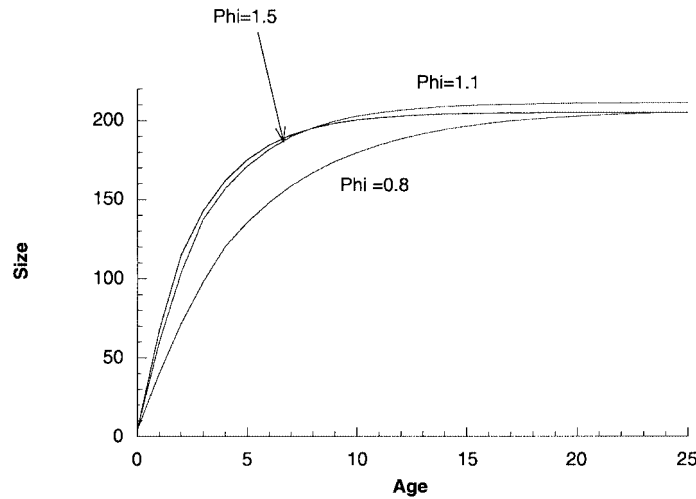


Fig. 2. Variation in growth rate can blur the strong prediction of Stamps *et al.* (1998) of an inverse relationship between age at maturity and asymptotic size. Here we modified the base case parameters to $B = 2.4$ and $s_c = 0.000002$ and show trajectories for $\phi = 1.5$ ($r = 0.107$), 1.1 ($r = 0.112$) and 0.8 ($r = 0.105$) with corresponding age of maturity = 2 years ($\phi = 1.5$), 3 years ($\phi = 1.1$) or 4 years ($\phi = 0.8$).

workers have observed relationships between dominance and growth, such that dominant juveniles grow more rapidly than subordinate ones (e.g. Stamps and Eason, 1989; Schuller, 1995). If one assumes a positive relationship between growth and fitness, it follows that individuals compete for high social status because this ensures access to resources that yield a high growth rate (Metcalf, 1993). Our approach provides a different perspective on this issue. If strong trade-offs between growth and mortality produce equivalent fitness for fast- and slow-growing individuals, then animals whose physiology is set for high growth may be more likely to compete aggressively for high social status, because they need more of the resources that are required to sustain high growth rates. In other words, the first (traditional) hypothesis argues that individuals grow faster because they are dominant, whereas the second hypothesis turns the causal arrow around and argues that individuals compete for dominant status because they are fast growers (Metcalf *et al.*, 1995). In addition, to the extent that initiating aggressive interactions increases mortality rates (e.g. because of the energetic, immune or elevated predation risk associated with aggression), then the aggressive behaviour associated with a high growth trajectory might contribute directly to inverse relationships between growth and survival rates.

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