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The evolution of growth trajectories: what limits growth rate?

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ABSTRACT

According to life-history theory, growth rates are subject to strong directional selection due to reproductive and survival advantages associated with large adult body size. Yet, growth is commonly observed to occur at rates lower than the maximum that is physiologically possible and intrinsic growth rates often vary among populations. This implies that slower growth is favoured under certain conditions. Realized growth rate is thus the result of a compromise between the costs and advantages of growing rapidly, and the optimal rate of growth is not equivalent to the fundamental maximum rate.

The ecological and evolutionary factors influencing growth rate are reviewed, with particular emphasis on how growth might be constrained by direct fitness costs. Costs of accelerating growth might contribute to the variance in fitness that is not attributable to age or size at maturity, as well as to the variation in life-history strategies observed within and among species. Two main approaches have been taken to study the fitness trade-offs relating to growth rate. First, environmental manipulations can be used to produce treatment groups with different rates of growth. Second, common garden experiments can be used to compare fitness correlates among populations with different intrinsic growth rates. Data from these studies reveal a number of potential costs for growth over both the short and long term. In order to acquire the energy needed for faster growth, animals must increase food intake. Accordingly, in many taxa, the major constraint on growth rate appears to arise from the trade-off between predation risk and foraging effort. However, growth rates are also frequently observed to be submaximal in the absence of predation, suggesting that growth trajectories also impact fitness *via* other channels, such as the reallocation of finite resources between growth and other traits and functions.

Despite the prevalence of submaximal growth, even when predators are absent, there is surprisingly little evidence to date demonstrating predator-independent costs of growth acceleration. Evidence that does exist indicates that such costs may be most apparent under stressful conditions. Future studies should examine more closely the link between patterns of resource allocation to traits in the adult organism and lifetime fitness. Changes in body composition at maturation, for example, may determine the outcome of trade-offs between reproduction and survival or between early and late reproduction. A number of design issues for studies investigating costs of growth that are imposed over the long term are discussed, along with suggestions for alternative approaches. Despite these issues, identifying costs of growth acceleration may fill a gap in our understanding of life-history evolution: the relationships between growth rate, the environment, and fitness may contribute substantially to the diversification of life histories in nature.

Key words: life-history evolution, trade-offs, compensatory growth, time constraints, condition, predation, physiology.

CONTENTS

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I. INTRODUCTION

Large body size is positively correlated with a number of fitness traits, including higher fecundity and offspring quality, mating success and longevity, though there are numerous exceptions (Shine, 1988; Roff, 1992; Honek, 1993; Andersson, 1994; Blanckenhorn, 2000; Bonduriansky, 2001; Sokolovska, Johansson & Rowe, 2000). This link between size and fitness is so well established that body size is frequently used as a surrogate for fitness when direct measurement of lifetime fitness is impractical or impossible. Assuming that selection on size is usually directional and positive, the most commonly cited constraint on size at maturity is development time (Kingsolver & Pfennig, 2004). Prolonged development time may reduce viability (particularly in seasonal environments) or reduce a population's inclusive fitness by increasing generation time (e.g. Schluter, Price & Rowe, 1991; Rowe & Ludwig, 1991; Abrams & Rowe, 1996; Nylin & Gotthard, 1998). On the other hand, animals that develop quickly can benefit in a number of ways, including higher intrinsic rate of increase, prolongation of the breeding season and improved access to resources, including mates (Sibly & Calow, 1986; Blanckenhorn & Demont, 2004).

Growth rates change quickly in response to environmental variation, and are closely linked to resource availability and foraging risk. Typically, animals that have free access to food mature both sooner and at a larger size. When resource levels decline, animals tend to grow more slowly, reaching maturity later and at smaller size (Morey & Reznick, 2000; Day & Rowe, 2002; Lind, Persbo & Johansson, 2008). Given that higher growth rates effectively decouple the trade-off between size and development time, it is sometimes assumed that animals grow at the maximum rate that is physiologically possible given resource availability. This assumption may seem reasonable at first glance, yet it begs the question of what constrains the evolution of faster intrinsic growth rates. Moreover, even animals with unlimited access to food are often observed to grow at rates lower than their fundamental maximum. This implies that the optimal growth rate may in some circumstances be lower than the maximum possible, and that high growth rates are costly.

In addition to growth rate plasticity, populations of a given species may also demonstrate local adaptive variation in growth rate depending on the local environment, and these differences in growth rate persist when animals are reared in common environments. The factors underlying genetic differentiation in growth rate among populations (such as increasing intrinsic growth rates along a latitudinal cline) parallel those favouring plastic increases in growth rate within populations (for example, higher growth rates observed in late-hatching animals). Thus, a closer study of plasticity in response to these environmental variables may contribute to a better understanding of the selective pressures acting on growth trajectories.

In short, growth rate plasticity is an apparently universal feature of animal life histories (Abrams & Rowe, 1996), and it is clear from the variation observed within and among populations that genetic variability in growth rates is also abundant (e.g. Riska, Atchley & Rutledge, 1984; Atchley & Zhu, 1997; Billerbeck, Lankford & Conover, 2001; Laugen

et al. 2003, Wilson *et al.*, 2007). Growth rate is a key life-history trait: rather than being shaped only indirectly *via* selection on age and size at maturity, it may itself affect individual fitness. The costs and benefits of accelerated growth may determine in part the optimal life-history strategy for individuals and species. Studies of the short- and long-term consequences of plastic acceleration of growth, in combination with comparisons among populations with different intrinsic growth rates, can be used to identify specific constraints that shape growth rate.

Herein, I synthesize current research on the ecological and evolutionary forces shaping growth rate. The advantages and costs of accelerated growth are discussed in turn, with particular emphasis on studies investigating direct costs of growth on adult fitness, which has only recently been the focus of empirical research. The majority of studies investigating constraints on growth rate use phenotypic manipulations to induce accelerated growth, or compare populations with different intrinsic growth rates reared under identical conditions. Both approaches have significant limitations, particularly with regards to identifying long-term costs of growth. However, careful experimental design and interpretation of data can overcome many of these issues.

II. GROWTH RATE IN LIFE-HISTORY THEORY

Optimization models are often applied to the study of life-history evolution and can be used to predict how trade-offs among life-history traits respond to environmental change. Suites of life-history traits are assumed to evolve in response to internal trade-offs, such as between development time and mortality, or between development time and size at maturity. For example, age and size at maturity are often assumed to be in direct conflict, such that the optimal time to mature is the one that maximizes the fecundity benefits of reaching a large size while minimizing the cost of delaying reproduction. The resolution of these trade-offs will depend both upon the specific characteristics of the organism as determined by its evolutionary history, as well as on local environmental conditions. Local conditions can influence the shape of the relationship between a trait and fitness. Models in which these functions are varied can be used to identify the most environmentally sensitive traits and to develop predictions about how changing environmental variables such as time constraints, predation and food availability will affect the resolution of trade-offs between life-history characters and thus the optimal trait value. Typically in life-history optimization models, overall fitness is a positive function of body size, which is itself assumed to be positively correlated with reproductive success (Rowe & Ludwig, 1991; Abrams & Rowe, 1996). It is also a decreasing function of development time, since juvenile mortality increases with time (Roff, 1980). The trade-off between age and size has been the subject of extensive theoretical analysis (Stearns & Koella, 1986; Kozlowski, 1992; Roff, 1992; Stearns, 1992; Day & Rowe, 2002). However, many models do not permit flexible growth rates. In these models, the optimal growth rate is equal to, or greater than, the physiological maximum. However, such flexibility may be necessary to explain fully the diversification of life-history strategies observed, particularly because variation in maturation traits is often less than expected (e.g. Bjorndal *et al.*, 2003). Considering flexible growth produces a more realistic model of life-history optimization. In such models, constraints on growth are usually modeled as instantaneous survival costs, since faster-growing individuals are assumed to experience an increased risk of predation. However, it is increasingly recognized that non-predation costs of growth may also shape life histories, and that these costs may be incurred across longer time scales that are typically modeled. The major ecological factors influencing growth rates include food availability, predation risk of foraging (including the shape of the relationship, size-selective effects and indirect effects of predation on access to resources), and time constraints (Table 1). In the following discussion, I review the predictions of life-history optimization models that include flexibility in growth rate. These models offer useful predictions regarding growth rate optimization in different ecological contexts.

(1) Models of growth: when should growth rate be accelerated?

Apart from resource variation, to which growth rates are extremely sensitive, models also investigate how changes in other ecological variables influence growth trajectories. Growth rates are also expected to vary in response to time constraints on development time, such as those that must arise in seasonal environments. Imposing a time constraint effectively intensifies the trade-off between age and size at maturity. When growth rate is fixed, the optimal size at maturity decreases as the season progresses (Rowe & Ludwig, 1991). However, a more complicated picture results when growth rates are flexible. Typically, juvenile mortality risk is modeled as an increasing function of growth rate to reflect a realistic conflict between reaching a large body size and limiting predation risk for active foragers (Houston, McNamara & Hutchinson, 1993; Abrams *et al.*, 1996). The outcome of the trade-off is mediated by other environmental factors or by simplifying assumptions in the model, such as a fixed age or size at maturity. When maturation is assumed to occur at some fixed target size, the optimal growth rate generally increases as season length declines (Houston *et al.*, 1993). When both development time and maturation size are permitted to vary in response to a time constraint, development time typically decreases, but growth rate either increases or stays the same depending on the sensitivity of growth rate to risk (Abrams *et al.*, 1996). If animals are under additional pressure to mature quickly due to a decreasing window of time in which to complete development, either growth rate must increase, or maturation size must decrease (Ludwig & Rowe, 1990; Rowe & Ludwig, 1991; Abrams & Rowe, 1996).

Increasing predation risk usually causes a reduction in the optimal growth rate (see Section II.2*a*). However, under

Table 1. Predictions of theoretical models for the effects of environmental variables on growth

Variable	Effect on growth rate	References
Immediate effects		
Time constraint	+(=)	Sibly *et al.* (1985); Abrams *et al.* (1996); Ludwig & Rowe (1990)
Time constraint + predation risk	+/− Outcome depends on shape of mortality-growth rate relationship	Abrams *et al.* (1996); Werner & Anholt (1993)
Predation risk (cumulative)	− Slower growth when growth rate is highly sensitive to predation risk	Abrams & Rowe (1996)
Predation risk (small prey preference)	+	Werner & Gilliam (1984); Day *et al.* (2002)
Predator-independent mortality	+	Sibly *et al.* (1985)
Indirect effects of predation	+ Fewer competitors leads to higher food availabilty	Werner & Gilliam (1984)
Food restriction	−	Roff (1992); Stearns (1992)
Delayed effects		
Delayed viability cost	+ Increasing the delay leads to a higher optimal growth rate	Yearsley *et al.* (2004)
Cumulative damage (long term)	+ Accelerated growth more likely later in development	Mangel & Munch (2005)
Trade-off between growth and a signal trait	− Lower optimal growth rate as fitness benefit of signal trait increases	Lindstrom *et al.* (2005)

some conditions, predation can actually lead to a prediction of growth acceleration. First, indirect effects of predation (e.g. thinning) can increase the amount of food available *per capita* in food-limited environments (Werner & Gilliam, 1984; Ernsting *et al.*, 1999). Theory also suggests that predator-independent mortality favours higher foraging rates (and thus higher growth rates) because it increases the cost of delaying maturity (Werner & Anholt, 1993). Second, size-selective predation is predicted to favour higher growth rates in some conditions. Although the instantaneous risk of predation is higher for fast-growing individuals, when risk of predation is highest for smaller size classes fast growth might reduce the total cumulative risk of mortality (Werner & Gilliam, 1984; Day *et al.*, 2002). Similarly, Abrams *et al.* (1996) modeled juvenile mortality as a decelerating function of growth rate (effectively simulating a predator preference for small individuals), and showed that both growth rate and body size were greater than when mortality was an increasing function of time.

(2) Models of growth: factors that limit growth rate

Growth may incur costs over different time scales. Costs such as increased predation risk occur immediately, and are concurrent with the period of growth. Long-term costs, by contrast, are defined as fitness costs imposed after growth has ceased, and are of distinct interest because they are capable of producing the submaximal growth that is sometimes observed in benign conditions.

(*a*) *Short-term costs of fast growth*

The most common type of short-term cost for growth acceleration in real populations is increased predation risk. Because foraging activity is often positively correlated with predation risk, organisms generally decrease foraging time, activity, or time spent in risky habitats when predators are present (Sih, 1982; Lima & Dill, 1990). Rates of juvenile mortality independent of predation are also known to be higher in fast-growing populations or selection lines of insects. This indicates that growth rate trades off with tolerance to subsequent periods of food stress (Gotthard, Nylin & Wiklund, 1994; Teuschl, Reim & Blanckenhorn, 2007). Abrams & Rowe (1996) modeled the effect of increasing predator density on the optimal growth rate and age and size at maturity, and showed that as predator density increases, optimal growth rate declines.

(*b*) *Costs of accelerated growth (long term)*

Optimization models that consider only those costs of growth imposed during the period of growth provide a limited view of the factors constraining growth rate. First, these models of immediate growth costs do not account for the submaximal growth rates that are frequently observed when predators (or other immediate risks) are absent (Metcalfe & Monaghan, 2001; Ali, Nicieza & Wootton, 2003). By considering only costs imposed during the period of growth, models lack a key constraint on growth rate. Fitness may not be adequately estimated from survival to, or size at, maturity. Variation in components of fitness during the adult stage should also be considered when constructing optimization models. Ignoring long-term costs is likely to result in overestimation of the optimal growth rate. Such costs could explain why growth trajectories are often lower than can be explained by juvenile mortality regimes alone.

Long-term costs of growth are assumed to arise from the inevitable conflict among different traits drawing on

the same finite pool of energy. Growth consumes a large proportion of an animal's resource intake (e.g. Wieser, 1994; Peterson, Walton & Bennett, 1999; Bayne, 2000), and accelerating it requires additional energy that must come at the expense of allocation to other traits and functions. This could conceivably have long-term consequences for fitness *via* changes in allocation to maintenance or reproductive reserves. Two studies (Yearsley, Kyriakis & Gordon, 2004; Mangel & Munch, 2005) modeled the effect of delayed costs on the optimal growth rate, with the usual assumption that fecundity increases with body size. In both cases, the cause of accelerated growth was a brief period of growth suppression imposed during early development, followed by a period of accelerated "compensatory" growth. Mangel & Munch (2005) hypothesized that growth rates might be linked to longevity *via* the accumulation of reactive oxygen species (ROS). Faster growth is thought to increase the rate of ROS production, which in turn reduces lifespan due to the resulting oxidative damage (Mangel & Munch, 2005). Yearsley *et al.* (2004) did not explicitly define the nature of the long-term costs in their model, but note that they might come in the form of trade-offs between growth rate and immune response or growth rate and somatic development.

Mangel & Munch (2005) modeled growth as a function of size, activity-related resource acquisition, damage and energy required to repair damage. Assuming that reproductive success increases with body size, accelerated growth was most likely to occur late in development, particularly when rate of damage was high or repair rate low. This result is intuitive given that the model assumes that growth-related damage is cumulative; the later it is incurred, the lower the probability that an individual will die prior to reproduction. More surprisingly, given that compensatory growth acceleration is so frequently observed in natural populations, the model predicted that it would occur only rarely. One possible explanation is that this model used a fixed development time. Flexible development time may be a critical component of realistic models of compensatory growth; for example, accelerated growth may be particularly advantageous under time constraints.

Yearsley *et al.*'s (2004) model varied the duration of periods of food deprivation and examined their effect on the trade-off between growth rate acceleration and delay in maturation. Mass at maturity was fixed, and the rate of growth was discounted by a viability cost. The timing of the imposition of this cost was varied in order to determine the effect of delaying the cost of increasing growth. In general, the longer the delay, the higher the optimal rate of growth during the period of compensation. This follows intuitively from the observation that the strength of selection decreases with age (Medawar, 1952).

Interestingly, the measure of fitness used in the model determined whether accelerated growth was expected to occur. When R_0 (reproductive rate) was the measure of fitness, accelerated growth was predicted for a wide range of parameter values. By contrast, if fitness was modeled as r (the intrinsic rate of population increase), accelerated growth was not produced. Yearsley *et al.* (2004) suggested that, in biological terms, this result is due to the advantage of late-life reproduction under density-dependence when modeling fitness in terms of the population. The effect may also explain why Sibly *et al.* (1985), modeling r, found that faster growth was never expected unless a time constraint (favouring earlier maturation) was included in the model. In most of the species in which compensatory growth acceleration has been studied, some type of time constraint is probable (e.g. seasonality in temperate species; Arendt, 1997; Metcalfe & Monaghan, 2001; Ali *et al.*, 2003). Thus, the evolution of compensatory growth acceleration may require the existence of a time constraint.

Although both models (Yearsley *et al.*, 2004; Mangel & Munch 2005) include a fitness benefit for increasing body size, Lindstrom, Metcalfe & Royle (2005) extended the general model by explicitly dividing reserve energy into maintenance, growth of somatic tissue (which is assumed to correlate with survival) and production of sexual ornaments (which is assumed to improve reproductive success). Their model predicts that compensation for body size may not be prioritized when the fitness benefit of increasing allocation to sexual ornaments is sufficiently great. However, this assumes that reproductive fitness depends on the size of the ornament and is independent of body size, which may not hold in a large number of species, because ornament size appears to depend strongly on underlying condition (Zahavi, 1975, 1977; Grafen, 1990). Further exploration of how available energy is allocated among components of fitness is certainly of interest, particularly in terms of allocation to reproduction *versus* survival. Resource allocation models (e.g. Broekhuizen *et al.*, 1994; Gurney *et al.*, 2003) suggest the importance of considering different components of size separately. Dividing size into two classes, reserves (mobilizable) and structural size (fixed), produces an outcome for compensation of reserve and structural size that is much closer to the pattern observed in real populations of fish. Plasticity in allocation to reserve and structural growth can have numerous implications for future fitness, since the latter is not available for metabolism (during subsequent periods of food restriction) and structural size is fixed at maturity for taxa exhibiting determinate growth. These results suggest that optimization might consider how energy is allocated between mobilizable and fixed tissues and how the optimal allocation to structure *versus* mass might differ depending on seasonality (i.e. onset of winter may favour fat reserves over structural size) or other environmental variables.

In summary, existing models for the optimization of age and size at maturity may provide a useful basic framework for studying the causes of, and constraints on, growth rate acceleration. Growth acceleration is most likely to occur when time constraints (either on development time or on the reproductive period) are imposed, though this effect may be moderated by the presence of predators. In general, activity-related mortality is expected to constrain growth rate, while sources of mortality that are unrelated to resource acquisition and growth may have the opposite effect (Sibly *et al.*, 1985).

More recent models examining how growth rate might be optimized considering long term costs suggest that such costs have the potential to play a key role in the evolution of juvenile growth trajectories observed in nature, and that compensatory growth is most likely when time is somehow constrained (Yearsley *et al.*, 2004; Mangel & Munch, 2005).

III. EMPIRICAL STUDIES OF FLEXIBLE GROWTH: CAUSES AND COSTS

In natural and laboratory populations, growth rate variation is observed in response to a range of environmental factors, including resource availability, predation risk, competition, time constraints and population density (reviewed in Lima & Dill, 1990; Arendt, 1997; Nylin & Gotthard, 1998). Each of these factors can vary considerably within a single generation, contributing to selection for high levels of phenotypic plasticity in growth trajectories. Experimental manipulation of these environmental variables can be used to produce faster rates of growth in accordance with the theoretical predictions outlined in the previous section. Growth trajectories are also known to diverge among populations along ecological gradients. For example, populations along a latitudinal cline may have higher intrinsic growth rates at higher latitudes due to the decreasing length of the growth season. This leads to the question of what constrains the evolution of higher rates of growth at lower latitudes.

In the following sections, the results of empirical studies investigating the factors underlying growth rate variation are reviewed, beginning with a discussion of the ecological conditions that favour accelerated growth. In the second part, I review evidence of direct fitness costs of growth acceleration from both phenotypic manipulation studies and comparisons of populations with different intrinsic growth rates. Some limitations of the experimental designs typically used in the study of constraints on growth rate are addressed, along with a discussion of future avenues for research, particularly at the intersection of physiology and life-history evolution.

(1) Benefits of rapid growth

(a) *Phenotypic manipulations: compensatory growth*

Variation in resource availability is virtually ubiquitous in nature, and growth is extremely sensitive to prevailing conditions. When food is restricted, growth rates often decline rapidly and immediately and available resources are allocated to maintenance. Equally, when conditions improve, growth rates quickly recover (e.g. Broekhuizen *et al.*, 1994). In some cases, the growth rate achieved may significantly exceed that of controls fed *ad libitum*, allowing growth trajectories to converge by the end of the growth period. Acceleration of growth rate in a way that reduces or eliminates the difference in final body size is referred to as "compensatory" or "targeted" growth (Tanner, 1963; Monteiro & Falconer, 1966; Metcalfe & Monaghan, 2001), though the latter term has also been used to describe a genetic phenomenon in which genetic variance in a trait decreases as the animal reaches its maturation size (Riska *et al.*, 1984). For the purposes of this review, I use the term compensatory growth in reference to a significant increase in the rate of growth relative to that normally achieved under non-limiting conditions. Because it has the effect of reducing the phenotypic variation generated by food or temperature stress (e.g. Bjorndal *et al.*, 2003), compensatory growth can be viewed in terms of canalization. Canalized traits remain constant in a range of environmental or genetic backgrounds, though more variation may become apparent under extreme conditions. This robustness of traits despite environmental perturbation may evolve if the optimal range of trait values is narrow or relatively insensitive to the environment (de Visser *et al.*, 2003). In the case of body size, adult size is expected to experience stronger selective pressure than size at any earlier point during development for two reasons. First, adult size corresponds closely to fitness, and second, growth is asymptotic or ceases at maturity. As a result, adult size appears to be less sensitive to environmental perturbations relative to size at earlier stages (Riska *et al.*, 1984; Flatt, 2005).

Compensatory growth is a common phenomenon and has been observed in a range of taxa following periods of low food or temperature that suppress growth rate relative to unstressed animals (e.g. Arendt, 1997; Nicieza & Metcalfe, 1997; Maclean & Metcalfe, 2001; Ali *et al.*, 2003; Hurst *et al.*, 2005; Huang, Wei & Zhang, 2008). Once the food or temperature restriction has been lifted, animals accelerate growth rate beyond that of controls (e.g. Fig.1A), causing them to mature at or near the same size.

Compensatory growth acceleration following a period of food restriction or deprivation is well known among vertebrates. Usually, accelerated growth is fueled by increased food intake (Johansson *et al.*, 2001; Ali *et al.*, 2003; Berger & Gotthard, 2008), but this is not always the case (Boujard *et al.*, 2000; Dmitriew, Cooray & Rowe, 2007). Fish in particular have been the subject of extensive study (reviewed by Ali *et al.*, 2003; see also Carlson, Hendry & Letcher, 2004; Nikki *et al.*, 2004; Royle, Lindstrom & Metcalfe, 2005; Àlvarez & Nicieza 2005; Johnsson & Bohlin, 2005; 2006; Myszkowski, Kaminski & Kamler, 2006), as have a few isolated species of frogs and birds (Bize, Metcalfe & Roulin, 2006; Arnold *et al.*, 2007; Capellan & Nicieza, 2007; Criscuolo *et al.*, 2008). Invertebrates also appear to be a highly tractable system for the study of compensatory growth; several species demonstrate a strong capacity for compensatory growth, including mites, damselflies, butterflies, caddis flies and beetles (Hakalahti, Bandilla & Valtonen, 2005; Shama & Robinson, 2006; Strobbe & Stoks, 2004; Tammaru *et al.*, 2004; Dmitriew & Rowe, 2005, 2007).

Humans and other mammals are also known to accelerate growth in order to catch up following periods of stress early in development (e.g. Roseboom *et al.*, 2000; McMillen & Robinson, 2005; Ozanne & Hales, 2004, 2005). Studies investigating growth acceleration in humans and models such

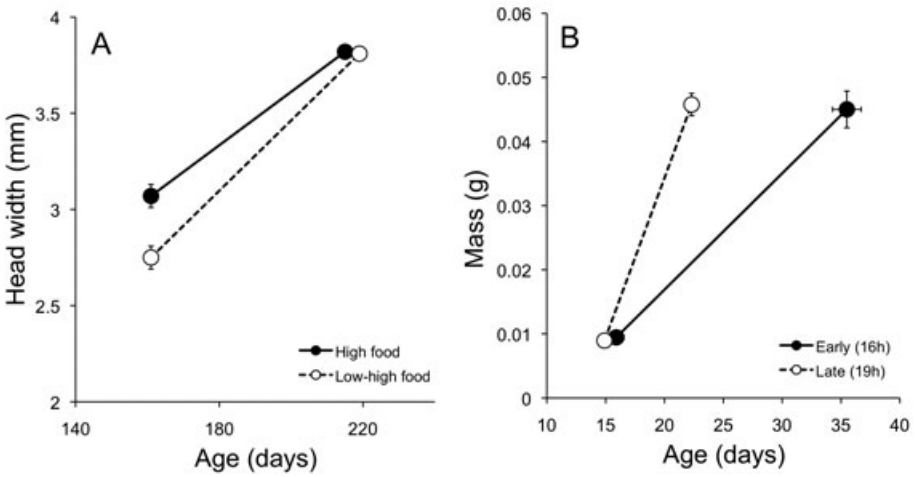

Fig. 1. Growth acceleration induced by manipulation of resource levels and time of season. (A) Growth in the damselfly *Ischnura verticalis* following a period of food restriction (open circles, broken line) and in a group reared at unrestricted food throughout development (filled circles, solid line). Values are means +/− S.E.M.; growth rate (slope) differs significantly between treatments ($F_{1,95} = 10.9$, $P = 0.0014$). Modified from Dmitriew & Rowe (2005). (B) Third-instar growth rate in the butterfly *Pararge aegeria*; third-instar growth rate depended on perceived photoperiod. Larvae grew significantly faster in the late season (19h:5h L:D) photoperiod treatment (open circles, dashed line) than in the early season (16h:8h L:D) photoperiod (filled circles, solid line). Values are means +/− S.E.M; relative growth rate differs significantly between treatments ($F_{1,18} = 84.8$, $P < 0.001$). Modified from Gotthard (2000).

as rats and mice are generally more mechanistic and focus on the specific physiological pathways underlying accelerated growth. The pathways underlying growth are now being described in increasing detail with the goal of understanding how early life conditions influence human health in the long term. As such, they may provide useful information as to how accelerated growth might be translated into fitness costs over the long term. One key area of research is how early resource conditions are linked to overall fitness *via* insulin sensitivity. Insulin is a hormone central to metabolic processes that controls growth efficiency and resource storage to a large degree. Animals initially exposed to restrictive growth conditions tend to exhibit faster growth than controls when resources are abundant; changes in the insulin-signaling pathway are thought to be largely responsible (reviewed in McMillen & Robinson, 2005).

(*b*) *Phenotypic manipulations: time constraints*

In species inhabiting seasonal environments, individuals are under constant pressure to reach maturation or other critical developmental milestones within a limited window of time. For example, at increasing latitudes, the length of the productive season decreases, leading to stronger pressure on growth and development rates (Blanckenhorn & Demont, 2004). However, predicting the effects of time constraints on growth rate may be complicated by interactions among factors such as season length, environmental productivity and the number of generations per year (as a sudden shift in voltinism can actually favour slower growth rate at very high latitudes) (Mousseau, 1997; Blanckenhorn & Demont, 2004). Many animals are highly sensitive to reliable cues, such as photoperiod, indicating time of season. It is expected that a forward-shifted photoperiod should elicit accelerated growth because the subject will perceive a reduced window of time in which to complete development (Rowe & Ludwig, 1991; Abrams & Rowe, 1996). Even organisms in tropical environments may be subject to time constraints since certain seasons are more conducive to reproduction or growth than others (e.g. Brown & Shine, 2006).

Deteriorating conditions such as declining water or food levels or increasing population density also act as a form of time constraint. Species having an aquatic juvenile stage may further be subject to hydroperiod constraints, since longer development time increases the risk that the pool of water will dry out before an individual reaches maturity. Analogously, faster growth may also be observed in conditions where reaching a size threshold quickly is always advantageous, either because it represents a size refuge from predators (Sibly *et al.*, 1985; Arendt, 1997; Day *et al.*, 2002) or because individuals become able to access larger and more productive prey classes (e.g. Arendt & Wilson, 1997; 2000).

(*i*) *Photoperiod.* A number of experimental studies have used manipulation of the photoperiod to test hypotheses about how time constraints affect the trade-off between size and time of maturity, though they are for the most part limited to insects. Insect growth trajectories are often sensitive to photoperiodic cues, which are highly reliable signals of point in the season. In all species that have been studied to date, the primary effect of manipulating photoperiod is for development time to decrease as the season progresses and for size at maturity to be reduced (e.g. Johansson *et al.*, 2001; Gotthard, 2004; Shama & Robinson, 2006). However, this does not mean that growth rates are not accelerated,

only that growth acceleration is insufficient to match body size to that reached by the control treatment. In fact, there is some empirical evidence, summarized below, that growth rate itself does vary in response to photoperiod manipulation.

In butterflies, larval growth rates are greater for individuals experiencing long daylength ("late" photoperiod) compared to short daylength ("early" photoperiod) (Fig. 1B; Nylin, Gotthard & Wiklund, 1996; Gotthard, 2000). A similar effect was observed for weight gain in the alpine beetle *Oreina elongata* and in caddis flies (Margraf, Gotthard & Rahier, 2003; Shama & Robinson, 2006). Among odonates, however, there is little evidence that growth rate increases substantially in individuals experiencing a "late" photoperiod (e.g. De Block & Stoks, 2004), although this may be a consequence of how growth is measured. In a variety of damselfly species, larvae experiencing a "late" photoperiod matured at a lower mass than those reared under an early photoperiod (Johansson & Rowe, 1999; Johansson *et al.*, 2001; De Block & Stoks, 2004; Strobbe & Stoks, 2004). In one study investigating time constraints in the damselfly *Enallagma cyathigerum*, both mass and head width were assessed. As in other studies, accumulation of mass was not accelerated. However, growth rate of head width was higher in the late photoperiod group (Strobbe & Stoks, 2004). Thus, it is possible that the capacity of damselflies to increase growth rate in response to time constraints depends on whether size is measured as mass or as structural size (see Section III.2*b* and Nicieza & Àlvarez, 2009).

Intriguingly, De Block & Stoks (2004) found that rate of mass gain did increase significantly among eggs collected from natural populations of the damselfly *Lestes viridis* late in the season relative to clutches collected earlier in the year. In this species, photoperiodic cues did not affect mass growth rate, suggesting that while damselflies hatching late in the season are physiologically capable of accelerating growth, photoperiod manipulation alone is insufficient to induce this response. Higher growth rates of late-hatching eggs may therefore require some unknown environmental cue or maternal effect to be induced.

(*ii*) *Predation.* Theory predicts that the presence of predators will usually favour slower growth rate because increasing foraging activity tends to increase vulnerability to predation (Werner & Anholt, 1993; Abrams & Rowe, 1996). In some special cases, however, predation may exert a pressure that is analogous to that of a time constraint: species that grow more rapidly might reach size refugia sooner, thereby reducing the cumulative risk of predation (Werner & Gilliam, 1984; Abrams & Rowe, 1996; Rudolf & Rodel, 2007). Nevertheless, even though accelerated growth might theoretically reduce overall exposure to predation, most studies that experimentally increase predation risk show that foraging rate declines, and consequently that development time increases and maturation size decreases (Lima & Dill, 1990; Benard, 2004). Gape-limited predation may serve as an exception to this pattern, selecting for faster growth in the prey species (Urban, 2007). While high growth rates may reduce survival in the short term, reaching a size refuge at an earlier age can reduce the cumulative mortality risk. As predicted under this scenario, Urban (2008) demonstrated that salamanders originating from ponds with high densities of gape-limited predators grew faster than those from populations with a lower predation risk when reared in a common environment. Similar results have been found among populations of pumpkinseed sunfish (*Lepomis gibbosus*), which appear to have evolved higher rates of growth when local predation risk is high (Arendt & Wilson, 1999). Few manipulative experiments have demonstrated accelerated growth rates in response to increasing predation risk. However, male house finches (*Carpodacus mexicanus*) exposed to nest ectoparasites exhibited higher rates of growth than did non-parasitized males; individuals that grow faster fledge sooner, thereby reducing the cumulative risk of contracting parasites (Badyaev *et al.*, 2006).

(*iii*) *Hydroperiod.* Hydroperiod is an important constraint in taxa such as odonates, flies and amphibians that complete juvenile development in aquatic environments. Drying rates vary by site, often unpredictably, which may contribute to the striking diversity of reaction norms for development time and size at maturity within and among such species. As with other forms of time constraint, experimentally increasing the rate of pond drying often causes a decline in development time (Juliano & Stoffregen, 1994; Altwegg, 2002; Laurila *et al.*, 2002; Merila, Laurila & Lindgren, 2004; DeBlock & Stoks, 2005; Shama & Robinson, 2006). The most common effect is for size at maturity to decrease as the hydroperiod is shortened. In all of these studies, size was measured as mass. A notable exception to the usual pattern of reduced size under hydroperiod constraint was in the mosquito *Ochlerotatus sticticus*, which emerged earlier when water levels were low but at the normal body size (Schafer & Lundstrom, 2006). In this experiment, body size was measured as wing length rather than mass, and may therefore represent another case in which an insect's allocation to structural size is prioritized even under stress.

Hydroperiod manipulations can also result in an underestimation of growth potential if the time constraint represented by decreasing water levels is confounded with correlated changes in resource availability, temperature or environmental quality. Generally, increasing temperature causes increased rates of growth (e.g. Atkinson, 1994); effects of increased population density, such as higher solute concentration or waste accumulation may have an opposing effect (e.g. Morey & Reznick, 2001, but see Juliano & Stoffregen, 1994). This problem can be minimized in the laboratory by careful control of food levels, solute concentrations (Juliano & Stoffregen, 1994) and temperature, which fluctuates to a higher degree as water levels fall, and which may have a variety of adaptive or non-adaptive consequences for growth rate (e.g. Frisch & Santer, 2004).

(*iv*) *Summary.* Among empirical studies, the most common effects of experimentally imposed time constraints are that development time and maturation size decline, as predicted by theory (Ludwig & Rowe, 1990; Rowe & Ludwig, 1991; Abrams *et al.*, 1996). However, despite theoretical

predictions that growth rate might be accelerated under size-selective predation (e.g. Sibly *et al.*, 1985; Abrams & Rowe, 1996), a survey of the empirical literature suggests that this is rare. This may be because in most cases the increased per unit growth risk outweighs the benefit of shortening the vulnerable period. Empirical evidence that growth is accelerated under time constraints is also limited, although a shortening of the developmental period is often observed. However, most experiments use mass as the sole measure of size, which may cause the potential for growth acceleration under time constraints to be underestimated. Experimental studies including some measure of structural size indicate that growth may be accelerated under photoperiod manipulation. Alternatively, this relative lack of evidence could be attributable to the evolutionary history of these organisms; those species inhabiting time-stressed or transient environments may be approaching physiological maxima or the limits of genetic variation for this trait (e.g. De Block, McPeek & Stoks, 2008*a*; but see Schultz, Lankford & Conover, 2002; De Block & Stoks, 2004). Environmental manipulations of conditions such as hydroperiod might also suppress growth incidentally due to correlated declines in the quality of the environment.

(*c*) *Comparing populations with different growth rates*

Intrinsic growth rates vary to a large extent among populations of a species, as well as within them. Common garden experiments demonstrate that intrinsic growth rates often increase along latitudinal or altitudinal clines. This implies that genetic variation in growth exists and that growth rates can evolve in both directions depending upon local selective pressures. These predictable evolutionary differences in growth trajectories parallel the plastic responses observed within populations exposed to variable conditions in laboratory settings. For example, just as experimental manipulations that simulate lateness of season can cause growth rate to be accelerated (e.g. Nylin *et al.*, 1996; Margraf *et al.*, 2003), growth rates tend to be higher with increasing latitude. By comparing populations along environmental clines, we can find corroborating evidence for those ecological pressures that are most important in shaping growth trajectories.

The majority of studies comparing growth trajectories among populations examine variation along latitudinal clines. Despite the fact that productivity tends to decline as latitude and altitude increases (Laugen *et al.*, 2003; Blanck & Lamouroux, 2007; De Block, McPeek & Stoks, 2008*b*), faster growth is nevertheless expected to evolve for several reasons. At high latitudes, the length of the season is shorter and there may be selection favouring large body size in colder temperatures (Bergmann clines; Blanckenhorn & Demont 2004; Gotthard, 2004). Furthermore, populations at higher latitudes may experience less intense selection against high foraging rates, since predator densities tend to be lower (e.g. Laurila, Lindgren & Laugen, 2008). Increasing growth rate at higher latitudes is well known among populations e.g. in insects, fish and frogs (e.g. Fig. 2; Conover & Present, 1990; Billerbeck, Schultz & Conover, 2000; Laugen *et al.*, 2003; Blanckenhorn & Demont, 2004; Lindgren & Laurila, 2005; Armbruster & Conn, 2006; Yamahira & Takeshi, 2008; Laurila *et al.*, 2008). Exceptions to this pattern may occur if changes in voltinism occur along the range studied. At some latitude, the season length becomes too short to support the number of generations per year that is optimal at lower latitudes. This results in saw-tooth patterns in growth rate along a latitudinal cline as the life history changes from bivoltine to univoltine or to semivoltine (Roff, 1980; Nylin & Gotthard, 1998).

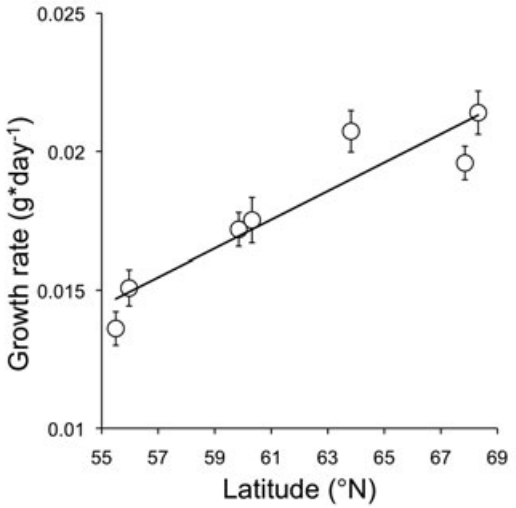

Fig. 2. Mean growth rate *versus* latitude of population of origin in the common frog, *Rana temporaria*, reared under common conditions (at 18 °C). Values are mean growth rate +/− S.E.M ($N = 15 - 20$ individuals per population). Modified from Lindgren & Laurila (2009).

Several studies in fish have investigated population differences in growth rate in greater detail and suggest that accelerated growth within populations at high latitudes is elicited by changes in behavioural and physiological traits such as activity level, metabolic rate or food conversion efficiency (Billerbeck *et al.*, 2000; Schultz *et al.*, 2002; Arnott, Chiba & Conover, 2006). A number of other ecological differences among populations may also lead to the evolution of different intrinsic growth rates. For example, Fraser *et al.* (2007) compared growth between populations of Atlantic salmon (*Salmo salar*) from similar latitudes that undergo different migratory distances. Longer migratory distance effectively shortens the time available for growth, causing that population to grow faster than short-distance migrants. Intrinsic variation in growth can also evolve in response to differing predation pressures. Arendt & Wilson (1997, 2000) found that populations of pumpkinseed sunfish originating from ponds containing a competitor species exhibited higher intrinsic growth rate than those inhabiting pumpkinseed-only ponds. This result is in accordance with the prediction that pressure to reach a size refuge (whether from competition or predation) favours higher rate of growth. Similar studies comparing populations with size-specific predation might

also be conducted to test the prediction that faster growth will be expressed when predation risk for small individuals is higher (e.g. Werner & Gilliam 1984; Abrams *et al.*, 1996). For example, certain insect species may inhabit aquatic systems that contain fish as well as those that do not; in the latter case, predation may originate primarily from larger size classes preying on smaller individuals.

(2) Costs of accelerated growth

There is an increasing amount of empirical evidence to suggest that accelerated growth comes at a cost to fitness. These costs can be grouped broadly according to the age at which the cost is imposed. The short-term cost of growth acceleration is mortality coincident with the growth period. Long-term costs are defined as those that are incurred subsequent to the cessation of growth. While short-term costs have been well described in both the theoretical and empirical literature, evidence of long-term costs is only recently coming to light, though the empirical evidence must not be overstated.

(*a*) *Short-term costs of rapid growth*

(*i*) *Predation risk.* Faster growth is most commonly achieved by increasing foraging effort, and predation risk often scales with foraging rates (Ali *et al.*, 2003). As a result, predation is a major constraint on growth rates (Houston *et al.*, 1993), most often causing maturation to occur later and/or at a smaller size (e.g. Cushman, Rashbrook & Beattie, 1994; Blanckenhorn, 2000; Johansson *et al.*, 2001; Peckarsky *et al.*, 2001). Higher pressure to acquire food when under resource stress may also lead to increased foraging activity and competition for food, leading to injury or death by predators or conspecifics (Fig. 3; Sundstrom, Lohmus & Devlin, 2005; Nicieza & Metcalfe, 1999). Animals at risk of predation reduce mortality risk by decreasing foraging effort, switching to safer habitats, or altering temporal activity patterns (Sih, 1982; Lima & Dill, 1990; Johansson *et al.*, 2001; Brodin & Johansson, 2004; Fraser *et al.*, 2004; Berger & Gotthard, 2008). All of these behavioural changes cause growth to be suppressed, which potentially reduces fitness *via* delayed development or smaller body size. However, conflicting pressures such as time constraints may cause animals to increase risk-acceptance while foraging, thereby increasing juvenile mortality (Gotthard, 2000; Laurila *et al.*, 2008). Accelerated growth may also be achieved without increasing foraging effort by changes in metabolism, digestive efficiency or patterns of resource allocation (Boujard *et al.*, 2000; Dmitriew & Rowe, 2005; Lindgren & Laurila 2005; Stoks, De Block & McPeek, 2006).

Common garden comparisons of populations with different intrinsic growth rates also suggest that there is great potential for predation risk to be elevated in fast-growing populations of amphibians and fish. Mortality risk increased with intrinsic growth rate of anuran populations along a latitudinal cline in Sweden. There was a positive association between activity level in a common environment and latitude of origin

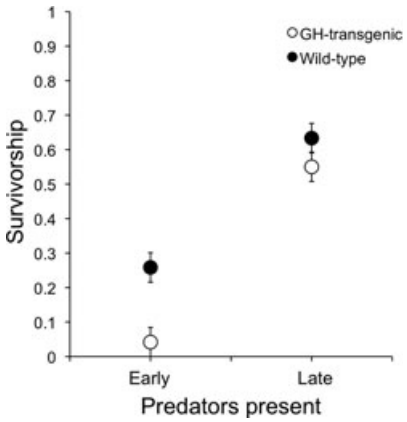

Fig. 3. Predation rates on coho salmon (*Oncorhynchus kisutch*) by older conspecifics. Growth-hormone transgenic coho salmon (open circles) were significantly less likely to survive to day 48 relative to slower growing, wild-type genotypes (filled circles) ($F_{1,11} = 12.34$, $P = 0.0008$). This was true both when predators were present at hatching ("early" predator presence) and when they were introduced 24 days after hatching ("late" predator presence). Values are means +/− 1 S.E.M. Modified from Sundstrom *et al.* (2005). [Corrections were introduced to the paper on 24 June 2010 after its first online publication on Wiley InterScience on 11 April 2010.]

in both *Rana arvalis* and *Rana temporaria* tadpoles reared in a common environment (Laurila, Pakkasmaa & Merila, 2006; Laurila *et al.*, 2008). While all populations of *R. temporaria* reduced activity in the presence of a caged predator, this effect was less pronounced for high-latitude tadpoles. Populations at high latitudes may be approaching their growth rate limits due to the pressure to develop quickly when seasons are short, reducing the scope for plastic acceleration in growth rate. As a result of these differences in behaviour, fast-growing tadpoles suffered higher mortality rates in the presence of a free-ranging predator (Laurila *et al.*, 2006, 2008).

Atlantic silverside (*Menidia menidia*) originating from the coast of Nova Scotia gain mass at around twice the rate of those from the coast of North Carolina, but experience higher mortality rates due to predation over both the short (24 h) and long term (30 days; Lankford, Billerbeck & Conover, 2001; Munch & Conover, 2003). Rapid growth of high-latitude fish is apparently achieved by a combination of behavioural and physiological differences among the populations, both of which contribute to the increased predation risk. Fast-growing individuals left predation refuges sooner than did slower growing fish when predators were in the vicinity (Chiba, Arnott & Conover, 2007), while both swimming endurance and burst swim speed were lower in the fast-growing population. This could hinder the ability to avoid predator attacks (Billerbeck *et al.*, 2001; Munch & Conover, 2004).

Many of the predation-dependent costs of rapid growth have the potential to carry over into the adult stage *via* negative effects on locomotor performance. Sablefish (*Anoplopoma fimbria*) undergoing compensatory growth have lower swim speed during the period of food deprivation as

well as after recovery (Sogard & Olla, 2002). Transgenic coho salmon (*Oncorhynchus kisutch*) containing genes for increased growth hormone production, which have much higher growth rate potential than wild-type salmon, were shown to suffer higher mortality as fry relative to wild-types (Sundstrom *et al.*, 2005). Domesticated brown (*Salmo trutta*) and rainbow trout (*Oncorhynchus mykiss*) that have undergone intense artificial selection for high growth rates forage as juveniles in riskier habitats and for longer periods of time, respectively, causing both species to experience lower survivorship relative to wild-type varieties (Àlvarez & Nicieza, 2003; Biro *et al.*, 2004).

Longer term studies of the effects of juvenile growth conditions on endurance swimming have been conducted in green swordtails *Xiphophorus helleri*. Though neither fast-start swimming performance nor swimming endurance differed among growth treatments when initially assessed (Royle, Lindstrom & Metcalfe, 2006*a*; Royle, Metcalfe & Lindstrom, 2006*b*), compensating individuals had poorer swimming endurance, which can affect long-term fitness by negatively impacting foraging efficiency or the ability to escape predation (Royle *et al.*, 2006*a*). In a rare long-term study of growth rate in a natural population, growth and survival were tracked in a population of lemon sharks *Negaprion brevirostri* (Dibattista *et al.*, 2007). Because of the low emigration rate of juvenile sharks, selection on traits such as body length, condition and growth rate could be estimated. Growth rate during the first year was a strong predictor of mortality within four years, with the fastest growing individuals being far more likely to die than the slowest growing sharks. The authors suggest this could be due to increased predation risk in the most productive foraging sites (Dibattista *et al.*, 2007).

(*ii*) *Predator-independent effects.* Several studies have found that fast-growing juveniles suffer higher mortality rates even when predation is not a major source of mortality. This implies that predation risk is not the only factor selecting against higher growth rates. A physiological cost for high growth rate is also implied by the results of selection experiments in two species of dipterans. In *Drosophila melanogaster*, faster growing lines had lower pre-adult viability (Partridge & Fowler, 1993), while in yellow dung flies (*Scathophaga stercoraria*), lines selected for large body size grew faster under good conditions, but performed more poorly than slow-growing lines when food was limited (Teuschl *et al.*, 2007). Similarly, rapidly growing, time-constrained dung flies suffered higher mortality rates independent of predation (Blanckenhorn, 1998), as did common frogs originating at high latitude (Laurila *et al.*, 2008). Reduced starvation resistance was also found in natural populations of butterflies having higher intrinsic growth rates that experienced higher rates of mass loss during a subsequent period of food restriction (Gotthard *et al.*, 1994). These results suggest that physiological costs of growth exist even in the short term, although so far the mechanism for this effect is unknown. One possibility is that during growth fewer resources are allocated to energy reserves, leaving individuals more vulnerable to future periods of resource stress (e.g. Bayne, 2000). There might also be an increased probability of death due to higher rates of oxidative damage at the molecular level, as proposed by Mangel & Munch (2005).

In summary, both selection experiments and common garden comparisons of populations with different intrinsic growth rates provide good evidence that acceleration of growth may be accompanied by a greater risk of mortality during the period of growth. These costs, which are essentially lethal consequences of accelerating growth prior to reproduction, exert an obvious downward selection pressure on the optimal growth rate. The best evidence of such costs comes from the study of predation risk relating to growth effort, but we still lack a general explanation for the propensity for so many animals to grow at submaximal rates in the absence of predators.

(*b*) *Long-term costs of rapid growth*

Costs of growth that are not incurred immediately could, nevertheless, contribute to creating strong selective pressure on growth trajectories, particularly if they occur early in the reproductive stage. Such costs might in fact play a key role in the prevalence of submaximal growth. However, the identification of such costs is fraught with numerous challenges. First, it is necessary that individual fitness traits, such as fecundity, mating success and longevity, be tracked over the long term. Second, the putative costs must be attributable to accelerated growth and not to some correlated but not functionally related trait. As a result, few studies succeed in demonstrating a trade-off between growth rate and any measure of adult fitness, though an increasing number of studies seek to do so. Most studies to date are based on temporary food restriction followed by compensatory growth; the papers discussed below are assumed to use this approach unless specified otherwise. This approach is not without drawbacks; these are discussed in Section IV.1*b*.

Ultimately, many of the costs attributed to compensatory growth arise from trade-offs in allocation among various traits and functions that depend on the same finite pool of resources. In many cases, these allocation-related costs are obvious. For example, compensatory growth may be observed for some measures of size but not others; in damselflies and mosquitoes, compensatory growth has been shown to occur in structural size but not mass (Strobbe & Stoks, 2004; Dmitriew & Rowe, 2005; Schafer & Lundstrom, 2006). This pattern of allocation may be adaptive because in insects structural size is fixed at eclosion, while adults can continue to accrue resources (e.g. Moya-Laraño *et al.*, 2003). Nevertheless, lower mass might still imply a reduction in fitness, since it implies a reduction in energetic reserves, which might reduce reproductive output or the ability to withstand subsequent periods of stress (Barrett *et al.*, 2009; Scharf, Filin & Ovadia, 2009; C. Dmitriew & L. Rowe, in preparation).

A different effect is observed among temperate fish species, which are indeterminate growers. In fish, body condition (mass per unit length) is rapidly corrected following periods of poor growth (Nicieza & Metcalfe 1997; Nikki *et al.*, 2004;

Àlvarez & Nicieza, 2005; Johnsson & Bohlin, 2005, 2006). Exceptions to this pattern occur in certain tropical fish species, which compensate for both skeletal size and mass simultaneously, or for skeletal size first (Takagi, 2001; Royle *et al.*, 2005; N.J. Royle, personal communication), though additional taxa must be studied for a conclusive pattern to be established. The tendency for temperate fish species to compensate for mass first may be due to the importance of stored energy reserves for determining overwinter survival (e.g. Kirjasniemi & Valtonen, 1997; Biro *et al.*, 2004; Finstad *et al.*, 2004; Pangle, Sutton & Kinnunen, 2004). When compensation for structural size does occur in salmonids, it is secondary to mass (e.g. Johnsson & Bohlin, 2005; 2006; Myszkowski *et al.*, 2006). The failure to compensate for skeletal size may nevertheless be costly, given that individuals with larger structural size may have a considerable competitive advantage (Nicieza & Metcalfe, 1999; Maclean & Metcalfe, 2001; Àlvarez, Cano & Nicieza, 2006).

More detailed analyses of body composition also suggest that compensatory growth masks differences among treatment groups in physiological traits. Damselflies and ladybird beetles undergoing compensatory growth differ in the proportion of body mass that is composed of triglyceride fats compared to controls (Stoks *et al.*, 2006; Dmitriew, Carroll & Rowe, 2009). Allocation to fat over other tissues, including structural size or reproductive tissues, may improve survival, particularly during subsequent periods of stress or during dispersal (e.g. Dmitriew & Rowe, 2007; Scott *et al.*, 2007). The consequences of differences in allocation to protein *versus* fat for fitness is apparent from studies comparing allocation to dispersal and reproductive morphs of the cricket *Gryllus firmis* (Zera & Denno, 1997). The conditions experienced during development may dictate the relative benefit of dispersal; if conditions are poor, allocation to dispersal traits rather than to immediate reproduction may be an adaptive strategy (Zera & Brink, 2000; Boggs, 2009). Since body composition may differ even when overall body size or mass does not, this may contribute to some of the variation in fitness observed among animals that grow at different rates.

Apart from these gross differences in mass or size, various other traits have been assayed in adults following compensatory growth. Comparing pumpkinseed sunfish from two populations with different intrinsic growth rates revealed that ossification was delayed (Arendt & Wilson, 2000) and that scale strength was negatively correlated with growth rate in six populations (Arendt, Wilson & Stark, 2001). Both effects are likely to have implications for swimming ability and may explain why swimming speed and endurance are lower in faster growing fish (e.g. Billerbeck *et al.*, 2001; Munch & Conover, 2004; Royle *et al.*, 2006*a*).

Tissue-specific patterns of resource allocation are likely to impose some cost on overall fitness, though they may nevertheless represent an adaptive compromise that depends upon species-specific ecology. A useful approach may be to make predictions about optimization of allocation of limited energy among traits (i.e. structural size *versus* energetic reserves) in light of the importance of a specific trait in determining overall fitness. For example, in species for which dispersal is an important component of the life history, limited energy may be allocated to traits that improve dispersal capability at the cost of a high early reproductive rate (Zera & Brink, 2000).

(*i*) *Long-term survival after compensatory growth.* Studies tracking long-term survival or reproduction can provide direct evidence of fitness costs following accelerated growth independent of age and size at maturity, but to date such experiments are relatively uncommon and their results are mixed. Johnsson & Bohlin (2005, 2006) found no measurable effect on recapture rates of brown trout tracked for a year after the compensatory growth, but given that this species can live for several years in the wild, longer term studies are needed. Carlson *et al.* (2004) found no correlation between growth rate and the probability of recapture after one year in the same species, although initial size differences were due to natural variation in the population and not to experimental manipulation of initial condition. In a recent laboratory study, lifetime fitness was tracked in two groups of three-spined sticklebacks (*Gasterosteus aculeatus*; Inness & Metcalfe, 2009). In the first treatment group, fish were subjected to intermittent feeding, which caused cycles of depressed and accelerated growth. In the second, food level was constant, and the overall mean growth rate was equal to that of the variable-growth treatment. There was no effect of treatment on reproductive output, but mortality rates were higher under variable growth (Inness & Metcalfe, 2009).

Invertebrates may be a more promising group for studies of this nature, since they have rapid generation times and are thus more easily tracked throughout their entire life. The ectoparasitic mite *Argulus coregoni* accelerated growth following a period of time off host, and subsequently suffered a shorter mean lifespan (Hakalahti *et al.*, 2005). Ladybird beetles (*Harmonia axyridis*) that had compensated for resource restriction early in life died sooner than controls, but mortality rate was only affected if food stress was imposed late in life (Dmitriew & Rowe, 2007). This may indicate either that investment in nutritional reserves was reduced, or that the rate of senescence was higher independent of food availability. A subsequent study found that body composition (whole body fat and protein content) in this species depended on rearing conditions even though overall mass and body size were the same (Dmitriew *et al.*, 2009). Another cost that could potentially affect long-term survival is a reduction in learning ability, as was demonstrated in zebra finches (*Taeniopygia guttata*) compensating for poor early growth (Fisher, Nager & Monaghan, 2006). This could in turn lead to reduced foraging efficiency (e.g. Dukas & Bernays, 2000).

Long-term costs of rapid growth might also arise from links between growth rate and oxidative damage (see Mangel & Munch, 2005), though evidence is largely circumstantial (Monaghan, Metcalfe & Torres, 2009). There is some indication that rate of growth is positively correlated with measures of oxidative damage (Alonso-Alvarez *et al.*, 2007, Nussey *et al.*, 2009), and levels of antioxidant enzymes were

higher in adult damselflies (*Lestes viridis*) that compensated for very brief periods of starvation in the final instar (De Block & Stoks, 2008*a*). This increase is assumed to indicate upregulation of the repair response in rapidly growing individuals. However, whether this upregulation is sufficient or whether damage nevertheless accumulates at a higher rate is unclear. Higher metabolic rate is associated with faster production of reactive oxygen species (ROS) and thus oxidative stress and reduced lifespan (Finkel & Holbrook, 2000). Both compensatory growth (Criscuolo *et al.*, 2008) and elevated levels of growth hormone (GH) raise resting metabolic rates (Hauck & Bartke, 2001; Bartke & Brown-Borg; 2004), leading to higher ROS production. Yet, in fast-growing, GH-transgenic mice, antioxidant defences were not correspondingly higher. This suggests that oxidative stress might be higher, causing greater net damage (Hauck & Bartke, 2001; Bartke & Brown-Borg, 2004). Rosa *et al.* (2008) examined the relationship between higher rates of growth in GH-transgenic zebrafish (*Danio rerio*) and showed that higher growth rates produced higher ROS levels, but although the antioxidant enzymes measured did not demonstrate a parallel increase in activity, no increased oxidative damage could be measured. Nevertheless, the lack of compensatory increases in antioxidant defences is suggestive of an increased vulnerability to cellular damage following rapid growth. The relationship between rapid growth and aging has great potential as a mediator of growth rate, but further research is needed to establish causal links between growth rate and oxidative damage. Given the tractability of insects for the study of long-term costs of accelerated growth, further study of the relationship between growth, oxidative damage and antioxidant defenses in this group would be of great value, and could test the hypothesis that oxidative damage due to growth leads to accelerated rates of senescence.

(*ii*) *Reproductive consequences of compensatory growth.* Apart from what might be inferred from studies demonstrating reduced investment in fat or other storage molecules, direct evidence for reproductive costs of accelerated growth is limited. To date, few studies have tracked reproductive success following accelerated growth. Those that did used food restriction followed by refeeding to induce compensatory growth acceleration. In the ladybird beetle *Harmonia axyridis*, a period of food restriction followed by compensatory growth of mass and structure had no effect on rate of egg production, total fecundity or hatching success within the first three months after eclosion (Dmitriew & Rowe, 2007). In the same study, male mating success, latency to copulation and copulation duration were also unaffected, and females did not demonstrate a preference for males reared under constant high-food conditions (Dmitriew & Rowe, 2007). Green swordtails demonstrate a similarly robust compensatory response to temporary food shortage, with males maturing at the same size as individuals maintained on constant rations. As in ladybird beetles, female swordfish did not demonstrate a preference for males of either treatment (Walling *et al.*, 2007). However, compensating males were less likely to achieve dominant status in the population (Royle *et al.*, 2005).

IV. DISCUSSION: IDENTIFYING THE COSTS OF GROWTH ACCELERATION

(1) Methodological issues

Despite the growing body of research examining constraints on growth rate, a number of issues, particularly with respect to the assessment of the long-term costs of growth, remain unresolved. Of particular note is the potential for costs of growth acceleration to be confounded with those arising from the stressor used to elicit it. Most studies looking at long-term costs of growth have used periods of resource restriction in order to induce compensatory growth acceleration. The interpretation of results may be biased depending upon the researchers' primary interest. For instance, two studies using nearly identical designs (i.e. lower growth rate induced by temporary resource restriction) could arrive at different conclusions depending upon whether these costs are attributed to growth acceleration (in which case the confounding effect of the food manipulation is downplayed) or whether they are linked to the initial period of food restriction, in which case possible costs of compensation, should they occur, are not taken into account.

The problem of disentangling costs of growth and stress is therefore a significant challenge for the study of the costs of growth acceleration. However, there are a number of possible ways in which to overcome this issue. First, rather than comparing fitness between the two growth rate treatments, the maximum growth rate attained by an individual can be used as the dependent variable (e.g. Fisher *et al.*, 2006; Alonso-Alvarez *et al.*, 2007); a negative relationship between individual growth rates and the measure of fitness used would be evidence for a cost of rapid growth. However, this approach is also problematic because differences in genetic quality or underlying condition (of subjects collected in the field) could lead to correlations between growth rate and fitness. Thus, a causal relationship between growth rate and fitness cannot be assumed.

(*a*) *Choosing measures of body size*

Compensatory growth, even when complete, is often accompanied by changes in body composition and allometry. This underscores the importance of choosing the appropriate measures of body size or condition when investigating trade-offs of growth acceleration. Though this has been pointed out numerous times in past reviews (Strobbe & Stoks, 2004; Nicieza & Àlvarez, 2009), it has been largely ignored in subsequent experimental designs. The most obvious issue is the lack of consistency among studies in how size is measured. Growth trajectories are typically based on repeated measures of mass or some aspect of structural size, such as body length in fish or pronotum width in insects. Although these traits may

be highly correlated with one another, it appears that they are not equally affected by stresses such as food restriction or time constraints during development (e.g. Strobbe & Stoks, 2004; Dmitriew & Rowe, 2005). Nevertheless, these measures are used interchangeably in literature reviews, and may obscure real patterns in allocation among animals growing at different rates. Authors making generalizations about the expected relationship between body size and fitness should explicitly state whether mass, structural size, or a composite measure of the two is used. Size-specific mass, often referred to as "condition" in the literature, may be a more accurate predictor of fitness than mass or length alone (e.g. Kirjasniemi & Valtonen, 1997; Biro *et al.*, 2004; Finstad *et al.*, 2004), although it has been suggested that density (resources per unit volume) may be more informative still (Moya-Laraño *et al.*, 2008). Mass is a good proxy for overall nutrient storage (Schulte-Hostedde *et al.*, 2005), although detailed analysis of the composition of the body tissues such as lipid or carbohydrate content provides more predictive power regarding future fitness, particularly in a specific environmental context (e.g. Barrett *et al.*, 2009; Monaghan, 2008). When such detailed physiological analyses are not possible or practical, it is advisable to measure both mass and at least one aspect of structural size, bearing in mind that the magnitude of the growth acceleration can vary among size traits. Identifying these differences might allow fitness costs and benefits of accelerating growth rate to be identified more easily, since investment in condition and investment in structural size have different implications for long-term fitness.

It is also known from theory that the manner in which size is measured may have important implications for growth rate plasticity in response to stress. For example, "size" is often defined in terms of either mass (e.g. Yearsley *et al.*, 2004) or some unspecified measure (such as total carbon content) that is not divided into structural *versus* reserve tissues (e.g. Abrams & Rowe, 1996; Mangel & Munch, 2005). However, the proportion of this "size" that is made up of structural size or mass appears to be a key component of energy budget models that seek to explain patterns of compensatory growth (Broekhuizen *et al.*, 1994; Gurney *et al.*, 2003). This may be particularly relevant for species facing fluctuating growth conditions, since resources allocated to structure rather than mass may differ in their accessibility during periods of stress. Energy allocated to skeletal growth is not available to be re-mobilized for maintenance of basic metabolic functions under periods of stress, while fat reserves are. Distinguishing between mobilizable and non-mobilizable energy appears to be critical for accurate description of real growth patterns observed during and subsequent to periods of food restriction in fish (Broekhuizen *et al.*, 1994; Gurney *at al.*, 2003). Whether resources are allocated to structural size (resources that cannot later be re-mobilized), or energetic reserves (which can), is likely to influence reproduction and survival in different ways.

(*b*) *Measuring growth trajectories*

Studies investigating costs of compensatory growth acceleration often use only two measurement points (e.g. before and after a period of recovery from food restriction) to estimate average growth rate over some period of time. However, since animal growth trajectories are actually non-linear (Tammaru & Esperk, 2007), it is possible that key information is lost in this simplified approach. What appears to be accelerated growth following a period of growth restriction when comparing growth at a given age may in fact be size-specific growth that is equal to that of the control treatment (see Nicieza & Àlvarez, 2009). However, this does not imply that compensatory growth is expected to occur without costs, since even though the end point of growth is the same, the growth trajectories are nevertheless highly divergent. This point is illustrated in Fig. 4 using data from the ladybird beetle, *H. axyridis*. As is typically observed in insects (Tammaru & Esperk, 2007), the rate of mass accumulation in *H. axyridis* decelerates prior to eclosion in the group receiving consistently high rations throughout development. Larvae undergoing compensatory growth recover by continuing to grow along a steeper trajectory right until eclosion. While a higher growth rate than in the high-food control group is achieved during this period, the highest instantaneous growth rate occurred in the controls. In studies on compensatory growth acceleration, the problem may not be how to explain submaximal growth

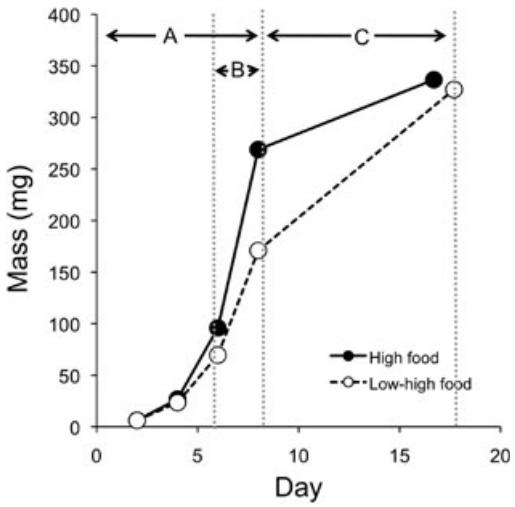

Fig. 4. Example of typical *Harmonia axyridis* growth trajectories at high food (solid line, $N = 231$) and food restriction (region A) followed by high food (dotted line, $N = 198$) to illustrate the difficulty in attributing long-term fitness costs to growth rate. Which treatment is undergoing the highest growth rate depends on when growth rate is measured. In this example, the maximum growth rate is higher in the high food treatment (region B). During the final period (region C), rate of growth of the high food treatment decelerates, causing the relative growth rate to be higher in the restricted food treatment (C. Dmitriew, unpublished data).

but rather why growth might decelerate prior to pupation under natural conditions.

(*c*) *Identifying costs of growth*

Given the above issues with food manipulations to investigate the costs of growth acceleration, it may be worth using alternatives. Manipulation of time constraints can avoid some of the major problems outlined above, since it does not require control of food levels. Photoperiod manipulation may be particularly useful since it is not accompanied by other changes in environmental quality that could be associated with other types of time constraint such as hydroperiod. Not all taxa respond to photoperiod by adjusting growth or development rates (Section III.1*b*). Nevertheless, for many species, it may be a valuable alternative approach to identifying the lifetime fitness costs of growth acceleration. The use of pilot studies to find a photoperiod manipulation that produces adults of equal size at maturity is also recommended, since this may eliminate the need to correct for differences in body size. One reason that some of the species studied to date appear not to catch up entirely when grown under a late-season photoperiod is that studies typically compare growth under light regimes simulating the extremes of the species' growth season (e.g. Johansson & Rowe, 1999).

The confounding effects of food manipulation can also be avoided by comparing populations with different intrinsic growth rates. The deficiency of this comparative method is that it assumes that putative costs correlated with growth rate are causal, and not incidental (e.g. due to genetic linkages). The cost might also depend on the environment in which the population evolved. For example, a negative correlation between the mean intrinsic growth rate of a population and swimming speed is often observed in common garden experiments on aquatic animals (e.g. Billerbeck *et al.*, 2001; Lankford *et al.*, 2001; Arendt & Hoang, 2005). This is assumed to indicate a direct cost of growth, since the ability to escape predation is compromised. Yet, it is also conceivable that selection on swimming speed is simply less intense in colder climates, because predation risk tends to be lower at increasing latitudes (Laurila *et al.*, 2008).

A second major issue is that evidence of a link between a response variable and fitness is often lacking in studies of the costs of variable growth. Even body size, while often positively correlated with fitness, does not explain all of the variance in fitness among individuals of a species (reviewed in Blanckenhorn, 2000). Yet, it is a commonly used proxy for fitness, even when support for a correlation between size and fitness in a given species has not been shown. Of course, direct measurement of fitness is often impractical, but effort should be made to provide evidence that so-called costs of rapid growth do, in fact, have negative effects on fitness components. This is particularly true of traits that are highly variable among individuals and that may change over short time scales. One example of such a trait is phenoloxidase (PO) concentration, which is commonly assayed as an indicator of immunocompetence. Studies linking decreased levels of PO to growth rate are commonly cited as evidence for a cost of rapid growth (e.g. Metcalfe & Monaghan, 2001, 2003; Dmitriew *et al.*, 2007; Campero *et al.*, 2008; De Block & Stoks, 2008*b*), yet PO has only been tenuously linked to fitness, if at all (e.g. Adamo, 2004; Schwarzenbach & Ward, 2007; Bailey & Zuk, 2008). PO levels are highly variable and often differ in effect from other measures of immunocompetence within the same individual or in response to the same experimental manipulations (e.g. Siva-Jothy & Thompson, 2002; De Block & Stoks, 2008*b*). It appears that PO itself is not particularly costly to produce, which might explain in part its variability (Armitage *et al.*, 2003; Mucklow, Visozo & Jensen, 2004; but see Schwarzenbach & Ward, 2006); if there is a growth-immunocompetence trade-off it may be more likely to occur at a molecule downstream of PO that is more costly to produce.

(2) Linking growth to fitness: integration of physiology and evolutionary biology

Direct or short-term costs of accelerated growth such as predation risk are straightforward to establish: death during the juvenile stage equals zero fitness. However, in other cases, the mechanisms connecting growth rate to reduced fitness are less obvious. This review has largely been concerned with predation-independent costs of growth, the existence of which are thought to underlie the evolution or expression of slower growth rates in the absence of predators. If growth rate is constrained primarily by energy-based trade-offs, it would be instructive to identify and study in greater detail the developmental and endocrinological pathways underlying these trade-offs. In doing so, we might identify broad patterns in growth rate variation among populations of species, to further resolve of our understanding of life-history evolution. Functional links between growth rate and adult morphological traits are still poorly understood; why some traits are affected by accelerated growth and others are not remains largely unknown.

(3) The maintenance of genetic variation in growth trajectories

Just as phenotypic variation in growth persists despite the presumed benefits of accelerated growth, genetic variation in this trait appears to persist despite the presumption of positive directional selection on growth rate. Intrinsic growth rates vary among species and among populations (e.g. across environmental clines), evidence that genetic variance in growth trajectories existed in the past. Moreover, extant genetic variance is implied by the rapid evolution of growth rate in response to artificial selection on adult body size or experimental evolution experiments in which growth rates respond rapidly to changing environmental conditions (e.g. Partridge and Fowler, 1993; Partridge *et al.*, 1994; Teuschl *et al.*, 2007; but see Partridge *et al.*, 1999). Finally, studies measuring genetic variance in growth trajectories directly also suggest that it is considerable (e.g. Riska, *et al.*, 1984;

Rauter & Moore, 2002; Wilson *et al.*, 2007; Dmitriew, Blows & Rowe, in press).

How genetic variance is maintained in traits under directional or stabilizing selection is a question of major interest to evolutionary biologists (Lande, 1975; Turelli & Barton, 2004). A number of mechanisms have been proposed for the maintenance of genetic variation in traits under selection, many of which apply to growth rate (e.g. Houle, 1998). In general, interactions between the genotype and its environment may act to maintain genetic variation within populations (e.g. Gillespie & Turelli, 1989; Turelli & Barton, 2004). In the case of genetic variability in growth trajectories, the apparent paradox (that variance is maintained in spite of directional or stabilizing selection on growth rate) may be resolved by the fact that the relative fitness of growth genotypes depends upon the environment; in particular, the relative costs and benefits of accelerating growth depend on current local conditions. The fact that there may be a substantial time lag before the cost for rapid growth is incurred further complicates the picture, particularly if conditions change over the course of a single generation. Genetically determined growth trajectories that have high fitness relative to other genotypes in one environment may have lower fitness in another. Because environmental conditions (in particular, resource availability) may vary to a considerable degree over short time spans, and because growth is so strongly dependent upon resource acquisition, growth conditions that change over the short term may cause selective pressures to fluctuate rapidly. Consider the hypothetical example of a damselfly species that completes development in ephemeral ponds with variable drying rates. In such a species, temporal and/or spatial variation in hydroperiod length will determine in part the optimal rate of growth; which genotype is favoured by selection will depend on the year, pond type and size, and the time of season. Fast-growing genotypes may be selected in smaller ponds, or late in the season, while other environments may favour slower growing genotypes because of fitness costs associated with rapid growth. Thus, such genotype by environment interactions may allow a large number of genetic growth strategies to persist on a local scale.

V. CONCLUSIONS

(1) The optimal rate of growth is achieved by balancing the fitness benefits of reaching a large body size in a short period of time and the costs of accelerating growth rate. While the advantages of growing rapidly are well known, the factors constraining growth rate have received less attention.

(2) It is well known that organisms demonstrate considerable plasticity in growth rate in response to variables such as resource availability and predation risk. There is some evidence that growth may also be accelerated in response to time constraints (signaled by photoperiod or hydroperiod), and in response to poor growth induced by brief periods of food restriction.

(3) Genetically based differences in growth rates have also been observed among populations, and along latitudinal clines. In general, growth rates tend to increase with latitude. At low latitudes where season length is longer, slower growth appears to be favoured.

(4) The prevalence of submaximal growth within populations, and the tendency for slower growth to predominate in certain populations, suggests that costs of growth may be considerable. Increasingly, studies seek to address the nature of these costs by comparing correlates of fitness among populations with different intrinsic growth rates, or by inducing growth rate variation by experimental manipulation of condition.

(5) Recently studies have investigated whether higher rates of growth might be constrained by long-term costs of growth acceleration. There is some evidence that accelerated growth may lead to poorer performance by adults, particularly when they are subsequently exposed to stress. However, based on the evidence so far, the primary constraint on growth rate appears to be increased juvenile mortality due to predation.

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VII. REFERENCES

Abrams, P. A., Leimar, O., Nylin, S. & Wiklund, C. (1996). The effect of flexible growth rates on optimal sizes and development times in a seasonal environment. *American Naturalist* **147**, 381–395.

Abrams, P. A. & Rowe, L. (1996). The effects of predation on the age and size of maturity of prey. *Evolution* **50**, 1052–1061.

Adamo, S. A. (2004). How should behavioural ecologists interpret measurements of immunity? *Animal Behaviour* **68**, 1443–1449.

Ali, M., Nicieza, A. & Wootton, R. J. (2003). Compensatory growth in fishes: a response to growth depression. *Fish and Fisheries* **4**, 147–190.

Altwegg, R. (2002). Predator-induced life-history plasticity under time constraints in pool frogs. *Ecology* **83**, 2542–2551.

Alonso-Alvarez, C., Bertrand, S., Faivre, B. & Sorci, G. (2007). Increased susceptibility to oxidative damage as a cost of accelerated somatic growth in zebra finches. *Functional Ecology* **21**, 873–879.

Àlvarez, D., Cano, J. M. & Nicieza, A. G. (2006). Microgeographic variation in metabolic rate and energy storage in brown trout: countergradient selection or thermal sensitivity? *Evolutionary Ecology* **20**, 345–363.

Àlvarez, D. & Nicieza, A. G. (2003). Predator avoidance behaviour in wild and hatchery-reared brown trout: the role of experience and domestication. *Journal of Fish Biology* **63**, 1565–1577.

Àlvarez, D. & Nicieza, A. G. (2005). Compensatory response 'defends' energy levels but not growth trajectories in brown trout, *Salmo trutta* L. *Proceedings of the Royal Society B Biological Sciences* **272**, 601–607.

Andersson, M. (1994). *Sexual Selection*. Princeton University Press, Princeton, New Jersey.
Arendt, J. D. (1997). Adaptive intrinsic growth rates: an integration across taxa. *The Quarterly Review of Biology* **72**, 149–177.
Arendt, J. D. & Hoang, L. (2005). Effects of food level and rearing temperature on burst speed and muscle composition of Western Spadefoot Toad (*Spea hammondii*). *Functional Ecology* **9**, 982–987.
Arendt, J. D. & Wilson, D. S. (1997). Optimistic growth: competition and an ontogenetic niche-shift select for rapid growth in pumpkinseed sunfish (*Lepomis gibbosus*). *Evolution* **51**, 1946–1954.
Arendt, J. D. & Wilson, D. S. (1999). Countergradient selection for rapid growth in pumpkinseed sunfish: Disentangling ecological and evolutionary effects. *Ecology* **80**, 2793–2798.
Arendt, J. D. & Wilson, D. S. (2000). Population differences in the onset of cranial ossification in pumpkinseed (*Lepomis gibbosus*), a potential cost of rapid growth. *Canadian Journal of Fisheries and Aquatic Sciences* **57**, 351–356.
Arendt, J., Wilson, D. S. & Stark, E. (2001). Scale strength as a cost of rapid growth in sunfish *Oikos* **93**, 95–100.
Armbruster, P. & Conn, J. E. (2006). Geographic variation of larval growth in North American *Aedes albopictus* (Diptera: Culicidae). *Annals of the Entomological Society of America* **99**, 1234–1243.
Armitage, S. A. O., Thompson, J. J. W., Rolff, J. & Siva-Jothy, M. T. (2003). Examining costs of induced and constitutive immune investment in *Tenebrio molitor*. *Journal of Evolutionary Biology* **16**, 1038–1044.
Arnold, K. E., Blount, J. D., Metcalfe, N. B. Orr, K. J., Adam, A., Houston, D. & Monaghan, P. (2007). Sex-specific differences in compensation for poor neonatal nutrition in the zebra finch *Taenopygia guttata*. *Journal of Avian Biology* **38**, 356–366.
Arnott, S. A., Chiba, S. & Conover, D. O. (2006). Evolution of intrinsic growth rate: Metabolic costs drive trade-offs between growth and swimming performance in *Menidia menidia*. *Evolution* **60**, 1269–1278.
Atchley, W. R. & Zhu, J. (1997). Developmental quantitative genetics, conditional epigenetic variability and growth in mice. *Genetics* **147**, 765–776.
Atkinson, D. (1994). Temperature and organism size—a biological law for ectotherms? *Advances in Ecological Research* **25**, 1–58.
Badyaev, A. V., Hamstra, T. L., Oh, K. P. & Seaman, D. A. A. (2006). Sex-biased maternal effects reduce ectoparasite-induced mortality in a passerine bird. *Proceedings of the National Academy of Sciences of the United States of America* **103**, 14406–14411.
Bailey, N. W. & Zuk, M. (2008). Changes in immune effort of male field crickets infested with mobile parasitoid larvae. *Journal of Insect Physiology* **54**, 96–104.
Barrett, E. L. B., Hunt, J., Moore, A. J. & Moore, P. J. (2009). Separate and combined effects of nutrition during juvenile and sexual development on female life-history trajectories: the thrifty phenotype in a cockroach. *Proceedings of the Royal Society B-Biological Sciences* **267**, 3257–3264.
Bartke, A. & Brown-Borg, H. (2004). Life extension in the dwarf mouse. *Current Topics in Developmental Biology* **63**, 189–225.
Bayne, B. L. (2000). Relations between variable rates of growth, metabolic costs and growth efficiencies in individual Sydney rock oysters (*Saccostrea commercialis*). *Journal of Experimental Marine Biology and Ecology* **251**, 185–203.
Benard, M. F. (2004). Predator-induced phenotypic plasticity in organisms with complex life histories. *Annual Review of Ecology Evolution and Systematics* **35**, 651–673.
Berger, D. & Gotthard, K. (2008). Time stress, predation risk and diurnal-nocturnal foraging trade-offs in larval prey. *Behavioural Ecology and Sociobiology* **62**, 1655–1663.
Billerbeck, J. M., Lankford, T. E. & Conover, D. O. (2001). Evolution of intrinsic growth and energy acquisition rates. I. Trade-offs with swimming performance in *Menidia menidia*. *Evolution* **55**, 1863–1872.
Billerbeck, J. M., Schultz, E.T & Conover, D. O. (2000). Oecologia. Adaptive variation in energy acquisition and allocation among latitudinal populations of the Atlantic silverside. *Oecologia* **122**, 210–219.
Biro, P. A., Morton, A. E. Post, J. R. & Parkinson, E. A. (2004). Overwinter lipid depletion and mortality of age-0 rainbow trout (*Oncorhynchus mykiss*). *Canadian Journal of Fisheries and Aquatic Sciences* **61**, 1513–1519.
Bize, P., Metcalfe, N. B. & Roulin, A. (2006). Catch-up growth strategies differ between body structures: interactions between age and structure-specific growth in wild nestling Alpine Swifts. *Functional Ecology* **20**, 857–864.
Bjorndal, K. A., Bolten, A. B., Dellinger, T., Delgado, C. & Martins, H. R. (2003). Compensatory growth in oceanic loggerhead sea turtles: Response to a stochastic environment. *Ecology* **84**, 1237–1249.
Blanck, A. & Lamouroux, N. (2007). Large-scale intraspecific variation in life-history traits of European freshwater fish. *Journal of Biogeography* **34**, 862–875.
Blanckenhorn, W. U. (1998). Adaptive plasticity in growth, development and body size in the yellow dung fly. *Evolution* **52**, 1394–1407.
Blanckenhorn, W. U. (2000). The evolution of body size: What keeps organisms small? *Quarterly Review of Biology*, **75**, 385–407.
Blanckenhorn, W. U. & Demont, M. (2004). Bergmann and converse Bergmann latitudinal clines in arthropods: Two ends of a continuum? *Integrative and Comparative Biology* **44**, 413–424.
Boggs, C. L. (2009). Understanding insect life histories and senescence through a resource allocation lens. *Functional Ecology* **21**, 27–37.
Bonduriansky, R. (2001). The evolution of male mate choice in insects: a synthesis of ideas and evidence. *Biological Reviews* **76**, 305–339.
Boujard, T., Burel, C., Medale, F., Haylor, G. & Moisan, A. (2000). Effect of past nutritional history and fasting on feed intake and growth in rainbow trout *Onchorhynchus mykiss*. *Aquatic Living Resources* **13**, 129–137.
Brodin, T. & Johansson, F. (2004). Conflicting selection pressures on the growth/predation risk trade-off in a damselfly. *Ecology* **85**, 2927–2932.
Broekhuizen, N., Gurney, W. S. C., Jones, A. & Bryant, A. D. (1994). Modelling compensatory growth. *Functional Ecology* **8**, 770–782.
Brown, G. P. & Shine, R. (2006). Why do most tropical animals reproduce seasonally? Testing hypotheses on an Australian snake. *Ecology* **87**, 133–143.
Campero, M., De Block, M., Ollevier, F. & Stoks, R. (2008). Correcting the short-term effect of food deprivation in a damselfly: mechanisms and costs. *Journal of Animal Ecology* **77**, 66–73.
Capellan, E. & Nicieza, A. G. (2007). Non-equivalence of growth arrest induced by predation risk or food limitation: context-dependent compensatory growth in anuran tadpoles. *Journal of Animal Ecology* **76**, 1026–1035.
Carlson, S. M., Hendry, A. P. & Letcher, B. H. (2004). Natural selection acting on body size, growth rate and compensatory growth: an empirical test in a wild trout population. *Evolutionary Ecology Research* **6**, 966–973.
Chiba, S., Arnott, S. A. & Conover, D. O. (2007). Coevolution of foraging behaviour with intrinsic growth rate: risk-taking in naturally and artificially selected growth genotypes of *Menidia menidia*. *Oecologia* **154**, 237–246.
Conover, D. O. & Present, T. M. C. (1990). Countergradient variation in growth rate: 34compensation for different length of the growth-season among Atlantic silversides from different latitudes. *Oecologia* **83**, 316–324.
Criscuolo, F., Monaghan, P., Nasir, L. & Metcalfe, N. B. (2008). Early nutrition and phenotypic development: 'catch-up' growth leads to elevated metabolic rate in adulthood. *Proceedings of the Royal Society B: Biological Sciences* **2575**, 1565–1570.
Cushman, J. H., Rashbrook, V. K. & Beattie, A. J. (1994). Assessing benefits to both participants in a lycaenid-ant association. *Ecology* **75**, 1031–1041.
Day, T., Abrams, P. A. & Chase, J. M. (2002). The role of size-specific predation in the evolution and diversification of prey life histories. *Evolution* **56**, 877–887.
Day, T. & Rowe, L. (2002). Developmental thresholds and the evolution of reaction norms for age and size at maturity. *American Naturalist* **159**, 338–350.
De Block, M., Mcpeek, M. A. & Stoks, R. (2008*a*). Life-history evolution when Lestes damselflies invaded vernal ponds. *Evolution* **62**, 485–493.
De Block, M., Mcpeek, M. A. & Stoks, R. (2008*b*). Life history plasticity to combined time and biotic constraints in Lestes damselflies from vernal and temporal ponds. *Oikos* **117**, 908–916.
De Block, M., Mcpeek, M. A. & Stoks, R. (2008*c*). Stronger compensatory growth in a permanent-pond Lestes damselfly relative to temporary-pond Lestes. *Oikos* **117**, 245–254.
De Block, M., Slos, S., Johansson, F. & Stoks, R. (2008*d*). Integrating life history and physiology to understand latitudinal size variation in a damselfly. *Ecography* **31**, 115–123.
De Block, M. & Stoks, R. (2004). Life-history variation in response to time constraints in a damseflly. *Oecologia* **140**, 68–75.
De Block, M. & Stoks, R. (2005). Fitness effects from egg to reproduction: Bridging the life history transition. *Ecology* **86**, 185–197.
De Block, M. & Stoks, R. (2008*a*). Compensatory growth and oxidative stress in a damselfly. *Proceedings of the Royal Society Series B: Biological Sciences* **275**, 781–785.
De Block, M. & Stoks, R. (2008*b*). Short-term larval food stress and associated compensatory growth reduce adult immune function in a damselfly. *Ecological Entomology* **33**, 796–801.
De Visser, J. A. G. M., Hermisson, J., Wagner, G. P., Meyers, L. A., Bagheri, H. C., Blanchard, J. L., Chao, L., Cheverud, J. M., Elena, S. F., Fontana, W., Gibson, G., Hansen, T. F., Krakauer, D., Lewontin, R. C., Ofria, C., Rice, S. H., Von Dassow, G., Wagner, A. & Whitlock, M. C. (2003). Evolution and detection of genetic robustness. *Evolution* **57**, 1959–1972.
Dibattista, J. D., Feldheim, K. A., Gruber, S. H. & Hendry, A. P. (2007). When bigger is not better: selection against large size, high condition and fast growth in juvenile lemon sharks. *Journal of Evolutionary Biology* **20**, 201–212.
Dmitriew, C., Blows, M. & Rowe, L. (In press). Ontogenetic change in genetic variance in size depends on growth conditions. *American Naturalist*.
Dmitriew, C., Carroll, J. & Rowe, L. (2009). Effects of early growth conditions on body composition, allometry, and survival in the ladybird beetle *Harmonia axyridis*. *Canadian Journal of Zoology* **87**, 175–182.
Dmitriew, C., Cooray, M. & Rowe, L. (2007). Effects of early resource-limiting conditions on patterns of growth, growth efficiency and immune function at emergence in a damselfly (Odonata: Coenagrionidae). *Canadian Journal of Zoology* **85**, 310–318.
Dmitriew, C. & Rowe, L. (2005). Resource limitation, predation risk and compensatory growth in a damselfly. *Oecologia* **142**, 150–154.

Dmitriew, C. & Rowe, L. (2007). Effects of early resource limitation and compensatory growth on lifetime fitness in the ladybird beetles (*Harmonia axyridis*). *Journal of Evolutionary Biology* **20**, 1298–1310.
Dukas, R. & Bernays, E. A. (2000). Learning improves growth rate in grasshoppers. *Proceedings of the National Academy of Sciences of the United States of America* **97**, 2637–2640.
Ernsting, G., Brandjes, G. J., Block, W. & Isaaks, A. A. (1999). Life-history consequences of predation for a subantarctic beetle: evaluating the contribution of direct and indirect effects. *Journal of Animal Ecology* **68**, 741–752.
Finkel, T. & Holbrook, N. J. (2000). Oxidants, oxidative stress and the biology and ageing. *Nature* **408**, 239–247.
Finstad, A. G., Ugedal, O. Forseth, T. & Naesje, T. F. (2004). Energy-related juvenile winter mortality in a northern population of Atlantic salmon (*Salmo salar*). *Canadian Journal of Fisheries and Aquatic Sciences* **61**, 2358–2368.
Fisher, M. O., Nager, R. G. & Monaghan, P. (2006). Compensatory growth impairs adult cognitive performance. *PLOS Biology* **4**, 1462–1466.
Flatt, T. (2005). The evolutionary genetics of canalization. *Quarterly Review of Biology* **80**, 287–317.
Fraser, D. F., Gilliam, J. F., Akkara, J. T., Albanese, B. W. & Snider, S. B. (2004). Night feeding by guppies under predator release: Effects on growth and daytime courtship. *Ecology* **85**, 312–319.
Fraser, D. J., Weir, L. K., Darwish, T. L., Eddington, J. D. & Hutchings, J. A. (2007). Divergent compensatory growth responses within species: linked to contrasting migrations in salmon? *Oecologia* **153**, 543–553.
Frisch, D. & Santer, B. (2004). Temperature-induced responses of a permanent pond and a temporary pond cyclopoid copepod: a link to habitat predictability? *Evolutionary Ecology Research* **6**, 541–553.
Gillespie, J. H. & Turelli, M. (1989). Genotype-environment interactions and the maintenance of polygenic variation. *Genetics* **121**, 129–138.
Gotthard, K. (2000). Increased risk of predation as a cost of high growth rate: an experimental test in a butterfly. *Journal of Animal Ecology* **69**, 896–902.
Gotthard, K. (2004). Growth strategies and optimal body size in temperate Paraginii butterflies. *Integrative and Comparative Biology* **44**, 471–479.
Gotthard, K., Nylin, S. & Wiklund, C. (1994). Adaptive variation in growth rate: life-history costs and consequences in the speckled wood butterfly, *Pararge aegeria*. *Oecologia* **99**, 281–289.
Grafen, A. (1990). Biological signals as handicaps. *Journal of Theoretical Biology* **144**, 517–554.
Gurney, W. S. C., Jones, W., Weitch, A. R. & Nisbet, R. M. (2003). Resource allocation, hyperphagia, and compensatory growth in juveniles. *Ecology* **84**, 2777–2787.
Hakalahti, T., Bandilla, M. & Valtonen, E. T. (2005). Delayed transmission of a parasite is compensated by accelerated growth. *Parasitology* **131**, 647–656.
Hauck, S. J. & Bartke, A. (2001). Effects of growth hormone on hypothalamic catalase and Cu/Zn superoxide dismutase. *Free Radical Biology and Medicine* **28**, 970–978.
Honek, A. (1993). Intraspecific variation in body size and fecundity in insects: a general relationship. *Oikos* **66**, 483–492.
Houston, A. I., Mcnamara, J. M. & Hutchinson, J. M. C. (1993). General results concerning the trade-off between gaining energy and avoiding predation. *Philosophical Transactions of the Royal Society London Series B* **341**, 375–397.
Houle, D. (1998). How should we explain variation in the genetic variance of traits? *Genetica* **102/103**, 241–253.
Huang, G., Wei, L. & Zhang, T. G. (2008). Compensatory growth of juvenile brown flounder *Paralichthys olivaceus* (Temminck & Schlegel) following thermal manipulation. *Journal of Fish Biology* **72**, 2534–2542.
Hurst, T. P., Spencer, M. L., Sogard, S. M. & Stoner, A. W. (2005). Compensatory growth, energy storage and behavior of juvenile Pacific halibut *Hippoglossus stenolepis* following thermally induced growth reduction. *Marine Ecology Progress Series* **293**, 233–240.
Inness, C. L. W. & Metcalfe, N. B. (2009). The impact of dietary restriction, intermittent feeding and compensatory growth on reproductive investment and lifespan in a short-lived fish. *Proceedings of the Royal Society Series B: Biological Sciences* **275**, 1703–1708.
Johansson, F. & Rowe, L. (1999). Life history and behavioural responses to time constraints in a damselfly. *Ecology* **80**, 1242–1252.
Johansson, F., Stoks, R., Rowe, L. & De Block, M. (2001). Life history plasticity in a damselfly: effects of combined time and biotic constraints. *Ecology* **82**, 1857–1869.
Johnsson, J. I. & Bohlin, T. (2005). Compensatory growth for free? A field experiment on brown trout, *Salmo trutta*. *Oikos* **111**, 31–38.
Johnsson, J. I. & Bohlin, T. (2006). The cost of catching up: increased winter mortality following structural growth compensation in the wild. *Proceedings of the Royal Society Series B: Biological Sciences* **273**, 128–1286.
Juliano, S. A. & Stoffregen, T. L. (1994). Effects of habitat drying on size at and time to metamorphosis in the tree hole mosquito *Aedes triseriatus*. *Oecologia* **97**, 369–376.
Kingsolver, J. G. & Pfennig, D. W. (2004). Individual-level selection as a cause of Cope's rule of phyletic size increase. *Evolution* **58**, 1608–1612.
Kirjasniemi, M. & Valtonen, T. (1997). Size-dependent over-winter mortality of young-of-the-year roach, *Rutilus rutilus*. *Environmental Biology of Fishes* **50**, 451–456.
Kozlowski, J. (1992). Optimal allocation of resources to growth and reproduction: Implications for age and size at maturity. *Trends in Ecology & Evolution* **6**, 15–19.
Lande, R. (1975). Maintenance of genetic-variability by mutation in a polygenic character with linked loci. *Genetical Research* **26**, 221–235.
Lankford, T. W., Billerbeck, J. M. & Conover, D. O. (2001). Evolution of intrinsic growth and energy acquisition rates. II. Trade-offs with vulnerability to predation in *Menidia menidia*. Evolution **55**, 1873–1881.
Laugen A. T., Laurila, A., Rasanen, K. & Merila, J. (2003). Latitudinal countergradient variation in the common frog (*Rana temporaria*) development rates: evidence for local adaptation. *Journal of Evolutionary Biology* **16**, 996–1005.
Laurila, A., Lindgren, B. & Laugen, A. T. (2008). Antipredator defenses along a latitudinal gradient in *Rana temporaria*. *Ecology* **89**, 1399–1413.
Laurila, A., Pakkasmaa, S., Crochet, P. A. & Merila, J. (2002). Predator-induced plasticity in early life history and morphology in two anuran amphibians. *Oecologia* **132**, 524–230.
Laurila, A., Pakkasmaa, S. & Merila, J. (2006). Population divergence in growth rate and antipredator defences in *Rana arvalis*. *Oecologia* **147**, 585–595.
Lima, S. L. & Dill, L. M. (1990). Behavioural decisions made under the risk of predation- A review and prospectus. *Canadian Journal of Zoology* **68**, 619–640.
Lind, M. I., Persbo, F. & Johansson, F. (2008). Pool desiccation and developmental thresholds in the common frog, *Rana temporaria*. *Proceedings of the Royal Society Series B Biological Sciences* **275**, 1073–1080.
Lindgren, B. & Laurila, A. (2005). Proximate causes of adaptive growth rates: growth efficiency variation among latitudinal populations of *Rana temporaria*. *Journal of Evolutionary Biology* **18**, 820–828.
Lindgren, B. & Laurila, A. (2009). Physiological variation along a geographical gradient: is growth rate correlated with routine metabolic rate in *Rana temporaria* tadpoles? *Biological Journal of the Linnean Society* **98**, 217–224.
Lindstrom, J., Metcalfe, N. B. & Royle, N. J. (2005). How are animals with ornaments predicted to compensate for a bad start in life? A dynamic optimization model approach. *Functional Ecology* **19**, 421–428.
Ludwig, D. & Rowe, L. (1990). Life-history strategies for energy gain and predator avoidance under time constraints. *American Naturalist* **135**, 686–707.
Maclean, A. & Metcalfe, N. B. (2001). Social status, access to food, and compensatory growth in juvenile Atlantic salmon. *Journal of Fish Biology* **58**, 1331–1346.
Mangel, S. B. & Munch, M. (2005). A life-history perspective on short- and long-term consequences of compensatory growth. *American Naturalist* **166**, E155–E176.
Margraf, N., Gotthard, J. & Rahier, M. (2003). The growth strategy of an alpine beetle: maximization or individual growth adjustment in relation to seasonal time horizons? *Functional Ecology* **17**, 605–610.
Mcmillen, I. C. & Robinson, J. S. (2005). Developmental origins of the metabolic syndrome: Prediction, plasticity and programming. *Physiological Reviews* **85**, 571–633.
Medawar, P. B. (1952). *An Unsolved Problem of Biology*. H.K. Lewis, London.
Merila, J., Laurila, A. & Lindgren, B. (2004). Variation in the degree and costs of adaptive phenotypic plasticity among *Rana temporaria* populations. *Journal of Evolutionary Biology* **17**, 1132–1140.
Metcalfe, N. B. & Monaghan, P. (2001). Compensation for a bad start: Grow now, pay later? *Trends in Ecology and Evolution* **16**, 254–260.
Metcalfe, N. B. & Monaghan, P. (2003). Growth versus lifespan: perspectives from evolutionary ecology. *Experimental Gerontology* **38**, 935–940.
Monaghan, P. (2008). Early growth conditions, phenotypic development and environmental change. *Philosophical Transactions of the Royal Society London Series B* **363**: 1635–1645.
Monaghan, P., Metcalfe, N. B. & Torres, R. (2009). Oxidative stress as a mediator of life history trade-offs: mechanisms, measurements and interpretation. *Ecology Letters* **12**, 75–92.
Monteiro, L. A. & Falconer, D. S. (1966). Compensatory growth and sexual maturity in mice. *Animal Production* **8**: 179–1992.
Morey, S. & Reznick, D. (2000). A comparative analysis of plasticity of larval development in three species of spadefoot toads. *Ecology* **81**, 1736–1749.
Morey, S. & Reznick, D. (2001). Effects of larval density on postmetamorphic spadefoot toads (*Spea hammondii*). *Ecology* **82**, 510–522.
Mousseau, T. A. (1997). Ectotherms follow the converse to Bergmann's Rule. *Evolution* **51**, 630–632.
Moya-Laraño, J., Macias-Ordonez, R., Blanckenhorn, W. U. & Fernandez-Montraveta, C. (2008). Analysing body condition: mass, volume or density? *Journal of Animal Ecology* **77**, 1099–1108.
Moya-Laraño, Orta-Ocana, J. M., Barrientos, J. A., Bach, C. & Wise, D. H. (2003). Intriguing compensation by adult female spiders for food limitation experienced as juveniles. *Oikos* **101**, 539–548.
Mucklow, P. T., Visozo, D. B. & Jensen, K. H. (2004). Variation in phenoloxidase activity and its relation to parasite resistance within and between populations of *Daphnia magna*. *Proceedings of the Royal Society of London Series B: Biological Sciences* **271**, 1175–1183.

Munch, S. B. & Conover, D. O. (2003). Rapid growth results in increased susceptibility to predation in *Menidia menidia*. *Evolution* **57**, 2119–2127.
Munch, S. B. & Conover, D. O. (2004). Nonlinear growth cost in *Menidia menidia*: Theory and empirical evidence. *Evolution* **58**, 61–664.
Myszkowski, L., Kaminski, R. & Kamler, E. (2006). Compensatory growth and matter or energy deposition in *Vimba vimba* juveniles fed natural food or a formulated diet. *Folia Zoologica* **55**, 211–222.
Nicieza, A. G. & Àlvarez, D. (2009). Statistical analysis of structural compensatory growth: how can we reduce the rate of false detection? *Oecologia* **159**, 27–39.
Nicieza, A. G. & Metcalfe, N. B. (1997). Growth compensation in juvenile Atlantic salmon: Responses to depressed temperature and food availability. *Ecology* **78**, 2385–2400.
Nicieza, A. G. & Metcalfe, N. B. (1999). Costs of rapid growth: the risk of aggression is higher for fast-growing salmon. *Functional Ecology* **13**, 793–800.
Nikki, J., Pirhoen, J., Jobling, M. & Karjalainen, J. (2004). Compensatory growth in juvenile rainbow trout, *Oncorhychus mykiss* (Walbaum), held individually. *Aquaculture* **235**, 285–296.
Nussey, D. H., Pemberton, J. M., Pilkington, J. G. & Blount, J. D. (2009). Life history correlates of oxidative damage in a free-living mammal population. *Functional Ecology* **23**, 809–817.
Nylin, S. & Gotthard, K. (1998). Plasticity in life-history traits. *Annual Review of Entomology*. **43**, 63–83.
Nylin, S., Gotthard, K. & Wiklund, C. (1996). Reaction norms for age and size at maturity in Lasiommata butterflies: predictions and tests. *Evolution* **50**, 1351–1358.
Ozanne, S. E. & Hales, C. N. (2004). Lifespan - Catch-up growth and obesity in male mice. *Nature* **427**, 411–412.
Ozanne, S. E. & Hales, C. N. (2005). Poor fetal growth followed by rapid postnatal catch-up growth leads to premature death. *Mechanisms of Ageing and Development* **126**, 852–854.
Pangle, K. L., Sutton, T. M. & Kinnunen, R. E. (2004). Overwinter survival of juvenile lake herring in relation to body size, physiological condition, energy stores and food ration. *Transactions of the American Fisheries Society* **133**, 1235–1246.
Partridge, L., Barrie, B., Fowler, K. & French, V. (1994). Thermal evolution of pre-adult life-history traits in *Drosophila melanogaster*. *Journal of Evolutionary Biology* **7**, 645–663.
Partridge, L. & Fowler, K. (1993). Responses and correlated responses to artificial selection on thorax length in *Drosophila melanogaster*. *Evolution* **47**, 213–226.
Partridge, L., Langelan, R. Fowler, K., Zwaan, B. & French, V. (1999). Correlated responses to selection on body size in *Drosophila melanogaster*. *Genetical Research*. **74**, 43–54.
Peckarsky, B. L., Taylor, B. W., Mcpeek, M. A. & Lytle, D. A. (2001). Variation in mayfly size at metamorphosis as a developmental response to risk of predation. *Ecology* **82**, 740–757.
Peterson, C. Walton, B. M. & Bennett, A. F. (1999). Metabolic costs of growth in free-living garter snakes and the energy budgets of ectotherms. *Functional Ecology* **13**, 500–507.
Rauter, C. M. & Moore, A. J. (2002). Quantitative genetics of growth and development time in the burying beetle *Nicrophorus pustulatus* in the presence and absence of post-hatching parental care. *Evolution* **56**, 96–110.
Riska, B., Atchley, W. R. & J. J. Rutledge. (1984). A genetic analysis of targeted growth in mice. *Genetics* **107**, 79–101.
Roff, D. A. (1980). Optimizing development time in a seasonal environment—the ups and downs of clinal variation. *Oecologia* **45**, 202–208.
Roff, D. A. (1992). The evolution of life histories: Theory and analysis. Chapman and Hall, New York.
Rosa, C. E., Figueiredo, M. A., Lanes, C. F. C., Almeida, D. V., Monserrat, J. M. & Marins, L. F. (2008). Metabolic rate and reactive oxygen species production in different genotypes of GH-transgenic zebrafish. *Comparative Biochemistry and Physiology B: Biochemistry and Molecular Biology* **149**, 209–214.
Roseboom, T. J., Van Der Meulen, J. H. P., Osmond, C., Barker, D. J. P., Ravelli, A. C. J., Schroeder-Tanka, J. M., Van Montfrans, G. A., Michels, R. P. J. & Bleker, O.P. (2000). Coronary heart disease after prenatal exposure to the Dutch famine, 1944–1945. *Heart* **84**, 595–598.
Rowe, L. & Ludwig, D. (1991). Size and timing of metamorphosis in complex life cycles: Time constraints and variation. *Ecology* **72**, 413–427.
Royle, N. J., Lindstrom, J. & Metcalfe, N. B. (2005). A poor start in life negatively affects dominance status in adulthood independent of body size in green swordtails *Xiphophorus helleri*. *Proceedings of the Royal Society B: Biological Sciences* **272**, 1917–1922.
Royle, N. J. Lindstrom, J. & Metcalfe, N. B. (2006*a*). Effect of growth compensation on subsequent physical fitness in green swordtails *Xiphophorus helleri*. *Biology Letters* **2**, 29–42.
Royle, N. J., Metcalfe, N. B. & Lindstrom, J. (2006*b*). Sexual selection, growth compensation and fast-start swimming performance in Green Swordtails, *Xiphophorus helleri*. *Functional Ecology* **20**, 662–669.
Rudolf, V. H. W. & Rodel, M. O. (2007). Phenotypic plasticity and optimal timing of metamorphosis under uncertain time constraints. *Evolutionary Ecology* **21**, 121–142.
Schafer, M. L. & Lundstrom, J. O. (2006). Different responses of two floodwater mosquito species, *Aedes vexans* and *Ochletotatus sticticus* (Diptera: Culicidae), to larval habitat drying. *Journal of Vector Ecology* **31**, 123–128.
Scharf, I., Filin, I. & Ovadia, O. (2009). A trade-off between growth and starvation endurance in a pit-building antlion. *Oecologia* **160**, 453–460.
Schluter, D., Price, T. D. & Rowe, L. (1991). Conflicting selection pressures and life-history trade-offs. *Proceedings of the Royal Society of London Series B: Biological Sciences* **246**, 11–17.
Schulte-Hostedde, A. I., Zinner, B., Millar, J. S. & Hickling, G. J. (2005). Restitution of mass-size residuals: Validating body condition indices. *Ecology* **86**, 155–163.
Schultz, E. T., Lankford, T. E. & Conover, D. O. (2002). The covariance of routine and compensatory juvenile growth rates over a seasonality gradient in a coastal fish. *Oecologia* **133**, 501–509.
Schwarzenbach, G. A. & Ward, P. I. (2006). Responses to selection on phenoloxidase activity in yellow dung flies. *Evolution* **60**, 1612–1621.
Schwarzenbach, G. A. & Ward, P. I. (2007). Phenoloxidase activity and pathogen resistance in yellow dung flies *Scathophaga stercoraria*. *Journal of Evolutionary Biology* **20**, 2192–2199.
Scott, D. E., Casey, E. D., Donovan, M. F. & Lynch, T. K. (2007). Amphibian lipid levels at metamorphosis correlate to post-metamorphic terrestrial survival. *Oecologia* **153**, 521–532.
Shama, L. N. S. & Robinson, C. T. (2006). Sex-specific life-history responses to seasonal time constraints in an alpine caddisfly. *Evolutionary Ecology Research* **8**: 169–180.
Shine, R. (1988). The evolution of large body size in females: A critique of Darwin's fecundity advantage model. *American Naturalist* **131**: 124–131.
Sibly, R. & Calow, P. (1986). Why breeding earlier is always worthwhile. *Journal of Theoretical Biology* **124**, 311–319.
Sibly, R., Calow, P. & Nichols, N. (1985). Are patterns of growth adaptive? *Journal of Theoretical Biology* **112**, 553–574.
Sih, A. (1982). Foraging strategies and the avoidance of predation by an aquatic insect, *Notonecta hoffmani*. *Ecology* **63**, 786–796.
Siva-Jothy, M. T. & Thompson, J. J. W. (2002). Short-term nutrient deprivation affects immune function. *Physiological Entomology* **27**, 206–212.
Sogard, S. M. & Olla, B. L. (2002). Contrasts in the capacity and underlying mechanisms for compensatory growth in two pelagic marine fishes. *Marine Ecology Progress Series* **243**, 165–177.
Sokolovska, N., Rowe, L. & Johansson, F. (2000). Fitness and body size in mature odonates. *Ecological Entomology* **25**, 239–248.
Stearns, S. (1992). *The Evolution of Life Histories*. Oxford University Press, Oxford.
Stearns, S. C. & Koella, J. C. (1986). The evolution of phenotypic plasticity in life-history traits: Predictions of reaction norms for age and size at maturity. *Evolution* **40**, 893–913.
Stoks, R., De Block, M. & Mcpeek, M. A. (2006). Physiological costs of compensatory growth in a damselfly. *Ecology* **87**, 1566–1574.
Strobbe, F. & Stoks, R. (2004). Life history reaction norms to time constraints in a damselfly: differential effects on size and mass. *Functional Ecology* **83**, 187–196.
Sundstrom, L. F., Lohmus, M. & Devlin, R. H. (2005). Selection on increased intrinsic growth rates in coho salmon, *Oncorhynchus kisutch*. *Evolution* **59**, 1560–1569.
Takagi, Y. (2001). Effects of starvation and subsequent refeeding on formation and resorption of acellular bone in tilapia, *Oreochromis niloticus*. *Zoological Science* **18**, 632–629.
Tammaru, T. & Esperk, T. (2007). Growth allometry of immature insects: larvae do not grow exponentially. *Functional Ecology* **21**, 1099–1105.
Tammaru, T., Nylin, S. Ruohomaki, K. & Gotthard, K. (2004). Compensatory responses in lepidopteran larvae: a test of growth rate maximisation. *Oikos* **107**, 352–362.
Tanner, J. M. (1963). Regulation of growth in size in mammals. *Nature* **199**, 845–850
Teuschl, Y., Reim, C. & Blanckenhorn, W. U. (2007). Correlated responses to artificial body size selection in growth, development, phenotypic plasticity and juvenile viability in yellow dung flies. *Journal of Evolutionary Biology* **20**, 87–103.
Turelli, M. & Barton, N. H. (2004). Polygenetic variation maintained by balancing selection: Pleiotropy, sex-dependent allelic effects and GxE interactions. *Genetics* **166**, 1053–1079.
Urban, M. C. (2007). The growth-predation risk trade-off under a growing gape-limited predation threat. *Ecology* **88**, 2587–2597.
Urban, M. C. (2008). Salamander evolution across a latitudinal cline in gape-limited predation risk. *Oikos* **117**, 1037–1049.
Walling, C. A., Royle, N. J., Metcalfe, N. B. & Lindstrom, J. (2007). Early nutritional conditions and growth trajectories and mate choice: does compensatory growth lead to a reduction in adult sexual attractiveness? *Behavioural Ecology and Sociobiology* **61**, 1007–1014.
Wieser, W. (1994). Cost of growth in cells and organisms: general rules and comparative aspects. *Biological Reviews* **69**, 1–33.
Werner, E. E. & Anholt, B. R. (1993). Ecological consequences of the trade-off between growth and mortality rates mediated by foraging activity. *American Naturalist* **142,** 242–272.

Werner, E. E. & Gilliam, J. F. (1984). The ontogenetic niche and species interactions in size structured populations. *Annual Review of Ecology and Systematics* **15**, 393–425.

Wilson, A. J., Pemberton, J. M., Pilkington, J. G., Clutton-Brock, T. H., Coltman, D. W. & Kruuk, L. E. B. (2007). Quantitative genetics of growth and cryptic evolution of body size in an island population. *Evolutionary Ecology* **21**, 337–356.

Yamahira, K. & Takeshi, K. (2008). Variation in juvenile growth rates among and within latitudinal populations of the medaka. *Population Ecology* **50**, 3–8.

Yearsley, J. M., Kyriakis, I. & Gordon, I. J. (2004). Delayed costs of growth and compensatory growth rates. *Functional Ecology* **18**, 563–570.

Zahavi, A. (1975). Mate selection: selection for a handicap. *Journal of Theoretical Biology* **53**, 205–214.

Zahavi, A. (1977). Cost of honesty: further remarks on handicap principle. *Journal of Theoretical Biology* **67**, 603–605.

Zera, A. J. & Brink, T. (2000). Nutrient absorption and utilization by wing and flight muscle morphs of the cricket *Gryllus firmus*: implications for the trade-off between flight capability and early reproduction. *Journal of Insect Physiology* **46**, 1207–1218.

Zera, A. J. & R. F. Denno. (1997). Physiology and ecology of dispersal polymorphism in insects. *Annual Review of Entomology* **42**, 207–230.