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The Long-Term Effects of Juvenile Food Availability on Adult Reproductive Decisions and Success in Trinidadian Guppies (*Poecilia reticulata*)

A Dissertation submitted in partial satisfaction
of the requirements for the degree of

Doctor of Philosophy

in

Evolution, Ecology, and Organismal Biology

by

Sonya Karissa Auer

March 2011

Dissertation Committee:
Dr. David N. Reznick, Chairperson
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Chapter One is reproduced with permission from the following published manuscript: Auer, S. K., J. D. Arendt, R. Chandramouli, and D. N. Reznick. 2010. Juvenile compensatory growth has negative consequences for reproduction in Trinidadian guppies (*Poecilia reticulata*). Ecology Letters 13:998-1007.

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ABSTRACT OF THE DISSERTATION

The Long-Term Effects of Juvenile Food Availability on Adult Reproductive Decisions and Success in Trinidadian Guppies (*Poecilia reticulata*)

by

Sonya Karissa Auer

Doctor of Philosophy, Graduate Program in Evolution, Ecology, and Organismal Biology
University of California, Riverside, March 2011
Dr. David Reznick, Chairperson

Food availability during the juvenile stage can a have profound impact on adult performance and fitness. Low food availability early in life generally leads to slower growth and development and maturation at an older age and smaller size, which can then lead to reduced survival and lifetime reproductive success. There is increasing evidence that organisms can respond to these early setbacks and in ways that might be adaptive. However, our current understanding of the diversity of compensatory mechanisms utilized, the costs and benefits of these responses, and the relative importance of these responses in the wild is still in a nascent stage. For this dissertation, I investigated how juvenile food availability influences adult reproductive decisions and success in Trinidadian guppies (*Poecilia reticulata*).

First, I examined whether juvenile compensatory growth has negative consequences for reproduction. I found that juvenile compensatory growth did not affect adult growth rates, litter production rates, or investment in offspring size, but had negative effects on litter size. Second, I investigated whether individuals during the adult

stage can mitigate the negative effects of early setbacks caused by low food availability. I found that females reared on low food matured at a later age, a smaller size and with less energy reserves than females reared on high food. In response to this setback, they changed their investment in growth, reproduction and fat storage throughout the adult stage such that they were able to catch up in body size, increase their reproductive output and restore their energy reserves to levels comparable to those of females reared on high food. Finally, I explored the relative effects of juvenile versus adult environmental conditions on adult growth strategies in the wild. I found that events occurring during early and later parts of an individual's ontogeny both had important consequences for adult growth strategies, but the direction of their influence differed. Poor conditions during the juvenile stage had a positive effect on adult growth rates, while those same conditions in the recent past and in the present had a negative effect on adult growth.

Table of Contents

List of Tables

List of Figures

Introduction

Many organisms live in variable environments where conditions experienced at one time in their life history can differ markedly from those encountered at an earlier time and life stage. For example, many organisms have life cycles in which juveniles and adults live in distinct habitats and exploit different resources (Moran 1994). Many organisms also face temporal changes within a given habitat through stochastic fluctuations or regular seasonal changes in factors such as temperature and food availability (Poulin et al. 1992; Farley and Fitter 1999). Among these organisms, there is increasing evidence that past conditions can have persistent effects on individual performance (Lummaa and Clutton-Brock 2002; Harrison et al. 2010). These enduring effects are thought to arise because conditions at one time influence the internal physiological state of the individual and thereby affect its decisions and performance in the future (McNamara and Houston 1996). Understanding how and why organisms respond to their current environment therefore demands that we study the effects of previous environments on these responses and their fitness consequences.

Food availability during critical stages of early growth and development is thought to have a particularly profound impact on the developing phenotype and its performance as an adult (Monaghan 2008). For example, metabolic, cardiovascular, behavioral, reproductive and immune functions are all sensitive to early food levels (Bateson 2001; Kuzawa 2005; Jasienska et al. 2006; Schilder and Marden 2006), and can impact many different aspects of the phenotype, including its morphology (Stevens et al. 2000; Ohlsson and Smith 2001), physiology (Desai and Hales 1997; Saino et al. 1997;

Birkhead et al. 1999), and behavior (Henry and Ulijaszek 1996; Bateson 2001). Early food levels can also have lasting effects on fitness through their impact on reproductive success (e.g. Lummaa 2003; Reid et al. 2003; Blount et al. 2006; Descamps et al. 2008) and longevity (e.g. Osmond and Barker 2000; Alonso-Alvarez et al. 2006).

Long-term fitness costs of low early food levels often arise when reduced rates of growth and development associated with low food levels lead to maturation at a smaller size and/or at an older age. A smaller size at maturation can reduce fitness because smaller adults generally have a lower reproductive potential and are more vulnerable to predation while delayed maturation can reduce fitness because it increases generation time and can decrease the reproductive lifespan (Roff 2002). Low food availability during the juvenile stage of life can also reduce energy reserves at maturation and lead to increased susceptibility to starvation, disease and predation in the adult stage of life (Lindström 1999; Morgan and Metcalfe 2001; Lummaa and Clutton-Brock 2002; McMillen and Robinson 2005).

Given that poor early conditions can be detrimental to future performance, selection might be expected to favor mechanisms that mitigate the negative effects of early setbacks, provided that the costs of such responses are not too high (Metcalfe and Monaghan 2001). Indeed, there is increasing evidence that organisms can respond to these early insults and in ways that might be adaptive (Metcalfe and Monaghan 2001). Brain sparing and the preservation of critical organs at the expense of less critical ones has been observed in human children and insects and is thought to be an adaptive response to poor food levels early in life (Finch and Kirkwood 2000; Stevens et al. 2000).

During the juvenile stage, many organisms can accelerate their growth when conditions improve after a period of growth restriction, a response called compensatory growth, and can thereby recoup their lost energy reserves and catch up in body size to the size they would have achieved under more favorable conditions (Metcalfe and Monaghan 2001). While these responses during the juvenile stage can trade-off with other fitness components later in life (Johnsson and Bohlin 2006; De Block and Stoks 2008; Auer et al. 2010), they are thought to be adaptive strategies that make the best of a bad situation (Metcalfe and Monaghan 2001). However, our current understanding of the diversity of compensatory mechanisms utilized, the costs and benefits of these responses, and the relative importance of these responses under environmental variability in the wild is still in a nascent stage.

The purpose of this dissertation is to investigate how early food availability influences adult reproductive decisions and success. Specifically, I address the following questions: 1.) Does juvenile compensatory growth, in response to an increase in food levels following a period of growth restriction, have negative consequences for reproduction? 2.) Can individuals during the adult stage mitigate the negative effects of early setbacks caused by low food availability? And 3.) What is the relative importance of juvenile versus adult environmental conditions on adult growth strategies in the wild? I address these questions in a small, freshwater fish, the Trinidadian guppy (*Poecilia reticulata*) using longitudinal studies in both controlled laboratory settings and in the field.

In Chapter One, I examine whether there are reproductive costs to juvenile compensatory growth. Compensatory or "catch-up" growth may be an adaptive mechanism that buffers the growth trajectory of young organisms from deviations caused by reduced food availability. Theory generally assumes that rapid juvenile compensatory growth impacts reproduction only through its positive effects on age and size at maturation, but potential reproductive costs to juvenile compensatory growth remain virtually unexplored. I used a food manipulation experiment to examine the reproductive consequences of compensatory growth in female guppies. Specifically, I compared different components of reproduction between guppies that were experimentally manipulated to undergo either routine or compensatory growth as juveniles.

I found that juvenile compensatory growth did not affect adult growth rates, litter production rates, or investment in offspring size. However, compensatory growth had negative effects on litter size, independent of the effects of female body length, resulting in a 20% decline in offspring production. These results demonstrate that rapid juvenile compensatory growth not only has costs for survival, but can also be detrimental to reproductive success.

In Chapter Two, I examine whether adults can mitigate the negative effects of low food availability during early growth and development. Low food during the early stages of life can potentially have negative effects on later success because it can lead to sexual maturation at a later age, smaller size and with lower energy reserves. Phenotypic plasticity in adult life history decisions may help to mitigate these potential costs, yet adult life history responses to juvenile food conditions remain largely unexplored. I used

a food manipulation experiment with female guppies to examine age-related changes in adult life history responses to early food conditions, whether these responses varied across different adult food conditions, and how these responses affected overall reproductive success. I reared juvenile guppies on either a low or high food level. Once they reached sexual maturity, half of the females were switched to the opposite food level and the other half were kept on the same ration trajectory received during the juvenile stage for a full factorial design of two rations levels across two life stages. Females then remained on their respective adult food rations until they gave birth to three litters.

I found that guppy females reared on low food as juveniles matured at a later age, a smaller size and with less energy reserves than females reared on high food as juveniles. In response to this setback, they changed their investment in growth, reproduction and fat storage throughout the adult stage such that they were able to catch up in body size, increase their reproductive output and restore their energy reserves to levels comparable to those of females reared on high food as juveniles. The net effect was that adult female guppies did not merely mitigate, but surprisingly were able to fully compensate for the potential long-term negative effects of poor juvenile food conditions on reproductive success. These results demonstrate that flexibility in growth and reproductive strategies can mitigate potential setbacks caused by a poor start to life and importantly that low food availability during early growth and development does not always spell doom for adult performance.

In Chapter Three, I explore the relative effects of conditions in the juvenile stage and those in the more recent past on adult somatic growth rates in wild guppies. Current

environmental conditions as well as those in the recent past and more distant juvenile stage can have significant effects on individual performance and population dynamics, but their relative importance remains unexplored. I used bimonthly estimates of food availability and population density coupled with longitudinal measurements of female body size to examine female growth responses to current environmental conditions and how those growth rates were affected by conditions in the immediate past as well as in the juvenile stage.

I found that events occurring during the juvenile and adult stage of an individual's ontogeny both had important consequences for adult growth strategies, but the direction of their influence differed. Poor conditions during the juvenile stage had a positive effect on adult growth rates, while those same conditions in the recent past and in the present had a negative effect on adult growth. These results demonstrate that the same conditions at different life stages can have different effects on the state of the organism and its subsequent short- and long-term growth strategies.

This dissertation demonstrates that early food availability can have a profound influence on adult reproductive decisions and success. First, it shows that juvenile compensatory growth can come at a cost to future reproduction and may therefore influence how compensatory and other growth strategies evolve. Second, it highlights that how individuals fare as adults is not merely a passive consequence of the limits imposed by early conditions, but rather that flexibility in adult growth and reproductive strategies can mitigate the effects of growing up in a poor environment. Finally, it illustrates that effects of early conditions on adult growth strategies are as important as

those of more recent conditions and suggests that a simultaneous consideration of events in both the recent and distant past may improve predictions for individual responses to environmental change.

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Chapter 1

Juvenile Compensatory Growth Has Negative Consequences for Reproduction in Trinidadian Guppies (*Poecilia reticulata*)

Abstract

Compensatory or “catch-up” growth may be an adaptive mechanism that buffers the growth trajectory of young organisms from deviations caused by reduced food availability. Theory generally assumes that rapid juvenile compensatory growth impacts reproduction only through its positive effects on age and size at maturation, but potential reproductive costs to juvenile compensatory growth remain virtually unexplored. I used a food manipulation experiment to examine the reproductive consequences of compensatory growth in Trinidadian guppies (*Poecilia reticulata*). Compensatory growth did not affect adult growth rates, litter production rates, or investment in offspring size. However, compensatory growth had negative effects on litter size, independent of the effects of female body length, resulting in a 20% decline in offspring production. I discuss potential mechanisms behind this observed cost to reproduction.

Introduction

Young organisms often face considerable challenges in acquiring resources because of the heightened mortality risks, increased competitive pressures and reduced physical capabilities associated with their small body size (Conover and Schultz 1997). These tough circumstances can be exacerbated if young are born during a time of low resources, if they are relatively small because of reduced parental provisioning, or if they are born late in the season (Lindstrom 1999; Metcalfe and Monaghan 2001). Reduced food intake during early growth and development can then have negative consequences for fitness. Low resources early in life can lead to slower rates of growth and development and thereby to maturity at an older age and smaller size. Delayed maturation decreases fitness because it increases generation time and can decrease the reproductive lifespan (Roff 1992). Smaller body size at maturation can also lead to reduced fitness because of the general positive effect that body size has on survival and reproductive success (Roff 1992). Given these potential fitness costs, selection might be expected to favor mechanisms that mitigate the negative effects of early setbacks in size at age, provided that the costs of such responses are not too high (Metcalfe and Monaghan 2001).

Compensatory or ‘catch-up’ growth is a common form of growth plasticity in which growth is accelerated to a rate above that of normal routine growth in response to an increase in resources following a period of growth restriction (Metcalfe and Monaghan 2001). Compensation can occur through recoupment of energy reserves and through increased investment in structural growth and thereby lead to partial, full or even

overcompensation in energy reserves and body size (Ali et al. 2003; Metcalfe and Monaghan 2003). Compensatory growth is commonly financed through a hyperphagic response to an increase in resource levels (Ali et al. 2003), but it can also be achieved through changes in allocation rules that direct energy towards growth at the expense of other functions and activities (Sogard and Olla 2002).

Compensatory growth may be an adaptive mechanism that buffers the growth trajectory of young organisms from deviations caused by reduced resources early in life (Metcalfe and Monaghan 2001). However, compensatory growth and rapid growth in general are known to have negative consequences for survival. When increased growth rates are financed by increased food intake, higher foraging rates can lead to increased risk of predation in the short-term (Lankford Jr. et al. 2001). Compensatory growth can also increase mortality risk and reduce lifespan through its long-term detrimental effects on disease resistance, starvation resistance, oxidative stress and over-winter mortality (Forsen et al. 2004; Dmitriew and Rowe 2005; Johnsson and Bohlin 2006; De Block and Stoks 2008). These survival costs are, in turn, thought to shape the evolution of the compensatory growth response (Yearsley et al. 2004; Mangel and Munch 2005).

While great emphasis has been placed on understanding survival costs to compensatory growth, its impact on reproduction, an equally important component of fitness, remains virtually unexplored (but see Morgan and Metcalfe 2001; Dmitriew and Rowe 2007). Current theory generally assumes that compensatory growth impacts reproduction only through its positive effects on age and size at maturation (Abrams et al. 1996; Ali et al. 2003; Metcalfe and Monaghan 2003; Yearsley et al. 2004; but see

Lindstrom et al. 2005; Mangel and Munch 2005 for exceptions). However, many of the developmental and physiological mechanisms thought to underlie survival costs of compensatory growth may also have negative impacts on reproduction. For example, the increases in metabolic demands (Stoks et al. 2006) and reductions in cognitive function (Fisher et al. 2006), energy reserves (Stoks et al. 2006), and physical performance (Royle et al. 2006) associated with compensatory growth could negatively impact the reproductive success of the organism as well as its survival. Alternatively, reproductive costs to juvenile compensatory growth could arise as an indirect result of a trade-off between compensatory growth and some other trait closely tied to reproduction (Reznick 1985). For example, if juvenile compensatory growth reduces later adult survival, then as adults, individuals may respond by investing more energy in survival at the expense of reduced current reproduction. If compensatory growth has negative consequences for later reproduction, then these costs have the potential to influence how compensatory and other plastic growth strategies evolve.

I examined the reproductive consequences of juvenile compensatory growth in Trinidadian guppies (*Poecilia reticulata*) by comparing different components of reproduction between guppies that were experimentally manipulated to undergo either routine or compensatory growth as juveniles. Specifically, I examined the effects of juvenile growth history on adult rates of litter production and investment in offspring size and offspring number. Because allocation of energy to adult growth comes at the expense of investment in reproduction, I also examined treatment differences in adult growth rates. In addition, I looked at the effects of compensatory growth and subsequent

reproduction on female energy reserves. I predicted that if there is a reproductive cost to compensatory growth, then guppies that underwent compensatory growth as juveniles would exhibit slower rates of litter production, reduced adult growth rates, fewer offspring per litter and/or smaller, lower quality offspring relative to individuals that exhibited normal routine growth rates during the juvenile stage of life.

Methods

Guppies are small poeciliid fishes that have internal fertilization and bear live young. Young develop rapidly and can give birth to their first litter as early as 60-70 days of age (Reznick and Bryga 1996). They initiate a new litter of young during or shortly after the birth of the prior litter and can give birth at regular intervals of approximately 25 days. Energy used to fuel each litter is taken in part from the female's somatic reserves as well as from energy accrued during the time of yolking (Reznick and Yang 1993). Females used in the assay were offspring of laboratory born descendents of fish collected in 2003 from the Turure River in Trinidad, West Indies.

Juvenile growth

I initially reared offspring from a given litter together at densities of 8-10 individuals in 8 liter aquaria. Offspring were from 22 litters, each litter from a different female. Fish were fed a diet of liver paste in the morning and *Artemia* brine shrimp nauplii in the evening. Juveniles in group tanks were fed 2 microliters per individual in the morning and 2 microliters per individual in the evening, amounting to roughly 24 J per individual per day. At age 14 days I randomly chose 2 sibling females of similar body mass and length from each of the 22 litters. Females were sexed based on the triangular

pattern of melanophore development in the abdominal region that is otherwise absent in males (Reznick 1990). Females were assigned to their own 8 liter aquaria and to one of two food treatments. One sibling per litter was fed an ad libitum ration throughout the experiment, hereafter control fish (n = 22). The food ration of the other sibling was reduced by half for the next two weeks, hereafter referred to as the "growth restriction period", from age 14-28 days and then fed ad libitum throughout the remainder of the experiment, hereafter experimental fish (n = 22). The food level used for experimental fish during the growth restriction period – 1 microliter in the morning and 1 microliter in the evening – was predetermined to allow some growth and maintain fat reserves comparable to those observed in the wild (Reznick et al. 2001). Food levels were otherwise adjusted each week to compensate for changes in female size and maintain rations predetermined to be at an ad libitum level (Reznick 1980). Ad libitum food rations were always only partially consumed, and uneaten food was removed daily. I used a randomized block design to control for variation among individuals in their pedigree and microenvironment experienced in the laboratory: sister pairs were placed next to one another in a randomly assigned position in the laboratory.

I measured standard length and mass each week during the juvenile stage. Individuals were anesthetized in a solution of neutrally buffered MS-222. Each fish was measured under a dissecting scope using digital calipers and then weighed using an Ohaus electronic balance (Pinebrook, NJ, USA). Aquaria were cleaned every two weeks with a partial water change to remove build up of nitrogenous waste.

Female reproduction

Females were mated overnight each week beginning at age 35 days, approximately a week before the earliest estimated age at maturation in ad libitum fed guppies (Reznick 1980). Females were mated overnight once a week with males randomly selected from the same stock of stud males. Aquaria were checked twice daily for newborn offspring. Offspring were collected when born, euthanized with a fatal overdose of MS-222 and preserved in 5% formalin. Female body length and mass were measured on the day each litter was born. Females were euthanized and preserved in the same manner after giving birth to their fourth litter.

I calculated litter size (number of offspring per litter), the mean offspring lean dry mass, and the mean extractable fat content per offspring for the first four litters of each female as well as the length of the three interlitter intervals between the four successive litters. Interlitter interval was calculated as the number of days between 2 consecutive litters. To determine the mean offspring lean dry mass, I first dried each litter overnight in an oven at 60 deg C. I then weighed the dry mass of each litter and submerged it in anhydrous ether to remove triglycerides until it reached a constant lean dry weight (Reznick and Endler 1982). The mean lean dry mass per offspring was then calculated as the total lean dry mass divided by the number of young in the litter. The difference between the dry weight and lean weight of a litter represents the fat content of that litter, which when divided by the number of offspring in a given litter, gives the mean extractable fat content (in mg) per offspring. Offspring size as well as body

condition (fat reserves) are indices of the quality of the offspring since they are both known to influence offspring growth and survival (Berkeley et al. 2004; Bashey 2008).

Juvenile and adult body condition

I examined treatment differences in fat reserves in juveniles at the end of the growth restriction period and in adults at the birth of their fourth litter. To assess juvenile fat content, I reared 14 additional pairs of female siblings, each pair from a different mother, under the same conditions as the treatments described above. One sibling of each pair was assigned to the control and the other to the experimental treatment. These siblings were raised until the end of the two week growth restriction period (28 days of age), euthanized, preserved and analyzed for fat content, as described above. Fat content of females preserved at the birth of their fourth litter was also analyzed using these same procedures.

Statistical analysis

I used a linear mixed model approach to examine the effects of treatment on juvenile growth and adult reproductive traits. For analyses where the dependent variable was measured on only one occasion for each individual, I included block as a random effect to account for variation in pedigree and microenvironment in the laboratory. For analyses of longitudinal data, I accounted for the random effects of female identity- her intercept and slope - and thereby controlled for the non-independence of repeated measures on the same female (Singer and Willett 2003; Bolker et al. 2009). I used a model selection approach whereby I tested hypotheses about the contribution of fixed effects in nested models. I started with a full model for each trait and used backward

model selection, sequentially eliminating terms with the lowest F-values until all terms in the model were significant. In general, a fixed factor or its interaction with another predictor was dropped from the model when its effect was not significant. However, because I was testing hypotheses about the effect of treatment on the different dependent variables, the main effect of treatment was always retained in the final model. I compared alternative models produced by maximum likelihood estimation, but report parameter estimates for the final model produced by restricted maximum likelihood estimation. If one of the random effects was not estimable due to model overparameterization, I excluded that effect.

Juvenile and adult growth: I first tested for differences in body size of experimental and control fish at the beginning and at the end of the two week growth restriction period (age 14 and 28 days). I also examined treatment differences in growth during the growth restriction period. I then tested for treatment differences in juvenile growth in body mass and length once experimental fish returned to ad libitum food conditions. Because experimental and control fish were different sizes at the end of the growth restriction period and because growth is often size-dependent, I compared growth rates among treatment groups using a "size/time" approach whereby growth trajectories of experimental fish during the compensation phase were 'slid back in time' to a common starting size with control fish (Mangel and Munch 2005; Nicieza and Alvarez 2009). Because experimental fish grew during the growth restriction period, I could not simply slide them back in time to the beginning of the growth restriction period when the control fish were 14 days old. So, I compared body sizes (mass and length) of the control fish at

ages 15, 16, and up to 20 days with those of the experimental fish at age 28 days to find the age at which the control fish were the same size as the experimental fish. The mean daily growth rates in mass and length of the control fish calculated for the week spanning ages 14-21 days were used to estimate their mass and length, respectively, on each of these days. When the control fish were 17 days old, they did not differ in body mass ($F_{1,41}$ = 1.33, p = 0.26) or length ($F_{1,41}$ = 3.49, p = 0.07) from experimental fish at age 28 days. Thus, experimental fish were slid back 11 days to a common starting size with the control fish at age 17 days. I analyzed growth across four ages (controls: 17, 21, 28, and 35 days of age; experimental fish: 28, 35, 42 and 49 days of age). Growth was analyzed until experimental fish were 49 days old, the age when our back-calculations indicated most of these individuals were mature. Finally, I examined whether experimental fish caught up in body size to control fish by the time they were 49 days old.

I then tested for treatment differences in adult growth in body mass and length. The yolking of eggs occurs at defined intervals in guppies and requires energy otherwise used for growth (Reznick and Yang 1993), so weekly changes in adult body size can depend on stage of pregnancy (Auer unpublished data). I therefore controlled for stage of pregnancy by analyzing growth rates between litters, i.e. between the age at which a female yolked the eggs for the first litter (i.e. at sexual maturity) and each of the ages at which she yolked a subsequent litter. Because guppies are livebearers, sexual maturity in females is only visibly apparent at the birth of the first litter. However, age at maturation can be approximated by subtracting the length of one gestation period from the age at first parturition (Reznick 1982). To estimate age at maturation, I first calculated the

interval between the first and second litter for each individual, and then subtracted that interlitter interval (the gestation period) from the age that individual first gave birth. I then used my weekly measurements of body size up until first parturition to estimate body size at the estimated age at maturation for each individual. For subsequent litters, the age and size at yolking corresponded to measurements taken at the birth of the previous litter. Adult growth was calculated as (body $\text{size}_{\text{litter } i+1}$ – body $\text{size}_{\text{litter } i}$) /interlitter interval.

Female reproduction: I first tested individually for differences in age and size at sexual maturation between treatment groups. I then tested for treatment differences in four reproductive traits – interlitter interval, mean offspring lean dry mass, mean extractable fat per offspring and litter size – and how those traits changed with litter number and female body length. Because the effect of treatment on litter size changed with adult body length, I re-centered the intercept in the final model on litter number and the grand mean female length for each litter and tested for treatment differences in litter size at each of the four litters (sensu Singer and Willett 2003; West et al. 2006).

Juvenile and adult body condition: I examined potential differences in fat reserves between treatments in the subset of female fish preserved at age 28 days and also in females preserved after the birth of their fourth litter. Comparisons of fat content controlled for effects of lean dry body mass (Christians 1999; Packard and Boardman 1999). Residual body mass (after controlling for body length) was a good predictor of the fat content measured in females preserved after the birth of their fourth litter ($F_{1,42} = 6.09$, $p = 0.02$), so I also tested for treatment differences in body condition at maturation.

In all analyses, diagnostics were run to ensure that the functional form of the model was linear or quadratic and to inspect the distribution of the residuals. With the exception of litter size, all traits were normally distributed. Litter sizes are count data and their variance was greater than the mean, so models testing effects of treatment on litter size assumed a quasi-Poisson distribution. Main effects and interactions were regarded as significant when $P < 0.05$. All models were run using SAS version 9.2 (SAS Institute, NC, USA). Because I was interested in the fixed effects, I present results of parameters estimates and significance values for both fixed and random effects in each final model, but only discuss results for fixed effects.

Results

Juvenile and adult growth

At age 14 days there was no significant difference between experimental and control fish in either body mass (mean ± 1SE: 17.3 ± 0.6 and 16.4 ± 0.6 mg respectively; treatment: $F_{1,21} = 2.28$, $p = 0.15$; block: Wald $Z = 2.22$, $p = 0.01$ on ln-transformed data) or standard length (9.5 ± 0.9 and 9.4 ± 0.1 mm respectively; treatment: $F_{1,21} = 4.51$, $p = 0.05$; block: Wald $Z = 2.54$, $p = 0.01$ on ln-transformed data). During the growth restriction period, experimental fish grew at a significantly slower rate than control fish in both mass (0.5 ± 0.1 vs. 3.8 ± 0.2 mg/day, respectively; treatment: $F_{1,19} = 98.8$, $p < 0.001$; initial mass: $F_{1,19} = 85.8$, $p < 0.001$; block: Wald $Z = 0.86$, $p = 0.19$) and length (0.1 ± 0.01 vs. 0.4 ± 0.01 mm/day, respectively; treatment: $F_{1,19} = 123.8$, $p < 0.001$; initial length: $F_{1,19} = 36.4$, $p < 0.001$; block: Wald $Z = 0.30$, $p = 0.38$) and as a result were significantly smaller than controls at 28 days of age in both mass (24.0 ± 1.2 vs. 70.2 ±

2.5 mg, respectively; treatment: $F_{1,21}$ = 534.8, p <0.001; block: Wald Z = 1.52, p = 0.06 on ln-transformed data) and length (10.9 ± 0.2 vs. 14.8 ± 0.2 mm, respectively; treatment: $F_{1,21}$ = 415.5, p <0.001; block: Wald Z = 1.37, p = 0.09 on ln-transformed data).

Once returned to ad libitum rations, experimental fish exhibited a compensatory growth response in both body mass and length, growing at a significantly faster rate than control fish (Table 1.1, Figure 1.1). Compensatory growth in body mass in experimental fish, when controlling for effects of mass at the beginning of each growth period, began immediately after the growth restriction period ended and continued until they were 49 days old; growth of experimental fish during this time was roughly 30% faster than control fish (Figure 1.1 *A*,*C*). Compensation in body length in the experimental fish, on the other hand, did not commence until the second week after the growth restriction period ended. Daily growth in length in experimental fish was initially roughly 90% of that exhibited by control fish, but then accelerated to a rate approximately 30% higher than the controls (Figure 1.1 *B*,*D*). By 49 days of age when most experimental fish had matured, experimental fish were still significantly smaller than control fish in both mass (161.3 ± 6.1 vs. 242.5 ± 6.6 mg, respectively; treatment: $F_{1,21}$ = 153.4, p <0.001; block: Wald Z = 1.80, p = 0.04 on ln-transformed data) and length (19.1 ± 0.2 vs. 22.0 ± 0.2 mm, respectively; treatment: $F_{1,21}$ = 338.1, p <0.001; block: Wald Z = 1.57, p = 0.06 on ln-transformed data) and therefore had only partially compensated in body size.

During the adult stage, daily growth rates in both mass and length declined with litter number in both treatments, but experimental fish grew at the same rate as control

fish (Table 1.2, Figure 1.2 *A*,*B*). Thus, differences in size between experimental and control fish at 49 days of age persisted through adulthood.

Female reproduction

Experimental fish matured at a significantly later age than control fish (49.4 ± 1.3 vs. 43.3 ± 1.5 days of age, respectively; treatment: $F_{1,21} = 30.9$, $p < 0.001$; block: Wald $Z = 1.02$, $p = 0.15$). However, there was no significant difference in body size between experimental and control fish in either mass (167.4 ± 12.4 vs. 187.4 ± 16.8 mg, respectively; treatment: $F_{1,21} = 1.20$, $p = 0.29$; block: Wald $Z = 0.58$, $p = 0.28$ on ln-transformed data) or length (19.6 ± 0.4 vs. 20.1 ± 0.5 mm, respectively; treatment: $F_{1,21} = 0.91$, $p = 0.35$; block: Wald $Z = 1.37$, $p = 0.09$ on ln-transformed data).

Interlitter interval increased with litter number, while mean offspring lean dry mass and mean offspring extractable fat increased with female body length. However, none were affected by treatment (Table 1.3, Figure 1.3 *A*-*C*). In contrast, treatment had a significant effect on litter size and how it changed with female body length (Table 1.3, Figure 1.3 *D*). When accounting for effects of litter number, litter size increased at a slower rate with female length in experimental fish relative to controls (Table 1.3, Figure 1.3 *D*). Because of this significant treatment by female length interaction, litter size was similar during the first litter (Table 1.3), but increasingly lower in the second (intercept: $t = 24.0$, $p < 0.001$, estimate ± 1SE: 28.1 ± 1.2; treatment: $t = -3.4$, $p < 0.001$, estimate: -5.47 ± 1.6), third (intercept: $t = 31.9$, $p < 0.001$, estimate: 42.0 ± 1.3; treatment: $t = -4.19$, $p < 0.001$, estimate: -7.6 ± 1.8), and fourth (intercept: $t = 32.8$, $p < 0.001$, estimate: 54.1 ± 1.7; treatment: $t = -4.42$, $p < 0.001$; estimate: -9.2 ± 2.1) litters when estimated at the

grand mean length at the yolking of each of the litters. Predicted differences in litter size therefore amounted roughly to 5, 8 and 9 offspring in the second through fourth litters, respectively. This difference reflected an approximate 20% decline in litter size in experimental relative to control fish.

Juvenile and adult body condition

In the subset of individuals preserved at age 28 days, experimental fish had a significantly lower amount of fat reserves than control fish when controlling for lean dry body mass (Figure 1.4; treatment: $F_{1,12} = 9.70$, p = 0.01; lean dry body mass: $F_{1,12} = 8.26$, p = 0.01; block: Wald $Z = 1.43$, p = 0.08 on ln-transformed data). Differences in body condition between treatment groups, estimated as residual ln-transformed body mass after removing effects of ln-transformed body length, were no longer evident at the estimated age of maturation (treatment: $F_{1,20} = 0.64$, p = 0.43; length: $F_{1,20} = 259.8$, $p < 0.001$; block: Wald $Z = 0.97$, p = 0.17). By the end of the experiment when females gave birth to their fourth litter, there was no significant difference in fat reserves between treatments (Figure 1.4; treatment: $F_{1,20} = 2.71$, p = 0.12; lean dry body mass: $F_{1,20} = 66.3$, $p < 0.001$; block: Wald $Z = 0.17$, p = 0.43 on ln-transformed data).

Discussion

The food manipulation was successful in inducing compensatory growth in both mass and length. Experimental fish accelerated their growth rates after food returned to ad libitum levels, increasing growth in mass immediately but not increasing their growth in length until one week after the growth restriction period ended. They then maintained the same growth rates as controls during the adult stage after only partially compensating

in mass and length. Experimental fish matured approximately 6 days later but at the same mass and length as controls. Interlitter interval, mean offspring lean dry mass, and mean offspring extractable fat were not affected by juvenile growth history. In contrast, juvenile compensatory growth led to reduced fecundity. After controlling for the effects of litter number and female length, litter size was similar in the first litter but was increasingly lower in the second, third and fourth litters in experimental relative to control fish. While compensatory growth has been shown to reduce the occurrence of precocious sexual maturation in males (Morgan and Metcalfe 2001), this is the first study, to our knowledge, to demonstrate that it can have negative effects on female reproduction and that the magnitude of these effects increases throughout adulthood.

Costs to reproduction could arise through a direct trade-off between compensatory growth and later reproduction. For example, compensatory growth could have negative impacts on reproductive development and physiology if it decreases the efficiency with which energy is assimilated or if it interferes with the development of reproductive structures. There is some evidence suggesting that rapid juvenile growth can interfere with the development of non-reproductive structures (Ricklefs et al. 1994; Arendt et al. 2001; Arendt 2003), but its impact on reproductive development and physiology are unknown. Compensatory growth could also negatively affect reproduction if it increases metabolic needs and thereby decreases the amount of energy available for reproduction. However, the effects of compensatory growth on metabolic rates are currently equivocal (Wieser et al. 1992; Stoks et al. 2006; Criscuolo et al. 2008).

Alternatively, the observed trade-off between compensatory growth and later reproduction could arise if juvenile growth impacts another trait closely tied to reproduction and alters how the trade-off between that trait and reproduction is resolved. Investment in reproduction is intimately connected to investment in adult growth and survival. Because energy is a finite resource, adults must 'decide' how to partition energy among the competing demands of these fitness-related traits (Roff 2002). How these trade-offs are resolved often depends on the state of the individual (McNamara and Houston 1996). Thus, if compensatory growth has long-term negative effects on adult growth potential or survival probability, these impacts may change how the trade-off between these traits and reproduction is resolved. For example, the well-known long-term negative effects of rapid juvenile growth on adult survival may favor increased investment in survival at the expense of reproduction and lead to the reproductive costs observed. While I did not measure investment in adult survival or changes in growth efficiency, these potential mechanisms certainly remain plausible.

Finally, the observed reduction in litter size could stem from long-term effects of the period of early growth restriction. Low food levels during early growth and development can have long-term negative effects on reproduction (Lindstrom 1999; Lummaa and Clutton-Brock 2002). However, they are also known to have no effect in some studies (Taborksy 2006), or positive effects on different components of reproduction in other studies (Painter *et al.* 2008). In our study, experimental fish were subjected to a food level during the growth restriction period that allowed them to grow approximately 1.3 mm during the two week restriction period, a rate of growth that falls

well within the range of 0.25-2.25 mm growth per two week period observed in the wild (Grether et al. 2001; Reznick et al. 2001). In addition, treatment differences in body condition present at the end of the growth restriction period were no longer evident at maturation. Thus, it seems unlikely that there were any long-term negative effects of early growth restriction. However, I cannot discount the possibility that the long-term reduction in reproductive potential is a direct consequence of the growth restriction period.

Events during the juvenile stage are thought to be tightly coupled with fitness in the adult stage through the size at which maturation occurs. In our study, juvenile growth history had a significant impact on litter size that was independent of the effects of female size. However, it is presently unclear what mechanisms underlie this observed cost to reproduction and why costs were delayed until later litters. While further studies are needed to fully tease apart the separate effects of the initial growth restriction from the subsequent growth acceleration, our results suggest that reproductive costs to accelerated juvenile growth may play an important role in the evolution of compensatory growth and other rapid juvenile growth responses.

Table 1.1. Results of final linear mixed model testing for effects of juvenile food level regime (treatment) on juvenile growth in body mass (mg per day) and body length (mm per day). Results are given for control (= 0) and experimental (= 1) fish. Size and growth trajectories of experimental fish starting at the end of the two week growth restriction period (28 days of age) were slid back in time back to a common starting size with the control fish (17 days of age). Parameter estimates for each model are centered on age 28 days for experimental fish and age 17 days for control fish.

	Juvenile growth in mass (mg per day)			Juvenile growth in length (mm per day)		
	Fixed Effects					
	Estimate	SE	*t*	Estimate	SE	*t*
Intercept	1.75	0.33	5.35***	0.38	0.01	32.3***
Age†	0.31	0.07	4.37***	0.01	< 0.01	3.19**
Age^2	-0.02	0.01	-4.14***	< -0.01	< 0.01	-5.14***
Initial size‡	0.05	0.01	4.89***	n/s		
Treatment	1.29	0.23	5.70***	-0.03	0.02	-1.84
Age x Treatment	n/s			0.01	< 0.01	4.96***
Age^2 x Treatment	n/s			n/s		
	Random Effects					
	Estimate	SE	Wald *Z*	Estimate	SE	Wald *Z*
Variance in female slope	0.01	< 0.01	2.09*	< 0.01	< 0.01	1.37
Residual variance	1.14	0.18	6.49***	< 0.01	< 0.01	6.59***

***P < 0.001; **P < 0.01; *P < 0.05; $^{n/s}$P > 0.05 and term dropped from model.

† Age is measured in days.

‡ Initial size at the beginning of each growth period; initial mass (in mg) was used for the juvenile growth in mass model and initial length (in mm) was used in the juvenile growth in length model.

Table 1.2. Results of final linear mixed model testing for effects of juvenile food level regime (treatment) on adult daily growth in body mass (mg per day) and body length (mm per day) between litters. Results are from control (= 0) and experimental (= 1) treatment fish. Parameter estimates are centered on litter 1.

	Adult growth in mass (mg per day)			Adult growth in length (mm per day)		
	Fixed Effects					
	Estimate	SE	*t*	Estimate	SE	*t*
Intercept	9.34	0.52	17.9***	0.25	0.01	27.1***
Litter	n/a			-0.12	0.01	-7.95***
Litter^2	-0.20	0.04	-5.75***	0.01	< 0.01	4.11***
Treatment	-0.31	0.66	-0.47	< -0.01	0.01	-0.30
Litter*Treatment	n/s			n/s		
Litter^2*Treatment	n/a			n/s		
	Random Effects					
	Estimate	SE	Wald *Z*	Estimate	SE	Wald *Z*
Variance in female intercept	3.07	1.09	2.80**	< 0.01	< 0.01	3.35***
Residual variance	7.87	0.86	7.87***	< 0.01	< 0.01	7.89***

***P < 0.001; **P < 0.01; $^{n/s}$P > 0.05 and term dropped from model; $^{n/a}$ term not included in model.

Table 3. Results of final linear mixed model testing for effects of juvenile food level regime (treatment) on adult changes in interlitter interval (days), mean offspring lean dry mass (mg) and mean offspring extractable fat (mg). Results are given for control (= 0) and experimental (= 1) fish. Parameter estimates for each model are centered on litter 1 and the grand mean body length (19.8 mm) at the yolking of the first litter.

	Interlitter interval (days)		Mean offspring lean dry mass (mg)		Mean offspring extractable fat (mg)		Litter size	
	Fixed effects							
	Estimate (SE)	*t*	Estimate (SE)	*t*	Estimate (SE)	*t*	Estimate (SE)	*t*
Intercept	23.22 (0.30)	77.0***	0.56 (0.02)	32.8***	0.10 (0.01)	16.8***	10.67 (1.21)	8.82***
Litter	0.53 (0.11)	4.97***	n/s		n/s		7.56 (1.29)	5.88***
Body length†	n/a		0.01 (< 0.01)	6.91***	0.01 (< 0.01)	11.0***	1.67 (0.28)	5.96***
Treatment	-0.05 (0.42)	-0.12	0.03 (0.02)	1.61	0.01 (0.01)	1.92	-2.15 (1.63)	-1.31
Litter*Treatment	n/s		n/s		n/s		n/s	
Body length*Treatment	n/a		n/s		n/s		-0.56 (0.15)	-3.66***
	Random effects							
	Estimate (SE)	Wald *Z*	Estimate (SE)	Wald *Z*	Estimate (SE)	Wald *Z*	Estimate (SE)	Wald *Z*
Variance in female intercept	1.33 (0.43)	3.08**	0.01 (< 0.01)	3.31***	< 0.01 (< 0.01)	2.59**	18.93 (5.83)	3.24***
Residual variance	2.44 (0.31)	7.96***	0.01 (< 0.01)	7.64***	< 0.01 (< 0.01)	7.83***	1.14 (0.15)	7.69***

***P < 0.001; **P < 0.01; *P < 0.05; n/s P > 0.05 and term dropped from model; n/a term not included in model.

† Body length is measured in millimeters.

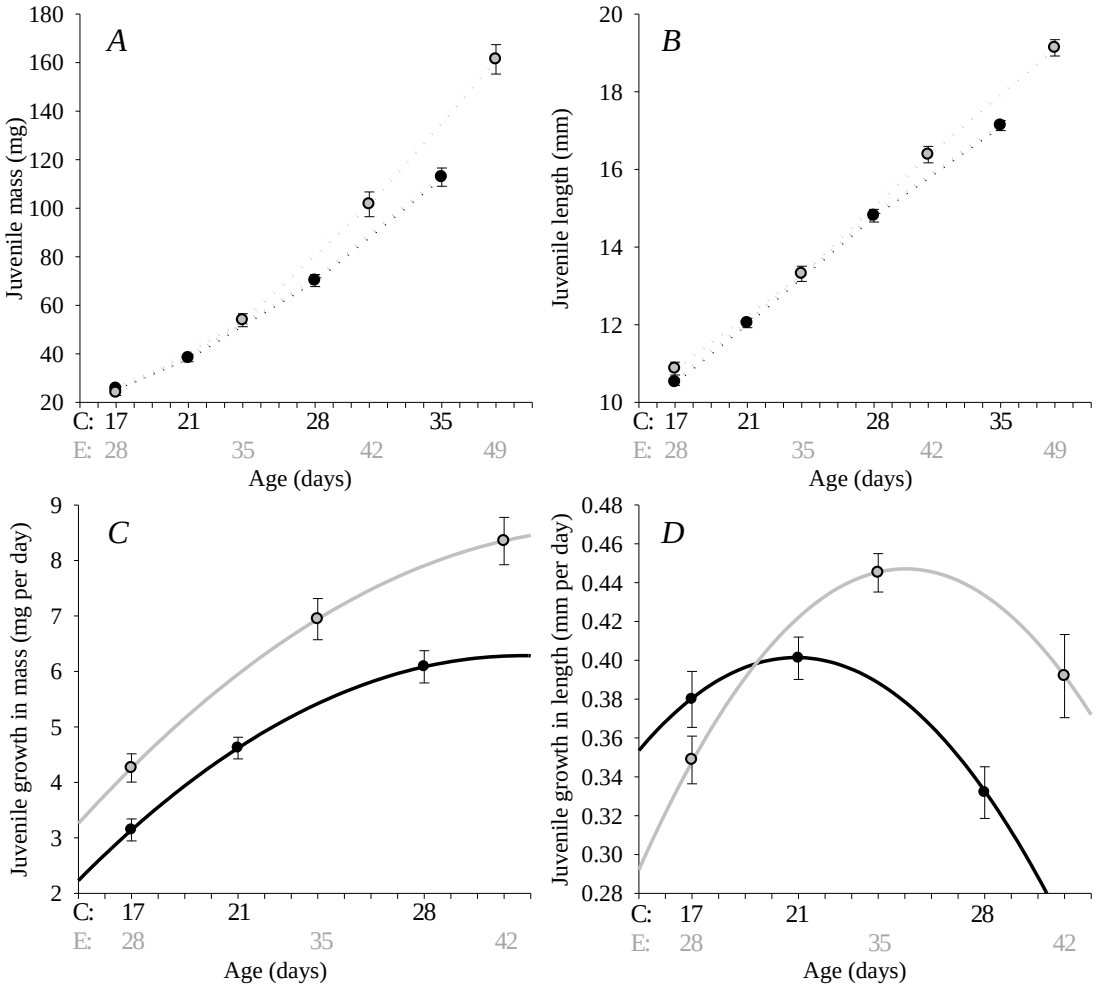

Figure 1.1. Trajectories of (*A*) juvenile body mass, (*B*) juvenile body length, (*C*) juvenile daily growth in mass, and (*D*) juvenile daily growth in length in experimental (grey) and control (black) fish. Trajectories of juvenile body size and juvenile growth are during the post-growth restriction period. For juvenile growth analyses, size and growth trajectories (for both mass and length) of experimental fish at 28 days of age (the end of the growth restriction period) were slid back in time to a common starting size with the control fish at 17 days of age. Estimates are presented as means ± 1SE; solid lines are predicted growth trajectories for each treatment.

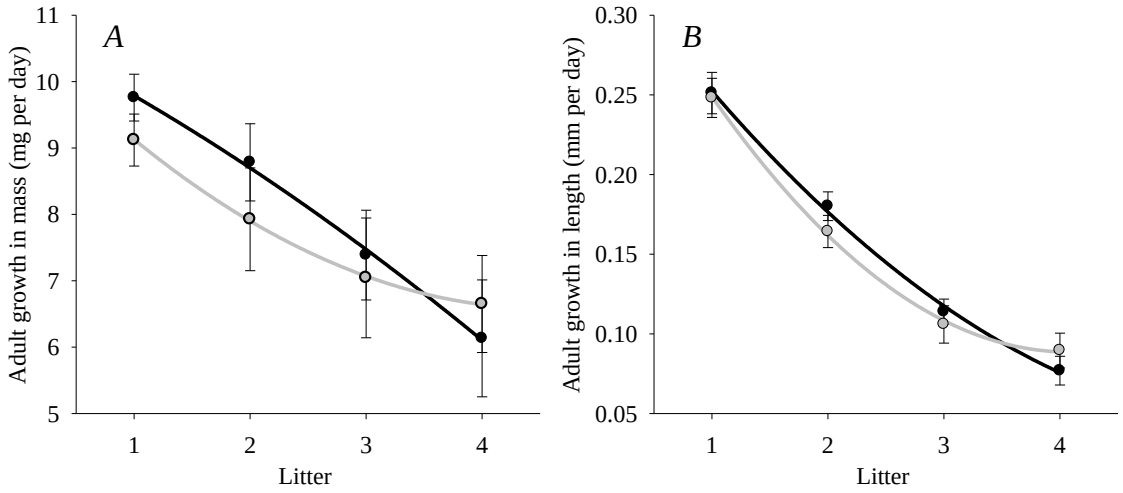

Figure 1.2. Trajectories of (*A*) adult daily growth in mass and (*B*) adult daily growth in length in experimental (grey) and control (black) fish. Estimates are presented as means ± 1SE; solid lines are predicted growth trajectories for each treatment.

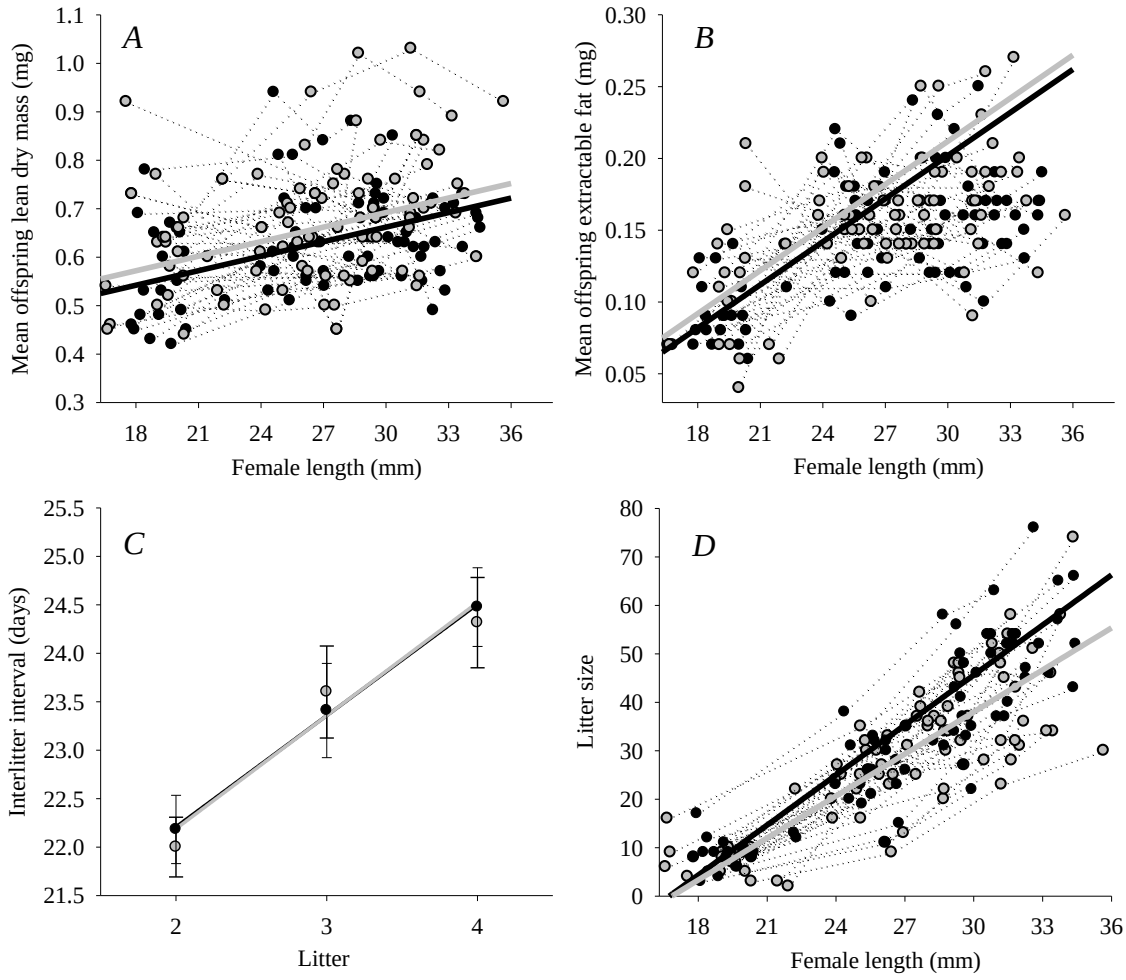

Figure 1.3. Trajectories of (*A*) mean offspring lean dry mass, (*B*) mean offspring extractable fat content, (*C*) interlitter interval, and (*D*) litter size for experimental (grey) and control (black) fish. For mean offspring lean dry mass, mean offspring extractable fat and litter size, dotted lines represent observed individual trajectories; solid lines represent predicted trajectories for each treatment. For interlitter interval, estimates are presented as means ± 1SE; solid lines are predicted trajectories for each treatment. Note that because estimated values for interlitter interval for experimental and control fish are nearly identical, only one trajectory is visible.

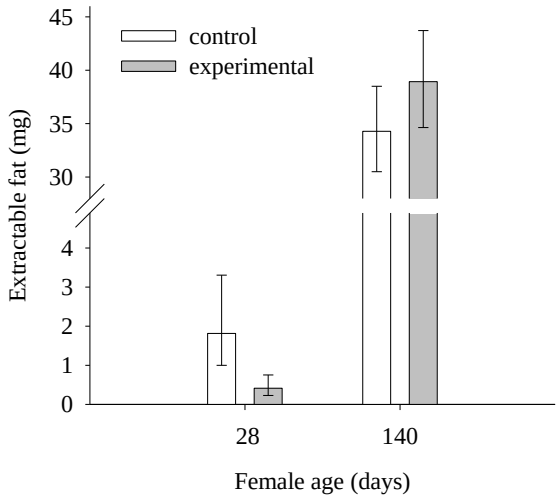

Figure 1.4. Extractable fat (mean ± 95% CI) for a subset of experimental and control females preserved at the end of the growth restriction period (age 28 days) and for experimental and control females preserved at the birth of their fourth litter (mean age = 140 days). Estimates of fat content are estimated marginal means after controlling for variation in lean dry body mass.

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Chapter 2

Phenotypic Plasticity in Adult Life History Strategies Compensates For a Poor Start in Life in Trinidadian Guppies (*Poecilia reticulata*)

Abstract

Low food availability during early growth and development can have long-term negative consequences for reproductive success. Phenotypic plasticity in adult life history decisions may help to mitigate these potential costs; yet adult life history responses to juvenile food conditions remain largely unexplored. I used a food manipulation experiment with female Trinidadian guppies (*Poecilia reticulata*) to examine age-related changes in adult life history responses to early food conditions, whether these responses varied across different adult food conditions, and how these responses affected overall reproductive success. Guppy females reared on low food as juveniles matured at a later age, a smaller size and with less energy reserves than females reared on high food as juveniles. In response to this setback, they changed their investment in growth, reproduction and fat storage throughout the adult stage such that they were able to catch up in body size, increase their reproductive output and restore their energy reserves to levels comparable to those of females reared on high food as juveniles. The net effect was that adult female guppies did not merely mitigate, but surprisingly were able to fully compensate for the potential long-term negative effects of poor juvenile food conditions on reproductive success.

Introduction

Organisms often live in environments where conditions can change within and across generations. Phenotypic responses to these changes in the environment can depend on the current physiological state of the organism as well as ambient external conditions (Houston and McNamara 1992; Schlichting and Pigliucci 1998). An organism's response to the current environment may also be influenced, to varying degrees, by its history via the effects of environments encountered during earlier stages of life and even in previous generations (Mousseau and Fox 1998; Schlichting and Pigliucci 1998; Ehrlen 2000; Taborsky 2006). How this dynamic interplay between genes and the environment molds different aspects of the phenotype throughout the lifetime and across generations can then have important consequences for individual fitness and population dynamics.

Food availability during early growth and development can have long-term effects on the adult phenotype and its subsequent performance (Lindström 1999; Monaghan 2008). Low food availability during the juvenile stage of life typically has a negative effect on future reproductive success (Lindström 1999; Lummaa and Clutton-Brock 2002). Long-term costs of low early food levels can arise when reduced rates of growth and development lead to maturation at a smaller size and/or at an older age; smaller size can reduced fitness because smaller adults generally suffer from decreased reproductive success and increased mortality while delayed maturation can reduce fitness because it increases generation time and can decrease the reproductive lifespan (Roff 2002). Low food availability during the juvenile stage of life can also have negative effects on energy reserves at maturation and susceptibility to starvation, disease and predation in the adult

stage of life (Lindström 1999; Morgan and Metcalfe 2001; Lummaa and Clutton-Brock 2002; McMillen and Robinson 2005).

During the juvenile stage, organisms exhibit a diverse array of phenotypic life history responses that can mitigate the negative long-term effects of poor early conditions (Metcalfe and Monaghan 2001). For example, juveniles of many organisms undergo compensatory growth when conditions improve after a period of growth restriction, whereby they are able to recoup their lost energy reserves and catch up in body size to the size they would have achieved under more favorable conditions (Metcalfe and Monaghan 2001). Organisms may also mitigate the effects of poor early conditions by delaying maturation until they reach a larger size (Roff 2002). While these responses during the juvenile stage can trade-off with other fitness components later in life (Johnsson and Bohlin 2006; De Block and Stoks 2008; Auer et al. 2010), they are thought to be adaptive strategies that make the best of a bad situation (Metcalfe and Monaghan 2001).

Phenotypic plasticity in life history decisions during the adult stage of life may also help to mitigate the negative effects of poor early conditions on overall reproductive success. Because resources are limited, organisms must 'decide' how to allocate resources to each of the different life history traits – growth, reproduction and survival- that ultimately determine their overall reproductive success (Roff 2002). Thus, when low food during the juvenile stage leads to fewer energy reserves, older age, and smaller body size at maturation, adults may be able to compensate for their poor early start through changes in how they invest in these different functions during the adult stage. For example, individuals reared as juveniles on low food may devote less to early

reproduction and growth and more to fat storage to recoup their reserves. They might compensate for delayed maturation by increasing their rate of reproduction or by investing more in each episode of reproduction. Organisms with indeterminate growth (i.e. those that continue to grow during the adult stage) may compensate for their small body size by first investing more in growth, catching up in body size, and then maintaining the same reproductive output as individuals reared under high food levels. These age-related changes in adult life history decisions have the potential to lessen the magnitude and eventually obliterate the effects of early food conditions on adult reproductive performance, but remain unexplored.

An individual's ability to mitigate the negative effects of poor early conditions may also depend on food conditions in the adult stage of life and whether conditions improve or remain constant across life stages. Empirical work has focused primarily on comparing adult survival and reproduction under high food conditions between individuals born under either good or poor food conditions (but see Taborsky 2006 for exception). For example, many epidemiological studies have taken advantage of data on disease prevalence and reproductive success in human cohorts that experienced famine or good conditions during the early stages of life and compared the fitness of these different cohorts under non-famine conditions (reviewed in Bateson 2001; Lummaa 2003; Lummaa and Tremblay 2003). Likewise, ecological studies that experimentally manipulate early food conditions tend to focus exclusively on reproductive success under good or *ad libitum* adult food conditions (e.g. De Block and Stoks 2005; Alonso-Alvarez et al. 2006; Blount et al. 2006; Barrett et al. 2009). These studies generally find that

individuals reared as juveniles on low food suffer from reduced reproductive success under high food adult conditions (but see Taborsky 2006; Painter et al. 2008 for exceptions). However, under low adult food levels, individuals reared as juveniles on low food may actually enjoy a reproductive advantage over individuals reared as juveniles under high food levels because of their smaller body size and lower maintenance costs (Monaghan 2008). Taborksy (2006) found that the long-term effects of rearing environment did not depend on the quality of the adult environment in a cichlid fish (*Simochromis pleurospilus*), but the generality of these long-term negative effects across different adult environments still needs to be investigated.

I examined adult life history responses to early food conditions and whether these responses changed with age and across different adult food conditions in female Trinidadian guppies (*Poecilia reticulata*). Guppies are small, livebearing poeciliid fishes that inhabit freshwater streams on the island of Trinidad, West Indies. Females used in the experiment were the offspring of first generation descendents of fish collected in 2008 from a downstream, high predation site on the Aripo River in the Northern Range Mountains. Food availability in this and other streams fluctuates on a seasonal basis through distinct alternating wet (low food) and dry (high food) seasons (Reznick 1989; Kohler 2010). Because guppies reproduce throughout the year, their lifetime can span either an entire wet or dry season or can span the transition between seasons such that food conditions may either remain constant or change from the juvenile to the adult stage of life. Guppies exhibit distinct phenotypic life history responses to changes in food availability in both the juvenile and the adult stages of life (Reznick 1989; Reznick and

Yang 1993; Bashey 2006). However, effects of juvenile food levels on adult life history decisions have not yet been examined.

I performed a food manipulation experiment with a 2 x 2 factorial design that had 2 juvenile and 2 adult food levels. Female guppies were raised on either low (L) or high (H) food until they matured. At sexual maturation, half of the females from each juvenile food level were switched to the opposite adult food level (L or H) while the other half remained on the same ration trajectory received during the juvenile stage (L or H). I measured adult responses in somatic growth, reproductive rate, reproductive investment, fecundity, offspring size and female body condition to juvenile growth history, how these responses changed with age and how they affected overall reproductive success under low and high adult food conditions.

Methods

Rearing of first generation fish

Twenty females were collected from the wild, brought into the laboratory and housed in individual 8 L aquaria until they gave birth (Reznick 1982). Litters of first generation (F1) offspring from each of the 15 wild-caught females that gave birth were collected when born and housed communally in larger 19 L aquaria. Offspring from each litter were sexed once they reached 28 days of age, females being distinguished from males by the triangular pattern of melanophore development in their abdominal region (Reznick 1990), and separated by sex into their own communal tanks. When offspring reached 35 days old, a week before the earliest recorded age at maturation in guppies (Reznick 1980), one F1 female was randomly chosen from each litter and housed

individually with one randomly selected unrelated F1 male. The 15 F1 male-female pairs were kept in the laboratory until females reached a size large enough to produce litters containing more than 20 offspring (approximately 6 months).

Parental as well as F1 fish were fed a diet of liver paste in the morning and *Artemia* brine shrimp nauplii in the evening. Rations for female F1 fish started at 10 μL per day and were increased by 20 μL every two weeks to adjust for increases in body size. Since male guppies stop growing once they reach maturity, their rations were increased only up until 20 μL per day.

Study Design

Twenty F2 offspring from a litter from each of the 15 pairs were separated at birth and housed in separate aquaria. F2 siblings were housed next to one another in blocks in the laboratory. One half of the litter was randomly assigned to a low food (L) ration, while the other half received a high food (H) ration. Offspring were sexed once they reached 28 days of age, as done for F1 offspring. Three females and 1 male from each food level from each litter that were similar in body mass (L food: $F_{3,56} = 0.61$, $p = 0.61$; H food: $F_{3,56} = 0.72$, $p = 0.55$) and length (L food: $F_{3,56} = 1.45$, $p = 0.24$; H food: $F_{3,56} = 1.73$, $p = 0.17$) were then randomly selected for the experiment. At maturation, one female from each ration level was randomly chosen from each litter, measured, euthanized with an overdose of MS-222, and preserved in formalin to estimate body condition at maturation (see below). Of the two remaining female siblings for each juvenile food level, one was switched to the other ration level and the second was kept on the same ration level for a full factorial design of two ration levels across two life stages:

LL, LH, HH, and HL. Female siblings were kept on that adult food ration until they gave birth to three litters.

To estimate age at maturation and thereby the timing of switches in food levels, I used the observable sexual development of each male sibling as an index of his sister's maturation. Female guppies give birth to live young, so maturity is not apparent until first parturition. However, age at maturation is best approximated by male age at maturation; male age at maturity is roughly equal to female age at first parturition (± 1-2 days) minus one gestation period or interlitter interval, the number of days between litters (Reznick 1982). Sexual development of a male sibling under his respective juvenile food levels, measured by the developmental stage of his intromittent organ – the gonapodium, was therefore used as an index of the timing of his sister's maturation (Reznick 1990).

Fish were fed a diet of liver paste in the morning and *Artemia* brine shrimp nauplii in the evening. Food levels were increased every two weeks. In the low food treatment, total daily food levels for the first three 2-week periods were 2, 4, and 7.5 μL and then increased by 5 μL each two week period thereafter. Fish in the high food treatment always received twice the amount allocated to fish in the low food treatment. These low and high food level trajectories were set according to rations that have been predetermined to lead to growth rates and female body size at maturation that fall within the range observed across guppy populations in the wild; growth rates under these rations range from 0.5-2.25 mm per two week period in the laboratory versus 0.25-2.25 mm per two week period in the wild (Grether et al. 2001; Reznick et al. 2001), while female body size at maturation on these same rations ranges from 13-17 mm in the laboratory and

from 12-17 mm in the wild (Reznick and Endler 1982). Tanks were checked for uneaten food after each feeding, but no incidences of uneaten food were observed.

Standard length (SL) of each fish was measured every two weeks from the time it was born until the end of the experiment. Body mass was measured starting at age 15 days. Each fish was anesthetized in a solution of neutrally buffered MS-222 and measured under a dissecting scope using digital calipers and then weighed using an electronic balance. Females were mated overnight each week with a male randomly selected from a stock tank of unrelated stud males once they reached 35 days of age and were approaching maturity (Reznick 1980). Aquaria were then checked twice daily for newborn offspring. Offspring were collected, euthanized with an overdose of MS-222 and preserved in 5% formalin on the day they were born. Female SL was also measured on the day each litter was born. Females were measured and weighed, euthanized and then preserved, as done for offspring, on the day they gave birth to their third litter for analyses of fat reserves.

Adult growth rate, reproductive rate, litter dry weight, fecundity, and offspring size were determined for the first three litters. Adult growth rate was calculated as $[(SL_{\text{litter } i} - SL_{\text{litter } i\text{-}1}) / (\text{the number of days between each litter})]$, where *i* is the litter number. Interlitter interval represents the rate of reproduction and was calculated as the number of days between consecutive litters. Because age at maturation was estimated, only intervals between the births of the first, second and third litters were calculated. Litter dry weight, measured after drying each litter overnight in an oven at 60 deg C, provided an index of total investment per litter. Offspring standard length was measured

in 3-5 offspring randomly chosen from each litter. To estimate changes in adult body condition between maturation and the birth of the third litter, females from the subset preserved at maturation and those preserved at the birth of their third litter were dried overnight in an oven at 60 deg C and then submerged in anhydrous ether to remove triglycerides until they reached a constant lean dry weight (Reznick and Endler 1982). Extractable fat content per female was then calculated as the difference between the dry and lean dry weight.

Statistical Analyses

I used a linear mixed model approach to examine adult life history responses to juvenile growth history and how these responses differed with age and the quality of the adult environment. For analyses where the dependent variable was measured only once on each individual, block was included as a random effect to control for variation in pedigree and microenvironment in the laboratory. For analyses of longitudinal data, female identity was included as a random effect to control for the non-independence of repeated measures on the same female (Singer and Willett 2003; Bolker et al. 2009). For each trait, I began with a full model and used backward model selection, sequentially eliminating terms with the lowest F-values until all terms in the model were significant. For analyses of mean effects across the three litters, the full model contained all main effects and their interactions. For analyses of age-trajectories, the full model for each trait included all main effects, covariates and all possible 2 and 3-way interactions between age and stage-specific food levels.

For the timing of first reproduction, I tested for effects of juvenile food level on the age and size at maturation, as estimated by male sexual development. However, because female age at first parturition is a more direct measure of maturation, I also compared age and size at first parturition between juvenile food treatments. For this latter analysis, I compared LL and HH females but excluded HL and LH females because of confounding effects of their adult food levels.

For adult growth and reproduction, I first tested for treatment differences in the intercept and age trajectory of each trait. I examined models that included and then models that excluded female SL as a covariate. Age was included as a time-varying covariate to account for temporal changes in reproductive traits and to control for age-specific changes in food rations. Because females differed in their reproductive schedules but food levels were dependent on age, all analyses were centered on the grand mean age (57 days) and grand mean SL (15.3 mm) at the yolking of the first litter. When the effect of juvenile or adult food levels on a reproductive trait changed with age, I re-centered the intercept in the final model on the grand mean age and grand mean female SL at the yolking of both the second (age: 84 days, SL: 18.9 mm) and third litter (age: 108 days, SL: 21.6 mm) and tested for treatment differences in that trait at those two litters as well (sensu Singer and Willett 2003; West et al. 2006). I then tested for overall effects of juvenile and adult food levels on the mean interlitter interval, mean adult growth rate, total litter dry weight, total fecundity, and mean offspring size across all three litters. If there was a significant interaction between effects of juvenile and adult levels on a specific trait, I examined differences in that trait value between LL and HL females and

between LH and HH females. Finally, because both the timing of maturation and the rate of reproduction can impact overall reproductive success, I tested for effects of juvenile and adult food levels on overall reproductive success, in terms of total litter dry weight and total fecundity across all litters, by controlling for the number of days it took a female to produce all three litters (i.e. her age at the birth of the third litter).

The effects of juvenile and adult food conditions were examined in 60 females, 15 females per treatment. One HH female never reproduced, so reproductive estimates are from only 59 individuals. Five individuals (1 LH, 1 LL, and 3 HL females) did not produce their third litter due to premature termination of the experiment, so they were omitted from analyses of total litter dry weight and total number of offspring produced across all three litters. In all analyses, I ran diagnostic analyses to ensure the functional form of the model and to check that residuals were normally distributed. With the exception of fecundity, all traits were normally distributed. For fecundity, I specified a quasi-Poisson distribution because the data were counts wherein the variance was greater than the mean. Main effects and interactions were regarded as significant when $P < 0.05$. All models were run using SAS version 9.3 (SAS Institute, Cary, NC). Estimates for each trait are given as least square means ± 1SE.

Results

Juvenile Growth Rates

Standard length of newborn females at age 1 day was 6.54 ± 0.03 mm and did not differ between low and high food treatments (juvenile food level: Juv: $F_{1,14} = 0.16$, $p = 0.69$; block: Wald $Z = 1.94$, $p = 0.02$). Mean daily growth was slower in females reared on low

food as juveniles (Ljuv) relative to females reared on high food (Hjuv) between the time they were born and the time they matured when accounting for variation in their SL at age 1 day (0.18 ± 0.004 vs. 0.27 ± 0.004 mm per day, respectively; Juv: F1,14 = 480.3, p < 0.01, initial SL: F1,42 = 9.64, p < 0.01; block: Wald Z = 2.14, p = 0.01; Figure 2.1 A).

Timing of Reproduction

Sexual maturation occurred at a later age in Ljuv females relative to Hjuv females (64.9 ± 1.0 vs. 50.1 ± 1.1 days, respectively; Juv: $F_{1,13}$ = 148.18, p < 0.01; block: Wald Z = 1.31, p = 0.09). L_{juv} females matured at a somewhat smaller SL and body mass relative to H_{juv} females (SL: 15.0 ± 0.2 vs. 15.5 ± 0.2 mm, respectively; body mass: 74.4 ± 3.5 vs. 81.8 ± 3.6 mg, respectively); treatment differences in SL (Juv: $F_{1,13}$ = 3.55, p = 0.08; block: Wald Z = 1.19, p = 0.11) and body mass (Juv: $F_{1,13}$ = 3.68, p = 0.07; block: Wald Z = 1.53, p = 0.06) were not statistically significant but did have an important effect on fecundity in the first litter (see fecundity analyses below). These differences in age and size at maturity between juvenile treatment groups were also evident at first parturition; first parturition occurred roughly two weeks later in L_{juv} females relative to H_{juv} females (92.2 ± 2.0 vs. 78.9 ± 2.1 days, respectively; Juv: $F_{1,13}$ = 27.8, p < 0.01; block: Wald Z = 0.99, p = 0.16). First parturition in L_{juv} females also occurred at a smaller SL (Juv: $F_{1,13}$ = 36.2, p < 0.01; block: Wald Z = 1.37, p = 0.09) and smaller body mass (Juv: $F_{1,13}$ = 21.0, p < 0.01; block: Wald Z = 0.78, p = 0.21) relative to H_{juv} females (SL: 18.2 ± 0.2 vs. 19.7 ± 0.2 mm, respectively; body mass: 131.5 ± 6.5 vs. 169.5 ± 6.7 mg, respectively).

Interlitter interval changed with female age and was affected by both adult and juvenile food levels (Table 1; Figure 2.2 *A,B*). Interlitter interval was longer in females in

the low food adult environment (L_{Ad}) relative to females in the high adult food environment (H_{Ad}) for both the second and third litter (Table 2.1; Figure 2.2 *A*,*B*). There was a significant effect of juvenile food level on interlitter interval and how it changed with age (Table 2.1; Figure 2.2 *A*,*B*). Interlitter interval was shorter in L_{Juv} relative to H_{Juv} females for the second litter (Table 2.1), but was similar between L_{Juv} and H_{Juv} females for the third litter (t = -0.21, p = 0.83). Female SL had no effect on interlitter interval so it was not included in the model. Overall, juvenile food level had no effect, while an increase in adult food level led to a shorter mean interlitter interval across the last two litters (Ad: $F_{1,14}$ = 22.12, p < 0.01; block: Wald Z = 1.52, p = 0.06; Figure 2.2 *C*).

Adult Growth Rates

Juvenile and adult food levels both had a significant effect on adult growth rates and how they changed with age (Table 2.1, Figure 2.1 *A*, Figure 2.2 *D*,*E*). When female SL was included as a covariate in the model, daily growth was slower in L_{Ad} relative to H_{Ad} females across all three litters (Table 2.1). In contrast, daily growth was faster in L_{Juv} relative to H_{Juv} females leading up to the birth of the first (Table 2.1) and second (t = 2.54, p = 0.01) litters but then similar among L_{Juv} and H_{Juv} females leading up to the birth of the third litter (t = 1.69, p = 0.09). When female SL was excluded from the model, juvenile and adult food levels both had a significant effect on adult growth and how it changed with age (Table 2.1, Figure 2.2 *D*,*E*). Daily growth was slower in L_{Ad} relative to H_{Ad} females leading up to the birth of the first (Table 2.1), second (t = -13.01, p < 0.01) and third (t = -4.94, p < 0.01) litters. In contrast, daily growth was faster in L_{Juv} relative to H_{Juv} females leading up to the birth of the first (Table 2.1), second (t = 9.16, p < 0.01) and

third (t = 2.65, p = 0.01) litters. In addition, there was a significant interaction between juvenile and adult food levels; the difference in growth rate between LL and HL females was less than the difference between LH and HH females across all litters (Table 2.1, Figure 2.2 *D*,*E*).

Overall, juvenile food level had a negative effect ($F_{1,14}$ = 72.59, p < 0.01), adult food had a positive effect ($F_{1,14}$ = 294.52, p < 0.01), and there was a significant interaction between the effects of juvenile and adult food levels on the mean daily adult growth rate across all three litters (Juv by Ad (adult): $F_{1,10}$ = 5.75, p = 0.04; block: Wald Z = 1.79, p = 0.03; Figure 2.2 *F*); the difference in mean growth rate between LL and HL females (9.9 ± 0.3 vs. 8.3 ± 0.2 mm x 10^2 per day, respectively) was less than the difference between LH and HH females (15.1 ± 0.3 vs. 12.2 ± 0.3 mm x 10^2 per day, respectively).

Higher growth rates in L_{juv} females eventually led to a convergence in body size between LL and HL fish and between LH and HH fish (Figure 2.1 *A*). At age 57 days, LL fish were significantly smaller than HL fish (14.1 ± 0.1 vs. 16.1 ± 0.1 mm, respectively; t = 13.40, p < 0.01), and LH fish were significantly smaller than HH fish (14.3 ± 0.1 vs. 16.5 ± 0.1 mm, respectively; t = 15.09, p < 0.01). By age 113 days and at the mean age at which LL females yolked their last litter (113.3 ± 1.3 days), LL had caught up in SL to HL fish (t = 1.62, p = 0.11). By 127 days and approximately 3 weeks after the mean age at which they yolked their last litter (108.9 ± 1.7 days), LH fish had caught up in SL to HH fish (t = 0.76, p = 0.45).

Adult Body Condition

L_{Juv} females had significantly less body fat at sexual maturation than H_{juv} females (Juv: $F_{1,10} = 6.10$, p = 0.03; lean dry body mass: $F_{1,10} = 15.44$, p = 0.03; block: Wald Z = 1.47, p = 0.07; see Figure 2.1 *B* for comparison of percent body fat). However, by the birth of the third litter, there was no effect of juvenile or adult food levels on fat content when accounting for differences in lean body mass (Juv: $F_{1,14} = 0.19$, p = 0.67; Ad: $F_{1,14}$ = 0.50, p = 0.48; lean dry body mass: $F_{1,41} = 18.89$, p < 0.01; block: Wald $Z = 0.32$, p = 0.37; see Figure 2.1 *B* for comparisons of percent body fat).

Reproductive Investment

Juvenile and adult food levels both had significant effects on litter dry weight and how it changed with age (Table 2.2, Figure 2.3 *A,B*). When female SL was included as a covariate in the model, litter dry weight was similar among L_{Ad} and H_{Ad} females for the first litter (Table 2.2), but was lower in L_{Ad} females relative to H_{Ad} females in the second (t = -3.12, p < 0.01) and third (t = -0.63, p < 0.01) litters. In contrast, litter dry weight was similar among L_{Juv} and H_{Juv} females for the first litter (Table 2.2) but was greater in L_{Juv} females relative to H_{Juv} females in the second (t = 2.24, p = 0.02) and the third (t = 4.04, p < 0.01) litters. When female SL was excluded from the model, juvenile and adult food levels both had significant effects on litter dry weight and how it changed with age (Table 2.2, Figure 2.3 *A,B*). Litter dry weight was similar among L_{Ad} and H_{Ad} females for the first litter (Table 2.2), but was lower in L_{Ad} females relative to H_{Ad} females in the second (t = -8.72, p < 0.01) and third (t = -9.98, p < 0.01) litters. In contrast, litter dry weight was lower for the first litter (Table 2.2), similar in the second litter (t = 1.77, p = 0.08), and

higher in the third litter (t = 2.06, p = 0.04) in L_{Juv} relative to H_{Juv} females. Overall, juvenile food level had a negative effect ($F_{1,14}$ = 4.82, p = 0.04), while adult food level had a positive effect ($F_{1,14}$ = 73.78, p < 0.01; block: Wald Z = 0.57, p = 0.28) on the total dry weight of all three litters (Figure 2.3 *C*).

Juvenile and adult food levels both had significant effects on fecundity and how it changed with age (Table 2.3, Figure 2.3 *D,E*). When female SL was included as a covariate in the model, fecundity was similar among L_{Ad} and H_{Ad} females for the first litter (Table 2.3), but was lower in L_{Ad} females relative to H_{Ad} females in the second (t = -3.20, p < 0.01) and third (t = -3.64, p < 0.01) litters. In contrast, fecundity was similar among L_{Juv} and H_{Juv} females for the first litter (Table 2.3), but was greater in L_{juv} females relative to H_{Juv} females in the second (t = 1.97, p = 0.05) and third litters (t = 3.96, p < 0.01). When female SL was excluded from the model, juvenile and adult food levels both had significant effects on fecundity and how it changed with age (Table 2.3, Figure 2.3 *D,E*). Fecundity was increasingly lower in L_{Ad} females relative to H_{Ad} females in the first (Table 2.3), second (t = -8.31, p < 0.01) and third (t = -11.53, p < 0.01) litters. In contrast, fecundity was lower for the first (Table 2.3), similar in the second (t = 1.34, p = 0.19), but greater in the third (t = 2.43, p = 0.01) litter in L_{Juv} relative to H_{Juv} females. In addition, there was a significant interaction between juvenile and adult food levels on fecundity; the difference in fecundity between LL and HL females was less during the first and second litter but greater in the third litter relative to the difference between LH and HH females across those litters (Figure 2.3 *D,E*). Overall, juvenile food level had a negative effect ($F_{1,14}$ = 5.36, p = 0.03) while adult food level had a positive effect (Ad: $F_{1,14}$ =

76.00, $p < 0.01$; block: Wald $Z = 1.30$, $p = 0.09$) on total fecundity across all three litters, L_{Juv} producing roughly 2 more offspring than H_{Juv} females (Figure 2.3 *F*).

Offspring SL increased with age, but was not affected by adult food levels (Table 2.3, Figure 2.3 *G*,*H*). Juvenile food level did have an effect on the age trajectory of offspring SL (Table 2.3), but because this effect was so slight, there was no difference in offspring SL between L_{Juv} and H_{Juv} females at the first (Table 2.3), second ($t = -0.17$, $p = 0.87$) or third ($t = -1.32$, $p = 0.19$) litter. Female SL had no effect on offspring SL so it was not included in the model. Overall, juvenile and adult food levels had no effect on the mean offspring SL across all three litters (Juv: $F_{1,14} = 0.60$, $p = 0.45$; Ad: $F_{1,14} = 0.03$, $p = 0.87$; block: Wald $Z = 1.88$, $p = 0.03$; Figure 2.3 *I*).

Overall reproductive success

When the number of days taken to produce all three litters was accounted for (female age at birth of third litter), only adult food levels had a significant effect on total dry weight invested in all three litters (Juv: $F_{1,14} = 0.03$, $p = .86$; Ad: $F_{1,14} = 103.07$, $p < 0.01$; age at third litter: $F_{1,35} = 11.88$, $p < 0.01$; block: Wald $Z = 0.98$, $p = 0.16$). Similarly, only adult food level had a significant effect on the total number of offspring produced (Juv: $F_{1,14} = 0.57$, $p = 0.46$; Ad: $F_{1,14} = 78.04$, $p < 0.01$; age at third litter: $F_{1,35} = 3.60$, $p = 0.06$; block: Wald $Z = 1.15$, $p = 0.12$).

Discussion

Environmental conditions during the juvenile stage can potentially have long-term negative consequences for reproductive performance through their effects on age, size and energy reserves at maturation. Here I show for the first time that phenotypic

plasticity in age-related adult life history decisions can not only mitigate, but, surprisingly, can fully compensate for setbacks caused by poor early food conditions and regardless of the quality of the adult environment. L_{Juv} females matured at a later age, a smaller size and with lower fat reserves relative to H_{Juv} females. In response to their juvenile history, they changed how they invested in growth, reproduction and energy storage throughout the adult stage. In both low and high food adult environments, L_{Juv} females first invested more in growth and less in reproduction relative to H_{Juv} females. Next, they continued to grow at a faster rate but also invested the same in reproduction as H_{Juv} females. They also produced the second litter at a faster rate relative to H_{Juv} females. Towards the end of the experiment, they then invested the same in growth and reproduced at the same rate, but invested more in reproduction relative to H_{Juv} females. A faster rate of increase in body size in L_{Juv} females facilitated this concomitant increase in their litter dry weight and fecundity. However, L_{Juv} females also compensated for their smaller size in later litters by increasing their size-specific investment in reproduction and producing litters with a higher dry weight and a higher number of offspring than H_{Juv} females. By the time they gave birth to their third litter, they had also restored their fat reserves to the same levels found in H_{Juv} females. These age-related changes in life history decisions effectively allowed them to catch-up in body size to H_{Juv} females, to compensate for their delayed maturation, and to recoup their energy reserves in each adult environment. The overall effect was that L_{Juv} females produced a slightly higher total litter dry weight and greater total number of offspring across the three litters but

achieved the same reproductive success as H_{Juv} females when accounting for the time it took to produce all three litters.

Overall, L_{Juv} females grew at faster rates, but they also had a higher total litter dry weight and produced a greater number of offspring across the three litters than H_{Juv} females. This higher total investment was accomplished despite having lower fat reserves at maturation and without any compromise to their fat levels at the end of the experiment. How were L_{Juv} females able to invest more in growth and reproduction relative to H_{Juv} females? One potential reason is the difference in metabolic needs associated with differences in body size between females reared on low versus high food levels as juveniles. In each adult environment, faster growth in L_{Juv} females eventually led to convergence in body size with H_{Juv} females, but this occurred only after the yolking of the third litter. Thus, L_{Juv} females were always smaller for their age and therefore may have had more resources left over after maintenance to devote to growth and reproduction (Monaghan 2008). Alternatively, treatment differences in growth and reproduction may have stemmed from differences in how L_{Juv} and H_{Juv} females invested in other processes such as immune function and the repair of oxidative damage that may enhance future survival; L_{Juv} females may have prioritized growth and reproduction at the expense of survival relative to H_{Juv} females (see further discussion below).

Females with a shared juvenile history exhibited strikingly similar plastic responses in growth and reproduction across low and high food adult environments. Moreover, age trajectories of three traits – interlitter interval, offspring SL, and size-specific growth – did not depend on adult environment at all but varied only in response

to juvenile food level. These patterns suggest that individuals with a shared juvenile history are adhering to a common resource allocation rule dictated by their similar internal state – age, body size and energy reserves – at maturation (McNamara and Houston 1996; Jørgensen and Fiksen 2006). This common life history response among L_{Juv} females allowed them to fully compensate for their poor start in life in both adult environments and therefore regardless of whether conditions improved or remained constant across life stages. However, both mortality risk and the timing of reproduction, in addition to reproductive investment, also influence fitness, so the adaptive value of these responses is not yet clear.

Compensatory responses during the adult stage may have detrimental effects on survival by increasing intrinsic mortality. While no deaths occurred during the production of the first three litters in this study, it is feasible that compensation during the adult stage may have delayed effects on intrinsic mortality. Compensatory responses during the juvenile stage are known to heighten the risk of intrinsic mortality later in life (Metcalfe and Monaghan 2001; Forsen et al. 2004), but the fitness consequences of compensation during the adult stage are not yet clear. Higher growth and reproduction in L_{Juv} relative to H_{Juv} females may come at a cost to future survival. This trade-off could be mediated simply by changes in energy allocation, energy intended for future survival being diverted instead towards growth and reproduction (Roff 2002). Negative effects on survival may also be compounded by the effects of increased growth and reproduction on oxidative stress (Alonso-Alvarez et al. 2004; Costantini 2008; Monaghan et al. 2009; Dmitriew 2010). Increased somatic growth and reproduction, through their positive

effects on metabolic rate, may increase the production of reactive oxygen species (ROS) (Alonso-Alvarez et al. 2004; Monaghan et al. 2009; Dmitriew 2010). ROS are a normal byproduct of aerobic metabolism but can damage cellular components such as DNA, lipids and proteins (Finkel and Holbrook 2000). Imbalances in the production of ROS and the antioxidant defense and repair systems needed to combat their negative effects can lead to oxidative stress and may thereby have detrimental effects on survival and longevity (Monaghan and Haussmann 2006; Costantini 2008; De Block and Stoks 2008; Monaghan et al. 2009; Nussey et al. 2009). However, it is unclear how effects of growth rate on the production of ROS and their counteracting antioxidant defense and repair mechanisms during both the juvenile and adult stage may interact to influence oxidative stress and survival during the adult stage. L_{Juv} females invested more in growth and reproduction during the adult stage, but they also grew less during the juvenile stage relative to H_{Juv} females. Slower growth during the juvenile stage should therefore demand less investment in cellular repair or antioxidant defense systems, but whether faster growth during the juvenile stage incurs less, more or comparable damage to faster growth and increased reproduction in the adult stage remains unexplored.

Extrinsic sources of mortality are also likely to moderate the ability of individuals to compensate for a poor start in life in the wild. Because predation-driven mortality in guppies is known to be size-selective, smaller individuals suffer from increased vulnerability to predation risk relative to larger individuals and may therefore not live long enough to compensate for their poor start to life (Reznick et al. 1996). In addition, L_{Juv} females matured at a significantly later age and at the time when H_{Juv} females were

roughly half way through the production of their first litter. Increased investment in reproduction by L_{Juv} females may compensate somewhat for their delayed reproduction, but predator-induced selective pressures are known to put a premium on early reproduction in wild guppies (Bronikowski et al. 2002). When these differences in the timing of reproduction are integrated with the risk of mortality, females that experience low food as juveniles in nature may well have substantially lower reproductive success than those that experienced high food levels as juveniles.

Finally, age-related changes in the effects of juvenile rearing conditions on adult reproductive traits may have important consequences for population dynamics. Delayed effects of rearing conditions are known to increase heterogeneity in reproductive performance among individuals or cohorts in a population and thereby influence population dynamics (Sæther 1997; Beckerman et al. 2002; Benton et al. 2006). Lindström and Kokko (2002) show theoretically that cohort effects can have either a stabilizing or destabilizing effect on population dynamics, depending on whether the dynamics of the population are inherently unstable or stable, respectively. Their model assumes that early conditions have a permanent impact on the quality of the individual. Here I show that this assumption may not always be met; low juvenile food conditions had a negative effect on reproduction in the first litter, no effect on reproduction in the second litter and a positive effect on reproduction in the third litter in guppies. This change in the magnitude of early environmental effects may impact population dynamics, but has not yet been examined.

Understanding the long-term effects of early environmental conditions is of primary importance to evolutionary biologists studying the adaptive value of phenotypic responses to environmental change (Monaghan 2008), to ecologists investigating the effects of environmental variation on population dynamics (Lindström and Kokko 2002), and to biomedical researchers interested in the developmental origins of human disease (Lummaa and Clutton-Brock 2002; Bateson et al. 2004). Early environments, through their effects on growth and development, can potentially limit adult reproductive output and lifespan. Yet, life history trajectories, as demonstrated here, are not just passive consequences of the constraints imposed by early conditions; individuals can respond to variation in the environment through flexibility in growth, reproduction and energy storage. Adult responses to poor early conditions may therefore help organisms mitigate potential long-term costs to reproduction and survival, but their underlying mechanisms and adaptive value require further attention.

Table 2.1. Final linear mixed models for interlitter interval and adult growth as a function of female age, juvenile (Juv) and adult (Ad) food levels.

	Interlitter interval (days)		Adult growth (mm per day x 10^2)			
			Size-specific model		Absolute model	
Fixed effects	Estimate (SE)	*t*	Estimate (SE)	*t*	Estimate (SE)	*t*
Intercept	23.88 (.29)	80.83**	16.0 (.4)	33.30**	14.1 (.4)	35.15**
Age	.01 (.01)	1.13	0.1 (.01)	5.14**	-.1 (.01)	-8.74**
SL	n/a		-1.4 (.2)	-8.99**	n/a	
Juv	-.81 (.38)	-2.16*	2.2 (.6)	3.60**	5.8 (.6)	10.03**
Ad	1.16 (.33)	3.47**	-6.5 (.5)	-13.60**	-5.6 (.5)	-10.30**
Age x Juv	.03 (.01)	2.06*	-.04 (.01)	-3.13**	-.1 (.01)	-5.62**
Age x Ad	n/s		n/s		.1 (.01)	6.10**
Juv x Ad	n/s		n/s		-1.6 (.7)	-2.40*
Random effects	Estimate (SE)	Wald *Z*	Estimate (SE)	Wald *Z*	Estimate (SE)	Wald *Z*
Female	1.06 (.31)	3.45**	1.8 (.6)	3.15**	.5 (.3)	1.47
Residual	.87 (.17)	5.08**	2.2 (.3)	6.96**	3.1 (.4)	7.37**

Note: Adult growth models include female standard length (SL) as a covariate (size-specific model) and exclude female SL as a covariate (absolute model). Juvenile food levels are low (= 1) and high (= 0). Adult food levels are low (= 1) and high (= 0). For interlitter interval, parameter estimates are centered on the grand mean age of the yolking of the second litter (84 days). For adult growth, parameter estimates are centered on the grand mean age (57 days) and the grand mean female SL (15.31 mm) at the yolking of the first litter. * $P < 0.05$; ** $P < 0.01$, $^{n/s}P > 0.05$; $^{n/a}$ term not included in model.

Table 2.2 Final linear mixed models for litter dry weight as a function of female age, juvenile (Juv) and adult (Ad) food levels.

	Litter dry weight (mg)			
	Size-specific model		Absolute model	
Fixed Effects	Estimate (SE)	*t*	Estimate (SE)	*t*
Intercept	2.05 (.32)	6.38**	3.06 (.31)	10.07**
Age	.01 (.02)	.50	0.13 (.01)	16.83**
SL	.89 (.15)	5.95**	n/a	
Juv	.02 (.46)	.04	-1.84 (.38)	-4.84**
Ad	-.15 (.33)	-.45	-.37 (.37)	-1.01
Age x Juv	.03 (.01)	2.86**	.05 (.01)	5.40**
Age x Ad	-.04 (.01)	-3.10**	-.08 (.01)	-8.75**
Random Effects	Estimate (SE)	Wald *Z*	Estimate (SE)	Wald *Z*
Female	.41 (.19)	2.18*	.61 (.24)	2.54**
Residual	1.42 (.19)	7.29**	1.65 (.23)	7.32**

Note: Female standard length (SL) is included as a covariate (size-specific model) and is excluded as a covariate (absolute model). Juvenile food levels are low (= 1) and high (= 0). Adult food levels are low (= 1) and high (= 0). Parameter estimates are centered on the grand mean age (57 days) and the grand mean female SL (15.31 mm) at the yolking of the first litter. * $P < 0.05$; ** $P < 0.01$, $^{n/s}P > 0.05$; $^{n/a}$ term not included in model.

Table 2.3. Final linear mixed models for fecundity per litter and offspring standard length (SL) as a function of female age, juvenile (Juv) and adult (Ad) food levels.

	Fecundity per litter				Offspring SL (mm)	
	Size-specific model		Absolute model		Absolute model	
Fixed Effects	Estimate (SE)	*t*	Estimate (SE)	*t*	Estimate (SE)	*t*
Intercept	3.07 (.42)	7.27**	4.27 (.32)	13.54**	6.18 (.05)	151.5**
Age	.02 (.02)	.66	.15 (.01)	15.35**	.01 (<.01)	7.29**
SL	.94 (.19)	4.82**	n/a		n/a	
Juv	-.16 (.61)	-.28	-2.76 (.47)	-5.88**	.07 (.06)	1.08
Ad	-.25 (.44)	-.58	-.92 (.42)	-2.15*	n/s	
Age x Juv	.04 (.01)	2.94**	.06 (.01)	5.47**	-.003 (.001)	-2.30*
Age x Ad	-.05 (.01)	- 3.00**	-.10 (.01)	-9.02**	n/s	
Juv x Ad[†]	n/s		1.23 (.59)	2.07*	n/s	
Random Effects	Estimate (SE)	Wald *Z*	Estimate (SE)	Wald *Z*	Estimate (SE)	Wald *Z*
Female	.62 (.33)	1.94*	.88 (.38)	2.30*	.03 (.01)	3.88**
Residual	2.56 (.35)	7.28**	2.80 (.38)	7.33**	.03 (<.01)	7.43**

Note: Female SL is included as a covariate (size-specific model) and excluded as a covariate (absolute model). Juvenile food levels are low (= 1) and high (= 0). Adult food levels are low (= 1) and high (= 0). For all models, parameter estimates are centered on the grand mean age (57 days) and the grand mean female SL (15.31 mm) at the yolking of the first litter. * $P < 0.05$; ** $P < 0.01$, $^{n/s}P > 0.05$; $^{n/a}$ term not included in model. [†] Estimates are given for juvenile food = L and adult food = L.

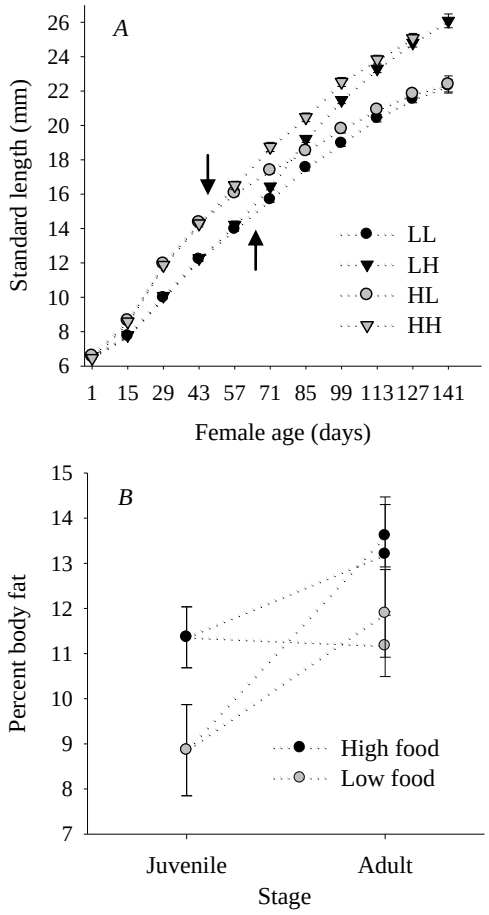

Figure 2.1. (*A*) Changes in standard length (mm) in females with high-high (HH), high-low (HL), low-high (LH) and low-low (LL) food across the juvenile and adult stages. Arrows point to mean age at maturation when the food level switch for HL (upper arrow) and LH (lower arrow) occurred. (*B*) Percent body fat at the end of the juvenile stage (Juvenile) and at the birth of the third litter in the adult stage (Adult) in females that were reared as juveniles under low (gray) or high (black) food levels that experienced low (gray) or high (black) food levels during the adult stage. Given are means ± 1SE.

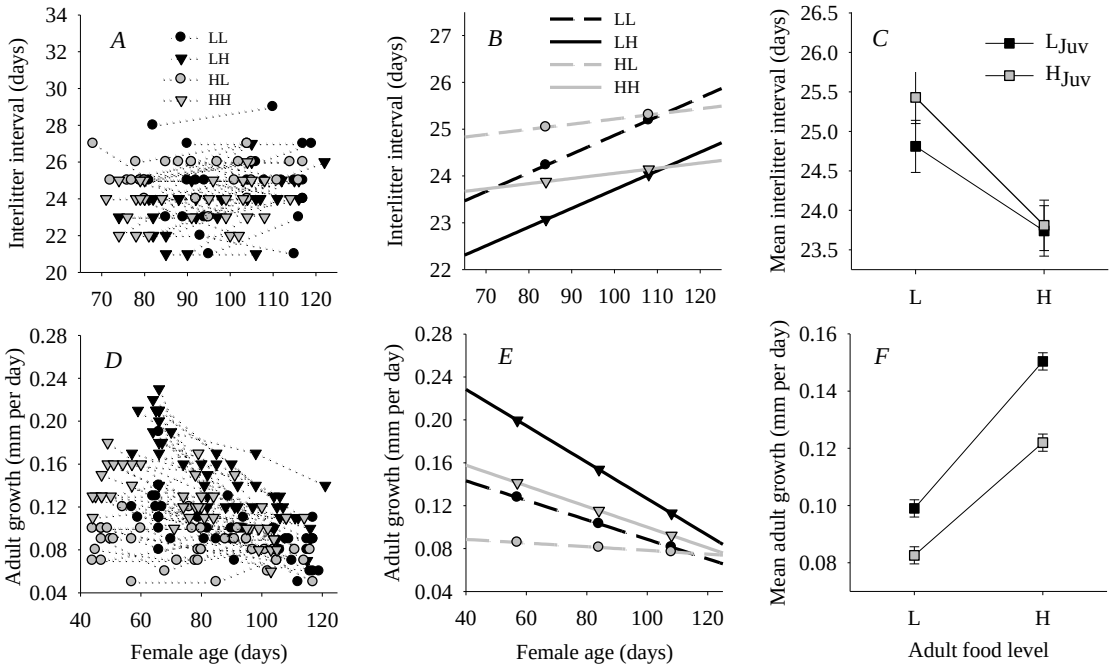

Figure 2.2. Observed trajectories (left), predicted trajectories (center) and reaction norms (right) for interlitter interval (*A-C*) and adult growth in length (*D-F*) under different juvenile and adult food levels. Trajectories are for females with high-high (HH), high-low (HL), low-high (LH) and low-low (LL) food levels across the juvenile and adult stage. For predicted trajectories, symbols represent the grand mean age at each of the three litters. Reaction norms are for those same females reared on low (L_{Juv}) and high (H_{Juv}) juvenile food levels under low (L) and high (H) adult food levels.

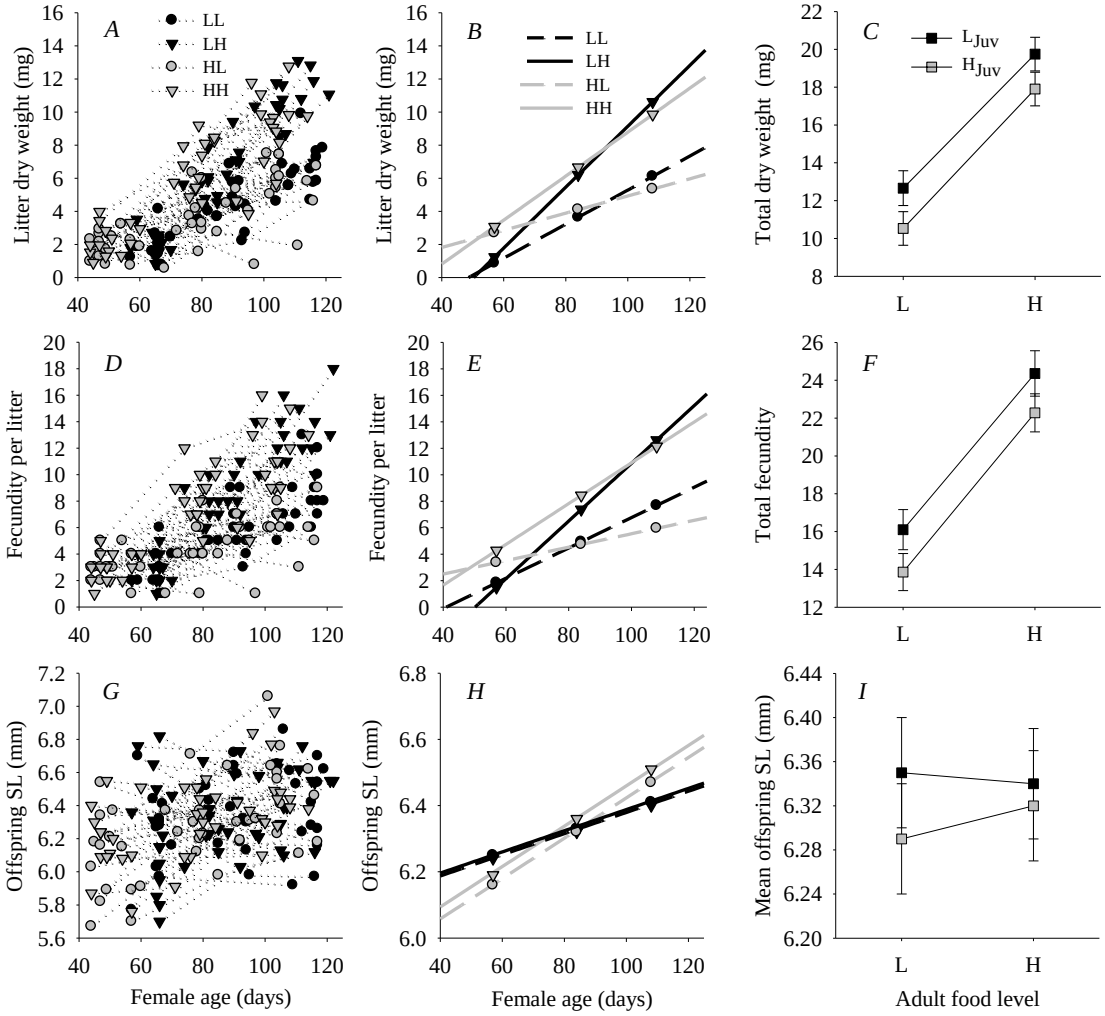

Figure 2.3. Observed trajectories (left), predicted trajectories (center) and reaction norms (right) for litter dry weight (*A-C*), fecundity (*D-F*), and mean offspring standard length (SL; *G-I*) under different juvenile and adult food levels. Trajectories are for females with high-high (HH), high-low (HL), low-high (LH) and low-low (LL) food levels across the juvenile and adult stage. For predicted trajectories, symbols represent the grand mean age at each of the three litters. Reaction norms are for those same females reared on low (L_{Juv}) and high (H_{Juv}) juvenile food levels under low (L) and high (H) adult food levels.

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Chapter 3

Life histories have a history:

Effects of past and present conditions on adult somatic growth rates in wild guppies

Abstract

Current environmental conditions during the adult stage as well as those in the recent past and during the juvenile stage can have significant effects on adult performance and population dynamics, but their relative importance remains unexplored. I examined the influence of food availability in the present, in the recent past and during the juvenile stage on adult somatic growth rates in Trinidadian guppies (*Poecilia reticulata*). I found that poor conditions during the juvenile stage, as reflected by low food availability, had a positive effect on adult growth rates, while the same conditions in the recent past generally had negative effects on adult growth. These results suggest that the same conditions at different stages may have different effects on the state of the organism and its subsequent long- and short-term growth strategies, respectively. A simultaneous consideration of events in both the recent and distant past may therefore improve predictions for individual- and population-level responses to environmental change.

Introduction

Most organisms face considerable environmental variation over their lifetime. For example, numerous organisms have life cycles in which juveniles and adults live in different habitats and capitalize on different resources (Moran 1994). Migratory species experience a wide range of habitat conditions during migration and while residing on their breeding and wintering grounds (Webster et al. 2002). Many organisms also face temporal variation within a given habitat through stochastic fluctuations or regular seasonal changes in factors such as temperature and food availability (Poulin et al. 1992; Farley and Fitter 1999). Throughout an organism's life history, conditions experienced at one moment can therefore differ markedly from those encountered at an earlier time and life stage.

Among these organisms, there is increasing evidence that past conditions can have lasting effects on individual performance. Behavioral and reproductive decisions, such as where to forage and when to reproduce, can depend on current conditions, but are also contingent on the individual's history of past environmental exposure (Metcalfe and Monaghan 2001; Lummaa and Clutton-Brock 2002; Harrison et al. 2010). These delayed effects are thought to occur because environmental conditions at any given time influence the internal state of the individual – such as its body size and condition – which in turn affects its performance in the future (McNamara and Houston 1996; Jørgensen and Fiksen 2006).

Conditions experienced during early growth and development can have long-term consequences for the adult phenotype (Henry and Ulijaszek 1996; Monaghan 2008). For

example, a suite of traits – including metabolic, cardiovascular, behavioral, reproductive and immune functions – has been shown to vary with nutrition during early growth and development (Bateson 2001; Jasienska et al. 2006; Schilder and Marden 2006) and have lasting effects on individual life history traits and subsequent reproductive success (Descamps et al. 2008; Hamel et al. 2009; Auer et al. 2010). Long-term effects of early environments on individual and cohort performance, called 'delayed life history effects' or 'delayed quality effects' or 'developmental effects', have been demonstrated in numerous organisms and are thought to play an important role in population dynamics (Sæther 1997; Beckerman et al. 2002; Lindström and Kokko 2002; Beckerman et al. 2003; Benton et al. 2006; Allen and Marshall 2010).

Adult performance is also influenced by more recent conditions experienced in the adult stage (Bearhop et al. 2004; Brommer et al. 2004; Cook et al. 2004; Norris 2005; Harrison et al. 2010). 'Carry-over effects' or 'seasonal interactions' can occur when events in one season affect the state of the individual and its success during the following season (Norris 2005; Harrison et al. 2010). For example, winter food quality can have positive effects on reproductive success in the upcoming breeding season (Norris and Marra 2007; Robb et al. 2008; Sorensen et al. 2009). Environmental conditions can also have more immediate consequences for adult performance within a season. For example, some organisms rely on stored energy acquired only recently to finance reproduction (Reznick and Yang 1993; Warner et al. 2008; Stephens et al. 2009). Like the long-term effects of juvenile environments, these delayed effects during the adult stage on

individual and cohort performance are thought to play an influential role in population dynamics (Runge and Marra 2005; Norris and Marra 2007; Harrison et al. 2010).

An individual's current state and its ability to respond to change may therefore reflect an integrated history of exposure to past environments in both the adult stage and the juvenile stage. However, because the delayed effects of environmental variation within and across life stages have traditionally been studied in isolation, their relative influence and subsequent importance remain unexplored. On one hand, conditions during critical stages of early growth and development can affect the structure and *modus operandi* of the developing organism and may have permanent effects on the emergent phenotype and its performance (Henry and Ulijaszek 1996; Metcalfe and Monaghan 2001). On the other hand, phenotypic plasticity in life history strategies or subsequent environmental heterogeneity may weaken or obliterate these earlier effects over time (Auer 2010). In the latter case, more recent conditions in the adult stage may have a larger effect on the present state of the adult organism and how it responds to variation in the current environment. In predicting individual responses to environmental change and their subsequent effect on population dynamics, it remains unclear if we need to account for the entire histories of individuals within a population or if events at one life stage or time are better predictors of individual performance than others.

I examined the effects of juvenile, recent and current environmental conditions on adult somatic growth rates in cohorts of wild Trinidadian guppies (*Poecilia reticulata*). Guppies are small, livebearing fishes that inhabit freshwater streams on the island of Trinidad, West Indies. Food availability in these streams fluctuates on a seasonal basis

through distinct alternating wet and dry periods (Reznick 1989; Kohler 2010). Because food availability can change substantially within only a few months, growth conditions can vary widely throughout the lifetime of a guppy. Studies demonstrate that guppy growth rates are highly sensitive to variation in both juvenile and adult environmental conditions (Grether et al. 2001; Auer 2010). Because of their effect on body size and vital rates, somatic growth rates are known to have important consequences for individual fitness and for the demographic structure and dynamics of guppy populations (Rodd and Reznick 1997; Bronikowski et al. 2002). Conditions during both the early and later stages of life therefore have the potential to show delayed effects on adult growth and subsequent population demographics.

Here I present data on seasonal variation in adult growth rates of wild guppy females. Using bimonthly estimates of food availability coupled with longitudinal measurements of female body size, I examined female growth responses to current conditions, investigated how those growth rates were affected by conditions in the immediate past as well as in the juvenile stage, and evaluated the relative importance of early, recent and current environments as predictors of adult growth.

Methods

Study area

The study was conducted in a small tributary to the upper reaches of the Guanapo River in the Northern Range Mountains of Trinidad, West Indies where a population of guppies was introduced in March 2008 and has been monitored since. The tributary is a small, first order stream with a distinct pool-riffle structure. It is located above a barrier

waterfall that excludes all but one fish species, Hart's killifish (*Rivulus hartii*) that may prey on smaller guppies (Liley and Seghers 1975). As part of a large-scale study on the feedbacks between ecological and evolutionary processes, 38 pregnant female and 38 male guppies were transplanted to this natural upstream tributary in March 2008 from a downstream site. Each female and male was tagged with a unique color combination of subcutaneous visible implant elastomer marks (NorthWest Marine Technology, Washington, USA). This marking technique allows identification of individuals without sacrificing their chances of survival in the field (Reznick et al. 1996). The introduction area comprises approximately 100 m of stream between two waterfalls, the downstream waterfall preventing immigration into the site from downstream populations.

Study design

I examined adult growth responses to current and previous food availability in recruits from this founding population. Guppies larger than 14 mm in standard length were netted each month and brought back into the laboratory. All recruits were individually marked once they reached 14 mm, as done for the founding females and males. Standard length was measured from photographs taken of each fish upon its initial capture and each successive recapture. For each photograph, the fish was anesthetized in a neutrally buffered solution of MS-222, and straightened over a plate next to a ruler. Its picture was then taken with a digital camera mounted on a tripod above. Standard length was then determined using the program Image J (US NIH, Maryland, USA). Guppies were tracked from May 2008 to July 2009, the first cohorts recruiting two months after the initial introduction in March 2008.

Food availability was estimated by sampling both benthic algae and benthic invertebrates that guppies are known to prey on (Bassar *et al.* 2010). Three transects were established in pools 30-50 meters apart and were sampled on a bimonthly basis from March 2008 to July 2009. Pools are the habitat most used by guppies and were defined as those deeper sections of stream that accumulated fine material and leaves under low flow velocities.

To estimate algal abundance, epilithon samples were collected using a modified version of a Loeb sampler (5.07cm^2, Loeb 1981). Epilithon was sampled from five to ten randomly-selected areas in the vicinity of each transect and combined into a single slurry. Quantitative subsamples taken from each slurry were used to estimate biomass as ash free dry mass (AFDM). AFDM was analyzed by filtering a known amount of homogenous sample through a pre-weighed ashed (at 450ºC for two hours) 47 mm Whatman GF/F filter. The material was dried in an oven at 50-55ºC until constant dry mass was achieved, and then placed in a desiccator until analysis. The material and filter together were weighed, ashed in a muffle furnace at 500ºC for one hour, and reweighed to calculate AFDM per m2 (Steinman et al. 2006).

To estimate benthic invertebrate abundance, macroinvertebrate samples were collected from pools on the same bimonthly basis as the epilithon samples described above using a Hess sampler with a basal area of 0.032 m2 and 250 μm mesh size (Wildlife Supply Company, Florida, USA). The substratum was disturbed by hand to suspend invertebrates which were then collected in a downstream net. The samples were rinsed through a 250 μm sieve, and retained materials were rinsed into a sample bag and

preserved with ≥70% ethanol containing rose bengal dye to facilitate sorting. In the laboratory, samples were rinsed through stacked 1 mm and 250 μm sieves to separate coarse (>1mm) and fine (>250 μm, <1mm) fractions. All invertebrates were removed from coarse fractions, whereas fine fractions were subsampled if necessary. Subsampling was accomplished using a Folsom plankton splitter (Aquatic Research Instruments, Idaho, USA) to divide samples (up to ⅛), and subsampled fractions were processed completely to acquire the target of 100 organisms (75 minimum). Sorted invertebrates were identified to the lowest practical taxonomic level (usually genus), counted, and measured to the nearest millimeter using a stage micrometer. AFDM was quantified using length-mass regressions calculated following the procedures of Benke et al. (1999) or from published values (Benke et al. 1999; Sabo et al. 2002; Baumgartner and Rothhaupt 2003; Hall et al. 2006; Miyasaka et al. 2008).

Because guppies are known to feed on both algae and invertebrates (Bassar *et al.* 2010), the mean of the summed AFDM of both invertebrates and algae per month across the three transects was used as a measure of total food availability. To control for effects of population density on per capita food availability, guppy population density was also estimated each month by counting all individuals in the populations known to be alive, whether because they were caught that particular month or because they were caught at other monthly census dates both before and after a given census date.

Statistical analyses

I examined how food availability during the juvenile and adult stage affected adult growth rates in the current environment. I defined the effects of three periods of

exposure to such conditions: current conditions during the adult stage, recent conditions in the adult stage, and conditions experienced during the juvenile stage (Figure 3.1). Current conditions were defined as those present during each monthly growth measurement. Since food availability estimates were available every other month, current conditions were defined for every other month. Recent conditions were defined as those present during the previous sampling episode two months prior. Finally, early juvenile conditions were defined as those present at the time of first capture (Figure 3.1). I considered only those individual females that were first captured at a standard length smaller than 21 mm; size at maturation ranges from 15 to 20 mm in the field, so juvenile conditions could not be reliably assigned to individuals larger than 21 mm (Reznick and Endler 1982). Because the juvenile stage in guppies is known to range from 1.5 to 2 months in duration (Reznick and Bryga 1996; Auer et al. 2010), individuals that first recruited during and directly after a month in which food was measured were considered part of the same cohort (Figure 3.1). For example, if food availability was measured during January, I assumed that during their two month stage as juveniles, that individuals recruiting in February had also experienced conditions present in January. In order to ensure that the juvenile stage did not coincide with the recent past stage two months prior, I considered only individuals that were tracked for at least three months after they first recruited. There were a total of 208 females that met these criteria. The analyzed data included 1-6 monthly growth measurements per female with an average of 2.4 growth measures per female, totaling 494 non-independent data points.

I used a linear mixed model approach to examine effects of food availability in the past and present on adult growth responses. Standard length and its square at the beginning of each monthly growth period served as covariates for all measurements of growth since growth is known to be a convex decreasing function of length (Reznick and Bryant 2007). Food level and density during the juvenile stage were used as time-invariant individual covariates. Current food level, current population density, food levels two months prior, and population density two months prior were modeled as time-varying covariates since they changed throughout the lifetime. Female identity was nested within cohort and included as a random effect to control for the non-independence of repeated measures on the same female and to control for conditions unique to each cohort that were not measured (Singer and Willett 2003; Bolker et al. 2009). Monthly recapture intervals ranged from 26-33 days. Monthly adult growth rate was calculated as $(SL_{recapture}–SL_{last\ capture})$ /(the number of days between captures) and then multiplied by 30 (mm $month^{-1}$). Parameter estimates were centered around 23 mm, the mean adult size at which growth measurements were obtained (sensu Singer and Willett 2003; West et al. 2006). Residuals were normally distributed, so growth was analyzed using the Mixed procedure in SAS version 9.3 (SAS Institute, North Carolina, USA).

Food availability in the current environment was not correlated with food levels two months prior (r = 0.3, p = 0.6) or with juvenile food level (r = - 0.06, p = 0.8), nor was food availability two months ago correlated with juvenile food (r = 0.2, p = 0.5), so measures at each stage provided independent measures of food availability. Food availability and population density were not correlated either (r = 0.1, p = 0.6). However,

correlation coefficients between population densities at the different stages were all significant (all $r > 0.45$, all $p < 0.05$). Therefore, only current density was included in the model to control for per capita food availability.

To find the model that best described adult monthly growth, I started with a full model that included all main effects and first order interactions between food availability at the three time stages and then used backward model selection, sequentially removing terms with the lowest *F* values until all terms in the model were significant. Terms in the model were evaluated using maximum likelihood estimation, but estimates presented for the final model are based on restricted maximum likelihood (Zuur *et al.* 2009).

Results

During the study period, food availability fluctuated significantly, in some instances increasing or decreasing 12 fold across a span of two months (Figure 3.2 *A*). Population density also changed through time, but generally increased with only minor fluctuations in its upward trend (Figure 3.2 *B*).

Individual and cohort growth trajectories

Standard lengths for adult growth measurements ranged from 17.24 – 32.62 mm and averaged 23.4 mm. Adult growth ranged from 0 – 3.63 mm per month and averaged 0.56 ± 0.03 mm per month across all months and all size classes. All individuals grew rapidly at first and then slowed their growth as they reached larger sizes (Table 3.1, Figure 3.3 *A*). There was significant variation among individual females in their asymptotic body size, some slowing their growth appreciably at 22 mm and others at 28 mm. Size-dependent growth trajectories also differed considerably among cohorts, some

cohorts growing twice as much in one month as other cohorts in the smaller size classes (Figure 3.3 *B*).

Effects of the past and present

Food availability at all time stages – those during the present, 2 months prior, and as far back as the juvenile stage – all had a significant effect on adult growth rates after controlling for effects of body size and population density (Table 3.1). However, the direction of these effects differed among time stages and, in some cases, their magnitude depended on food levels at other time stages. Current food availability and food availability experienced two months prior both had a positive effect on monthly growth rates (Table 3, Figure 3.4 *A, B*). In contrast, food availability during the juvenile stage generally had a negative effect on adult growth (Table 3.1, Figure 3.4 *C*). However, there was also a significant positive interaction between food levels experienced during the juvenile stage and 2 months prior such that the effects of juvenile food level became less and less negative as the food level experienced 2 months prior increased (Table 3.1, Figure 3.5 *B)*. Overall, individuals reared as juveniles under lower food grew faster as adults than those reared as juveniles under higher food levels in all but one case: when food levels experienced 2 months prior were at their highest, individuals reared as juveniles under lower and higher food grew at the same rate (Figure 3.5).

Discussion

An individual's history of exposure to past environmental conditions in the adult stage and as far back as the juvenile stage is known to affect its decisions in the current environment. However, despite the role that past events in these two life stages are

thought to play in processes such as population dynamics, their effects on and relative importance for individual performance have not yet been evaluated simultaneously. Here I show that past events occurring in the juvenile and adult stage can both have important delayed consequences for adult growth strategies, but the direction of these effects can differ among life stages and their magnitude can depend on food levels at other life stages.

Poor conditions during the juvenile stage, as reflected by low food availability, generally had a positive effect on adult growth rates, while the same conditions in the recent past had negative effects on adult growth. These contrasting effects suggest that the same conditions during the juvenile and adult stages may have different effects on the state of the organism and its subsequent long- and short-term growth strategies, respectively. However, the dampening of juvenile food effects by food levels experienced more recently in the adult stage also suggests that heterogeneity in the adult stage can, in some cases, obliterate the long-term effects of juvenile environments.

The negative effect of low food experienced 2 months prior that I report here is consistent with effects observed in other studies. Poor conditions in the adult stage, such as low food availability or high population density, generally have negative effects on physiological condition and subsequent performance (Ratikainen et al. 2008; Harrison et al. 2010). Poor conditions can increase stress levels and have negative effects on aspects of immune function such as parasite resistance which can then carry-over to negatively affect future performance, including growth (Pickering 1993; Norris and Evans 2000; Karell et al. 2007; Kopp and Medzhitov 2009). Negative effects of low food levels and

high population density on muscle mass and energy reserves can also be detrimental to future performance (Bearhop *et al.* 2004; Robb *et al.* 2008; Sorensen *et al.* 2009). Conditions in the recent past can place constraints on an organism's ability to grow or reproduce. For example, the probability of pregnancy in elk is contingent on them storing up a threshold level of 8-10% body fat during the previous non-breeding season (Cook *et al.* 2004). Recent conditions may also favor reallocation of resources to other functions at the expense of growth. For example, organisms that experience an increase in food levels following a period of food restriction often divert resources to replenish their energy stores before resuming structural growth (Nicieza and Metcalfe 1997; Auer et al. 2010).

Why and how poor juvenile conditions had a positive effect on adult guppy growth rates is less clear. Studies typically find that poor conditions during the juvenile stage, like recent conditions during the adult stage, have a negative effect on the physiological state of the organism at maturation and its subsequent performance as an adult (Lindström 1999; Lummaa and Clutton-Brock 2002). Low food availability during the juvenile stage can lead to smaller adult body size, reduced energy reserves, increased stress levels and increased susceptibility to starvation, disease, and predation (Lindström 1999; Morgan and Metcalfe 2001; Lummaa and Clutton-Brock 2002; McMillen and Robinson 2005; Davis and Maerz 2009), which in turn can lead to reduced somatic growth, reproductive success and survival in the adult stage of life (Madsen and Shine 2000; De Block and Stoks 2005; Descamps et al. 2008). However, there is also evidence from guppies suggesting that poor juvenile conditions do not always spell doom for adult performance. Guppies reared as juveniles under low food levels in the laboratory are

older, smaller and have fewer energy reserves at maturation than females reared on higher food levels, but they are able to compensate for these initial setbacks through phenotypic plasticity in their growth and reproductive strategies during the adult stage (Auer 2010). As adults, females reared as juveniles on low food replenished their fat reserves, increased their growth rate to make up for their small body size, and increased their production of babies to compensate for their delayed maturity such that overall they were able to enjoy the same reproductive success as females reared on high food as juveniles (Auer 2010).

Why do poor juvenile conditions have negative long-term effects in some cases and positive long-term effects in others? Variation in how juvenile conditions affect later adult performance may stem from differences in the magnitude of the early insult observed across studies; extremely poor early conditions may have delayed pathological effects on adult physiology and health, while more moderate reductions in the quality of the rearing environment may permit organisms to mitigate or even compensate for any early setbacks later on in life (Monaghan 2008). Variation in species' responses may also reflect differences in their evolutionary history; those species that have evolved in variable environments may be more likely to be able to cope with early perturbations through increased flexibility or phenotypic plasticity in their anatomy, physiology and behavior relative to species that have evolved in more constant environments (Pigliucci 2001; DeWitt and Scheiner 2004; Monaghan 2008).

Differences in adult growth strategies may also explain why poor conditions during the juvenile stage have contrasting long-term effects across species. One of the

primary ways that early conditions can influence adult performance is through effects on juvenile growth rates and subsequent adult body size. Deviations in the quality of the environment during the juvenile stage can have significant effects on an individual's size-at-age because this is the time when growth is the fastest (Metcalfe and Monaghan 2001). A smaller size-at-age caused by low early food availability can have potentially negative consequences for future size-dependent survival and reproduction (Metcalfe and Monaghan 2001; Roff 2002). In organisms with determinate growth, juvenile conditions can therefore have a permanent effect on adult body size and subsequent success. However, organisms with indeterminate growth still have the opportunity during adulthood to increase their growth rate and catch up to the body size they would have achieved under more favorable conditions. Compensatory growth during the juvenile stage is a well known phenomenon (Metcalfe and Monaghan 2001; Ali et al. 2003). However, adult growth responses to juvenile conditions have only recently been examined and their prevalence in organisms with indeterminate growth is still uncertain. Madsen and Shine (2000) found a negative effect of poor early growth conditions on adult growth rates of free ranging pythons, while Taborsky (2006) found that adult growth rates were influenced only by current food availability in a mouth brooding cichlid. In contrast, I have previously demonstrated in a laboratory study that female Trinidadian guppies reared on low food as juveniles generally grow faster for their size as adults as reported here (Auer 2010). The dampening of juvenile food effects on adult growth by more recent food levels in the adult stage observed in this study are also consistent with those effects observed in the laboratory (Auer 2010). When adult food

levels are high, individuals reared in the laboratory under low food levels initially grow faster but then maintain the same growth rate as individuals reared under high food levels after a period of time under those high adult food levels. This congruence between effects observed in the laboratory and field suggest that the patterns I observed in this study represent phenotypic plasticity of adult growth in response to early conditions.

Alternatively, the faster somatic growth I observed in adults that had recruited during periods of low food availability may be due to sources other than the direct effects of the external environment on the state of the individual. Importantly, variation in adult growth rates could reflect genetic differences among cohorts either because of seasonal differences in the composition of reproductive individuals that produced these cohorts or because of viability selection. Guppies breed during periods of both low and high food availability (Reznick 1989), but the number of reproductively active females changes seasonally, more females reproducing during the dry season when food levels are highest (Alkins-Koo 2000). Thus, if only high quality females are able to breed under the low food conditions during the wet season, then cohorts born during these rough times may be of higher quality themselves and therefore exhibit higher growth as adults. The higher growth I observed in females that recruited under poor juvenile conditions may also be due to viability selection causing a temporal shift in genotype frequency within a cohort, whereby only higher quality individuals born in the wet season survive to the size at which I started tracking them. However, guppy individuals that are followed from birth into adulthood in the laboratory exhibit the same growth responses as observed here

(Auer 2010), so it is likely that these growth responses reflect phenotypic plasticity, at least in part.

There is an increasing appreciation that conditions in both the recent and distant past can produce variation in demographic life history traits among cohorts and individuals and that this heterogeneity in performance can then have important consequences for population size, structure and dynamics (Sæther 1997; Beckerman et al. 2002; Lindström and Kokko 2002; Benton et al. 2006; Ratikainen et al. 2008). Since population dynamics are a consequence of the summed life history responses of all individual constituents, predicting how populations will respond to changes in the environment therefore hinges on how well we understand the link between the environment and individual performance (Beckerman et al. 2002; Benton et al. 2006). Previous studies have demonstrated repeatedly that individual performance is influenced by conditions in the recent and distant past, and these effects are now increasingly being incorporated, albeit separately, into demographic models (Lindström and Kokko 2002; Ratikainen et al. 2008). While these models typically assume that individual performance is influenced in a positive manner by the quality of conditions in the past, here I found that this assumption is met with respect to the effect of recent conditions but not the effect of juvenile conditions on adult growth rates. These differences between guppies and other organisms in how they respond as adults to conditions experienced during the early stages of life underscore the need to better understand how and why responses to early conditions may differ among species. Second, this study highlights the importance of simultaneously considering events occurring in both the recent and distant past.

Many species face new challenges as ecosystems worldwide undergo rapid human-induced changes (Vitousek et al. 1997; McCarty 2001; Walther et al. 2002). Given these recent changes in climate and habitat size and distribution, there is now an urgent need to better understand how organisms are affected by, and cope with, changes in environmental conditions experienced in different life stages. Here I show that individual variation in a key demographic trait, adult somatic growth rate, reflects an integrated history of exposure to environmental conditions in both the past and present. However, the mechanisms underlying these individual growth responses and how these growth responses translate into population level processes still require further attention if we are to improve our predictions for how individuals and populations might be expected to respond to current environmental change.

Table 3.1. Final linear mixed model of the effects of standard length (SL) and its square (SL^2), current population density (CurDen), and food availability in the present (CurFood), two months prior (2moFood) and during the juvenile stage (JuvFood) on adult somatic growth rates in adult female Trinidadian guppies (*Poecilia reticulata*). Food availability is calculated as summed ash free dry mass (g per m^2) of both algae and invertebrates. SL is centered on 23 mm.

Fixed effects	Estimate	SE	df	*t*	p
Intercept	1.35	0.10	206	13.10	< 0.001
SL	- 0.10	0.01	277	- 8.83	< 0.001
SL^2	0.01	0.003	277	4.16	< 0.001
CurDen	- 0.003	0.0003	277	- 9.95	< 0.001
CurFood	0.024	0.003	277	7.48	< 0.001
2moFood	0.018	0.004	277	4.74	< 0.001
JuvFood	- 0.016	0.003	277	- 4.90	< 0.001
JuvFood*2moFood	0.0002	0.0001	277	2.99	< 0.01
Random Effects	Estimate	SE	*Wald Z*		p
Female intercept	0.036	0.018	2.04		0.02
Residual	0.22	0.019	11.38		< 0.0001

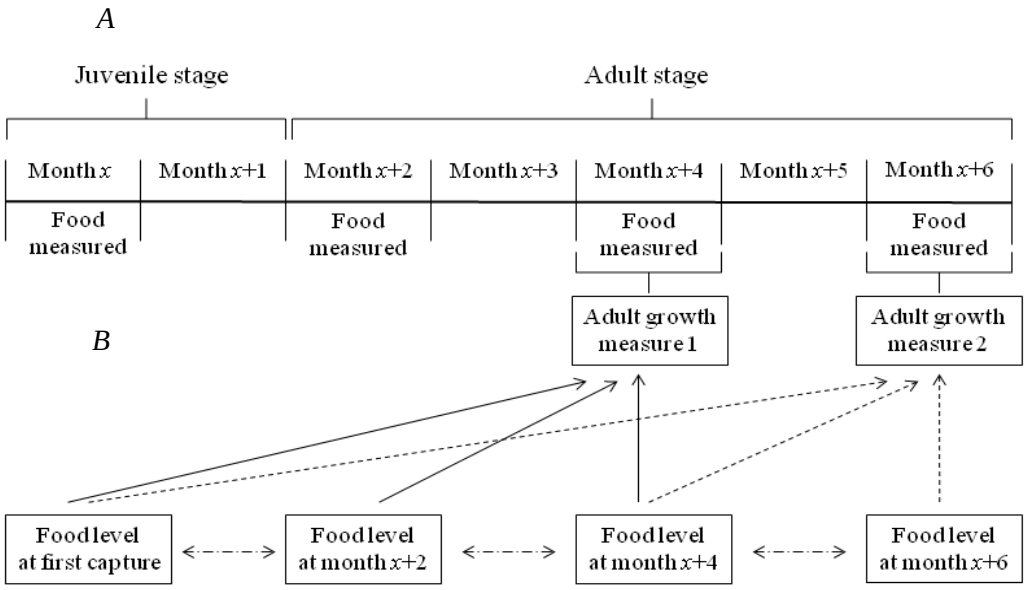

Fig. 3.1. Schematic illustrating (*A*) the schedule of food availability measurements and adult growth measurements for a given fish, and (*B*) food conditions during the juvenile stage, 2 months prior, and in the present and their interactions that could influence an individual's growth rate during the adult stage.

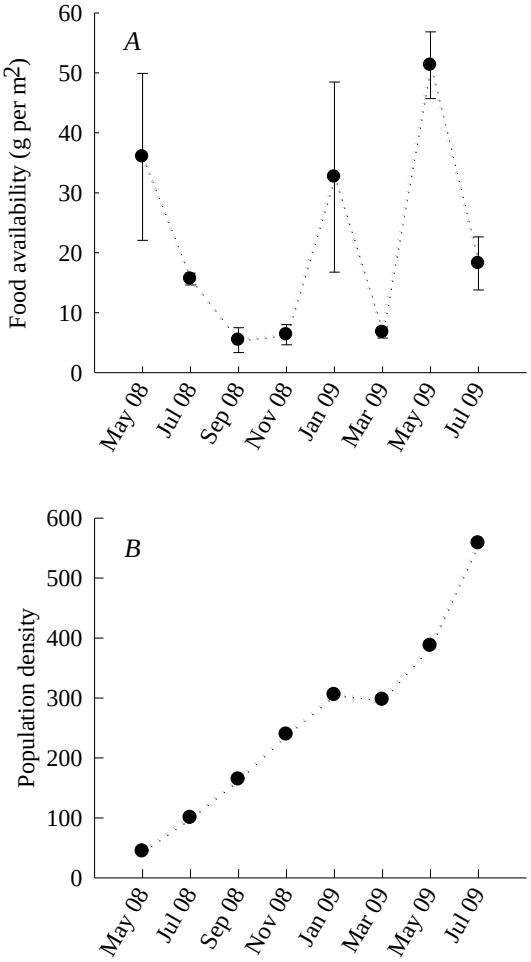

Fig. 3.2. Temporal variation in (*A*) food availability and (*B*) population density in a tributary to the upper Guanapo River in Trinidad, West Indies. Food availability is calculated as the summed ash free dry mass (g) of both algae and invertebrates per square meter. Estimates of population density do not include individuals smaller than 14 mm in length.

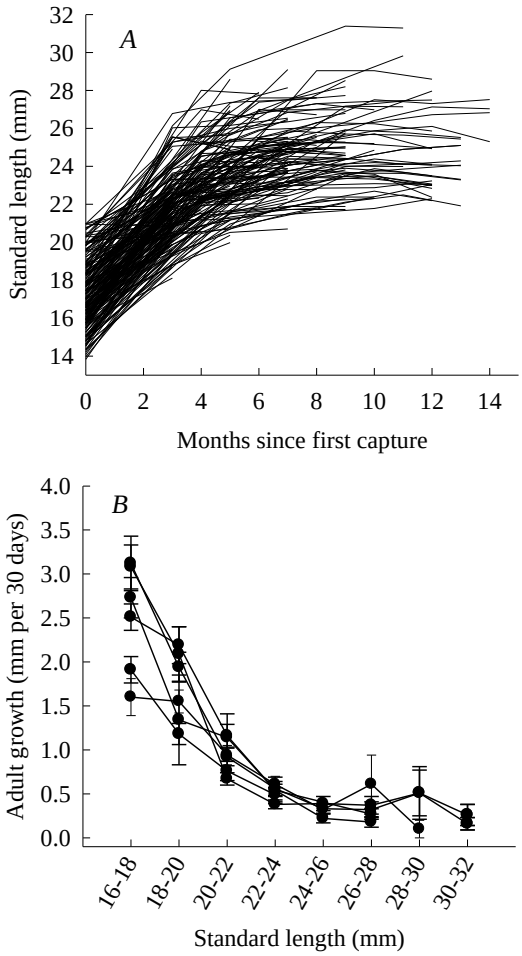

Fig. 3.3. (*A*) Size trajectories of 208 individual females, and (*B*) growth trajectories of those same females from 6 different cohorts born between March 2008 and May 2009 in a tributary to the upper Guanapo River in Trinidad, West Indies.

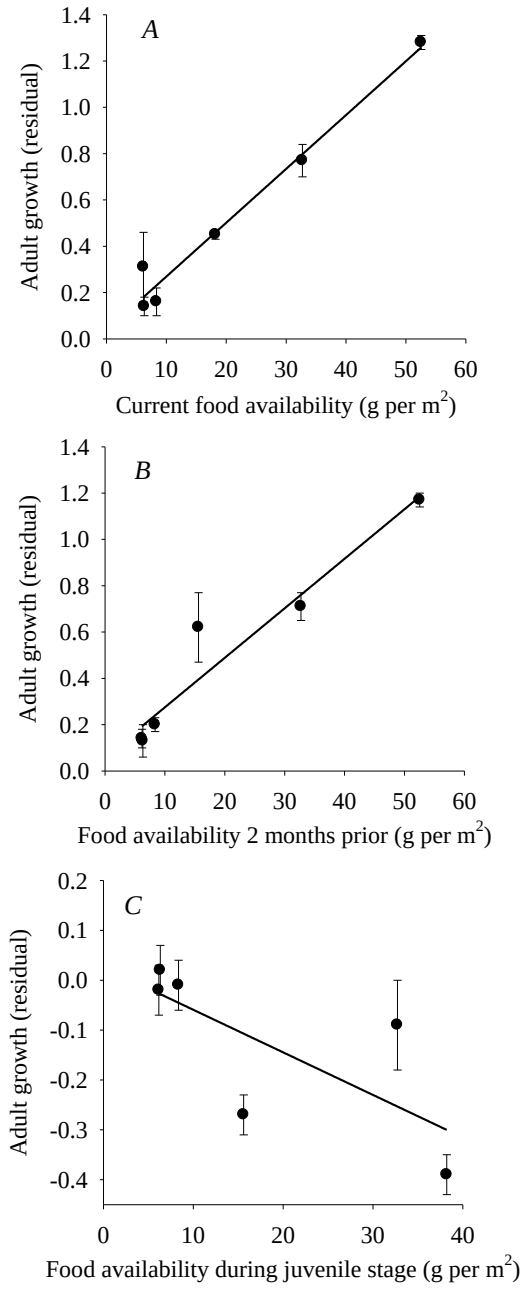

Fig. 3.4. Adult growth as a function of food availability in the present (*A*), two months prior (*B*) and during the juvenile stage (*C*) in Trinidadian guppies in a tributary to the Guanapo River in Trinidad, West Indies. Food availability is calculated as the summed ash free dry mass (g) of both algae and invertebrates per square meter. Shown are partial residuals when accounting for all other environmental variables and their interactions.

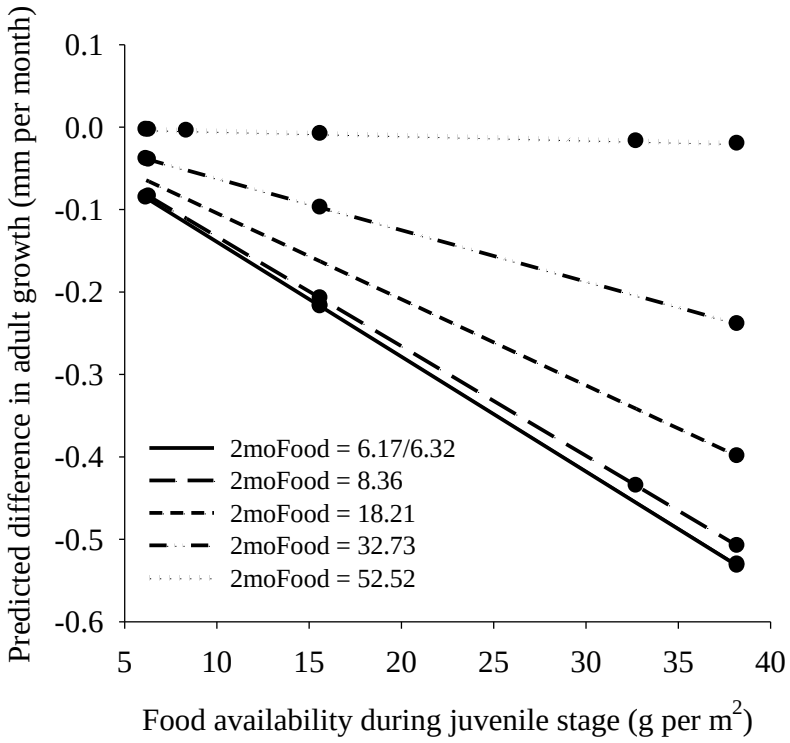

Fig. 3.5. Predicted difference in growth from mean adult growth in response to juvenile food level as a function of food availability 2 months prior (2moFood) in Trinidadian guppies in a tributary to the Guanapo River in Trinidad, West Indies. Food availability is calculated as the summed ash free dry mass (g) of both algae and invertebrates per square meter. Food levels of 6.17 and 6.32 g AFDM m^{-2} are shown together since their predicted trajectories overlap. Points show growth rate predicted for combinations of food levels actually observed.

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Concluding remarks

Food availability during early growth and development can have a profound effect on the adult phenotype and its performance. Low food availability is generally thought to have permanent negative impacts on adult components of fitness. There is increasing evidence that organisms can respond to these early setbacks through changes in their life history strategies. However, our current understanding of the diversity of compensatory mechanisms utilized, the costs and benefits of these responses, and the relative importance of these responses under environmental variability in the wild is still in a nascent stage. My dissertation research investigated how juvenile food availability influences adult reproductive decisions and success in Trinidadian guppies (*Poecilia reticulata*).

Chapter One demonstrated that juvenile compensatory growth can have negative effects on reproductive success. Previous studies have demonstrated a cost to the timing of male maturation, but this is the first study to demonstrate a cost to reproduction in females. These reproductive costs may play an important role in the evolution of compensatory growth and other rapid juvenile growth responses to variation in the environment. However, the physiological mechanism(s) underlying this observed cost is presently unknown and requires further study. Additionally, the overall adaptive value of compensatory growth is not clear; theory generally assumes that compensatory growth is costly only to survival, but should now consider reproductive costs when determining the optimal growth decision in a given environment.

Chapter Two demonstrated that low food availability during early growth and development does not always spell doom for adult performance. Long-term effects of early food levels have typically been studied in organisms with determinate growth where the effects of juvenile environments on adult final body size are permanent. This is the first study to show that phenotypic plasticity in age-related adult life history decisions can not only mitigate but, surprisingly, can fully compensate for setbacks caused by poor juvenile food conditions. However, the adaptive value of these responses still requires further attention. Because growth and reproduction can be costly to current and future survival, respectively, an examination of the overall fitness consequences of these responses in the wild would be most informative.

Chapter Three demonstrated that past events occurring during the juvenile and adult stage both had important consequences for adult growth strategies, but the direction of their influence differed; poor conditions during the juvenile stage had a positive effect, while those same conditions during the adult stage had a negative effect on adult growth rate. Past events in the juvenile and in the adult stage have traditionally been studied in isolation. This is the first study to consider these past events simultaneously and to evaluate their relative effects on adult growth responses. While the observed effects of recent events are consistent with the findings of other studies, the negative effect of juvenile food availability on adult growth rates in guppies, as observed here and in Chapter Two, contrasts with those effects found in other organisms. That juvenile food level can have a positive or no effect on adult growth in some species and a negative effect in others underscores the need to examine adult growth responses in multiple

species under common conditions and thereby evaluate whether variation in species' responses arise merely because of differences in the severity of the initial setback across study designs or because of actual differences in their evolutionary history and the types of environments in which they have evolved.

Understanding the long-term effects of early environments is if central importance to biomedical researchers examining the developmental origins of human disease, to evolutionary biologists interested in the adaptive value of phenotypic responses to environmental change, and to ecologists predicting the effects of heterogeneity among individuals on population level processes. This dissertation research demonstrates that early environments can have long-term effects on adult reproductive decisions and success. First, it shows that juvenile compensatory growth to an increase in food levels after a period of growth restriction is costly to reproduction. Second, it highlights that adult life history strategies are not just a passive consequence of the limitations imposed by the environment but that adults can respond flexibly to variation in both past and present environments and in ways that might be adaptive. Finally, it demonstrates that past events in both the juvenile and adult stage can have equally important effects on adult growth strategies and should both be considered when predicting individual- and population-level responses to environmental change.