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CROSS-GENERATIONAL ENVIRONMENTAL EFFECTS AND THE EVOLUTION OF OFFSPRING SIZE IN THE TRINIDADIAN GUPPY *POECILIA RETICULATA*

FARRAH BASHEY[1]
Department of Biology, University of California, Riverside, California 92521

Abstract.—The existence of adaptive phenotypic plasticity demands that we study the evolution of reaction norms, rather than just the evolution of fixed traits. This approach requires the examination of functional relationships among traits not only in a single environment but across environments and between traits and plasticity itself. In this study, I examined the interplay of plasticity and local adaptation of offspring size in the Trinidadian guppy, *Poecilia reticulata*. Guppies respond to food restriction by growing and reproducing less but also by producing larger offspring. This plastic difference in offspring size is of the same order of magnitude as evolved genetic differences among populations. Larger offspring sizes are thought to have evolved as an adaptation to the competitive environment faced by newborn guppies in some environments. If plastic responses to maternal food limitation can achieve the same fitness benefit, then why has guppy offspring size evolved at all? To explore this question, I examined the plastic response to food level of females from two natural populations that experience different selective environments. My goals were to examine whether the plastic responses to food level varied between populations, test the consequences of maternal manipulation of offspring size for offspring fitness, and assess whether costs of plasticity exist that could account for the evolution of mean offspring size across populations. In each population, full-sib sisters were exposed to either a low- or high-food treatment. Females from both populations produced larger, leaner offspring in response to food limitation. However, the population that was thought to have a history of selection for larger offspring was less plastic in its investment per offspring in response to maternal mass, maternal food level, and fecundity than the population under selection for small offspring size. To test the consequences of maternal manipulation of offspring size for offspring fitness, I raised the offspring of low- and high-food mothers in either low- or high-food environments. No maternal effects were detected at high food levels, supporting the prediction that mothers should increase fecundity rather than offspring size in noncompetitive environments. For offspring raised under low food levels, maternal effects on juvenile size and male size at maturity varied significantly between populations, reflecting their initial differences in maternal manipulation of offspring size; nevertheless, in both populations, increased investment per offspring increased offspring fitness. Several correlates of plasticity in investment per offspring that could affect the evolution of offspring size in guppies were identified. Under low-food conditions, mothers from more plastic families invested more in future reproduction and less in their own soma. Similarly, offspring from more plastic families were smaller as juveniles and female offspring reproduced earlier. These correlations suggest that a fixed, high level of investment per offspring might be favored over a plastic response in a chronically low-resource environment or in an environment that selects for lower reproductive effort.

Key words.—Adaptive phenotypic plasticity, food limitation, life history, maternal environmental effects, propagule size, reproductive effort, resource availability.

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Interest in plasticity has grown to the point where demonstrating adaptive phenotypic plasticity is the primary goal of many studies (Gotthard and Nylin 1995). Nevertheless, understanding the nature of the adaptation and how plasticity evolves is still often elusive. In some cases, such as induced morphological defenses against predators (Lively 1986) or the acceleration of development in response to ephemeral habitats (Morey and Reznick 2000), the adaptive value of the plastic response is quite evident. However, one of the most prevalent forms of environmental heterogeneity, variation in resource availability, usually produces dramatic changes in phenotype whose adaptive significance remains unclear.

Life-history traits are especially plastic in response to quantitative changes in resource availability, yet this plasticity is mostly interpreted as a passive, physical response to environmental variation rather than as an adaptation (Smith-Gill 1983). Indeed, the adaptive value of lower growth or reproduction in response to food limitation is often difficult to determine (but see Calow and Woolhead 1977; Blanckenhorn 1998). Food limitation can also have cross-generational effects. Resource-limited mothers often produce smaller offspring (in plants: Donohue and Schmitt 1998; arthropods: Fox and Czesak 2000; mammals: Jones and Friedman 1982). But, several studies have shown that resource limited mothers produce larger offspring (e.g., Brody and Lawlor 1984; Schmitt et al. 1992).

Although producing larger offspring often comes at the cost of producing fewer offspring, this fecundity cost may be outweighed if larger offspring have increased fitness (Smith and Fretwell 1974; Lloyd 1987). Offspring size is thought to have a greater effect on offspring fitness in low-resource or highly competitive environments, thus affording larger offspring a greater selective advantage and increasing the optimal offspring size in low-resource environments (Brockelman 1975; Sibly and Calow 1982, 1983; Parker and Begon 1986; Winemiller and Rose 1993). Therefore, if mothers respond to reduced food availability by producing larger offspring, then this environmental maternal effect may represent adaptive plasticity (Mousseau and Fox 1998).

While adaptive plasticity is frequently claimed when the change in phenotype makes sense from an adaptive perspective, any putative case of adaptive plasticity requires empirical evaluation for the adaptive hypothesis to be upheld (Newman 1992). In fact, even highly plausible adaptive-plasticity

[1] Present address: 1001 East Third Street, 142 Jordan Hall, Department of Biology, Indiana University, Bloomington, Indiana 47405; E-mail: fbasheyv@indiana.edu.

hypotheses have been shown to be unsupported when evaluated with well-designed and statistically powerful empirical tests (e.g., Winn 1999). Moreover, empirical tests that have supported the adaptive-plasticity hypothesis have revealed complexities that were previously unrecognized (e.g., Dudley and Schmitt 1996; Spitze and Sadler 1996). Thus, it is necessary to test the hypothesis that the larger offspring produced by food-limited mothers is an adaptive response to the low-food environment, particularly because larger offspring often fail to have a fitness advantage in more competitive conditions (Kaplan 1985; Ruohomaki et al. 1993; Svensson et al. 2001). To date, only a few examples of adaptive maternal effects mediated by offspring size have been clearly demonstrated (e.g., Gliwicz and Guisande 1992; Fox et al. 1997; Donohue and Schmitt 1998).

If phenotypic plasticity is an effective means of enabling an organism to appropriately match its phenotype to its environment, then why isn't adaptive plasticity ubiquitous? The evolution of all traits can be limited by functional constraints or genetic correlations with other traits (Arnold 1992). We usually think of trait correlations within a single environment, but when traits are plastic it is necessary to look at these relationships across environments (Via and Lande 1985). In fact, a more holistic view of trait evolution is one that examines reaction norms in a context where environment-specific selection pressures and relationships among traits can be evaluated (Travis 1994; Schlichting and Pigliucci 1998). In addition, correlations between plasticity itself (independent of the actual trait value) and other traits can exist. When these correlations result in a plastic organism having a lower fitness than a nonplastic organism while both are exhibiting the same trait value, this cost of plasticity can restrict the evolution of adaptive plasticity (DeWitt et al. 1998). Furthermore, with a trait such as offspring size, which is subject to selection in both maternal and offspring generations, it is necessary to measure the fitness consequences and functional relationships among traits in both generations (Kirkpatrick and Lande 1989).

In this study, I examine the interplay of selection and functional constraints on the evolution of phenotypic plasticity of offspring size in the Trinidadian guppy, *Poecilia reticulata*. Female guppies have been shown to grow less, reproduce less, and bear fewer, larger offspring in response to food limitation (Reznick and Yang 1993). Interestingly, this plastic difference in offspring size is of the same order of magnitude as evolved genetic differences among populations (Reznick 1982; Reznick and Bryga 1987, 1996). Genetic differences in guppy offspring size are associated with variation in the community of predators to which guppies are exposed. However, the competitive environment experienced by guppies also varies with predation regime (Reznick et al. 2001), and recent evidence suggests that differentiation in guppy offspring size is consistent with local adaptation to the competitive environment (F. Bashey, unpubl. ms.). The similarity between plasticity and genetic differences in this system provides a good opportunity to study how traits evolve as reaction norms. Specifically, if mothers are able to alter the size of their offspring in response to food limitation, then why do fixed genetic differences in offspring size evolve when guppies are exposed to more competitive environments?

To explore this question, I examined plasticity in offspring size in response to maternal food level and the consequences for offspring fitness for guppies from two natural populations that experience different selective environments. My goals were to examine whether the plastic responses to food level varied between populations, test the consequences of maternal manipulation of offspring size for offspring fitness, and assess whether costs of plasticity exist that could account for the evolution of mean offspring size across populations rather than a plastic response to resource competition. In each population, full-sib sisters were exposed to either a low- or high-food treatment. For each of these mothers, I examined how investment per offspring varied as a function of maternal size and fecundity at both the family and population levels. I then tested the consequences of maternal investment per offspring on offspring fitness by raising the offspring of low- and high-food mothers in either low- or high-food environments. Furthermore, I examined whether the magnitude of the plastic response itself affected components of maternal and offspring fitness by examining correlations between plasticity in investment per offspring and maternal and offspring fitness components.

Materials and Methods

Study System

Guppies (*P. reticulata*) are small, livebearing fish found natural habitats that can be classified dichotomously into high- and low-predation communities (for reviews, see Endler 1995; Reznick et al. 2002). The competitive environment experienced by guppies also varies with predation regime (Reznick et al. 2001). Low-predation sites are upstream of high-predation sites; they tend to be smaller streams, with more canopy cover and lower levels of light and primary productivity. In addition, due to the lower level of predation, guppies maintain a higher biomass per unit area at these sites and, thus, experience lower somatic growth rates (Reznick et al. 2001).

Offspring size, litter size, and age and size at maturity have been shown to evolve in response to experimental changes in predation regime (Reznick et al. 1990). In low-predation sites, guppies produce litters that are 40% smaller in total mass than those from high-predation sites, yet they produce individual offspring that are 50% larger in dry weight (Reznick and Endler 1982; Reznick and Bryga 1987; Reznick et al. 1996c). Common garden experiments on F_2 guppies show a 20% difference in litter mass and a 15% difference in offspring mass, indicating a heritable genetic component to these traits and a significant effect of the environment (Reznick 1982; Reznick and Bryga 1996). In addition, food-limited females produce litters that are almost 30% smaller and offspring that are almost 20% larger than high-food females (Reznick and Yang 1993). Thus, environmental effects on offspring size are of the same order of magnitude as evolved genetic differences.

Source of Experimental Animals

The guppies used in this study were derived from two natural populations from streams in the Northern Range Mountains, Trinidad. One population is from a tributary (grid reference: PS 842 894) of the Marianne River (MT), which is a resource-limited, low-predation population (Reznick et al. 1996c; Grether et al. 2001), and the other is from the Aripo River (AR; PS 942 777), which is a high-predation, high-resource population (Reznick and Endler 1982; Reznick et al. 1990). Approximately 20 adult females and males were collected from each locality in September 1997.

Environmental and maternal effects on plasticity were controlled by raising individuals in a common environment for two generations. Female guppies store sperm, so the offspring in one litter can be sired by more than one male. Wild-caught female guppies were isolated in 8-L aquaria, and offspring were kept as separate lineages for future crosses. F_1 offspring were reared in densities of up to six per 8-L aquarium until they were approximately three weeks old. At that age, immature males were separated from immature females and kept at densities of two per 8-L aquarium. At approximately two to three months of age, one F_1 female per lineage was mated with a unique, wild-caught male or an unrelated F_1 male. To ensure enough F_2 offspring of each sex, up to 16 siblings were kept at densities up to eight per 8-L aquarium. At approximately 30 days, of age, F_2 offspring were sexed and up to six females and two males were kept from each litter. F_2 siblings were housed in single-sex pairs in 8-L aquaria until approximately two to three months of age. Nineteen F_2 families were reared from the MT population and 17 from the AR population.

Maternal Food Manipulation Experiment

The maternal food environment was manipulated to determine whether plasticity in offspring size varies across families and between populations. For each population, two sisters from each family were randomly assigned to a high-food treatment and two to a low-food treatment. The four F_2 sisters from each family were all mated to the same F_2 male from an unrelated lineage. Females were isolated and fed ad libitum until they gave birth to their first litter. After their first litter, females were started on quantified food levels. Females on the low-food treatment were fed 5 μl of liver paste in the morning and 10 μl of brine shrimp nauplii in the evening, whereas the high-food females were fed 30 μl liver paste and 40 μl of brine shrimp. The high-food fish received five times the energy content as low-food fish (404.26 vs. 81.13 joules per day).

Because guppies fully provision their young in the first five or six days after the birth of their previous brood (Reznick and Yang 1993), characteristics of their second litter largely represent the ad libitum feeding that the females received prior to the birth of the first litter. Characteristics of the third litter represent the effect of the different food levels that the females received after the birth of the first litter (Fig. 1a; Reznick and Yang 1993). Females were mated to their original male after the birth of each litter.

The offspring from the third litter were counted; a subset was preserved in 5% formalin and a subset was used in the offspring food manipulation experiment (see below). Up to 10 individual offspring per female were measured in standard length (SL, to 0.01 mm). Offspring were then dried at 60°C for four days and weighed individually (to 0.001 mg). Dried offspring were then extracted twice with ethyl ether and reweighed. Fat content was measured as the difference between the dried and lean weights of the offspring. Lean offspring were then ashed at 550°C for 8 h; ashing combusts the organic content of a specimen, which in this case was assumed to be protein. Energetic content was calculated by converting the fat and protein content to joules (Kleiber 1975). I define maternal investment per offspring as the mean energetic content of individual offspring in a female's third litter.

Mothers were weighed (to 0.0001 g) after the birth of each litter. After the birth of the third litter, females were preserved in 5% formalin. Females were later dissected and the energetic content of their ovaries and soma were determined by indirect calorimetry following the procedure used for the offspring, except that three ether extractions were performed.

Offspring Food Manipulation Experiment

To determine whether the fitness of offspring was affected by the environment of their mother, I reared offspring of low- and high-food mothers under low- and high-food conditions. Although two sisters were exposed to each maternal food level, the offspring of only one sister per maternal food level were raised (Fig. 1b). Offspring from a female's third litter were divided into low- and high-food treatments. Offspring were raised with their littermates at densities of four individuals per 8-L aquarium. Fish were fed with either brine shrimp or liver paste two times per day. High-food fish were given approximately twice as much food (on a joule per gram basis) as the low-food fish, so that one treatment was maintained just below ad libitum and the other was just above the minimal requirement to sustain growth. Food levels were increased weekly, in an approximately quadratic fashion to accommodate growth. Low food levels started at 1.03 joules per fish and were capped after 12 weeks at 40.57 joules per fish; high food levels started at 12.23 joules per fish and were capped after 18 weeks 404.27 joules per fish. Food levels were reduced when a death occurred in a tank, to maintain a constant per capita food level.

Juvenile guppies were measured weekly until offspring were large enough for the sexes to be distinguished. Sexes could be distinguished at three weeks of age in the high-food treatment, but not until eight weeks for in the low-food treatment. I then kept one male and one female per tank until they reached sexual maturity (males) or gave birth to their first broods (females). Brother and sister were housed together and measured weekly. An unrelated adult F_2 male was added to the tank to allow the female access to mature sperm. Males were defined as mature based on the development of the anal fin (Turner 1941). As males approached maturity they were observed daily; upon morphological maturity (but before sperm maturation), they were measured and preserved, thereby preventing mating between siblings. The females were kept singly until they gave birth to their first litter. The female and her F_4 offspring were measured and then preserved.

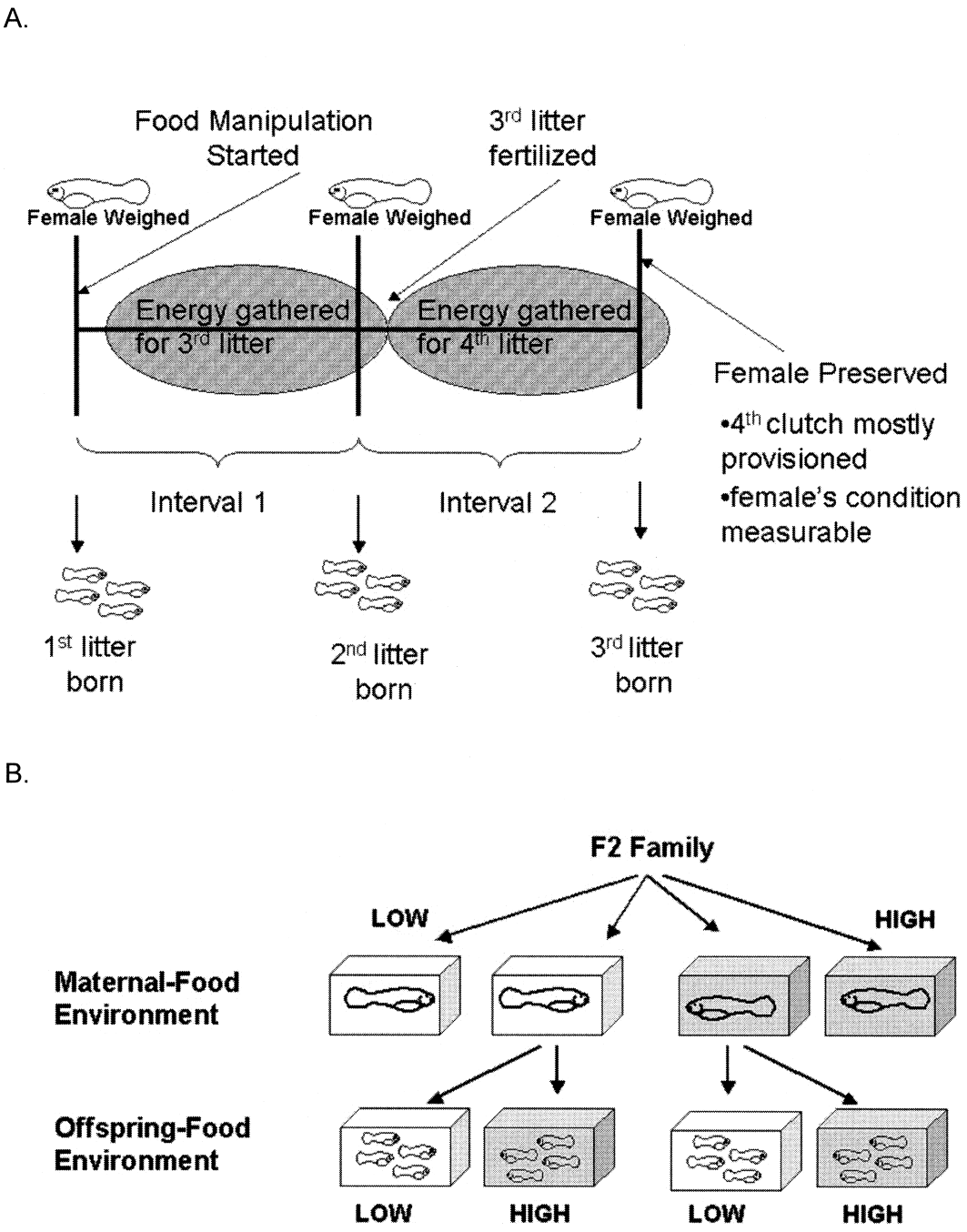

FIG. 1. Schematic representations of (a) the reproductive cycle of the guppy over the time-course of the experiment and (b) the experimental design of the study. A total of 36 F_2 families were reared from two natural guppy populations. Mothers were exposed to either a high- or low-maternal-food treatment. Offspring of 138 females were preserved at birth for measurement of reproductive output and offspring size. For each family, offspring of the third litter for one female from each maternal food level were split between a high- and low-offspring-food treatments and raised until reproductive maturity.

Statistical Analyses

Unless otherwise specified, all analyses were conducted using the mixed procedure in SAS version 8.2. (Littell et al. 1996). Population and maternal food level were considered fixed factors, while family nested within population and its interactions were considered random effects. Restricted maximum likelihood was used to estimate variance components. Below I describe the analyses performed in more detail.

Initial offspring characteristics

Analyses of variance (ANOVAs) were performed on the mean SL, mass (dry, lean, and lipid), and energetic content of offspring in a female's third litter. Offspring from the AR population were slightly more variable, so separate error variances were used for each population (Milliken and Johnson 2002). Variance components were estimated from one-way ANOVAs for each population and maternal food level and

used to calculate broad-sense heritabilities of offspring energetic content: $H^2 = 2\sigma^2_{\text{family}}/(\sigma^2_{\text{family}} + \sigma^2_{\text{error}})$. The genetic correlation of offspring energetic content across maternal food levels was estimated as $r^2_G = \sigma^2_{\text{family}}/(\sigma^2_{\text{family}} + \sigma^2_{\text{family}\times\text{food}})$ and the heritability of plasticity in offspring energetic content as $H^2_{\text{plasticity}} = 2\sigma^2_{\text{family}\times\text{food}}/(\sigma^2_{\text{family}\times\text{food}} + \sigma^2_{\text{error}})$ using variance components from two-way ANOVAs performed separately for each population (Roff 1997; Lynch and Walsh 1998). I examined how both offspring number and female body size (wet mass after the birth of the second litter) affected offspring energetic content by using both as covariates in a mixed-model ANCOVA with population and maternal food as main effects.

Offspring survival

I analyzed the probability of surviving the juvenile period using the GLIMMIX macro of SAS, which allows random effects to be included in logistic regression analyses (Littell et al. 1996). I first performed a joint analysis examining the effect of population and maternal food, and then analyzed each population separately to see whether initial offspring characteristics (energy content, lean, and lipid mass) increased offspring survival.

Offspring growth

I performed a repeated-measures, mixed-model ANOVA on the average size (ln[mass]) of offspring in each tank. I performed separate analyses for each offspring food level because at the high food level the juvenile period lasted only three weeks, whereas at the low food level the juvenile period lasted eight weeks. In each analysis, population and maternal food level were considered between-subject effects and time was the within subjects effect. I estimated the error variance for each population separately (Milliken and Johnson 2002). I also examined the relationship between initial offspring characteristics and offspring size over the juvenile period for each population separately.

Offspring maturity

I examined male size (wet mass) and age (in days) at maturity and female size, age, and number of offspring at first reproduction using mixed-model ANOVAs. Because competition between males and females within a tank could affect maturity, relative size at the end of the juvenile period was evaluated as an additional, fixed factor in these analyses of offspring maturity. I performed separate analyses for each offspring food level.

Correlates of plasticity in investment per offspring

Plasticity in investment per offspring was calculated by subtracting the family mean investment per offspring at high maternal food level from the family mean at low maternal food level. Costs of plasticity are defined as negative partial regression coefficients for plasticity from a multivariate regression of trait values and plasticity on fitness (DeWitt 1998). This definition controls for differences in fitness due to the expressed phenotype and isolates the independent effect of plasticity. I evaluated whether plasticity for investment per offspring had any effects on fitness components in both maternal and offspring generations by using individual values for investment per offspring and fitness components, coupled with the family-based measure of plasticity. This approach, dubbed ''the Van Tienderen model'' by DeWitt (1998), is analogous to a full family-means model but is more powerful. In addition, family was used as a random effect in all analyses.

In the mothers, I examined the effect of plasticity on somatic investment and on investment in future reproduction by looking at the energetic content of maternal somatic and reproductive tissues after the birth of the third litter, by which point, the female has mostly invested in her fourth litter (Fig. 1a). I examined the effect of plasticity on somatic investment by a mixed-model ANCOVA on somatic investment with population as a main effect and maternal weight at the second litter, investment per offspring, and its plasticity as covariates. I used maternal weight as a covariate because I did not want the association between maternal size and offspring size to confound the examination of whether plasticity per se had an effect on the future condition of the female. I examined the effect of plasticity on future reproductive investment by the mother by a mixed-model ANCOVA on reproductive investment with population as a main effect and maternal somatic investment (to control for the known association between female size and fecundity), investment per offspring, and its plasticity as covariates. Separate analyses were performed for each maternal food level.

I examined the effect of plasticity on the following fitness components of the offspring: survival over the juvenile period, offspring size over the course of the juvenile period, and male and female characteristics of maturity. In each case, I performed analyses of the same type as described for the maternal effects analyses: logistic regression for survival, repeated-measures ANCOVA for juvenile weight, and univariate ANCOVAs for offspring maturity. Each analysis used population as a discrete, fixed factor, family as a random effect, and investment per offspring and its plasticity as regression variables. In each case, I ran separate analyses for each offspring food level.

RESULTS

Effects of Maternal Environment on Maternal Investment in Offspring

Low-food mothers produced offspring that were longer in SL, heavier in dry and lean mass, and had a lower lipid mass than offspring of high-food mothers (Table 1, Fig. 2). AR offspring were larger than MT offspring in all of these traits. None of the interactions between maternal food level and population were significant. However, the populations differed in the magnitude of their response, with AR having a greater increase in SL (2.7% vs. 1.4%), dry mass (6.0% vs. 2.6%), and lean mass (9.2% vs. 6.1%) in response to low maternal food and MT having a greater increase in lipid mass in response to high maternal food (12.9% vs. 7.6%). Thus, while the energetic content of AR offspring increased in the low-maternal-food treatment, the energetic content of MT offspring did not (Fig. 2c).

Broad-sense heritabilities for offspring energetic content

TABLE 1. Fixed effects from mixed-model analyses of variance of offspring characteristic at birth. *F*-values are given for each effect with the corresponding *P*-value below each. Family (population) and the family (population) × maternal food interaction were considered as random effects in each analysis.

	df	Standard length (mm)	Dry mass (mg)	Lean mass (mg)	Lipid mass (mg)	Energy (joules)
Population	1,33	58.72	11.65	14.65	3.17	9.88
		<0.0001	0.0017	0.0005	0.0841	0.0035
Maternal food	1,33	17.11	4.63	16.19	6.50	0.66
		0.0002	0.0388	0.0003	0.0156	0.4234
Population × maternal food	1,33	2.01	0.85	1.03	0.25	0.79
		0.1653	0.3634	0.3176	0.6199	0.3800

were high, and there was a significant effect of family for the AR population at both food levels (Table 2). In the MT population, heritabilities at each food level were lower and family effects not significant. However, offspring energetic content was tightly correlated across environments in the MT population, indicating a strong family component to offspring energetic content and negligible genetic variance for plasticity. In the AR population, offspring energetic content was still highly correlated across environments, yet there was more (albeit not significant) variation among families in their plastic response to food level (Table 2).

When maternal body size and the number of offspring produced were used as covariates in the analysis of offspring energetic content, populations expressed different functional relationships for both covariates (Fig. 3), as indicated by significant heterogeneity of slopes between populations. In other words, populations differed significantly in the relationship between maternal weight and offspring energetic content ($F_{1,31} = 6.48$, $P = 0.0035$) and in the relationship between offspring energetic content and offspring number ($F_{1,32} = 9.95$, $P = 0.0035$). As females from the AR population grew larger, they invested more energy in each individual offspring (Fig. 3a; $F_{1,14} = 17.11$, $P = 0.0010$), whereas females from MT did not (Fig. 3b; $F_{1,17} = 0.37$, $P = 0.5526$). Furthermore, MT females did not show a trade-off between offspring energetic content and offspring number (Fig. 3d; $F_{1,17} = 0.40$, $P = 0.5347$). In contrast, AR females invested more per offspring at the expense of offspring number (Fig. 3c; $F_{1,15} = 34.33$, $P < 0.0001$), and this trade-off was more pronounced under low-food conditions (maternal food × offspring number: $F_{1,14} = 8.70$, $P = 0.0105$).

Maternal Environmental Effects on Offspring Life History

Offspring survival

No natural mortality occurred in the high-offspring-food treatment. In the low-offspring-food treatment, the maternal food environment had no significant effect on offspring survivorship ($F_{1,31} = 1.72$, $P = 0.1989$). Offspring from the AR population did have a greater probability of surviving the juvenile period than offspring from the MT population ($F_{1,31} = 7.24$, $P = 0.0114$). Within each population there was no significant relationship between offspring survival and initial offspring characteristics.

Offspring growth

The effect of the maternal food environment on juvenile size depended on the offspring food level and the population (Table 3). Under high-offspring-food conditions, there was no detectable effect of the maternal food environment on offspring size over the juvenile period, nor did the populations differ in their maternal effects. In contrast, under low-offspring-food conditions, populations varied significantly in how the maternal environment affected juvenile size (Fig. 4a). In fish from the AR population, offspring whose mothers were kept under low-food conditions tended to achieve a greater mass than offspring whose mothers were kept under high-food conditions. In contrast, in fish from the MT population, offspring whose mothers were kept under low-food conditions tended to be smaller than their counterparts.

In both populations, initial energetic content of offspring had a positive effect on juvenile size when offspring were raised under low-food conditions, suggesting that differences in maternal investment mediated the difference in maternal effects. Populations did not differ significantly in how increased maternal investment in offspring affected juvenile size; however, the relationship was only significant in the AR population (AR: $F_{1,14} = 13.06$, $P = 0.0028$; MT: $F_{1,16} = 2.98$, $P = 0.1036$). The weaker association in MT population is perhaps due to the strong association of both lean and lipid mass with juvenile size in the AR population (lean mass: $F_{1,15} = 11.32$, $P = 0.0043$; lipid mass: $F_{1,15} = 7.62$, $P = 0.0146$), but a strong association of only lean mass in the MT population (lean mass: $F_{1,17} = 5.95$, $P = 0.0260$; lipid mass: $F_{1,17} = 0.02$, $P = 0.8885$).

Offspring maturity

The effect of the maternal food environment on the maturity of male offspring paralleled the influence of the maternal food environment on juvenile size (Table 4). At the high offspring food level, there were no significant effect of the maternal food environment on male maturity, nor was there difference between populations in how the maternal food environment affected male maturity. In contrast, at the low offspring food level, populations varied significantly in how the maternal food environment affected male size at maturity (Fig. 4b). Males from the AR population whose mothers were kept at low food tended to be larger at maturation than offspring whose mothers were kept at high food. Males from the MT population showed an opposite and smaller effect of the maternal food environment. Despite the low food level, no significant maternal effects or population by maternal food interactions were detectable on male age at maturity, suggesting that males were able to gain in size without a delay in maturation (Table 4).

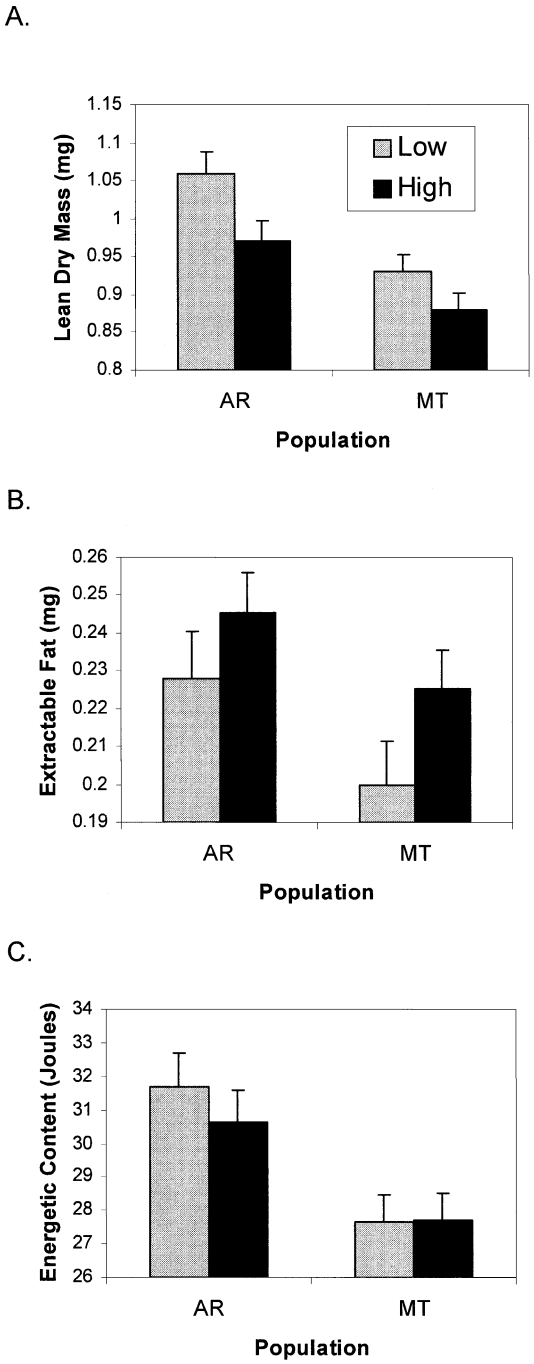

FIG. 2. Mean (±1 SE) (a) lean mass, (b) lipid mass, and (c) energetic content of offspring at birth from the mixed-model ANOVAs presented in Table 1 shown as a function of maternal food level and population. Both populations produce larger, leaner offspring in response to low food levels. However, in the MT population these responses balance each other out such that offspring energetic content does not vary with maternal food level, while in the in the AR population low-food mothers invest more per offspring.

No significant maternal effects were observed on female age, size, and number of offspring at first reproduction. Nevertheless, under low offspring food levels, females (from either population) who were larger at the end of the juvenile period matured significantly earlier than those who were smaller ($F_{1,31} = 5.96$, $P = 0.0206$). This suggests that even though the maternal food environment did not have a detectable direct effect on female maturity, it could have a cascading influence via juvenile size. At high offspring food levels, size at the end of the juvenile period did not have a significant effect on female age at maturity ($F_{1,32} = 0.22$, $P = 0.6399$).

Correlates of Plasticity

Maternal generation

Plasticity in investment per offspring was correlated with the condition of the mother at the end of the experiment (Table 5). At low food levels, females from more plastic families had a significantly lower somatic investment (Fig. 5a), suggesting that plasticity comes at a cost of maternal condition. However, these same families show a significantly higher investment in future reproduction at low food levels (Fig. 5b), indicating that, to increase investment per offspring in response to a low-food environment, females must also increase reproductive effort. At high food levels, females from more plastic families invested more in soma and less in future reproduction, but these patterns were not significant (Table 5). Populations did not differ in the relationships between plasticity and maternal investment.

Offspring generation

Two offspring fitness components were significantly affected by the level of plasticity: juvenile size and female maturity. Families that had a greater level of plasticity produced offspring that were relatively smaller in juvenile size under the low-offspring-food treatment (Fig. 5c, $F_{1,31} = 5.46$, $P = 0.0260$). This effect of plasticity opposes the direct effect increasing investment per offspring has of increasing juvenile size. In addition, female offspring from more plastic families tended to reproduce earlier and at a smaller size (Table 6, Fig. 5d). Populations did not differ from each other in the relationships between plasticity and female maturity or juvenile size; however, only the AR population showed significant effects. These results from the offspring generation are in concordance with those seen in the maternal generation, both suggesting that plasticity is positively correlated with reproductive effort and negative correlated with somatic condition.

DISCUSSION

This study demonstrates that changes in maternal investment per offspring in response to the maternal environment can affect components of offspring fitness. These fitness effects depend on the offspring environment and conform to predictions of the adaptive plasticity hypothesis. Moreover, the functional relationships underlying these plastic shifts in offspring size vary between populations. Plasticity in offspring size was found to be correlated with traits associated with higher reproductive effort. Below, I first discuss aspects of my study that appear contrary to earlier studies. I then discuss the populational variation in plasticity and its consequences for offspring fitness. Finally, I conclude by pro-

TABLE 2. Broad-sense heritabilities of offspring energetic content, its genetic correlation across environments, and the heritability of plasticity in offspring energetic content. Values were calculated as explained in the methods. In parentheses beside each value, the denominator degrees of freedom and *P*-values are given for the *F*-test associated with the corresponding factor (family or family × food).

Population	Heritability		r_G	$H^2_{plasticity}$
	Low food	High food		
AR	0.88 (16, 0.0355)	0.93 (16, 0.0276)	0.82 (16, 0.0196)	0.24 (32, 0.2285)
MT	0.027 (17, 0.3799)	0.63 (18, 0.1071)	1.00 (17, 0.0001)	0.00 (35, 0.9837)

posing how offspring size and its plasticity might have evolved across populations of guppies.

Generality of Population and Maternal Environment Effects on Offspring Size

In apparent contradiction to the accepted pattern, offspring from the low-predation population in this study (MT) were smaller than those from the high-predation population (AR; Fig. 2). The most likely cause of this is the geographic source of these populations: the MT population is from the north slope of the Northern Range Mountains, while the AR population is from the south slope. Although, low-predation guppies from both slopes have larger offspring than their counterparts from high-predation locales, south-slope guppies have larger offspring than similarly sized guppies from the north slope (Reznick and Bryga 1996). Both the MT and AR populations have been studied previously (see Materials and Methods for references) and can be considered good representatives of low- and high-predation locales, respectively (D. Reznick, pers. comm.). The goal of this study was not to reestablish mean differences in offspring size, but rather to examine patterns of plasticity in offspring size among populations.

Females guppies from both populations responded to food limitation by making larger, leaner offspring (Fig. 2). These results contrast earlier work on a third population of guppies (a laboratory stock derived from a high-predation locale) that found female guppies made larger, fatter offspring in response to food limitation (Reznick and Yang 1993). However, Reznick and Yang did not report offspring fat content, rather they assumed that because offspring lean weight did not significantly differ between low- and high-food mothers, but dry weight did, that these offspring must be fatter. Subsequent reexamination of Reznick and Yang's data has shown this conclusion to be incorrect (D. Reznick, pers. comm.). Reznick and Yang (1993) did not include maternal weight as a covariate in their analysis of offspring lean weight but did in their analysis of offspring dry weight. Maternal weight significantly and positively affected offspring lean weight in their study ($F_{1,33} = 5.52$, $P = 0.0250$), and when included in the analysis, low-food mothers produced offspring that were larger in lean weight ($F_{1,33} = 6.07$, $P = 0.0192$). Moreover, analyses of offspring lipid mass showed that there is no significant difference in the fat content of low- and high-food mothers in their study (with covariate: $F_{1,33} = 2.48$, $P = 0.1249$; without covariate: $F_{1,34} = 1.17$, $P = 0.2871$). A comparison of all three populations shows an overall similarity of response: larger, leaner offspring in response to low food.

Theoretical models assume that larger offspring have an environment-specific fitness advantage over smaller offspring and thus predict that optimal offspring size should be larger in more competitive environments (Brockelman 1975). While plasticity of offspring size in guppies is in the adaptive direction, other members of the family Poecilidae do not appear to share this trait. For example, *Poecilia latipinna* and *Heterandria formosa* did not alter offspring size or make smaller offspring, respectively, in response to food limitation (Reznick et al. 1996b; Trexler 1997). In contrast, *Priapichthys festae*, like guppies, produced larger offspring in response to low food (Reznick et al. 1996b). *Poecilia latipinna* and *H. formosa* differ in the timing of maternal provisioning relative to *P. reticulata* and *P. festae*; the former display matrotrophy, meaning that mothers continue to provision offspring after fertilization, while the latter stop provisioning prior to fertilization. Reznick et al. (1996b) suggested that matrotrophic females might be unable to adaptively increase offspring size in response to food limitation because of physiological constraints of their reproductive mode, while Trexler (1997) proposed that the relationship between offspring size and fitness might be more variable and less predictable in these matrotrophic species. While more work is clearly needed to examine the interplay between reproductive mode and adaptive plasticity in offspring size, recent work on *H. formosa* indicated that mothers can increase the size of their offspring in response to high densities, and that this response positively increased fitness of offspring reared at high densities (F. H. Rodd, J. Leips, J. M. L. Richardson, and J. Travis, unpubl. ms.). Thus, while reproductive physiology might constrain how a female can respond to environmental heterogeneity, these responses can undoubtedly be molded by selective forces.

Populational Variation in Plasticity and the Consequences for Offspring Fitness

Although mothers from both populations increased offspring lean mass and decreased offspring lipid mass in response to low food, the magnitude of these responses varied such that only in the AR population did investment per offspring increase in response to low food (Fig. 2c). In addition to the lower magnitude of plasticity in the MT population, guppies from the MT population exhibited less genetic variation for offspring energetic content and plasticity of offspring energetic content than those from AR (Table 2). While lower genetic variation could be indicative of lower overall genetic diversity in low-predation populations (Shaw et al. 1994), there is no a priori reason to suspect that low genetic diversity would affect the magnitude of the plastic response.

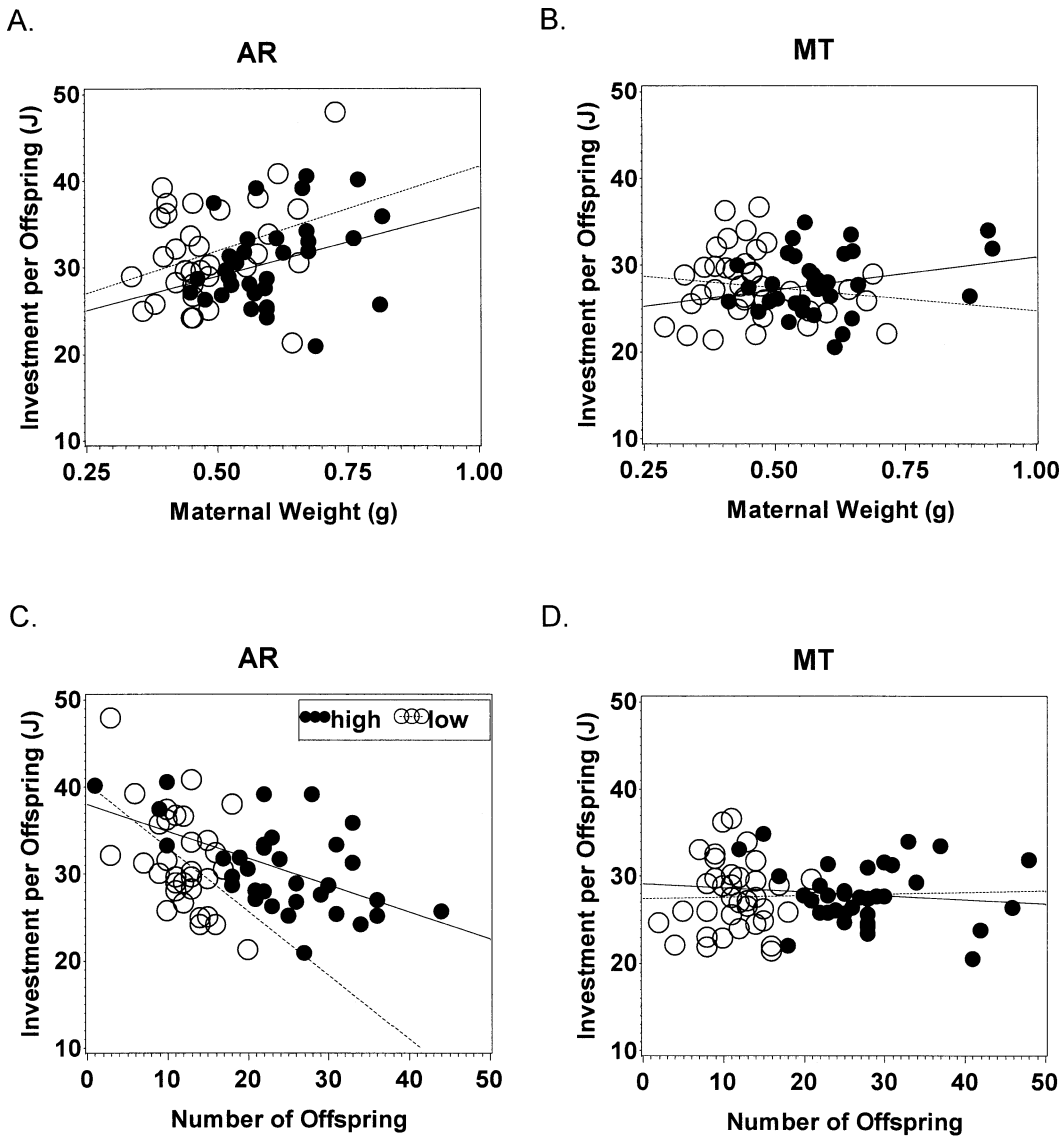

FIG. 3. Relationship between the energetic investment in individual offspring versus (a, b) maternal weight and (c, d) the number of offspring for each population and maternal food level. Populations are significantly different in these relationships. Although populations do not differ in total variation in investment per offspring, maternal weight and fecundity explain this variation in the AR population but not in the MT population.

TABLE 3. Fixed effects from mixed-model repeated-measures analysis of variance of offspring size as juveniles. Family (population) and family × maternal food were included as random effects. *F*-values are given for main effects with the corresponding *P*-value below each.

	Low offspring food		High offspring food	
	df	log(mass)	df	log(mass)
Population	1,32	4.39	1,31	0.47
		0.0001		0.4992
Maternal food	1,32	0.16	1,31	0.05
		0.6947		0.8295
Population × maternal food	1,32	6.64	1,31	0.28
		0.0148		0.6004
Time	6,506	1402.95	1,89	5190.08
		<0.0001		<0.0001

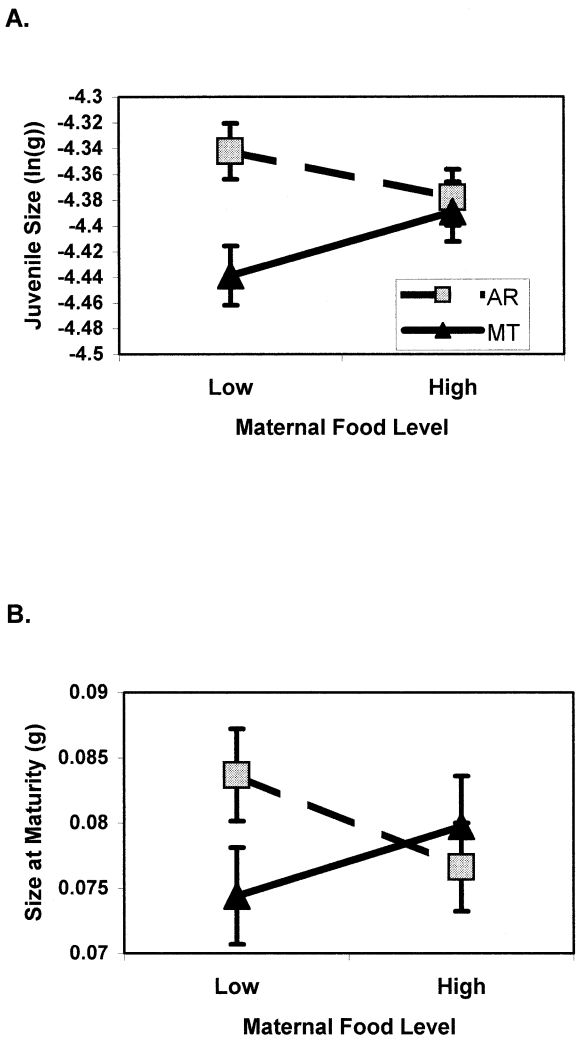

FIG. 4. The effect of the maternal food environment on (a) juvenile size and (b) male size at maturity for offspring from each population raised in a low-food environment. Least-square means (±1 SE) from the analyses presented in Tables 3 and 4 are given.

TABLE 4. Fixed effects from mixed-model analyses of variance of characteristics of maturity for male offspring. Family (population) was included as a random effect. *F*-values are given for main effects with the corresponding *P*-value below each. Relative size classified the size of each male relative to the size of his female sibling who shared a tank with him. The MT population was more greatly affected by relative size. The interaction between population and maternal food level tests for a difference in the effect of the maternal environment between populations.

		Male offspring at maturity	
	df	Weight	Age
Low offspring food			
Population	1,31	0.53	0.35
		0.4729	0.5608
Maternal food	1,27	0.08	0.11
		0.7733	0.7441
Population × maternal food	1,27	4.55	1.65
		0.0421	0.2096
Relative size	1,27	48.43	207.19
		<0.0001	<0.0001
Population × relative size	1,27	5.77	4.15
		0.0234	0.0516
High offspring food			
Population	1,33	0.99	2.86
		0.3270	0.1002
Maternal food	1,26	0.06	0.28
		0.8078	0.6021
Population × maternal food	1,26	0.66	0.13
		0.4246	0.7247
Relative size	1,26	8.13	28.62
		0.0084	<0.0001
Population × relative size	1,26	7.49	5.43
		0.0110	0.0278

The proportional change in offspring dry weight between low- and high-maternal-food treatments was more than twice as great in the AR population than in the MT population. Furthermore, populations differed in how investment per offspring varies with maternal weight and offspring number, indicating fundamental differences in reproductive physiology (Fig. 3).

The effects of the maternal environment on offspring size at birth affected the fitness of raised offspring. In support of the adaptive plasticity hypothesis, maternal effects on offspring fitness components varied across populations and environments. For offspring raised under high food levels, no maternal effects were detected, suggesting maternal fitness would be maximized by increasing fecundity rather than offspring size in noncompetitive environments. In contrast, under the low offspring food levels, the magnitude and direction of the effect of the maternal environment varied between the two populations (Fig. 4). In the AR population, offspring produced by low-food mothers and raised in the low-food treatment tended to be larger as juveniles than offspring produced by high-food mothers. In addition, male offspring of low-food mothers tended to be larger at maturity. These results support the adaptive plasticity hypothesis because larger juveniles have higher survivorship in the field (Reznick et al. 1996a; Bashey 2002). Furthermore, females from some populations prefer larger male guppies (Reynolds and Gross 1992), and larger males have been found to have higher escape velocities (Odell 2002).

In contrast, in the MT population, maternal effects on offspring fitness components were in the opposite direction (Fig. 4). Because mothers from the MT population did not increase their investment per offspring in response to low-food, the fact that offspring from low food mothers did not have increased fitness is not surprising. In fact, within each population investment per offspring was positively associated with juvenile size, suggesting that differences in investment per offspring is the cause of the maternal environmental effect on juvenile size.

To date, the studies that have demonstrated cross-generational adaptive plasticity via the manipulation offspring size have been mainly in invertebrates and plants (e.g., Gliwicz and Guisande 1992; Fox et al. 1997; Donohue and Schmitt 1998). In contrast, in vertebrates most forms of maternal effects have been shown to be the result of sacrificing offspring fitness to increase parental fitness (Price 1998). This difference maybe due to the longer period of parental care in many vertebrates leading to increased, optimistic invest-

TABLE 5. Effect of plasticity on maternal fitness components as determined from mixed-model analyses of covariance of maternal somatic and reproductive investment. Family (population) was included as a random effect. Fixed effects are given. No interactions were significant. At low maternal food, the slope of the relationships between plasticity and maternal fitness components are significantly different from zero (Fig. 5a, b).

	Low maternal food			High maternal food		
	df	*F*	*P*	df	*F*	*P*
Maternal somatic investment						
Population	1,31	0.00	0.9513	1,30	0.50	0.4846
Maternal weight	1,30	48.19	<0.0001	1,31	43.38	<0.0001
Investment per offspring	1,30	29.21	<0.0001	1,31	7.84	0.0087
Plasticity in investment per offspring	1,30	7.11	0.0122	1,31	3.18	0.0844
Maternal reproductive investment						
Population	1,31	0.27	0.6067	1,30	0.04	0.8408
Maternal soma	1,32	5.92	0.0207	1,33	24.46	<0.0001
Investment per offspring	1,32	0.15	0.7034	1,33	0.84	0.3663
Plasticity in investment per offspring	1,32	5.65	0.0236	1,33	1.25	0.2710

ment early in the reproductive season followed by sacrificing these young when conditions are worse than average. In fact, other vertebrates with minimal parental care do show adaptive increases in parental investment per offspring as offspring conditions deteriorate (Sinervo 1998; Rodd et al., unpubl. ms.). Thus, maternal manipulation of offspring size may be an important, general mechanism by which mothers can increase their fitness in systems with minimal parental care.

Evolution of Guppy Offspring Size and Its Plasticity

Historically, as guppies invaded upstream, low-predation sites from downstream, high-predation sites, they moved from a less to a more competitive environment. If guppies already possessed the ability to plastically respond to changes in the resource environment by increasing offspring size, why did guppy populations respond by genetically increasing offspring size? Furthermore, assuming that the lower level of plasticity in investment per offspring in the low-predation MT population, relative to the two other (high-predation) guppy populations studied so far, is indicative of differences in plasticity across predation regimes, why might the evolution of larger offspring be linked with a lower level of plasticity? Here, I discuss three mechanisms that could be affecting the evolution of offspring size and its plasticity in the guppy system: differential selection on the ability to plastically respond to changes in the resource environment, differential selection on offspring size resulting in changes in plasticity, and differential costs of plasticity.

Undoubtedly, differences in temporal and spatial heterogeneity of resource availability must exist across populations, so selection for different levels of plasticity most likely exist. However, whether selection for offspring size plasticity varies between predation regimes is currently unknown and in need of study. Nonetheless, the results from the current study parallel work in another system where adaptive divergence in plasticity between populations has been shown (Donohue et al. 2000). In woodland sites, selection always favors earlier flowering and longer internodes in *Impatiens capensis*, while in open-canopy sites, selection on these traits is density dependent. Thus, open-canopy populations have been selected to show a greater level of plasticity in response to density relative to the woodland populations. If a similar mechanism is operating in the guppy system, we would expect chronically low resource levels at the MT site that always favor larger offspring and higher, more variable resources at the AR site, which favor a smaller mean offspring size and increased plasticity.

In addition to selection on plasticity per se, plasticity can result from selection of different trait means in different environments (Falconer 1990; Via 1993). Environment-specific correlations between trait means and plasticity in plant morphology have been found and are thought to contribute to divergence of plasticity across populations (Donohue et al. 2000). In addition, a selection experiment on egg size in the seed beetle *Stator limbatus* found that selection for increased egg size resulted in increased plasticity; while the selection for decreased egg size resulted in decreased plasticity (M. E. Czesak and C. W. Fox, unpubl. data). Surprisingly, however, this response occurred in only one environment, suggesting different genetic correlations between egg size and its plasticity in the two environments. In the current study, correlations between family mean investment per offspring and plasticity in offspring investment were positive in the low-resource maternal environment ($r = 0.61$, $P = 0.0001$) and negative in the high-resource environment ($r = -0.34$, $P = 0.0457$). Therefore, in a low-resource environment, selection for increased offspring size will have the indirect effect of increasing the plastic response, while in a high-resource environment selection for decreased offspring size will indirectly increase the plastic response. This result suggests that differential selection on trait means across the predation regime could be important for maintaining the generalized plastic response seen across populations.

In contrast, correlates of plasticity seen in this study (Fig. 5) might explain why plasticity might differ between predation regimes. First, while larger investment per offspring increased juvenile size, offspring from more plastic families had lowered juvenile size in the low-offspring-food treatment. Coupled with selection for increased offspring size in low-resource environments, this negative relationship between plasticity and a component of juvenile fitness suggests that it would be selectively advantageous for families that encounter low-food environments frequently to have a fixed, high level of investment rather than to respond plastically,

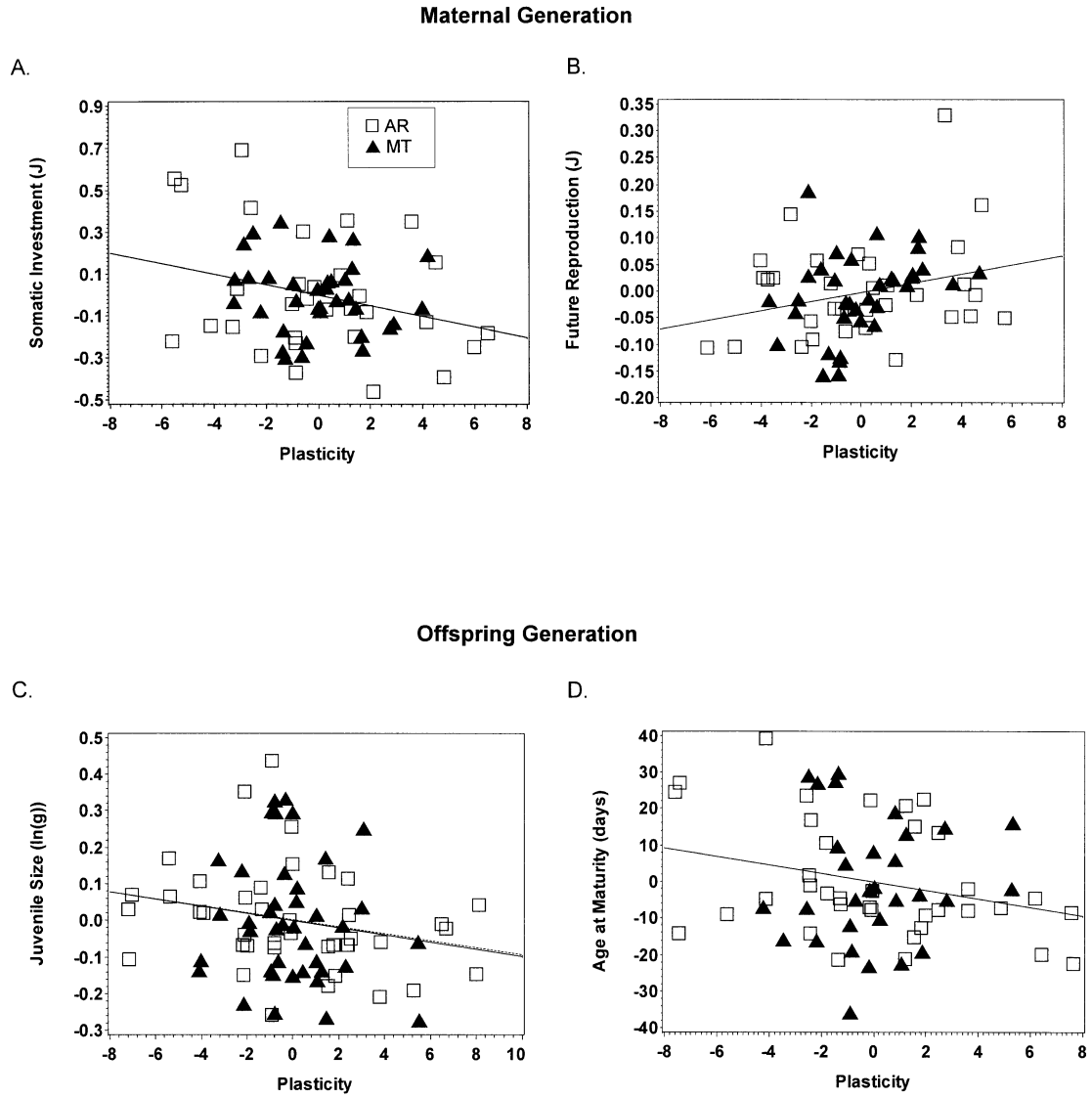

FIG. 5. Relationship between plasticity in investment per offspring and (a) maternal somatic investment at low maternal food, (b) maternal future reproductive investment at low maternal food level, (c) juvenile size of offspring raised in a low-food environment, and (d) age at maturity of female offspring raised in a low-food environment. Residuals are shown to control for the effect of the other factors in the analyses (e.g., population, family, investment per offspring) and isolate the relationship between plasticity and the dependent variable. The regression lines shown represent the slope of this relationship.

because a plastic response would incur a cost relative to the fixed response.

A second potential limit on the evolution of plasticity in this system may arise via a positive correlation between plasticity and reproductive effort (Fig. 5). More plastic families produced female offspring that matured earlier and at a smaller size, indicative of increased reproductive effort. Mothers from more plastic families also had increased allocation to reproduction and decreased allocation to growth in low-food environments. These correlations between plasticity and reproductive effort suggests that plasticity will be decreased in environments that favor decreased reproductive effort, such as is found in low-predation populations (Reznick et al. 1990). These correlations also suggest that one of the fundamental assumptions of optimal offspring size theory (e.g., Smith and Fretwell 1974), namely that maternal fitness is maximized by maximizing the number of surviving offspring in the current brood, may be incorrect. Despite the clear possibility of physiological integration between offspring size and other maternal life-history decisions, this assumption has received very little theoretical or empirical attention (but see Winkler and Wallin 1987; Caley et al. 2001; Czesak and Fox 2003).

In summary, this study demonstrated populations do differ in plasticity in investment per offspring in ways that can have fitness consequences for both mother and offspring. Mothers from the MT population showed less variation in investment per offspring in response to food limitation, maternal weight, and litter size than mothers from the AR population. In low-food environments, maternal environmental effects were found to influence offspring size throughout the juvenile period and characteristics of offspring maturity. These maternal

TABLE 6. Effect of plasticity on maturity of female offspring as determined by mixed-model analyses of covariance of the age and weight of female offspring at maturity. Family (population) was included as a random effect. *F*-values are given for fixed effects with the corresponding *P*-value below each. No interactions were significant. The relationship between plasticity and the age at maturity of female offspring raised under low food is depicted in Figure 5d.

		Female offspring at maturity	
	df	Age	Weight
Low offspring food			
Population	1,31	0.98	0.99
		0.3301	0.3266
Investment per offspring	1,31	0.00	0.42
		0.9492	0.5223
Plasticity in investment per offspring	1,31	4.51	6.25
		0.0418	0.0181
High offspring food			
Population	1,30	5.39	3.67
		0.0272	0.0648
Investment per offspring	1,32	1.05	0.80
		0.3133	0.3778
Plasticity in investment per offspring	1,32	5.39	4.53
		0.0267	0.0411

effects appear to be mediated by differences in initial investment per offspring. In addition, plasticity in investment per offspring was correlated with lower growth and higher reproductive effort in both maternal and offspring generations. Thus, the evolution of offspring size and its plasticity may not be affected only by selection balancing the tradeoff between offspring size and number of a given litter but may be subject to the influence of selection on maternal size and other maternal life-history decisions. A formal analysis of what offspring size maximizes maternal fitness, and whether this size should be achieved by plasticity will need to assess how offspring-size decisions and plasticity per se affect the life history of both the mother and her offspring. Further, this analysis would have to be performed separately for different environmental contexts.

Offspring size, a trait influenced both genotypically and by the maternal environment, may exhibit complex responses to selection (Kirkpatrick and Lande 1989). In guppies, the interaction between differential selection on mean offspring size across guppy populations and the effect of maternal food limitation on offspring size suggests future work on this system may afford insights into how traits evolve as reaction norms (Schlichting and Pigliucci 1998; Tufto 2000). As a first step, a study examining the plastic responses of several low- and high-predation populations, paired by drainage, is needed to assess whether the correlates of plasticity and the differences between populations seen in this study can be generalized. If these patterns hold, then the next, more difficult, step will be to characterize the temporal and spatial heterogeneity of the selective environment of several low- and high-predation populations. A useful approach would be studies that disentangle the predation environment from the resource environment (e.g., Grether et al. 2001). Finally, artificial selection and random mating experiments could be used to determine to what degree traits are functionally linked or in linkage disequilibrium due to past correlated selection (Lynch and Walsh 1998).

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