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QUANTITATIVE GENETIC AND OPTIMALITY ANALYSES OF LIFE-HISTORY PLASTICITY IN THE EASTERN MOSQUITOFISH, *GAMBUSIA HOLBROOKI*

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Life-history theory predicts that organisms should allocate resources to maintenance, growth, reproduction, and storage in such a way that fitness is maximized (Gadgil and Bossert 1970; Smith and Fretwell 1974; Brockelman 1975; Giesel 1976; Stearns and Crandall 1984; Stearns and Koella 1986; McGinley et al. 1987; Winkler and Wallin 1987). Although much life-history theory is framed in terms of population-level responses to different environments, similar expectations can be applied to individual-level responses that are the product of phenotypic plasticity (Via 1987). Modification of the phenotype according to the environment (i.e., phenotypic plasticity) should evolve in habitats where organisms encounter equally frequent, but variable patches, as long as the cost of this phenotypic plasticity is low, suitable genetic variation is available, and the environmental cues are highly correlated with the environment of selection (Via and Lande 1985; Schlichting 1986; Via 1987; Van Tienderen 1991; Gomulkiewicz and Kirkpatrick 1992; Scheiner 1993).

A number of life-history optimality models make testable predictions about the timing, level, and packaging of reproductive investment in environments that differ in food availability or "scope for growth" (for reviews, see Roff [1992] and Stearns [1992]). Phenotypic plasticity in age and size at maturity in high- relative to low-growth environments has been addressed in a variety of models, with a number predicting earlier age at maturity and increased size at maturity (Roff 1984, 1986), later age at maturity at a larger size (Ko-

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TABLE 1. Predictions of several optimality models relating growth environment to life-history traits.

Trait	Model	Growth environment	
		Low	High
Age at maturity	Roff 1984, 1986	later	earlier
	Stearns and Koella 1986	later	earlier
	Kozlowski and Wiegert 1987	earlier	later
	Hutchings 1993	earlier[1]	later[1]
Size at maturity	Roff 1984, 1986	smaller	larger
	Stearns and Koella 1986	variable[2]	variable[2]
	Kozlowski and Wiegert 1987	smaller	larger
Reproductive allotment	Hutchings 1993	higher[1]	lower[1]
Offspring size	Smith and Fretwell 1974	larger	smaller
	Morris 1987		

[1] Growth environment defined as adult/juvenile growth rate.
[2] Size at maturity depends on juvenile and adult mortality rates.

zlowski and Uchmanski 1987; Kozlowski and Wiegert 1987), or earlier age at maturity and a variety of sizes at maturity (depending on environmentally induced mortality rates) in high growth environments relative to low growth ones (Stearns and Crandall 1984; Stearns and Koella 1986; Houston and McNamara 1992; Kawecki and Stearns 1993; Berrigan and Koella 1994; see Table 1). Recently, Hutchings (1993) used a different, but related approach, relying on differences in juvenile and adult growth rate to predict strategies in timing and overall quantity of reproductive investment. Hutchings (1993) compared environments on the basis of relative growth for adults and juveniles, and predicted that environments with higher relative adult growth rates should have lower reproductive allotments and later ages at maturity relative to lower adult growth environments. A second group of models relate the effects of food availability and juvenile survival on the "packaging" of reproductive investment per offspring (Smith and Fretwell 1974; Morris 1985, 1987; McGinley et al. 1987, Schultz 1991). In these models, poorer environments are predicted to select for larger investment per offspring (Table 1).

All of the above models make testable predictions on the directionality of responses for several important life-history traits for organisms in low relative to high food environments (Table 1). However, experimental falsification of these predictions for any population can always be argued against on the basis of genetic constraints on the traits being tested. Specifically, lack of additive genetic variation for norms of reaction will not allow a plastic phenotypic response to evolve, even when such a response is adaptive (Via and Lande 1985). Therefore, any experimental tests of the above predictions that do not find plastic responses in the directions predicted (Table 1) can be explained by assuming the responses, though optimal, were not possible due to genetic constraints in the population examined. Thus, a complete test of these optimality models requires an additional step to quantify the underlying genetic variation in the traits examined (Rose et al. 1987; Leroi et al. 1994).

A need for a combined approach to phenotypic plasticity, in which one tests the predictions of the models of adaptive phenotypic plasticity, along with quantification of the genetic variation in those responses, is the motivation for the current study. To test the influences of two environments (differing in resource quantity) on life-history variation, we used a common freshwater poeciliid, the eastern mosquitofish, *Gambusia holbrooki*, raised under controlled conditions in the laboratory. The population used was collected from Pond C at the US Department of Energy's Savannah River Site near Aiken, South Carolina. This natural (i.e., nonintroduced) population was chosen because of a history of exposure to short-term resource fluctuations. Pond C was part of a nuclear reactor cooling system (Bennett and Goodyear 1978; Block et al. 1984), and thus underwent numerous cycles of extreme temperature change from 1958 to 1988. The length of the thermal cycles was random, ranging from thermal input for a few hours to several months. These cycles resulted in concomitant fluctuations in resources several times during the year, which caused "boom and bust" cycles for the *Gambusia* in this system (Meffe 1991). Therefore, we expect that such patterns of resource and thermal fluctuations may have selected for adaptive phenotypic plasticity in Pond C *Gambusia* (Giesel 1976).

We examined the responses of fitness-related characters (growth, age and size at maturity, fecundity, egg size, reproductive allotment [proportion of biomass in reproductive tissues], and lipid storage) in *G. holbrooki* from Pond C raised under two levels of resource stress to quantify the plasticity of responses of the above characters to decreased resource availability. We then compared these data to optimality models that predict investment strategies in high and low food environments (Table 1). We also combined this analysis with a quantitative genetic examination of the heritability of norms of reaction to address the possibility of genetic constraints on optimal phenotypic plasticity.

MATERIALS AND METHODS

Twenty-four first lab-generation (F_1) *G. holbrooki* females were mated to eight wild-caught males in a nested mating design (three females per male). F_1 females were used to reduce the potential for carry-over of maternal effects from the field (Reznick 1981). After mating, the F_1 females were isolated in individual 3.7-liter plastic jugs with artificial grass (contained within 220-liter aquaria), and allowed to produce offspring. Each jug had 4 × 8 cm, mesh-covered holes cut in each wall to allow continuous water exchange.

Twelve F_2 offspring per F_1 female were randomly chosen from brood two, and these were measured for standard length and then placed into individual jugs in the 220-liter aquaria described above. All length measurements were made by capturing a dorsal image of the fish on a computer image analysis system, with MorphoSys® software. Standard length (measured from tip of the upper lip to the beginning of the caudal fin) was then calculated by averaging measures from three separate images of each fish.

Twelve 220-liter aquaria were used. Each aquarium could hold 24 of the 3.7-liter jugs. Therefore, one offspring from each of the 24 families was placed into each of the 12 aquaria, for a total of 288 individuals. Fish in six of the aquaria were fed a high food diet and the other six were fed a low food diet. Fish were fed brine shrimp nauplii when they were juveniles, and were gradually switched over to flake food as they aged. This feeding regime has proven to produce maximal growth conditions (at the high diet) in previous experiments using this mosquitofish population (Meffe, unpubl. data).

For the brine shrimp feeding, one gram of brine shrimp eggs (Argent Chemical Laboratories, Redmond, WA) was added to one liter of saltwater and incubated for 48 h. The hatched nauplii were flushed with freshwater, and concentrated into 500 ml of freshwater. From this solution, 2, 3, 4, and 5 ml of suspended nauplii were fed to the low-diet fish at intervals of 1–7, 8–14, 15–28, and 29–40 d, respectively. The high-diet fish received three times these volumes during the same time intervals. Between 41 d and 48 d, the food alternated between nauplii one day and flake food (Prime Flakes Tropical Fish Food, Zeigler Bros., Inc., Gardners, PA) the next day. The flake food was fed using shaker bottles, which provided a little more than three times the amount of flake food for high-diet versus low-diet treatments (high-diet = 10.4 ± 1.0 mg; low-diet = 3.1 ± 0.5 mg). From day 49 to 70, fish in both treatments received only flake food once a day.

Growth was estimated every two weeks by measuring standard length as above. Juvenile growth was defined as growth during the first two growth increments (2 and 4 weeks); adult growth was the average of the remaining intervals (6, 8, and 10 weeks).

After the third week, each fish was checked every two days for sexual maturity. Sexual maturity in males was based on the elongation of the anal fin into the gonopodium, and was determined in accordance with the descriptions of Turner (1941). Sexual maturity in females was based on the appearance of a black spot dorsal to the anus, which enlarges to its maximal size at sexual maturity (Constanz 1989). When the fish had matured, standard length was measured and the day of maturity was recorded.

All fish were sacrificed at 70 days, and females were dissected to remove their reproductive tissues. Eggs were categorized as immature, mature, or atretic (post mature; see Wetherington et al. 1989). Fecundity was defined as the combination of mature and atretic eggs. Egg size was measured on the mature eggs by selecting the three largest eggs and measuring their maximum and minimum diameters (see Weeks 1993). Weight of the reproductive tissues (used in calculations of reproductive allotment) was measured by weighing all the dried eggs and other supportive ovarian tissues (see below).

For somatic lipid extraction, male and female fish were diced into small pieces and placed in tared, 3.5-ml thimbles. The intestines did not need to be removed, because the fish were not fed for over 24 h before sacrifice. For ovarian lipid extraction, eggs and ovarian tissues were placed in tared, 2-ml thimbles. Procedures for lipid extraction are outlined in Meffe and Snelson (1993). The difference in pre- and postextraction mass is the amount of storage lipid contained in the tissues.

Statistical Methods

All nine life-history traits were analyzed using a mixed-model ANOVA. The independent variables were block (i.e., 220-liter aquarium), food treatment (hereafter "treatment"), sex, sire, and dam. Treatment and sex were considered fixed effects while the other variables were considered random. The statistical analysis followed Via (1984) and Fry (1992). Because the treatments were applied per tank, the appropriate statistical analysis is to nest replicate tanks within feeding treatments (Hicks 1982). The only interaction with sex that was significant was the sex*treatment interaction. Therefore, all other interactions involving sex were removed from the models, and the ANOVAs were recomputed. In the juvenile growth analysis, differences between sexes were nonsignificant, and sex was removed from the model. The ANOVAs were then recomputed. Also, sex was not a factor in the analyses of fecundity, egg size, ovarian lipid content, and reproductive allotment, because these variables were only measured in females. Five of the nine traits were affected by blocks, though none of the interaction terms including blocks were significant, and thus were not included in the analyses.

There was no mortality during the 70 d of the experiment. However, there was some imbalance in the statistical design because some juveniles developed spinal scoliosis, and were removed from all analyses, and some dam*treatment combinations did not have both sexes represented. Because of the imbalance, Type IV sums of squares were used to calculate F-ratios (SAS 1985), and these F-tests should be considered approximations (Fry 1992). Tests of significance were computed using the Satterthwaite approximation in the RANDOM statement in PROC GLM (SAS 1985). The appropriate F-ratios for the analyses with and without sex are those given by the "SAS model" (Fry 1992). All dependent variables conformed to the expectations of the ANOVA models, and therefore transformations were not used on any variate.

In the analyses of reproductive allotment, somatic lipids, and ovarian lipids, some measure of size was used as a covariate. For reproductive allotment, ovarian dry weight (before lipid extraction) was the dependent variable and somatic dry weight (before lipid extraction) was the covariate. This analysis is similar to using a "gonadosomatic index," which is the ratio of reproductive weight to body weight (e.g., Reznick et al. 1993). For lipid content in the somatic and ovarian tissues, lipid-free somatic dry weight and lipid-free ovarian dry weight were used as the covariates, respectively. In these analyses, differences between treatments indicated that individuals had more fat in the respective tissues, relative to body size. Use of ANCOVAs were substituted for ANOVAs

of ratios because ANCOVAs are more robust due to the specific tests of the assumption of homogeneity of slopes. For somatic lipids, the number of days that the females were mature was negatively correlated with the somatic lipid content. Therefore, "days mature" was used as a second covariate in analysis of somatic lipids. The relationships of the three dependent variables with the covariates were homogeneous across the independent variables for all analyses, as assumed by the ANCOVA procedure.

Results and Discussion

In the current experiment, six of the nine fitness-related traits were significantly affected by the two feeding treatments (Table 2; Fig. 1), and thus displayed phenotypic plasticity in response to food availability. In what follows, we briefly describe the responses of the six fitness characters that exhibited significant plasticity, and then interpret these responses both in terms of optimality models, where possible, and in terms of genetic constraints.

The initial effect of the feeding treatments was to increase juvenile growth rate for the high- relative to the low-diet fish (Table 2; Fig. 1). This increased growth rate presumably triggered an earlier investment in reproduction at a slightly (but not significantly) larger size, and an increased reproductive allotment that resulted in a greater number of eggs per female in the first 70 d relative to the low-diet fish (Table 2; Fig. 1). Also, the proportion of the somatic and ovarian tissues that was due to storage lipids was lower in the high-diet fish (Table 2; Fig. 1). Because the ovarian tissues are composed primarily of eggs by weight, and since egg size did not differ among feeding treatments (Table 2), these results indicate a greater amount of storage lipid per egg in the low- relative to the high-diet fish, which is consistent with previous work on guppies (Reznick and Yang 1993). Note that estimates of egg volume in both feeding treatments (~4.0 μl) is similar to volumes reported in related lecithotrophic poeciliid species in the genus *Poeciliopsis* (Weeks 1993), suggesting that the eggs measured in the current study were fully developed.

Optimality Models

We now compare these responses to those predicted by several optimality models that relate life-history variation to environments that differ in food availability or potential for growth. Three models specifically predict optimal age and size at maturity from differences in growth environment (Table 1). The first (Roff 1984, 1986) maximizes net reproductive rate, and predicts optimal age and size at maturity using a simple relationship between growth and mortality rates. Assuming mortality rates are similar across environments, increased growth should be associated with decreased age at maturity and increased size at maturity. A similar, but more complex, model has been developed by Stearns and Koella (1986), and since has been extended by several authors (Houston and McNamara 1992; Kawecki and Stearns 1993; Berrigan and Koella 1994). This model predicts four separate optimal norms of reaction for individuals in different growth environments. In addition to these four norms of reaction, Berrigan and Koella (1994) have identified several others. In all scenarios, optimal age at maturity decreases as growth

Table 2. Mean square values for life-history traits. Abbreviations are as follows: Block(Trt) = block nested within treatment; Trt*Sex = treatment by sex interaction; Trt*Sire = treatment by sire interaction; Dam(Sire) = dam nested within sire; Trt*Dam = treatment by dam within sire interaction; Juv Grth = juvenile growth rate; Adult Grth = adult growth rate; AAM = age at maturity; SAM = size at maturity; Som Lpds = somatic lipid content; Egg Vol = egg volume; Ova Lpds = ovarian lipid content; and Rep Allt = reproductive allotment. No values are given for Sex and Trt*Sex for five of the nine traits because these components were not included in the model for these traits (see text).

	Life history trait															
	Juv Grth		Adult Grth		AAM		SAM		Som Lpds		Fecundity		Egg Vol		Ova Lpds		Rep Allt	
Component	MS	*P*	MS	*P*	MS	*P*	MS	*P*	MS	*P*	MS	*P*	MS	*P*	MS	*P*	MS	*P*
Treatment	22.9	.0001	0.1	.4206	1157	.0298	8.22	.3438	66.7	.0064	1966	.0025	0.21	.6133	11.4	.0186	166.0	.0270
Block(Trt)	0.3	.0008	1.4	.0001	177	.0064	7.41	.0007	4.5	.1322	107	.0523	0.95	.0095	0.9	.7633	18.5	.1744
Sex			87.1	.0001	833	.0006	5.78	.1158	269.7	.0001								
Trt*Sex			2.7	.0013	0	.9353	0.88	.5386	14.7	.0272								
Sire	0.4	.7972	0.4	.8888	262	.4415	9.39	.5391	11.4	.2084	94	.4179	0.63	.2338	6.2	.1234	23.1	.5763
Trt*Sire	0.1	.2626	0.3	.0401	66	.1621	3.54	.0618	4.6	.3309	68	.1794	0.24	.6548	1.4	.5176	21.2	.0489
Dam(Sire)	0.6	.0001	0.8	.0001	225	.0004	8.58	.0004	5.0	.2747	53	.2927	0.33	.4694	2.6	.1736	14.1	.1384
Trt*Dam	0.1	.6502	0.1	.9862	37	.9275	1.42	.8711	3.7	.2402	40	.7705	0.32	.5760	1.6	.3276	7.8	.8553
Error	0.1		0.3		70		2.32		3.0		56		0.36		1.4		12.8	

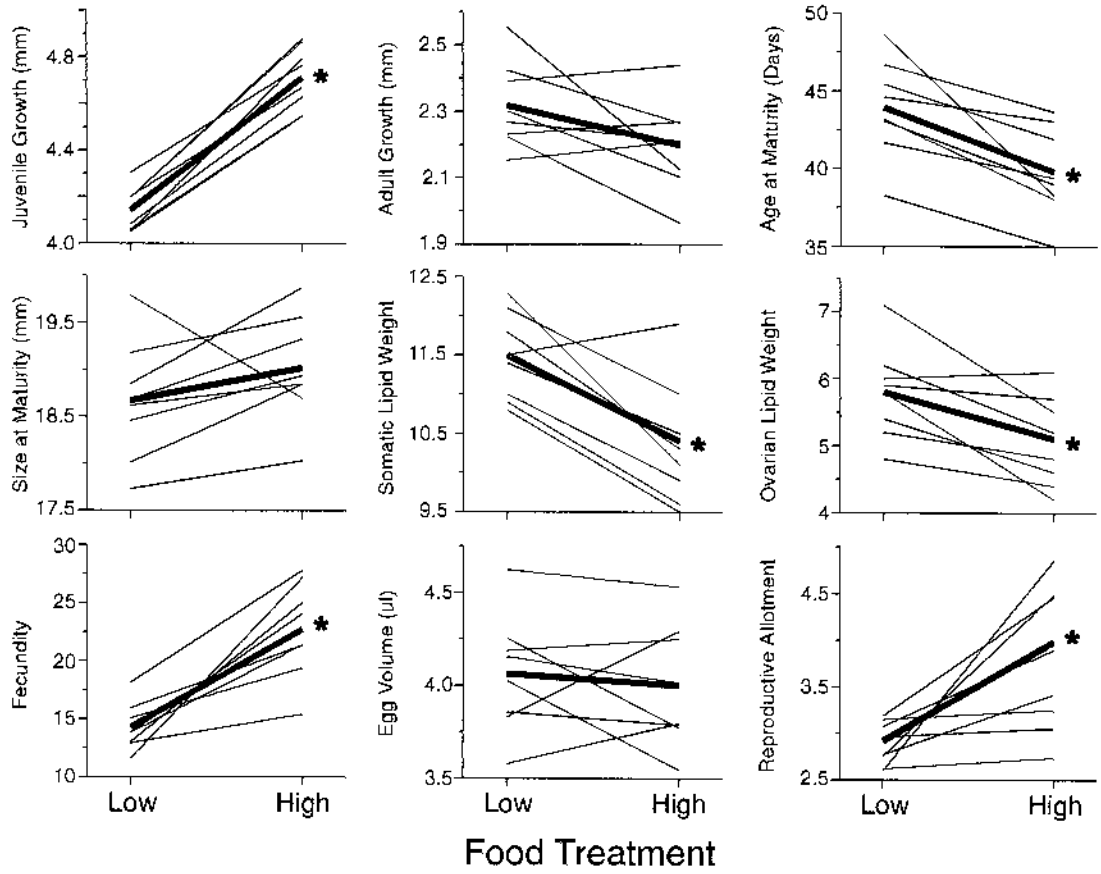

FIG. 1. Reaction norms for half-sib families. Thicker lines show the average response of all families, and asterisks denote significant ($P < 0.05$) phenotypic differences in high- and low-food treatments. For somatic lipid weight, ovarian lipid weight, and reproductive allotment, the units are not portrayed because the responses of these three variables have been corrected for covariates (see text).

increases. However, size at maturity can either increase, decrease, or stay the same in different environments, depending on the effects of growth on juvenile and adult mortality rates (Stearns and Koella 1986) and whether growth is determinate or indeterminate (Berrigan and Koella 1994). The third model (Kozlowski and Wiegert 1987) also maximizes net reproductive rate, and uses a geometric approach for the calculation of lifetime fitness. In this model, increased growth rate is predicted to cause delayed age at maturity and maturation at a larger body size (Table 1).

In the current study, age at maturity was significantly decreased for fish on high- relative to low-diet treatments (Fig. 1), which is consistent with the models of Roff (1984, 1986) and Stearns and Koella (1986), but not with the Kozlowski and Wiegert (1987) model (Table 1). In addition, size at maturity was slightly, but not significantly, larger in high- relative to low-diet treatments. These results also contrast with the predictions of the Kozlowski and Wiegert (1987) model, indicating that this model does not correctly predict the responses of this population. The later age at maturity and the slight increase in size at maturity on high diets is consistent with either the Roff (1984, 1986) or Stearns and Koella (1986) models, thus not allowing us to distinguish between the two. To obtain a more precise estimate of the predictions of the these two models, one needs an estimate of the effects of growth environment on juvenile and adult mortality rates. To date, estimates of mortality rates in the Pond C *Gambusia* population have been unsuccessful. The population is too large for mark-release-recapture estimates, and so far, determining age of Pond C *Gambusia* using otoliths has been unsuccessful. Therefore, although we can eliminate the Kozlowski and Wiegert (1987) model as descriptive of the plastic responses of age and size at maturity of these fish, further tests of the two remaining models must await better estimates of natural mortality rates in Pond C.

Recently, Hutchings (1993) suggested a novel approach to relating age at maturity, as well as reproductive allotment, to growth environment. Hutchings suggested defining environments on the basis of the relative growth rates of the juvenile and adult phases of an organism's life history. Individuals with low adult-to-juvenile growth ratios (i.e., low relative adult growth rates) should reproduce earlier and have a higher reproductive allotment than individuals with high adult/juvenile growth (Hutchings 1993). These predictions are based on the expectations for future reproduction, which should be lower in environments with lower relative adult growth, assuming fecundity is positively related to size. Low expectations of future reproduction should select for increased current reproductive effort (Williams 1966; Schaffer 1974). Hutchings (1993) finds that several populations of

TABLE 3. Calculation of adult-to-juvenile growth ratios in the two feeding treatments, and their respective ages at maturity and reproductive allotments. Means are calculated for females only.

Trait	High (SE)	Low (SE)
Juvenile growth (mm/2 weeks)	4.71 (0.05)	4.16 (0.04)
Adult growth (mm/2 weeks)	2.97 (0.06)	2.89 (0.03)
Adult/juvenile growth ratio	0.63	0.69
Age at maturity (days)	37.8 (0.44)	42.5 (0.61)
Reproductive allotment (%)	33.2 (1.0)	29.9 (0.8)

brook trout (*Salvelinus fontinalis*) have ages at maturity and reproductive allotments that are consistent with these expectations. In the current experiment, the high-diet environment caused lower adult/juvenile growth relative to the low-diet treatment (Table 3), due to a significantly higher juvenile growth rate in the former (Fig. 1). Age at maturity was significantly reduced and reproductive allotment was significantly greater in the low relative to the high adult/juvenile growth environment as expected in the Hutchings (1993) model. Therefore, mosquitofish again appear to have responded to food limitation in an adaptive direction in age at maturity and also in reproductive allotment.

There are a number of optimality models that relate investment in offspring to food availability (Smith and Fretwell 1974; McGinley et al. 1987; Morris 1987; Winkler and Wallin 1987; Schultz 1991). Environments with low food availability should favor greater investment per offspring (Table 1), as long as this increased investment confers a large enough increase in offspring fitness to compensate for the reduction in total offspring number (Smith and Fretwell 1974; Morris 1987). This prediction has recently been supported in a study on juvenile brook trout (Hutchings 1991). In the current study, egg size was slightly larger (but not significantly so) and there was significantly greater amounts of lipids in the low- relative to the high-diet fish (Table 2). Because egg size was basically unchanged, these results indicate that energetic investment per offspring (in the form of increased lipids) was greater in the low-diet fish, consistent with the expectations of the optimality models. Increased offspring weight, which was largely a result of increased lipids, was also found in a recent study of female *Poecilia reticulata* raised in low-diet treatments (Reznick and Yang 1993). It is possible that increased lipid stores allow a greater resistance to starvation in juvenile fish, and thus would be adaptive in low-diet environments. A comparison of survival of offspring under starvation stress produced by mosquitofish females raised in high- versus low-diet environments would clarify the fitness effects of such an allocation strategy, and thus clarify this potentially adaptive response.

Genetic Constraints

Tests of adaptive phenotypic plasticity are incomplete without examinations of the underlying genetics of the traits measured. There are two potential genetic constraints on the evolution of optimal phenotypic plasticity: genetic correlations and lack of sufficient additive genetic variation (Via 1984; Via and Lande 1985). The former would constrain the simultaneous evolution of optimal phenotypes in two or more correlated traits in one or more environments, whereas the latter would constrain the ability of natural selection to mold an adaptive response (Fisher 1930; Falconer 1981; Via and Lande 1985; Via 1987). In particular, for selection of adaptive phenotypic plasticity, there must be an additive genetic component to norms of reaction. This component can be measured either by finding that the genetic correlation (r_G) of a trait expressed in two or more environments is less than $\pm$ 1 (Via 1984, 1987), or that there is a significant additive component to the genotype by environment interaction (Via 1984). The low numbers of sires used in the current experiment did not allow us to estimate significant levels of r_G with much power. However, power to detect significant levels of additive G $\times$ E (calculated using the treatment by sire interactions) was high ($> 80\%$) in this experimental design, assuming r_G was low (< 0.4) and heritability was moderate to high (> 0.40) in the five nonreproductive traits (see Fry [1992] for power calculations). Power to detect significant levels of additive G $\times$ E in the four reproductive traits was $> 60\%$ when r_G was very low (< 0.2) and heritability was high (> 0.80). Therefore, to estimate genetic constraints in this population, we concentrated on the detection of additive genetic variation in norms of reaction using the estimates of sire-by-treatment interactions.

Significant levels of sire-by-treatment variation were found for three of the nine life-history traits (adult growth, size at maturity and reproductive allotment), indicating detectable levels of additive G $\times$ E in these traits (Table 2; Fig. 1). Thus, it appears that sufficient levels of additive variation exists for selection of adaptive norms of reaction in these traits in this population of mosquitofish. Two of these three traits (adult growth and size at maturity) showed no significant plastic response to feeding environment, yet appear to have sufficient genetic variation for natural selection to shape an adaptive norm of reaction, if one exists. Thus, the lack of a significant plastic response of size at maturity (see above) cannot be attributed to a genetic constraint in this population. For the third trait, reproductive allotment, the phenotypic response to feeding environment appears to be in the direction predicted, and there appears to be sufficient genetic variation for continued selection. The significant level of additive G $\times$ E can either indicate that selection is still continuing toward a single optimal norm of reaction, that there are many optima in this population, or that genes for reproductive allotment are genetically correlated with genes for other characters that preclude the simultaneous evolution of optimal characters in all environments (Falconer 1981; Via and Lande 1985).

The other six traits did not reveal significant levels of additive G $\times$ E (Table 2). This can either indicate that there really is no additive variation for norms of reaction remaining in this population for these traits, or that the levels of variation are too low to be detected using the current sampling design. If the levels of additive variation are truly negligible for these traits, then continued selection for adaptive plasticity in traits such as age at maturity and ovarian lipids will be impossible in this population in the near future. So, although these two traits appear to have responded to the feeding environments in the directions predicted by the optimality models (Table 1), the population may be constrained from

attaining the single most adaptive plastic response because of a lack of sufficient genetic variation. An alternate explanation for the lack of genetic variation in these traits is that a single most adaptive plastic response has been attained, and that all other variants have been selected against, which would reduce the additive variation to nondetectable levels (Falconer 1981). Estimating the optimal responses of these traits in these environments (Schluter 1988) would be helpful to distinguish between these alternatives.

Because this study was conducted in the laboratory, it remains unclear if our estimates of additive G × E reflect conditions in the field (see Trexler and Travis 1990a,b). In a review of several quantitative genetic studies on a variety of organisms, Hill and Caballero (1992) reported that nearly 40% of the genetic analyses that measured both expected heritabilities (calculated from either sib correlations or offspring-parent regressions) and realized heritabilities (calculated from selection experiments) on the same population found a less than 50% correspondence between these two measures. In fact, 25% of these studies showed significant differences (at the $P < 0.05$ level) between expected and realized heritabilities, suggesting that expected heritabilities can often be misleading. Thus, extrapolations from laboratory studies to natural systems, such as the current study, should be made with caution.

Conclusions

Overall, the mosquitofish in the current study appeared to exhibit adaptive phenotypic plasticity in response to food stress. Patterns of plasticity in age at maturity, reproductive allotment, and investment in offspring were consistent with optimality models for these traits. Further studies, both comparative and experimental, are required before we can claim the observed plasticity is the optimal response to the feeding environments imposed (Orzack and Sober 1994). In this regard, visualizations of the fitness surface for life-history variants in the two feeding environments (Schluter 1988) would potentially help to understand the different selective regimes in the two environments. Nevertheless, we can claim that the average responses of these fish were in the directions predicted by several optimality models, and thus suggest adaptive phenotypic plasticity has been selected in this Pond C mosquitofish population.

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