Change of Genetic Architecture in Response to Sex

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ABSTRACT

A traditional view is that sexual reproduction increases the potential for phenotypic evolution by expanding the range of genetic variation upon which natural selection can act. However, when nonadditive genetic effects and genetic disequilibria underlie a genetic system, genetic slippage (a change in the mean genotypic value contrary to that promoted by selection) in response to sex may occur. Additionally, depending on whether natural selection is predominantly stabilizing or disruptive, recombination may either enhance or reduce the level of expressed genetic variance. Thus, the role of sexual reproduction in the dynamics of phenotypic evolution depends heavily upon the nature of natural selection and the genetic system of the study population. In the present study, on a permanent lake *Daphnia pulicaria* population, sexual reproduction resulted in significant genetic slippage and a significant increase in expressed genetic variance for several traits. These observations provide evidence for substantial genetic disequilibria and nonadditive genetic effects underlying the genetic system of the study population. From these results, the fitness function of the previous clonal selection phase is inferred to be directional and/or stabilizing. The data are also used to infer the effects of natural selection on the mean and the genetic variance of the population.

A traditional view of sexual reproduction is that it provides populations with greater adaptability by generating greater genetic variability through segregation and recombination that occur during meiosis (LANDE 1975; LYNCH and GABRIEL 1983; MICHOD and LEVIN 1988; CHARLESWORTH 1990, 1993). This view might be true were there no nonadditive genetic effects and/or genetic disequilibria were in repulsion, *i.e.*, alleles of dissimilar effects were clustered together statistically.

In their theoretical treatment, biologists differ to a certain extent with regards to the roles of nonadditive genetic effects and genetic disequilibria as influential factors in determining genetic architecture of populations (means and genetic variation-covariation) (MATHER 1942, 1943; GRIFFIN 1960; BULMER 1971; LANDE 1975; THOMPSON 1976; LYNCH and GABRIEL 1983; KONDRASHOV 1988; BARTON and TURELLI 1989; FALCONER 1989; HOULE 1989; CHARLESWORTH 1990; BURGER 1993; GAVRILETS and HASTINGS 1995). Experimental evidence for nonadditive genetic effects and genetic disequilibria is also somewhat unbalanced. There seems to be substantial evidence for nonadditive genetic effects within a locus (dominance), as revealed by the almost universal phenomenon of inbreeding depression (CHARLESWORTH and CHARLESWORTH 1987; RALLS *et al.* 1988; FALCONER 1989). Evidence for nonadditive genetic effects among loci (epistasis) is not as substantial (but see DOBZHANSKY *et al.* 1959; MUKAI 1969; CHARLESWORTH and CHARLESWORTH 1975; HEATH *et al.* 1984; CHAO 1988; HARD *et al.* 1992; WILLIS 1993; MORENO 1994). Despite a large number of studies, evidence for genetic disequilibria in natural populations is still controversial (*e.g.*, BARKER 1979; LEWONTIN 1985; SMIT-MCBRIDE *et al.* 1988; ZAPATA and ALVAREZ 1992, 1993).

Genetic disequilibrium can influence the standing genetic architecture of populations (means and genetic variation-covariation) in two ways (throughout, genetic disequilibrium will refer to both gametic-phase disequilibrium and/or HARDY-WEINBERG disequilibrium). First, in populations in genetic disequilibrium, the genetic (co)variation for quantitative traits that is revealed at the phenotypic level is only the expressed genetic (co)variation. Virtual genetic (co)variation is that expected in the absence of genetic disequilibrium. Expressed genetic (co)variation will be higher than virtual genetic (co)variation, if prevailing genetic disequilibria are in coupling, and lower if in repulsion (LANDE 1975; LYNCH and GABRIEL 1983; GAVRILETS and HASTINGS 1993; LYNCH and DENG 1994) (Note hereafter, unless otherwise specified, genetic variability will refer to the expressed genetic variability). Second, when both genetic disequilibria and nonadditive genetic effects are present, the genotypic mean may differ from its equilibrium value (LYNCH and DENG 1994). The implications of these principles for the evolutionary dynamics of quantitative traits are threefold (LYNCH and DENG 1994). Upon random mating, (1) the mean of a quantitative trait will change if there are nonadditive genetic effects in genetic disequilibria; (2) genetic (co)variation of quantitative traits will increase if genetic disequilibria

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are in repulsion, or decease if in coupling; and (3) the direction of change of the mean and genetic (co)variation (if any) will be opposite to that promoted by previous selection.

Though nonadditive genetic effects and genetic disequilibria are relevant to all organisms, the issues are investigated empirically most easily in cyclical parthenogens (LYNCH and DENG 1994). In natural populations, each year, cyclical parthenogens typically experience several consecutive generations of asexual reproduction. During such extended periods of clonal selection, the magnitude of genetic disequilibria, and hence the consequences of sexual reproduction on genetic architecture, are magnified. Therefore, it is relatively easier to detect change of the genetic architecture (if any) brought about by sex in cyclical parthenogens than in a purely sexual population. Additionally, the following features can facilitate some genetic studies.

1. In the laboratory, genotypes of cyclical parthenogens can be preserved intact, barring mutation, by asexual reproduction (HEBERT 1987), and hence can be replicated within and among experiments. Therefore, parental genotypes and their sexually produced offspring genotypes can be assayed side-by-side in one controlled environment. Any change in the genetic architecture across sexual generations will thus not be confounded by any temporal environmental changes and can only be attributed to the genetic changes brought about by sexual reproduction.

2. By replicating and acclimating genotypes within experiments (LYNCH 1985), the total expressed genetic (co)variation [sum of additive and nonadditive genetic (co)variation] can be estimated in both parental and offspring generations across a generation of sexual reproduction (LYNCH 1984; LYNCH *et al.* 1989; LYNCH and DENG 1994). In purely sexual species, unless monozygotic twins are readily available or there are no nonadditive sources of genetic variation, the total expressed genetic (co)variation can not be estimated without bias (FALCONER 1989).

3. Estimation of covariance between the parent and sexually produced offspring can yield information on additive genetic (co)variation (FALCONER 1989). A comparison of the additive genetic (co)variation and the total genetic (co)variation provides insight into the magnitude of the nonadditive genetic contribution to the total genetic (co)variation.

The present study investigates the influences of sexual reproduction on the genetic architecture of a large permanent lake Daphnia population. The results are used to infer natural selection on several characters, and the contribution of genetic disequilibria and nonadditive genetic effects to the population's genetic architecture.

MATERIALS AND METHODS

Study organism: Daphnia is a freshwater microcrustacean. It lives in either ephemeral (seasonal ponds) or permanent (lakes and reservoirs) environments. Most Daphnia populations reproduce by cyclical parthenogenesis, others by purely asexual reproduction. Normally, cyclically parthenogenetic populations are initiated by hatching resting eggs (reproduced by sexual reproduction). All hatchlings are females, initially reproducing asexually. In permanent environments, dozens of generations of clonal reproduction are common. At the end of the growing season, when environments begin to deteriorate, males are produced asexually by females, and females switch to sexual reproduction. Mating is normally random in natural populations, as concluded by extensive electrophoretic studies [for reviews, see HEBERT (1987) and LYNCH and SPITZE (1994)]. The sexually produced eggs are resting eggs, wrapped in a modified carapace, called an ephippium. They are the forms through which Daphnia populations survive harsh environmental periods.

Study population: In October 1992, a permanent lake Daphnia population was found to be undergoing a phase of sexual reproduction. The population is located in Dorena Reservoir, Cottage Grove, Oregon. The reservoir has an area of at least 7 km^2. Over 2 consecutive days, thousands of adult individuals were sampled from 10 randomly chosen locations throughout the lake (five near the shore and five near the middle, with each location being at least 20 m apart). The population contained approximately 15% sexual females and 8% males. All isolated animals were identified morphologically (BROOKS 1957) as members of the *Daphnia pulex* group.

Production of outbred progeny: About 1000 females bearing ephippia (sexually produced resting eggs) were isolated into individual beakers containing the green alga *Scenedesmus* in filtered water from Dorena. Within the next 3 days, about half of the females died. For those surviving, the shed ephippia were isolated into small individual vials containing about 5 ml of filtered lake water. Subsequently, the vials were maintained at 4° in complete darkness for 7 days and then were taken out and placed at 20° in a photoperiod of approximately 10 hours of light/day, and were monitored for hatching for 7 days. Normally, seven to 35 resting eggs would hatch during this period, with a hatching peak occurring between 2.5 to 4 days after the vials were introduced to the light/warm condition. By alternating dark/cold and light/warm cycles 10 times, hatchlings from approximately 230 parental clones were obtained and cloned, yielding sexually produced offspring clones. After shedding ephippia, the originally isolated females resumed asexual reproduction, providing parental clones for subsequent experiments.

Determination of species identity and breeding system: Cellulose acetate gel electrophoresis (HEBERT and BEATON 1989) was performed on 10 allozyme loci: LDH, FUM, MPI, AD, ME, MDH, HEX, APK, PGM, and PGI. The diagnostic locus LDH was fixed for the F allele, identifying this population as *Daphnia pulicaria* (HEBERT *et al.* 1988, 1989). The PGI and PGM loci were polymorphic, each having four alleles. At the PGI locus, in the parental generation, the population was significantly out of HARDY-WEINBERG proportions (sample size $n = 108$, G TEST (SOKAL and ROHLF 1981), $P < 0.01$). However, in the offspring generation, data from a comparable sample size (n = 101) did not detect significant HARDY-WEINBERG deviation ($P > 0.10$). Data for the PGM locus did not reveal any significant HARDY-WEINBERG deviation in either generation (for both generations, $n = 48$, $P > 0.05$). Consistency with HARDY-WEINBERG proportions at the PGI locus in the offspring generation and at the PGM locus in both generations supports the idea that the population reproduces sexually and that mating is effectively random. Direct evidence of

sexuality came from the observed segregation of alleles in the offspring from the parents heterozygous at the two polymorphic loci (PGM and/or PGI) during routine electrophoretic analyses.

Life-table experiments: Two life-table experiments were performed, employing a total of 85 pairs of parents and sexually produced offspring. The experiments utilized a standard life-table design (LYNCH 1985; LYNCH *et al.* 1989). The temperature was 10°, and the photoperiod was set to a 16-hr light:8-hr dark cycle. The food for each individual animal was 100 ml filtered Dorena Lake water with a density of the green alga *Scenedesmus* of about 300,000 cells per ml, which was replenished every other day. Each clone had three replicate lines in the experiments. All replicate lines were acclimated to the defined experimental conditions for two generations to ensure that maternal and grandmaternal effects did not contribute to the between-clone component of variance in the final analyses (LYNCH 1985; SPITZE *et al.* 1991). Under the experimental temperature, the developmental rate of Daphnia is low; therefore, individuals were only measured every other day for different life-history traits, such as instar-specific body size, clutch size and age at first reproduction, *etc.* Detailed experimental procedures have been described extensively elsewhere (*e.g.,* LYNCH 1985; LYNCH *et al.* 1989).

Data analysis: For the data of different life history traits from the two life-table experiments, nested ANOVA analyses were performed respectively for each trait, using PROC GLM (SAS INSTITUTE 1990), specifying replicates nested within clones and clones nested within the two life-table experiments (block). The results, adjusted by a simultaneous inference procedure for the number of the traits tested (sequential Bonferroni technique) (HOLM 1979; RICE 1989), revealed that the between-life-table experimental effects were not significant at the 5% level. Thus, data from the two life-table experiments were combined for further analyses. Using statistical procedures in LYNCH *et al.* (1989) and LYNCH and DENG (1994), univariate analyses were conducted for each life-history trait in both generations (parent and offspring), to estimate the broad-sense heritability (H^2, the ratio of total genetic variance to phenotypic variance), narrow-sense heritability (h^2, the ratio of additive genetic variance to phenotypic variance), and the change of the mean phenotypic value and total genetic variance across generations. Note again, unless otherwise specified, genetic variability refers to the expressed genetic variability, and heritability refers to the expressed heritability. With genetic disequilibria, the virtual genetic variability and heritability are not estimable; further, what is relevant to the short-term evolution is the expressed genetic variability and heritability. In computing h^2, the additive genetic variance was estimated by twice the covariance of the means of the parental and offspring clones, and the phenotypic variance is estimated by the phenotypic variance among parental individuals.

Correlations were investigated at the phenotypic, additive genetic and total genetic levels for three kinds of comparisons: offspring number and offspring size, clutch size and investment in growth (increment in body-size), clutch size and age at reproduction. The analyses of correlations at the total genetic and phenotypic levels followed SPITZE *et al.* (1991) and were conducted by a bootstrapping program supplied by K. SPITZE. The analyses of additive genetic correlations from parent and offspring data followed FALCONER (1989, pp. 314–317), and the computation was performed by a bootstrapping program developed by us.

RESULTS

Broad-sense and narrow-sense heritabilities: For the seven life-history traits analyzed, in both generations, the broad-sense heritability (an index of total genetic variability) was significantly greater than zero (significance is at the 5% level unless otherwise specified), ranging from 0.25 to 0.69 (Table 1), with an average for all characters over both generations of 0.47. On the other hand, the narrow-sense heritability (an index of additive genetic variability) was relatively low and significant for only three traits, with an average value of 0.27 (Table 1). Since the difference between the broad- and narrow-sense heritabilities is approximately equal to the fraction of the total phenotypic variance that has a nonadditive genetic basis, these observations suggest a relatively high magnitude of nonadditive genetic variance in the study population. Averaged over the two generations and the seven traits, nonadditive genetic variance composed about 20% of the total phenotypic variance, and about 43% of the total genetic variance.

Heritability may not be the best measure of genetic variability in certain situations (HOULE 1992). In order for the measures of variability appropriate for a variety of situations to be calculated, means and genetic variances for the life-history traits are tabulated in Table 2.

Release of hidden genetic variance: For all but one trait, the broad-sense heritability in the offspring generation was equal to or greater than that of the parental generation, suggesting release of hidden genetic variation upon sexual reproduction (data columns 1 and 2 in Table 1). Direct comparison of the total genetic variance (data column 4 in Table 1) revealed that the total genetic variance was higher in the offspring than in the parental generation for all traits. Significant increases of genetic variance were observed for all three body-size measurements (instar-specific body size, body size at birth and body size at first reproduction). On average, in units of the mean phenotypic variance in the two generations, sex caused the total genetic variance to increase by about 18%.

Genetic slippage: Changes in the genotypic means for the life-history traits were calculated in units of the mean phenotypic standard deviation in the two generations (data column 5 in Table 1). Except for age at first reproduction and clutch size, the relative genotypic value for all the traits decreased after sex. For three traits (instar-specific body size, body size at birth and adult growth rate), the decrease was significant, averaging 0.30.

Correlation patterns at genetic and phenotypic levels (Table 3): Negative genetic correlation between offspring number and offspring size, and between clutch size and investment in growth (increment in body-size), and positive genetic correlation between clutch size and age at reproduction would suggest the presence of genetic trade-offs for the evolution of the fitness traits. However, for none of these situations were significant trade-offs detected at either the additive or the total genetic levels. The signs of almost all genetic correlation estimates were opposite to that expected under

TABLE 1

Summary of the genetic parameter estimates for the life-table experiments

Character	H_p^2	H_o^2	h_{po}^2	$\Delta V_{G_{sex}}$	$\Delta\bar{g}$
Instar-specific body size	0.63** (0.11)	0.69** (0.10)	0.28 (0.15)	0.26* (0.12)	−0.20* (0.07)
Body size at birth	0.43** (0.07)	0.69** (0.06)	0.45* (0.14)	0.46* (0.19)	−0.33* (0.14)
Body size at first reproduction	0.40** (0.06)	0.61** (0.04)	0.23 (0.16)	0.31* (0.13)	−0.23 (0.15)
Juvenile growth rate	0.39** (0.08)	0.39** (0.08)	0.21 (0.23)	0.13 (0.18)	−0.16 (0.19)
Adult growth rate	0.44* (0.18)	0.46** (0.09)	0.29** (0.08)	+0.00 (0.01)	−0.36* (0.17)
Age at first reproduction	0.25** (0.08)	0.30** (0.09)	0.06 (0.22)	0.06 (0.21)	0.04 (0.11)
Clutch size	0.48** (0.10)	0.44** (0.07)	0.40* (0.17)	0.02 (0.08)	0.03 (0.07)

H_p^2 and H_o^2 are the broad-sense heritabilities in the parent and offspring generations respectively. h_{po}^2 is the narrow-sense heritability. $\Delta V_{G_{sex}}$ is the change of expressed total genetic variance after sex measured in units of the mean phenotypic variance in the parent and offspring generations. $\Delta\bar{g}$ is the slippage of the mean genotypic value following sex, in units of the mean phenotypic standard deviation in the two generations. The standard errors are given in parentheses. The standard errors for the broad-sense heritabilities were obtained by the TAYLOR expansion method (LYNCH 1985; DENG 1995), while those for the narrow-sense heritabilities were obtained by the method in FALCONER (1989).

For instar-specific body size, separate analyses were performed for each of the first seven instars, and the average of the instar-specific results is reported; for clutch size, the reported estimate was obtained in the same way using data for the first three adult instars.

Significance is judged by whether an estimate differs from 0 by more than two standard errors (* $P < 0.05$), or 2.56 standard errors (** $P < 0.01$).

The juvenile growth rate is calculated by [ln (body size at first reproduction) − ln (body size at birth)]/(age at first reproduction), where body size = body length from the top of the head to the base of the tailspine in mm. The adult growth rate is equal to [ln (body size of the third adult instar) − ln (body size of the first adult instar)]/(time between these two instars).

genetic trade-offs (Table 3). The only significant trade-off detected was for the total number of eggs of second and third clutches and the duration of the first two adult instars at the phenotypic level in the offspring generation. Interestingly, the total genetic correlation in the offspring generation is significantly greater than that in the parental generation for the number and the average size of offspring released from the second clutch, and nearly significantly smaller for the number of eggs in the first clutch and the time to maturity (P-value from bootstrapping analysis is 0.07), suggesting a tendency of more trade-offs in the parental generations. These observations, together with the observations of significant release of hidden genetic variance, suggest significant changes in the genetic (co)variation structure across sexual generations.

DISCUSSION

The present study investigated the consequences of sexual reproduction for a population's genetic architecture (genotypic means, genetic variances, and genetic correlations) for life-history traits. Substantial nonadditive genetic effects were revealed by an observed genetic slippage of the mean genotypic value in response to sex and by direct comparison of narrow- and broad-sense heritabilities. Substantial repulsion genetic disequilibria were revealed by significant changes of total genetic variances and total genetic correlations across generations. Theoretical results of LYNCH and DENG (1994) suggest that changes in genotypic means and variances in response to sex will almost always be opposite in direction to that promoted by clonal selection. Thus,

TABLE 2

Summary of the supplementary genetic parameter estimates for the life-table experiments

Character	$\bar{z}_p$	$\bar{z}_o$	V_G (P)	V_G (O)	V_A
Body size at birth	0.65 (0.01)	0.63 (0.01)	0.0023 (0.0004)	0.0052 (0.0005)	0.0029 (0.0010)
Body size at first reproduction	1.59 (0.02)	1.56 (0.02)	0.0059 (0.0017)	0.0110 (0.0020)	0.0038 (0.0022)
Juvenile growth rate	0.068 (0.001)	0.066 (0.001)	2.1E-5 (0.3E-5)	3.3E-5 (1.1E-5)	1.3E-5 (1.1E-5)
Adult growth rate	0.013 (0.002)	0.011 (0.001)	5.2E-6 (1.2E-6)	5.4E-6 (0.4E-6)	3.2E-6 (1.4E-6)
Age at first reproduction	12.36 (0.22)	12.42 (0.30)	0.48 (0.08)	0.60 (0.11)	0.12 (0.04)

$\bar{z}_p$ and $\bar{z}_o$ are the mean genotypic values in the parental and offspring generations respectively. V_G (P) and V_G (O) are the total genetic variances in the parental and offspring generations respectively, and V_A is the additive genetic variance. Standard errors are given in parentheses. Parameter estimates are not given for the composite traits in Table 1, such as instar specific body-size and clutch size, since the values for the mean and genetic variance differ considerably across component traits, such as the different instar specific body sizes and the first three clutch sizes. Body-size is measured in mm, and age in days.

TABLE 3

Summary of the correlations at additive genetic (r_A), total genetic (r_G), phenotypic (r_P) levels in the parent (p) and offspring (o) generations, and the change of r_G across sexual generations ($\Delta r_G = r_{G(o)} - r_{G(p)}$)

Traits	r_A	$r_{G(p)}$	$r_{G(o)}$	$r_{P(p)}$	$r_{P(o)}$	Δr_G
Number and average size of offspring released from the second clutch	0.20	0.05	0.72*	0.29*	0.45*	0.67*
Number of eggs in the first clutch and immature growth investment	−0.40	0.04	0.20	0.42*	0.66*	0.16
Total number of eggs of second and third clutches and mature growth investment	0.54*	0.61*	0.85*	0.85*	0.70*	0.23
Number of eggs in the first clutch and time to maturity	−0.50	−0.25*	−0.98*	0.06	0.35	−0.74
Total number of eggs of second and third clutches and duration of the first two adult instars	−2.45*	−0.30	−0.23	0.16	0.37*	0.07

All correlation estimates in the table are from the mean of the 1000 bootstrap samples. Significance is judged by the 2.5% or 97.5% quantiles of the distribution of the 1000 bootstrapping estimates.

Immature growth investment = first adult instar body size − newborn body size; mature growth investment = third adult instar body size − first adult instar body size.

Genetic correlation should be between −1.0 and 1.0 by definition. However, since r_A is estimated from parent-offspring covariance analyses, it is not a product-moment correlation, and such estimates may fall out of the range of [−1.0, 1.0] due to sampling error (FALCONER 1989; LYNCH and WALSH 1996).

for body size and growth rate, for which the genetic variance increases or the genotypic mean changes significantly after sex, the nature of clonal selection in the natural population can be inferred to have directional and/or stabilizing components. Technically, the increase of genetic variance implies that the second derivative of the log of relative fitness with respect to the phenotype is negative (SHNOL and KONDRASHOV 1993), which is equivalent to saying that selection is directional and/or stabilizing. During the clonal reproduction phase, the net effects of directional selection must have caused increases in instar-specific body sizes, body sizes at birth, and adult growth rate, since the mean of these characters decreased after sex.

The interpretation of our narrow-sense heritability (h^2_{po}) estimates as estimates of the relative magnitude of additive genetic variance assumes that mating is random. Although mating was effectively random with respect to the two polymorphic loci studied (PGM and PGI), it might not have been so with respect to the traits studied. Molecular marker studies in this and many other studies (HEBERT 1987; LYNCH and SPITZE 1994) indicate that inbreeding is extremely uncommon in Daphnia. It is conceivable that assortative mating for life-history traits (such as body size attributes) may occur, but there is no direct evidence for it. Positive assortative mating causes coupling gametic-phase disequilibria and inflates estimates of h^2_{po} (FALCONER 1989). If this were to occur, our conclusions about nonadditive genetic effects and repulsion disequilibrium would be rendered more conservative and thus stronger.

The present study is consistent with two previous studies (EBERT *et al.* 1993; LYNCH and DENG 1994) in that all three studies revealed significant nonadditive genetic effects. Although EBERT *et al.* (1993) did not detect any significant change of genetic variance upon sexual reproduction, with only 23 parent-offspring pairs, the statistical power of their study was relatively small. With about 90 parent-offspring pairs employed by LYNCH and DENG (1994) and by the present study, changes in genotypic means and genetic variances were observed readily. However, the direction of the change of genetic variances observed in the present study and in LYNCH and DENG (1994) differs, as the latter study detected a significant reduction of total expressed genetic variation in response to sex. Thus, the modes of genetic disequilibria (whether coupling or repulsion) and the nature of natural selection (whether stabilizing or disruptive) appear to differ between these two populations. These two contrasting results on the change of genetic variability suggest the complexity of natural selection among different populations.

The inferred stabilizing selection on body size in the present study is consistent with previous ecological work on the selection imposed upon cladoceran species by invertebrate and vertebrate predation (reviewed by LYNCH 1980). Generally, both of these selection forces operate simultaneously on permanent lake Daphnia populations, and they conflict. Selection from invertebrate predation favors larger body size, while selection from vertebrate predation favors smaller body size. Thus, the co-occurrence of both types of predation can impose stabilizing selection on body size. In the current study, the significant release of hidden genetic variance for body size supports the idea of an intermediate optimal body size, but directional selection may also be

responsible for the change in genetic variance (LANDE and ARNOLD 1983; SHNOL and KONDRASHOV 1993). Indeed, significant genetic slippage for instar-specific body size and body size at birth suggest the tendency for natural selection to increase body size, implying that in the study population invertebrate selection (mainly from copepods) predominates over vertebrate predation (mainly from fish). This implication is contrary to the general idea, based on circumstantial evidence, that predation pressure from fish predominates over that from invertebrates in permanent lakes (LYNCH 1980). However, a predominance of invertebrate predation in lakes harboring dense fish populations has been noted before in a study on size-specific mortality (LYNCH *et al.* 1981).

Results from our multivariate correlation analysis corroborate earlier results from Daphnia genetic studies (LYNCH 1984; SPITZE *et al.* 1991; LYNCH and SPITZE 1994), all of which have yielded many total genetic correlations between life-history traits that are opposite in sign to those expected under the antagonistic pleiotropy hypothesis (SIBLY and CALOW 1986; ROSE 1991; STEARNS 1992). These observations might arise from genotype-environment interaction caused by assaying individuals in a novel environment (SERVICE and ROSE 1985). Although it is almost impossible to mimic completely the conditions animals experience in the wild, genotype-environment interaction is minimized in the present study and in our previous studies (LYNCH and SPITZE 1994; LYNCH and DENG 1994) by raising animals in their natural source water, feeding them with a natural phytoplankton species, and setting photoperiod and temperature to those encountered in nature. Our empirical observations might also arise as a consequence of mutations influencing resource acquisition outnumbering those influencing resource allocation (LYNCH 1985; VAN NOORDWIJK and DE JONG 1986; HOULE 1991). Finally, SPITZE *et al.* (1991) suggested that if the signs of additive and total genetic correlations differ, then genetic constraints may conflict for the asexual selection phase and the overall evolutionary dynamics, which may help explain the empirical observations in the present study, and in LYNCH (1984) and SPITZE *et al.* (1991). Nevertheless, this kind of conflict is not suggested by the present study, since the signs of additive and total genetic correlations are identical in all cases, except for the number of eggs in the first clutch and immature growth investment (and for this pair of traits, no significant genetic correlation is involved).

Although the present study revealed a significant contribution to the genetic architecture from nonadditive genetic effects, it does not provide a basis for separating the individual contributions of dominance and epistasis. Quantifying and isolating different genetic variance components from the resemblance between relatives is notoriously difficult and generally assumes that populations are in genetic equilibrium (HARDY-WEINBERG equilibrium and gametic-phase equilibrium). However, the genetic equilibrium assumption has seldom, if ever, been checked. Many studies have attempted to isolate and quantify dominance genetic variance by comparing parent-offspring covariance estimates with those of full-sibs (FALCONER 1989; LYNCH and WALSH 1996). However, this approach assumes that epistatic variance is negligible and maternal effects are absent. Although direct and unbiased quantification of the magnitude of the epistatic genetic effects may not be possible for quantitative traits, their contribution to the genetic architecture is potentially large. If there are n loci underlying a trait, the number of additive genetic effects and dominance genetic effects is $O(n)$ (on the order of n, while that of the first-order (additive by additive) epistatic genetic effects is $O(n^2)$, and that of second-order (additive by additive by additive, and additive by dominance) epistatic genetic effects is $O(n^3)$. Thus, even if individual epistatic genetic effects are much smaller than additive and dominance genetic effects, their total contribution can be at least comparable. Finally, it is important to note that a small magnitude of nonadditive components of genetic variance implies little, if anything, about the presence of nonadditive genetic effects, since nonadditive genetic effects can contribute greatly to the additive component of genetic variance (FALCONER 1989; CHEVERUD and ROUTMAN 1995)

Nonadditive genetic effects and genetic disequilibria are not only relevant to large, randomly mating populations. They are particularly relevant to small inbreeding populations, which occur in important ecological contexts (such as habitat colonization by a few propagules), and in some biological conservation practices (captive breeding of a few individuals of an endangered species). In theory, as gene and/or genotype frequencies change with random genetic drift and/or inbreeding, the average effects of segregating alleles may change and some of the epistatic (COCKERHAM 1984a,b; GOODNIGHT 1987, 1988; COCKERHAM and TACHIDA 1988; TACHIDA and COCKERHAM 1989) and dominance (ROBERTSON 1952; WILLIS and ORR 1993) genetic variance may be converted to additive genetic variance. Some experiments have demonstrated inflated additive genetic variance after population bottlenecks (POWELL 1978; RINGO *et al.* 1985; BRYANT *et al.* 1986, 1990; MEFFERT and BRYANT 1991). Together with the present study and LYNCH and DENG (1994), these observations provide direct evidence for the role of nonadditive genetic effects and genetic disequilibria in determining the genetic architecture of populations.

With estimates of h^2_{po} (narrow-sense heritability estimated from parent-offspring regression), $\Delta\bar{g}$ (genetic slippage), h^2_p (broad-sense heritability in the parental generation) and h^2_o (broad-sense heritability in the offspring generation), the magnitude of the total selection in the wild can be estimated in principle. From a univar-

iate perspective, we first consider the influence of selection on the mean, then on the genetic variance.

From standard quantitative genetics, we have

$$R_S = h^2 \cdot S \tag{1a}$$

$$R_a = H^2 \cdot S \tag{1b}$$

where R_a and R_s are responses to selection due to total and additive genetic variability respectively. Specifically, R_a is the difference between the mean genotypic value after clonal selection (but before sex) and that before clonal selection, and R_s is the difference between the mean genotypic value after clonal selection and after sex and that before clonal selection. H^2 and h^2 are the mean broad- and narrow-sense heritabilities over the entire asexual period, respectively, and in a strict sense, both may change with the operation of selection and build up of genetic disequilibria. Here, we use the mean of the broad-sense heritabilities in the parent and sexually produced offspring generations as an approximation for H^2 [*i.e.*, $H^2 = (H_o^2 + H_p^2)/2$], and h_{po}^2 for h^2. S is the cumulative selection differential over the entire asexual period, which is the selection differential on the average member of the population. As introduced in the "Study organism" sub-section in MATERIALS AND METHODS, males are only present in the final generation, and they are clonal replicates of their mothers. We assume that in the final generation, selection differential of males (S_m) is equal to that of females (S_f). Violation of this assumption should not be problematic, since if there are many clonal generations before sex, $S = S_1 + S_2 + \ldots + S_{k-1} + [(S_m + S_f)/2]$ (where S_i is the selection differential in the ith clonal generation, and k is the number of clonal generations before sex). Hence, even if $S_m \neq S_f$, contribution of the last generation of selection to S is small. In our study population, the number of clonal generations before sex is estimated to be more than 10 (the population was established in early February, reproduced sexually in October, and a clonal generation takes about 2 wk). Since the genetic slippage of the mean is defined to be

$$\Delta\bar{g} = R_s - R_a, \tag{2}$$

by Equations 1a, 1b, and 2, we have

$$S = \Delta\bar{g}/(h^2 - H^2) \tag{3}$$

Note, intuitively or from the theory of LYNCH and DENG (1994), we know that if there were no nonadditive genetic effects (*i.e.*, $h^2 = H^2$), there would be no slippage. On the other hand, if there were no additive genetic effects, the slippage would be equal to the selection gain during the previous phase of clonal selection (*i.e.*, $H^2 \cdot S$). Equation 3 correctly predicts these results, a way to verify our derivation.

If a large population is at equilibrium, then for every cycle of sexual reproduction and clonal selection, the change of genetic variance by clonal selection should be balanced by that induced by mutation and sexual reproduction, *i.e.*,

$$\Delta V_{g_{sel}} = -(V_{g_{mut}} + \Delta V_{g_{sex}}) \tag{4}$$

where ΔV_{gsel} is the change of genetic variance due to selection; ΔV_{gmut} is the input of genetic variance from mutation per cycle; and ΔV_{gsex} is the change of genetic variance due to sexual reproduction. Since ΔV_{gmut} is positive and usually very small (LYNCH 1985, 1988), we have:

$$\Delta V_{g_{sel}} \cong -\Delta V_{g_{sex}} \tag{5}$$

Equation 5 implies that the change in genetic variance in response to sex provides a close estimate (opposite in sign) of the change of genetic variance due to natural selection.

The above estimation procedure is based on a simple univariate consideration. It is useful when applied to each of a set of uncorrelated traits or some single composite fitness measurements, such as the intrinsic rate of natural increase (CROW and KIMURA 1970). When a set of correlated traits are involved, the application of the above procedure to each trait respectively will likely result in biased estimation because of the indirect selection and correlated response. However, the same logic above will easily lead to the corresponding multivariate version of the estimation procedures. From the well-known multivariate selection-response equation of LANDE (1979) and LANDE and ARNOLD (1983), we have:

$$\mathbf{R}_s = \mathbf{G}_A\mathbf{P}^{-1}\mathbf{S} = \mathbf{G}_A\boldsymbol{\beta} \tag{6a}$$

$$\mathbf{R}_a = \mathbf{G}_T\mathbf{P}^{-1}\mathbf{S} = \mathbf{G}_T\boldsymbol{\beta} \tag{6b}$$

where $\mathbf{R}_s$ and $\mathbf{R}_a$ are the column vectors of responses to selection due to additive and total genetic variability, respectively; $\mathbf{G}_A$ and $\mathbf{G}_T$ are additive and total genetic variance-covariance matrices, respectively; and $\mathbf{P}^{-1}$ is the inverse of the phenotypic variance matrix. $\mathbf{S}$ and $\boldsymbol{\beta}$ are the column vectors of selection differentials and selection gradients. $\mathbf{S}$ contains the compound information of direct and indirect selection on the phenotypes. The elements of $\boldsymbol{\beta}$ ($\boldsymbol{\beta} = \mathbf{P}^{-1}\mathbf{S}$) describe the forces of selection operating directly on the traits [for detailed definition, see LANDE (1979) and LANDE and ARNOLD (1983)]. The column vector of genetic slippage $\Delta\bar{\mathbf{g}}$ is

$$\Delta\bar{\mathbf{g}} = \mathbf{R}_S - \mathbf{R}_a \tag{7}$$

by Equations 6a, 6b and 7, we have

$$\boldsymbol{\beta} = (\mathbf{G}_A - \mathbf{G}_T)^{-1}\Delta\bar{\mathbf{g}} \tag{8}$$

This is the corresponding multivariate version of Equation 3. Under the present experimental design, all the information of $\mathbf{G}_A$, $\mathbf{G}_T$ and $\Delta\bar{\mathbf{g}}$ are available; thus the selection gradients can be computed by Equation 8.

Although the multivariate technique may separate direct and indirect components of selection parameters

TABLE 4

The elements of the additive (G_A) and total genetic (G_T) variance-covariance matrices

	Body size at birth	Body size at 1st reproduction	Adult growth rate
		G_A	
Body size at birth	0.0029		
Body size at 1st reproduction	0.0018	0.0038	
Adult growth rate	2.8E-5	3.4E-5	3.2E-6
		G_T	
Body size at birth	0.0038		
Body size at 1st reproduction	0.0043	0.0085	
Adult growth rate	5.1E-5	7.7E-5	5.2E-6

in theory, in practice it might be compromised by the need to determine how many and what traits to be included in the analysis (LANDE 1979; LANDE and ARNOLD 1983). Since body size at birth, body size at first reproduction and adult growth rate are the three single characters exhibiting significant genetic slippage and/or change of genetic variability, the selection on them is likely to be of significance. We use Equation 8 to estimate the selection gradients and Equation 5 to approximate the change of genetic variance due to selection ($\Delta V_{g_{sel}}$). The computation of in the parent and offspring generations and $\mathbf{G}_A$ was described in MATERIAL AND METHODS. The $\mathbf{G}_T$ was taken to be the average of those in the parent and offspring generations. The $\mathbf{G}_T$ and $\mathbf{G}_A$ are summarized in Table 4. Elements of $\Delta\bar{\mathbf{g}}$ used were on the original scale (not, as in Table 1, standardized by the mean phenotypic standard deviations in the parent and offspring generations) and can be converted from data in Tables 1 and 2. $\boldsymbol{\beta}$ was then calculated and, together with $\Delta V_{g_{sel}}$, summarized in Table 5. With the caveat that only the three traits likely to be of importance were analyzed in the multivariate analyses, it is interesting to note that all the three characters were selected to increase, and their genetic variance was decreased by clonal selection. Since adult growth rate is a compound character of body size, these observations are consistent with the previous inference that body size is under conflicting selection pressures from vertebrate and invertebrate predation and that invertebrate selection overwhelms vertebrate predation, causing a directional component of selection for increase in body size.

TABLE 5

Summary of the estimates of natural selection parameters

Character	$\boldsymbol{\beta}$	$\Delta V_{g_{sel}}$
Body size at birth	0.41	−0.26
Body size at 1st reproduction	5.91	−0.46
Adult growth rate	4.90	−0.00

$\Delta V_{g_{sel}}$ is defined in Equation 4 and measured in the same units as $\Delta V_{g_{sex}}$ in Table 1; $\boldsymbol{\beta}$ is defined in Equation 6 and measured on the original scale of each trait. $\boldsymbol{\beta}$ can be interpreted, using body-size at 1st reproduction as an example, as such: holding all other traits unchanged, the relative fitness of the population will increase nearly fivefold with an increase of 1 mm for body-size at 1st reproduction.

With a purely additive genetic system, sexual reproduction may increase a population's evolutionary potential by releasing hidden genetic variance (LYNCH and GABRIEL 1983; CHARLESWORTH 1993; LYNCH and LANDE 1993). However, purely additive genetic systems may not exist at all. Selection works on the total outcome of all genetic interactions (the joint additive and nonadditive genetic effects underlying the genetic architecture), and advances different, but equally favorable combinations of genes. Segregation and recombination during sexual reproduction then disrupt these adaptive combinations and reassort genes into less favorable combinations, resulting in erosion of some advancement due to previous selection (genetic slippage). This phenomenon is equivalent to outbreeding depression (SHIELDS 1982) at the clonal level. When both genetic slippage and release of hidden genetic variance occur simultaneously, a critical question concerning a particular bout of sexual reproduction is as follows: Will this bout of sexual reproduction enhance a population's adaptability? More specifically, can the release of hidden genetic variance, which increases evolutionary potential, compensate for genetic slippage, which temporarily reduces a population's evolutionary response? A similar question arises when inbreeding depression is accompanied by an inflation of additive genetic variance when a population experiences population bottlenecks. Theoretical and empirical investigation of these issues needs to be pursued.

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