

Both Costs and Benefits of Sex Correlate With Relative Frequency of Asexual Reproduction in Cyclically Parthenogenic *Daphnia pulex* Populations

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ABSTRACT

Sexual reproduction is generally believed to yield beneficial effects via the expansion of expressed genetic variation, which increases the efficiency of selection and the adaptive potential of a population. However, when nonadditive gene action is involved, sex can actually impede the adaptive progress of a population. If selection promotes coupling disequilibria between genes of similar effect, recombination and segregation can result in a decrease in expressed genetic variance in the offspring population. In addition, when nonadditive gene action underlies a quantitative trait, sex can produce a change in trait means in a direction opposite to that favored by selection. In this study we measured the change in genotypic trait means and genetic variances across a sexual generation in four populations of the cyclical parthenogen *Daphnia pulex*, which vary predictably in their incidence of sexual reproduction. We show that both the costs and benefits of sex, as measured by changes in means and variances in life-history traits, increase substantially with decreasing frequency of sex.

THE evolution and continued pervasiveness of sexual reproduction despite its apparent costs (FISHER 1930; MULLER 1932; MAYNARD SMITH 1978) have been the focus of research for over a century. WEISMANN (1889) originally suggested that sex functions as a mechanism to produce selectable genetic variation through the shuffling of existing genetic material, an idea often widely accepted as one of the major explanations of the benefit of sex (BARTON and CHARLESWORTH 1998; BURT 2000). By this means, segregation and recombination facilitate the purging and fixation of deleterious and beneficial mutations, respectively (FISHER 1930; MULLER 1932, 1964), increasing the efficiency of natural selection and potentially population fitness. However, while sometimes creating new adaptive genetic combinations, sex can also disassemble previously successful multilocus genotypes (FISHER 1930; MAYNARD SMITH 1978; FELDMAN *et al.* 1980). If the underlying gene action is nonadditive, the breakup of these combinations by sexual reproduction can impose a cost in the form of recombination load (FISHER 1930; FELDMAN *et al.* 1980; CHARLESWORTH and BARTON 1996) or genetic slippage (LYNCH and DENG 1994), both of which refer to the offspring population being maladapted relative to the parent population.

A substantial body of theoretical research provides clear predictions of these potential costs and benefits,

but quantifying the effects of sex in an ecologically relevant manner has proved difficult. Laboratory manipulations of asexually reproducing organisms induced to reproduce sexually have added to our empirical understanding of the effects of sex. In particular, several recent laboratory experiments were able to attribute changes in fitness and rates of adaptation to sexual reproduction (COLEGRAVE 2002; KALTZ and BELL 2002; GODDARD *et al.* 2005). In *Chlamydomonas*, for example, sex resulted in increased fitness variance in sexual relative to asexual lines, and despite an initial decrease in mean fitness in the generations immediately subsequent to sex (recombination load), long-term fitness increased at a faster rate when there were larger increases in variance (KALTZ and BELL 2002). This work supports some Weismann–Fisher–Muller models of a short-term cost to sex accompanied by a long-term benefit due to increased variance (COLEGRAVE *et al.* 2002; KALTZ and BELL 2002). However, the extent to which these types of laboratory findings are applicable to natural populations is untested.

Measuring changes in the underlying genetic architecture of populations solely due to sex, specifically changes in genotypic means and genetic variances, is highly feasible in species that reproduce via cyclical or facultative parthenogenesis (LYNCH and DENG 1994; DENG and LYNCH 1996). The intervening period of asexual reproduction between sexual episodes enables beneficial combinations of genes to undergo selection, resulting in a buildup of genetic disequilibrium. Periodic sex

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breaks up these disequilibria, leading to changes in expressed genetic variance. Expressed genetic variance is predicted to increase if the population is experiencing repulsion disequilibria (nonrandom associations between genes of opposite effect) and decrease if there are coupling disequilibria (associations between genes of similar effect) (KONDRASHOV 1993; LYNCH and DENG 1994).

In addition to changes in genetic variance, sex can also result in a deleterious change in trait means. If there is nonadditive gene action involving epistasis or dominance underlying the genetic combinations selected during periods of asexual reproduction, recombination will result in a change in the genotypic mean of the selected traits in the direction opposite to that previously favored by selection (genetic slippage) (LYNCH and DENG 1994). Because these changes in variances and means are due to the accumulation of genetic disequilibria during the period of asexual reproduction, their magnitude across a sexual generation is predicted to be proportional to the length of the preceding asexual period (LYNCH and GABRIEL 1983). While less frequent sex should result in bursts of genetic variance promoting higher rates of evolution, it will also result in a greater short-term cost in terms of genetic slippage (LYNCH and DENG 1994).

We tested this hypothesis using four natural lake populations (CACERES and TESSIER 2004) of the cyclically parthenogenic microcrustacean *Daphnia pulicaria* that differ consistently and predictably in their frequency of sexual reproduction. Although all four populations are capable of sexual reproduction, the actual frequency of sex, previously calculated using the proportion of males and sexually reproducing females (CACERES and TESSIER 2004), spans an ~30-fold difference between the high- and low-sex populations (CACERES and TESSIER 2004). The *D. pulicaria* system offers extraordinary power because in addition to variation in the frequency of sex in nature, sampled individuals can be maintained clonally in the laboratory, allowing the essentially permanent propagation of wild-caught parental genotypes along with their sexually produced offspring. Both parent and offspring genotypes can thus be assayed simultaneously, at the same developmental stages in a common environment, ensuring that measured changes in means and variances across the sexual generation are a consequence only of changes in disequilibrium and not a result of environmental differences or selection on the offspring generation. In this study, we address the question of how the frequency of sex in natural populations affects the costs and benefits of recombination by quantifying the change in genetic variance and genotypic means for life-history traits in ~100 wild-caught parent-offspring pairs from each of the four lake populations. The life-history traits used in this study are extensively documented as undergoing selection pressure due to vertebrate and invertebrate predat-

ors, and resource competitors, in *Daphnia* populations (LYNCH 1977, 1980; MURTAUGH 1981; SPITZE 1991; DECLERCK and DE MEESTER 2003).

MATERIALS AND METHODS

Population and sampling: *D. pulicaria* are small planktonic Cladocera that reproduce by cyclical parthenogenesis; *i.e.*, individuals can reproduce both sexually and asexually. Populations reproduce asexually for extended periods ranging from weeks to years before engaging in a bout of sex, often in response to changing or deteriorating environmental conditions. One or two sexually produced eggs are deposited in a desiccation-resistant capsule, known as an ephippium, formed by modification of the carapace. The ephippia are readily identifiable, allowing sexually reproducing females to be easily distinguished from those reproducing asexually. Once released from the female, the eggs within the ephippium can remain dormant until receiving suitable hatching cues.

We sampled four lake populations of *D. pulicaria* from Barry County in southern Michigan, chosen because of their dramatic and consistent differences in the incidence of sexual reproduction (CACERES and TESSIER 2004). The difference in frequency of sex is believed to be due to ecological factors influencing the ability of animals to persist in the water column year-round. These populations are described in detail elsewhere (CACERES and TESSIER 2004), but briefly they are as follows: Little Long Lake (high sex), with ~15–30% of the population reproducing sexually each year; Bristol Lake and Warner Lake (both medium sex), with ~3–10% reproducing sexually every year; and Baker Lake (low sex), which reproduces asexually for multiple years before engaging in sexual reproduction.

Each lake population was sampled during the peak sexual periods by making 20–30 vertical tows of a standard plankton net across multiple locations in each lake. Collected individuals were sorted in the laboratory and ~1000 ephippial females (that had mated with males and were still carrying the ephippia) were isolated from each population into individual beakers. Once an ephippium was released from the female, it was placed under conditions to induce hatching: 14 hr light at 10°:10 hr dark at 7°, with periodic placement in 4° dark to stimulate development. One of the strengths of the *Daphnia* system is that all wild-collected females (parent genotype) and hatched individuals (offspring genotype) can be maintained via clonal reproduction in individual beakers in the laboratory until sufficient numbers of family pairs (parent and hatched offspring) are available. Each hatching sample was initiated with 10-fold more ephippia than needed so that parent-offspring pairs for the final assay could be randomly chosen from a larger sample. When both eggs from an ephippium hatched, one offspring was chosen randomly for the assay. This does not completely remove the possibility of within-lab or within-family selection, as some ephippia did not hatch and some hatched offspring died, but if any selective effect on particular parent-offspring genetic combinations systematically influenced changes in trait means or genetic variances, it most likely would have led to the loss of offspring with low fitness resulting in downwardly biased estimates of changes in means and variances. For each population, the life-history assay began when ~100 parent-offspring pairs were firmly established. On average the time from sampling the population to initiating the assay was ~2.5 months.

Life-history assay: Life-history assays were performed for each of the four populations separately, but parents and offspring within each population were assayed simultaneously.

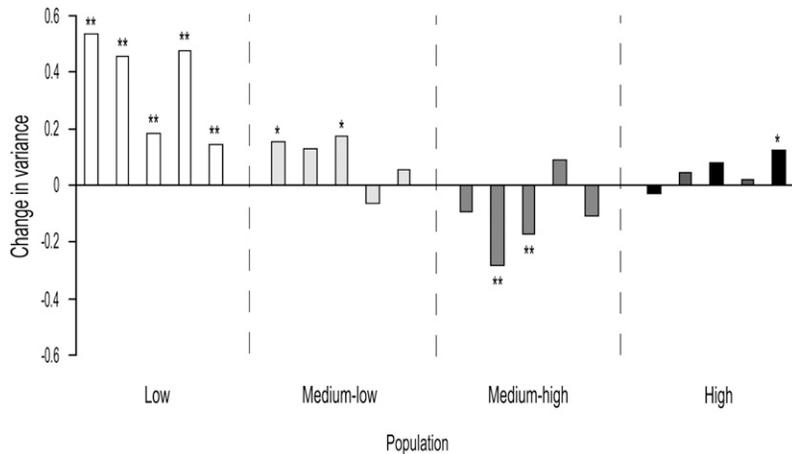


FIGURE 1.—Changes in expressed genetic variances, measured as the difference between the offspring genetic variance and the parent genetic variance, standardized by the average phenotypic variance. For each of the four populations, trait categories from left to right are size at birth, adult size, clutch size, adult age at stage, and juvenile growth rate. Significance is judged as whether an estimate differs from zero by >2.0 standard errors ($*P < 0.05$) or >2.6 standard errors ($**P < 0.01$).

Detailed descriptions of the standard life-history assay are given elsewhere (LYNCH *et al.* 1989; LYNCH and DENG 1994; DENG and LYNCH 1996). Briefly, prior to the assay of each population, three replicate clonal lines were established from each of the ~ 200 genotypes (100 parent and 100 offspring), for a total of 600 individuals per population assay. All replicate clonal lines were maintained for two generations in the experimental conditions prior to the assay to remove covariances due to maternal and grandmaternal environment effects. All four assays were done in standard experimental conditions of 12 hr light:12 hr dark and a constant temperature of 20° .

All individuals were assayed for a total of 22 fitness-related traits: body length at birth, at maturity, and at each of the first four clutches; age at maturity and at the first four clutches; numbers of offspring in each clutch; and growth rate from juvenile to maturity, from maturity to first clutch, and pairwise between each of the adult body stage measurements (PFRENDER and LYNCH 2000). Measurement of growth in this way is possible because *Daphnia* grow in discrete stages, such that the release of each clutch is coincident with the shedding of the old carapace and its replacement by a new, generally larger one.

Analyses: Prior to analysis all trait distributions were examined by population and generation using the Univariate procedure in SAS (SAS INSTITUTE 1999). One-way ANOVAs were used to estimate the phenotypic variances and their genetic and environmental components (LYNCH *et al.* 1989; LYNCH and DENG 1994) by setting mean squares equal to expected mean squares, using the model

$$Y_{ij} = u + A_i + E_{ij},$$

where Y_{ij} is the trait measured on the j th replicate of the i th clone, A_i is the genotypic value of the i th clone within a generation (parent or offspring), and E_{ij} is the residual deviation of the j th replicate of the i th clone. The within-clone variance, or variance among the replicates of each genetically distinct clone, provides a measure of the environmental variance. The variance among clones (genotypes) provides an estimate of the expressed genetic variance. The phenotypic variance (total variance) is the sum of the within- and among-clone variances. Standard errors were obtained using the delta method (LYNCH and WALSH 1998). Each trait was analyzed separately for parents and offspring in each of the four populations.

For each trait in each population, the change in expressed genetic variance and phenotypic trait means across generations (*i.e.*, the difference between parents and offspring due to sex) were calculated. For each trait, the change in expressed genetic variance (ΔV_g) due to sex was calculated as the genetic variance in the offspring generation (V_{go}) minus the genetic

variance in the parent generation (V_{gp}). This measure was standardized by the average phenotypic variance (V_p) of the parent and offspring generations: $\Delta V_g = (V_{go} - V_{gp}) / [(V_{po} + V_{pp}) / 2]$. Equality of genetic variances in the parent and offspring generations within each population was tested using F -tests, prior to standardizing the change in variance. The change in genotypic trait means ($\Delta \hat{g}$) due to sex was calculated as the difference between the offspring generation trait mean (\hat{g}_o) and the parent generation trait mean (\hat{g}_p) and was standardized by the average phenotypic standard deviation (SD_p) of the parent and offspring generations: $\Delta \hat{g} = (\hat{g}_o - \hat{g}_p) / [(SD_{po} + SD_{pp}) / 2]$. Significance of change in trait means between the parent and the offspring generations within each population was determined using a one-way ANOVA prior to standardizing the measure of change in means. All significance tests were Bonferroni corrected for multiple comparisons within each population (SOKAL and ROHLF 1995).

RESULTS

All four populations experienced a significant change in expressed genetic variance after sex for at least some of the life-history traits measured (Figure 1). In three of the four populations, sexual reproduction led to an overall increase in expressed genetic variance in the offspring generation relative to that seen in the parent generation. However, in the fourth population (medium-high sex), there was a statistically significant decrease in expressed genetic variance after sex for adult body size and clutch size and an observable although nonsignificant decrease in variance for most other traits (Figure 1).

A change in the genotypic means across the sexual generations was seen in all but one trait category across the four populations (Figure 2). The magnitude of genetic slippage was highest in the two lower-sex populations. It was statistically significant for size at birth, adult size, and clutch size in the lowest-sex population and for both size at birth and adult body size in the medium-low-sex population.

The aim of this study was not only to measure changes in variances and trait means due to sex, as these effects have been documented in other studies (LYNCH and DENG 1994; DENG and LYNCH 1996; VIA and SHAW 1996;

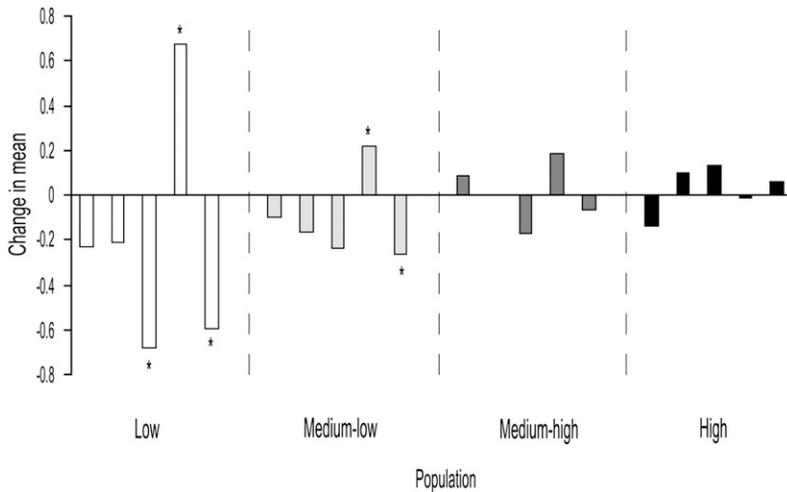


FIGURE 2.—Change in trait means measured as the difference between the offspring population trait mean and the parent population trait mean, standardized by the average phenotypic standard deviation. Population categories and significance estimates are as in Figure 1.

PFRENDER and LYNCH 2000), but also to examine how the population frequency of sex affects the magnitude of these changes. Because the measured changes in means and variances are both positive and negative (Figures 1 and 2), for Figure 3 we took the absolute value of all measures and calculated an average of the 22 traits within each population to specifically illustrate the relationship between the population frequency of sex and the magnitude, regardless of direction, of change in trait means and variances. The population with the highest frequency of sex experienced relatively little change in genetic variance, but as the population frequency of sex decreased the magnitude of change in variance across the sexual generation became progressively larger (Figures 1 and 3). As with the pattern of change in variance, the population with the highest frequency of sex experienced relatively small changes in genotypic trait means, but again as the population frequency of sex decreased the magnitude of the change in trait means across the sexual generation increased (Figures 2 and 3).

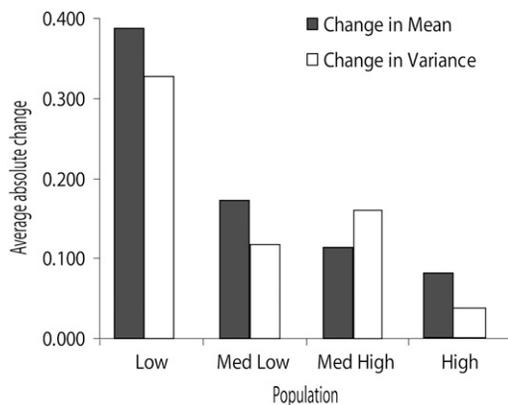


FIGURE 3.—Changes in variance and means were calculated individually for each trait as described previously, in units of phenotypic variance and phenotypic standard deviation, respectively. These values were converted to absolute numbers and then averaged across all 22 traits within each population.

DISCUSSION

In three of the four populations, the direction of change in expressed genetic variance is consistent with the idea that repulsion disequilibria accrue in populations under selection. The buildup of such disequilibria is a major assumption underlying theoretical explanations that invoke increased fitness variance as one of the major benefits of sexual reproduction (FELSENSTEIN 1974; KONDRASHOV 1993; BARTON 1995). Theory suggests that an increase in variance after sex will facilitate a faster rate of adaptation and higher fitness. This idea has been supported empirically in *Chlamydomonas*, where experimental populations manipulated to reproduce sexually were able to adapt to novel conditions at a faster rate than their asexual competitors (COLEGRAVE *et al.* 2002; KALTZ and BELL 2002). While we did not specifically measure fitness in these *Daphnia* populations in their natural habitat (*i.e.*, the assays were laboratory based), the study populations were clearly products of natural selection and the life-history traits measured here are well documented as being fitness related (LYNCH 1980; LYNCH *et al.* 1989). Given this, the expectation for these three populations is that the rate of adaptation and increase in fitness will be highest immediately after sex, but will gradually decrease until the next sexual episode. This pattern, illustrated in Figure 4, is expected to be the same in all three populations, but because the buildup of disequilibrium is less in populations with more frequent sexual reproduction, the magnitude of change in fitness variance across each sexual generation will also be smaller. The implication is that populations that undergo periodic asexual and sexual reproduction will experience cycles of expressed and hidden genetic variance and thus cycles of adaptive potential, and that the magnitude of these cycles will be inversely proportional to the population frequency of sex (LYNCH and GABRIEL 1983; KALTZ and BELL 2002).

Contrasting this increased adaptive potential and opportunity for long-term evolutionary change engen-

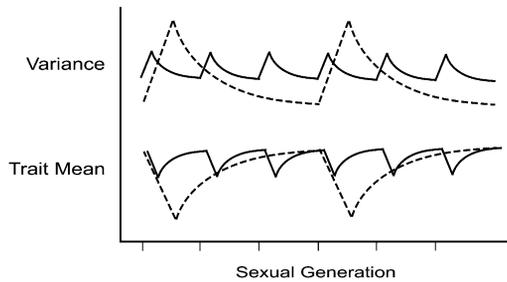


FIGURE 4.—Theoretical cycles of expressed genetic variance and genotypic trait means in relation to the population frequency of sexual reproduction. The magnitude of cycles of variance (top lines) and trait means (bottom lines) differs at high (solid line) and low (dashed line) frequency of sex. An episode of sex results in an increase in expressed variance and a slippage of trait means, whereas selection during the asexual period drives the mean back toward its optimum and reduces genetic variance. In cyclical parthenogens, this process leads to opposing cycles of variance and mean fitness, the magnitude of which increases with decreasing frequency of sex. This process can occur if directional selection is moving a trait mean toward a new optimum (not shown) or under stabilizing selection where the trait optimum does not change but genetic slippage moves the mean away from the optimum, re-imposing directional selection after each sexual generation.

dered by the release of genetic variance is the short-term cost in the form of maladaptive change in the mean phenotype of fitness-related traits. The impact of genetic slippage is substantial, with the average change across all traits within each of the four populations ranging from 0.05 to 0.3 phenotypic standard deviations (Figure 3) and that for individual traits being as high as 0.7 phenotypic standard deviations (Figure 2). As with the observed changes in genetic variance, the magnitude of slippage increased as the population frequency of sex decreased (Figure 3), implying that the previously discussed cycles of expressed genetic variance are diametrically opposed by cycles of increased and reduced mean fitness (Figure 4). This supports the contention that there are immediate fitness costs to sexual reproduction that offset the beneficial increase in expressed genetic variance.

This balance between the long-term benefit of increased variance and the short-term reduction in mean fitness is key in arguments for the maintenance of sex and the evolution of increased recombination (BARTON 1995). However, a decrease in expressed genetic variance after sex was observed in one of the four populations studied here and also twice in a previously studied population (LYNCH and DENG 1994; PFRENDER and LYNCH 2000), suggesting that in nature, selection sometimes favors genetic combinations that result in coupling disequilibrium. This result implies that the effect of sex in some populations may actually be an overall deleterious change in the fitness distribution.

This study quantifies the effect of reproducing sexually on accumulated genetic associations. Ideally, as

these genetic associations were both formed and broken down in nature, the consequent effects would have also been measured in nature. As this is not feasible in *Daphnia*, this study suffers the criticism that the genetic effects being measured may differ between the natural and the laboratory environments, and specifically, the sign of epistatic interactions may change across environments (AGRAWAL 2006). Clearly, as with previous attempts to measure the effect of sex on fitness (CHARLESWORTH and CHARLESWORTH 1975; COLEGRAVE *et al.* 2002; KALTZ and BELL 2002), this possibility cannot be ruled out. However, the results in this study are robustly supported by previous assays of *Daphnia* populations (LYNCH 1984; LYNCH and DENG 1994; DENG and LYNCH 1996; PFRENDER and LYNCH 2000) that consistently document the accumulation of disequilibria for life-history traits during periods of asexual reproduction and changes in expressed variance and trait means after sex.

Two assumptions underlying this study are that all genotypes within each population are equally likely to reproduce sexually during periods of sexual reproduction and that mating is random among sexually reproducing individuals. However, if there was within-population genotypic variation in the propensity to reproduce sexually, the sampled females from each population potentially have less accumulated disequilibria than their respective populations in entirety. This would result in our measures of the magnitude of change in means and variances for each population being smaller than if the samples from each population included individuals that invested in sex less frequently, but should have no effect on the relative magnitude of changes across populations. Our estimates of the cost of sex would also be rendered more conservative if the assumption of random mating were violated. Nonrandom mating in the form of sexual selection, where males with higher fitness have greater mating success, would result in the magnitude of slippage measured being smaller than if less-fit males contributed equally to the offspring generation (AGRAWAL 2001; SILLER 2001). There is currently no evidence for sexual selection in *Daphnia*; however, if this does occur in these populations there is no reason to expect it would vary among populations. Thus, while the magnitude of our measures of slippage may be affected, the pattern seen across populations due to the frequency of sex would not change.

Our results provide a firm empirical foundation for the theoretical expectation that the pervasiveness of nonadditive gene action may universally result in genetic slippage of mean phenotypes of adaptive traits following sexual reproduction (LYNCH and WALSH 1998). The observation that the magnitude of change in both genetic variances and means of fitness-associated phenotypes increases with decreasing frequency of sex reflects the potentially inextricable connection between these two opposing products of sexual reproduction. To resolve the paradox of the evolution

and maintenance of sexual reproduction, future work will need to evaluate whether the benefit of increased genetic variance is sufficient to offset the cost of genetic slippage.

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