APPARENT DIRECTIONAL SELECTION OF BODY SIZE IN *DROSOPHILA BUZZATII*: LARVAL CROWDING AND MALE MATING SUCCESS

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The multivariate phenotypic approach of natural selection suggests that selection can be adequately described by combining measurements of selection on phenotypic traits and estimates of heritabilities and genetic correlations (Lande and Arnold 1983). Under this framework (i.e., ignoring the consequences of pleiotropic effects of unconditionally deleterious mutations), two conclusions can be reached when directional selection on a genetically variable trait is detected: (1) that the population is not at an evolutionary equilibrium; or (2) that if the population is indeed at equilibrium, then there exists a “trade-off” between opposing selective forces (see also Robertson 1955). However, when external, nonheritable traits are included in this multivariate approach, a third conclusion is also possible, namely that an apparent selection differential can persist at equilibrium (i.e., without a genetic response) due to a correlation between the nonheritable and the heritable, phenotypic trait (Price et al. 1988; Kirkpatrick et al. 1990; Rausher 1992; van Tienderen and de Jong 1994).

Natural selection on body size has long been of interest in Drosophila as well as in other insects. Several laboratory studies have shown a positive phenotypic correlation between adult body size and fitness components in Drosophila (Partridge and Fowler 1993 and references therein), and Wilkinson (1987) provided indirect evidence that the selection favoring large adult flies is countered by the advantage of faster developing larvae that produce genetically smaller adults. Subsequently, Partridge and Fowler (1993), and Santos et al. (1992a, 1994) showed that *D. melanogaster* flies artificially selected for large body size had a longer development time and a lower viability under high larval densities than their corresponding control lines. The results of these studies show that body size is heritable and suggest that there is a trade-off between the performance of adult and juvenile stages.

A positive phenotypic correlation between male size and mating success has also been found in the field for several *Drosophila* species (Partridge et al. 1987b; Markow 1988; Santos et al. 1988, 1992b; Taylor and Kekic 1988; James and Jaenike 1992; Markow and Ricker 1992). Wild flies are usually highly variable for body size. Even though there is a genetic component to this variability, much of it is environmental in origin. Thus, Prout and Barker (1989) and Santos et al. (unpubl. manuscript), working with *D. buzzatii*, showed that approximately 50% of the phenotypic variance in body size is due to differences between rot means (i.e., individual rotting cactus cladodes used as breeding sites), and more than 95% of the between-rot component and 70% of the within-rot component of the phenotypic variance is attributable to environmental causes, most likely differences in larval food supply and fluctuations in temperature across rots. It is thus quite possible that the mating success of a wild male, which is presumably a function of the time it spends in outcompeting other males and in pursuing females (Partridge et al. 1987a), will be influenced by external conditions such as the quantity and energetic quality of food consumed by the individual at the larval stage that, in turn, has effects on its final size.

Leibowitz et al. (1995) have recently addressed whether the phenotypic differences between wild caught mating and single *D. buzzatii* males could be attributable to genetic differences between the samples. Though the laboratory-reared progeny of the mating males were on average larger (but not significantly so), less phenotypically variable, and developed faster than those of single males, their results did not provide clear evidence for directional selection acting on the genetic component of body size, and could be explained by assuming that the mating males carry a lower than average frequency of generally deleterious mutations that affect body size, mating success, and development time. This last interpretation would favor the view that a mutation-selection balance of deleterious alleles with pleiotropic side effects is maintaining additive genetic variation in phenotypic traits (Barton 1990; Kondrashov and Turelli 1992; Gavrilets and de Jong 1993; Caballero and Kightley 1994).

It was the aim of the present study to determine the effect of environmentally induced variation of body size on male mating success in *D. buzzatii*, and to test whether or not this trait responds to phenotypic selection when environmental factors may affect both phenotype and fitness. Briefly, I will show that a male’s body size (as measured by wing length) has no impact on mating success in competition among males that grew up under uncrowded conditions, but that size is important among males that grew up under crowded conditions. The phenotypic directional selection on wing length, however, did not translate into a genetic response when the distributions of body size among the offspring were compared.

**Materials and Methods**

The *D. buzzatii* flies used in the experiments originated from 40 isofemale strains derived from Carboneras (Almería,
southeastern Spain) in September 1993. A large outbreeding population was founded in October 1994 and maintained since then by serial transfer (Mueller 1985), at one-week intervals, in 130-mL bottles with standard commal-agar-yeast food at 25°C (12:12 light/dark cycle) and uncontrolled humidity. The outbred stock was cultured for approximately six generations before the start of the experiments in February 1995.

In this study, wing length, measured from the proximal junction of fourth and fifth longitudinal veins to the intersection of the third longitudinal vein with the distal wing margin (see Leibowitz et al. 1995), was used as an indicator of male size. Experiments conducted to determine the mating success of males were performed at room temperature (22–24°C) in groups of 15 nonvirgin but receptive females (see below) and 20 virgin males introduced into a 500-mL Erlenmeyer flask (Starmer et al. 1988). Females were introduced first and 10 min afterwards, the males. As frequent female remating is a characteristic feature of Drosophila species of the repleta group (Markow 1982, 1985; Barbadilla et al. 1991), I used a 0.75:1 female : male ratio because under field conditions the number of males seeking mates is likely to exceed the number of receptive females, and the intensity of male-male competition is presumably stronger as the receptive female:male ratio decreases (Sharp 1982). Each mating pair was gently removed by aspiration after half of the males had mated, so each mating trial provided 10 “winners” and 10 “losers.” Throughout the experiments CO2 was used when anaesthesia was necessary. The transfers of flies that initiated the mating trials were performed without anaesthetization.

Two different sets of experiments were carried out. In the first experiment, all flies were reared at 25 ± 0.1°C under controlled low-density conditions. This was achieved by placing 80 first instar larvae (± 4 h) in vials (108 mm depth, 30 mm diameter) containing 20 mL of fresh food. Emerging flies were collected daily, separated by sex, and the males to be tested were stored in groups of 20 flies in 130-mL bottles at 25°C. The females were kept with the same number of males for the first four days after emergence, after which the males were discarded and the females stored in groups of 15 and transferred to bottles with fresh food every day until the start of the experiment. This procedure allows the use of receptive but somewhat “choosy” females. Dead flies were replaced with new ones of the same age to maintain a constant number of adults in each bottle. All flies were 7–10 d old when tested, and all groups of competing males were matched for age. Copulation time in D. buzzatii ranges from about 1–3 min (Patterson 1947; Spieth 1952; Barker and Fredline 1985), and a mating was assumed to take place if the male mounted in the normal position and remained there for at least 20 s. Twenty-five trials, giving a total of 250 nonmating and 250 mating males, were performed. It took usually no more than 30–45 min until 10 males had courted and mated in each test, so four to five tests were made each day and the whole experiment was finished within six days. The males were fixed in 3:1 alcohol : glycerol. One wing was measured for each of the 500 males.

In the second experiment, all procedures were the same as above, but the males used in the mating tests were daily collected from 130-mL culture bottles, with high larval densities, during 10 days from the time of first adult emergence. This resulted in an increase in size variability that was, presumably, mainly environmental in origin. The experimental males were 6–11 d old when tested, taken at random from different culture bottles initiated on different days, placed together in groups of 20 in 130-mL bottles, starved for 2 h before each test by transferring them into empty bottles plugged with wet cotton at 25°C, and were not matched for age. Field experiments at Carboneras have shown that starved males (and females) are the norm, rather than the exception, throughout most of the year, but probably not during the fruit season from August to November (Santos et al. 1992b, unpubl. obs.). The females used in each mating trial were reared under controlled low-density conditions, were nonvirgin but receptive, and stored together in 130-mL bottles at 25°C as previously described.

A sample of nonmating (S) and mating (P) males from the second experiment were individually crossed (n = 198 of S and n = 187 of P) to four well-fed virgin females between four and six days old. This was done because if crowding has any effect on male mating success as related to body size, a test of the hypothesis that the phenotypic selection produces a response requires estimation of genetic parameters. Flies were left to mate for 48 h, after which time the males were fixed in 3:1 alcohol : glycerol and the females transferred to a new vial. For practical considerations, I had previously decided to obtain data for two families per male (sire), and to achieve a balance design so that the data could be readily analyzed by using a standard least-square (ANOVA) procedure (see below). Twenty eggs (0–12 h old) from each of two or three randomly chosen females mated to each male were individually placed in a vial with 5 mL of food at 25 ± 0.1°C. Emerging progeny were removed from the vials and fixed in alcohol and glycerine. Two random sons from two females per male that produced two or more male progeny were measured for wing length.

The final dataset was a balanced two-way nested classification for S (154) and P (152) males. All data were In-transformed. The model of analysis is

\[ y_{ij} = \mu + \alpha_i + \delta_{(i)} + e_{ij}, \]  

where \( \mu \) is the overall grand mean, \( \alpha_i \) is the random effect of the ith male (sire), \( \delta_{(i)} \) is the random effect of the jth female (dam) within the sire i, and \( e_{ij} \) is the residual error associated with the ln (wing length) of the ijth individual. Least-squares (ANOVA) estimates of the components of variance and their standard errors were obtained following Becker (1984). Several estimates of heritability of wing length were made for each sample. Heritability in the offspring environment was estimated from the covariance between half-sibs

\[ \hat{h}^2_{(O)} = \frac{4\hat{\sigma}^2_f}{\hat{\sigma}^2_f + \hat{\sigma}^2_{h} + \hat{\sigma}^2_c} = \frac{V_{A,O}}{V_{P,O}} \]  

and full-sibs

\[ \hat{h}^2_{(F)} = \frac{2(\hat{\sigma}^2_f + \hat{\sigma}^2_{h})}{\hat{\sigma}^2_f + \hat{\sigma}^2_{h} + \hat{\sigma}^2_c} = \frac{V_{A,F}}{V_{P,F}} \]  

(“caret notation” denotes an estimator of a parameter). I am
Table 1. Mean wing lengths (in mm) in the two experiments.

<table>
<thead>
<tr>
<th>Group</th>
<th>N</th>
<th>Mean ± SD</th>
<th>F*</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Optimum density</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>All males</td>
<td>500</td>
<td>2.093 ± 0.049</td>
<td>0.99 ± 1.25</td>
<td>0.319 ± 0.056</td>
</tr>
<tr>
<td>Nonmating</td>
<td>250</td>
<td>2.091 ± 0.052</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mating</td>
<td>250</td>
<td>2.095 ± 0.046</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Uncontrolled density</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>All males</td>
<td>500</td>
<td>1.871 ± 0.103</td>
<td>42.91 ± 1.28</td>
<td>&lt;.001 ± 0.038</td>
</tr>
<tr>
<td>Nonmating</td>
<td>250</td>
<td>1.842 ± 0.105</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mating</td>
<td>250</td>
<td>1.900 ± 0.093</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mating (S)</td>
<td>154</td>
<td>1.841 ± 0.110</td>
<td>22.93 ± 1.52</td>
<td>&lt;.001 ± 0.011</td>
</tr>
<tr>
<td>Matings (P)</td>
<td>152</td>
<td>1.895 ± 0.089</td>
<td></td>
<td></td>
</tr>
<tr>
<td>All offspring</td>
<td>1224</td>
<td>2.086 ± 0.052</td>
<td>0.34 ± 1.04</td>
<td>0.559 ± 0.526</td>
</tr>
<tr>
<td>S-males offspring</td>
<td>616</td>
<td>2.085 ± 0.053</td>
<td></td>
<td></td>
</tr>
<tr>
<td>P-males offspring</td>
<td>608</td>
<td>2.088 ± 0.051</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*F*-ratios for average comparisons of ln (wing length) between mating and nonmating samples are based on standard one-way ANOVAs. For the offspring, the *F*-ratios compare the distribution of ln (wing length) of P-males to that of all males. Significance of variance ratios for mating and nonmating males were empirically tested by sampled randomization tests (see text for details).

assuming that nongenic paternal effects on offspring wing length are negligible, which is probably the case. Following Riska et al. (1989) three estimates of the narrow sense heritability in the parental environment were made. The first,

\[ \frac{\sigma^2_{\hat{h}^2}}{V_{A_{(O)}}} = \frac{V_{A_{(O)}}}{V_{P(F)}} \]  

(4)

is the ratio of the additive genetic variance in the offspring environment to the phenotypic variance in the parental environment and assumes that additive genetic variance does not differ between environments. The second, \(2\hat{\beta}_{O,F}\), is the twice the offspring-father regression coefficient and assumes no genotype-environment interaction (i.e., no differences in additive genetic variances and perfect correlation of the expression of genotypes in the two environments). The third estimate is

\[ V_{P(F)} \cdot (2\hat{\beta}_{O,F})^2 = \gamma^2 \hat{h}^2, \]  

(5)

which is a lower bound of the heritability in the parental environment and is available whenever the additive genetic variance in the offspring environment is higher than zero, but is an underestimate whenever the additive genetic correlation across environments (\(\gamma\)) is less than one.

Results

Mating Success and Environmental Conditions

No attempt was made to test for differences between replicated mating trials, and summary statistics are given in Table 1 for both experiments. No deviation from normality was detected in the distributions of ln (wing length) in any case (Kolmogorov-Smirnov tests; Optimum density: \(D = 0.028, P > 0.05\); uncontrolled density: \(D = 0.037, P > 0.05\)). When the experimental males were reared under optimum density, no statistically significant differences were detected in the distribution of wing length between mated versus unmated. However, when size variation was increased as a result of the differences in crowding conditions and/or the decline in size as the adult emergence proceeded in the culture bottles (the amount of phenotypic variation in the second experiment was approximately 4.4 times greater than in the first experiment), larger males had higher mating success and were phenotypically less variable than smaller, nonmating males.

The experiments involved a finite population of individuals, and the mating tests were designed so that the individuals in copula were removed from the population until half of the males had mated. Tests for the significance of mean wing length between mating and nonmating males in Table 1 are based on the comparison of the two groups, but these means are clearly not obtained from independent samples. However, the same conclusion is reached when comparing the average wing length before and after selection (see Endler 1986, pp. 171–173). Thus, in the second experiment the standardized selection differential was \(i = 0.28\) (\(t\)-test = 3.78, \(P < 0.001\)) for the whole sample of males (\(n = 500\)), or \(i = 0.27\) (\(t\)-test = 2.82, \(P = 0.005\)) for the subsample of males that were used as sires. The differences in variance between mating and nonmating males were tested by means of sampled randomization test after performing 5000 random partitions (Sokal and Rohlff 1995, pp. 808–809), and translate to a proportional change in the variance of \(j^2 = 0.22\) for the whole sample of males (\(j^2 = 0.29\) for the subsample of males used as sires) when the experimental males were reared under uncontrolled densities.

No statistically significant differences were detected in the distribution of wing length between the progenies of S- and P-males. It seems, therefore, that the phenotypic differences observed in the parental generation for wing length do not translate into differences in the offspring generation.

Variance Components and Heritability Estimates

ANOVA estimates of variance components (between sires, between dams, and error) are given in Table 2. Delete-one-sire family jackknife data resampling was carried out because it provides a robust test of significance to detect the genetic components of variance (Mitchell-Olds and Bergelson 1990). Dominance variance and/or maternal effects were estimated as \(\hat{\sigma}^2_{dm} = \hat{\sigma}^2_{a} - \hat{\sigma}^2_{r}\), and seem to be of little importance in both samples.

Estimates of the components of genetic variation and heritabilities are shown in Table 3. All estimates of heritability
Table 2. ANOVA estimates of variance components for wing length (in \( \ln \text{mm}^2 \times 10^6 \)). \( \sigma^2_S \), \( \sigma^2_D \), \( \sigma^2_{DE} \), and \( \sigma^2_e \) represent components of variance attributable to sires, dams, dominance and/or maternal effects, and within family variation, respectively. Jackknife estimates are from a delete-one-sire family jackknife. Confidence limits were obtained from normal-approximation jackknife interval estimators (Sokal and Rohlf 1995, pp. 820–823).

<table>
<thead>
<tr>
<th>Variance component</th>
<th>Direct estimate</th>
<th>Jackknife estimate</th>
<th>Upper and lower 95% limits</th>
<th>Lower (one-tailed) 95% limit</th>
</tr>
</thead>
<tbody>
<tr>
<td>S-males</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>( \sigma^2_S )</td>
<td>0.785875</td>
<td>0.785874</td>
<td>1.430158</td>
<td>0.141590</td>
</tr>
<tr>
<td>( \sigma^2_D )</td>
<td>1.292243</td>
<td>1.292231</td>
<td>2.209897</td>
<td>0.374565</td>
</tr>
<tr>
<td>( \sigma^2_{DE} )</td>
<td>0.506369</td>
<td>0.506357</td>
<td>1.872039</td>
<td>-0.859325</td>
</tr>
<tr>
<td>( \sigma^2_e )</td>
<td>4.357827</td>
<td>4.357938</td>
<td>5.125795</td>
<td>3.590082</td>
</tr>
<tr>
<td>P-males</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>( \sigma^2_S )</td>
<td>0.752569</td>
<td>0.752546</td>
<td>1.480474</td>
<td>0.024618</td>
</tr>
<tr>
<td>( \sigma^2_D )</td>
<td>0.843361</td>
<td>0.843343</td>
<td>1.602296</td>
<td>0.084564</td>
</tr>
<tr>
<td>( \sigma^2_{DE} )</td>
<td>0.090792</td>
<td>0.090884</td>
<td>1.331509</td>
<td>-1.149741</td>
</tr>
<tr>
<td>( \sigma^2_e )</td>
<td>4.297934</td>
<td>4.297912</td>
<td>4.969265</td>
<td>3.626559</td>
</tr>
</tbody>
</table>

are statistically significant, clearly indicating that there is substantial additive genetic variation for wing length on which sexual selection could act. For both samples, I found that \( \gamma^2 \sigma^2_S > 2| \beta_{OF} | \sigma^2_{DE} \), a situation that shows that the additive genetic variance was larger in the parental environment than in the offspring environment (Riska et al. 1989). If we assume that wing length was the only target of selection, and that gene action is additive (which seems to be the case), the expected difference between the mean of the progenies of P-males to that of the offspring population is given by \( R = \gamma^2 \sigma^2_S \), where \( S \) is the selection differential that describes the relationship between the parental phenotypes and fitness (Falconer 1981). Using the difference in means between the phenotypic value of the mating males to that of all sires (in \( \ln \text{mm} \)), and the lower bound estimate of the heritability in the parental environment (\( \gamma^2 \sigma^2_S \) = 0.281), \( R = 0.0022 \). The probability of rejecting the null hypothesis of no differences between the offspring means would be approximately 0.87 (two-tailed test). This power point indicates, by definition, that most of the trials would give a statistically significant F-value.

A potential problem with this experiment, and with any experiment that looks at the effect selection of parents in one environment has on their progeny in another environment, is the presence of genotype-environment interaction. Note, however, that in the present situation the cross-environmental additive genetic correlation (\( \gamma \)) is positive because the offspring-father regression (2\( \beta_{OF} \)), which is a linear function of the cross-environment correlation (Riska et al. 1989), is positive and statistically significant (Table 3). The predicted response (\( R = \gamma^2 \sigma^2_S \gamma \)) is also a linear function of the cross-environment correlation, and the value \( R = 0.0022 \) is a lower bound of the predicted response because \( \gamma^2 \sigma^2_S \gamma \) in this case. Accordingly, it seems reasonable to conclude that genotype-environment interaction is not masking the potential genetic response that would have been detected if selection was acting on body size and the offspring were reared under conditions similar to that found by their parents. The important conclusion to be obtained is, therefore, that there seems to be no genetic correlation underlying the phenotypic correlation seen between body size and male mating success in spite of substantial heritability for wing length.

Table 3. ANOVA estimates of genetic variance components (±SE) for wing length (in \( \ln \text{mm}^2 \times 10^6 \)) and heritabilities.

<table>
<thead>
<tr>
<th>Component</th>
<th>S-males</th>
<th>P-males</th>
</tr>
</thead>
<tbody>
<tr>
<td>( V_{A(S)} )</td>
<td>3.1435 ± 0.3474</td>
<td>3.0103 ± 0.3085</td>
</tr>
<tr>
<td>( V_{A(D)} )</td>
<td>4.1562 ± 0.4302</td>
<td>3.1919 ± 0.3827</td>
</tr>
<tr>
<td>( V_{P} )</td>
<td>6.4359 ± 0.3890</td>
<td>5.8939 ± 0.3541</td>
</tr>
<tr>
<td>( \gamma^2 )</td>
<td>35.8388 ± 4.0579</td>
<td>22.2996 ± 2.5413</td>
</tr>
<tr>
<td>( h^2_{(S)} )</td>
<td>0.488 ± 0.216</td>
<td>0.511 ± 0.209</td>
</tr>
<tr>
<td>( h^2_{(D)} )</td>
<td>0.646 ± 0.121</td>
<td>0.542 ± 0.120</td>
</tr>
<tr>
<td>( h^2_{(P)} )</td>
<td>0.088 ± 0.039</td>
<td>0.135 ± 0.055</td>
</tr>
<tr>
<td>( 2\beta_{OF} )</td>
<td>0.157 ± 0.034</td>
<td>0.173 ± 0.041</td>
</tr>
<tr>
<td>( \gamma )</td>
<td>0.281</td>
<td>0.222</td>
</tr>
</tbody>
</table>

\( ^a \) From the 154 S-males and the 152 P-males in Table 1.

\( ^b \) SE obtained as \( \sqrt{\frac{V_{A(S)}}{V_{P}}} \).

Discussion

The results presented here for the first experiment suggest that when a group of well-fed \textit{D. buzzatii} males are supplied with females, little or no phenotypic selection for body size will occur. This finding agrees with previous results where size was measured as thorax length and virgin females were used in the mating tests with a 1:1 receptive female: male ratio (Barbadilla 1992). Wild flies, however, are not likely to experience optimum growing conditions, and the question was raised to what extent an evolutionary response of body size is dependent on larval rearing density. In the second experiment, the phenotypic variance in body size was environmentally increased by using males that had grown under conditions of larval crowding and had emerged at different intervals of time. Under these circumstances, larger males had higher mating success than smaller ones, in accordance with what has been previously found in the laboratory for \textit{D. melanogaster} (Partridge and Farquhar 1983). Therefore, the importance of male mating success as a component of fitness in \textit{D. buzzatii} seems to be dependent on the set of conditions encountered by flies during the growing period.

The most important finding from the present study was that the phenotypic selection for large adult body size detected in the second experiment, and comparable to that found in the natural population where the amount of phenotypic vari-
ation is approximately six times greater than that in the optimum laboratory environment (Santos et al. 1988, 1992b; Leibowitz et al. 1995), did not result in a different average body size between the offspring of mating and nonmating males in spite of substantial additive variation for wing length. This result apparently contradicts the genetic correlation between male mating success and body size detected by Wilkinson (1987) for D. melanogaster. He observed that the mean wing length of mating males was significantly higher than the mean of all males in the base population, and that the offspring of inseminated flies that had mated in chambers where 80 males were present had longer wings compared with the offspring of females mated with randomly chosen males. It was implied from these data that the offspring of larger males had higher preadult mortality. The results are, however, open to alternative interpretations. Because the larval densities used by Wilkinson were unlikely to be very high (see also Partridge and Fowler 1993), a mortality difference could take place only in the case that flies with lower fitness had a higher than average frequency of unconditionally deleterious alleles that might have pleiotropic effects on body size. Alternatively, variation in larval densities across vials in the “choice” and “no choice” females allowed 24 h to oviposit could result in a difference between samples for average body size. In any case, it is also possible that the situation in D. melanogaster is different than that in D. buzzatii. In fact, there is some evidence from sibling analyses of a negative genetic correlation between thorax length and developmental time in D. buzzatii (J. S. F. Barker pers. comm. 1995).

Zamudio et al. (1995) have shown that male territorial success in D. melanogaster is affected by the developmental temperature at which the flies have been raised, independent of their body size. They suggest that size per se may be a “red herring” with respect to territorial success, and the results in the present paper show that this is indeed the case for mating success in D. buzzatii. As well as decreasing adult size, larval crowding also increases the concentration of waste products in Drosophila cultures (Botella et al. 1985). The latter flies to emerge from a crowded culture are not only smaller, but probably have also experienced a lower quality environment and might be less vigorous. A positive phenotypic correlation between body size and general vigor could explain the lack of a genetic response for body size when comparing the progeny of the S- and P-males. In any case, what is clear here is that body size does not seem to be the target of selection, and the question is open to whether or not the same might be true in wild populations.

If much of the genetic variance for body size in the natural population is contributed by recessive or partially recessive deleterious alleles that tend to decrease the genotypic value of the trait (e.g., by reducing the efficiency of enzymes involved in regulatory pathways that, in turn, might affect growth rate), wild males successful at mating are likely to be larger and to produce fitter and less variable offspring than unmated males. It might explain the reduction we detected in both the phenotypic variance and the dominance component in the offspring of wild mating males when compared to that of single males, and the negative genetic correlation between size and development time in the unmated males (Leibowitz et al. 1995). This last finding is clearly inconsistent with what we would expect if there were conflicting effects of genetic variation for body size on larval and adult performance. The flies used in the present experiments were obtained from isofemale strains kept in isolation for a number of generations (approximately 20) before establishing the outbred stock (see materials and methods). The increase in consanguinity in the isofemale strains has probably purged the populations of deleterious alleles with relatively large effects, and most of the genetic variance for body size in the stock might be due to the segregation of additive mutants with little or no effects on fitness.

In conclusion, no differential mating success with respect to body size is detected when D. buzzatii males are reared under optimal conditions, but larger males are more successful at mating than smaller ones when the flies are grown under conditions of larval crowding. The distribution of wing length in their progeny are not, however, statistically different in spite of substantial heritability. Whether body size reflects an evolutionary compromise between the conflicting effects of genetic variation on larval and adult performances in Drosophila remains to be demonstrated, but our data suggest no causal relationship of body size to fitness.

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