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THE RESPONSE TO SELECTION FOR FAST LARVAL DEVELOPMENT IN *DROSOPHILA MELANOGASTER* AND ITS EFFECT ON ADULT WEIGHT: AN EXAMPLE OF A FITNESS TRADE-OFF

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Abstract.—A selection experiment using *Drosophila melanogaster* revealed a strong trade-off between adult weight and larval development time (LDT), supporting the view that antagonistic pleiotropy for these two fitness traits determines mean adult size. Two experimental lines of flies were selected for a shorter LDT (measured from egg laying to pupation). After 15 generations LDT was reduced by an average of 7.9%. The response appeared to be controlled primarily by autosomal loci. A correlated response to the selection was a reduction in adult dry weight: individuals from the selected populations were on average 15.1% lighter than the controls. The lighter females of the selected lines showed a 35% drop in fecundity, but no change in longevity. Thus, there is no direct relationship between LDT and adult longevity. The genetic correlation between weight and LDT, as measured from their joint response to selection, was 0.86. Although there was weak evidence for dominance in LDT, there was none for weight, making it unlikely that selection acting on this antagonistic pleiotropy could lead to a stable polymorphism. In all lines, sex differences in weight violated expectations based on intrasex genetic correlations: Females, being larger than males, ought to require a longer LDT, whereas there was a slight trend in the opposite direction. Because the sexual dimorphism in size was not significantly altered by selection, it appears that the controlling loci are either invariant or have very limited pleiotropic effect on developmental time. It is suggested that they probably control some intrinsic, energy-intensive developmental process in males.

Key words.—Body size, development time, fecundity, fitness trade-off, genetic correlation, life-history evolution, longevity, selection.

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Underpinning many evolutionary arguments is the concept that species trade off one ability as another is improved. Within a species, understanding such trade-offs can provide insight into how the resulting stabilizing selection influences both the optimum value of a trait and its genetic variability. However, demonstrations of such negative genetic correlations for fitness-related traits have proved surprisingly elusive (see Bell and Koufopanou 1986; Stearns 1992), and many of these have been focused on a single problem, the trade-off between early fecundity and longevity (or late fecundity) in *Drosophila melanogaster* (see Rose 1991). In this paper, another trade-off in fitness in *D. melanogaster* is established: the negative genetic correlation between fast development and high fecundity.

In the absence of trade-offs, it is expected that a fitness-related trait would be maintained at its optimum value and would exhibit very little genetic variation (because selection would quickly fix all favorable variants). Given a trade-off between two such traits, the mean of both is affected, because the joint optimum is no longer at the optimum of either trait considered alone. It is less clear what effect such a trade-off would have on genetic variation. Rose (1982, 1985) discussed the role of antagonistic pleiotropy in promoting stable polymorphism through heterozygote advantage. However, such polymorphisms occur only if stringent dominance criteria are satisfied and, in general, heterozygotes must exhibit beneficial directional dominance for both traits (see Curtsinger et al., 1994). Thus, a necessary consequence of such polymorphism is the presence of significant dominance genetic variance.

Many fitness traits may appear to be subject to strong directional selection, in that there are no a priori reasons why, in the absence of a trade-off, they should have an intermediate optimum; for example, higher fecundity is always beneficial. This suggests an approach to the investigation of trade-offs: first, pick a trait for which there are clear reasons to expect strong directional selection; second, demonstrate through selection that genetic variation exists to drive the trait mean further in the direction favored by natural selection; and third, investigate the correlated response in traits that a priori seem likely to exhibit antagonistic pleiotropy.

One trait that is appropriate for this kind of study is larval developmental time in *D. melanogaster*. In their natural environment, the larvae of *D. melanogaster* develop on rotting fruit. This is a transient resource and larvae developing quickly will pupate before the resource is exhausted. In addition, as more and more eggs are laid on a piece of fruit, the level of competition increases (Nunney 1990). Both of these factors favor individuals able to develop quickly; hence, developmental time in *Drosophila* has generally been considered as a trait subject to directional selection for faster development (see, for example, Tantawy and El-Helw 1970). Because directional selection would reduce genetic variation, this view has been reinforced by the observation that selection for a shorter larval period has had very limited success (Sang and Clayton 1957; Sokal and Hunter 1958; Clarke et al. 1961).

To investigate whether or not there was significant variation for a shorter larval period, I initiated an experiment to select for faster larval development. No attempt was made to select for slower development. This decision was based on two related points: first, direct natural selection on this trait favors faster development such that the genetic variation favoring slower development is of limited interest in the pres-

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ent study; and second, most deleterious mutants slow larval development, and hence lines selected for slow development will tend to accumulate pathological traits that have little bearing on the evolutionary potential of the species. Mukai and Yamazaki (1971) estimated an average delay of 2.5 h in developmental time (to eclosion) per homozygous mutant accumulating in an initially isogenic stock of *D. melanogaster*. The presence of this type of low fitness allele can generate aberrant correlations among fitness traits. For example, Hiraizumi (1961), using the same species, found that developmental time (to eclosion) was positively correlated with subsequent female fecundity when the eclosion time was short, but negatively correlated when it was long. He argued that the negative correlation was due to low fitness genotypes exhibiting both slow development and low fecundity.

The positive correlation observed by Hiraizumi (1961) among faster developing genotypes suggests a trade-off between the higher fitness of a shorter developmental time and of larger size. Size shows a positive genetic relationship to female fecundity in *Drosophila* (Robertson 1957); indeed, larger size has been linked to higher fitness in males as well, through increased mating success (reviewed in Partridge and Fowler 1993). However, the existence of this trade-off between developmental time and size is not established. Partridge and Fowler (1993) found that flies selected for smaller body size showed no decrease in developmental time.

Here, measurements on individuals from two experimental lines selected for faster larval development and from their controls were used to establish that significant genetic variation existed for faster larval development; to resolve the conflicting patterns of phenotypic and genetic correlation between developmental rate and adult weight; and to confirm that the genetic link between body size and lifetime fecundity is realized in the selected lines. Given the importance of dominance in the determining the potential for antagonistic pleiotropy to maintain polymorphism, the patterns of dominance and X-chromosome involvement in the two traits were examined through F_1 hybrids. In addition, the pattern of sexual dimorphism of these traits in selected and control populations was used to provide insight into the relationship between intrasex and intersex variation.

Materials and Methods

Selection Procedure

The base population was established from several hundred females caught in the University of California, Riverside campus orange grove over a period of 2 mo. This population was maintained as a large serial-transfer bottle population (> 1000 adults) for 18 months prior to the beginning of the selection experiment. This time period was intended to enable the population to become adapted to the laboratory environment and prevent spurious genetic correlations due to selection for adaptation during the experiment (Service and Rose 1985). Two replicates were initiated (A and B), each from 60 randomly chosen inseminated females. The founding females of each replicate laid eggs in two sets of bottles, one set becoming the selection treatments (labeled "exp" for experimental) and the other becoming the control lines ("con"). Thus, there were two lines (expA and expB) selected for faster larval development each paired with its control line (conA and conB). This pairing was included in the design to control for both the effects of initial genetic variation and for the inevitable variations implicit in the selection procedure (see below); each "con" treatment was always handled in the same way as its "exp" partner.

Each new generation of the four lines was initiated in the same way. Egg laying was synchronized by allowing the approximately 120 flies (of both sexes) to lay eggs for 1–2 h in each of four to five bottles. There was some variation in laying time and the number of bottles depending how many eggs could be seen in the bottles. Despite attempts to standardize conditions, the number of eggs laid per bottle varied widely and bottles containing very few eggs (fewer than about 50) were not used. The first bottle was always discarded to avoid the problem of eggs retained for some period in the body of the females (Bakker 1959), because these eggs would lead to a spurious environmentally induced shortening of developmental time. Excess yeast was provided to larvae in the remaining bottles and they were maintained under constant light at 25°C. A regime of constant light was employed to minimize the synchronization of pupation with any diurnal cues.

After about 90 h, the experimental bottles were examined at increasingly frequent intervals until the first larvae began to pupate. The bottles were then examined every hour until the first 30 to 40 pupating individuals in each bottle had been collected. The pupae for a given line were placed together in a fresh bottle at 25°C. The target was to collect 120 pupae; hence, the number of pupae collected per bottle varied with the total number of bottles. To keep the intensity of selection high, pupae were collected in rough proportion to the number of larvae in the bottle. No attempt was made to estimate the intensity of selection, because it was believed that variation in the synchrony of the eggs and in the larval density (which averaged 400 to 500) would make any estimates approximate at best.

The controls were treated in a manner identical to the experimentals (in particular, larval densities were low and excess yeast was provided throughout larval development), except that pupae were randomly chosen on the fifth day, by which time all of the larvae had pupated. For each "con" line, the number of pupae selected from each bottle and the number of bottles used was always equal to the numbers used for the "exp" line of the same replicate.

After eclosion, the adults were held in bottles for 1–3 wk before the cycle was restarted. The total generation time averaged 22 d over the first 15 generations.

Developmental Time Experiment

Fifty virgin females were collected from each of the four lines at generation 15 of the selection experiment. Half were crossed to males of their own type and half were crossed to males from the other line of the same replicate set, creating four cross types from each replicate: the two parental types, "con" and "exp," plus two hybrid types, "cxe" (conA females × expA males and conB females × expB males) and the reciprocal "exc" (expA [or expB] females with conA [or conB] males). Thus, in the hybrid crosses the first letter de-

fines the female parent (*e*xp or *c*on). The hybrid crosses were included to investigate maternal, X-chromosome, and dominance effects.

Eggs were collected on agar plates over a period of 1.5 h, and 90 eggs from each cross were placed in 3 vials (30/vial). The vials were provided with excess yeast, which together with the low density, ensured maximal survivorship. It also closely approximated the larval rearing conditions prevailing in the populations. To minimize external cues, the vials were kept under constant light at 24°C. Larvae were collected (and timed) as they pupated and were kept individually in small glass tubes. The time to eclosion was measured. Adults were sexed, dried, and weighed. Thus, for each individual, its larval developmental time, pupal period, dry weight, and sex were recorded; however, the data for pupal period will be discussed elsewhere. For the analysis, dry weight was log-transformed to test proportion-based hypotheses; normality and homoscedasticity were both improved by the transformation.

Statistical Analysis

The analysis of the experiment was based on analysis of variance (ANOVA) and analysis of covariance (ANCOVA) using SAS 6.07. The fixed-effect treatments were cross type (''exp,'' ''con,'' ''exc,'' or ''cxe'') and sex (male or female). Random effects were replicate (''rep'' A or B) and the rearing vials, with ''vial'' nested within type and replicate. It is usual in selection experiments to consider the effect of line as a nested random effect within type, on the assumption that each experimental population reflects a random sample of the gene pool of the species being considered. This traditional analysis is presented; however, the present experiment was designed to violate that assumption. The selected/control pair making up each replicate set was initiated with exactly the same parents and thus represents a single sample of the gene pool. The pair was also treated in a similar manner when each new generation was chosen (described above). This a priori pairing allowed an examination of dominance in the F_1 cross between the selected and control populations within a replicate set. It also allowed an investigation of the differences between the replicates. When the analysis revealed significant interactions involving replicate (or, in some cases, sex), these effects were investigated in detail by subdividing the analysis. This increased the number of tests performed on the data, and the appropriate Bonferroni correction was applied to all significance levels in the subdivided analyses (e.g., see Sokal and Rohlf 1994).

The preliminary testing of the data included all effects and their interactions, but for the final analysis an interaction was set to zero following the rules outlined in Sokal and Rohlf (1994). There is some debate in the statistical literature concerning the pooling of interaction effects (because preliminary testing affects the sampling distribution of the statistics). Some (e.g., Kirk 1982) advocate pooling if the level of significance of an interaction (*P*) is greater than 0.25. The procedure used was more conservative; in particular, interactions that were not a priori expected to be zero were pooled only if $P > 0.75$. Interactions treated this way were CROSS (or SELECTION) × SEX, CROSS × REP, SEX × REP, and CROSS × SEX × REP. Based on both the design and experience, no interactions with VIAL were anticipated and these were evaluated at the $P > 0.25$ level. In each ANCOVA, the analysis was subdivided if any interactions with the covariate were significant ($P < 0.05$ using sequential sums of squares), or else these interactions were omitted.

Where appropriate, the results were further investigated using both a priori and a posteriori contrasts. A posteriori comparisons of means were made using Tukey's studentized range. Three a priori orthogonal contrasts were used. The first (''selection'') examined the direct effects of selection by comparing ''con'' and ''exp'' results. The second (''direction'') determines whether the direction of the hybrid cross (''cxe'' versus ''exc''), had an influence on the traits being measured. The interpretation of this comparison was sex dependent. A comparison of females provided a one-tailed test of maternal effects because females from ''exc'' matings carried the maternal influence of the selected line. In the absence of maternal effects, a comparison of the males provided a one-tailed test of the influence of the X chromosome since males from ''exc'' matings carried an X chromosome from the selected line. The third contrast (''dominance'') used data from all four cross types. In the absence of net dominance, the mean of the two hybrid crosses was expected to be the same as the mean of the two parental lines (the midpoint). In male offspring, this comparison was a direct test of autosomal dominance; in females, it also included the X chromosome.

Analysis of Genetic Correlation

Two of the genetic correlations between larval developmental time and adult weight were investigated: the correlation of additive effects and the correlation of dominance effects. These genetic correlations can be calculated from the correlated response to selection by comparing a selected line to its paired control. Specifically, the additive correlation can be calculated using the variances and covariance of the two traits measured as deviations from the joint mean of the control and selected populations; similarly, the dominance correlation can be calculated using the deviation of the F_1 hybrids from the same joint mean.

This approach requires that two assumptions are satisfied. First, that the response to selection was due primarily to alleles that were rare in the initial gene pool (and hence rare in the controls) increasing in frequency to near fixation. Under these conditions, the additive and dominance correlations depend primarily on additive and dominance genetic effects (see Appendix). The assumption appears to be reasonably well satisfied for trait of larval developmental time, because earlier workers (Sang and Clayton 1957; Sokal and Hunter 1958; Clarke et al. 1961) noted that it was difficult to select for a shorter developmental time, as would be expected if the favored alleles were initially at low frequency; and the additional response to selection after generation 15 was negligible, which would support the view that favored alleles were close to fixation. This last point was confirmed by measuring the larval developmental time again after a further 23 generations of selection.

The second assumption is that environmental effects could be estimated and factored out. This was achieved by basing

the statistical analysis on the trait means per vial. The variance/covariance measures taken over all vials includes both genetic and environmental effects. However, the same measures, taken within replicate and within cross type, eliminates the major genetic influences. It was assumed that the genetic differences among vials of the same line and cross type were negligible, because each vial contained 30 individuals randomly chosen from a source expected to exhibit little genetic variance for the two traits.

The first step in the statistical analysis tested the null hypothesis that additive and dominance correlations between the two traits were the same. Because this null hypothesis was upheld, then the second step was to derive a single estimate of the combined genetic correlation.

In the first step, the additive and dominance genetic correlations were compared in two ways: statistically using ANCOVA and qualitatively using linear regression. For the ANCOVA, the null hypothesis tested was that the overall correlation (genetic plus among-vial effects) estimated from "pure" types ("con" and "exp") was the same as that estimated from "hybrid" types ("cxe" and "exc"), because these groups reflected additive and dominance effects, respectively. The analysis examined the vial means of adult weight, calculated separately for each sex and classified into the two groups, with mean larval developmental time as the covariate. The null hypothesis was tested by the interaction between group and the covariate.

For the regression analysis, the "pure" and "hybrid" vial means were analyzed separately. The approach was identical for both groups, except that the regression of the "hybrid" lines was constrained to pass through the midpoint of the "pure" group data. For both groups, two regressions were run: an "overall" regression, partitioning out the effect of sex (by coding it as a 1, −1 variable); and an "environmental" regression, partitioning out the genetic effects of replicate and cross type (again using 1, −1 coding). The differences between the resulting variances and covariance of the two regressions estimated the genetic effects that were eliminated from the "environmental" regression. The estimates were used to calculate the genetic regression and correlation coefficients. These coefficients could be compared only qualitatively between "pure" and "hybrid" groups because they lacked the degrees of freedom required to test for a difference between the additive and dominance coefficients.

Because the initial ANCOVA showed that the "pure" and "hybrid" groups were homogeneous, they were analyzed jointly. The genetic correlation was estimated in a way analogous to the qualitative regression analysis described above; however, because the constraint on the hybrid regression was now removed, the more powerful ANCOVA approach could be used. (ANCOVA did not require the class variables to be coded as numbers, and hence interactions could be included). The "overall" phenotypic correlation of the two traits was estimated using the mean larval developmental time per vial as the covariate of mean adult weight. The resulting correlation, being based on all vial means, included genetic differences among lines (selected versus control versus hybrid; replicate A versus B) plus environmental differences among vials. (Note that the "overall" phenotypic effects pooled environmental factors acting within vials). The between-vial "environmental" correlation was estimated by repeating the ANCOVA with variance due to genetic sources removed by including "cross type" and "replicate" in the model. The "overall" and "environmental" estimates of the trait variances and their covariance were used to calculate the genetic correlation and regression slope that showed how the traits were linked through selection.

Fecundity and Longevity

Lifetime fecundity and longevity of female flies from each of the "pure" lines was estimated in the following way. For each of the four lines, 22 sets of four females were maintained at 25°C, and these were transferred to a new vial twice a week (every Monday and Friday) until all the females died. Each set of four females also contained four males from the base population of *D. melanogaster,* and these males were replaced as they died. Survivorship was recorded weekly. A subset of six of the sets were used to estimate lifetime fecundity by scoring eclosion from the laying vials. The laying vials were not supplemented with yeast, but after the adults were transferred to a new vial, yeast was added to the old vial to maximize larval survival. Over the next 16 d, the offspring production was recorded, by which time eclosion of the offspring was complete; this was before any of the next generation eclosed such that there was no danger of overlapping generations in the vial.

The analysis of fecundity was based on the (log transformed) lifetime productivity of each replicate of four females using ANOVA. The analysis of survivorship was based on the proportional death rate calculated for each weekly period. This proportion, arcsine/square-root transformed, was used as the dependent variable in a weighted ANCOVA, with time as the covariate, cross type ("exp" and "con") as a fixed effect, replicate (A and B) as a random effect, and the sample size at each time interval as the weight. This method of analysis, in common with almost all methods of analyzing survival curves, makes the assumption that each time interval is statistically independent, which requires that the surviving individuals remain a random sample of the total population (see Tatar et al. 1993). If this assumption is violated (e.g., because particular phenotypes survive longer), then there is a danger of finding spurious statistical significance. It will be seen that this possibility does not adversely affect the interpretation of the results.

RESULTS

Larval Developmental Time

Larval developmental time (defined as the time from egg laying to pupation) was the trait subject to direct selection. Two questions were addressed in the analysis of this trait: first, was there a significant response to selection; and second, if so, what was the genetic basis of the response? To evaluate the response to selection data from only the "pure" populations ("con" and "exp") were used; however, the investigation into the genetic basis of the response required the use of data from "pure" and "hybrid" populations.

The Response to Selection.—There was evidence from the initial analysis of the "pure" populations that the two lines

TABLE 1. Analysis of variance of the response to selection on the selected trait, larval developmental time (h), and on adult weight (log(μg)) in the two replicate sets, A and B. The "line-nested" analysis ignored the pairing of the lines into replicates; hence, line was nested within selection ("con" and "exp").

Source	df	REP A†		REP B†		Line nested‡	
		MS	*F*	*MS*	*F*	*MS*	*F*
I. The selected trait: Larval developmental time							
SELECTION	1	3941.9	55.49**	1601.6	36.49**	5244.2	41.65*
SEX	1	0.9	0.08	85.9	7.15*	53.1	2.88
SELECTION×SEX	1	15.2	1.26	3.7	0.30	16.8	0.91
LINE	2	—	—	—	—	125.9	1.96
LINE×SEX	2	—	—	—	—	18.4	1.53
VIAL	4	69.7	5.78***	43.3	3.60*	56.5	4.69***
Error	—	12.1		12.0		12.0	
(Error df)		(133)		(123)		(256)	
Variance explained (%)		72.8		55.7		66.8	
II. The correlated response: Adult weight							
SELECTION	1	0.7387	10.99*	1.1156	64.64**	1.8407	54.76*
SEX	1	1.7293	262.58***	1.5604	189.03***	3.2857	1838.0***
SELECTION×SEX	1	0.0015	0.23	0.0125	1.52	0.0115	6.44
LINE	2	—	—	—	—	0.0336	0.91
LINE×SEX	2	—	—	—	—	0.0018	0.24
VIAL	4	0.0671	10.18***	0.0171	2.07	0.0421	5.69***
Error	—	0.0066		0.0083		0.0074	
(Error df)		(133)		(123)		(256)	
Variance explained (%)		78.9		74.4		76.8	

Note: all tables use the significance convention: * 5%; ** 1%; *** 0.1%.

† SELECTION was tested against VIAL mean square; all other tests used Error mean square. The Bonferroni correction for two tests was used to evaluate significance.

‡ For the nested analysis, VIAL had 8 df. SELECTION was tested using LINE mean square; SEX and SELECTION×SEX were tested using LINE×SEX mean square; and LINE was tested using the composite mean square of LINE×SEX+VIAL−ERROR, modified slightly because of unequal sample size, with 6.8 df.

responded slightly differently to selection (the interactions selection × line was close to significance at $P = 0.07$). Thus, to address the basic question of whether or not the selected lines showed a significant response, the lines were analyzed separately (Table 1). The response to selection (see Fig. 1) was highly significant ($P < 0.01$) in both lines, equaling 10.7h (9.4% decrease in development time) in line A and 7.0 h (6.3% decrease) in line B. Both sexes responded to selection to the same degree (no sex × selection interaction), but there was an overall tendency for female larvae to develop slightly faster than males (Fig. 1). In line B, the sex difference of 1.6 h was significant. When the pairing of the line A and line B populations was ignored, and the data were analyzed with line as a nested effect, the effect of selection was again significant (Table 1).

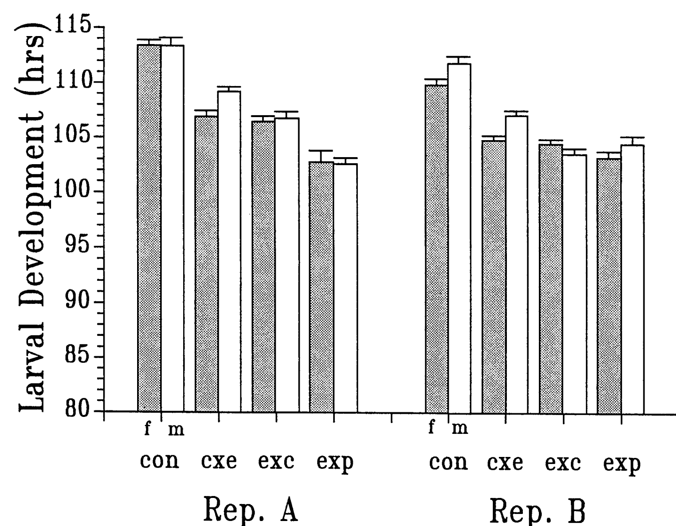

FIG. 1. Larval developmental time in control (con), selected (exp), and hybrid lines (cxe and exc) in replicates A and B. The results for females (f) are shaded and those for males (m) are unshaded. The error bars show 1 SE.

The response to selection was also evaluated in the investigation of the genetic basis of the response. The initial analysis of the complete data set showed a significant interaction between cross type and sex ($P < 0.05$) and a potentially nonzero interaction between cross type and line ($P < 0.1$). This reinforced the view that the two lines responded slightly differently to selection. In addition, it was possible that dominance and X-chromosome effects could generate interactions with sex. For these reasons, the analysis was subdivided by line and sex (Table 2). In all four of the a priori sex/line comparisons of "con" and "exp," the effect of selection was significant at $P < 0.01$ (Table 2).

The data analyzed apply to generation 15 of the selection experiment. The effect of selection was not tracked each generation; however, two features of the response not apparent from the generation 15 tests are worthy of comment. First, the response over time of the selected populations can be seen by examining the selection threshold used in each generation (Fig. 2). The selection threshold for the selected lines was defined as the larval developmental time of the slowest individuals included in the next generation and was determined by the need to collect approximately 120 larvae. The

TABLE 2. Analysis of variance of larval developmental time for each sex in each replicate. A priori contrasts were used to document the significance of selection, direction of the cross (maternal and X-chromosome effects) and dominance. Note: significance levels include the Bonferroni correction for four tests.

		REP A				REP B			
		Female		Male		Female		Male	
Source	df	*MS*	*F*	*MS*	*F*	*MS*	*F*	*MS*	*F*
CROSS†	3	573.8	25.16***	796.0	21.47**	295.1	11.12*	426.7	24.81***
VIAL	8	23.1	2.01	38.6	4.42**	26.8	3.41**	17.5	1.68
Error	—	11.5		8.7		7.9		10.4	
(Error df)		(134)		(135)		(127)		(137)	
Variance explained (%)		57.9		69.0		51.0		51.8	
A priori contrasts (1 df):									
SELECTION									
con vs. exp		1579.4	68.26***	2288.6	59.26**	718.2	26.75**	880.7	50.38***
DIRECTION									
cxe vs. exc		6.0	0.26	97.1	2.51	4.1	0.15	202.0	11.56*
DOMINANCE									
con/exp vs. cxe/exc		72.9	3.15	0.2	0.01	151.9	5.66	247.2	14.14*

† CROSS (con, cxe, exc and exp) was tested against the VIAL mean square.

equivalent parameter was estimated for the control lines by determining the threshold that would have been needed to collect the same number of larvae. By the end of generation 14, the difference between selected and experimental populations appeared to be stabilizing, and this was confirmed by measurement of the mean larval developmental time in generation 38 (Fig. 2), which showed no significant differences from those measured in generation 15. Note that the selection thresholds are much faster than the mean values because they are defined by the fastest developing larvae in the population.

The Genetic Basis of the Response.—The subdivided analysis of the complete data set, including the hybrid lines, provided information on the possible genetic basis of the response to selection through examination of the two orthogonal comparisons, "direction" and "dominance" (Table 2). The female "direction" comparison was not significant; hence, there was no evidence of maternal effects. Given the absence of maternal effects, the "direction" comparison in males tested for X-chromosome effects. The results provided some weak support for X-chromosome involvement: there was a nonsignificant difference of 2.5 h in line A and a similar significant difference of 2.6 h in line B ($P < 0.05$).

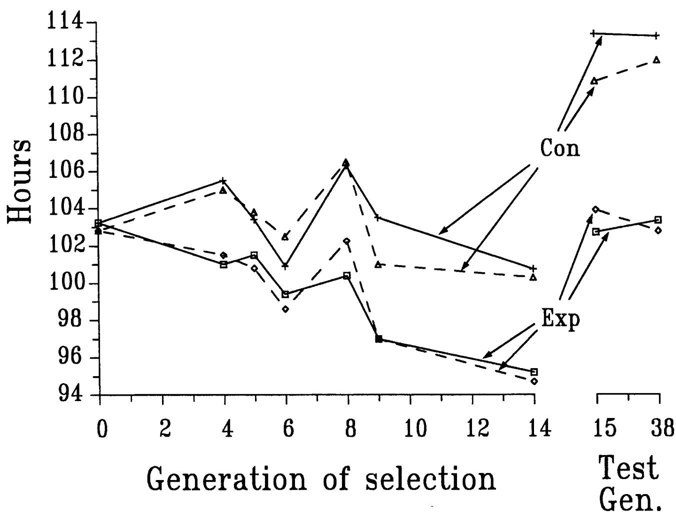

FIG. 2. The response to selection for shorter larval developmental time. The response up to the end of generation 14 is shown in terms of the average selection threshold used to collect pupating larvae for the next generation. For the control lines, this threshold is the value that would have been needed to collect the required number of individuals for the next generation, if selection had been applied. The mean developmental time of larvae in each line was tested in the experiment described in this paper at generation 15 and again at generation 38. Replicates A and B are shown, respectively, by solid and dashed lines.

Examination of the "dominance" contrast (Table 2), shows that the males of line B were again the only group to show a significant effect ($P < 0.05$), and it was equal to 1.9 h in the direction of faster development. In contrast, the hybrid males of line A were precisely intermediate between the parental types. Both types of female hybrid were biased toward faster development, by an average of 1.7 h. Thus, overall, there was weak evidence of directional dominance favoring fast development.

Adult Dry Weight

Correlated Response to Selection.—The first hypothesis to be tested was whether selection for faster larval development indirectly selected for smaller flies. A second hypothesis concerned whether the two sexes were equally affected. For ease of comparison, the analysis of the indirect response to selection was structured identically to the analysis of the direct response (Table 1). The ANOVA of log(weight) showed a significant reduction in weight due to selection for fast larval development in both lines (see Fig. 3). The reduction was the same in both sexes (no selection × sex interaction), although it was slightly higher in females (17%) than in males (13%). Pooling across sexes, the weight loss equaled 14% in line A ($P < 0.05$) and 17% in line B ($P < 0.01$). The nested analysis also showed the weight change to be significant ($P < 0.05$). Females were on average 26% heavier than males

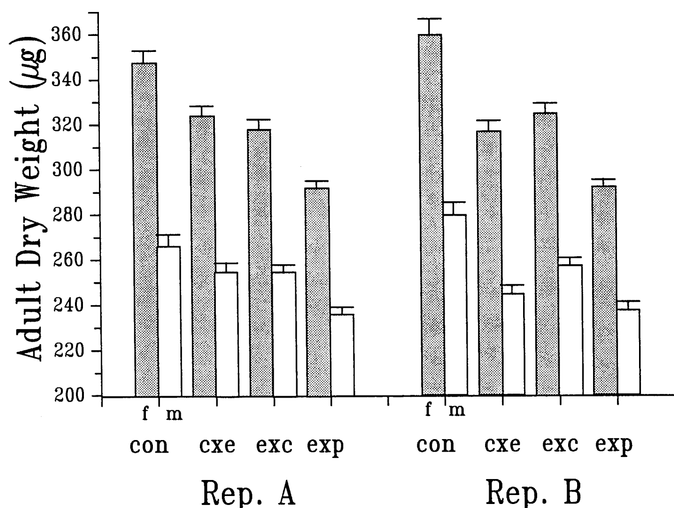

FIG. 3. Adult dry weight in control (con), selected (exp), and hybrid lines (cxe and exc) in replicates A and B. The results for females (f) are shaded and those for males (m) are unshaded. The error bars show 1 SE. These values were calculated from the untransformed data.

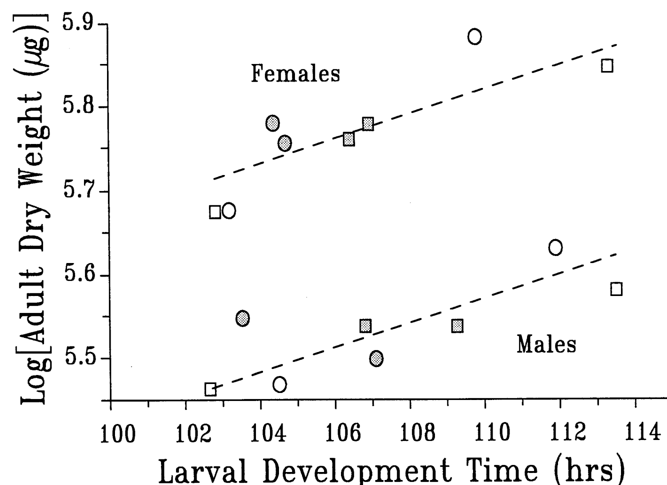

FIG. 4. The relationship of adult dry weight to larval developmental time. The mean values (across vials) for each cross type, split by sex, for both replicate A (squares) and B (circles) are plotted. Data points from the hybrids (cxe and exc) are stippled and those for the pure lines (con and exp) are open. The slope of the two dashed regression lines is the estimated genetic relationship between the two traits, equaling 0.0147 log(μg)/h.

($P < 0.001$); 30% in control populations verses 23% in the selected populations.

To examine the genetic basis of the response, the analysis was subdivided by line and sex, as it had been for the analogous analysis of larval development. Although the effect of selection remained significant in all four groups ($P < 0.05$), there were no indications of maternal, X-chromosome, or dominance effects (Table 3).

Regression on Larval Development Time.—The data can be used to investigate the genetic relationship between adult dry weight and larval developmental time through their joint response to selection. For this investigation, the analysis was based on vial means.

The initial step in the genetic analysis was to test the null hypothesis that the additive and dominance correlations were the same. The statistical test of this null hypothesis employed an ANCOVA in which the effects of sex (but not of line) were partitioned out. This "overall" analysis showed that the relationship between adult weight and developmental time was not significantly different ($P > 0.1$) between "pure" ("con" and "exp" populations) and the "hybrid" ("cxe" and "exc" populations). Thus, the data from both groups fall along a single regression (Fig. 4); however, the hybrid data, considered alone, would fall along a much flatter line. This difference, although not statistically significant, could be used to argue against pooling the "pure" and "hybrid" data. One possible reason for this apparent difference is that environmental effects were not excluded from the analysis, and the flat distribution of the hybrid lines could reflect primarily environmental effects, given the relatively small genetic differences among the hybrid lines. This possibility was investigated in more detail using a regression analysis that factored out the environmental effects. As noted in the Materials and

TABLE 3. Analysis of variance of the correlated response of adult dry weight for each sex in the two replicates. For further explanation see Table 2.

		REP A				REP B			
		Female		Male		Female		Male	
Source	df	*MS*	*F*	*MS*	*F*	*MS*	*F*	*MS*	*F*
CROSS	3	0.1181	4.73	0.1087	4.57	0.2263	9.60*	0.1684	5.53
VIAL	8	0.0256	5.16***	0.0247	3.44**	0.0239	3.32**	0.0314	4.22***
Error	—	0.0050		0.0072		0.0072		0.0074	
(Error df)		(134)		(135)		(127)		(137)	
Variance explained (%)		51.2		32.5		49.2		42.9	
A priori contrasts (1 df):									
SELECTION									
con vs. exp		0.3434	13.34*	0.3153	12.76*	0.6559	27.47**	0.4266	13.58*
DIRECTION									
cxe vs. exc		0.0088	0.34	0.0001	0.01	0.0197	0.83	0.0211	0.67
DOMINANCE									
con/exp vs. cxe/exc		0.0080	0.31	0.0088	0.36	0.0020	0.09	0.0514	1.64

TABLE 4. Environmental and genetic covariation between adult dry weight and larval developmental time. Based on ANCOVA analyses of mean adult dry weight (per vial) with mean larval developmental time (MLARVA) as the covariate. In the "overall" analysis, the covariate accounted for both environmental differences between vials and genetic differences between lines. In the "environmental" analysis, the covariate accounted only for environmental differences between vials. The percent variance explained relates to the complete model; the correlation coefficient relates only to the covariate. Regression units: log(μg)/h. Note: SEX × CROSS/REP interaction was eliminated from the model after preliminary analysis ($P > 0.75$).

		Source of variation					
		Overall		Environmental		Genetic	
Source	df	*MS*	*F*	*MS*	*F*	*MS*	*F*
MLARVA	1	0.1155	39.18***	0.0004	0.21	0.1272	16.39**
SEX	1	0.7275	246.77***	0.5752	295.27***	—	
CROSS/REP†	7	—		0.0084	4.30**	–	
Error	—	0.0029		0.0019		0.0078	
(error df)		(45)		(38)		(6)	
Variance explained (%)		85.4		90.7		73.2	
Regression slope ± SE		0.0130 ± 0.0021		0.0021 ± 0.0047		0.0147 ± 0.0036	
Correlation coefficient		0.682***		0.074		0.856**	
Sex difference ± SE		0.2489 ± 0.0158		0.2367 ± 0.0138		—	

† CROSS and REP were combined into a single random effect that partitioned out all genetic differences between vials; this simplification does not affect the calculation of the genetic parameters.

Methods, this analysis did not have the statistical power to compare the "pure" (i.e., additive) and "hybrid" (i.e., dominance) results; however, the environmental correlations were estimated at 0.162 and −0.087, respectively, whereas the genetic correlations were estimated at 0.974 and 0.684, respectively. Thus, these results fully supported the view that the additive and genetic correlations were similar and that the flat distribution of the "hybrid" values in Figure 4 was due to the low environmental correlation between the two traits.

Because the null hypothesis that additive and dominance effects were the same could not be rejected, the two groups ("pure" and "hybrid") were considered together. The "overall" ANCOVA (that partitioned out only the effects of sex) was repeated, again using the mean developmental time (per vial) as a covariate regressed against mean weight. The sex difference in weight was highly significant, but the regression of weight on developmental time did not differ between the sexes, with a joint slope of 0.013 log(μg)/h (Table 4). It is worth noting that when the analysis was repeated, but with the genetic effects due to replicate removed (by including it as an effect in the model), the replicate effect was nonsignificant ($P = 0.10$), and the slope was virtually unchanged at 0.014 log(μg)/h.

The "environmental" ANCOVA estimated the environmental effects acting between vials because it partitioned out the effects of sex plus the effects of replicate, cross-type, and their interaction. This analysis showed that, within lines, the vial means for log weight and mean larval developmental time were uncorrelated ($r = 0.074$) and were linked by a nonsignificant slope of 0.002 log(μg)/h (Table 4). These estimates of "environmental" and "overall" variation among vials permitted the genetic effects to be estimated (Table 4), giving a genetic correlation between the two traits of 0.856 ($P < 0.01$) and a slope of 0.0147 log(μg)/h (see Fig. 4).

TABLE 5. Lifetime female fecundity in control and selected lines.

I. Analysis of variance based on log-transformed productivity data. Note: SELECTION×REP interaction was eliminated from the model after preliminary analysis ($P > 0.75$).

Source	df	*MS*	*F*
SELECTION	1	1.106	55.70***
REP	1	0.046	2.32
Error	21	0.020	
Variance (%)		73.4	

II. Mean values. The mean ± SE of the log-transformed productivity data (four females per replicate) is given in parentheses.

Replicate	Control	Selected
A	296.2 (7.08 ± 0.07)	200.8 (6.69 ± 0.05)
B	282.6 (7.03 ± 0.04)	176.6 (6.56 ± 0.07)

Female Fecundity and Longevity

The mean lifetime fecundity was significantly lower in the selected lines (Table 5). The reduction was substantial, with the selected lines having on average only 65% of the lifetime fecundity of the control lines. There was no significant difference between the replicates, although the individuals in line A had a higher mean fecundity than those in line B. This difference was probably due to the slightly higher death rate observed in line B, although this difference was not significant ($P = 0.14$). There was no indication that the control and selected lines differed in longevity, such that the difference in their lifetime fecundity was not due to longevity differences. The analysis also showed that the survivorship curves were fitted closely by assuming a linearly increasing death rate (Table 6; Fig. 5); indeed the statistical model explained 84% of the variance in the data (Table 6).

DISCUSSION

Selection for fast larval development revealed sufficient genetic variation to reduce developmental time by an average of 7.9% in 15 generations. The response was the same in both sexes (7.7% in females and 8.1% in males). Given the ecology of *D. melanogaster,* with the larvae developing in

TABLE 6. Analysis of covariance of survivorship data from control and selected lines, using the arcsine/square-root transformation of the weekly death rate with TIME as a covariate.

Source	df	*MS*	*F*
TIME	1	145.345	153.88***
SELECTION†	1	0.704	2.87
REP†	1	4.251	17.02
SELECTION×REP	1	0.245	0.26
Error	30	0.946	
Variance (%)		83.8	
Regression slope ± SE		0.0194 ± 0.0016	

† SELECTION and REP were tested against the SELECTION×REP mean square. The denominator mean squares and degrees of freedom were corrected slightly for unequal sample size.

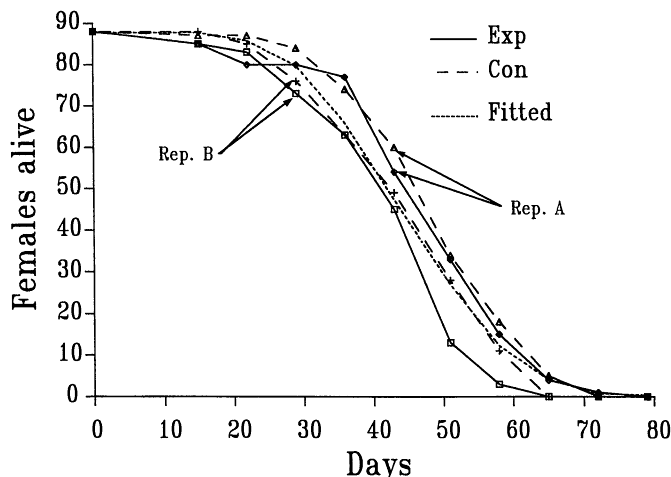

FIG. 5. Survivorship curve of control (con) and selected (exp) adult females of replicates A and B. The best fit curve to the data (dotted line) assumes a linear increase with time in the arcsine/square-root transformed death rate (see Table 6).

the transient resource of rotting fruit, rapid development would be expected to be highly advantageous. Thus, selection would be expected to minimize developmental time. Indeed Burnet et al. (1977) found that, although larvae of *D. melanogaster* could be successfully selected for faster feeding rate, the speed of development was not improved. However, it appears that this is not the whole story. Selection would not minimize developmental time if some other fitness-related trait was adversely affected and, in this study, it has been shown that there is a strong antagonistic pleiotropy between developmental time and another important fitness trait, size.

Trade-off with Adult Weight

Selection for fast larval development resulted in a correlated response that reduced weight by 17.2% in females and by 13.0% males. In this process, females changed from being an average of 29.7% larger than males to 23.4% larger; however, the sex difference in response was not statistically significant.

Size is an important determinant of fecundity in females and mating success in males (see Partridge and Fowler 1993). Here, the relationship of size with fecundity was confirmed; the 17% reduction in size of females translated into a 35% reduction in lifetime fecundity. However, this reduction was not due to a reduced longevity of the selected flies; there was no significant difference in the survivorship of the control and selected populations. Partridge and Fowler (1992) found that flies selected for late reproduction lived longer and had a longer larval period. This raised the possibility that the extended larval period was linked to the extended adult longevity and would lead to the expectation that the flies selected for rapid larval development would have reduced longevity; however, this was not the case. Thus, in different kinds of selection experiment, longer larval development may lead to greater longevity, no change or, in the case of the flies selected for larger size by Hillesheim and Stearns (1991, 1992), decreased longevity. There is apparently no direct relationship between larval developmental time and adult longevity, although Zwaan et al. (1995) also found that selection for faster developmental time (in their case, egg to adult) had no effect on longevity.

The trade-off between shorter developmental time and larger size was highly significant and the estimated genetic correlation (r_g) was −0.86. Zwaan et al. (1995) found a similar trade-off in an experiment selecting for faster egg-to-adult development. In their experiments, the change in developmental time was probably due to a change in both the larval and pupal period; in fact, Tantawy and El-Helw (1970) showed that the heritability of the pupal period was about twice that of the larval period, suggesting that the bulk of the response observed by Zwaan et al. (1995) may have been in the pupal period. However, even though their selection regime included the pupal period, the correlated response of weight reduction was similar to that seen in the experiments reported here.

In contrast to the results that I obtained, Zwaan et al. (1995) found no significant reduction in fecundity due to selection for faster development; however, they showed very clearly the potential for such an effect. They showed that the phenotypic relationship between wing length (their measure of size) and lifetime fecundity is very steep, such that the large 35% decrease from 287 offspring to 189 offspring that I observed (see Table 5) would arise from a mean difference in wing length of only about 2–3%. This degree of change in a linear measurement is consistent with the 17% change that I observed in dry weight.

The same genetic trade-off between developmental rate and size also has been observed in the beetle, *Callosobruchus maculatus* (Møller et al. 1989). Such a trade-off leads to stabilizing selection for an intermediate body size and this has been modeled for both *D. melanogaster* (Roff 1981) and *C. maculatus* (Sibly et al. 1991). In the first of these two models, a short developmental time is advantageous because of its effect on intrinsic growth rate in an age-structured population; in the second, it is advantageous because of an artificially imposed selection threshold for developmental time (the culture interval). A third model has been suggested by Prout and Barker (1989, 1993). They suggested that in *Drosophila* populations, high levels of larval competition in some resource patches select for genetically small flies, be-

cause these genotypes develop quickly and pupate before the resources are exhausted.

Given that selection for faster developmental rate resulted in a correlated reduction in body size, the reverse experiment of selecting for small size would also be expected to produce a correlated response in larval development. This response should be predicted by a slope of 58.23 h/log(μg) ($=r_g^2/b$, where b is the slope defining the correlated response of weight to reduced developmental time [Table 4]; see Falconer 1981). Hillesheim and Stearns (1991) selected for increased and decreased wet weight in both rich and poor environments. In the rich environment, there was no significant correlated response in developmental time. In the poor environment, the smaller flies indeed developed faster; however, both large and small lines showed a progressive shortening of the developmental period suggesting that even in the absence of size selection, the environment was selecting for faster development. This result supports the basic premise of the Prout and Barker (1989, 1993) hypothesis (discussed above) that a poor environment favors rapid larval development. Partridge and Fowler (1993) also selected for increased and decreased body size, using thorax length as their measure. They found that the large-selected flies exhibited an extended developmental period, but they found no evidence for a change in larval developmental time of the small-selected flies. However, there are two factors that could have made their detection of such a change difficult. First, their technique was not designed to detect a response of less than 12 h and the regression slope of 58.23 h/log(μg), derived from the experiments reported here, suggests that flies would have to lose more than 20% of their body weight before the response in developmental time reached that figure. Second, environmental factors acting among the vials (such as those caused through variations in larval density) would act to obscure the relationship. Notwithstanding these complications, there is actually good evidence of a significant difference between their control and small-selected flies: out of 16 sex/cross comparisons, 14 show the small-selected flies developing faster and only 1 comparison is in the opposite direction ($P < 0.01$ using a sign test).

The Genetic Basis of the Traits

The use of hybrid crosses allowed some analysis of the genetic basis of the response to selection. There was an indication of a small X-chromosome involvement in the change in larval developmental rate, and there was some suggestion of dominance acting in the direction of faster development. However, both effects were, at best, marginally significant, and it appears that the bulk of the response to selection involved autosomal loci with primarily additive effects. This view is further supported by the absence of X-chromosome or dominance effects in the analysis of weight, because the high genetic correlation between weight and developmental time would predict similar genetic patterns across the two traits. However, the approximately twofold greater coefficient of variation in weight differences compared with developmental time differences would make the detection of small effects on weight difficult to detect. For the same reason, the apparent differences in the genetic determination of the two traits were insufficient to create a difference between the correlation of additive effects and the correlation of dominance (and male X-chromosome) effects.

Curtsinger et al. (1994) showed that, in general, a precondition for a stable polymorphism to result directly from antagonistic pleiotropy is substantial dominance variance. Specifically, in the fitness trade-off between fecundity and developmental rate, polymorphism would be favored by directional dominance favoring high fecundity (i.e., large body size) and fast development. The was no indication of directional dominance for body size. For developmental time, there were indications of directional dominance in replicate B, but not in replicate A; this dominance can be seen as a left shift of the shaded circles in Figure 4. Such low levels of dominance make it unlikely that antagonistic pleiotropy acts to maintain substantial levels of polymorphism.

Sexual Dimorphism

A consistent feature of *D. melanogaster* is that adult females are significantly larger than males (see Bakker 1959) and in the present experiment, the control females were almost 30% heavier than the males. Another consistent feature is that females generally eclose before males, creating an apparent paradox of larger size and shorter developmental time in females compared with males. In fact, the early eclosion of females is largely due to their shorter pupal period (see Bakker and Nelissen 1963; Nunney 1983); however, this does not account for all of the paradox. To account for the large female size, we would expect that female larval development would take longer than males. In fact, sex differences in larval developmental time are small, strain specific, and may be in either direction (Powsner 1935; Nunney 1983). For example, in the present experiment, there was no sex difference in replicate A but there was a small, but significant, difference in replicate B, and this was in the direction of faster female development. Thus, in comparing the sexes, it is clear that there is no direct relationship between the duration of the larval period and adult weight. The most probable explanation is that there is some intrinsic feature of male development that causes them to be smaller, that is, males are intrinsically less efficient at converting the gains from a given feeding period into adult weight. A likely cause of this loss of efficiency is some aspect of gonadal development. It is notable that larval testes are much larger than larval ovaries (Kerkis 1931). Partridge (pers. comm., 1993) has suggested that spermatogenesis may be a costly process that slows male development; it is certainly the case that some meiosis is initiated as early as the time of pupation (see Lindsley and Tokuyasu 1980). An alternative, if unlikely, possibility is that males are generally subject to less stringent selection for developmental rate than females. In the present experiment, any sex-limited genetic variation that promoted faster development would have been favored. If there was more such variation in males than females (as might be expected if males were historically subject to weaker selection), then this bias would have been revealed by a greater response of male developmental time to selection. The experiment failed to reveal any such effect.

Regardless of the cause, it is clear that the strong positive genetic correlation between larval developmental time and

weight seen within the sexes does not apply to the differences between the sexes. This raises the question of whether the sexual dimorphism for size is controlled by genes that have no pleiotropic effects on developmental time. If so, selection on developmental time would have no effect on the degree of dimorphism. As noted earlier, there was a nonsignificant trend (duplicated almost precisely in both lines) for females to lose proportionately more weight than males (females 17.2%; males 13.0%). However, the females remained 23% heavier than males, such that, even if the greater female loss was a real effect of the selection, the effect was small and the pattern of dimorphism was largely unaltered. This further supports the view that some intrinsic feature of male development is the cause of the sexual dimorphism.

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Literature Cited

Bakker, K. 1959. Feeding period, growth, and pupation in larvae of *Drosophila melanogaster.* Entomol. Exp. Appl. 2:171–186.

Bakker, K., and F. X. Nelissen. 1963. On the relations between the duration of the larval and pupal period, weight and diurnal rhythm in emergence in *Drosophila melanogaster.* Entomol. Exp. Appl. 6:37–52.

Bell, G., and V. Koufopanou. 1986. The cost of reproduction. Oxf. Surv. Evol. Biol. 3:83–131.

Burnet, B., D. Sewell, and M. Bos. 1977. Genetic analysis of larval feeding behaviour in *Drosophila melanogaster.* II. Growth relations and competition between selected lines. Genet. Res. 30:149–161.

Clarke, J. M., J. Maynard Smith, and K. C. Sondhi. 1961. Asymmetrical response to selection for rate of development in *Drosophila subobscura.* Genet. Res. 2:70–81.

Curtsinger, J. W., P. M. Service, and T. Prout. 1994. Antagonistic pleiotropy, reversal of dominance, and genetic polymorphism. Am. Nat. 144:210–228.

Falconer, D. S. 1981. Introduction to quantitative genetics. 2d ed. Longman, London.

Hillesheim, E., and S. C. Stearns. 1991. The responses of *Drosophila melanogaster* to artificial selection on body weight and its phenotypic plasticity in two larval food environments. Evolution 45:1909–1923.

———. 1992. Correlated responses in life-history traits to artificial selection for body weight in *Drosophila melanogaster.* Evolution 46:745–752.

Hiraizumi, Y. 1961. Negative correlation between rate of development and female fertility in *Drosophila melanogaster.* Genetics 46:615–624.

Kerkis, J. 1931. The growth of the gonads in *Drosophila melanogaster.* Genetics 16:212–242.

Kirk, R. E. 1982. Experimental design: Procedures for the behavioral sciences. 2d ed. Brooks/Cole, Monterey, CA.

Lindsley, D. L., and K. T. Tokuyasu. 1980. Spermatogenesis. Pp. 225–294 *in* M. Ashburner and T. R. F. Wright, eds. The genetics and biology of *Drosophila,* Vol 2. Academic Press, London.

Møller, H., R. H. Smith, and R. M. Sibly. 1989. Evolutionary demography of a bruchid beetle. I. Quantitative genetical analysis of the female life history. Funct. Ecol. 3:673–681.

Mukai T., and T. Yamazaki. 1971. The genetic structure of natural populations of *Drosophila melanogaster.* X. Developmental time and viability. Genetics 69:385–398.

Nunney, L. 1983. Sex differences in larval competition in *Drosophila melanogaster*: The testing of a competition model and its relevance to frequency-dependent selection. Am. Nat. 121: 67–93.

———. 1990. *Drosophila* on oranges: Colonization, competition, and coexistence. Ecology 71:1904–1915.

Partridge, L., and K. Fowler. 1992. Direct and correlated responses to selection on age at reproduction in *Drosophila melanogaster.* Evolution 46:76–91.

———. 1993. Responses and correlated responses to artificial selection on thorax length in *Drosophila melanogaster.* Evolution 47:213–226.

Powsner, L. 1935. The effects of temperature on the duration of the developmental stages of *Drosophila melanogaster.* Physiol. Zool. 8:474–520.

Prout, T., and J. S. F. Barker. 1989. Ecological aspects of the heritability of body size in *Drosophila buzzatii.* Genetics 123: 803–813.

———. 1993. *F*-statistics in *Drosophila buzzatii*: Selection, population size and inbreeding. Genetics 134:368–375.

Robertson, F. W. 1957. Studies in quantitative inheritance. XI. Genetic and environmental correlation between body size and egg production in *Drosophila melanogaster.* J. Genet. 55:428–443.

Roff, D. 1981. On being the right size. Am. Nat. 118:405–422.

Rose, M. R. 1982. Antagonistic pleiotropy, dominance, and genetic variation. Heredity 48:63–78.

———. 1985. Life history evolution with antagonistic pleiotropy and overlapping generations. Theor. Popul. Biol. 28:342–358.

———. 1991. Evolutionary biology of aging. Oxford University Press, Oxford.

Sang, J., and G. A. Clayton. 1957. Selection for larval development time in *Drosophila.* J. Hered. 48:265–270.

Service, P. M., and M. R. Rose. 1985. Genetic covariation among life-history components: The effect of novel environments. Evolution 39:943–944.

Sibly, R. M., R. H. Smith, and H. Møller. 1991. Evolutionary demography of a bruchid beetle: IV. Genetic trade-off, stabilizing selection, and a model of optimal body size. Funct. Ecol. 5: 594–601

Sokal, R. R., and P. E. Hunter. 1958. Environmentally caused fluctuations in quantitative characters of *Drosophila.* 10th. Int. Congr. Entomol. 2:842–854.

Sokal, R. R., and F. J. Rohlf. 1994. Biometry. 3d ed. W. H. Freeman, New York.

Stearns, S. C. 1992. The evolution of life histories. Oxford University Press, Oxford.

Tantawy, A. O., and M. R. El-Helw. 1970. Studies on natural populations of *Drosophila.* IX. Some fitness components and their heritabilities in natural and mutant populations of *Drosophila melanogaster.* Genetics 64:79–91.

Tatar, M., J. R. Carey, and J. W. Vaupel. 1993. Long-term cost of reproduction with and without accelerated senescence in *Callosobruchus maculatus*: Analysis of age-specific mortality. Evolution 47:1302–1312.

Zwaan, B. J., R. Bijlsma, and R. F. Hoekstra. 1995. Artificial selection for development time in *Drosophila melanogaster* in relation to aging: Direct and correlated responses. Evolution 49: 635–648.

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Appendix

Genetic Correlations Measured between Lines

Consider a perfectly pleiotropic polymorphic locus, with allele C at high frequency (p_c) in the control line and allele E at high frequency (q_e) in the experimental line. Let the genotypic values for CC, CE, and EE be a_1, d_1, $-a_1$ for trait 1 and a_2, d_2, and $-a_2$ for trait 2 (see Table).

Genotype	CC	CE	EE
Frequency in control line	p_c^2	$2p_cq_c$	q_c^2
Frequency in experimental line	p_e^2	$2p_eq_e$	q_e

Effect on trait 1	a_1	d_1	$-a_1$
Effect on trait 2	a_2	d_2	$-a_2$

From the table it follows that the mean effect in pop j on trait i = $a_i(p_j^2 - q_j^2) + 2p_jq_jd_i$.
The variance in trait i generated by the mean line differences is

$$\mathrm{Var}(i) = 2[p_e - p_c]^2[a_i + d_i(1 - p_e - p_c)]^2,$$

and the covariance between the traits created by these differences is

$$\mathrm{Cov}(1, 2) = 2[p_e - p_c]^2[a_1 + d_1(1 - p_e - p_c)][a_2 + d_2(1 - p_e - p_c)].$$

Assume (1) that in the control line, EE is rare: $q_c^2 \approx 0$; (2) that in the experimental line, CC is rare: $p_e^2 \approx 0$; and (3) that the difference in the frequency of the rare alleles is small: $q_c - p_e \approx 0$ (and hence, from (1) and (2), $q_cp_e \approx 0$). Then, we have:

$$\mathrm{Var}(i) = 2a_i^2(1 - 2q_c - 2p_e);$$
$$\mathrm{Cov}(1, 2) = 2a_1a_2(1 - 2q_c - 2p_e).$$

These variance and covariance terms depend only on the additive effects of the locus on the two traits, offset by the sum of the frequency of the rare alleles.

Next, we consider the deviations of the F_1 hybrid from the midpoint between the two parental lines as a method of evaluating the dominance effects at the locus. The frequency of each genotype in the F_1 is Freq. CC = p_cp_e; Freq. EC = $p_cq_e + q_cp_e$; Freq. EE = q_cq_e, from which the mean for each trait is calculated. The squared deviation of this mean for trait i from the mean of the two parental lines is

$$Vd(i) = d_i^2(1 - 3q_c - 3p_e) + 2a_id_i(q_c - p_e) + a_i^2(q_c + p_e),$$

such that if both parental lines were fixed ($q_c = p_e = 0$), then $Vd(i) = d_i^2$. Similarly, the covariation of the two trait means around the midpoint is defined by

$$\mathrm{Covd} = d_1d_2(1 - 3q_c - 3p_e) + (a_1d_2 + a_2d_1)(q_c - p_e) + a_1a_2(q_c + p_e).$$

Assuming $q_c - p_e \approx 0$ gives

$$\mathrm{Vd}(i) = [1 - 2(q_c + p_e)]d_i^2 + (q_c + p_e)(a_i^2 - d_i^2);$$
$$\mathrm{Covd} = [1 - 2(q_c + p_e)]d_1d_2 + (q_c + p_e)(a_1a_2 - d_1d_2).$$

and, because q_c and p_e are expected to be small, dominance effects determine these values. Only if dominance effects are small are these measures likely to be biased by the additive effects.