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MALE GENOTYPE AFFECTS FEMALE LONGEVITY IN *DROSOPHILA MELANOGASTER*

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Abstract.—Several recent studies suggest that interactions with conspecific males can reduce the longevity of female *Drosophila melanogaster* or support the idea that male and female fitness components are involved in antagonistic interactions. Here we report that males from third-chromosome isogenic lines demonstrated significant genetic variation in male reproductive performance and in the longevity of their mates. Increased male performance was marginally significantly associated with one measure of increased female survival rate. However, there was no indication of trade-offs or negative correlations between male reproductive success and female survival. We discuss alternative hypotheses for the cause of the induced variation in female longevity.

Key words.—*Drosophila*, life history, longevity, mortality, sexual antagonism, sexual conflict, sperm competition.

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Recent studies suggest that interactions with conspecific males can reduce the longevity of female *Drosophila*, even

if female reproductive rate is held constant (Partridge et al. 1986; Fowler and Partridge 1989; Chapman et al. 1995). At least part of this life-shortening effect of exposure to males has been attributed to mating itself, rather than to nonmating interactions among males and females (Fowler and Partridge 1989; Rice 1996). In addition, some evidence suggests that

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the mating effect can be largely accounted for by exposure to seminal fluid, rather than other mating interactions such as copulation duration or courtship activity (Chapman et al. 1995).

Another experiment (Rice 1996) lends additional support to the idea that male reproductive activity has deleterious consequences for female survival and suggests that male and female fitness components are involved in antagonistic interactions. Rice (1996) allowed the evolution of males within a population, while preventing female co-evolution. Compared to controls, males quickly achieved higher net fitness (total offspring production under competitive conditions), higher remating rates, and higher sperm-competitive ability. These increases in male fitness were accompanied by greatly increased mortality in their mates, but no significant change in female fecundity. Thus, increases in male reproductive fitness were associated with increased deleterious effects on the longevity of their mates.

This suite of studies provides a convincing case that male reproduction has deleterious effects on female longevity. To our knowledge, however, there have been no previous reports that within-population genetic variation among males can lead to variation in the longevity of their mates. Within-population genetic variation in male mating success (Hughes 1995a,b) and sperm competitive ability (Clark et al. 1995; Hughes 1997) have been reported for the randomly mating laboratory-adapted *Ives* (*IV*) population of *D. melanogaster*. Here we report that male genotypes derived from this same population demonstrated significant genetic variation in both male reproductive performance and in the longevity of their mates. These results are particularly striking because the females were exposed to males of different genotypes for only a short time (24 h).

MATERIALS AND METHODS

Flies used in this experiment were all derived from the *IV* population, an inversion-free, randomly mating population that has been maintained at large population size in a laboratory environment for many generations (Charlesworth and Charlesworth 1985). The population is adapted to the laboratory environment and is presumably in mutation-selection-drift equilibrium with respect to genetic variation for life-history traits (Rose and Charlesworth 1981a,b; Rose 1984).

A balancer chromosome placed on the *IV* genetic background (*IV*; *TM6b*) was used to produce *IV* third-chromosome isogenic lines in the usual way; see Hughes (1995a) for a detailed description. Placing the balancer on the *IV* background avoids introducing genetic variance from the balancer stock and avoids the effects of hybrid dysgenesis. We used two replicate sublines within lines. Each of the two sublines is isogenic for the same third chromosome, but have independently derived X and second chromosomes. Use of sublines allows us to check for genetic background effects and to remove these effects when attributing variation to the third chromosome (Hughes 1995a).

Thirty-three isogenic lines were assayed for male performance in sperm-competition trials. At the same time, the females mated to these males were assayed for longevity and age-specific survival. Male performance was measured by

first mating females to males from a standard marker stock, then mating the same females a few days later to males from one of the isogenic lines. Both the female and the male used as her first mate were homozygous for a visible mutation (*ebony* [*e*] in this case) that had been placed on an *IV* background. Offspring produced after the second mating could then be unambiguously assigned to one of the two possible sires. Different isogenic lines were then compared for relative performance when competing against males from the mutant stock.

Hughes (1997) provides detailed protocols for assays of second-male reproductive success. Briefly, density effects were partially controlled by two generations of constant and low parental density (five females and males per rearing vial). Parental flies were removed after day 7, and offspring were collected on day 11. We collected males and females for the experiment as virgins and held them in separate-sex vials at constant density until they were 4 days old. *Ebony* females were then mated to *ebony* males by placing one virgin female in a vial with two males. Males were removed after 2 h. We then held the female 4 days (this interval yields a reasonable remating rate and allowed us to insure that every female had produced live larvae from her first mating; see Hughes 1997). Each female was placed in a vial with two wild-type males from one of the isogenic lines, and males were removed 24 h later. After removal of males, we transferred each female to a new vial every 7 days until she died or for a maximum of 7 transfers if she lived that long (except in block 4 which was ended after the fifth transfer). We recorded all deaths or escapes, as well as the phenotype (*ebony* or wild type) and sex of all offspring. If the assay ended before the death of a particular female or if the female escaped, her survival time was considered to be right-censored. All females ceased producing offspring before the fifth transfer. Similar methodology has been used in several genetic studies of sperm competition (Prout and Bundgaard 1977; Clark et al. 1995; Hughes 1997).

We measured reproductive performance of second males using a competitive index (CI), calculated as $a/(b + 1)$, where a is the number of progeny sired by the second male and b is the number of progeny sired by the first male (Gilchrist and Partridge 1997; Hughes 1997). This estimator of relative success is approximately unbiased (Haldane 1955). Although larval density and female remating are not strictly controlled in this protocol, we showed in a previous experiment that larval viability could not account for the genetic variation in CI among male genotypes from this same population (Hughes 1997). We also showed that CI was uncorrelated with female remating probability (Hughes 1997). These results suggest that the genetic variation in CI was due to differences in male fertility and not to differences in larval competitive ability or in the number of copulations completed. Male productivity, measured by a , was used as a second measure of male reproductive performance. This measure was based only on the offspring produced in the females' second vial to avoid confounding female survival with male productivity (all females included in the analysis survived at least through the end of the second laying period).

In a given block of the experiment, we conducted two replicate assays for each subline (a total of four replicates

for each third chromosome). All replicates in a given block were assayed simultaneously. We performed a total of four complete blocks, yielding 16 measures of male performance and female longevity for each isogenic line. Females did not always produce offspring sired by the second male; these females and their offspring were excluded from analyses of male reproductive ability and female longevity. The number of replicates included per line varied from two to 14 (mean = 7.67 ± 0.47 ; mode = 9).

We evaluated the performance of males from different isogenic lines by analysis of variance, with line and subline as random effects. We used the general linear models procedure of the JMP statistical analysis software (SAS Institute 1997). Subline effects (effect of genetic background) were nested within lines and blocks. Both CI and a were transformed using the Box-Cox procedure to improve normality and homoscedasticity of residuals. After transformation, residuals were approximately normally distributed (CI: Shapiro-Wilk $W = 0.9843$, $P = 0.59$; a : Shapiro-Wilk $W = 0.9844$, $P = 0.60$) and homoscedastic. A few line-by-block combinations did not yield any useful male performance data because females did not produce any wild-type offspring. The line-by-block interaction effect was therefore not estimable.

We analyzed effects of male genotype on female survival by first calculating product-limit (Kaplan-Meier) survival estimates ($S[t] = [1 - d_i/n_i]$, where d_i is the number dying in interval i and n_i is the number that were alive at the beginning of the interval) for each census time for females mated to males of each genotype (Kalbfleisch and Prentice 1980). We then fit exponential, log-normal, and Weibull distributions to these product-limit estimates. Some recent analyses of *Drosophila* survival data have used a logistic or gamma-Gompertz distribution that includes a term specifying mortality deceleration at late ages (e.g., Khazaeli et al. 1998; Service et al. 1998a,b). However, sample sizes needed to detect significant deceleration are much larger than what we had available for our isogenic lines. We therefore confined our interest to the exponential, Weibull and log-normal distributions. A Weibull distribution

$$S[t] = \exp[-(\lambda t)^\kappa] \quad (1)$$

fit the data better than other models, producing approximately straight and parallel survival curves, whereas the other models produced dramatically nonlinear survival curves. The Weibull distribution describes a modified exponential survival curve in which the instantaneous death rate at time t , the hazard rate, is a function of time:

$$h(t) = \left\{ -\frac{d}{dt} [\log S(t)] \right\} = \kappa \lambda (\lambda t)^{\kappa-1}. \quad (2)$$

Under this model there is a linear relationship between age-at-death and $\log(-\log S[t])$. Because of this relationship, we could use a regression model in which the Weibull hazard is conditional on a set of covariates, \mathbf{z} , with associated regression parameters β (Kalbfleisch and Prentice 1980). We used this analysis to determine if there were significant differences in the hazard functions of females mated to different male genotypes and to determine if there were significant subline or block effects. That is, the Weibull hazard function for

females was assumed to be proportional to a function of male genotype, subline, and block (Kalbfleisch and Prentice 1980; JMP survival analysis module, SAS Institute 1997). In this analysis, we tested the significance of these effects by likelihood-ratio tests.

We used several different methods to determine if there was an association between female survival and the reproductive performance of her mates. First, we included male reproductive characters as covariates in the regression analysis described above to determine if male reproduction was significantly associated with hazard rate variation among females. Second, we calculated Spearman-rank correlations between CI and the median survival time for females mated to males of each line. We also calculated Spearman-rank correlations between CI and the Weibull κ and λ parameters (κ is the slope of the regression of on age at death and corresponds to a measure of the rate of increase of mortality with age; λ is the 63.2 percentile of the failure time distribution). Finally, because Sgro and Partridge (1999) have recently shown that costs of reproduction in females may be very age specific and that they may be delayed until the onset of senescence, we tested for correlations between CI and age-specific mortality.

RESULTS

Line medians for CI and line means for κ and λ are shown in Table 1. Weibull survival curves for the females are shown in Figure 1. Each line in the figure is the survival curve generated from all females mated to males of a particular third-chromosome genotype.

Analysis of variance indicated significant variation among isogenic lines for male reproductive performance, but no significant variation for male productivity (Table 2). Block effects contributed significant variation to both of these traits, whereas subline effects (effect of two different genetic backgrounds) were not significant for either.

The Weibull hazard rate for females was significantly affected by male genotype, genetic background, and block (Table 3). The significant genotype effect indicates that males of different genotypes differentially affected the survival of females with which they mated. The significant effect of subline suggests that these male genetic effects on female survival were not confined to genes on chromosome III; that is, the X and/or chromosome II also contain genes causing differential male effects on female survival. Neither CI nor a (after Box-Cox transformation) significantly improved the fit of the regression model, indicating that these male reproductive parameters were not significantly associated with variation in female survival (CI: likelihood ratio $\chi^2 = 0.03$, $P = 0.81$; a : $\chi^2 = 0.21$, $P = 0.65$).

Correlations between line means showed that CI was not significantly correlated with median survival time (Spearman's $\rho = -0.07$, $P = 0.72$), with the λ parameter of the Weibull distribution ($\rho = 0.07$, $P = 0.72$), or with the κ parameter of the Weibull distribution ($\rho = -0.16$, $P = 0.39$). Correlations between CI and age-specific mortality were all nonsignificant at $P > 0.25$ after correction for multiple tests using the sequential Bonferonni technique (Rice 1989).

Because CI includes the number of offspring sired by the

TABLE 1. Least-square line means for mate survival time and for CI. Median survival times and estimates of the Weibull parameters are also shown. Numbers in parentheses represent standard errors of least-square means (standard deviations for survival times). Line means for Weibull κ and λ (calculated as described in the text) are also given. Empty cells represent cases in which particular parameters were not estimable because of limited sample size.

Line	Female longevity	Median longevity	λ	κ	CI
4	55.43 (6.77)	67.5	60.27	3.66	4.96 (4.01)
7	46.67 (8.08)	47.0	51.16	4.74	-0.49 (6.97)
11	39.77 (5.48)	47.0	50.02	2.66	7.35 (4.65)
13	46.06 (5.76)	53.5	50.95	3.92	9.13 (4.33)
18	52.15 (3.31)	53.5	55.88	7.06	9.12 (3.56)
22	39.83 (4.27)	39.5	43.13	4.90	0.82 (4.53)
24	53.50	53.5			3.09 (8.50)
25	46.91 (6.66)	53.5	52.27	2.93	10.82 (4.10)
26	49.30 (2.61)	53.5	52.78	7.64	9.30 (3.87)
28	67.50	67.5			1.96 (4.33)
29	50.85 (3.78)	47.0	53.66	8.58	3.30 (5.58)
31	44.50 (5.35)	53.5	51.60	3.58	2.28 (4.67)
33	46.50 (3.81)	53.5	49.36	6.98	1.31 (4.92)
35	48.06 (4.01)	53.5	52.63	4.49	5.35 (3.52)
40	47.00	47.0			1.01 (6.01)
41	57.00 (3.50)	60.5	58.77	20.73	0.61 (4.53)
44	51.43 (3.06)	53.5	54.14	7.39	5.78 (3.87)
45	50.00 (3.50)	53.5	51.64	17.15	9.03 (6.04)
46	54.36 (2.40)	53.5	56.39	13.99	6.17 (4.16)
47	38.50 (1.13)		49.18	5.66	8.54 (4.53)
48	41.98 (3.99)	47.0	46.20	4.92	5.08 (4.11)
49	39.50 (7.98)	39.5	44.60	2.77	5.02 (5.59)
52	48.71 (4.40)	47.0	53.35	4.35	11.72 (3.74)
53	45.25 (6.23)	53.5	51.72	3.73	14.57 (5.07)
57	48.43 (5.75)	53.5	55.78	3.75	6.93 (4.53)
63	38.17 (4.89)	39.5	43.59	3.79	0.13 (4.53)
67	47.31 (6.09)	53.5	53.88	3.27	4.81 (4.44)
68	30.43 (4.85)	39.5	41.71	1.99	13.38 (4.65)
71	25.38 (3.14)	32.5	43.65	1.59	6.79 (4.11)
73	39.50 (3.79)	32.5	42.88	4.54	8.14 (4.65)
74	41.17 (4.54)	53.5	48.22	4.94	-0.18 (4.92)
75	45.53 (2.25)		49.75	6.79	8.62 (4.26)
77	48.97 (4.12)	60.5	51.19	4.75	3.49 (3.74)

wild-type experimental male in the numerator (a) and the number of offspring sired by the *ebony* competitor male in the denominator (b), we tested whether the correlations between survival parameters and CI might obscure underlying correlations between survival parameters and wild-type or *ebony* male reproductive success. To eliminate the confounding effect that long-lived females produced more total offspring simply because they lived longer, we restricted this analysis to include only those offspring produced in the first and second laying vials, before any females died or were censored. Using these measures, median female survival time was not correlated with productivity of her wild-type mate, a ($\rho = -0.05$, $P = 0.78$) nor with productivity of her *ebony* mate, b ($\rho = 0.03$, $P = 0.89$). λ was also uncorrelated with a ($\rho = 0.04$, $P = 0.82$) and b ($\rho = -0.06$, $P = 0.86$). However, κ was significantly correlated with a ($\rho = -0.42$, $P = 0.02$) and marginally significantly correlated with b ($\rho = 0.33$, $P = 0.07$). The significant correlation between κ and a indicates that females that mated to less productive wild-type males tended to have mortality rates that increased more quickly with age, whereas those that mated to highly productive wild-

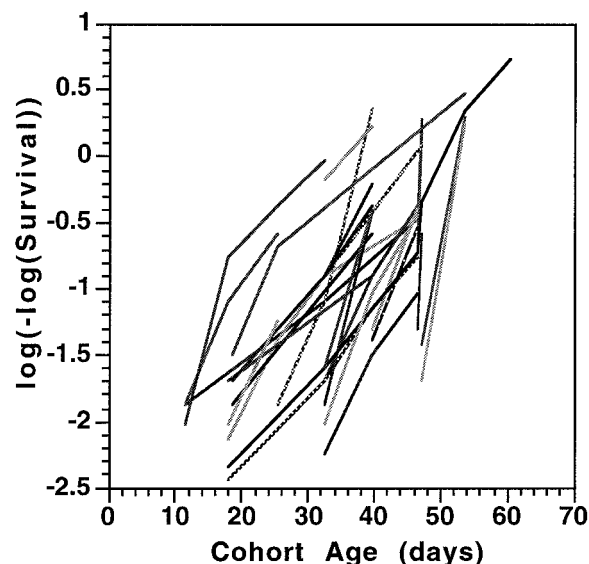


FIG. 1. Weibull survival curves for females mated to males of 33 different third chromosome isogenic lines, showing the relation between age and survival probability for females mated to males from each of the 33 isogenic lines. The vertical axis shows a transformation of the age-specific survival probability ($\log[-\log S(t)]$) for each genotype. The horizontal axis shows the age of females mated to each male genotype. The $S(t)$ values are product-limit (Kaplan-Meier) survival probabilities.

type males had mortality rates that increased more slowly. Note, however, that these correlations are no longer significant after correction for multiple tests. Correlations between age-specific mortality and productivity of each male type (a and b) were all nonsignificant at $P > 0.35$ after correcting for multiple tests (Rice 1989).

DISCUSSION

We found significant differences in the reproductive performance of wild-type males from a set of isogenic lines and significant differences in the life span of females mated to these males. Female survival was unaffected by the number of *ebony* or wild-type offspring that they produced. There was no indication of a trade-off or negative correlation between wild-type male performance (CI) and female survival. We did find a nominally significant correlation suggesting females mated to highly productive wild-type males (which were the second mates in our mating scheme) had mortality

TABLE 2. Effect of line, subline, and block on male reproductive performance (CI) and on total male productivity (a). Both dependent variables were transformed using the Box-Cox procedure before analysis.

Trait	Source of variation	df	SS	F
CI	block	3	100.0	2.97*
	line	32	766.6	2.14***
	subline (line, block)	63	708.8	1.07
Productivity	block	3	27451	66.28****
	line	32	5017	1.13
	subline (line, block)	63	8697	0.99

* $P < 0.05$; *** $P < 0.001$; **** $P < 0.0001$.

TABLE 3. Effect of line, subline, and block of males on the Weibull hazard rate (h). Analysis was conducted by parametric survival analysis (see text) and likelihood-ratio χ^2 -values are reported.

Trait	Source of variation	df	χ^2
h	block	3	32.27****
	line	32	59.65**
	subline (line, block)	63	84.62**

** $P < 0.01$; **** $P < 0.0001$.

rates that increased slowly with age. However, no strong inference can be made from these correlations because they were not significant after correcting for multiple tests.

Drosophila melanogaster males are known to show substantial intrapopulation genetic variation in courtship behavior (Ritchie et al. 1994), mating success (Hughes 1995a,b; Charlesworth and Hughes 1996), sperm-competitive ability (Clark et al. 1995; Hughes 1997), and many other traits. Although the data presented here do not directly support an antagonistic relationship between male fitness and female longevity, as suggested by Rice (1996), the critical male trait may simply not have been measured. We did not measure male behavior, copulation success or attempts, or the components or amount of seminal fluid transferred. Genetic variation in any of these male traits could potentially lead to differential female survival and all are worthy of future investigation. In addition, the genetic effects measured here are due to whole-chromosome effects, which may differ in direction and magnitude from effects of individual loci. That is, some alleles at loci on the third chromosome could well have antagonistic effects, but these effects might be obscured by effects at other loci. Finally, because our data were based on isogenic lines, the genetic variation we documented was due to homozygous effects of alleles. Determining whether there are substantial heterozygous effects will require testing male haplotypes or using half-sib or parent-offspring mating designs.

What our results do reveal is that male genotypic effects on female survival are polymorphic within the *IV* population and these genotypic effects are measurable even when females are exposed to males for a very brief time (24 h or less). Holland and Rice (1999) recently reported on the results of 34–45 generations of artificial selection in *D. melanogaster* under conditions that were divergent with respect to mating system and effective population size (single-pair matings vs. a 3:1 sex ratio). The selected populations differed in male effects on female longevity and in female survival under continuous exposure to males. Our results differ from those of Holland and Rice (1999) in that the genetic variability among males in our experiment reflects standing genetic variation within a single population, rather than evolved divergence between populations. In long-term selection experiments, mutational variance can contribute substantially to response to selection (Hill 1982; Hill and Rasbash 1986), so selection response cannot necessarily be attributed to standing variation in the ancestral population. Selection can also change the magnitude and sign of genetic correlations among traits (Falconer and Mackay 1996, pp. 199, 332). Thus, correlated responses to long-term selection do not directly reflect standing variation in the base population. Our experiment

therefore confirms that genetic variation among males in their effects on female survival can show substantial levels of standing variation within populations. The forces responsible for maintaining this variation are as yet undetermined, although theoretical models and quantitative genetic studies have suggested some possibilities (Prout and Clark 1996; Hughes 1997; Clark et al. 1999).

In addition to ruling out number of offspring as a direct cause of differential female survival, we were also able to evaluate some other potential causes. One of these is that isogenic lines could vary in the ability of males to induce females to increase their reproductive rate, thereby decreasing female longevity. Such an effect has been demonstrated by Service and Vossbrink (1996). We found no significant genetic variation among isogenic lines in male productivity, and as shown above, we found no association between number of wild-type offspring and female longevity. These results suggest that variation in male productivity is not the cause of variation in female longevity.

Another cause of differential female mortality could be that males of some genotypes may be able to induce females to mate more often, and the increased mating frequency may affect female longevity. Rice (1996) found that males that caused higher female mortality also induced higher female remating rates. We could partially address this possibility by ranking male genotypes by their success in getting nonvirgin females to mate with them. That is, each male genotype had the opportunity to mate with 16 nonvirgin females. As stated above, the number of these females that actually produced offspring from these matings varied from two to 14 per genotype. However, the correlation between proportion of females remating and median female longevity was small and not significant (Spearman's $\rho = 0.16$, $P > 0.35$). By this measure at least, the differences in female longevity were not associated with male success in inducing females to remate. Additional, more direct tests of this hypothesis are needed.

Gilchrist and Partridge (1997) have suggested that genetic variation in sperm-competitive ability may be partly or wholly attributable to genetic variation in the larval viability of the offspring produced by experimental males. If our isogenic lines varied genetically in larval viability, this could be the cause of the genetic variation in CI. However, results from a previous experiment indicate that larval viability differences cannot account for genetic variation in CI in this population (Hughes 1997). Even if larval viability differences did contribute to differences in CI, the male genetic variation on female longevity would remain unexplained.

Another possible cause of the male effect on female survival is variation in male behavior. Males that interact more with females, perform more courtship behavior, or are more aggressive in interactions may reduce female longevity. Partridge and Fowler (1990) demonstrated an effect of exposure to males on female longevity after controlling for differences in reproductive rate. Females that were exposed to *fruitless* or to microcauterized males (both of which will court but cannot mate) had shorter life spans than females not exposed to any males. In addition, microcauterized males courted more than *fruitless* males, and females lived longer when they were exposed to *fruitless* males. These results suggest that

male courtship activity can affect female longevity, irrespective of any effect on reproduction. It is therefore reasonable to hypothesize that genetic variation among isogenic lines in male behavior could be responsible for the observed effects on female longevity. An investigation of this hypothesis is currently underway.

In conclusion, we have measured an interesting and puzzling effect of male genotype on female longevity in sperm-competition trials. Whatever the cause, it must produce differential female mortality after short-term exposure to males, because females were exposed to males of different genotypes for only 24 h in our experiment.

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LITERATURE CITED

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