GENETICS OF LIFE HISTORY IN *DROSOPHILA MELANOGASTER*. II. EXPLORATORY SELECTION EXPERIMENTS

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ABSTRACT

Two types of small-scale selection experiments were performed. (1) Artificial selection experiments were performed on age-specific female fecundity. Selection for early fecundity over three generations produced a statistically detectable direct response. There was no detectable indirect response in other life-history characters. Selection for late fecundity over three generations did not produce any detectable direct response. Indirect responses were detected: early egg-laying decreased and longevity increased. (2) Natural selection for late-age fitness components increased late fecundity, female longevity, and the duration of female reproduction, while early fecundity and mean egg-laying rate decreased.

THE quantitative genetics of life-history characters are among the most important features of natural populations. The nature of the genetic variation for Drosophila viability (MUKAI *et al.* 1974), early fecundity (ROBERTSON 1957) and longevity (MAYNARD SMITH 1959) has received attention in the past, but the remainder of the Drosophila life history has been largely neglected, except for the works of GOWEN and JOHNSON (1946) and GIESEL (1979) on inbred lines.

Recent progress in age-structured population genetics theory (CHARLESWORTH 1980) motivated us to perform a small-scale sib analysis of complete adult female life histories (ROSE and CHARLESWORTH 1981). Some of our findings could not be tested statistically because of a confounding design variable. In any case, the small numbers involved in that experiment (about 1,200) necessitated further corroborative experiments.

Here we report the results of selection experiments that provide an exploratory check on the findings of ROSE and CHARLESWORTH (1981). The first of these experiments entailed artificial selection on both early and late age-specific fecundity. In the second experiment, natural selection on later life-history characters was contrived by reproducing a large population culture using only old females.

MATERIALS AND METHODS

The experimental population, culture conditions and phenotypic assay methods are described in ROSE and CHARLESWORTH (1981). Here we outline only the selection procedures.

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A. *Artificial selection experiment*: Two selection lines, "B" and "O", were paired with control lines simultaneously reproduced in identical fashion. The initial generations were started using the same methods as those used to obtain progeny for the sib analysis of ROSE and CHARLESWORTH (1981), except that the female parents of the initial generation were produced using larvae obtained from the tubes employed in the phenotypic assay of the mother's fecundity. For the B lines, these larvae were from tubes of assay day 5. For the O lines, they were from tubes of assay days 19 and 20.

Selection proceeded by choosing the parents with the highest 5-day fecundity record, from days 1 to 5 for the selected B line, and from days 21 to 25 are selected O line. (Only those females that laid eggs during the 5-day measurement period are discussed here.) The best 30% of those laying were chosen, but not all females produced sufficient larvae for the rearing procedure. Those that did not were discarded. Each remaining female was used as a source of larvae for the same number of tubes, and larval numbers were standardized over tubes, as was routine for the assay procedure. The control lines were reproduced using the same number of females, tubes per female, and larvae per tube, the only difference being arbitrary choice of females as parents.

There were three generations of selection. The initial generations of B and O lines were sampled simultaneously from the same base-population generation. The two experiments were thereby subject to the same seasonal laboratory conditions. The standarized selection differentials (FALCONER 1960) were about one per generation in both lines, giving a cumulative standardized differential after three generations of 3.18 for the selected B line and 3.31 for the selected O line. About 50 to 60 flies were assayed for the selected character per generation for each of the four lines. Complete adult female life-history assays were performed on all four lines during the fourth generation.

B. *Natural selection experiment*: All the available females from one generation of the base population were used to lay eggs first for the next generation of the base population, here called "CB", and then for the first generation of a new population culture, called "CO". For the next twelve generations, adults were collected from CO culture bottles at ages of 1 to 6 days from the time of eclosion. They were then supplied with 10 to 16 fresh bottles every 4 to 5 days until all adults were at least 21 days past eclosion. These adults were then used to lay eggs in 16 culture bottles for the next CO generation, and discarded to ensure discrete generations. Meanwhile, the CB population culture was reproduced, using adults of 1 to 6 days of age. Both population sizes remained large throughout. After the 12th CO generation, and over 20 CB generations, adults from the CO and CB populations were used to produce 104 sample progeny from *each* population for simultaneous life history assay.

RESULTS

A. *Artificial selection for age-specific fecundity*: Table 1 gives the response of the selected characters through the three generations of selection. The *t*-tests for differences of mean between selected and control lines suggest that the selected B line responded to selection, while the selected O line did not.

Table 2 compares the selected and control B lines after three generations of selection. (Longevity and the last day of egg laying are given in assay days, while the rate of laying is in eggs laid per assay day, while alive). There is no evidence of an indirect response to selection during the assay period.

Table 3 compares the selected and control O lines after three generations, demonstrating evidence of substantial indirect responses to selection. Early fecundity and egg-laying rate fell in the selected line relative to the control, while longevity and the length of the egg-laying period increased. This situation is puzzling, because there was no detectable *direct* response. However, the least-

TABLE 1

Direct response to selection over three generations

Generation	Selected		Control		t-test	
	Mean	Stan. dev.	Mean	Stan. dev.	d.f.	t
A. Selection for egg-laying days 1–5						
1	362.55	48.65	356.43	71.32	94	0.484
2	541.69	53.72	458.58	63.24	103	7.182†
3	547.00	88.42	501.02	53.79	104	3.204†
4	498.45	90.50	454.30	109.23	108	2.291*
B. Selection for egg-laying days 21–25						
1	248.36	104.46	224.78	100.17	115	1.235
2	230.00	124.21	189.03	120.07	110	1.739
3	179.86	99.99	161.34	90.11	121	1.080
4	188.60	114.38	192.77	91.82	59	−0.153

* Significantly different at $p < 0.05$.
† Significantly different at $p < 0.01$.

squares linear regression through the points giving the difference in daily egg-laying between selected and control *versus* day of assay was significant, having a slope of 0.9503 and a y-intercept of −15.68. This yields a *predicted* selected-line fecundity level much greater than that of the control line after assay day 18. This, in turn, supports the hypothesis that there was some, albeit extremely limited, direct response to selection, but that it was not detected because of en-

TABLE 2

Response to three generations of selection for egg laying during days 1 to 5

Character	Selected		Control		t-test	
	Mean	Stan. dev.	Mean	Stan. dev.	d.f.	t
Five-day egg laying:						
days 1–5	498.45	90.50	454.30	109.23	108	2.291*
days 6–10	439.80	144.12	444.65	149.34	104	−0.169
days 11–15	392.57	119.88	380.49	147.61	89	0.422
days 16–20	270.66	145.89	266.95	170.11	77	0.103
days 21–25	183.66	120.88	186.67	119.76	54	−0.091
Cumulative egg laying:						
days 1–5	498.45	90.50	454.30	109.23	108	2.291*
days 1–10	920.91	238.13	882.48	261.02	108	0.800
days 1–15	1229.36	408.42	1213.70	416.97	108	0.197
days 1–20	1420.50	537.66	1401.56	557.09	108	0.175
days 1–25	1525.45	627.16	1446.63	654.59	108	0.639
total	1612.64	719.34	1551.96	731.96	108	0.435
Longevity:	26.20	11.18	26.15	13.08	108	0.021
Last day of laying:	23.00	10.98	21.04	10.72	108	0.938
Rate of laying:	72.93	19.90	74.87	20.22	108	−0.503

* Significantly different at $p < 0.05$.

TABLE 3

Response to three generations of selection for egg laying during days 21 to 25

Character	Selected		Control		*t*-test	
	Mean	Stan. dev.	Mean	Stan. dev.	d.f.	*t*
Five--day egg laying:						
days 1–5	418.64	78.33	518.65	99.11	158	—7.037†
days 6–10	406.32	84.86	425.19	94.76	153	—1.298
days 11–15	378.23	115.42	360.05	120.21	148	0.939
days 16–20	240.95	143.08	222.39	123.21	113	0.744
days 21–25	188.60	114.38	192.77	91.82	59	—0.153
Cumulative egg laying:						
days 1–5	418.64	78.33	518.65	99.11	158	—7.037†
days 1–10	809.38	184.50	916.41	229.79	158	—3.228†
days 1–15	1173.43	289.35	1244.96	339.94	158	—1.433
days 1–20	1338.64	389.91	1401.89	423.39	158	—0.977
days 1–25	1422.40	454.25	1482.31	489.07	158	—0.798
total	1466.74	480.14	1487.35	524.90	158	—0.258
Longevity:	24.14	10.73	21.05	8.62	158	1.996*
Last day of laying:	21.58	9.63	18.88	7.37	158	1.979*
Rate of laying:	70.40	19.06	79.79	17.96	158	—3.187*

* Significantly different at $p < 0.05$.
† Significantly different at $p < 0.01$.

vironmental variation of greater magnitude. The extremely low heritability estimate for egg-laying days 21 to 25 found previously (ROSE and CHARLESWORTH 1981) bolsters the case for this hypothesis.

B. *Natural selection for late fertility*: Table 4 and Figure 1 compare the aggregated life-history assay data for CO and CB populations. (Here, we assume that sample variance heterogeneity does not render a comparison *t*-test invalid if it produces a result with a probability less than 0.01). Early fecundity and egg-laying rate are depressed in the CO population, while late fecundity, longevity and the length of the laying period are enhanced. Many of these effects correspond to the apparent effects of artificial selection for increased late fecundity.

DISCUSSION

A. *Interpretation of the results*: The first thing to be said about such unreplicated experiments is that they can not be used to provide quantitative estimates of genetic parameters (FALCONER 1977a). At most, they can indicate broad qualitative features of, or differences between, the populations subjected to selection. Therefore, the present experiments cannot be used to check the specific genetic parameter estimates given in ROSE and CHARLESWORTH (1981).

However, the present experiments do support the following findings of that article. First, the significant artificial selection response of early fecundity compared with the lack of response of late fecundity corroborates the earlier finding that the heritability of early fecundity is greater than that of late fecundity in *D. melanogaster*. Second, the negative indirect response of early fecundity to both

TABLE 4

Comparison of samples from the CO and CB populations

Character	CB Sample		CO Sample		t-test	
	Mean	Stan. dev.	Mean	Stan. dev.	d.f.	*t*
Five-day egg laying:						
days 1–5	551.30	56.84	421.91	88.47	201	—12.364‡
days 6–10	472.03	67.43	479.50	108.05	197	0.585
days 11–15	323.37	104.17	393.19	133.02	190	4.044†
days 16–20	238.78	113.78	286.69	131.18	165	2.431*
days 21–25	136.79	89.91	182.85	108.19	110	2.515*
Cumulative egg laying:						
days 1–5	551.30	56.84	421.91	88.47	201	—12.364‡
days 1–10	1018.73	116.90	890.56	199.33	200	—5.573‡
days 1–15	1332.68	203.06	1256.95	329.27	200	—1.966‡
days 1–20	1535.34	302.95	1488.64	455.86	200	—0.856
days 1–25	1611.04	360.59	1607.68	541.82	200	—0.052
total	1657.63	407.71	1699.68	623.42	200	0.567
Longevity:	26.79	9.07	30.25	11.53	200	2.283*
Last day of laying:	23.00	8.56	26.12	10.70	200	2.363*
Rate of laying:	76.93	16.68	68.61	18.66	200	—3.327†

* Significantly different at $p < 0.05$.
† Significantly different at $p < 0.01$.
‡ Significantly different at $p < 0.01$, but evidence for variance heterogeneity.

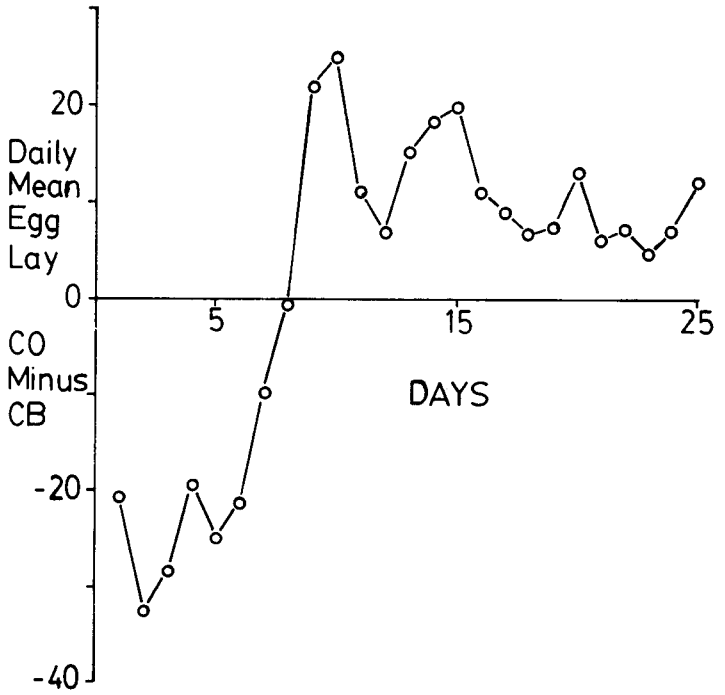

FIGURE 1.—Differences in mean daily conditional fecundities between samples of the CO and CB populations during assay days 1–25.

(a) artificial selection for late fecundity and (b) natural selection for late fertility suggests that there is indeed antagonistic pleiotropy among genes affecting early and late life-history attributes, as the sib analysis of covariance had tentatively suggested (ROSE and CHARLESWORTH 1981).

It could be argued that the absence of negative late life-history character responses to selection for early fecundity contradicts the second conclusion, but this need not follow. The laboratory culture schedule used to maintain the base population, discrete generations of 13 to 15 days length, strongly selected for early fertility. Under this regime, few genes that increased early fertility and depressed later life-history attributes alone would remain available for further selection. Selection would have much more of an opportunity to depress early fecundity, while increasing late fecundity, longevity, etc. Such asymmetries are not surprising on theoretical grounds (BOHREN, HILL and ROBERTSON 1966).

An experiment comparable to the natural selection experiment favoring late fertility was performed by WATTIAUX (1968) on a *Drosophila subobscura* population. Relative to cultures reproduced using young adults, a culture reproduced using old adults exhibited enhanced late fecundity and longevity, as well as depressed early male mating success and early fecundity, although not all of these differences were statistically significant. By selecting for early fertility, SOKAL (1970) and MERTZ (1975) depressed longevity in *Tribolium castaneum*. Despite the contradictory interpretations offered by WATTIAUX (1968), SOKAL (1970) and MERTZ (1975), all of these results may be explained in terms of the same pattern of antagonistic pleiotropy between early and later life history as that found in both the present selection experiments and the analysis of covariance of ROSE and CHARLESWORTH (1981). Though these earlier results *may* be explained in terms of pleiotropy, as the present results must be, they could also be explained in terms of mutation accumulation, since genetic variances and covariances were not estimated in these experiments. Clear evidence for the validity of the mutation-accumulation theory in such experiments would require evidence that the additive genetic variance of longevity is greater in populations with depressed longevity, due to selection for early fitness, compared with unselected controls of greater longevity. Of course, if the additive genetic variance is *not* greater, then the WILLIAMS (1957) pleiotropy theory is the only one that would be tenable.

LINTS and HOSTE (1974, 1977) performed Drosophila experiments that produced sporadic, perplexing fluctuations in absolute longevity and fecundity. Given the absence of controls, the hybrid origin of the stocks used and thus the lack of genetic equilibrium, as well as the variation in the age at the time of reproduction of some of the experimental populations, it seems difficult to relate their results to those given here. Certainly, it is not the case that these results challenge the validity of all selection experiments on life history, as these authors claim (LINTS and HOSTE 1977, p. 402; LINTS 1978, p. 102). RASMUSSON (1956) obtained absolute fluctuations in fecundity similar to those of LINTS and HOSTE (1977), but concluded instead that controls were needed to compensate for recondite environmental factors.

B. *Overall conclusions*: It is apparent that the results of the selection experiments reported here and the sib analysis of ROSE and CHARLESWORTH (1981) are in broad, qualitative agreement. The high heritability estimated for early fecundity in the sib analysis is reflected in the ease of increasing early fecundity by artificial selection. More generally, the genetic variability for life-history characters found in the sib analysis is proven by the variety of direct and indirect responses to selection, both artificial and "natural." In particular, the pattern of antagonistic indirect responses of egg-laying rate and early fecundity to increases in lifespan and late fecundity corresponds to the negative additive genetic correlations found for these characters. It could be argued that the indirect response to natural selection for late fertility was due to the accumulation of mutations depressing early fecundity alone, but this case cannot be made for the indirect response to artificial selection for late fecundity. In that experiment, both selected and control populations were free from natural selection against mutations depressing early fecundity. Even in the absence of any possibility of differential mutation accumulation, antagonism between early and late life-history characters is observed.

Certainly, this series of experiments is not entirely free of factors that suggest caution in interpretation. Nonetheless, two conclusions appear to be virtually indisputable. First, there is abundant genetic variability for life-history characters, variation that can respond to selection. Second, there appear to be appreciable antagonistic pleiotropic effects between early and late life-history characters. While these are modest conclusions, useful corollaries with relevance to a number of important issues may be derived.

(1) *Quantitative genetics of fitness*: As FALCONER (1977b) has pointed out, it is not possible to infer the genetic variance components of *fitness* from the genetic variance components of *components of fitness* if there is pleiotropy between fitness components that has not been fully delineated. Since such pleiotropy does indeed seem to exist, and moreover is antagonistic, it is *not* possible to reason from the abundant additive genetic variance for fecundity to the existence of abundant additive genetic variance for fitness itself. Indeed, all pleiotropic fitness effects could cancel out and the detected fitness-component variability could be neutral. Alternatively, as FALCONER (1977b) suggested, strictly additive genetic variability for fitness components coupled with antagonistic pleiotropy between them could give rise to heterozygote superiority for fitness itself and thus to polymorphic selective equilibria at which there is no additive genetic variance whatsoever for fitness itself.

(2) *Fisher's Fundamental Theorem*: From this, it is clear that, in cases of this kind, there is no ready conclusion to be drawn about the validity of Fisher's Fundamental Theorem (FISHER 1930) from the genetics of a subset of fitness components. In the present instance, other fitness components, such as male mating success (ANDERSON *et al.* 1979), remain unexamined, but could also be subject to pleiotropy; therefore, no conclusions about the genetics of total fitness can be obtained. Indeed, the apparently widespread additive genetic variation in the life-history characters of many species (*e.g.*, DERR 1980; DINGLE, BROWN

and HEGMANN 1977; ISTOCK 1981) can be seen to have no necessary relevance to the Fundamental Theorem. Considerably more research must be done before genetic data that can test the Fundamental Theorem will be available.

(3) *The evolution of senescence*: By contrast, the presence of antagonistic pleiotropy between life-history characters is of definite significance for the evolution of senescence. This is the first clear evidence for the sort of pleiotropic allelic effects postulated by WILLIAMS (1957). While the known phenotypic correlations were suggestive (*e.g.*, SNELL and KING 1977), such correlations are not an infallible guide to genetic correlations. In poultry, there are well-characterized instances where phenotypic correlations involving life-history characters, such as egg-laying, are opposite in sign to the genetic correlations (FALCONER 1960, pp. 315–316). Though some of additive genetic correlation estimates found in ROSE and CHARLESWORTH (1981) may be of the wrong sign, there are enough cases of positive phenotypic correlation for characters with negative genetic correlation estimates to suggest that the former is often a poor guide to the latter, insofar as Drosophila life history is concerned. More generally, it may be the case that antagonistic pleiotropy between life-history characters predominates among high-fitness alleles, as the results of SIMMONS, PRESTON and ENGELS (1980) suggest.

It might be thought that evidence for antagonistic pleiotropy based on the effects of segregating alleles could be attacked on the grounds that, while it may reflect the present state of a population, it does not show that such alleles were of importance in earlier stages of the evolution of senescence. However, formal models of pleiotropy between fitness components show that, of the simple genetic systems with antagonistic pleiotropy, some will go to fixation equilibria, while others will remain polymorphic (ROSE in preparation). The detection of the latter class of pleiotropic alleles is thus evidence for a larger class of such alleles, many of which would have been fixed in the past. In such cases, these are the genes that must have established senescence. On the other hand, in cases where the mutation-accumulation theory is correct, segregating variability due to deleterious late-acting alleles must remain in large populations, because such alleles are nearly neutral. Thus segregating genetic variability is, in general, an adequate guide to the evolutionary causes of senescence.

However, there is one important qualification to be made to the present evidence in favor of WILLIAMS' (1957) pleiotropy theory of senescence: there is no incontrovertible reason for supposing that this theory is correct for all other species. Depending on the physiological genetics of the organism concerned, either mutation accumulation or antagonistic pleiotropy could predominate in the evolution of senescence. However, it is nevertheless conceivable that the latter always predominates.

(4) *Reproductive effort and life-history evolution*: The antagonistic pleiotropy theory of WILLIAMS (1957) leads naturally to the reproductive-effort theory of evolutionary ecology (WILLIAMS 1966; GADGIL and BOSSERT 1970). Indeed, these are merely translations of one another. With pleiotropy, the evolution of senescence becomes bound up with the evolution of life history as a whole. Most

importantly, it appears to be the case that genes do not simply either enhance or depress every fitness component, and there is thus a genetic "trade-off" between life-history characters. However, in view of the substantial genetic variability that apparently may remain segregating for life-history characters, it seems unlikely that life-history phenotypes are precisely optimized, whether or not mean fitness is maximized or the Fundamental Theorem is in force.

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