GENES AFFECTING PRODUCTIVITY IN NATURAL POPULATIONS OF *DROSOPHILA MELANOGASTER*¹

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ABSTRACT

Two hundred second chromosomes were extracted from a Japanese population in October of 1972, and the viabilities and productivities of homozygotes and heterozygotes from them were examined. Viability was measured by the *Cy* method and productivity by the number of progeny produced per female. The frequency of lethal-carrying chromosomes was 0.315. When the average heterozygote viability was standardized as 1.000, the average homozygote viability was 0.595 including the lethal lines, and 0.866 excluding them. The frequency of recessive sterile chromosomes among 131 non-lethal lines was 0.092 in females and 0.183 in males. There were two instances in which homozygosis for the second chromosome caused sterility in both sexes, which was close to the number expected (2.2) on a random basis of $0.092 \times 0.183 \times 131$. When the average heterozygote productivity of 200 lines was standardized as 1.000, the average homozygote productivity was 0.532 including female steriles, and 0.584 excluding them. The ratio of detrimental load to lethal load was 0.383, while the ratio of partial sterility load to complete sterility load was 5.767. The average viability of lethal heterozygotes was slightly, but not significantly, lower than that of lethal-free heterozygotes, while the average productivity of lethal heterozygotes was significantly lower than that of lethal-free heterozygotes. There was a significant association of sterility in either sex with low viability of homozygotes. However, no statistically significant differences in viability and productivity were detected between sterile heterozygotes and non-sterile heterozygotes. The heterozygous effects of viability and productivity polygenes were examined by regressions of the heterozygotes on the sum of corresponding homozygotes. The regression coefficients were slightly positive for both viability and productivity if lethal and sterile chromosomes were excluded. The correlation between viability and productivity in homozygotes was significantly positive when sterile chromosomes were included, but the significance disappeared when the sterile chromosomes were excluded. In the heterozygotes there were no detectable correlations between them.

MOST references to genetic loads in populations of *Drosophila* have been concerned with viability, especially as measured using marker chromosomes. Fertility and fecundity are as important as viability, although their measurements are much more difficult and laborious. However, several studies of sterility

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genes in populations of *D. melanogaster* have been reported (SWEET and SPIESS 1962; TEMIN 1966; HOENIGSBERG, CASTRO and GRANOBLES 1969; and OSHIMA and WATANABE 1973).

The genetic variations which cause partial sterility of homozygous *Drosophila* females were revealed by TEMIN (1966), MARINKOVIC (1967a, b) and WATANABE and OSHIMA (1973) in various ways. TEMIN (1966) used a natural population of *D. melanogaster*; she measured the frequency of fertile individuals among ten flies for each line and regarded this as fertility. MARINKOVIC (1967a, b) used natural populations of *D. pseudoobscura*; he measured fecundity by the number of eggs laid and fertility by the number of adult progeny per female per six days of egg laying. WATANABE and OSHIMA (1973) measured productivity in a cage population of *D. melanogaster* by a technique similar to MARINKOVIC's.

The present study was undertaken to reveal genetic variations affecting productivity in a natural population and to compare it with those affecting viability. Attention was focused on the heterozygous effect of lethal-carrying chromosomes and polygenes controlling viability or productivity. Correlations between fitness components (viability, longevity and productivity) were also examined.

MATERIALS AND METHODS

Samples of *D. melanogaster* were collected in October, 1972 at Katsunuma, Japan. A total of 200 males ($+_i/+_j$) from these samples were crossed individually to three (*SM1*) *Cy/Pm* females. A single *Cy* male ($+_i/Cy$) from each cross was mated again with three (*SM1*) *Cy/Pm* females, and three *Cy* females ($+_i/Cy$) were crossed to three *Cy* males ($+_i/Cy$) in the next generation. Two hundred second chromosomes ($+_1, +_2, \dots, +_{200}$) were maintained as lines balanced with the (*SM1*) *Cy* chromosomes ($+_i/Cy$). The genetic backgrounds (*X, III, IV* chromosomes and cytoplasm) of these lines were made up of random chromosomes and cytoplasm from the same natural population, because the genetic backgrounds of (*SM1*) *Cy/Pm* flies had been replaced with chromosomes and cytoplasm from the same population.

Estimation of viability: Homozygote and heterozygote viabilities were estimated as follows. Crosses were made between 3 $+_i/Cy$ females and 3 $+_i/Cy$ males with 4 simultaneous replications in each chromosome line. In the offspring, $+_i/Cy$ and $+_i/+_i$ flies segregated with an expected ratio of 2:1. The viabilities of random heterozygotes were estimated similarly, except that two successively numbered lines were combined, i.e., $+_i/Cy \times +_{i+1}/Cy$, in order to secure random combinations of different chromosome lines. Three days after the crosses, all 6 flies were transferred to a second vial. Three days after the transfer, all flies were discarded. Flies emerging from both vials, the original and the transfer, were counted 3 times, until the 16th day after the cross or transfer. *Cy* and wild-type flies from the pair of vials were pooled and considered as a single observation. The viability was expressed as the percentage of wild-type flies among total number emerged. Forty lines were employed as a set, and five sets of replicates were made at five different times ($40 \times 5 = 200$). Homozygote and heterozygote viabilities were examined at the same time in each replication. All viabilities were standardized to the average viability of the heterozygotes for each replication.

Estimation of productivity: Productivity was examined in the generation immediately following measurement of viability. Two non-*Cy* females ($+_i/+_i$ in homozygotes, $+_i/+_{i+1}$ in heterozygotes) aged 48 hrs after eclosion were crossed to 4 Oregon-R males with 4 simultaneous replications for each line. Three days after the cross, all 6 flies in a vial were transferred to a second vial. Three days after the transfer, all flies were discarded. Flies from both vials were counted 3 times, until the 16th day after the cross or transfer, and pooled as a single

observation. The crosses, replications and standardizations in the estimation of productivity were done in the same way as for viability.

Adult viability (Longevity): Viability in this experiment can be considered as pre-adult viability or egg-to-adult viability. Viability of the adult flies is not included in it, because the adult viability is a part of longevity. Productivity was measured by the number of offspring from a female who laid eggs in her early adult life (from the 3rd to 8th day after eclosion). Some females died before the last day of their egg laying periods. However, they were not replaced with new females, since the adult viability of females also was a component of productivity. Eight females (2 females per cross $\times$ 4 simultaneous replications) per chromosome line were used for the productivity experiment. Therefore, the number of surviving females at 8th day ranged from 0 to 8. This was regarded as a measure of adult viability.

Male sterility: When the productivity of a chromosome in the homozygote was less than 0.1, the tested chromosome was regarded as having a female sterility gene. On the other hand, recessive male sterility genes were tested as follows. Five non-Cy males ($+_i/+_i$) from each chromosome line were crossed to five virgin Oregon-R females. If no larvae were observed after 5 days, the same cross was made in the next generation to confirm the male sterility.

All experiments were carried out at 25° under a fluorescent lamp, using 10 $\times$ 3 cm glass vials with about 10 ml of corn meal, agar, molasses and yeast medium. The total number of flies counted was 626,196 in the viability experiment and 502,400 in the productivity experiment.

RESULTS

Preliminary experiment on fecundity: To obtain data on the egg laying pattern of females, fecundity was examined daily from the 3rd to 22nd day after eclosion. One normal chromosome line was taken from the present material and was made heterozygous with Cy ($+_i/Cy$); it was also made homozygous ($+_i/+_i$). The two genotypes were inbred ($+_i/Cy \times +_i/Cy$ or $+_i/+_i \times +_i/+_i$), and transferred daily to a new medium. Each cross was replicated 32 times. The average number of eggs laid per female per day are shown in Figure 1. The peak of egg laying was at the 5th or 6th day after eclosion; the number of eggs decreased gradually after the 8th day in both homo- and heterozygotes. About half of the eggs were deposited by the 8th day. The ratio of eggs laid by the homozygous females to those laid by the heterozygous females was 0.80 when pooled through the 8th day, and 0.83 through the 22nd day. These results suggest that egg laying ability was maximal in the early 6 days (from 3rd day to 8th day), and the relative fecundity in the early days was an effective way to estimate the lifetime fecundity of *D. melanogaster*. Thus, estimating productivity in young females, as in the present experiment, will suffice to measure lifetime productivity.

Distributions of viabilities and productivities: The viabilities and productivities of homozygotes and heterozygotes for 200 second chromosomes were examined. The distributions are given in Table 1 and graphically presented in Figure 2. The number of lethal-carrying chromosomes that showed viability indices lower than 0.1 was 63. The frequency of lethals was therefore 0.315. About 60% of the second chromosome fell in the range of normals (from 0.75 to 1.25 of the viability indices). The distribution of viabilities of random heterozygotes was much narrower, and was rather clustered about 1.00.

FIGURE 1.—Average number of eggs produced per female each day for the two genotypes of *D. melanogaster*.

TABLE 1

Distributions of homozygote and heterozygote viabilities and productivities

Index	Viability		Productivity	
	Homozygotes	Heterozygotes	Homozygotes	Heterozygotes
0 -0.05	60		9	
0.05-0.15	5		9	
0.15-0.25	8		6	
0.25-0.35			9	
0.35-0.45	3		13	1
0.45-0.55	1		16	1
0.55-0.65	2		19	2
0.65-0.75	5		18	5
0.75-0.85	7	2	16	10
0.85-0.95	44	24	11	37
0.95-1.05	58	144	4	75
1.05-1.15	7	30	1	47
1.15-1.25				18
1.25-1.35				4
Total	200	200	131	200

FIGURE 2.—Frequency distributions of homozygote and heterozygote variabilities (top) and productivities (bottom) of second chromosomes.

Among 131 non-lethal chromosomes, the number of female sterile chromosomes that showed productivity indices lower than 0.1 was 12. The frequency was 0.092. The variation of homozygous productivity was very large, and the total productivity load was great. Only 25% of the non-lethal chromosomes showed normal productivity (from 0.75 to 1.25 of the productivity indices). The distribution of productivities of the random heterozygotes was also very broad (from 0.35 to 1.35 of the productivity indices).

The frequency of male sterile chromosomes among 131 non-lethal chromosomes was 0.183 (24/131). As reported by OSHIMA and WATANABE (1973), a clear parallelism among frequencies of lethal, male sterile and female sterile chromosomes in the ratio 1 : 1/2 : 1/4 was again observed; i.e., 0.315 : 0.183 : 0.092. In the present populations, chromosomes which led to sterility in the homozygotes of both sexes were detected. Table 2 shows the number of chromosomes causing no sterility (fertile), male sterility, female sterility and sterility in both sexes. Expected numbers were calculated under the assumptions that male sterility genes and female sterility genes are independent and that no genes cause sterility in both sexes. The number of chromosomes causing sterility in both sexes were very similar to that expected. This result was different from those

TABLE 2

Observed and expected numbers of chromosomes causing sterility when homozygous in males, females and in both sexes

Chromosome	Observed	Expected
Fertile	97	97.2
Male sterile	22	21.8
Female sterile	10	9.8
Both sex sterile	2	2.2
Total	131	131.0

obtained from the same population in 1968 and 1970 (OSHIMA and WATANABE 1973), when an excess of flies sterile in both sexes was found.

Homozygous load: The average viabilities of all homozygotes, non-lethal homozygotes and all heterozygotes are given in Table 3A. Using the formulae of GREENBERG and CROW (1960), the genetic loads due to viability genes were calculated; they are given in Table 3C. Because of a few semilethal (0.1–0.5 of viability indices) and subvital (0.5–0.75 of viability indices) chromosomes, the average viability of non-lethal homozygotes was very high (0.866). Consequently, the detrimental load was calculated to be small. The ratio of detrimental load to lethal load (D:L ratio) was 0.383. This figure is smaller than the 0.584 which was obtained by KOSUDA (1971) for the same natural population in 1968.

TABLE 3

Average liabilities (A) and productivities (B), and homozygous genetic loads (C) for second chromosomes from the Katsunuma population

A.			
Chromosome		<i>N</i>	Average viability
All homozygotes		200	0.595 ± 0.031
Non-lethal homozygotes		137	0.866 ± 0.020
All heterozygotes		200	1.000 ± 0.004
B.			
Chromosome		<i>N</i>	Average productivity
All homozygotes		131	0.532 ± 0.024
Non-sterile homozygotes		119	0.584 ± 0.022
All heterozygotes		200	1.000 ± 0.009
C.			
Viability		Productivity	
Total viability load	0.519	Total productivity load	0.631
Lethal load	0.375	Complete sterility load	0.093
Detrimental load	0.144	Partial sterility load	0.538
D:L ratio	0.383	P:C ratio	5.767

N indicates the number of lines or crosses.

TABLE 4

Average viabilities and productivities of lethal-free, single-lethal, and double-lethal heterozygotes

Genotype	No. of crosses	Average viability	Average productivity
Non-lethal/Non-lethal'	90	1.004 $\pm$ 0.005	1.030 $\pm$ 0.013*,**
Lethal/Non-lethal	94	.997 $\pm$ 0.006	.980 $\pm$ 0.015*
Lethal/Lethal'	16	.987 $\pm$ 0.011	.946 $\pm$ 0.021**

* and ** indicate statistical significance at the 5% and 1% levels, respectively.

The average productivities of 131 non-lethal homozygotes, non-sterile homozygotes and all heterozygotes are given in Table 3B. Using the formulae of TEMIN (1966), the genetic loads due to productivity genes were calculated and are presented in Table 3C. Because there were so few cases of complete sterility, the complete sterility load was only 0.093, while the partial sterility load was 0.538. The ratio of partial sterility load to complete sterility load (P:C ratio) was 5.767, which was more than 15 times the D:L ratio. From the above results it can be seen that the average second chromosome in the Katsunuma population contains about 0.5 lethal equivalent load and at least 0.6 sterility equivalent load in addition. The former is largely due to lethal genes and the latter to partial sterility genes which can be named productivity polygenes.

Viability and productivity of lethal heterozygotes: Out of 200 random heterozygotes, 90 were lethal-free, 94 were single-lethal and 16 were double-lethal. Single-lethal means that one of the homologous chromosomes carries at least one recessive lethal gene. Table 4 shows the results. The average viabilities of lethal-carrying heterozygotes were slightly lower than those of lethal-free heterozygotes, but there were no significant differences among them.

On the other hand, the average productivities of lethal-carrying heterozygotes were significantly lower than those of lethal-free heterozygotes. Thus, lethal genes reduce the productivity of their carriers, even if the lethal genes have no effects, or slight ones at best, on viability in the heterozygous condition (WATANABE and OSHIMA 1973; WATANABE, YAMAGUCHI and MUKAI 1975).

Effects of sterility genes: The average viability and productivities of sterile homozygotes are shown in Table 5. Chromosomes which caused sterility in

TABLE 5

Homozygote viabilities and productivities of sterile chromosomes

Chromosome	No. of lines	Average viability	Average productivity
Non-females sterile	119	.914 $\pm$ 0.015**	.584 $\pm$ 0.022**
Female sterile	12	.737 $\pm$ 0.068**	.022 $\pm$ 0.008**
Non-male sterile	107	.923 $\pm$ 0.013*	.541 $\pm$ 0.027
Male sterile	24	.784 $\pm$ 0.054*	.493 $\pm$ 0.059

* and ** indicate statistical significance at the 5% and 1% levels, respectively.

either female or male homozygotes were significantly associated with low viability. This relationship between low viability and sterility has been reported by SWEET and SPIESS (1962), TEMIN (1966) and OSHIMA and WATANABE (1973) in *D. melanogaster*, and by DOBZHANSKY, HOLZ and SPASSKY (1942) in *D. pseudoobscura*. The homozygous productivity of female sterile chromosomes, whose productivity indices were lower than 0.1, was of course close to 0 (0.022). Male sterile chromosomes were slightly deleterious in homozygous productivity, but a significant reduction was not detected. The reason may be the small number of the sterility lines which were examined.

Table 6 gives the average viability and average productivity of sterile heterozygotes. There were no detectable differences in viability or in productivity between fertile heterozygotes and sterile heterozygotes. More precise examinations using many sterile lines are needed to clarify the homozygous (pleiotropic) effect and also the heterozygous (dominance) effect of sterility genes.

Polygenes controlling viability or productivity: To test the heterozygous effect of chromosomes which do not carry a lethal gene but contain other deleterious mutations, regression coefficients (*b*) of the non-lethal heterozygote viabilities on the sum of corresponding homozygote viabilities were examined. A set of regressions were done, the first involving only chromosomes with viability indices larger than 0.1, the second those whose viability indices were larger than 0.2, and so on. If the polygenes manifest dominance the coefficient is expected to be positive, if they are recessive it will be zero, and if overdominant it will be negative. Table 7 shows the results. The regression coefficient was slightly positive for different viability cutoffs, and it increased to become significantly positive as the index became higher. This suggests that the genetic load is primarily mutational, since the effect of the chromosomes as homozygotes is qualitatively similar to the effect in heterozygotes.

The heterozygous effects of polygenes affecting productivity were also examined; the results are shown in Table 7. Sterile heterozygotes were, of course, excluded. The regression coefficients were again positive for every productivity index, and they were somewhat higher than those for viability. Thus, much of the partial sterility load in the population is eliminated by selective disadvantage in the heterozygotes.

TABLE 6

Average viabilities and productivities of sterile-free heterozygotes and single-sterile heterozygotes

Genotype	No. of crosses	Average viability	Average productivity
Non-female sterile/Non-female sterile'	68	1.001 $\pm$ 0.006	1.033 $\pm$ 0.015
Female sterile/Non-female sterile	13	1.008 $\pm$ 0.012	1.014 $\pm$ 0.023
Non-male sterile/Non-male sterile'	55	1.008 $\pm$ 0.006	1.031 $\pm$ 0.018
Male sterile/Non-male sterile	23	.990 $\pm$ 0.011	1.027 $\pm$ 0.019

TABLE 7

Regression coefficients (b) of heterozygotes viability and productivity on the sum of corresponding homozygote viability and productivity with standard deviations (sb)

Index†	Viability			Productivity		
	N	b	sb	N	b	sb
> 0.1	90	0.027	0.033	68	0.075	0.046
> 0.2	80	0.060	0.026	62	0.102*	0.051
> 0.3	78	0.033	0.029	48	0.129	0.066
> 0.4	78	0.033	0.029	39	0.151	0.082
> 0.5	76	0.032	0.033	29	0.108	0.097
> 0.6	74	0.043	0.044	17	0.241	0.146
> 0.7	68	0.062	0.055	9	0.873**	0.237
> 0.8	59	0.133	0.073			
> 0.9	45	0.243*	0.110			

* and ** indicate statistically significant differences from 0 at the 5% and 1% levels, respectively.

† Viability indices or productivity indices of the chromosomes used for the estimation of the regression coefficients.

N indicates the number of heterozygous crosses.

Pre-adult viability and adult viability: Table 8 gives the adult viabilities, which were calculated as the number of females alive on the 8th day after the eclosion. In 60% of homozygous lines, all females lived past the 8th day. Five lines among the 131 homozygotes had the shortest lives, with six females among 8 sisters dying before the 8th day. On the other hand, in 90% of heterozygous lines, all tested females lived past the 8th day. In no heterozygous line did more than 3 of 8 sisters die before the 8th day.

The relation between pre-adult viability and adult viability is shown in Figure 3. The average pre-adult viability of homozygotes, whose adult viability

TABLE 8

Adult viabilities measured as the number of females alive eight days after eclosion

Adult viability index	Homozygote		Heterozygote	
	N	%	N	%
8 (All alive)	78	59.5	179	89.5
7	29	22.1	18	9.0
6	10	7.6	3	1.5
5	6	4.6	—	—
4	1	0.8	—	—
3	2	1.5	—	—
2	5	3.8	—	—
1	—	—	—	—
0 (All dead)	—	—	—	—
Total	131		200	

N indicates the number of lines.

FIGURE 3.—Regression of pre-adult viability on adult viability in homozygotes ($n = 131$, $Y = 0.0427X + 0.5924$).

index was minimum (2), was 0.624, and that for adults whose viability index was maximum (8) was 0.944. There was a significant positive regression of pre-adult viability on adult viability ($b = 0.0427$, $sb = 0.0101$). Even when the first point (adult viability index = 2) is omitted the regression coefficient was significantly positive ($b = 0.0323$, $sb = 0.0142$). Thus, individuals more viable in the young life stages (egg, larva, pupa) will live longer as adults. HADORN (1961) ascribed such effects to polyphasic factors, and TEMIN (1966) obtained data for their existence.

Correlation between productivity and (pre adult) viability: In Table 9 are summarized the correlations between productivity and viability of the chromo-

TABLE 9

Correlation coefficients (r) between productivity and viability in homo- and heterozygotes

	N	r	t
Homozygotes:			
All	131	0.304	3.626**
Excluding male steriles and female steriles	109	0.105	1.094
Special†	78	0.057	0.498
Heterozygotes:			
All	200	0.030	0.418
Excluding lethals	90	0.053	0.496
Excluding lethals, male steriles and female steriles	45	-0.088	0.578

N indicates the number of combinations.

r = correlation coefficient.

$t = r\sqrt{(N-2)/(1-r^2)}$

† Chromosome lines whose homozygous females did not die during their egg laying period in the productivity experiment.

** indicates statistical significance at the 1% level.

some lines. The correlation in the homozygotes was significantly positive when sterile chromosomes were included. However, the correlation became smaller and non-significant when the sterile chromosomes were excluded. The complete sterility types were thus apparently associated with low viability, as already shown in Table 5. If the chromosome lines in which some sisters of the same genotypes died during their egg laying period were excluded (Special in Table 9), the correlation coefficient declined much more, to 0.057. In the heterozygotes, the correlations were very close to 0, even when lethals or sterile chromosomes were excluded. These results are basically similar to those reported by TEMIN (1966) and by WATANABE and OSHIMA (1973). There was no evidence for negative correlations between viability and productivity for high viability chromosomes (cf. HIRAIZUMI 1961).

DISCUSSION

A large amount of genetic variation controlling productivity is concealed in natural populations of *D. melanogaster*. The total productivity load, which was largely due to the partial sterility load, was 0.631 female sterile equivalents when non-lethal second chromosomes from the Katsunuma population were examined. In addition, the population maintained a viability load of 0.519 lethal equivalents. Thus, the average second chromosome carried the equivalent of 1.15 genes which made the flies to be lethal or female sterile when homozygous. If we take into consideration male fertility, adult viability and other fitness components, the homozygous genetic load would be doubled.

There are apparently different patterns in the distributions of viability and productivity. The former has a typical bimodal distribution with one peak at the lethals and another peak at the normals, while the latter distribution was rather unimodal, having a weak peak at the subfertiles. MARINKOVIC (1967a, b) and WATANABE and OSHIMA (1973) already reported similar results.

The deleterious major genic variants in *Drosophila* populations are mostly complete lethals and complete steriles. They originate as mutations at rates comparatively lower than for polygenic mutations mildly affecting viability (MUKAI 1964). However, most natural populations contain fewer lethals than the expectations calculated by a balance between mutation and selection on homozygotes. Therefore, some selection against heterozygotes must be expected (CROW 1964; NEI 1968). It has been a point of interest whether lethal mutations are deleterious or heterotic in the heterozygotes. Many experiments were conducted by many investigators. However, the effects of lethals on the viability of lethal heterozygotes were not always deleterious (DOBZHANSKY and SPASSKY 1968; MARINKOVIC 1967b; WALLACE and DOBZHANSKY 1962; WALLACE, ZOUROS and KRIMBAS 1966; BAND and IVES 1963). The present experiment shows the deleteriousness of the lethal heterozygotes in *productivity* but not in *viability*. This had previously been suggested by the results of WATANABE and WATANABE (1973) and of WATANABE, YAMAGUCHI and MUKAI (1975). Thus, lethal genes are selected by productivity in the heterozygotes.

Sterility genes are another class of major genic mutation. The frequencies of recessive sterility genes in natural and cage populations of *D. melanogaster* have been reported by several investigators (SWEET and SPIESS 1962; TEMIN 1966; HOENIGSBERG, CASTRO and GRANOBLES 1969; OSHIMA and WATANABE 1973). OSHIMA and WATANABE (1973) showed a clear parallelism among the frequencies of lethals, male steriles and female steriles, their respective proportions being 1, $\frac{1}{2}$ and $\frac{1}{4}$. The present study confirmed this finding. Homozygotes for genes conferring sterility in both males and females showed low viabilities. A pleiotropic effect of sterility genes on viability was thus suggested in the homozygotes, but no deleterious effect such as were found for lethals was detected in the heterozygotes. This may be due in part to the small numbers of sterile chromosomes.

Genetic variations, except the lethal and sterility genes, may be ascribed to polygenes which control viability or productivity. The homozygous effects are small and mild compared with those of lethals or steriles. The heterozygous effects of the polygenes were examined by the regression method. The regression coefficients of the heterozygote on the sum of corresponding homozygotes were consistently positive for both viability and productivity. Thus, the polygenes are, on the average, selectively disadvantageous in the heterozygotes. The regression coefficients in productivity were significantly positive even when chromosomes causing low productivity were used. This means that the low productivity chromosomes are also made up of accumulated polygenes rather than being the result of a single major gene which causes semisterility.

Finally, correlations between productivity and viability are scarce both in homozygotes and heterozygotes. TEMIN (1966) obtained a weak but not significant positive correlation; MARINKOVIC (1967a, b) found a similar result in a natural population of *D. pseudoobscura*. WATANABE and OSHIMA (1973) reported that no detectable correlations were observed, either in homozygotes or in heterozygotes in the analysis of a cage population of *D. melanogaster*. In the present experiment, a significantly positive correlation was detected only when sterile chromosomes were included. However, this was largely due to the pleiotropic effect of sterility genes on homozygous viability. Therefore, if the sterile chromosomes were excluded, the correlation was reduced to non-significance. The pre-adult viability and adult viability were, as mentioned previously, positively correlated. If chromosomes showing full survival in early adulthood of the homozygotes were used, the correlation coefficient drops to 0.057 ($n = 78$). From these figures, together with those from the heterozygotes, it can be concluded that female productivity polygenes are not correlated with viability polygenes and are in large part independent mutations.

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