VIABILITIES OF THIRD CHROMOSOMES OF *DROSOPHILA*
PSEUDOOBSCURA DIFFERING IN RELATIVE
COMPETITIVE FITNESS¹

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ABSTRACT

Arrowhead (AR) third chromosome arrangements of *Drosophila pseudoobscura*, whose competitive fitnesses had been determined in population cages, were tested for their genetic loads in homozygous, heterozygous (homokaryotypic), and heterokaryotypic (AR/CH) combinations. The results showed that their competitive population cage performances were correlated to their viabilities as homozygotes but were not correlated to their viabilities as heterozygotes or as heterokaryotypes. However, the results do not fit in too simply with the mutational model of population structure, since the improvement of homozygous viability with increased competitive fitness was not accompanied by a significant degree of dominance as measured by the regression of viabilities of heterozygotes on homozygotes. Only the AR chromosomes derived from the population with poorest competitive fitness showed marked partial dominance ($\bar{h} = .35$). The viabilities of heterokaryotypes were markedly uniform for all chromosomes tested and produced significantly greater numbers of flies per culture than the homokaryotypes. In general, the results show that the ranking of relative competitive fitnesses of these chromosomes is not a simple extrapolation of their viabilities, although marked changes in the populations tested have occurred. It is proposed that the differences in competitive fitness, homozygous viability, and degree of dominance observed among these chromosomes, arise from differences in genetic variability which enable different linkage relationships to be established for genes affecting these attributes.

GENETIC load has been defined as a reduction in the average fitness of a population, or in the fitness of the average genotype of a population caused by the presence of deleterious genes or of other factors which lead to differences in fitness between genotypes (MULLER 1950; MORTON, CROW and MULLER 1956; CROW 1958; and other publications). Differences in genetic loads of populations should therefore be reflected in comparisons between their relative fitnesses, that is, an inverse correlation may be expected between load and fitness. Experimentally, it would therefore be highly desirable if both these attributes of a population could be measured simultaneously. The present experiments have approached this problem by offering estimates of the genetic loads carried in third chromosomes of *Drosophila pseudoobscura* which were shown to differ in their relative competitive fitnesses as measured in population cages. Also, to distin-

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guish whether the loads observed reflect mutational or segregational origin (for review see DOBZHANSKY 1970) estimates were made of the dominance effects in heterozygous condition of chromosomes with known viabilities in homozygous condition. A further test was to place these chromosomes into particular heterokaryotypic combinations to determine whether their relative viabilities as heterokaryotypes also reflect their relative competitive fitnesses.

MATERIALS AND METHODS

The populations of *D. pseudoobscura* used to provide the test chromosomes of the present experiments have been previously described (STRICKBERGER 1969). They consist of individual laboratory populations maintained in cages which differ in respect to the numbers and histories of the Arrowhead (AR) arrangement third chromosome strains used to initiate them. In some instances, single AR strains were used, while in others 2, 4, 6, or 8 AR strains were used, all originating in the 1940's from Pinion Flats, California. In the experiments described (ibid.), various populations founded with these AR strains were permitted to undergo selection for population cage conditions for periods of about 20 months. Flies were then extracted, crossed to a common sets of strains carrying the Chiricahua (CH) arrangement, and the relative fitnesses of the AR chromosomes were then measured by observing their frequencies for a one-year period in population cages. Of the results obtained, the most relevant to the present experiments was that the greater the initial genetic variability (in terms of numbers of different strains) present in an AR third chromosome population, the greater the competitive fitness against CH under test conditions. Specifically, AR's derived from populations which had initially been started with eight different AR strains reached frequencies between 70-80% after one year's competition with CH; AR's from populations begun with four different AR strains reached frequencies between 50-60%; while AR's from a population carrying only a single AR strain (AR2 in the present experiment) maintained a frequency of only between 30-40%. In the present experiments, these three different AR populations were labeled respectively "A" (8 AR strains), "B" (4 AR strains) and "C" (1 AR strain). They were separately maintained while the competition tests were going on with CH, and the present experiments were begun immediately after the results of the competition were known.

Since the present purpose was to test the homozygous and heterozygous viabilities of third chromosomes, a marker stock was prepared carrying the Santa Cruz third chromosome arrangement (marked by the gene *Lobe*) and the Standard arrangement (marked by the genes *Blade* and *Scute*). The X and Y chromosomes of this stock as well as almost the entire autosomal background, other than the third chromosome, were derived from one of the AR stocks by backcrossing for 6 generations.

Extraction of AR chromosomes for viability tests from each of the three experimental populations was then begun by removing single males from the cages and crossing them to marker stock *L/Bl Sc* females. In the next generation, single AR/*L* male progeny from each original male were backcrossed to marker stock females. This produced replicates of single AR chromosomes in each culture which were then crossed AR/*L* $\times$ AR/*Bl Sc* to provide the four phenotypically distinct classes of offspring, AR/AR (+/+), AR/*L* (lobe), AR/*Bl Sc* (blade, scute), *L/Bl sc* (lobe, blade, scute), which were then counted. These final crosses were made by allowing eight pairs of parents to lay eggs successively in four replicate cultures (half-pint bottles of cornmeal-agar-molasses medium). Since the flies did not usually begin to lay eggs immediately, the parents were kept in the first culture for about 6 days, and in succeeding cultures thereafter for about 3 days. Homozygous AR_x/AR_x progeny were produced from crosses in which the parents were all derived from a single AR chromosome from a single AR male, whereas the heterozygous AR_x/AR_y progeny were produced from crosses in which the male parents (AR_x/*L*) came from one such male and the female parents (AR_y/*Bl Sc*) from another. The tests were made in weekly groups of about 5 to 10 chromosomes from each of the three populations, and were performed simultaneously both in homozygous and heterozygous combinations. Each chromosome was tested

in one homozygous combination and two heterozygous combinations, e.g., AR_1/AR_1 , AR_1/AR_2 , AR_2/AR_2 , AR_2/AR_3 . . . AR_5/AR_1 . In all, about 50 chromosomes each were tested from the three populations.

Heterokaryotypes (AR/CH) were tested after the homozygous and heterozygous tests were completed, and were produced by again extracting single AR chromosomes from each population using the *L/Bl Sc* marker stock. Single AR/*L* males were then backcrossed to *L/Bl Sc* females to replicate the AR chromosome, and in the next generation final crosses were made between AR/*L* males and females heterozygous for the Chiricahua and Standard arrangements (CH/*Bl Sc*). The CH chromosome used was identical in all AR/CH tests and had been isolated, using the marker stock and salivary chromosome analysis (for technique see STRICKBERGER 1963) from a population cage in which a single strain of AR (AR2: population "C") had been competing with CH. Each heterokaryotypic test was performed by permitting 6 pairs of parents to lay eggs in four replicate cultures as above, and then counting the four phenotypically distinct classes: AR/CH (+/+), CH/*L* (lobe), AR/*Bl Sc* (blade, scute), and *L/Bl Sc* (lobe, blade, scute). Again, weekly groups of 5–10 chromosomes were tested simultaneously from each of the three AR populations, and the tests were continued until a total of about 50 chromosomes from each were scored.

An average of 810 flies were counted for each chromosome tested in the homozygous and heterozygous crosses, and an average of 1,072 flies were counted for each chromosome in the heterokaryotype crosses. *In toto*, 406,495 flies were scored, all raised at 25°C.

RESULTS

Using the viability of the *L/Bl Sc* class as the standard (1.000), the relative viabilities of the other three segregating classes is given in Table 1 for each of the three AR populations and for each of the three types of crosses. These results shown an improvement in homozygous viability of AR chromosomes in the direction $A > B > C$, although in only one comparison (*A* vs. *C*) are the differences significant ($P < .05$). The viabilities of the heterozygous combinations of the three populations are obviously not correlated with the order of homozygous viabilities, nor are there significant differences between the heterozygous viabilities of the different populations. In two populations, B and C, heterozygous combinations appear more viable than homozygotes, whereas the reverse occurs for population A. However, these latter comparisons are not statistically significant. The heterokaryotype (AR/CH) genotypes show relative uniformity in viabilities which are all significantly below ($P < .001$) the viabilities of any of the homozygous or heterozygous AR/AR combinations.

In general, viabilities of the +/+ classes are higher than those reported by SPASSKY *et al.* (1960), but this may arise from the fact that the *L/Bl Sc* marker stock they used did not have the background chromosomes of the tested population. Another possible source for the relatively high homozygous +/+ viabilities in the present experiments lies in the selection method used to obtain AR chromosomes to found the three populations: as previously described (STRICKBERGER 1969), only AR chromosomes viable in *isogenic* condition were used. Thus, lethal and semi-lethal chromosomes were initially excluded, and did not apparently accumulate subsequently.

In respect to the *L*/+ and *Bl Sc*/+ classes in Table 1, two important observations can be made:

1. There is an obvious correlation between the average viabilities of the +/+ and *L*/+ classes ($r = .98$).
2. The average viabilities of the *Bl Sc*/+ class are

TABLE 1
Number of chromosomes tested and mean relative viabilities of given genotypes (with standard errors) from each set of crosses compared to the L/Bl Sc class (viability = 1.000)

Population	Homozygote crosses $L/+$			Heterozygote crosses $L/+$			Heterozygote crosses $L/+$			Heterozygote crosses $L/+$		
	Number	(AR_x/AR_x)	$Bl Sc/+$	Number	(AR_x/AR_x)	$Bl Sc/+$	Number	(AR_x/AR_x)	$Bl Sc/+$	Number	(AR/CH)	$Bl Sc/+$
A = AR1-8	50	1.678 $\pm .081$	1.709 $\pm .155$	49	1.556 $\pm .058$	.968 $\pm .032$	49	1.556 $\pm .058$	.937 $\pm .033$	53	1.207 $\pm .024$	1.219 $\pm .022$
B = AR1-4	50	1.511 $\pm .070$	1.531 $\pm .071$	49	1.769 $\pm .173$	1.028 $\pm .028$	49	1.769 $\pm .173$	1.037 $\pm .030$	50	1.199 $\pm .022$	1.203 $\pm .024$
C = AR2	48	1.446 $\pm .058$	1.462 $\pm .061$	48	1.570 $\pm .062$	.987 $\pm .024$	48	1.570 $\pm .062$	.992 $\pm .030$	54	1.225 $\pm .026$	1.245 $\pm .026$

uniform in all tests for all three populations. It is interesting to note that the correlation between the $+/+$ and $L/+$ viabilities exists whether the wild-type chromosome in the $L/+$ genotype is AR (homokaryotypic crosses) or CH (heterokaryotypic crosses). The reason for this $L/+$ and $+/+$ correlation seems unknown, although from other observations (unpublished) the relative viabilities of $L/+$ and $Bl\ Sc/+$ would seem to follow from the fact that the $Bl\ Sc$ marker is generally less viable than the L marker. The uniform average response of the $Bl\ Sc/+$ class in all experiments, and the fact that the $+$ chromosome in this genotype is always AR, can be used to indicate comparability of experimental conditions between the different tests. In a similar four-class segregation experiment in *D. melanogaster*, WALLACE (1958) used the uniform viabilities of his $Pm/+$ class for the same purpose.

The significant fall in AR/CH heterokaryotype viabilities compared to homokaryotypes seemed puzzling, and an experiment was undertaken to test the viability of the CH chromosome used. Flies from the *isogenic* CH stock were mated to the $L/Bl\ Sc$ marker stock, and crosses were then made ($CH/L \times CH/Bl\ Sc$) to produce four classes of offspring. In all, 10 such crosses were made with 6 pairs of parents in each, and each cross was replicated in successive culture bottles four times. Among the 13,495 progeny scored, the relative viabilities ($L/Bl\ Sc = 1.000$) were: $CH/CH = 1.198 \pm .075$, $L/CH = 1.039 \pm .048$, $Bl\ Sc/CH = 1.207 \pm .009$. Since the final crosses were made reciprocally, the results could be further partitioned and showed even lower viabilities of CH/CH progeny ($1.095 \pm .070$) when the mothers were $CH/Bl\ Sc$; that is, when the mothers had the same genotype as the mothers in the heterokaryotypic crosses. These findings may therefore help explain the relatively low AR/CH viabilities as a depressant effect of the CH chromosome, and perhaps an even further depressant maternal effect. It is also of interest that in both reciprocal lines of these crosses the $+/+$ and $Bl\ Sc/+$ viabilities now appear correlated while the $L/+$ viabilities occupy the approximate 1.000 viability position formerly occupied by the $Bl\ Sc/+$ class. Since the $L/+$ class represents the same genotype (L/CH) in both the CH/CH and heterokaryotypic crosses, the change in relative viability must arise from interaction between segregating genotypes: in the heterokaryotypic crosses AR is also segregating, whereas in the cross described above, only CH is segregating.

To identify the presence of chromosomal heterogeneity, analyses of variance were performed on the $+/+$ viability data, and the results are given in Table 2. Although the variances for homozygous viabilities increase in the progression from C to B to A, chromosomal heterogeneity as a cause for viability differences is only significant in populations A and C. In population A, the between-chromosome variance component is much higher in homozygous than in heterozygous combinations signifying a more uniform response among heterozygotes, perhaps along with an improvement in their "buffering" qualities as evidenced by the reduction in within-chromosome variance. This effect of heterozygosis on decreasing variances in the A population seems further reflected in all populations by the reduction of variance among the heterokaryotypes. Heterokaryotypes are, after all, the most heterozygous $+/+$ genotypes in the experiment. Nevertheless,

TABLE 2

Summary of analyses of variance of +/+ viabilities†

Population		Within chromosomes		Among chromosomes		F
		df	Mean sq.	df	Mean sq.	
A = AR1-8	Homozygotes	130	.9917	49	2.0327	2.0497***
	Heterozygotes	125	.3569	48	.6033	1.6901*
	Heterokaryotypes	157	.4520	52	.4225	.9347
B = AR1-4	Homozygotes	124	.9802	49	1.1821	1.2059
	Heterozygotes	128	.8991	48	1.2723	1.4151
	Heterokaryotypes	146	.1306	49	.1127	.8628
C = AR2	Homozygotes	132	.4365	47	.6517	1.4929*
	Heterozygotes	122	.4295	47	.6829	1.5900*
	Heterokaryotypes	156	.2144	53	.3419	1.5944*

* Significant at .05 level. *** Significant at .001 level.

† In a few cultures, few or none *L/Bl Sc* flies appeared, and such cultures were not included in the analysis of variance.

it should be kept in mind that only a single CH chromosome was used in all of the heterokaryotype tests, and may be partially responsible for the uniformity of results. At the same time, however, the reduced variances of heterokaryotypes compared to homozygotes in all three populations signifies that in spite of the fact that only a single AR chromosome appears in the +/+ class of each cross under both conditions, heterokaryotypic viabilities of different AR chromosomes are probably more alike.

TABLE 3

Number of flies counted in cultures of all tests

	Numbers of flies	Cultures		
		Number	$\bar{x}$	S.E.
Homozygotes				
A = AR1-8	41,818	187	223.62	7.28
B = AR1-4	36,441	177	205.88	8.02
C = AR2	41,811	181	231.00	7.10
Total	120,070	545	220.31	4.33
Heterozygotes				
A	39,027	178	219.25	6.44
B	40,776	186	219.23	7.37
C	38,385	176	218.10	7.19
Total	118,188	540	218.87	4.05
Heterokaryotypes				
A	58,525	210	279.69	8.44
B	55,826	193	289.25	8.47
C	53,886	211	255.39	7.92
Total	168,237	614	274.00	4.81

TABLE 4

Correlations (r), regression coefficients (b), and summary of analyses of variance of regressions of heterozygotes on homozygotes in the +/+ classes

Population	r	b	Error		Regression		F
			df	Mean sq.	df	Mean sq.	
A = AR1-8	.1339	.2162	47	.1716	1	.1473	.8584
B = AR1-4	.1195	.1538	46	.2063	1	.1523	.7382
C = AR2	.4947***	.7003***	44	.1374	1	2.0483	14.9075***

*** Significant at .001 level.

It is also of interest that the heterokaryotypes produced significantly greater ($P \ll .001$) mean numbers of flies per culture than either homozygotes or heterozygotes. As shown in Table 3, this significant increase occurs for all three populations, although to a somewhat greater degree for populations A and B than for population C. Since the heterokaryotypic tests were performed after the homozygous and heterozygous tests were completed, it is possible that a change in experimental conditions is responsible. On the other hand, comparability of the *Bl Sc*/+ viabilities between all of the tests may indicate a high degree of uniformity of experimental conditions.

Evaluation of dominance effects on viability was undertaken by estimating correlations between homozygous and heterozygous viabilities, where the former is the arithmetic mean of the two homozygous parental AR chromosomes. These results are given in Table 4, and show significant correlation and regression coefficients for only the C population, whose regression slope is also significantly different ($P < .05$) than that of either A or B. This can be interpreted to mean that only in the C population can we detect dominance for viability genes, and the degree of dominance can be calculated using the relationship given by MUKAI and YAMAZAKI (1968, formula 3):

$$\bar{h} = \frac{\text{cov (homozygote + heterozygote viabilities)}}{2\sigma_G^2}$$

which, in turn, is equal to half the regression slope since $b = \frac{\text{cov}}{\sigma^2}$. For population C, $\bar{h}$ is therefore .35.

Since each chromosome was tested in two heterozygous combinations, detection of dominance was also attempted by calculating the correlations between the two heterozygous viabilities for each chromosome. In this case, however, we would not expect as high a correlation as between a homozygote and heterozygote, since two different heterozygous combinations of a particular chromosome involves modifying effects of different chromosomes on each combination. The results, shown in Table 5, support this view, yet indicate again that C population chromosomes show relatively greater (although not now significant) dominance.

DISCUSSION

The present experiments enable comparisons to be made between viability

TABLE 5

Correlations, regression coefficients, and analyses of variance for regressions of viabilities of a chromosome in two heterozygous combinations

Population	<i>r</i>	<i>b</i>	Error		Regression		F
			df	Mean sq.	df	Mean sq.	
A = AR1-8	.0602	.0778	46	.1764	1	.0295	.1672
B = AR1-4	.0070	.0070	45	.2134	1	.0005	.0234
C = AR2	.2220	.2585	44	.1765	1	.4023	2.2793

studies that measure the genetic loads of AR third chromosomes and their relative fitnesses in population cage competition with CH. The relationships between the two sets of data, however, are certainly not simple. First, the heterozygous AR combinations, which we might suspect are important genotypes in establishing AR superiority in population cages, show no correlation between their ranking in viability tests and their ranking in competitive fitness tests ($A > B > C$, STRICKBERGER 1969). In the case of the A population, heterozygotes show a small (although not significant) reduction in viability compared to homozygotes. Second, the AR/CH heterokaryotype viabilities are uniformly lower than either the AR homozygotes or heterozygotes, yet in the population cages the AR/CH genotypes are almost universally heterotic. Only one example is known to the author where *complete* fixation for one of these two arrangements has taken place. (Unpublished results: This involved cage competition between AR chromosomes that had existed in a "monomorphic", i.e. homokaryotypic, population cage for two years *versus* CH chromosomes that had never been exposed to such conditions until the competition began—see Cage III in STRICKBERGER 1963.)

There are, of course, good reasons why one might not expect the results of viability and competitive tests to coincide: the marker chromosomes used undoubtedly have some effect on relative third-chromosome viabilities; the viability tests are only for a single generation of egg-to-adult development; only a single wild-type genotype is tested for viability within a culture whereas competitive fitness tests involve simultaneous interaction between AR/AR, AR/CH and CH/CH combinations; third chromosome karyotypes are restricted to one particular set of frequencies in viability tests but are not so restricted in competitive fitness tests; the viability tests are done in bottles, while the competitive tests are done in cages; fertility differences between genotypes are undoubtedly important in cages but are not tested in bottles; only one CH chromosome was used in the present viability tests; etc. These factors truly impose an "uncertainty" on extrapolating results from one experiment to the other.

Nevertheless, in spite of differences in experimental conditions between competitive tests and viability tests, some of the present results, used cautiously, may shed some light on some of the changes that have taken place between the three AR populations. First, it is of interest that genetic loads measured as chromosomes deleterious in homozygous condition show that improvement in homozygous viability of chromosomes is in the same ranking order ($A > B > C$) as their relative

competitive fitnesses in population cages (although only the A-C comparison shows a viability difference at the 5% level of significance.)

Second, there is a remarkable uniformity in viability for the third chromosomes from all of these populations in combination with the CH chromosome. This may signify that although marked differences in viability (see analyses of variances for homozygotes in Table 2) as well as in competitive fitness exist between these chromosomes, they produce more uniform viability responses when made heterozygous with a completely different gene arrangement. However, the cause for this may not be simply heterozygosity *per se* since both the AR and CH chromosomes involved have been selected, i.e., "coadapted", for the same geographic locality (DOBZHANSKY and PAVLOVSKY 1953; ANDERSON 1968). If the uniformity of heterokaryotype viabilities also exists in population cages, differences in fitness between AR chromosomes in such competitions may arise primarily from differences between them in homokaryotypic fitness rather than in heterokaryotypic fitnesses. Supporting evidence for this is the fact that AR competitive fitnesses can be significantly improved by allowing homokaryotypic selection to act on factors such as differences in the opportunity for recombination (STRICKBERGER 1965) and on differences in initial genetic variability (STRICKBERGER 1969).

The fact that the mean numbers of flies produced in the heterokaryotypic cultures are significantly greater than the mean numbers produced in the homozygous and heterozygous crosses also indicates a facilitative effect caused by the presence of heterokaryotypes (see also BEARDMORE, DOBZHANSKY and PAVLOVSKY 1960, and others). It is interesting to note, however, that this effect is not accompanied by the superior viability of the heterokaryotypes compared to the relative viabilities of the homozygous and heterozygous homokaryotypes. As in other instances (DAWOOD and STRICKBERGER 1969), facilitation can apparently be caused by genotypes whose relative viabilities may not necessarily be superior.

A further point of interest relates to whether these populations follow a "mutational" or "segregational" model, i.e., selection respectively for improved homozygotes or heterozygotes. As discussed previously, the data in Table 1 would, on first appearance, indicate that improvement of AR homozygous viability is occurring along with an increase in the relative fitness of AR chromosomes. However, chromosomes following a mutational model should also show correlation between the viabilities of homozygotes and heterozygotes, i.e., dominance. In the present experiments, this correlation between genotypes is highly significant for the least "fit" population (C) but not for the others showing greater fitness. In C, the degree of dominance observed through regression analysis is $\bar{h} = .35$. This value accords well with that of MUKAI and YAMAZAKI (1968) for newly arisen viability polygenes ($\bar{h} = .40$), and with that observed by WILLS (1966) for 17 nonlethal chromosomes from one of his natural populations ($\bar{h} = .30$).

The overall picture presented by these data is therefore a change from low competitive fitness, relatively low homozygous viability, and partial dominance in population C, to improved competitive fitness, increased homozygous viability, and lack of discernible dominance in A and B. What could account for these

changes? A possible explanation is simply the difference in initial variability between the three populations, since each was founded with a different number of AR chromosome strains. That is, an isogenic AR strain (population C) may after a period of time produce variability among its third chromosomes, but this variability is neither sufficient nor of the appropriate type to improve the competitive fitness of these chromosomes (STRICKBERGER 1969). Irradiated populations of single isogenic AR chromosomes have also shown such lack of improvement (ibid.). If, as explained above, competitive improvement of AR chromosomes is associated with improved homozygous viabilities, then the lack of such improvement in C may therefore arise from the lack of appropriate genetic variability.

Continuing further: What is the connection between genetic variability in these populations and dominance? It is, of course, possible that the C population bears only one dominant AR chromosome, and that some of the other AR chromosomes introduced into populations A and B are overdominant. Although only one such single AR chromosome population was tested in the present experiment, the uniform competitive response of different single AR chromosome populations (ibid.) makes it likely that different single AR strains may be somewhat alike in respect to homozygous viability and dominance, and it is rather the opportunity for recombination between these strains which enables competitive fitness, viability, and dominance to change. In other words, if linkage disequilibrium can be generated through selection on genetic variability so that dominance relationships change, then some of the observed results may be explained.

Many genes are undoubtedly selected for their epistatic effects on closely linked genes, and theoretical justifications for selection for linkage disequilibria have been presented by various authors (see KOJIMA and LEWONTIN 1970). WILLS, CRENSHAW and VITALE (1970) using computerized populations, have shown the establishment of linked groups containing both fixed and polymorphic alleles. The fact that recombination can modify the viability of natural chromosomes is not a novel idea. Chromosomes "normal" in viability in homozygous conditions, when allowed to recombine with each other, have yielded products which when tested again in homozygous condition, now show a complete range of viability from lethality and sterility, to supervitality (see for example, SPIESS 1959, KRIMBAS 1960). WALLACE and VETUKHIV (1955) have demonstrated that the heterotic activity of particular chromosome combinations can be reduced by recombination between them yet maintain a similar amount of heterozygosity. Also, the likelihood that recombination alone can lead to changes in dominance can be garnered from the results of MUKAI and YAMAZAKI (1968) for second chromosomes in *D. melanogaster*; when mutant polygenes are heterozygous in the coupling phase (linked together opposite a wild-type homologue) they show overdominance ($\bar{h} \cong -.27$), when heterozygous in the repulsion phase (on both homologues) they show partial dominance ($\bar{h} \cong .4$). If a similar mechanism can be considered to operate in the present experiment, the loss of dominance that appears to accompany the improvement in fitness may perhaps signify an increase in coupling relationships between genes.

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