Viability interactions between the left and right arms of the second chromosome of *Drosophila melanogaster* .

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ABSTRACT

Inter and intrachromosomal viability interactions have been detected in a few experimental studies. Computer simulations and analytical models have led to postulation of nonadditivity of gene action. This study reports evidence of strong nonadditive interactions between the arms of the metacentric second chromosome of Drosophila melanogaster. Mean viability for 40 homozygous lines of the second chromosomes was 0.720 ± 0.265 . Mean viability for 40 half homozygous second chromosomes was 0.928 ± 0.310 . Significant heterogeneity among and within lines was found in both groups of chromosomes, as well as a highly significant viability difference between the two groups. Comparison of observed viabilities with the expected values, according to the theories of additive and multiplicative gene action, was made for both groups. Highly significant departures from the expected values were found for over 90% of the lines in both groups of chromosomes, for both additive and multiplicative models of gene action.

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I am grateful to B. Dempsey, S. Espinet, B. Giles, and S. Nemeth for valuable help in preparing Drosophila medium and for many useful discussions. Dr. J.Barrett and Dr.MacKay helped with statistical analysis.

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INTRODUCTION
AND
LITERATURE SURVEY

Lewontin (1963) made the following statement :

> By the end of 1932 Haldane, Fisher, and Wright had said everything of truly fundamental importance about the theory of genetic change in populations... There remains for us, the epigonai, to reintroduce bit by bit the complexities of nature, to see to what extent the complexities really make a difference, whether qualitative or quantitative, in our basic formulations.

But later, in a paper reporting the results of multilocus computer simulations, he states (Franklin and Lewontin, 1970):

> The discovery that, when more than a couple of dozen genes are involved in linkage, the gene number is irrelevant has far-reaching implications for the theory of population genetics. While we commonly think of population genetics as the best example of the successful application of mathematical theory in biology, much of our confidence is unjustified. There is a striking discrepancy between the structure of genetic theory and the observations of experiment and natural history.

These two comments clearly show how little we know about the genetical changes involved in evolution and what happens to our mathematical theories when we re-introduce "the

complexities of nature".

Most mathematical formulations of population genetics treat single locus models from an allele frequency change or equilibrium point of view. Such models attempt to predict fitness effects of allele substitution at a locus. To arrive at theoretical conclusions relevant to the multilocus situation single locus effects are simply added together. Experimental testing of the predictions of single locus fitness theory is difficult because most observational and experimental work is, of necessity, done with whole phenomes where fitness depends on environmental conditions, and genomes in their totality. Even if we could measure the effects on fitness of single loci after randomization of the genetic background, the conclusions drawn might be misleading because of the experimental modifications of the background introduced in such studies. Single locus lethals are, of course, excluded from this consideration.

Mather (1973) has suggested that more attention must be paid to the genetic structure of populations where the effects of linkage and epistasis may play a major role. On the same theme Dobzhansky (1955) wrote :

> The linear seriation of the constituent genes in a chromosome is not fortuitous; a chromosome is an organized unit, the functioning of which depends on the spatial distribution of its parts. The

> linkage relationships of alleles in a multiple heterozygote are also not fortuitous: the developmental effects of genes may be different in the coupling and in the repulsion phase.

Working with chromosomal inversions, rearrangements, and recombinations, Dobzhansky became convinced of the existence of strong interactions among genes. He accepted the idea of a supergene, and he found it nothing short of amazing that the problem of interlocus interactions received so little attention. This lack of theoretical attention was obviously caused by the mathematical difficulties involved in formulating a theory adequate to frame the results of observations in terms used by experimental population geneticists, that is, in terms of chromosome segments, their map length, and their fitness effects per unit map length. In addition problems of experimental design and fitness measurement have to date precluded estimation of locus by locus interactions. Most theoreticians express a strong wish that experimental workers collect "more information about an essential parameter of the genetic system, namely the effect on fitness of homozygosity for a segment of chromosome" (Lewontin, 1974).

Very few experimental studies have been designed to detect the presence of interactions. The results of these few studies are to a certain extent in disagreement.

Temin, Meyer, Dawson, and Crow (1969) and Spassky, Dobzhansky, and Anderson (1965) attempted to assess the importance ofepistasis, linkage effects, and the deleterious effects of homozygosity in Drosophila melanogaster and in Drosophila pseudoobscura respectively. Both groups worked with whole second and third chromosomes where either one or both chromosomes were made homozygous by similar techniques as described below. Spassky et al (1965) found significant interactions between and within chromosomes in quasinormal cultures of Drosophila pseudoobscura. Quasinormal cultures are those containing expected or near to the expected ratio of flies homozygous for a given chromosome and heterozygous flies. Lethals and subvitals are not considered because of the strong effects these alleles exert by themselves. The lethals do not interact by definition, and severely detrimental genes are rare in the population, being eliminated by their primary effects on the individual homozygotes (Temin et al., 1969). On the other hand, the interaction value for the more common mildly detrimental genes, which are present in the quasinormal class of the synthetic homozygotes, might be an important factor in the action of selection against these genes. This is the reason why the interaction studies center on the quasinormal group of chromosomes.

As mentioned above, Spassky et al. (1965) found a significant positive interaction component both between and within the second and third chromosomes in Drosophila pseudoobscura quasinormal cultures. On the other hand , Temin et al. (1969) found that there is only very slight positive or reinforcing epistasis within and between the second and third chromosome in the quasinormal class of Drosophila melanogaster. This epistasis was not statistically significant. In addition, this study suggests that the distribution of mildly deleterious genes in the quasinormal class of flies is uniform on each given chromosome, rather than there being large interactions of opposite direction which effectively cancel each other out, leaving a small net positive epistasis.

Apart from these two studies, which attempted to measure directly the amount of interaction between chromosomes, there are only a few considerations of inbreeding depression and interaction. Levene (1965) found some evidence of positive epistasis in Tribolium, by looking at the inbreeding effect at the various levels of homozygosity (coefficient of inbreeding F = 0, 1/8, 1/4). The nonlinearity of the inbreeding effect (Figure 1) indicated positive epistasis.

Kidwell, Tracey, Glaser, and Kidwell (1971), using

a biometrical approach, analyzed x-ray induced genetic variance of wing length in Drosophila melanogaster. They found that there is a very large component of genetic variance attributable to 2 and 3 factor interactions. The epistasis was especially strong between the sex chromosome and the autosomes.

Mukai (1968) observed positive interaction between spontaneous mutant polygenes. He based this conclusion on the nonlinearity between generation number and average viability of second chromosomes which accumulated these mutants.

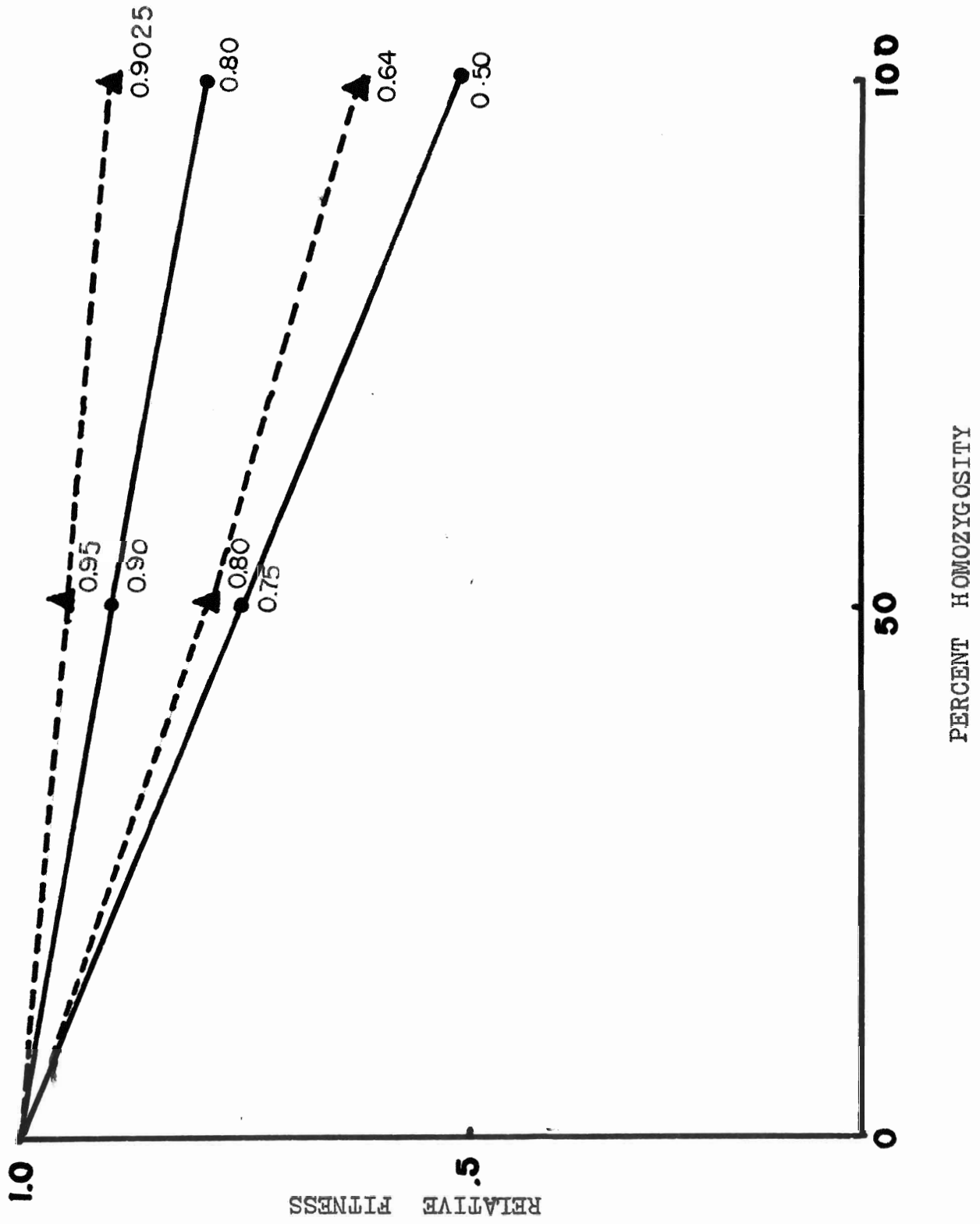
This study was designed to assess the fitness effects of homozygosity of one arm or of the entire second chromosome in Drosophila melanogaster. Under the multiplicative model of fitness interactions, as well as under the additive model, the decrease in fitness of the one arm homozygous flies should be such that average fitness reaches the point midway between the fitness of the flies totally homozygous for the whole second chromosome and the fitness of the original wild population (Figure 1). Departure from this midpoint would indicate that there are epistatic interactions either between the two arms of the second chromosome (which are nearly equal in length) or with the rest of the genome. The experimental demonstration of such inter-

actions might give experimental support and direction to theoretical studies of chromosome organization and locus by locus fitness interaction.

FIGURE 1

Expected Relative Fitness of Whole and Half Homozygous Chromosomes. The circles and solid lines represent the expected fitness declines on increasing homozygosity under an additive model for two chromosomes. The triangles and dashed lines represent the expected fitness declines on increasing homozygosity under a multiplicative model for two very similar chromosomes. That is the expected half chromosome fitnesses, under additivity are equal to $(1 - \frac{w}{2})$ where w is the relative fitness depression. The equivalent values under the multiplicative model are $(1 - w)^{\frac{1}{2}}$.

Note that the multiplicative model is not linear although it appears to be in the figure.


1.0
.5
0
50
100
RELATIVE FITNESS
PERCENT HOMOZYGOSITY
0.9025
0.80
0.64
0.50
0.95
0.90
0.80
0.75

MATERIALS AND METHODS

Stocks: *Drosophila melanogaster* were collected over buckets of fermenting bananas at Professor Peter Rand's Farm, Line 2 - Concession 2, Niagara-on-the-Lake, Ontario, during the evening of July 11, 1975. Two techniques were used to extract wild homozygous second chromosomes. These techniques are diagrammed in Figures 2,3, and 4. Balancer stocks used in the extractions are described in Table 1.

Extraction of Chromosomes:

In the first extraction procedure (Figure 2) wild females were individually crossed to G 23 balancer stock males. Thus each line was derived from only one wild fly, ie. one original chromosome. The balancer chromosome, which was present in the stock males, inhibits crossing over in the second chromosome because it contains a number of overlapping inversions with built in lethals. In homozygous state the balancer is therefore lethal. The F_3 generation flies are of two kinds : those heterozygous for the balancer and the wild chromosome and those which are homozygous for the same wild chromosome which is present in the heterozygous flies. The ratio of one homozygous to two heterozygous flies is expected because the flies homozygous for the balancer chromosome die as eggs, larvae or pupae.

FIGURE 2

Derivation of homozygotes for wild chromosomes II of *Drosophila melanogaster*. *Cy* represents a marker chromosome which suppresses recombination. The subscript i refers to a wild chromosome.

P Freshly collected wild ♀♀ x Cy G 23

F_1 Cy/II_i♀ x Cy G 23 (from stock)

F_2 Cy/II_i x Cy/II_i

F_3 Cy/Cy Cy/II_i II_i/II_i

Expected ratio 1 (dies): 2 : 1

FIGURE 3

Derivation of homozygotes for the left arm of the second chromosome of Drosophila melanogaster .

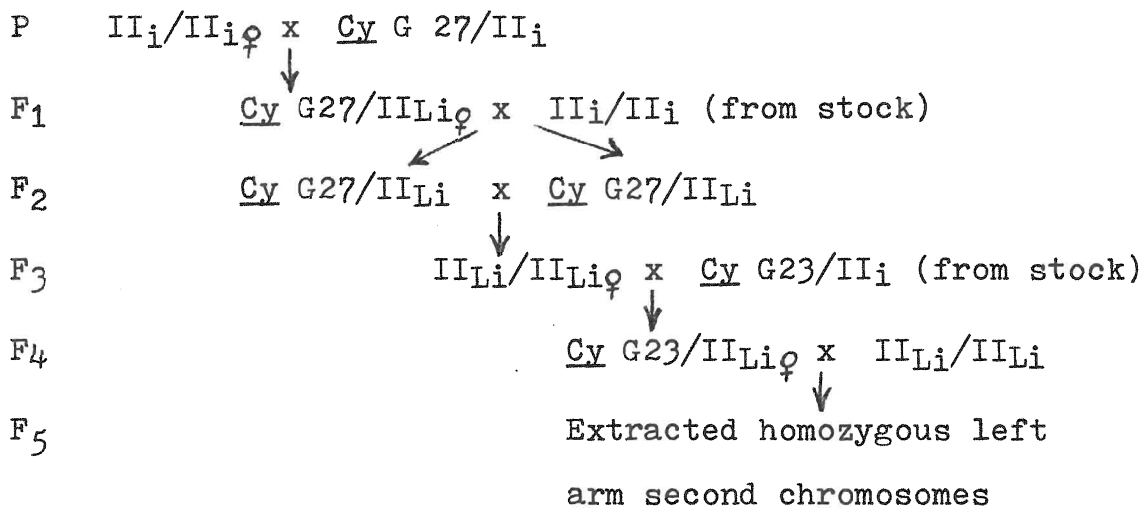

P
II_i/II_i♀ x Cy G 27/II_i
F_1
Cy G27/II_{Li}♀ x II_i/II_i (from stock)
F_2
Cy G27/II_{Li} x Cy G27/II_{Li}
F_3
II_{Li}/II_{Li}♀ x Cy G23/II_i (from stock)
F_4
Cy G23/II_{Li}♀ x II_{Li}/II_{Li}
F_5
Extracted homozygous left
arm second chromosomes

FIGURE 4

Derivation of both whole homozygous second chromosomes and left arm homozygous second chromosomes of Drosophila melanogaster by a combined method.

Cy G27 and Cy SM5 represent marker chromo - somes which suppress recombination in the left arm or in the whole chromosome, re - spectively. The subscripts i and j refer to wild chromosomes.

P	Cy G27/II_{Li}♀	x	Cy SM5/Pm	i = 1,4
F_1	Cy SM5/II_{Lij}♀	x	Cy SM5/Pm	i,j = 1,4; i = j
F_2	Cy SM5/II_{Lij}	x	Cy SM5/II_{Lij}	i,j = 1,4; i = j
F_3	Cy SM5/II_{LiRj}	x	Cy SM5/II_{LiRj}	i,j = 1,4; i = j and i,j = 1,4; i ≠ j

F_4 Cy SM5/II_{LiRj} : II_{LiRj}/II_{LiRj}

2 Curly : 1 wild

TABLE 1

The summary and sources of the stock *Drosophila melanogaster* flies .

Code name	Composition	Source
G 23	al,S,ast,ho/SM1,al^2,Cy,sp^2	I.Oster,Bowling Green
G 27	al^2,Cy,InL,lt^3/b,pr,Bl,lt^3 cn^2,In Cy R,L^4,sp^2	I.Oster, Bowling Green
SM5	BL,L^2/SM5,al^2,Cy,lt^2,sp^2	M.M. Green, University of California, Davis

Explanation of symbols (from Lindsley and Grell, 1972) :

L^4 (Lobe) , S (Star), ast (asteroid), lt^3 (light), pr (purple), and cn (cinnabar) are eye colour and/or shape mutants.

al (aristaless) missing or diminished aristae

sp (speck), and b (black) are body colour mutants.

Bl (Bristle) shortened bristles

ho (heldout), and Cy (Curly) are wing mutants

In L and In R inversion Left and Right (arms of the second chromosome)

SM Second Multiple (multiple inversions on the second chromosome).

TABLE 2

Drosophila medium components

Tegosept	80 ml
Molasses	1350 g
Water	8600 ml
Salt	70 g
Cream of wheat	1030 g
	500 vials

Size of vial : approximately 3.0 cm diameter x 10cm height; filled to approximately 3cm height.

The heterozygous flies were recognized by the presence of the dominant mutation Curly wing (Cy), which was built into the balancer chromosome.

The flies containing the homozygous left arm of the second chromosome were prepared (Figure 3) by crossing flies homozygous for the whole second chromosome, prepared as described above, to the G 27 stock flies. The G 27 stock flies with the Cy marker in the left arm of the second chromosome had been crossed to Randy flies in mass cultures for twelve generations to ensure the randomization of the right arm. Because these stock flies contained a balancer which was different from the balancer used to prepare the whole second chromosome homozygous flies, and because flies containing the same arm portion of the chromosome were needed for a meaningful comparison of viabilities between whole and half homozygous chromosomes, the original balancer chromosome G 23 was reintroduced in the F_3 generation (Figure 3). By this method flies were prepared which in the F_5 generation contained randomized right arms derived from the Randy wild chromosomes and homozygous left arms identical to those of the fully homozygous second chromosomes.

Unfortunately, this method did not produce consistent results and upon checking it was found that the balancer chromosome which was used either did not effectively

suppress recombination in the heterozygous flies or that the Cy mutant had been lost. In the first case portions of the balancer chromosome not containing the Curly marker, may have been introduced to the wild chromosome by crossing over. This would produce an abnormally high frequency of wild, apparently supervital, flies. The balancer chromo - some was prepared by H.J. Muller in or around 1948 and this is, as far as we know, the first reported instance of its failure to prevent recombination, or of Cy loss through back mutation.

A new extraction method was therefore employed (Figure 4) to resynthesize the required chromosomes. This consisted of crossing the flies with the left arm balancer and right arm wild chromosome to a new balancer (SM5; Figure 4) kindly supplied by Professor M.M. Green, University of California, Davis. Four sublines were established, using four male sibs from the parental line, and crossing them to the SM5 balancer. The F_3 generation thus consisted of four sublines each carrying the balancer chromosome and ex - tracted chromosomes with identical left arms and hetero- geneous though homozygous right arms. When one male and one female of the same subline were crossed, a progeny with entirely homozygous second chromosome resulted (the expected ratio was again 2 heterozygous flies to one homozygous fly),

but when a female of one subline was crossed to a male of a different subline the left arm homozygous flies resulted, together with heterozygous flies, as above.

Any deviations from the expected 1 homozygous : 2 heterozygous flies ratio are due to the viability differences, since no meiotic drive is known to occur in the heterozygous flies (Tracey and Ayala, 1974) and no evidence of meiotic drive was observed in these experiments. The flies were raised in vials on standard wheat hearts - molasses medium (Table 2) and kept in incubators at 25± 1°C.

RESULTS

Distribution of Viabilities . Figures 5 and 6 show the distribution of viabilities for whole and half homozygous chromosomes. Both distributions are roughly bimodal, with one mode representing the lethal chromosomes and the other the "quasinormal" chromosomes. The latter chromosomes include the mild detrimentals as well as super vital homozygous chromosomes. This bimodality is usually observed in studies of viability (see for example Tracey and Ayala, 1974; Dobzhansky and Spassky, 1963). Among the 53 whole second chromosomes, 9 were lethal and 2 were severely detrimental; among the 50 half homozygous chromosomes, 6 were lethals and 1 severely detrimental.

Viabilities (2a/b) of whole and half homozygous chromosomes, relative to the SM5 heterozygotes, are presented in Tables 3 and 4. Haldane's (1956) formula was used to estimate viability. The correction for small sample sizes has the following simple form :

$$V = \frac{2a}{b + 1}$$

a = number of homozygotes
b = number of heterozygotes

FIGURE 5

Distribution of viabilities of whole second chromosomes of Drosophila melanogaster .

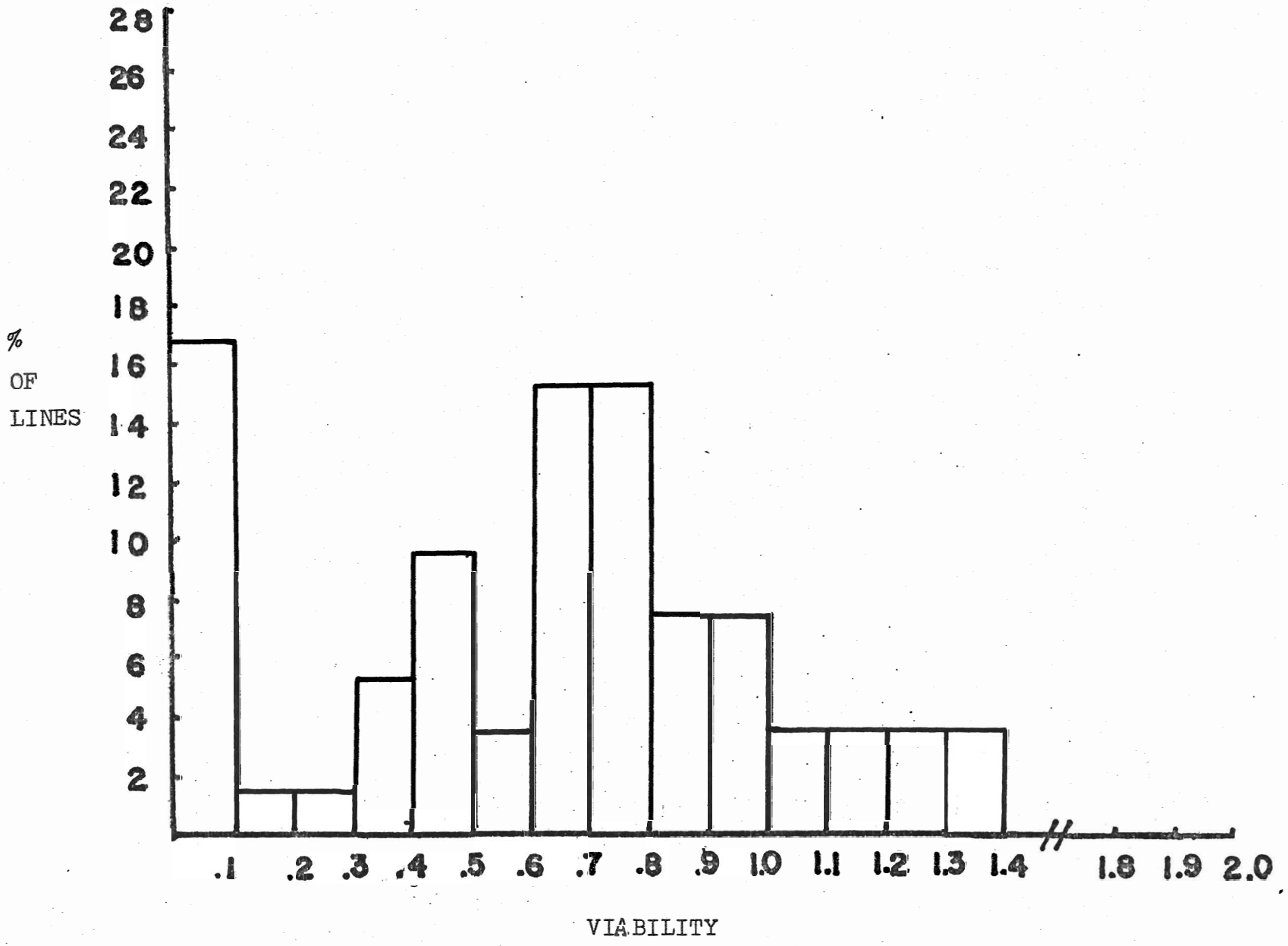

28
26
24
22
20
18
16
14
12
10
8
6
4
2
% OF LINES
.1 .2 .3 .4 .5 .6 .7 .8 .9 1.0 1.1 1.2 1.3 1.4 1.8 1.9 2.0
VIABILITY

FIGURE 6

Distribution of viabilities of half homo - zygous second chromosomes of *Drosophila melanogaster* .

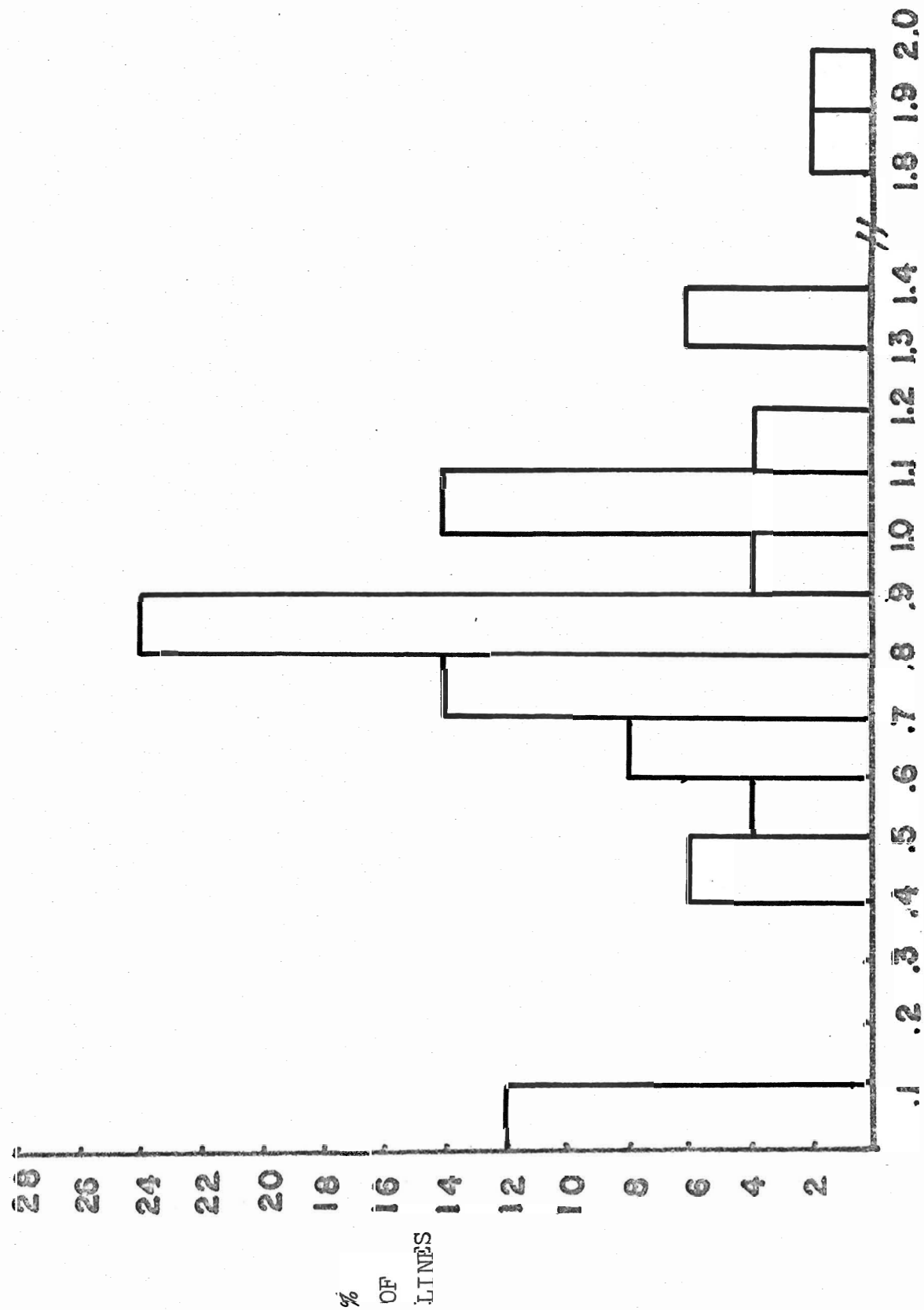

% OF LINES
25
26
24
22
20
18
16
14
12
10
8
6
4
2
.1
.2
.3
.4
.5
.6
.7
.8
.9
1.0
1.1
1.2
1.3
1.4
1.8
1.9
2.0
VIABILITY

TABLE 3

Viabilities of homozygous second chromosomes of Drosophila melanogaster relative to SM5 //+ heterozygotes. The combined brood viabilities were computed by summing phenotypes over broods (see Appendix I for data).

Line	Brood 1 Viability	Brood 2 Viability	Brood 1 + 2 Viability
1	.420±.130(2)	.106± (1)	.375±.175(2)
2	.452±.171(3)	.906 (1)	.448±.233(3)
3	.863±.184(4)	1.025±.189(4)	.937±.153(4)
4	.626±.296(4)	.970±.374(3)	.726±.276(4)
5	.337±.010(3)	1.420±.380(2)	.612±.232(3)
6	.575±.193(4)	.859±.136(3)	.726±.248(4)
7	1.253±.589(4)	1.046±.470(2)	1.327±.565(4)
8	1.693±.761(2)	.959±.117(2)	1.389±.361(2)
9	.504±.140(2)	1.098 (1)	.613±.249(2)
10	.718±.214(3)	.656±.207(3)	.727±.112(3)
11	.716±.039(3)	.830 (1)	.739±.043(3)
12	.766±.408(4)	----------	.473±.412(4)
13	.986±.272(3)	.932±.156(3)	.931±.196(3)
14	.507±.065(3)	.898±.069(3)	.638±.057(3)
15	.424±.032(2)	.316±.128(2)	.388±.076(2)
16	.811±.216(3)	.743±.081(3)	.781±.062(3)
17	.608±.404(4)	.850 (1)	.681±.338(4)
18	.880±.155(4)	1.210±.394(4)	1.005±.220(4)
19	.747±.237(3)	.887±.243(3)	.608±.042(3)
20	1.053±.370(3)	1.118 (1)	1.100±.317(4)
21	.353±.297(2)	.360 (1)	.287±.231(2)
22	1.124±.304(2)	.589±.189(2)	.608±.034(2)
23	1.251±.205(3)	.771±.091(2)	1.116±.226(3)
24	.450±.134(2)	.446 (1)	.513±.197(2)
25	.759±.178(4)	.806±.138(2)	.805±.097(4)
26	1.380±.358(3)	1.046±.124(2)	1.227±.449(3)
27	.779±.267(2)	.697±.103(2)	.653±.117(2)
28	.948±.448(2)	----------	.948±.448(2)
29	.925±.101(3)	----------	.925±.101(3)
30	.516±.044(3)	.698±.198(2)	.551±.123(3)
31	.362±.126(2)	.412 (1)	.348±.112(2)
32	1.201±.707(3)	.867±.099(3)	1.013±.371(3)
33	.824±.676(2)	.700 (1)	.709±.561(2)
34	.736±.122(2)	.564±.256(2)	.713±.139(2)
35	.559±.030(2)	.324±.046(2)	.450±.020(2)
36	.673±.280(3)	.705±.077(3)	.724±.112(3)
37	1.242±.265(4)	.666 (1)	1.221±.263(4)
38	.470±.237(4)	.872±.016(2)	.467±.284(4)
39	.184±.148(2)	.224±.088(2)	.193±.039(2)
40	.947±.050(3)	.826±.061(3)	.884±.071(3)
TOTAL			.720±.265
41	1.043±.177(2)	.452 (1)	.816±.206(2)
42	.830±.174(2)	.880±.320(2)	.880±.244(2)
43	.613±.153(3)	.644 (1)	.621±.153(3)
44	.459±.109(2)	.332 (1)	.454±.104(2)

NOTE: Number of replicates is in brackets following standard deviations. Lines 41-44 are not included in statistical analysis, because half homozygous replicates were not run for these lines. Nine lethals are not included in this table.

TABLE 4

Viabilities of homozygous left arm second chromosomes of Drosophila melanogaster relative to SM5 //+ heterozygotes . The combined brood viabilities were computed by summing phenotypes over broods (see Appendix I for data).

Line	Brood 1 Viability	Brood 2 Viability	Brood 1 + 2 Viability
1	.454±.342(3)	.587±.221(3)	.519±.268(3)
2	.680±.333(3)	.817±.293(2)	.764±.169(3)
3	.674±.147(5)	.881±.261(5)	.746±.161(5)
4	1.173±.985(6)	1.013±.209(6)	1.038±.424(6)
5	.450±.255(3)	.734±.154(3)	.601±.162(3)
6	.672±.261(5)	1.217±.346(3)	.877±.156(5)
7	.850±.117(6)	.949±.120(3)	.879±.078(6)
8	1.264±.479(3)	1.759±.901(2)	1.345±.567(3)
9	1.204±.235(5)	.879±.033(4)	1.060±.227(6)
10	.997±.117(4)	1.100±. (1)	1.015±.099(4)
11	1.189±.275(6)	.789±.061(2)	1.070±.213(6)
12	.304±.173(3)	.779±.218(3)	.475±.175(3)
13	1.160±.481(3)	.821±.049(3)	.883±.103(3)
14	.799±.179(6)	.596±.364(3)	.751±.137(6)
15	.767±.446(6)	.920±.231(3)	.807±.451(6)
16	1.142±.412(3)	----------	1.142±.412(3)
17	.675±.174(5)	1.224±.355(3)	.789±.251(5)
18	1.053±.215(6)	1.383±.453(3)	1.083±.197(6)
19	.825±.327(5)	.894±.362(5)	.861±.350(5)
20	1.080±.206(4)	.862±.325(3)	1.056±.229(4)
21	.719±.262(3)	1.005±.318(3)	.843±.302(3)
22	1.464±.736(2)	1.416 (1)	1.909±.291(2)
23	.769±.103(2)	.937±.137(2)	.846±.122(2)
24	.691±.151(3)	.618±.152(2)	.687±.149(3)
25	1.056±.391(5)	.985±.319(2)	1.016±.336(5)
26	1.189±.277(6)	1.120±.010(2)	1.199±.270(6)
27	.444±.306(2)	1.332 (1)	.529±.391(2)
28	.921±.824(4)	.880 (1)	.835±.890(4)
29	1.901±1.493(3)	1.028 (1)	1.899±1.494(3)
30	.821±.155(3)	.858±.082(2)	.818±.132(3)
31	.815±.072(3)	.907±.407(3)	.891±.133(3)
32	1.009±.365(4)	1.022±.357(3)	1.308±.705(4)
33	1.156±.413(3)	1.720 (1)	1.300±.298(3)
34	.547±.195(3)	1.148±.252(2)	.615±.223(3)
35	.710±.248(2)	----------	.710±.248(2)
36	.982±.285(4)	----------	.982±.285(4)
37	.845±.189(3)	1.109±.557(2)	.817±.119(3)
38	1.025±.512(6)	.697±.235(5)	.886±.356(6)
39	.509±.300(3)	.278 (1)	.471±.297(3)
40	.887±.193(3)	.657±.009(2)	.799±.246(3)
TOTAL			.928±.310
45	.484±.121(3)	.204±.072(2)	.431±.150(3)
46	.780±.135(3)	1.000 (1)	.698±.122(3)
47	.460±.118(6)	.958±.524(5)	.907±.211(6)
48	.562±.215(2)	.812 (1)	1.124±.304(2)

NOTE: Number of replicates is in brackets following standard deviations. Lines 45-48 are not included in statistical analysis, because whole homozygous replicates were not run for these lines. Six lethals are not included in this table.

The formula is useful if less than 100 flies are counted per culture as it corrects for the bias introduced by the statistical occurence of homozygotes in small samples. The correction was applied throughout because it does not appreciably affect the viability ratio if the number of flies is large.

Statistical Analysis of the Results. One way analysis of variance was performed on the combined broods. The results are shown in Table 5. There is significant heterogeneity both among lines and within lines (among sublines). The differences among lines are expected because each line represents a different wild chromosome with different genic content. The heterogeneity within each line, while not unexpected, is more difficult to explain. It probably reflects the reduced buffering capacity of homozygous lines; such lines should exhibit more drastic reaction to micro-environment variation such as differential crowding. On the other hand, genetic differences among sublines are expected. The I, III and IV chromosomes were not controlled and recombination in IIR generates different right arms among sublines. Brood heterogeneity was not significant at the 5% level (Table 6); therefore the two broods were combined for subsequent analysis. Table 6 shows the paired t test analysis which compares the viability values of the whole and

TABLE 5

One way analysis of variance of viabilities of whole and half homozygous second chromosomes of Drosophila melanogaster .

Whole homozygous second chromosomes combined broods

Source	Sum of squares	Degrees of freedom	Mean square
Among groups	9.0252	39	0.2314
Within groups	7.8748	77	0.1023
Total	16.9001	116	

F**= 2.2628

Half homozygous second chromosomes combined brood

Source	Sum of squares	Degrees of freedom	Mean square
Among groups	11.2792	39	0.2892
Within groups	22.6447	118	0.1919
Total	33.9239	157	

F* = 1.5071

NOTES : * significant at 0.05 level
** significant at 0.005 level

TABLE 6

Paired t tests to determine brood differences and differences between viabilities of the whole and half homozygous second chromosomes of Drosophila melanogaster .

Test of significance of differences between viabilities of brood one and brood two of the whole homozygous second chromosome .

Number of lines		41	
t value	t =	0.0985	N.S.
Degrees of freedom	df =	40	

Test of significance of differences between viabilities of brood one and brood two of the half homozygous second chromosome .

Number of lines		41	
t value	t =	1.6339	N.S.
Degrees of freedom	df =	40	

Test of significance of differences between viabilities of combined broods of the whole and half homozygous second chromosome .

Number of lines		40
t valune	t** =	3.5700
Degrees of freedom	df =	39

NOTE : ** significant at 0.001 level

N.S. not significant

half homozygous chromosomes, using the combined brood estimates for each comparison. There is a highly significant viability difference between these two sets of chromo - somes.

Table 7 shows the Chi-square comparison of these results with the viability values predicted by the additive and multiplicative models of gene action. If, for example, the whole homozygous chromosome has a certain viability, $(1-v_i)$, then the same chromosome should, according to the additive model, have viability, $(1-\frac{v_i}{2})$, when it is only half homozygous. Under a multiplicative model the equivalent values are $(1-v_i)$ and $(1-v_i)^{\frac{1}{2}}$. Note that both models assume that viability depression is the result of the cumulative effects of many mildly deleterious alleles homogeneously distributed along the chromosome.

In the table the predicted values were compared with the observed viabilities. The observed viabilities were also compared with predictions of the multiplicative model of gene action; highly significant departures from the values predicted by both models were found over all lines.

Another method of comparing the observed and expected half chromosome viabilities is presented in Table 8. Using the observed whole chromosome viability, $(1-ns)$, where n is the number of viability depressing loci and s is the

TABLE 7

Chi-square test of fit of the observed viability data with the values predicted by the additive and multiplicative models of gene action.

** ($P < 0.01$) $\chi^2_{1df} = 6.63$

* ($P < 0.05$) $\chi^2_{1df} = 3.84$

Line	Chi - square value Additive	Multiplicative	
1	42.307**	27.59**	
2	0.72	0.047	
3	10.78 **	10.74**	
4	0.01	0.08	
5	7.68 **	6.48**	
6	0.32	0.19	
7	8.99 **	8.43**	
8	1.12	1.35	
9	7.11 **	9.40**	
10	0.36	0.44	
11	0.79	0.99	
12	14.95 **	11.63**	
13	1.76	1.75	
14	0.006	0.07	
15	4.88 *	10.98**	
16	1.61	1.75	
17	2.69	3.28	
18	0.097	0.097	
19	0.15	0.55	
20	1.22	1.20	
21	1.36	8.61**	
22	25.36 **	27.66**	
23	3.02	2.98	
24	1.02	0.43	
25	0.80	0.89	
26	0.01	0.02	
27	0.30	0.18	
28	0.03	0.03	
29	0.42	0.43	
30	33.14 **	29.25**	
31	4.83 *	10.88**	
32	0.25	0.25	
33	3.97 *	4.45*	
34	2.44	2.03	
35	6.90 **	9.40**	
36	1.78	2.08	
37	6.81 **	6.57*	
38	0.52	2.77	
39	2.28	0.33	
40	8.70 **	8.59**	
Overall Probability	0.005	0.005	80 df

TABLE 8

Observed and Expected Half Chromosome Via - bilities. The whole chromosome and half chromosome observed viabilities are presented for each line in columns two and three. The expected half chromosome viabilities and 95% confidence limits for relative single locus fitnesses (1-S) of 0.99, 0.95 and 0.90 are tabulated in columns four and five. The number of loci and the 95% confidence limits for these two models are presented in columns six and seven. The numbers in column eight are the number of subline viabilities falling within the viability confidence interval; the final column presents the number of replicate sublines within each line. Where whole chromosome viabilities were greater than 1.00 they were adjusted to 1.00 to allow comparison with the multiplicative model (values greater than 1.00 generate negative numbers).The BASIC program used to compute the table is presented in Appendix II.

Line	Observed Viability		Expected Viabilities		Number of Loci		Number of Sublines	Sublines
	Whole	Half	Additive	Multiplicative	Additive	Multiplicative	in 95% Interval	
1	0.38	0.52	±0.10 0.69±0.22 ±0.31	±0.08 0.61±0.20 ±0.30	31.2± 9.8 6.2± 4.4 3.1± 3.1	48.8±12.3 9.6± 5.4 4.7± 3.8	1	3
2	0.45	0.76	±0.09 0.72±0.21 ±0.29	±0.08 0.67±0.19 ±0.29	27.6± 9.2 5.5± 4.1 2.8± 2.9	39.9±11.1 7.8± 4.9 3.8± 3.4	2	3
3	0.94	0.75	±0.02 0.97±0.03 ±0.05	±0.02 0.97±0.04 ±0.05	3.2± 1.6 0.6± 0.7 0.3± 0.5	3.2± 1.6 0.6± 0.7 0.3± 0.5	0	5
4	0.73	1.00	±0.03 0.86±0.06 ±0.09	±0.03 0.85±0.06 ±0.09	13.7± 2.7 2.7± 1.2 1.4± 0.9	15.9± 3.0 3.1± 1.3 1.5± 0.9	2	6
5	0.61	0.60	±0.08 0.81±0.17 ±0.24	±0.07 0.78±0.17 ±0.26	19.4± 7.7 3.9± 3.5 1.9± 2.4	24.4± 8.7 4.8± 3.8 2.3± 2.7	1	3
6	0.73	0.88	±0.03 0.86±0.07 ±0.10	±0.03 0.85±0.07 ±0.10	13.7± 3.2 2.7± 1.5 1.4± 1.0	15.9± 3.5 3.1± 1.6 1.5± 1.1	2	5
7	1.00	0.88	±0.002 1.00±0.004 ±0.005	±0.002 1.00±0.004 ±0.005	0.05±0.2 0.01±7.4 0.005±5.2	0.05±0.2 0.01±7.3 0.005± 5.1	0	6
8	1.00	1.35	±0.004 1.00±0.009 ±0.01	±0.004 1.00±0.009 ±0.01	0.05±0.4 0.01±0.2 0.005±0.1	0.05±0.4 0.01±0.2 0.005±0.1	0	3

continued...

Line	Observed Viability Whole	Half	Expected Viabilities Additive	Multiplicative	Number of Loci Additive	Multiplicative	Number of Sublines in 95% Interval	Sublines
			±0.03	±0.03	19.4 ± 3.3	24.3± 3.7		
9	0.61	1.06	0.81±0.07	0.78±0.07	3.9 ± 1.5	4.8± 1.6	1	6
			±0.10	±0.10	1.9 ± 1.0	2.3± 1.1		
			±0.04	±0.04	13.7 ± 4.2	15.9± 4.5		
10	0.73	1.02	0.86±0.09	0.85±0.09	2.7 ± 1.9	3.1± 2.0	2	4
			±0.13	±0.13	1.4 ± 1.3	1.5± 1.4		
			±0.03	±0.03	13.1 ± 2.7	15.0± 2.9		
11	0.74	1.07	0.87±0.06	0.86±0.06	2.6 ± 1.2	2.9± 1.3	2	6
			±0.08	±0.08	1.3 ± 0.8	1.4± 0.9		
			±0.09	±0.08	26.4 ± 9.0	37.2±10.7		
12	0.47	0.48	0.74±0.20	0.69±0.19	5.3 ± 4.0	7.3± 4.7	2	3
			±0.29	±0.29	2.6 ± 2.9	3.6± 3.3		
			±0.03	±0.03	3.5 ± 3.3	3.6± 3.3		
13	0.93	0.88	0.97±0.07	0.96±0.08	0.7 ± 1.5	0.7± 1.5	1	3
			±0.10	±0.11	0.3 ± 1.0	0.3± 1.0		
			±0.03	±0.03	18.1 ± 3.2	22.4± 3.5		
14	0.64	0.75	0.82±0.07	0.80±0.07	3.6 ± 1.4	4.4± 1.6	3	6
			±0.10	±0.10	1.8 ± 1.0	2.1± 1.1		
			±0.04	±0.03	30.6 ± 4.1	47.1± 5.1		
15	0.39	0.81	0.69±0.09	0.62±0.08	6.1 ± 1.8	9.2± 2.3	1	6
			±0.13	±0.11	3.1 ± 1.3	4.5± 1.6		
			±0.06	±0.06	11.0 ± 5.8	12.3± 6.2		
16	0.78	1.14	0.89±0.13	0.88±0.13	2.2 ± 2.6	2.4± 2.7	3	3
			±0.18	±0.20	1.1 ± 1.8	1.2± 1.9		

continued...

Line	Observed Viability		Expected Viabilities		Number of Loci		Number of Sublines	Sublines
	Whole	Half	Additive	Multiplicative	Additive	Multiplicative	in 95% Interval	
17	0.68	0.79	±0.04 0.84±0.08 ±0.11	±0.03 0.83±0.08 ±0.11	16.0 ± 3.5 3.2 ± 1.6 1.6 ± 1.1	19.1 ± 3.8 3.7 ± 1.7 1.8 ± 1.2	2	5
18	1.00	1.08	±0.002 1.00±0.004 ±0.005	±0.002 1.00±0.004 ±0.005	0.05 ±0.2 0.01± 0.07 0.005±0.05	0.05± 0.2 0.01± 0.07 0.005±0.05	1	6
19	0.61	0.86	±0.04 0.81±0.09 ±0.12	±0.03 0.78±0.08 ±0.12	19.6 ± 3.9 3.9 ± 1.7 2.0 ± 1.2	24.8 ± 4.4 4.9 ± 1.9 2.4 ± 1.3	3	5
20	1.00	1.06	±0.003 1.00±0.006 ±0.008	±0.003 1.00±0.006 ±0.008	0.05± 0.25 0.01± 0.11 0.005±0.08	0.05± 0.25 0.01± 0.11 0.005±0.08	0	4
21	0.29	0.84	±0.10 0.64±0.23 ±0.33	±0.08 0.54±0.20 ±0.30	35.7 ±10.5 7.1 ± 4.7 3.6 ± 3.3	62.1 ±13.8 12.2 ± 6.1 15.9 ± 4.3	2	3
22	0.61	1.91	±0.28 0.81±0.63 ±0.89	±0.29 0.78±0.82 ±1.40	19.6 ±28.1 3.9 ±12.6 2.0 ± 8.9	24.8 ±31.6 4.9 ±14.0 2.4 ± 9.8	1	2
23	1.00	0.85	±0.01 1.00±0.03 ±0.04	±0.01 1.00±0.03 ±0.05	0.05± 1.4 0.01± 0.6 0.005±0.4	0.05± 1.4 0.01± 0.6 0.005±0.4	1	2
24	0.51	0.69	±0.09 0.76±0.19 ±0.27	±0.08 0.72±0.18 ±0.29	24.4 ± 8.7 4.9 ± 3.9 2.4 ± 2.7	33.2 ±10.1 6.5 ± 4.5 3.2 ± 3.1	3	3

continued...

Line	Observed Viability Whole	Half	Expected Viabilities Additive	Multiplicative	Number of Loci Additive	Multiplicative	Number of Sublines in 95% Interval	Sublines
25	0.81	1.02	±0.03 0.90±0.06 ±0.09	±0.03 0.90±0.06 ±0.09	9.8 ± 2.7 2.0 ± 1.2 1.0 ± 0.9	10.8± 2.9 2.1 ± 1.3 1.0 ± 0.9	0	5
26	1.00	1.20	±0.002 1.00±0.004 ±0.005	±0.002 1.00±0.004 ±0.005	0.05± 0.2 0.01± 7.4 0.005±5.2	0.05± 0.2 0.01± 7.3 0.005±5.1	0	6
27	0.65	0.53	±0.26 0.83±0.59 ±0.84	±0.28 0.81±0.76 ±1.29	17.4 ±26.5 3.5 ±11.8 1.7 ± 8.4	21.2 ±29.3 4.2 ±12.9 2.0 ± 9.0	2	2
28	0.95	0.84	±0.02 0.97±0.04 ±0.06	±0.02 0.97±0.04 ±0.06	2.6 ± 1.8 0.5 ± 0.8 0.3 ± 0.6	2.7 ± 1.8 0.5 ± 0.8 0.3 ± 0.6	0	4
29	0.93	0.93	±0.13 0.96±0.28 ±0.40	±0.13 0.96±0.33 ±0.50	3.8 ±12.6 0.8 ± 5.6 0.4 ± 4.0	3.9 ±12.8 0.8 ± 5.7 0.4 ± 4.0	2	3
30	0.55	0.82	±0.08 0.78±0.19 ±0.26	±0.07 0.74±0.18 ±0.27	22.5 ± 8.3 4.5 ± 3.7 2.2 ± 2.6	29.7 ± 9.6 5.8 ± 4.2 2.8 ± 3.0	3	3
31	0.35	0.89	±0.10 0.67±0.22 ±0.32	±0.08 0.59±0.20 ±0.30	32.6 ±10.0 6.5 ± 4.5 3.3 ± 3.2	52.5 ±12.7 10.3 ± 5.6 5.0 ± 3.9	1	3
32	1.00	1.00	±0.02 1.00±0.04 ±0.06	±0.02 1.00±0.04 ±0.06	0.05± 1.8 0.01± 0.8 0.005±0.6	0.05± 1.8 0.01± 0.8 0.05± 0.5	1	4

continued...

Line	Observed Viability Whole	Half	Expected Viabilities Additive	Multiplicative	Number of Loci Additive	Multiplicative	Number of Sublines in 95% Interval	Sublines
33	0.71	1.30	±0.07 0.85±0.15 ±0.21	±0.06 0.84±0.15 ±0.22	14.6 ± 6.7 2.9 ± 3.0 1.5 ± 2.1	17.1 ± 7.3 3.4 ± 3.2 1.6 ± 2.2	1	3
34	0.71	0.62	±0.07 0.86±0.15 ±0.21	±0.06 0.84±0.15 ±0.22	14.4 ± 6.7 2.9 ± 3.0 1.4 ± 2.1	16.8 ± 7.2 3.3 ± 3.2 1.6 ± 2.2	2	3
35	0.45	0.71	±0.33 0.73±0.74 ±1.05	±0.33 0.67±0.99 ±1.80	27.5 ±33.3 5.5 ±14.9 2.8 ±10.5	39.7 ±40.0 7.8 ±17.7 3.8 ±12.4	2	2
36	0.72	0.98	±0.04 0.86±0.09 ±0.13	±0.04 0.85±0.09 ±0.13	13.8 ± 4.2 2.8 ± 1.9 1.4 ± 1.3	16.1 ± 4.5 3.1 ± 2.0 1.5 ± 1.4	1	4
37	1.00	0.82	±0.004 1.00±0.009 ±0.010	±0.004 1.00±0.009 ±0.010	0.05± 0.4 0.01± 0.2 0.005±0.1	0.05± 0.4 0.01± 0.2 0.005±0.1	0	3
38	0.47	0.89	±0.04 0.73±0.09 ±0.12	±0.03 0.68±0.07 ±0.11	26.7 ± 3.8 5.3 ± 1.7 2.7 ± 1.2	37.9 ± 4.6 7.4 ± 2.0 3.6 ± 1.4	1	6
39	0.19	0.47	±0.11 0.60±0.25 ±0.35	±0.08 0.44±0.19 ±0.30	40.4 ±11.2 8.1 ± 5.0 4.0 ± 3.5	81.8 ±15.9 16.0 ± 7.0 7.8 ± 4.9	1	3
40	0.88	0.80	±0.04 0.94±0.09 ±0.13	±0.04 0.94±0.10 ±0.14	5.8 ± 4.2 1.2 ± 1.9 0.6 ± 1.3	6.1 ± 4.4 1.2 ± 1.9 1.6 ± 1.3	0	3

viability coefficient (.01, .05, or .10) we estimated n for both models. For the additive model $n = (1-(1-ns))/s$ and the half chromosome expectation is $Exp(V) = (1-ns/2)$ For the multiplicative model $n = (1-ns)^{1/n}$ and the half chromosome expectation is $Exp(V) = (1-ns)^{n/2}$

95% confidence intervals were calculated for n by assuming a Poisson ($\bar{x} = s^2$) and for V by using the upper and lower n limits. This test generates expected half chromosome viabilities, using the whole chromosome viabilities, to generate expectations in accordance with one of the two theoretical models as in the previous test (Table 7). Here, however, three values of relative single locus fitnesses are assumed. This leads to an expectation of a certain number of loci which cause the given viability depression. The viability data are then checked against the expected values and the number of sublines, for each line, which fall within the 95% confidence limits of the expected viabilities is recorded. It was found that the most sublines fall outside of the 95% confidence limits for each viability class. Those which fall within the given confidence limits do so within the class of small number of loci with larger effects. This result could possibly be interpreted as contradictory to the idea of many loci with small effects and even distribution along the chromosome. Such interpretation is limited, however, by the fact that only three expected viability values were generated, using three theoretical single locus fitnesses out of an infinite possible number of values.

DISCUSSION

The results of this study, taken together, strongly support the conclusions of several theoretical papers on epistasis and intrachromosomal interaction, in that they show the presence of strong interactions between the two arms of the second chromosome. These interactions are synergistic and they do not conform either to the additive or to the multiplicative model of gene action. Dobzhansky, Spassky and Anderson (1965) also found significant synergistic interactions between the second and third chromosomes of Drosophila pseudoobscura and Temin et al. (1969) found slight reinforcing epistasis between the second and third chromosomes in Drosophila melanogaster .

Lewontin (1964 a,b) wrote a paper on the interaction of selection and linkage where he summarized earlier work (Kimura 1956; Lewontin and Kojima 1960; Bodmer and Parsons 1962) which indicated that even in the simplest cases (two loci, simple symmetrical selective values) linkage might have dramatic effects on the course of natural selection. The reverse is also true; natural selection may modify linkage relationships and recombination rates in populations.

Lewontin's computer simulation results of two and five locus interacting systems support the conclusions of previous studies in that :

1. loci may be kept in permanent linkage disequilibrium, by natural selection, despite gene frequency equilibrium;
2. disequilibrium can be maintained even for genes that are unlinked if epistasis is strong;
3. epistasis may be generated by simple multiplicative fitnesses;
4. linkage disequilibrium results in higher mean fitness.

In a subsequent computer simulation study Franklin and Lewontin (1970), working with up to 36 locus systems, various allele frequences, 200 simulated generations and incorporating varying amounts of recombination, showed that the degree of linkage disequilibrium between a pair of loci is not simply a function of the fitnesses of the two locus system. Disequilibrium may be largely determined by the average effects of many loci which form a linked complex with the loci under study. Thus the degree of disequilibrium is apparently also a function of the map length of the given chromosomal segment. The simulation results, under a variety of assumptions, such as different initial gametic

type frequencies and various selection pressures, were essentially the same. Particularly, the average correlation in gene frequency between a pair of loci on a chromosome segment was found to be largely independent of the number of loci in that segment. This means that such disequilibrium is practically independent of the average effects of a locus in the segment and, therefore, loci are not interacting multiplicatively, nor in an additive manner in these computer models.

Sved, Reed and Bodmer (1967) and King (1967) suggested another model of fitness which does not lead to unreasonably large fitness depressions on inbreeding. The method is based on a model which does not assume multiplicative interaction among loci; because such interaction would theoretically lead to large genetic loads and overestimates of fitness depression (but see Tracey and Ayala 1974 for an opposite point of view). The selection models of the above authors assume that some proportion of the population survives irrespective of the exact genotypic composition. The survival reflects the severity of the environment and the availability of niche space. Thus the mean adaptedness does not necessarily change as the population evolves, since this adaptedness is the proportion of the population which is surviving. But

the relative fitnesses of the genotypes in the population do change. Selection is by truncation, saving the phenotypes with the highest score on a normal distribution curve of phenotypes, which results from the multilocus determination of the character.

Another question arises - why, assuming the advantages of epistasis and the resultant close linkage, does not the genome coalesce into one large unit (Turner, 1967). Two answers have been proposed :

1. The large size of such a megalogene would probably interfere with the processes of meiosis and, perhaps, with other processes at the biochemical and physiological level. Thus the unichromosomal condition is not observed, because it would disrupt reproduction and perhaps function.
2. Wills and Miller (1976) suggest that "in an outbred population, selection for reduction in recombination allowing the buildup of epistatically interacting blocks of loci can be opposed by selection for random assortment".

They suggest that this is so, because linkage disequilibrium may slow the approach of polymorphisms to their selective equilibrium points. To illustrate this point, we may consider two loci, either unlinked or with no linkage disequilibrium between them. Each locus has two alleles. If one locus is

near its selective equilibrium point, while the other is far from it, strong selection will move the latter locus rapidly toward equilibrium point, without affecting the other locus in any appreciable way. If the two loci are strongly linked and in a state of linkage disequilibrium, a selective force acting on the distant locus will also move the other locus out of its equilibrium. This lessens the effectiveness of selection. The result indicates that the balance between long-term selection for linkage tightening in the case of favourable epistatic interactions, and long-term selection for high levels of heterozygosity is dependent on the distribution of equilibrium frequencies in the population. Wills and Miller (1976) found that random assortment has an advantage over linkage in the rapidity of movement of alleles toward their equilibrium points. Thus the populations with loose linkage should, at least theoretically, be able to adapt more rapidly to environmental changes, where adaptation depends on allele frequencies at single loci. The average relative fitness of organisms in such populations should also increase more rapidly than those with tight linkage.

CONCLUSION

Very recently Wright (1977) summarized extensively the early experimental work on inbreeding depression and heterosis in the plants and animals. He refers to a study by Robertson and Reeve (1955) who showed the depression of thorax and wing length, and egg production in two strains of inbred parental Drosophila melanogaster in relation to the F_1 generation. The depression was very significant in the case of the egg production. Analysis of variance indicated significant interaction among chromosomes in 22 of the 36 cases, with 19 at the 0.01 level. Wright (1977) concludes that although theoretically, on the assumption of additivity of locus effects, there is proportionality of inbreeding decline to the increase in the inbreeding coefficient, the evidence shows "important nonadditive interactions that cannot be overcome by any transformation of scale". Thus, the early work supports the results of Spassky et al (1965) and Temin et al (1969).

This study brings in another piece of evidence for nonadditive interactions at the hitherto very little studied intrachromosomal level.

REFERENCES

Bodmer,W.F.,and P.A.Parsons.1962. Linkage and recombination in evolution. Advan. Genet. 11: 1-100 .

Dobzhansky,T. 1955. A review of some fundamental concepts and problems of population genetics. Cold Spring Harb. Symp. Quant.Biol. 20: 1-15.

Dobzhansky, T., and B.Spassky. 1963. Genetics of natural populations. XXXIV. Adaptive norm, genetic load, and genetic elite in Drosophila pseudoobscura Genetics 48:1467-86 .

Franklin,I., and Lewontin,R.C. 1970. Is the gene the unit of selection? Genetics 65:707-734 .

Haldane, J.B.S. 1956. The estimation of viabilities. J.Genetics 54:294-96 .

Kidwell,J.F.,Tracey, M.L., Glaser,P., and M.M.Kidwell.1971. A chromosomal analysis of x-ray induced genetic variance of wing length in Drosophila melanogaster Can.J. Genet. Cytol. 13:600-606.

Kimura,M. 1956. A model of a genetic system which leads to closer linkage by natural selection. Evolution 10:278-87.

King,J.L. 1967. Continuously distributed factors affecting fitness. Genetics 55:483-92.

Levene,H.I., Lerner,M., Sokoloff,A., Ho,F.K., and I.R.Franklin. 1965. Genetic load in Tribolium. Proc.Natl.Acad. Sci.U.S. 53:1042-50.

Lewontin,R.C., and K.Kojima. 1960. The evolutionary dynamics of complex polymorphisms. Evolution 14:458-472.

Lewontin,R.C. 1963.The role of linkage in natural selection. In Geerts,S.J. ed. 1965. Genetics Today. Proceeding of the 11th Int. Congress of Genetics. Vol.3. Pergamon Press, Oxford.

Lewontin,R.C. 1964a. The interaction of selection and linkage. I. General considerations heterotic models. Genetics 49 :49-67.

Lewontin,R.C. 1964b. The interaction of selection and linkage. II. Optimum models. Genetics 50:757-782.

Lewontin,R.C. 1974. The Genetic Basis of Evolutionary Change. Columbia University Press, N.Y.

Mather,K. 1973. Genetical Structure of Populations. Chapman and Hall, London.

Mukai,T. 1969. The genetic structure of natural populations of Drosophila melanogaster. VII. Synergistic interaction of spontaneous mutant polygenes controlling viability. Genetics 61: 479-95 .

Robertson,F.W.,and E.C.R.Reeve. 1955. Studies in quantitative inheritance. VII. Crosses between strains of different body size in Drosophila melanogaster. Z.ind.Abst.Vererb.86:424-38.

Spassky,B., Dobzhansky,T., and W.W.Anderson. 1965. Genetics of natural populations. XXXVI. Epistatic interactions of the components of the genetic load in Drosophila pseudoobscura . Genetics 52:653-64

Sved, J.A., Reed,E., and W.J.Bodmer. 1967. The numbers of balanced polymorphisms that can be maintained in a natural population. Genetics 55:469-481.

Temin,R.G., Meyer,H.V., Dawson,P.S., and J.F. Crow. 1969 The influence of epistasis on homozygous viability depression in Drosophila melanogaster Genetics 61 : 497-519.

Tracey, M.L., and F.J.Ayala. 1974. Genetic load in natural populations : is it compatible with the hypothesis that many polymorphisms are maintained by natural selection ? Genetics 77: 569-89.

Turner,J.R. 1967. Why does the genotype not congeal? Evolution 21:645-56 .

Wills,C . and C. Miller. 1976. A computer model allowing maintenance of large amounts of genetic variability in Mendelian populations.II: The balance of forces between linkage and random assortment. Genetics 82 : 377-99.

Wright, S. 1977. Evolution and the genetics of populations. Vol.3. The University of Chicago Press, Chicago.

APPENDIX I

Experimental Data

a) Whole homozygous chromosomes

Line	Sublines	Brood 1		Brood 2		Combined broods	
		Homo-zygotes	Hetero-zygotes	Homo-zygotes	Hetero-zygotes	Homo-zygotes	Hetero-zygotes
1							
	1x1	11	39	---	---	11	39
	2x2	--	81	---	96	--	177
	3x3	--	77	---	84	--	161
	4x4	11	75	4	74	15	149
2							
	1x1	22	81	29	63	51	144
	2x2	--	--	--	--	--	--
	3x3	3	27	--	15	3	42
	4x4	10	32	--	--	10	32
3							
	1x1	25	59	16	26	41	85
	2x2	51	86	32	55	83	141
	3x3	13	36	16	29	29	65
	4x4	22	58	6	16	28	74
4							
	1x1	32	67	20	29	52	96
	2x2	18	53	33	57	51	110
	3x3	16	80	7	30	23	110
	4x4	2	7	--	--	2	7
5							
	1x1	10	52	--	--	10	52
	2x2	8	78	26	49	34	127
	3x3	8	36	18	19	26	55
	4x4	--	--	--	--	--	--
6							
	1x1	22	75	28	58	50	133
	2x2	14	93	20	59	34	152
	3x3	40	65	24	49	64	114
	4x4	3	9	--	--	3	9
7							
	1x1	44	40	--	--	44	40
	2x2	11	16	--	--	11	16
	3x3	16	62	21	72	37	134
	4x4	25	46	47	61	72	107
8							
	1x1	--	71	--	--	--	71
	2x2	27	21	8	18	35	39
	3x3	21	44	35	64	56	108
	4x4	--	--	--	--	--	--
9							
	1x1	19	58	28	50	47	108
	2x2	10	54	--	--	10	54
	3x3	--	47	--	--	--	47
	4x4	--	--	--	--	--	--
10							
	1x1	15	69	27	56	42	125
	2x2	38	80	4	14	42	94
	3x3	28	70	19	77	47	147
	4x4	--	--	--	--	--	--

Line	Sublines	Brood 1		Brood 2		Combined broods	
		Homo-zygotes	Hetero-zygotes	Homo-zygotes	Hetero-zygotes	Homo-zygotes	Hetero-zygotes
11							
	1x1	--	10	--	--	--	10
	2x2	5	12	--	--	5	12
	3x3	21	59	22	52	43	111
	4x4	19	55	--	--	19	55
12							
	1x1	5	27	--	--	5	27
	2x2	27	45	--	--	27	45
	3x3	4	49	--	--	4	49
	4x4	7	70	--	--	7	70
13							
	1x1	33	107	34	93	67	200
	2x2	34	61	5	8	39	69
	3x3	5	7	34	69	39	76
	4x4	--	--	--	--	--	--
14							
	1x1	8	37	6	12	14	49
	2x2	--	83	--	--	--	83
	3x3	21	72	14	28	35	100
	4x4	25	94	33	81	58	175
15							
	1x1	16	69	8	84	24	153
	2x2	11	45	12	53	23	98
	3x3	--	34	--	9	--	43
	4x4	--	37	--	--	--	37
16							
	1x1	34	60	22	68	56	128
	2x2	16	45	17	44	33	89
	3x3	--	--	--	--	--	--
	4x4	5	15	5	11	10	26
17							
	1x1	8	16	--	--	8	16
	2x2	5	39	--	--	5	39
	3x3	36	66	--	--	36	66
	4x4	8	97	31	72	39	169
18							
	1x1	27	64	20	43	47	107
	2x2	21	57	14	37	35	94
	3x3	31	74	29	34	60	108
	4x4	33	57	30	38	63	95
19							
	1x1	13	40	8	25	21	65
	2x2	9	33	11	39	20	72
	3x3	--	107	--	101	--	208
	4x4	7	12	17	62	24	74
20							
	1x1	20	42	19	35	39	77
	2x2	--	--	--	--	--	--
	3x3	32	94	--	--	32	94
	4x4	12	17	--	--	12	17

Line	Sublines	Brood 1		Brood 2		Combined broods	
		Homo-zygotes	Hetero-zygotes	Homo-zygotes	Hetero-zygotes	Homo-zygotes	Hetero-zygotes
21							
	1x1	--	42	--	--	--	42
	2x2	3	106	--	--	3	106
	3x3	--	--	--	--	--	--
	4x4	14	42	7	38	21	80
22							
	1x1	--	--	--	--	--	--
	2x2	--	--	--	--	--	--
	3x3	11	38	7	17	18	55
	4x4	15	20	10	66	25	86
23							
	1x1	25	34	--	--	25	34
	2x2	27	55	27	64	54	119
	3x3	34	49	18	52	52	101
	4x4	--	--	--	--	--	--
24							
	1x1	--	--	--	--	--	--
	2x2	--	--	--	--	--	--
	3x3	3	18	--	--	3	18
	4x4	14	47	18	42	32	89
25							
	1x1	7	16	--	--	7	16
	2x2	29	73	31	92	60	165
	3x3	11	22	--	--	11	22
	4x4	12	50	22	52	37	102
26							
	1x1	14	14	--	--	14	14
	2x2	--	36	--	--	--	36
	3x3	25	38	24	75	49	113
	4x4	28	55	35	75	63	130
27							
	1x1	--	35	--	--	--	35
	2x2	20	77	2	4	22	81
	3x3	--	--	--	--	--	--
	4x4	23	43	22	73	45	116
28							
	1x1	--	--	--	--	--	--
	2x2	--	--	--	--	--	--
	3x3	10	39	--	--	10	39
	4x4	30	42	--	--	30	42
29							
	1x1	35	81	--	--	35	81
	2x2	--	--	--	--	--	--
	3x3	31	57	--	--	31	57
	4x4	20	46	--	--	20	46
30							
	1x1	11	47	--	7	11	54
	2x2	26	98	39	86	65	184
	3x3	--	--	--	--	--	--
	4x4	22	77	7	27	29	104

Line	Sublines	Brood 1		Brood 2		Combined broods	
		Homo-zygotes	Hetero-zygotes	Homo-zygotes	Hetero-zygotes	Homo-zygotes	Hetero-zygotes
31							
	1x1	--	--	--	--	--	--
	2x2	2	16	--	--	2	16
	3x3	--	--	--	--	--	--
	4x4	22	89	13	62	35	151
32							
	1x1	27	80	28	66	55	146
	2x2	--	112	--	--	--	112
	3x3	28	75	13	33	41	108
	4x4	7	5	3	7	10	12
33							
	1x1	2	26	--	--	2	26
	2x2	--	--	--	--	--	--
	3x3	33	43	7	19	40	62
	4x4	--	--	--	--	--	--
34							
	1x1	23	74	2	12	25	86
	2x2	33	76	16	38	49	114
	3x3	--	--	--	--	--	--
	4x4	--	--	--	--	--	--
35							
	1x1	--	--	--	--	--	--
	2x2	--	--	--	--	--	--
	3x3	18	63	6	42	24	105
	4x4	30	107	12	64	42	171
36							
	1x1	24	60	4	12	28	72
	2x2	18	124	45	96	63	220
	3x3	35	73	23	65	58	138
	4x4	--	72	--	25	--	97
37							
	1x1	12	14	--	--	12	14
	2x2	17	25	2	5	19	30
	3x3	21	34	--	--	21	34
	4x4	30	69	--	--	30	69
38							
	1x1	21	84	16	35	37	119
	2x2	11	99	--	51	11	150
	3x3	38	89	33	76	71	165
	4x4	4	24	--	6	4	30
39							
	1x1	2	105	12	76	14	181
	2x2	--	46	--	--	--	46
	3x3	--	--	--	--	--	--
	4x4	13	77	6	86	19	163
40							
	1x1	30	62	7	17	37	79
	2x2	39	80	36	78	75	158
	3x3	19	49	20	75	49	124
	4x4	--	--	--	--	--	--

Line	Sublines	Brood 1		Brood 2		Combined broods	
		Homo-zygotes	Hetero-zygotes	Homo-zygotes	Hetero-zygotes	Homo-zygotes	Hetero-zygotes
41							
	1x1	13	29	12	52	25	81
	2x2	--	--	--	--	--	--
	3x3	23	44	--	--	23	44
	4x4	--	33	--	--	--	33
42							
	1x1	--	--	--	--	--	--
	2x2	27	51	27	44	54	95
	3x3	--	--	--	--	--	--
	4x4	20	60	7	24	27	84
43							
	1x1	23	107	--	--	23	107
	2x2	10	24	--	--	10	24
	3x3	--	--	--	--	--	--
	4x4	15	48	19	58	34	106
44							
	1x1	--	--	--	--	--	--
	2x2	10	56	--	--	10	56
	3x3	23	80	1	5	24	85
	4x4	--	--	--	--	--	--

APPENDIX I

Experimental Data

b) Half homozygous chromosomes

Line	Sublines	Brood 1		Brood 2		Combined broods	
		Homo-zygotes	Hetero-zygotes	Homo-zygotes	Hetero-zygotes	Homo-zygotes	Hetero-zygotes
1							
	1x2	--	11	--	--	--	11
	1x3	--	86	--	102	--	188
	1x4	12	96	25	74	37	170
	2x3	6	66	14	97	20	163
	2x4	--	--	--	--	--	--
	3x4	41	87	30	73	71	160
2							
	1x2	--	--	--	--	--	--
	1x3	2	3	--	--	2	3
	1x4	25	60	16	60	41	120
	2x3	--	--	--	--	--	--
	2x4	--	--	--	--	--	--
	3x4	9	81	35	62	44	143
3							
	1x2	29	81	29	54	58	135
	1x3	21	61	21	32	42	93
	1x4	28	71	22	49	50	120
	2x3	22	110	10	32	32	142
	2x4	15	36	11	36	26	72
	3x4	--	--	--	--	--	--
4							
	1x2	10	5	--	--	10	5
	1x3	16	41	33	51	49	92
	1x4	27	49	39	67	66	116
	2x3	19	52	28	52	47	104
	2x4	17	102	17	55	34	157
	3x4	25	59	17	36	42	95
5							
	1x2	8	78	27	61	35	139
	1x3	6	14	11	26	17	40
	1x4	--	--	--	--	--	--
	2x3	4	22	14	53	18	75
	2x4	--	--	--	--	--	--
	3x4	--	--	--	--	--	--
6							
	1x2	12	74	28	64	40	138
	1x3	--	91	--	--	--	91
	1x4	27	92	43	50	70	142
	2x3	36	75	--	--	36	75
	2x4	11	42	37	66	48	108
	3x4	9	17	--	--	9	17
7							
	1x2	30	67	27	66	57	133
	1x3	27	68	16	33	43	101
	1x4	19	44	--	--	19	44
	2x3	9	19	--	--	9	19
	2x4	26	49	--	--	26	49
	3x4	16	48	11	19	27	67

Line	Sublines	Brood 1		Brood 2		Combined broods	
		Homo-zygotes	Hetero-zygotes	Homo-zygotes	Hetero-zygotes	Homo-zygotes	Hetero-zygotes
8							
	1x2	47	49	20	14	67	63
	1x3	33	54	--	--	33	54
	1x4	--	--	--	--	--	--
	2x3	26	72	3	6	29	78
	2x4	--	--	--	--	--	--
	3x4	--	--	--	--	--	--
9							
	1x2	35	51	21	44	56	95
	1x3	24	41	--	--	24	41
	1x4	23	28	4	8	27	36
	2x3	48	96	38	89	86	185
	2x4	32	66	30	69	62	135
	3x4	--	--	--	--	--	--
10							
	1x2	30	59	--	--	30	59
	1x3	29	61	--	--	29	61
	1x4	23	52	11	19	34	71
	2x3	--	--	--	--	--	--
	2x4	26	43	--	--	26	43
	3x4	--	--	--	--	--	--
11							
	1x2	25	39	17	39	42	78
	1x3	20	49	--	--	20	49
	1x4	28	45	--	--	28	45
	2x3	28	35	24	65	52	100
	2x4	20	45	--	--	20	45
	3x4	36	49	--	--	36	49
12							
	1x2	1	24	13	23	14	47
	1x3	--	54	10	33	10	87
	1x4	--	17	--	--	--	17
	2x3	--	--	--	--	--	--
	2x4	6	23	13	38	19	61
	3x4	6	35	--	14	6	49
13							
	1x2	11	11	34	76	45	87
	1x3	44	95	32	83	76	178
	1x4	--	--	--	--	--	--
	2x3	37	100	36	87	73	187
	2x4	--	--	--	--	--	--
	3x4	--	--	--	--	--	--
14							
	1x2	10	27	--	--	10	27
	1x3	16	28	1	8	17	36
	1x4	25	57	10	41	35	98
	2x3	22	51	6	10	28	61
	2x4	12	46	--	--	12	46
	3x4	31	85	--	--	31	85

Line	Sublines	Brood 1		Brood 2		Combined broods	
		Homo-zygotes	Hetero-zygotes	Homo-zygotes	Hetero-zygotes	Homo-zygotes	Hetero-zygotes
15							
	1x2	29	81	18	54	47	135
	1x3	25	70	39	63	64	133
	1x4	31	64	4	8	35	72
	2x3	9	57	--	--	9	57
	2x4	17	20	--	--	17	20
	3x4	5	32	--	--	5	32
16							
	1x2	14	16	--	--	14	16
	1x3	--	--	--	--	--	--
	1x4	15	46	--	--	15	46
	2x3	45	78	--	--	45	78
	2x4	--	--	--	--	--	--
	3x4	--	--	--	--	--	--
17							
	1x2	20	42	15	33	35	75
	1x3	6	14	--	--	6	14
	1x4	21	59	30	34	51	93
	2x3	21	69	--	--	21	69
	2x4	2	9	--	--	2	9
	3x4	16	42	28	51	44	93
18							
	1x2	20	45	--	--	20	45
	1x3	23	51	6	5	29	56
	1x4	27	50	25	53	52	103
	2x3	33	70	22	35	55	105
	2x4	36	47	--	--	36	47
	3x4	21	38	--	--	21	38
19							
	1x2	14	34	5	12	19	46
	1x3	56	129	12	32	68	161
	1x4	8	48	4	13	12	61
	2x3	19	27	12	14	31	41
	2x4	--	--	--	--	--	--
	3x4	32	81	26	64	58	145
20							
	1x2	--	--	--	--	--	--
	1x3	23	46	23	34	46	80
	1x4	26	62	22	61	48	123
	2x3	--	--	--	--	--	--
	2x4	18	25	20	87	52	130
	3x4	30	52	9	31	39	83
21							
	1x2	--	--	--	--	--	--
	1x3	9	46	8	22	17	68
	1x4	31	81	21	47	52	128
	2x3	--	46	--	14	--	60
	2x4	--	--	--	--	--	--
	3x4	25	48	31	42	56	90

Line	Sublines	Brood 1		Brood 2		Combined broods	
		Homo-zygotes	Hetero-zygotes	Homo-zygotes	Hetero-zygotes	Homo-zygotes	Hetero-zygotes
22							
	1x2	--	--	--	--	--	--
	1x3	--	--	--	--	--	--
	1x4	--	--	--	--	--	--
	2x3	6	--	--	--	6	--
	2x4	22	19	--	--	22	19
	3x4	12	32	73	72	85	104
23							
	1x2	24	54	22	40	46	94
	1x3	--	--	--	--	--	--
	1x4	--	--	--	--	--	--
	2x3	--	--	--	--	--	--
	2x4	24	71	18	44	42	115
	3x4	--	--	--	--	--	--
24							
	1x2	--	--	--	--	--	--
	1x3	24	62	--	--	24	62
	1x4	22	52	10	25	32	77
	2x3	--	--	--	--	--	--
	2x4	--	--	--	--	--	--
	3x4	13	53	10	42	23	95
25							
	1x2	--	--	--	--	--	--
	1x3	25	42	--	--	25	42
	1x4	14	23	--	--	14	23
	2x3	9	30	--	--	9	30
	2x4	24	68	23	68	47	136
	3x4	31	36	30	45	61	81
26							
	1x2	6	6	--	--	6	6
	1x3	28	56	13	22	41	78
	1x4	22	36	--	--	22	36
	2x3	28	67	--	--	28	67
	2x4	14	21	--	--	14	21
	3x4	43	73	10	17	53	90
27							
	1x2	24	63	16	23	40	86
	1x3	--	--	--	--	--	--
	1x4	2	28	--	--	2	28
	2x3	--	--	--	--	--	--
	2x4	--	--	--	--	--	--
	3x4	--	--	--	--	--	--
28							
	1x2	--	--	--	--	--	--
	1x3	--	--	--	--	--	--
	1x4	1	17	--	--	1	17
	2x3	8	6	--	--	8	6
	2x4	2	7	--	--	2	7
	3x4	26	65	33	74	59	139

Line	Sublines	Brood 1		Brood 2		Combined broods	
		Homo-zygotes	Hetero-zygotes	Homo-zygotes	Hetero-zygotes	Homo-zygotes	Hetero-zygotes
29							
	1x2	--	--	--	--	--	--
	1x3	39	119	--	--	39	119
	1x4	39	73	39	75	78	148
	2x3	--	--	--	--	--	--
	2x4	--	--	--	--	--	--
	3x4	22	10	--	--	22	10
30							
	1x2	34	88	--	--	34	88
	1x3	--	--	--	--	--	--
	1x4	47	90	--	--	47	90
	2x3	--	--	--	--	--	--
	2x4	33	98	7	17	40	115
	3x4	--	--	--	--	--	--
31							
	1x2	--	--	--	--	--	--
	1x3	5	13	11	18	16	31
	1x4	--	--	--	--	--	--
	2x3	31	71	16	25	47	96
	2x4	--	--	--	--	--	--
	3x4	27	61	5	29	32	90
32							
	1x2	23	37	--	10	23	47
	1x3	14	37	15	48	29	85
	1x4	15	1	20	26	35	27
	2x3	28	94	44	36	72	130
	2x4	--	--	--	--	--	--
	3x4	--	--	--	--	--	--
33							
	1x2	--	--	--	--	--	--
	1x3	16	43	31	37	47	80
	1x4	20	38	--	--	20	38
	2x3	--	--	--	--	--	--
	2x4	--	--	--	--	--	--
	3x4	12	13	--	--	12	13
34							
	1x2	--	--	--	--	--	--
	1x3	--	--	--	--	--	--
	1x4	--	--	--	--	--	--
	2x3	14	51	7	9	21	60
	2x4	5	31	--	--	5	31
	3x4	30	75	30	66	60	141
35							
	1x2	--	--	--	--	--	--
	1x3	--	--	--	--	--	--
	1x4	34	70	--	--	34	70
	2x3	--	--	--	--	--	--
	2x4	--	--	--	--	--	--
	3x4	3	12	--	--	3	12

Line	Sublines	Brood 1		Brood 2		Combined broods	
		Homo-zygotes	Hetero-zygotes	Homo-zygotes	Hetero-zygotes	Homo-zygotes	Hetero-zygotes
36							
	1x2	10	32	--	--	10	32
	1x3	--	--	--	--	--	--
	1x4	30	42	--	--	30	42
	2x3	28	53	--	--	28	53
	2x4	--	--	--	--	--	--
	3x4	35	78	--	--	35	78
37							
	1x2	37	66	21	69	58	135
	1x3	26	78	--	--	26	78
	1x4	--	--	--	--	--	--
	2x3	--	--	--	--	--	--
	2x4	--	--	--	--	--	--
	3x4	22	56	10	11	32	67
38							
	1x2	22	68	13	47	35	115
	1x3	26	50	23	64	49	114
	1x4	13	20	--	--	13	20
	2x3	25	26	25	53	50	79
	2x4	10	94	14	81	24	175
	3x4	46	76	30	61	76	137
39							
	1x2	18	62	6	42	24	104
	1x3	--	--	--	--	--	--
	1x4	4	69	--	--	4	69
	2x3	--	--	--	--	--	--
	2x4	29	68	--	--	29	68
	3x4	--	--	--	--	--	--
40							
	1x2	35	60	--	--	35	60
	1x3	23	67	24	71	47	138
	1x4	--	--	--	--	--	--
	2x3	25	99	34	104	59	203
	2x4	--	--	--	--	--	--
	3x4	--	--	--	--	--	--
45							
	1x2	7	41	12	86	19	127
	1x3	--	--	--	--	--	--
	1x4	8	24	--	--	8	24
	2x3	--	--	--	--	--	--
	2x4	6	24	1	14	7	38
	3x4	--	--	--	--	--	--
46							
	1x2	5	17	--	--	5	17
	1x3	--	--	--	--	--	--
	1x4	27	64	2	3	29	67
	2x3	--	--	--	--	--	--
	2x4	21	60	--	--	21	60
	3x4	--	--	--	--	--	--

Line	Sublines	Brood 1		Brood 2		Combined broods	
		Homo-zygotes	Hetero-zygotes	Homo-zygotes	Hetero-zygotes	Homo-zygotes	Hetero-zygotes
47							
	1x2	17	25	5	18	22	43
	1x3	15	30	13	13	28	43
	1x4	21	53	25	39	46	92
	2x3	8	25	--	--	8	25
	2x4	33	77	14	47	47	124
	3x4	29	57	14	48	43	105
48							
	1x2	--	--	--	--	--	--
	1x3	--	--	--	--	--	--
	1x4	--	--	--	--	--	--
	2x3	41	99	39	95	80	194
	2x4	5	6	--	--	5	6
	3x4	--	96	--	--	--	96

APPENDIX II

Program computing expected viabilities and numbers of loci for the additive and multiplicative models.

THIS PROGRAM COMPUTES EXPECTED VIABILITIES AND NUMBERS OF LOCI
FOR THE ADDITIVE AND MULTIPLICATIVE MODELS

```
10 DIM  S(3),A(3),M(3),E(3),F(3),T(5),L(3),P(3),Q(3),R(3),X(3),Y
(3)
11 S(1)=0.99:S(2)=0.95:S(3)=0.90
12 T(1)=12.706:T(2)=4.303:T(3)=3.182:T(4)=2.776:T(5)=2.571
19 PRINT "V,H,N,D,W"
20 INPUT V,H,N,D,W
21 REM  V=2ND VIABILITY, H=HALF 2ND VIABILITY
22 REM  S(I) = LOCUS VIABILITIES;1=.99,2=.95,3=.90
23 REM  N= NO. OF SUBLINES;D=S.D. OF SUBLINES
24 REM  W= S.D. OVER SUBLINES FOR HALF CHROMOSOMES
25 PRINT "V=";V,"H=";H,"N=";N
26 PRINT "D=";D,"W=";W,
27 PRINT "S(1)=";S(1),"S(2)=";S(2),"S(3)=";S(3)
30 FOR I=1 TO 3:A(I)=(1-V)/(1-S(I)):NEXT I
31 REM  A(I)=NO. OF LOCI FOR ADDITIVE MODEL
35 PRINT "A(1)=";A(1),"A(2)=";A(2),"A(3)=";A(3)
40 FOR I=1 TO 3:M(I)=LOG(V)/LOG(S(I)):NEXT I
41 REM  M(I)=NO. OF LOCI FOR MULTIPLICATIVE MODEL
45 PRINT  "M(1)=";M(1),"M(2)=";M(2),"M(3)=";M(3)
46 REM  E(I) AND F(I) ARE HALF CHROMOSOME EXPECTATIONS
50 FOR I=1 TO 3:E(I)=1-0.5*A(I)*(1-S(I)):NEXT I
55 PRINT "E(1)=";E(1),"E(2)=";E(2),"E(3)=";E(3)
60 FOR I=1 TO 3:F(I)=S(I)!(0.5*M(I)):NEXT I
65 PRINT "F(1)=";F(1),"F(2)=";F(2),"F(3)=";F(3)
69 REM  COMPUTE 95 % CONFIDENCE LIMITS ON HALF
70      REM  CHROMOSOME VIABILITY BY ASSUMING A POISSON
71      REM  DISTRIBUTION OF NUMBER OF LOCI AND USING
72      REM  S.D.= MEAN TO CALCULATE LOCUS NUMBER LIMITS
73      REM  THEN  CALCULATE VIABILITY LIMITS
74 REM  L=95% CONFIDENCE LIMITS ON NUMBERS
75 REM  P=95% CONFIDENCE LIMITS ON VIABILITY
76 IF N=2 THEN 81
77 IF N=3 THEN 82
78 IF N=4 THEN 83
79 IF N=5 THEN 84
80 IF N=6 THEN 85
81 FOR I=1 TO 3:L(I)=T(1)*(0.5*SQR(A(I))/SQR(N)):NEXT I:GOTO 86
82 FOR I=1 TO 3:L(I)=T(2)*(0.5*SQR(A(I))/SQR(N)):NEXT I:GOTO 86
83 FOR I=1 TO 3:L(I)=T(3)*(0.5*SQR(A(I))/SQR(N)):NEXT I:GOTO 86
84 FOR I=1 TO 3:L(I)=T(4)*(0.5*SQR(A(I))/SQR(N)):NEXT I:GOTO 86
85 FOR I=1 TO 3:L(I)=T(5)*(0.5*SQR(A(I))/SQR(N)):NEXT I:GOTO 86
86 FOR I=1 TO 3:PRINT "95% ADD. LOCI LIMIT=";0.5*A(I),L(I):NEXT
I:GOTO 91
91 FOR I=1 TO 3:Q(I)=L(I)*(1-S(I)):NEXT I
92 FOR I=1 TO 3:PRINT "95% ADD. VIABILITY LIMIT=";E(I),Q(I):NEXT I
```

```
92 FOR I=1 TO 3:PRINT "95% ADD. VIABILITY LIMIT=";E(I),Q(I):NEXT
I
96 IF N=2 THEN 101
97 IF N=3 THEN 102
98 IF N=4 THEN 103
99 IF N=5 THEN 104
100 IF N=6 THEN 105
101 FOR I=1 TO 3:L(I)=T(1)*(0.5*SQR(M(I))/SQR(N)):NEXT I:GOTO 10
6
102 FOR I=1 TO 3:L(I)=T(2)*(0.5*SQR(M(I))/SQR(N)):NEXT I:GOTO 10
6
103 FOR I=1 TO 3:L(I)=T(3)*(0.5*SQR(M(I))/SQR(N)):NEXT I:GOTO 10
6
104 FOR I=1 TO 3:L(I)=T(4)*(0.5*SQR(M(I))/SQR(N)):NEXT I:GOTO 10
6
105 FOR I=1 TO 3:L(I)=T(5)*(0.5*SQR(M(I))/SQR(N)):NEXT I:GOTO 10
6
106 FOR I=1 TO 3:PRINT "95% MULT. LOCI LIMIT=";0.5*M(I),L(I):NEX
T I:GOTO 110
110 FOR I=1 TO 3:R(I)=F(I)-(S(I)!((0.5*M(I)-L(I)))):NEXT I
111 FOR I=1 TO 3:PRINT "95% MULT. VIABILITY  LIMIT=";F(I),-R(I):
NEXT I
112 B=W-D:PRINT "WHOLE-HALF S.D.S=";B
113 FOR I=1 TO 3:X(I)=H-E(I):NEXT I
114 FOR I=1 TO 3:PRINT "ADDITIVE OBS-EXP VIABILITIES=";X(I):NEXT
I
115 FOR I=1 TO 3:Y(I)=H-F(I):PRINT "MULTIPLICATIVE OBS-EXP VIABI
LITIES=";Y(I):NEXT I
116 END
```

SAMPLE OUTPUT

```
V,H,N,D,W
V= .5           H= .83          N= 6
D= .21          W= .186         S(1)= .99       S(2)= .95
S(3)= .9
A(1)= 50        A(2)= 10        A(3)= 5
M(1)= 68.96756393598            M(2)= 13.51340733395
M(3)= 6.578813478956
E(1)= .75       E(2)= .75       E(3)= .75
F(1)= .70710678119              F(2)= .70710678119
F(3)= .70710678119
95% ADD. LOCI LIMIT= 25          3.710918855208
95% ADD. LOCI LIMIT= 5           1.659573363855
95% ADD. LOCI LIMIT= 2.5         1.173495579447
95% ADD. VIABILITY LIMIT= .75    3.71091885E-02
95% ADD. VIABILITY LIMIT= .75    8.29786681E-02
95% ADD. VIABILITY LIMIT= .75    .1173495579447
95% MULT. LOCI LIMIT= 34.48378196799            4.358317839112
95% MULT. LOCI LIMIT= 6.756703666975            1.929207269492
95% MULT. LOCI LIMIT= 3.289406739478            1.346078050393
95% MULT. VIABILITY  LIMIT= .70710678119        3.16614499E-02
95% MULT. VIABILITY  LIMIT= .70710678119        7.35511651E-02
95% MULT. VIABILITY  LIMIT= .70710678119        .10774413062
WHOLE-HALF S.D.S=-2.40000000E-02
ADDITIVE OBS-EXP VIABILITIES= 8.00000000E-02
ADDITIVE OBS-EXP VIABILITIES= 8.00000000E-02
ADDITIVE OBS-EXP VIABILITIES= 8.00000000E-02
MULTIPLICATIVE OBS-EXP VIABILITIES= .12289321881
MULTIPLICATIVE OBS-EXP VIABILITIES= .12289321881
MULTIPLICATIVE OBS-EXP VIABILITIES= .12289321881
```