SEX-LINKED INHERITANCE OF A QUANTITATIVE TRAIT OF DROSOPHILA PSEUDOBOSCURA

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The analyses of genetic variations for most quantitative traits in diploid animals have often been made on the assumption that a majority of genes governing such traits are located on autosomal chromosomes. In a few instances, however, it has been found that the sex chromosomes might carry a considerable portion of the genes for a quantitative trait (e.g. chaetae number of D. melanogaster studied by MATHER 1942).

The basic principle for determining the existence of sex-linked genes for a certain trait is to investigate the pattern of variation associated with the transmission of sex chromosomes. Sometimes the investigation can be much facilitated by the use of tester stocks with appropriate markers. Unfortunately, however, such tester stocks are not readily available for the quantitative genetics studies with most organisms. The objective of this paper is to report the results of three different experiments which all point to the existence of sex-linked genes governing a major portion of genotypic variation of a quantitative trait in a population of Drosophila.

MATERIALS

The basic material was a random mating population of Drosophila pseudoobscura. This population descended from about a dozen impregnated females captured by PROFESSOR TH. DOBZHANSKY and his associates at Mather, California. These iso-female lines were intermixed, and have been kept in a standard plastic population cage at a constant temperature of 19°C for a number of generations. At the start of this study, the population was considered to have established an equilibrium with respect to its genetic structure. This population will be referred to as the original population (O).

From population O, a series of inbred lines were extracted without artificial selection by 12 or 13 generations of full-sib matings. Since then each line has been carried by mass culture of approximately ten pairs of adults in a one-half

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pint milk bottle. By this procedure 15 inbred lines were established; they will be called the inbred population (I). The actual inbreeding was not complete \( (F<1) \) in population I. However, it will be considered to be completely inbred for ease of description and discussion.

A new random mating population was synthesized from population I, by using each line as a male six times and as a female six times. This formed a balanced array of 90 of the possible 210 \( F_1 \) crosses. The offspring from the 90 crosses were placed in a cage to maintain the new population. This will be referred to as the synthesized population (S).

Thus, there were three populations, the last two of which were sequentially derived from the first. They are considered to represent three different phases of the same population, if the inbreeding was carried out truly at random with respect to the character to be examined.

All experimental flies were kept at 19°C, and were cultured on a cream-of-wheat medium \( (\text{DEMEREC and KAUFMANN 1957}) \).

The character studied was wing length, represented by the length of the third longitudinal vein marked by the asterisks in Figure 1. Both wings of each indi-

![Figure 1: Drosophila wing. The length between the asterisks is measured for representing wing length.](image)

vidual were cut off at their base, mounted on glass slides, and measured with an ocular micrometer. The calibration rate will be given for each experiment.

Three experiments and the findings from these experiments will be described in the order in which they were conducted, and a general discussion and joint interpretation of the results will be given later.

**EXPERIMENT I**

The design of Experiment I permitted a comparison of the magnitudes of genetic variance in each of the three populations. The methods used for the estimation of the genetic variances were an analysis of variance of full-sib progenies nested in half-sib groups (nested design) for populations O and S, and an analysis of diallel cross progenies for population I.

The nested design was set up by mating each male parent to three female parents, all taken at random from the respective population. Out of 60 such matings, 47 successful groups for O and 56 for S were obtained. After mating, the individual female parents were put into separate culture bottles.

The diallel design was made by crossing each of the 15 inbred lines to all others,
including reciprocal crosses. In this case, five female parents from each line, impregnated by three males from another line, were put together in each culture bottle. It was thought that this scheme would yield a larval density equivalent to that existing in the nested design of non-inbred flies, since the inbreds were less viable, less fertile and less fecund. Thus, “full-sib families” in the diallel design were not full-sib families in a strict sense, but were made up by F₁ individuals between two lines.

In all cases of Experiment I, five female progeny from each full-sib family were collected at the peak period of adult eclosion, and their wing length was recorded. Thus, the data of Experiment I were all on diploids with respect to both sex and autosomal chromosomes.

The results of analyses of the three populations are presented in Table 1. The

<table>
<thead>
<tr>
<th>Population</th>
<th>O</th>
<th>I</th>
<th>S</th>
</tr>
</thead>
<tbody>
<tr>
<td>σ²_q</td>
<td>1.6749 ± 0.66</td>
<td>2.3025 ± 0.40</td>
<td>0.4382 ± 0.06</td>
</tr>
<tr>
<td>σ²_r</td>
<td>1.8667 ± 0.92</td>
<td>1.6306 ± 0.80</td>
<td></td>
</tr>
<tr>
<td>σ²_f</td>
<td>5.7564 ± 1.08</td>
<td>4.8554 ± 0.94</td>
<td></td>
</tr>
<tr>
<td>σ²_o</td>
<td>4.9808 ± 0.32</td>
<td>4.1110 ± 0.19</td>
<td>6.3151 ± 0.37</td>
</tr>
<tr>
<td>σ²_p</td>
<td>0.3194 ± 0.00</td>
<td>0.3493 ± 0.00</td>
<td>0.4572 ± 0.02</td>
</tr>
</tbody>
</table>

Mean length in micrometer units: 108.47, 95.48, 105.95
Mean length in millimeter units: 2.1802, 1.9191, 2.1296

A diallel cross is used for I, and a nested crossing scheme is employed for O and S. All components are measured in micrometer units. For the identification of components, see the text.

components of variance for each population were obtained by equating the mean squares to their expectations in terms of the variance components. For the details of the computational procedure, reference should be made to books on statistical genetics (e.g. KEMPThORNE 1957; LERNER 1958; FALCONER 1960). The identification of the components of variance is as follows:

σ²_q: Variance of general combining ability in the diallel design. It is the component of variance associated with the marginal means of individual lines (half-sib means).

σ²_r: Variance of specific combining ability in the diallel design. It is the component of variance associated with the deviations of individual full-sib means from the expectations based on the marginal means of the parental lines.

σ²_p: Variance due to the differences between a line entering as a paternal or as
a maternal parent in the diallel cross. It is the component of variance associated 
with so called "maternal effects."

\( \sigma_r^2 \): Variance of reciprocal cross cultures in the diallel design. In this case it is 
the component of variance between the replicated cultures, since reciprocal 
crosses in this design have identical genotypes, and the component is adjusted 
for maternal effects.

\( \sigma_m^2 \): Variance due to genetic differences of male parents in the nested design. It is the component of variance among half-sib means.

\( \sigma_f^2 \): Variance due to genetic differences of female parents mated to the same 
male parents in the nested design. It is the component of variance among full-sib 
means within half-sib groups.

\( \sigma_o^2 \): Variance due to differences among individual offspring in full-sib families. 
In the diallel design, it is the component of variance due to environmental effects 
on individual offspring. In the nested design, it is the component jointly due to 
the effects of gene segregation within full-sib families and to environmental 
effects.

\( \sigma_w^2 \): Variance due to the differences between pairs of wings of single individuals. 
It is a measure of wing symmetry of individual flies and includes measuremental 
errors.

The last two rows in Table 1 contain the mean wing length for each test. The 
low value for population I probably indicates that the larval density in the diallel 
design was slightly greater than in the nested design as a result of five-female 
bottle culture. The calibration rate in this experiment is 99.5 units = 2mm.

The estimates of the corresponding components are approximately equal in the 
O and S populations. The estimates of \( \sigma_o^2 \) and those of \( \sigma_m^2 \) do differ significantly 
between O and S, when compared with the estimates of the corresponding stand-
ard errors. However, it is reasonably certain, in the view of the closeness of \( \sigma_m^2 \) 
and \( \sigma_f^2 \) for O and S, that the amount of genetic variation is quite similar in the 
two populations. This indicates that the process of inbreeding which separated 
the two was random with respect to wing length.

In such a situation, various components of variance in the diallel design of 
population I are expected to be comparable with those obtained from the two 
nested designs O and S. The estimate of \( \sigma_w^2 \) in the diallel is of the same magnitude 
as the estimates from the two others.

In the following it will be assumed that the genes at different loci do not inter-
act with each other (no epistasis). The component \( \sigma_o^2 \) in the nested design from 
a random mating diploid population is expected to contain one half the additive 
genetic variance, three-fourths the dominance variance and the environmental 
variance, while the same component in the diallel includes only the environ-
mental variance. The estimates agree roughly with such an expectation, but those 
from the nested designs (particularly the one from the original population) are 
too small in comparison to the estimate from the diallel.

The component \( \sigma_r^2 \) in a random mating diploid population contains one fourth 
the additive variance \((\sigma_A^2)\), one fourth the dominance variance \((\sigma_D^2)\) and the
variance between culture bottles. The magnitude of the between-bottle variance may be estimated by the component $\sigma_i^2$ in the diallel. Pooling the estimates over O and S, this yields,

$$\sigma_i^2 - \sigma_r^2 = (1/4) \sigma_A^2 + (1/4) \sigma_D^2 \approx (5.7564 + 4.8554)/2 - 2.9913 = 2.3146 \quad (1)$$

The component $\sigma_m^2$ under the usual assumptions includes one fourth the additive variance. Thus, pooling over the two populations,

$$\sigma_m^2 = (1/4) \sigma_A^2 \approx (1.8667 + 1.6306)/2 = 1.7486 \pm 0.61 \quad (2)$$

Hence, the estimates of genotypic components of variance will be

$$\sigma_A^2 \approx 6.9944 \pm 2.44 \text{ and } \sigma_D^2 \approx 2.2640 \pm 4.38$$

for the original and synthesized populations.

In the diallel design of population I, $\sigma_A^2$ and $\sigma_D^2$ contain $(1+F)/4$ times the additive variance and $[(1+F)/2]^2$ times the dominance variance, respectively. Since the value of the inbreeding coefficient $F$, is about 0.9 for population I, the estimates of the genotypic components of variance become

$$\sigma_A^2 \approx 3.5261 \pm 1.39 \text{ and } \sigma_D^2 \approx 2.5712 \pm 0.44 \quad (3)$$

The estimate of dominance variance is nearly the same in the two designs, but that of the additive genetic variance appears to be different (the difference between the two estimates of $\sigma_A^2$ is 3.4683 $\pm$ 1.99, which indicates statistical significance at 10 percent level).

Since the original and synthesized populations had approximately the same value for $\sigma_m^2$, the discrepancy between the results of the nested and diallel designs does not seem to be of reasonable magnitude in comparison to the size of the standard errors unless some basic assumptions involved in the interpretation of the genetic variability are at fault. One of the basic assumptions used in the computation of genotypic variances was that all genes for the character are on autosomal chromosomes. If, however, some genes are, in fact, located on sex chromosomes, an apparent inflation of the additive genetic variance is expected to occur in the estimate from the nested design. This point is illustrated in Table 2, which describes half-sib families with either autosomal or sex-chromosomal inheritance. The letters in the table represent either genes or chromosome segments. The top of the table shows the parental genotypes when a male is mated to three random females. The middle portion represents the female progeny arising from these matings, and gives the average effect of this half-sib family, i.e., the breeding value of the male parent.

In autosomal inheritance, the variance of half-sib averages, which gives rise to $\sigma_m^2$, is seen to be based upon the average effect of two genes (or chromosome segments) from the male. In sex-linked inheritance, however, the effect is of a single gene (or chromosome segment). The variance of single genes is expected to be larger than that of the averages of two gene effects. If autosomal and sex-chromosomal genes have effects of the same magnitude in the female, then the component of variance found in sex-linked inheritance is expected to be twice the component from autosomal inheritance.
This is one of the possible explanations for the apparent discrepancy in the estimates of $\sigma_m^2$. In order to provide more information on this point, Experiments II and III were conducted.

**EXPERIMENT II**

Males receive their X chromosomes from their maternal parent, but not from their paternal parent. On the other hand, the transmission of autosomes follows the same pattern for males as for females. The situation is contrasted in the lower part of Table 2. The breeding value of a male parent for male progeny is again

| Table 2 |

**Autosomal vs sex-chromosomal inheritance**

<table>
<thead>
<tr>
<th>Male parent</th>
<th>Autosomal inheritance</th>
<th>Sex-chromosomal inheritance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Female parents</td>
<td>CD EF GH</td>
<td>CD EF GH</td>
</tr>
<tr>
<td>Male progeny</td>
<td>AC AE AG</td>
<td>AC AE AG</td>
</tr>
<tr>
<td>Female progeny</td>
<td>BC BE BG</td>
<td>AD AF AH</td>
</tr>
<tr>
<td>Half-sib average (females)</td>
<td>$1/2 (\overline{A} + \overline{B})$</td>
<td>$\overline{A}$</td>
</tr>
<tr>
<td>Male progeny</td>
<td>Same as ♀ ♀ progeny</td>
<td>CY EY GY</td>
</tr>
<tr>
<td>Half-sib average (males)</td>
<td>$1/2 (\overline{A} + \overline{B})$</td>
<td>$\overline{Y}$</td>
</tr>
</tbody>
</table>

Capital letters other than Y represent individual chromosome segments or genes. The letter "Y" represents a Y chromosome. See the text for the details.

the average of two genes or chromosome segment values for autosomes, while for sex chromosomes the breeding value is determined only by the Y chromosome. If Y chromosomes are truly inert, there will be no contribution from the sex chromosomes to the value of $\sigma_m^2$ from male progenies of the nested design. Hence, it follows that the estimate of $\sigma_m^2$ from male progenies must be small for a trait which is strongly sex-linked.

From the same crosses used for the nested analysis of population S, five male progeny were taken from each full-sib family, and their wings were mounted on slides by the same procedure used for females. The calibration rate was the same as before. The estimates of the components of variance are given in Table 3.

From a biological standpoint there is no reason to assume that the magnitudes of these components are quantitatively comparable with those obtained from female progeny data. However, it is noted that the $\sigma_m^2$ estimate for females is significantly different from zero at 5 percent level, while that for males is not. The relative magnitudes such $\sigma_m^2/\sigma_f^2$ may also be compared for the two sexes. It is obvious from the data in Table 3 that the value $\sigma_m^2$ relative to all other compo-
A comparison of components of variance for female and male wing length in the synthesized population

<table>
<thead>
<tr>
<th></th>
<th>Females</th>
<th>Males</th>
</tr>
</thead>
<tbody>
<tr>
<td>$\sigma^2$</td>
<td>1.6306 ± 0.80</td>
<td>0.2514 ± 0.57</td>
</tr>
<tr>
<td>$\sigma_m^2$</td>
<td>4.8554 ± 0.94</td>
<td>4.9214 ± 0.89</td>
</tr>
<tr>
<td>$\sigma_f^2$</td>
<td>6.3151 ± 0.37</td>
<td>4.5584 ± 0.27</td>
</tr>
<tr>
<td>$\sigma_a^2$</td>
<td>0.4572 ± 0.02</td>
<td>0.3790 ± 0.01</td>
</tr>
<tr>
<td>Mean length in micrometer units</td>
<td>105.95</td>
<td>97.34</td>
</tr>
<tr>
<td>Mean length in millimeter units</td>
<td>2.1296</td>
<td>1.9565</td>
</tr>
</tbody>
</table>

Components in male progeny is essentially nil in comparison to that found in female progeny.

The female component $\sigma_f^2$, includes the variation due to the differences of sex-linked genes transmitted from different female parents mated to the same male parents, the variance arising from the autosomal chromosome differences, and the bottle-to-bottle environmental variance. The component $\sigma_a^2$ contains the environmental variance on individuals and a fraction of genotypic variance due to gene segregation in female parents. Their magnitudes are much larger than the value of $\sigma_m^2$, as expected. Thus, the evidence from the analysis of Experiment II also supports the hypothesis of sex-linked inheritance of the character.

**EXPERIMENT III**

Approximately one year after Experiments I and II, another experiment was set up from population I in order to obtain further evidence on this subject. Experimental conditions were not exactly the same as those of the two previous experiments.

For this experiment twelve inbred lines, which were still available from the lines in population I, were taken, and they were arbitrarily divided into two groups of six lines. The two groups were labeled A and B. Males from each line in group A were mated to females from each line in group B in a $6 \times 6$ factorial design. These 36 crosses were called set AB. Reciprocal crosses of set AB were also made and called set BA.

By a procedure similar to the one used for the diallel design, flies were reared, and wing length was measured on five male progeny and five female progeny of each cross in both sets. The analysis of data was conducted separately for each sex in each set using the usual analysis of a factorial design. The components of variance obtained are listed in Table 4. The top four are the components of variance associated with the differences among lines used as male parents or as female parents. These components are similar to $\sigma_f^2$ of Experiment I in their content, but different from $\sigma_m^2$ in that paternal and maternal half-sib families are treated separately for the components in this case. The subscripts of these components stand for the sexes of parents and the groups of lines, A and B.
All four estimates for female progenies are larger than the value of $\sigma^2$ in Experiment I. This may be accounted for partly by the difference of calibration rates (122 units = 2 mm in Experiment III, and partly by the sampling of lines used for Experiment III. In the female data, the difference due to the sex of the parent does not exist; or, at least, it is much smaller than the difference due to the arbitrary grouping of the twelve parental lines into two groups, A and B.

The components estimated from the male progeny exhibit an entirely different pattern. Here the size of the components due to differences between parents appear to be mainly determined by the sex of the parents. This is consistent with the hypothesis of sex-linked inheritance. Male paternal half-sibs do not receive an X chromosome from their male parent, while all maternal half-sib males receive an identical X chromosome from their female parent. Thus, the variance associated with marginal means of male parents is expected to be small and that of female parent to be larger under the assumption of sex-linked inheritance.

A valid statistical test of significance for the comparison, $(\sigma^2_{A_d} + \sigma^2_{B_d})$ vs $(\sigma^2_{A_f} + \sigma^2_{B_f})$, for the progeny of either sex can be made by using the F-ratio in which the denominator is the sum of the two mean squares corresponding to $\sigma^2_{A_d}$ and $\sigma^2_{B_d}$, and the numerator is the sum of the two mean squares corresponding to $\sigma^2_{A_f}$ and $\sigma^2_{B_f}$. Under the null-hypothesis of no sex-linked inheritance, the mean squares for the marginals of A δ and A θ have the same expectations. The same is true for the B δ and B θ mean squares. It is then seen that this F-ratio pools the information from the AB and BA sets. If the null hypothesis is false (i.e., there is some degree of sex-linked inheritance), the numerator of the F-ratio will tend to be larger than the denominator. In the male progeny, this F-ratio is 2.50 with ten degrees of freedom for the denominator and the numerator, indicating that the comparison is significant at $5\% < P < 10\%$. The F-ratio in the female progeny is 1.02 with the same numbers of degrees of freedom as in the male progeny, yielding $P = 50$ percent. Although the statistical significance in the male progeny is not extremely high, the data of Experiment III confirm the hypothesis of sex-linked inheritance.

*Partition of sex-linked and autosomal variation*

The evidence from each of the three experiments was all in support of the existence of sex-linked genes controlling a great portion of genotypic variability of this character. In this section an attempt will be made to assess the variation arising from sex-linked genes as a fraction of the total genotypic variation. This will be done for females and males, separately.

*Female progeny:* The data from Experiment I will be used for this analysis. In a random mating population ($F = 0$), the correlation between additive effects of sex-linked genes is three-fourths in full-sisters and that between dominance effects is one-half. The same correlations in half-sisters are one-half and zero, respectively. This leads to the following formulae for covariances among sisters due to sex-linked genes:

\[
\begin{align*}
\text{Covariance (half-sisters)} &= \Sigma (1/2) \sigma^2_d \\
\text{Covariance (full-sisters)} &= \Sigma (3/4) \sigma^2_d + \Sigma (1/2) \sigma^2_d
\end{align*}
\]
where the summation ($\Sigma$) is taken over all sex-linked genes, and $\sigma_a^2$ and $\sigma_d^2$ are the additive and dominance variances, respectively, of individual loci.

For autosomal genes the covariances are

\[
\text{Covariance (half-sisters)} = \sum (1/4) \sigma_A^2 \\
\text{Covariance (full-sisters)} = \sum (1/2) \sigma_A^2 + \sum (1/4) \sigma_D^2
\]

(6)

(7)

as usual for diploid populations. The covariances observed in the nested design of Experiment I are the sum of covariances due to autosomal and sex-linked genes, i.e., (4) + (6) for half-sisters and (5) + (7) for full-sisters.

Let $p$ be the proportion of "genes" on the X chromosome and $(1-p)$ the proportion on the autosomes. Then the overall covariances are expressed by

\[
\text{Covariance (half-sisters)} = \frac{1+p}{4} \sigma_A^2 \\
\text{Covariance (full-sisters)} = 2 \frac{1+p}{4} \sigma_A^2 + \frac{1+p}{4} \sigma_D^2
\]

where $\sigma_A^2$ and $\sigma_D^2$ are the total additive and total dominance variances, respectively. In terms of the genetic components of variance in the nested design.

\[
\sigma_m^2 = \text{Covariance (half-sisters)} = \frac{1+p}{4} \sigma_A^2 \\
\sigma_f^2 - \sigma_r^2 = \text{Covariance (full-sisters)} - \text{Covariance (half-sisters)} = 1/4 \sigma_A^2 + \frac{1+p}{4} \sigma_D^2
\]

(8)

(9)

In order to estimate the value of $p$, the estimate of $\sigma_A^2$ from the diallel design can be substituted into (8), since this estimate is not affected by the presence of sex-linked genes. Thus, using $\sigma_m^2 = 1.7486$ from (2) and $\sigma_A^2 = 3.5261$ from (3), equation (8) yields $p = 0.98 \pm 1.04$.

This proportion can be checked by computing the value of $\sigma_D^2$ from equation (9) and comparing it with the corresponding estimate given in (3). From (1), (9), $\sigma_A^2 = 3.5261$ and $p = 0.98$, it is found that,

\[
2.3146 = (1/4) 3.5261 + (1.98/4) \sigma_D^2
\]

which gives $\sigma_D^2 = 2.905$. The estimate from the diallel design is 2.5712 as given in (3), indicating fair agreement between the two values. It is concluded, therefore, that almost all "genes" (98 percent) governing the character are located on the X chromosomes.

The reliability of the estimated $p$ depends upon the magnitude of the standard error of $p$. Unfortunately in this case, the standard error computed approximately for $p$ is very large (1.04). However, this is consistent with the earlier finding that the difference between the two estimates of $\sigma_A^2$ was not statistically significant ($P = 0.10$).

Male progeny: When sex-linked genes are considered, the definition of gene actions for males is not the same as that for females. For example, there is no dominance effect for genes on X chromosomes of male progeny. Nevertheless, a partitioning of genotypic variability into sex-chromosomal and autosomal parts is still possible on a slightly different basis from the case of female progeny.
In Experiment III, the component of variance obtained from the marginal means of male parents ($\sigma^2_u$) contains only the additive variance due to autosomal genes, since male parents do not transmit their X chromosomes to male progeny. On the other hand, $\sigma^2_q$ contains the variance due to differences among genes on the X chromosomes transmitted from female parent lines, besides the variance from autosomal genes. Thus, taking values from Table 4,

### TABLE 4

The estimates of the components of variance of wing length for female and male progenies in Experiment III (a factorial crossing design of inbred lines)

<table>
<thead>
<tr>
<th></th>
<th>Females</th>
<th>Males</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Set AB</td>
<td>Set BA</td>
</tr>
<tr>
<td>$\sigma^2_\alpha$</td>
<td>3.11 ± 2.28</td>
<td>.78 ± 0.90</td>
</tr>
<tr>
<td>$\sigma^2_{B_Q}$</td>
<td>2.50 ± 1.96</td>
<td></td>
</tr>
<tr>
<td>$\sigma^2_A$</td>
<td>3.24 ± 2.49</td>
<td>1.89 ± 1.56</td>
</tr>
<tr>
<td>$\sigma^2_B$</td>
<td>2.49 ± 2.09</td>
<td>.10 ± 0.64</td>
</tr>
<tr>
<td>$\sigma^2_\beta$</td>
<td>5.13 ± 1.84</td>
<td>6.50 ± 2.28</td>
</tr>
<tr>
<td>$\sigma^2$</td>
<td>7.73 ± 0.95</td>
<td>8.22 ± 1.01</td>
</tr>
<tr>
<td>$\sigma^2_w$</td>
<td>0.86 ± 0.08</td>
<td>0.78 ± 0.08</td>
</tr>
<tr>
<td>Mean length in</td>
<td></td>
<td></td>
</tr>
<tr>
<td>micrometer units</td>
<td>127.47</td>
<td>125.42</td>
</tr>
<tr>
<td>Mean length in</td>
<td></td>
<td></td>
</tr>
<tr>
<td>millimeter units</td>
<td>2.0892</td>
<td>2.0556</td>
</tr>
</tbody>
</table>

For the identification of components, see the text.

$$ p = \frac{(3.07 + 1.89) - (0.10 + 0.78)}{(3.07 + 1.89) + (0.10 + 0.78)} = 0.70. $$

The standard error of this estimate is 0.35. This proportion is not as high as that obtained for the female progeny of Experiment I, but the difference is not statistically significant.

The estimate, $p = 0.70 ± 0.35$ is significantly different from zero. This strongly indicates the existence of sex-linked genes for the character.

### DISCUSSION

Each of the three experiments has its merits and demerits as a means of detecting sex-linked inheritance of a quantitative trait. A quantitative trait is considered to be one where the distribution of offspring phenotypes of a mating does not breakdown into discrete segregation classes, but instead shows a more continuous variation. This may result from circumstances where the proportion of nongenetic variability is rather large in comparison with that of genetic variability. These can render it impossible to identify the effect of single genes, and statistical methodology is required in the analysis of the genetic basis of such traits. The statistical methods used here are not concerned with the numbers of
genes on the X chromosome and on the autosomes, but merely estimate the proportion of the genetic variance arising from segregation of the X chromosome.

The method given in Experiment I is an analysis of data only of female progeny, which have diploid genotypes for both autosomes and sex-chromosomes. This is certainly an advantage of this approach over the other two, since critical comparisons are made strictly on the same sex, and gene effects are defined on the basis of diploidy for both sex-chromosomal and autosomal genes.

However, there are a few disadvantages of this method. Since the conclusion is based on the difference of genetic covariances among half-sibs expected from two different materials, it is necessary to show that the two materials are identical within an allowable limit of sampling when they are placed under the same mating system. An extra study is needed in order to test this point. In the present work this assurance was obtained by comparing the synthesized population with the original population in the nested design. In general, such a test may indicate the opposite result. If this is the case, the approach used in Experiment I would obviously be invalid. One may expect such circumstances when directional selection is unavoidable during the process of inbreeding.

Another drawback of this approach was pointed out to the authors by Dr. C. CLARK COKERHAM. The conclusion from this approach is based on observing the inflation of paternal half-sib covariance (male component of variance) in the nested design, over the magnitude expected from the amount of additive genetic variance found in the factorial design. However, the inflation of this kind may be caused by the lack of intrachromosomal recombination in Drosophila males, if genes on the same chromosomes exhibit epistasis of an additive type (e.g. additive $\times$ additive interactions). Thus, the observation of an inflated estimated of $\sigma^2_m$ does not necessarily support the presence of sex-linked genes.

The second method for detecting sex-linked genes (Experiment II) was the comparison of paternal half-sib covariance obtained from male progeny with that from female progeny. Thus, how critical the result is hinges on the similarity of gene actions in the two sexes. If the similarity is good, this approach is a valid one. Otherwise, the conclusion is confounded with sex differences. The advantage of this method over the first is that the males and females tested can be obtained from the same crosses made from members of the original population, eliminating need of developing inbred lines.

The third approach (Experiment III) utilizes the half-sib covariances among male progeny and among female progeny. However, the type of confounding that may make the second approach invalid does not always occur in this case. The quantities compared are the difference between maternal and paternal half-sib covariances in male progeny and the same difference in female progeny. Dissimilarities that may exist in the gene actions of autosomal genes in the two sexes, do not come into the comparison as directly as in the case of the second method. The difference, $(\sigma^2_m - \sigma^2_f)$, in female progeny reflects mainly the errors of sampling in dividing the lines into maternal and paternal parents. The same difference in male progeny includes the variance due to X chromosomes minus the variance due to Y chromosomes, and the variance due to the errors of sampling.
Thus, the difference is expected to be large in male progeny, when X chromosomes contribute a substantial variation to the total. In this sense, the third method is superior to the second.

The weakness of the third method becomes obvious when the information is desired about the original population from which the inbreds were derived. Here again it is necessary to establish whether the inbred lines represent the original population within an allowable limit.

Finally, the fact that some of the results in this paper did not give statistically significant conclusions is probably due to the limited sizes of the experiments rather than to a low proportion of sex-linked inheritance. For example, the number of degrees of freedom for the variances among half-sib means in Experiment III was only five for each set. Even when pooled over two sets, the number of degrees of freedom was only ten for the critical comparison. In this connection it was unfortunate that male data were not collected from the diallel cross in Experiment I. There the comparison similar to the one made in Experiment III would have had 15 degrees of freedom for each marginal mean.

Furthermore, because of the biases discussed above, no single method used is free of criticism. However, a combination of methods which indicate the same conclusion is much stronger evidence than the results of a single experiment.

Because of the combination of methods used, with all yielding an indication of sex-linked inheritance, it is felt that the above experiments, as a whole, provided definite evidence of the existence of at least partial sex-linked inheritance of the character studied.

SUMMARY

Three different experiments are described for the detection of partly sex-linked inheritance of a quantitative trait. The population used was a panmictic cage population of *D. pseudoobscura* which had been kept under laboratory conditions for a long period of time, and the trait analyzed was wing length.

The partitioning of the total genotypic variation indicated that a majority of the genetic variation of this trait was caused by sex-linked genes.

LITERATURE CITED


