Evidence for Balanced Linkage of X Chromosome Polygenes in a Natural Population of Drosophila

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ABSTRACT

Extensive levels of polygenic variation can be maintained in a population without creating a severe segregational load. One way to account for this is that the alleles are arranged on a chromosome so that different regions balance each other phenotypically. To test whether this occurs in a natural population, we isolated ten Drosophila melanogaster X chromosomes and mapped regions of polygenic activity affecting sternopleural bristle number. The chromosomes fell into a small number of groups based upon the similarity of their distributions of polygenic activity. The results are consistent with a model in which a large proportion of the variation can be attributed to a small number of segregating chromosome regions and in which the chromosomes show internal balance.

In studies that helped establish the foundation of modern biometrical genetics, Mather (1943, 1949, 1979), Mather and Jinks (1982) and others developed models of the genetic, or "polygenic," basis of quantitative variation. According to these models, polygenic traits were dependent upon the interaction of numerous genes, whose effects were essentially interchangeable and small relative to environmental sources of variation. This variation could be stored in the gene pool and released by recombination to serve as raw material for the action of selection.

This view of quantitative variation implies that a natural population faces two competing pressures. Polygenic heterozygosity (potential variability) needs to be maintained while minimizing the segregational load (free variation). Mather's model of polygenic balance addresses this problem in terms of genetic coadaptation. The phenotypic impact of segregation could be minimized in two ways. First, the alleles at a given locus on the chromosome could show overdominance in which a balanced polymorphism maintains both "+" and "−" alleles in the population. This is called relational balance. Alternatively, balance could be produced by the compensating effects of polygenic alleles arranged along the chromosome. This is called internal or epistatic balance (+ − + −).

Much of the evidence to test such models has come from whole chromosome substitution and polygene mapping studies of selection lines, comparison of isofemale lines from natural populations, and studies of selection line responses. For example, accelerated responses to selection for sternopleural bristle number have been traced to recombination between tightly linked loci in repulsion heterozygotes (Thoday, Gibson and Spickett 1964), and the segregation of different polygenic alleles can be detected in isofemale lines established from natural populations (e.g., Lee and Parsons 1968; Milkman 1970; Parsons 1980; Thompson and Hellack 1982; Thompson and Mascie-Taylor 1985; Thompson and Thoday 1979).

There is, however, little information about polygene distribution and polygenic balance in chromosomes isolated directly from natural populations. Early studies of polygenic activity on the X chromosome, for example, suggested that most effects were associated with only a few regions (Wigan 1949, and references therein), indicating that a relatively small number of polygenic loci accounted for the majority of the phenotypic variation in traits such as sternopleural bristle number. Unfortunately, these studies used wild-type strains that had been in the laboratory for many generations which makes it difficult to generalize from them to natural populations.

In this paper, we report a survey of polygene effects on sternopleural bristle number in ten X chromosomes sampled from one natural population. We find that a large proportion of the polygenic variation in bristle number can be traced to a relatively small number of segregating chromosome regions. In addition, the observed patterns of polygene linkage are consistent with a model in which internally balanced chromosomes are segregating in the natural population, although their organization appears to be more dynamic than that initially proposed by Mather.

MATERIALS AND METHODS

Male Drosophila melanogaster collected from a natural population in Noble, Oklahoma, provided the X chromosomes mapped in this study. Individual males were collected from a small fruit pile that we placed in a rural forested area near the South Canadian River. Experimental cultures
were maintained in half pint bottles at 25 ± 1° on cornmeal, molasses, and agar medium.

Each wild-caught male was mated to females from an inbred strain carrying the X balancer chromosome Base (LINDSLEY and GRELL 1967) with the recessive mutants heldout (ho, 2-4.0) and veinlet (ve, 3-0.2) marking its autosomes. From each original cross, a single heterozygous F1 female was backcrossed to Basc;ho;ve males. These females do not show hybrid dysgenesis. One +;ho;ve male was then mated back to Basc;ho;ve females to increase the homozygosity for the original marked autosomes, and homozygous strains carrying the X chromosomes from the original wild-type males were produced. Each strain therefore carries a different X chromosome isolated from the same natural population. Ten of these strains were used in the present study. No X chromosome aberrations were detected in this sample.

Mapping of the wild-type X chromosomes was accomplished by mating +;ho;ve males with females from an inbred strain carrying an X chromosome marked with yellow (y, 0.0), crossveinless (cv, 13.7), vermillion (v, 33.0) and forked (f, 56.7) and the same autosomes as the experimental lines, marked with ho and ve. F1 females were then backcrossed to y cv v f; ho; ve males. Sternoteleral bristle numbers were measured in five males of each parental and single recombinant class from each of five replicates for each strain (N = 200 males counted for each tested X chromosome). In some instances, fewer than five males were available in a recombinant class, and data were examined using analyses of variance with unequal sample sizes. Unweighted pair-group method using arithmetic averages (UPGMA) clustering was done with Biostat II Multivariate statistical software (PIMENTEL and SMITH 1985).

RESULTS

Ten individual X chromosomes were isolated from male Drosophila collected at the same time from a secluded area. Although we can not exclude the possibility that these flies came from different natural populations, they were attracted to the fruit bait over about a 2-day period and must, therefore, have at least originated from the same vicinity in the forest.

The correlation between female and male sternopleural bristle number in these lines is 0.93 (N = 25 flies of each sex in four replicates). An analysis of variance on the male data showed that, although there is significant phenotypic variation among cultures (4 replicates × 10 lines; F9,990 = 27.51, P < 0.001), the between-component of variance is significantly greater than the within-strain component (F5,990 = 14.12, P < 0.001). This between-strain variance is the focus of the remaining analyses, and for simplicity we limit our discussion to male data.

Mapping of X chromosome regions affecting sternopleural bristle number: Polygenic effects on wild-type X chromosomes were mapped in relation to a standard X chromosome marked with y cv v f. The average sternopleural bristle numbers in each noncrossover and single crossover class are given in Table 1. When the y cv v f control progeny were compared among the ten mapping experiments, there was no significant variation [for one-way analysis of variance (ANOVA), F9,235 = 1.41]. Thus, the differences ob-

served among recombinant classes can be attributed to X-linked genetic factors, rather than uncontrolled environmental variation.

The y cv v f chromosome therefore serves as an appropriate class against which each recombinant can be standardized. This also enables us to make direct comparisons among the polygenic effects in the ten tested X chromosomes. Unless otherwise specified, all of the following analyses were done on data derived by subtracting the average y cv v f sternopleural bristle number for a given strain from each of the other classes (Figure 1). The standardized effects of each recombinant class are shown in Table 2.

The ultimate goal of this study was to see whether the X chromosomes sampled from a natural population showed patterns of genetic similarity, as implied by the hypothesis that levels of quantitative variation can be significantly affected by a relatively small number of polygenic loci. To guide our evaluation of these ten chromosomes, we employed the UPGMA as a clustering technique (SNEATH and SOKAL 1973; ROHLF, KISHPAUGH and KIRK 1982; THOMPSON, HELLACK and SCHNELL 1986).

This method of cluster analysis produces a dendrogram that groups the strains on the basis of their degree of similarity, as measured by a “distance” derived from comparing the average bristle numbers in all noncrossover and crossover classes (Figure 2). This “distance” is therefore sensitive to the pattern of phenotypic variation caused by polygenic loci in the marked chromosome blocks. The more similar two strains are in the average bristle numbers associated with the various recombinant chromosomes, the more closely they will be linked together in the dendrogram.

This technique assumes that clusters exist in the data (SNEATH and SOKAL 1973), and it is very important to be aware of the limitations created by this assumption. Clustering will occur, even if there is no biological significance to the differences among strains. It is only an initial guide to the interpretation of data. In the following discussion, therefore, statistical tests are used to help evaluate the similarities and differences suggested by the clustering technique.

Six strains (7, 9, 11, 13, 17, and 23) appear to cluster together (distance = 0.16). For example, in all cases, the “++ ++” class had a lower bristle number and the y ++ class had a higher bristle number than the y cv v f control, as one would expect if there was a factor on the left end of the wild-type chromosome that reduced bristle number relative to the standard.

To confirm the similarity of these six strains, an ANOVA was carried out using the original data for all chromosomal classes. Sample sizes were unequal, with a maximum of 25 in each class (all but 15 of the 48 classes were complete). As expected, there was significant variance among strains (F5,1065 = 7.80, P <
somes show the same pattern of effect on bristle binant classes across all strains. It measures the extent these six chromosomes were compared (F35,1065 to which the marked regions of different consistency in bristle numbers among the various recombinant classes. On the other hand, is sensitive to the degree of consistency in sternopleural bristle numbers in parental and single crossover classes (mean \( \pm \text{se} \); all sample sizes = 25, except where denoted in the footnotes).

### Table 1

Male sternopleural bristle numbers in parental and single crossover classes (mean \( \pm \text{se} \); all sample sizes = 25, except where denoted in the footnotes).

<table>
<thead>
<tr>
<th>Strain</th>
<th>++ ++</th>
<th>ycv v f</th>
<th>++ ++</th>
<th>ycv + +</th>
<th>++ v f</th>
<th>ycv v +</th>
<th>++ + f</th>
</tr>
</thead>
<tbody>
<tr>
<td>3</td>
<td>21.85 ± 0.67*</td>
<td>19.70 ± 0.49*</td>
<td>21.68 ± 0.69*</td>
<td>19.50 ± 0.86*</td>
<td>22.35 ± 0.54*</td>
<td>21.21 ± 0.52*</td>
<td>21.06 ± 0.53*</td>
</tr>
<tr>
<td>7</td>
<td>19.08 ± 0.49</td>
<td>19.60 ± 0.54</td>
<td>20.51 ± 0.77</td>
<td>20.28 ± 0.49</td>
<td>21.16 ± 0.47</td>
<td>20.96 ± 0.45</td>
<td>21.40 ± 0.42</td>
</tr>
<tr>
<td>9</td>
<td>20.40 ± 0.36</td>
<td>20.64 ± 0.47</td>
<td>20.71 ± 0.50</td>
<td>20.14 ± 0.48</td>
<td>20.32 ± 0.56</td>
<td>19.65 ± 0.64</td>
<td>20.00 ± 0.51</td>
</tr>
<tr>
<td>10</td>
<td>20.92 ± 0.56</td>
<td>19.52 ± 0.33</td>
<td>20.29 ± 0.39</td>
<td>20.33 ± 0.67*</td>
<td>20.96 ± 0.52</td>
<td>19.75 ± 0.38</td>
<td>20.29 ± 0.41*</td>
</tr>
<tr>
<td>11</td>
<td>18.48 ± 0.40</td>
<td>20.04 ± 0.38</td>
<td>20.95 ± 0.52*</td>
<td>19.60 ± 0.60*</td>
<td>21.12 ± 0.57</td>
<td>18.73 ± 0.48*</td>
<td>19.96 ± 0.48*</td>
</tr>
<tr>
<td>13</td>
<td>19.48 ± 0.30</td>
<td>19.92 ± 0.41</td>
<td>20.37 ± 0.42</td>
<td>19.29 ± 0.34*</td>
<td>20.50 ± 0.33*</td>
<td>21.16 ± 0.39</td>
<td>21.28 ± 0.39</td>
</tr>
<tr>
<td>14</td>
<td>21.00 ± 0.35</td>
<td>20.68 ± 0.35</td>
<td>22.93 ± 0.76*</td>
<td>20.25 ± 0.37*</td>
<td>19.32 ± 0.40</td>
<td>19.17 ± 0.36*</td>
<td>20.17 ± 0.41*</td>
</tr>
<tr>
<td>17</td>
<td>19.40 ± 0.30</td>
<td>19.72 ± 0.41</td>
<td>21.30 ± 0.74*</td>
<td>18.41 ± 0.41*</td>
<td>20.75 ± 0.52*</td>
<td>20.05 ± 0.49*</td>
<td>21.38 ± 0.41*</td>
</tr>
<tr>
<td>21</td>
<td>19.64 ± 0.38</td>
<td>20.80 ± 0.44</td>
<td>20.35 ± 0.57*</td>
<td>19.76 ± 0.38*</td>
<td>20.76 ± 0.36</td>
<td>19.52 ± 0.49</td>
<td>20.64 ± 0.38</td>
</tr>
</tbody>
</table>

\*10, 12, 13, 14, 15, 16, 17, 18, 19, 20, 21, 22, 23, 24.

Strain 14 clustered with the first six strains at a distance of 0.30. When pooled with the first six strains, the S \( \times \) R interaction was not quite significant (F12,1238 = 1.32, \( P = 0.08 \)), but in pairwise comparisons with each of the six strains, it was significantly different from three (S \( \times \) R in strain 14 versus 9, F7,361 = 2.6, \( P < 0.01 \); versus 13, F7,346 = 5.23, \( P < 0.01 \); and versus 23, F7,365 = 4.86, \( P < 0.001 \)). Strain 14 differed from the others in several ways, including the y + + + + recombinant class in which average bristle number was much higher.

Strains 21 and 14 (distance = 0.72) are significantly different (S \( \times \) R interaction, F7,335 = 2.23, \( P < 0.05 \)). In addition, strains 3 and 10 differ from all other strains (distance = 1.14), but they are not significantly different from each other (distance = 0.04; S \( \times \) R interaction, F7,290 = 1.45, \( P = 0.18 \)).

We therefore hypothesize that these ten X chromosomes fall into as many as four categories that differ in the number and/or magnitude of the polygenic modifiers of sternopleural bristle number that they carry. Three of these categories are relatively distinct (group 7-9-11-13-17-23, group 3-10, and 21).

Models of the polygenic composition of ten X chromosomes: Based on the observation that the ten X chromosomes could be assigned to a small number of categories, we next turned our attention to the polygenic makeup of these chromosomal classes. The largest cluster of categories we next turned our attention to the polygenic makeup of these chromosomal classes. The largest cluster of strains included 7, 9, 11, 13, 17 and 23. Pooling the phenotypic effects associated with each recombinant class in these six strains yielded an alternating pattern (Figure 3). Using computer models of the X chromosome mapping protocol, we examined the minimum number and simplest arrangement of polygenic loci that would yield the recombinational patterns we had found.

The loci in our computer simulations fit the defi-
TABLE 2

Average contributions of X chromosomal regions to sternopleural bristle number

<table>
<thead>
<tr>
<th>Strain</th>
<th>++++</th>
<th>++++</th>
<th>+++vf</th>
<th>+++vf</th>
<th>+++vf</th>
<th>+++vf</th>
</tr>
</thead>
<tbody>
<tr>
<td>3</td>
<td>2.150</td>
<td>1.984</td>
<td>-0.200</td>
<td>2.653</td>
<td>1.511</td>
<td>1.356</td>
</tr>
<tr>
<td>7</td>
<td>-0.520</td>
<td>0.708</td>
<td>0.600</td>
<td>0.560</td>
<td>0.520</td>
<td>0.240</td>
</tr>
<tr>
<td>9</td>
<td>-0.200</td>
<td>0.068</td>
<td>-0.504</td>
<td>0.520</td>
<td>-0.080</td>
<td>0.760</td>
</tr>
<tr>
<td>10</td>
<td>1.400</td>
<td>0.766</td>
<td>0.813</td>
<td>0.800</td>
<td>0.304</td>
<td>0.480</td>
</tr>
<tr>
<td>11</td>
<td>-1.560</td>
<td>0.915</td>
<td>-0.440</td>
<td>0.080</td>
<td>-1.307</td>
<td>-0.083</td>
</tr>
<tr>
<td>13</td>
<td>-0.440</td>
<td>0.448</td>
<td>-0.634</td>
<td>1.040</td>
<td>-0.170</td>
<td>0.372</td>
</tr>
<tr>
<td>14</td>
<td>0.240</td>
<td>2.249</td>
<td>-0.458</td>
<td>-0.180</td>
<td>0.480</td>
<td>0.600</td>
</tr>
<tr>
<td>17</td>
<td>-0.320</td>
<td>1.250</td>
<td>-1.308</td>
<td>-0.400</td>
<td>-0.553</td>
<td>0.454</td>
</tr>
<tr>
<td>21</td>
<td>-1.160</td>
<td>-0.467</td>
<td>-1.055</td>
<td>-0.050</td>
<td>-0.555</td>
<td>0.581</td>
</tr>
<tr>
<td>23</td>
<td>-0.320</td>
<td>0.800</td>
<td>-0.320</td>
<td>0.120</td>
<td>-1.120</td>
<td>1.000</td>
</tr>
</tbody>
</table>

The bristle number in the y cv vf standard chromosome line has been subtracted from each recombinant category, so the values above represent the mean increase or decrease in bristle number produced by each chromosome type.

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Figure 2.—Dendrogram derived from a cluster analysis based upon the results of mapping ten different X chromosomes from a natural population. The average bristle numbers in each recombinant class were standardized as shown in Figure 1.

Distance

The bristle number in the y cv vf standard chromosome line has been subtracted from each recombinant category, so the values above represent the mean increase or decrease in bristle number produced by each chromosome type.

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(e.g., \(y\) to \(cv\); \(cv\) to \(v\); and so forth). Variables included the placement of polygenic effects and their relative magnitudes and sign (increasing or decreasing the phenotype). Recombinants were generated between chromosomes using random numbers, and the overall effect of a chromosome was determined by adding the effects of all of the polygenic loci it carried. As in the actual experiments, "chromosome effects" were standardized by subtracting the value of the hypothetical \(y\) \(cv\) \(v\) \(f\) chromosome.

The simplest situation is one in which the wild-type and standard chromosomes are heterozygous at only one locus. Since our standardization procedure focuses upon the size of the difference between these two chromosomes, half of the recombinant classes must be zero (i.e., they would have the same polygenic composition as the standard) no matter where along the chromosome the gene is located (Figure 4a). Thus, the actual pattern of allelic substitutions in the chromosomes we mapped must have been more complex than this.

It is, however, possible to define linkages of two loci that yield recombinant effects similar to those we found (Figure 4b). The two heterozygous loci must be at opposite ends of the chromosome and must be linked in trans (+ − and − +). The magnitudes of the two loci must also be unequal. Since the + + + + chromosome has a lower bristle number than the \(y\) \(cv\) \(v\) \(f\) standard, the left-hand end of the standard chromosome must have a larger effect on bristle number than the right-hand end of the wild-type chromosome (Figure 4b).

These two loci will create an alternating pattern like that shown in Figure 3, but at least one additional locus of smaller effect (Figure 4c) is needed to account for the observed phenotypes associated with each recombinant genotype. Since there are so many other possible parameters (e.g., closely linked pairs of loci, recombinational distance between polygenes and flanking markers, and gene magnitude), it is inappropriate to press the computer model too far (Mc-

**DISCUSSION**

Early models of quantitative variation assumed that there were large numbers of loci that had small, interchangeable effects upon a phenotype (Mather 1943, 1979). While this assumption played a valuable role in the development of biometrical genetics, it is now clear that segregation at a relatively small number of loci often accounts for most of the phenotypic variation in a trait (Thoday 1961; Spickett 1963; Thompson 1975, 1976; Shrimpton and Robertson 1988a,b; and references in Thompson and Thoday 1974, 1979, and in Weir et al. 1988). The chromosomal distribution and magnitude of individual polygenic effects are, therefore, measurable factors that can lead to a better understanding of the control of phenotypic variation in a population.

Although most mapping of individual polygenic loci has been done on autosomes, the \(X\) chromosome offers special advantages. The most important advantage is that males are hemizygous, and dominance does not mask alleles. But the \(X\) chromosome also poses unique problems for maintaining polygenic variability in a natural population. Selection in males, for example, should reduce the effectiveness of relational balance as a mechanism for maintaining polygenic heterogeneity, since the heterozygosity that could be maintained in females will segregate and be expressed in males. Indeed, it would not be too surprising to find the \(X\) chromosome relatively silent for many quantitative traits.

In one of the few studies of \(X\)-linked polygene distribution, Wigan (1949) found that several regions of the \(X\) chromosome carry polygenes that modify sternopleural bristle number. The largest effects were localized near the left and right tips. This observation is consistent with the idea that a small number of loci can account for the majority of the phenotypic variance. Unfortunately, he used laboratory wild-type strains that had originated from widely separated
areas in England and the United States, so his results can not be generalized to a natural population.

WIGAN's mapping of polygenic activity also utilized a series of chromosomes carrying different genetic markers. Polygene mapping is based upon a comparison of the phenotypic effects associated with a genetically marked chromosome (the "standard") and a wild-type chromosome. Wild-type segments can be substituted into the standard chromosome by recombination, and the level of phenotypic expression associated with each chromosomal region can be measured. It is a technique that focuses upon differences. If the wild-type and the standard chromosome carry the same alleles at a locus, the gene will not be detected. Similarly, if several different standards are used, the genetic differences among them add an extra source of background variation to the assay. The fact that WIGAN succeeded in identifying regional differences, in spite of the potential "noise" in his assay, is further evidence that polygenic modifiers of sternopleural bristle number are far from being uniform in magnitude or distribution along the chromosome.

As noted earlier, the goal of our study was to test whether X chromosomes sampled at the same time from a natural population showed patterns of genetic similarity, as would be expected if a small number of loci affecting bristle number were segregating in that population. To minimize uncontrolled variation, we employed one standard chromosome, y ÇJ, in all of our experiments. The autosomes were identical in all strains. The phenotypic differences among the ten wild-type strains are, therefore, due to polygenic differences in the X chromosomes they carry.

Clustering techniques and analyses of variance showed that some X chromosomes had similar patterns of polygenic activity. Although it is not possible to conclude that the same polygenic alleles were present in each case, the number of different chromosome "types" is small.

There are, however, two predictions from the classical internal balance model that are not substantiated by these data. The results do not provide evidence for a simple, alternating arrangement of complementary effects (+ - + - + -) along the length of the chromosome as implied by MATHER's original model of polygene organization. Furthermore, the population should be segregating for both types of complementary chromosomes in equal frequencies (that is, + - + - + - should be as common as - + - + - +).
Although a sample of ten chromosomes is too small to measure frequencies accurately, none of the collected chromosomes were complementary to the most common type. The pattern of polygene effects seen in the most common type of chromosome (the group of six) could be simulated by a model involving polygenes concentrated in only two or three regions. The gene effects were not equal, but the regions on the two ends of the chromosome complemented each other by having opposite effects upon the trait. Indeed, this is the kind of internal balance one might expect, since polygenes are not simply small, phenotypically interchangeable factors, as assumed in the classical model.

Experimental studies have shown that the phenotypic effects of individual polygenic loci are not equal (Thompson and Tодay 1979; Barton and Turelli 1989), so two or more regions of smaller effect may be needed to compensate for a larger one. In addition, the magnitude of gene expression is affected by environmental variables, such as temperature (Gupta and Lewontin 1982; Schnee and Thompson 1984a, b; Barton and Turelli 1989). Thus, a given combination of loci should not be expected to balance effectively under the complete range of conditions that an organism experiences in nature. In other words, the process of establishing and maintaining internally balanced polygenic effects in a chromosome will probably be a very dynamic one. This sample of chromosomes shows, however, that the effects of the process may still be detectable in a natural population.

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LITERATURE CITED


