THE DISTRIBUTION OF POLYGENIC ACTIVITY ON THE X CHROMOSOME OF *DROSOPHILA MELANOGASTER*

L. G. WIGAN
John Innes Horticultural Institution, Merton, London
Department of Genetics, Birmingham University

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INTRODUCTION

THE theory of polygenic balance supposes the expression of a quantitative character to be under the control of a number, probably a large number, of genes with small similar and supplementary effects. Within an established mating-group the polygenes must be balanced in such a way that the chromosomes coming together in any zygote will generally produce a phenotype approximating to the mode of the population, which will itself approximate to the optimum under the existing environmental conditions. As recombination occurs in every generation new arrangements of polygenes must continually arise ; the great majority of these will fit into the existing balance system, but some will not do so, they will be unbalanced and will provide the variation upon which natural selection can act, and the favoured recombinants will be those which tend to maintain the population near the ever-moving optimum point. Evolutionary success depends upon the release of variability at an appropriate rate, a process which itself depends in part upon the linkage relations of the polygenes (Mather, 1941, 1942, 1943).

It is on these grounds important to find out how the polygenes are arranged in relation to the chromosome length and to the frequency of chiasmata along it. Some indications may be drawn from previous work. Payne (1918) on testing a line selected for high number of scutellar bristles located part of the activity on the X chromosome near and perhaps to the right of the *w*^{e} locus. Mather (1941) selecting for abdominal chaeta number in *D. melanogaster* in a cross between *BB* and + found a correlation between *BB* and high chaeta number in F_2 and subsequent selected generations, although the connection had been the reverse in the parental lines. The same author (1942) studied chromosomes I and III from the selected lines more intensively. In the sex chromosomes the extreme left end was not followed and in the remaining portion no undoubted differences of activity were found, but there was strong indication that some of the lines tested differed in the region near *cv*. In the third chromosome differences were found in the *th* region, situated near the centromere. Sismanidis (1942) in lines selected for high number of scutellar bristles found that from a cross using *y w* and *B* stocks the latter was eliminated

in favour of the wild type, whereas both *y w* and the wild type left end persisted, so providing some evidence of activity at the right end of chromosome I.

To summarise ; indications of polygenic activity have been found on the X chromosome at the left end near *w* (Payne) and *cv* (Mather) and also in the region of *B* (Mather, Sismanidis), on the third chromosome near the centromere (Mather), and finally Mather (1944) has demonstrated the activity of the heterochromatic Y, and of the right end of the X.

The demonstration of polygenic activity, like that of all other genetic activity, depends upon finding differences and most of the experiments mentioned above compared only a few chromosomes, or chromosomes derived from only a single original cross, so that the chance of finding differences was less than might otherwise be the case. In the experiments reported here a survey of the X chromosome has been attempted using a greater number of chromosomes of many different origins.

STOCKS AND METHOD

Nine laboratory stocks of wild type *Drosophila melanogaster* were used : Amherst, Ealing, Ockley, Rothamsted, Samarkand, Sutton Bonnington and Wellington which had been kept in mass culture in this laboratory for at least a year, Florida-4, an originally homozygous stock similarly kept, and Oregon, brother-sister mated for over 170 generations. The activity of homologous regions of wild type chromosomes cannot be compared directly, but each can be compared with a standard marked chromosome and so with each other.

The following tester chromosomes were used :—

1. y^{39e} (0·0) ct^{6} (20·0) wy^{40a} (41·9) *B* (57·0).
2L. *B car* (62·5) *B* from tester 1.
2M. *B car B* from unrelated stock.
3. ct^{6} *v* (33·0) g^{2} (44·4).
4. *y. ec* (5·5).

The marked males were back-crossed to $\widehat{XX}$ *f* flies with Oregon autosomes for at least 6 generations before use and were maintained by back-crossing throughout the experiments. For use, the marked males were crossed to *y Hw dl-49* m^{2} g^{4} females, also with Oregon autosomes and the inversion heterozygotes crossed to a single male of the wild stock to be investigated. Daughters without *Hw* were mated in pairs to an excess of Oregon males and allowed to lay for two two-day transfers in half pint milk bottles. The eggs were layed and offspring emerged at a temperature of 25° C. The quantitative character chosen for investigation was the number of sternopleural chaetæ on both sides of the fly.

A female heterozygous for a wild type X chromosome and one

with 4 marker genes will (if multiple recombination is ignored) give 8 types of sons, and one with two markers 4 types. For example *B car*/+ will give the types of sons shown in table 1.

TABLE 1

Recombination in region	Phenotypes	Designation
0	+ +	a
	B car	b
1	+ *car*	c
	B +	d

Using the designation in column 3 table 1. **a** differs from **c** in the region of *car* which, in one is derived from the wild type chromosome, and in the other from the tester, while **d** differs from **b** in the same way ; therefore these two pairs of differences each provides an estimate of the relative activity of the chromosome region associated with the *car* locus.

The method of differencing used (Mather, 1942) is given in some detail in tables 2*a* and 2*b*. The sternopleural chaeta number was determined for all classes on a maximum of 10 males from each bottle ; if any class was entirely unrepresented the bottle was discarded. In table 2*a* the means of these determinations and the

TABLE 2*a*

Method of differencing

Column 1 Class	Mean bristle number of classes				Differences of class means			
	2 a (++)	3 b (*B car*)	4 c (+ *car*)	5 d (*B* +)	6 a−c	7 Weight	8 d−b	9 Weight
Bottle 1*a* . .	18·60 10	18·60 10	18·50 4	18·33 3	0·10	2·86	−0·27	2·31
,, 1*b* . .	17·70 10	17·90 10	18·78 9	18·90 10	−1·08	4·74	1·00	5·00
,, 2*a* . .	19·50 10	18·20 10	18·90 10	18·50 6	0·60	5·00	0·30	3·75
,, 2*b* . .	18·40 10	17·60 10	17·29 7	18·29 7	1·11	4·12	0·69	4·12
,, 3*a* . .	19·90 10	19·20 10	17·60 10	17·75 4	2·30	5·00	−1·45	2·86
,, 3*b* . .	19·30 10	18·60 10	19·00 9	17·88 8	0·30	4·74	−0·72	4·44
,, 4*a* . .	18·00 10	18·50 10	18·67 3	17·67 3	−0·67	2·31	−0·83	2·31
,, 4*b* . .	19·20 10	19·00 10	18·67 3	18·13 8	0·53	2·31	−0·87	4·44
Sum of products					15·3486	31·08	−4·7798	29·23
Weighted mean difference					0·49		−0·16	

car region of Amherst chromosome from experiment 5A with tester 2M (*B car*)

numbers of flies on which they were based are given in columns 2 to 5 for four replications each with two transfers ; since all comparisons are within the individual bottles environmental factors are reduced to a minimum. Generally, as in this case, 8 bottles were used, but

if any had to be discarded the results were based on the remainder without correction. Columns 6 and 8 give the differences between means in columns 2 and 4 and 5 and 3 respectively. As the means compared are based on varying numbers of individuals up to 10 they must be weighted; an appropriate system is described by Mather (1942) in which the weight used is the product of the two numbers on which the means are based, divided by their sum. In the first entry in column 6 for example, the difference is between a mean of 18·60 based on 10 observations and one of 18·50 based on 4, so that the appropriate weight is $\frac{10\times4}{14} = 2{\cdot}86$, as shown in column 7. Weights similarly obtained for the **d**—**b** differences are given in column 9. To obtain the mean weighted difference, the products of the individual differences and their weights are summed and divided by the sum of the weights. In table 2*b* the weighted mean differences are set out in columns 2 and 3 for the Amherst chromosome (used as the example in table 2*a*) and the four other chromosomes included in the fifth experiment.

As explained above, each of the two differences for any chromosome are independant estimates of the activity of the part of the chromosome under investigation relative to the activity of the tester chromosome, in this case the region round *car*; therefore half their differences (given in column 4 table 2*b*) provides a measure of the variation due to the conditions of the experiment. The total sum of squares of these differences is 0·190075 with 5 degrees of freedom, but one component of this statistic may be isolated as the overall difference between the two estimates. Such a difference would be due to the interaction of loci along the chromosome, and will be found as the square of the sum of the differences, divided by their number—the familiar " correction term " of this type of analysis. Subtracting this correction term gives a corrected sum of squares of 0·069950 with 4 degrees of freedom. The significance of the difference between the estimates in columns 2 and 3 may be assessed by comparison with the corrected mean square, the square root of the variance ratio (2·62) being a **t** with 4 degrees of freedom. In this case it approaches, but does not reach the 5 per cent. level of probability and need not be regarded as significant, so that the uncorrected mean square 0·038015 with 5 degrees of freedom may be used as the estimate of error in subsequent stages of the analysis.

Column 5 is half the sum of the two estimates and its variance measures the differences between the chromosomes tested. The corrected mean square of 0·442718 gives a ratio with the error variance of 11·65, which with N_1 and N_2, 4 and 5 respectively, has a probability between 0·01 and 0·001. The correction figure of column 5 shows the overall difference between the wild type chromosomes and the tester, which is not significant. Finally, inspection of column 5 suggests that the Oregon chromosome differs from all

the rest. To test the significance of this difference the Oregon item was multiplied by 4, the number of other chromosomes in the experiment and the sum of the rest was subtracted from it. The remainder squared and divided by 20 gives a mean square with

TABLE 2*b*

Analysis of variance

Column 1	2 a−c	3 d−b	4 Half of columns 2−3	5 Mean of columns 2+3
Amherst	0·49	−0·16	0·325	0·165
Ealing	0·79	−0·84	0·025	−0·815
Ockley	−0·40	−0·60	0·100	−0·500
Oregon	1·16	0·63	0·265	0·895
Samarkand	−0·24	−0·36	0·060	−0·300

	S.S.	D.F.	M.S.	Correction term/ corrected M.S.	$t\,(=\sqrt{\text{V.R.}})$	P
Error variance (column 4)—						
Between chromosomes	0·190	5	0·038	...	...	...
Between estimates (correction term)	0·120	1	0·120	6·87	2·62	0·10-0·05
Corrected S.S.	0·070	4	0·017	...	...	...
All chromosomes (column 5)—				V.R.		
Between chromosomes	1·832	5	...	...	...	...
Between tester and chromosomes (correction term)	0·062	1	0·062	1·62	1·27	0·30-0·20
Variation between chromosomes	1·771	4	0·443	11·65	...	0·01-0·001
All chromosomes without Oregon—						
Between chromosomes	1·031	4	...	...	...	...
Between tester and chromosomes (correction term)	0·526	1	0·526	13·83	3·72	0·02-0·01
Variation between chromosomes	0·506	3	0·169	4·44	...	0·20-0·05
Difference of Oregon and rest— $\left[\frac{(4\times 0\cdot 895)-(-1\cdot 45)}{20}\right]^2$	1·265	1	1·265	33·28	5·77	0·01-0·001
Internal error variance	0·190	5	0·038	...	...	...

one degree of freedom which is significant. The analysis of the remaining 4 chromosomes was carried out in the usual way and shows that in fact the significance of the variance of the 5 chromosomes was due almost entirely to the Oregon item which differs very significantly from the other four, which do not in this analysis differ significantly among themselves.

In this example the variances are compared with the error variance derived from the same experiment, but for convenience and precision it is desirable to pool the error variances of all the experiments. Not all comparisons are independent of each other,

for instance, the determination of the *car* region is based on comparisons between wild type and *car* and *B* and *B car* while that of the *B* region uses the same figures in comparing wild type with *B* and *car* with *B car*. One group was therefore made of the error variances from the left-most region in any test and any others independant of it (that is *y* and *wy* from tests with *y ct wy B* and *B* from those with *B car*) and another from the right-most region (*ct* and *B* from tester 1 and *car* from tester 2) and the error variance from the *y* region with tester 4 was included in the left-hand and that from the *v* region with tester 3 in the right-hand group. As the bias shown by the correction figures was not significant the uncorrected error mean squares were used throughout. The two groups given in table 3 were tested for hetero-

TABLE 3

Homogeneity of error variances from separate tests

Experiment	Region	S.S.	D.F.	Experiment	Region	S.S.	D.F.
1	*y*	0·656625	9	1	*ct*	0·685700	9
1	*wy*	0·416125	9	1	*B*	0·188950	9
2	*y*	0·302675	9	2	*ct*	0·631450	9
2	*wy*	1·219375	9	2	*B*	1·015050	9
3L	*B*	0·103200	5	3L	*car*	0·183500	5
3M	*B*	0·063475	5	3M	*car*	0·063450	5
4	*B*	0·070500	3	4	*car*	0·073275	3
5A	*B*	0·195050	5	5A	*car*	0·190075	5
B	*B*	0·257975	5	B	*car*	0·322975	5
C	*B*	0·447575	5	C	*car*	0·449275	5
D	*B*	0·243875	5	D	*car*	0·222525	5
7	*y*	0·057979	6	6	*v*	0·405100	6
Overall M.S. . .		0·053792	75	Overall M.S. . .		0·059084	75
Test for homogeneity $\frac{0·105709}{0·005788} = \chi^2_{(11)}$ 18·26 P 0·10—0·05				Test for homogeneity $\frac{0·068223}{0·006982} = \chi^2_{(11)}$ 9·78 P 0·70—0·50			

geneity by the formula due to W. L. Stevens (Fabergé, 1936). The pooled error variance from the second group 0·059084 being the larger and more homogeneous was used throughout in all tests of significance, so that N_2 is always 75. Where the larger variance has only one degree of freedom the square root of the ratio is treated as a normal deviate.

RESULTS.—THE RIGHT END

In the first experiment 9 wild type chromosomes were tested with *y ct wy B*. In table 4 the analysis of the sums of the two estimates of the effects of the right end in the region round *B* based on comparisons between wild type and *B* and *y ct wy* and *y ct wy B* is given. Although when all the chromosomes are considered they show a significant

difference, this on further investigation is found to consist solely of a difference between Oregon and all the rest, which do not differ significantly amongst themselves. There is also a highly significant difference between the tester and all chromosomes except Oregon. Since this experiment had tested X chromosomes which had been back-crossed to $\widehat{XX}f$ with Oregon autosomes for at least 12 generations, it was repeated, using chromosomes straight from the stock bottles : the results of this second experiment were very similar and are given in the last three columns of table 4. Therefore the lack of variation

TABLE 4

B *region from tests with* y ct wy B

	Experiment 1				Experiment 2		
	D.F.	M.S.	V.R. or *t*	P	M.S.	V.R. or *t*	P
Between all chromosomes . .	8	0·205	3·47	0·01-0·001	0·227	3·85	0·01-0·001
Between Oregon and rest . .	1	1·413	4·89	<0·001	1·243	4·59	<0·001
Between rest without Oregon .	7	0·033	...	...	0·082	1·39	>0·20
Difference between rest and tester	1	13·847	highly	significant	11·186	highly	significant
Pooled error variance . .	75	0·059	...		...	...	

The larger overall M.S. from table 3 is used as error variance in this and all later tables

in the region round *B* cannot be ascribed to any possible effect of its association with the Y chromosome from the $\widehat{XX}$ stock.

In order to investigate the right end of the chromosome further, two *B car* testers were made up. In one (2L) the *B* locus came from tester 1 and in the other (2M) from a supposedly unrelated stock. The locus of *car* came from the same source in both testers. A single male from each of 5 stocks (including Oregon) was tested with both these testers in experiments 3L and 3M. In these tests the chromosome is divided into two, for the estimation of polygenic variability ; the proximal part, extending from a point midway between *B* and *car* includes the main heterochromatic region of the chromosome and is marked by *car*, and the essentially euchromatic distal part is marked by *B*. In view of the extensive recombination to the left of *B* the data for this region are not critical ; the right end is, however, well marked by *car*. The results of the analysis are set out in the top two lines of table 5 ; both testers give very similar results, except for their difference from the 4 chromosomes without Oregon (last three columns). This difference is clearly due to sampling error, as tester 2M was used in all subsequent experiments, in which it shows a significant difference from these chromosomes.

Experiment 4 tested the two stocks (with Oregon) which showed the maximum range of variation in experiment 3M ; this variation was not recovered, presumably because the two stocks were not inbred.

To investigate the relation of X and Y chromosomes experiment 5 was set up. Males from 5 stocks (including Oregon) were tested with 2M and the identical chromosomes were kept for 3 generations against the same back-cross stock of $\widehat{XX}$ females used to keep the tester and

TABLE 5

car *region*. *All tests with* B car *testers*

Experiment	All chromosomes				Without Oregon			
	D.F.	M.S.	V.R.	P	D.F.	M.S.	V.R.	P
3L	4	0·349	5·90	<0·001	3	0·008	...	...
3M	4	0·266	4·50	0·01-0·001	3	0·092	1·55	0·20
4	2	0·214	3·62	0·05-0·01	1	0·001	...	...
5A	4	0·443	7·49	<0·001	3	0·169	2·85	0·05-0·01
B	4	0·255	4·32	0·01-0·001	3	0·013	...	...
C	4	0·121	2·04	0·20-0·05	3	0·056	...	...
D	4	0·534	9·04	<0·001	3	0·210	3·55	0·05-0·01
Sum of 5A-D .	...	...	...		12	0·112	1·89	0·05
Part of 3 and 5 .	...	...	...		12	0·103	1·73	0·20-0·05

Experiment	Oregon item			Difference of chromosomes (without Oregon) from tester		
	M.S.	*t*	P	M.S.	*t*	P
3L	1·370	4·82	<0·001	1·227	4·56	<0·001
3M	0·788	3·65	<0·001	0·051	...	...
4	0·427	2·69	0·01-0·001	0·120	1·42	0·20-0·10
5A	1·265	4·63	<0·001	0·526	2·98	0·01-0·001
B	0·981	4·08	<0·001	0·526	2·98	0·01-0·001
C	0·315	2·31	0·05-0·02	0·893	3·89	<0·001
D	1·507	5·05	<0·001	0·570	3·11	0·01-0·001

re-tested in each generation. Any influence of the Y in this region would tend to make the wild type chromosomes more like the tester and would be reflected in a decrease in significance over the last four entries in the final columns in table 5. Clearly no such decrease occurs in the course of 3 generations, but this is not surprising as Mather (1944) found considerable variation after 5 generations of crossing to $\widehat{XX}$, and its subsequent decay was irregular.

Since the 4 chromosomes remained unchanged throughout experiments 5*a* to 5*d* the results may be summed. This is done in the one from last line of table 5 and the variance ratio just attains the 5 per cent. level of probability (1·89 with 12D.F.). A further test with 12 degrees of freedom could also be made, because chromosomes from the Amherst, Ockley and Samarkand stocks were tested both in experiment 3 and 5. Although the chromosomes tested in experiment 3 may have differed from those tested in experiment 5*a* to 5*d* (as the

stocks from which they came were mass-cultured) any difference there might have been between experiments was not significant and the mean squares for the 3 chromosomes in all 6 tests may properly be summed (last line of table 5), the ratio approaching the 5 per cent. level of probability.

The consistently significant difference between the Oregon chromosome, and the testers on the one hand, and the other 8 chromosomes tested on the other, shows the right end of the chromosome may greatly affect sternopleural bristle number. The thorough analysis of the fifth experiment (which contains the greatest amount of data) corroborated by the analysis of the three stocks tested in the third and fifth experiments shows that even over and above this rather clear cut difference there are indications of further variation in this region.

CENTRAL PORTION

The *y ct wy B* tester used in the first two experiments gave estimates for the two regions centering on *ct* and *wy* and for the region embracing both these loci. The analysis is shown in table 6. The great increase in significance of the two regions taken together suggested that there was either a centre of variation about half-way between the two loci, or, if the two loci were each associated with a separate centre of activity, these must be acting in the same direction. The possibility of a single centre of variation was tested in experiment 6 using a *ct v g* chromosome as tester. Only the *v* comparison is reliable with this tester, and it failed to reveal any significant difference in this region, as shown in the bottom line of table 6. There must then be

TABLE 6

Central region

	ct region				*wy* region				*ct wy* region			
	D.F.	M.S.	V.R.	P	D.F.	M.S.	V.R.	P	D.F.	M.S.	V.R.	P
Between chromosomes experiment 1	8	0·130	2·19	0·05-0·01	8	0·084	1·42	>0·20	8	0·246	4·17	0·001
Between chromosomes experiment 2	8	0·228	3·85	0·01-0·001	8	0·170	2·88	0·01-0·001	8	0·350	5·92	0·001
									v region			
Between chromosomes experiment 6									5	0·057	...	...

two centres of activity, near *ct* and *wy* respectively, which act in the same direction in the 9 chromosomes tested, although the action is in a plus direction in some chromosomes and in a minus direction in others. Furthermore these centres of activity are divided by a region having no polygenic activity on sternopleural bristle number.

THE LEFT END

The analysis of the *y* region in the first two experiments is given in table 7. In both experiments the variance ratio is highly significant and this region is thus clearly very active in affecting the bristle number, though the average difference is little if any greater than the difference found at the right end, between the Oregon and tester chromosomes on one hand and the other chromosomes tested on the other. In order to localise the active region more exactly experiment 7 was set up using 6 chromosomes with tester 4 (*y ec*). The region marked by *ec* was rejected for similar reasons to those which led to rejection of the *B* data with *B car* testers. The results for the *y* region are given in the last line of table 7 and show the great activity of the

TABLE 7

y *region*

Between chromosomes experiment 1 .	8	1·005	17·02	<0·001
,, ,, ,, 2 .	8	0·968	16·38	<0·001
,, ,, ,, 7 .	5	1·968	33·30	<0·001

region from *y* to midway to *ec* (0·0-2·8). This result does not preclude the existence of variation also in the remainder of the region covered in experiments 1 and 2 (from 2·8—10·0) ; indeed the variance in experiment 7, being nearly twice that in the other two experiments, might suggest some activity in this region, having an opposite sign to that in the distal part.

DISCUSSION

From the results of these experiments a rough map of the distribution of polygenes controlling sternopleural bristle number begins to emerge, for the X chromosome. Maximum activity is in the left end from 0·0-2·8 on the genetical map ; in the middle part two centres associated with *ct* and *wy* show significant activity and are divided by an inactive region, and the right end from between *B* and *car* to the centromere shows a great difference between the Oregon chromosome and the rest and, in addition, some differences between the chromosomes other than Oregon. This activity is probably underestimated in these experiments since Mather (1944), using a different technique, has shown the existence of considerable activity in the pairing segment of the X and Y and the transference of genetic material between them. Failure to detect it in the present experiments may be connected with the exceptional behaviour of the Oregon chromosome found by Mather in this region, since the right ends of testers 1 and 2L and 2M may all have been ultimately derived from the Oregon stock which is generally used for outcrossing and extraction in this laboratory. In this connection it may be noted that

the difference of Oregon from the other chromosomes is of about the same order of magnitude as the largest difference reported by Mather between the Y chromosomes which he tested.

In considering the distribution of polygenes it must be remembered that in these experiments only one character has been followed. Although the observation of correlated responses (Wigan and Mather, 1942) shows that polygenes controlling different characters may occupy the same chromosome regions, different optimum rates for the release of variability in different characters would disfavour the existence of variability of all characters in all centres of activity. It must also be remembered that these results were obtained on the X chromosome ; *Drosophila melanogaster* shows relational balance (Wigan, 1944) and the polygenic organisation of the X chromosome, which is hemizygous in one sex, and must therefore also show internal balance, may not represent the condition in the autosomes, where no such compromise is called for.

For the polygenes controlling sternopleural bristle number the distribution is not continuous, the two centres of activity associated with the loci *ct* and *wy* being separated by an inactive region round *v*. Perhaps a more minute investigation of the chromosome would reveal further subdivisions of the polygenic distribution within the rather large blocks so far tested, and indeed the result of experiment 7 suggests this to be so in the longer region associated with *y* in experiments 1 and 2. It is interesting that the whole of this long region extending from half-way between *y* and *ct* to half-way between *wy* and *B*, a total length of 39·5 units (that is more than half the cross-over length of the chromosome) should be so organised that the two active regions recognised in it have effects in the same direction. If such an arrangement should prove to be general for this region of the X it suggests that the distribution of polygenic complexes having a plus or minus effect is governed not only by the distribution of chiasma frequency and the general requirements of balance but by other undetermined factors as well.

In fig. 1 the cross-over is compared with the salivary map and with the heterochromatin in the metaphase chromosome. The left end of the chromosome, which has great polygenic variability, is also known to contain a high proportion (relative both to its genetic and salivary length) of the recorded visible mutant loci and of chemically induced lethals (Slizynska and Slizynski, 1947) ; major genes and polygenes are not therefore antithetical in their distribution. Neither are they fully correlated, for the region round *v* shows more major genic loci but less polygenic variation than does the portion of the chromosome marked in these experiments by *car*. Polygenic variation may thus be associated with regions rich in major genes but it may also occur where they are absent.

It will be seen from the diagram that the polygenic activity of the regions tested is not directly proportional to their euchromatic length.

It is also clear that activity is not proportional to the amount of heterochromatin which has also been shown by Mather (1944), who found that females with an extra Y (which is almost entirely heterochromatic) did not differ significantly from their normal sisters.

In addition to the heterochromatic block at the proximal end there are probably many intercalary segments of heterochromatin within the euchromatic portion ; the evidence for this, with maps, is given by Slizynski (1945) and Slizynska and Slizynski (1947). Of the first 20 divisions of the salivary map, covering the whole X chromosome (Bridges and Brehme, 1944) only 4 divisions are not

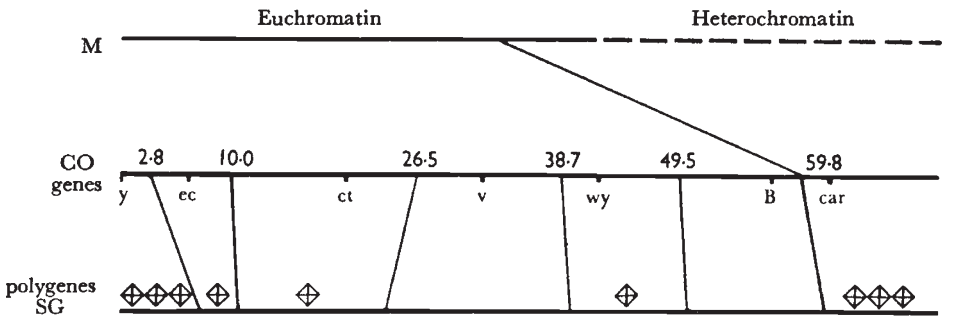

FIG. 1.—Relation of polygenic activity to length of eu- and heterochromatin. Metaphase chromosome above (after Dobzhansky, 1936). Crossing-over map centre. Salivary chromosome below (points estimated after Bridges and Brehme, 1944).

The lozenges indicate roughly the polygenic activity associated with the chromosome regions. At the extreme right end the differences were chiefly between Oregon and the other chromosomes tested, but lesser differences were also found between these latter.

reported to contain some heterochromatin. A single division of the salivary map being, on the average for the whole X chromosome, equivalent to about 3 genetical units of recombination, the regions tested probably each included several heterochromatic segments. Moreover, it has been shown (Prokofieva-Belgovskaja, 1947) that eu- and heterochromatin are not invariable in their limits even in the salivary nuclei of the same individual of *D. melanogaster*, a condition that has also been reported in *D. nebulosa* (Pavan, 1946) and *D. pallidipennis* (Dobzhansky, 1944), while Kosswig and Shengun (1947) show these limits to be variable in *Chironomus* in polytene nuclei in different tissues. It is not therefore possible from these results, to establish any causal connection between polygenic action and the presence of intercalary heterochromatin.

SUMMARY

The distribution of polygenic activity affecting the number of sternopleural bristles in *D. melanogaster* was examined in 9 X chromosomes from laboratory wild stocks.

Maximum activity was found at the left end (0·0-2·8) ; the regions round *ct* (10-31) and *wy* (31-49·5) both showed significant activity

which tended in the same direction in any one chromosome ; no activity was found near *v* (26·5-38·7). At the right end, in the region round *car* (59·7-centromere), there was a highly significant difference between the inbred Oregon chromosome and the testers on the one hand and the other 8 chromosomes tested on the other ; the other differences found in this region were significant just at the 5 per cent. level.

Polygenic activity was shown not to be directly proportional to cross-over length or to the amount of euchromatin shown on the salivary map ; nor was it proportional to the amount of heterochromatin in the metaphase chromosome. Owing to the general distribution of intercalary heterochromatin along the X chromosome no connection could be established between polygenic activity and heterochromatin.

In the chromosomes tested polygenic activity was not fully correlated with the distribution of major genic loci, but the greatest polygenic activity was associated with a region having a high concentration of visible and lethal mutations.

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