RECOMBINATION AND RESPONSE TO SELECTION IN *DROSOPHILA MELANOGASTER*

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ABSTRACT

Most biologists believe that recombination speeds response to selection for traits determined by polygenic loci. To test this hypothesis, sixteen *Drosophila melanogaster* populations were selected for positive phototaxis for twenty-one generations. In some populations, balancer chromosomes were used to suppress autosomal recombination, and in others the autosomes were free to recombine. Suppression of recombination had no effect on mean rate of response to selection, though it may have increased variability in the rate of response among replicate lines. Suppressed recombination lines did not shift selection response to the freely recombining *X* chromosomes, despite fairly large increases in *X* chromosome recombination. The results suggest that in populations of moderate size, sex does not accelerate short term response to selection.

BIOLOGISTS have acquiesced in the belief that sex accelerates evolution and that recombination among alleles at different polymorphic loci speeds response to selection, thereby facilitating evolutionary adaptation. Work by several people has converged to challenge this notion (MAYNARD SMITH 1968; ESHEL and FELDMAN 1970; ESHEL 1971; KARLIN 1973; THOMPSON 1976; WILLIAMS 1975). Sex, it now appears, may act most often to slow response to selection, to buffer populations against too rapid adaptation to shifting environments. The question is under debate. But debate to date has been limited to abstract models, bolstered to some degree by natural history observations. Relevant experiments have been scarce, inconclusive, and, worst, largely ignored. Here I present evidence that suppression of recombination in *Drosophila melanogaster* populations of moderate size has no effect on mean response to selection. By inference, sex does not accelerate evolution.

MATERIALS AND METHODS

D. melanogaster individuals always exhibit gender; they are male or female, obligate outbreeders and crossers. But the males show no crossing over and no recombination between loci on homologous chromosomes (for rare exceptions see HIRAZUMI 1971; SLATKO and HIRAZUMI 1973). Balancer chromosomes can be used to eliminate recombination in females too. Inversions and other structural rearrangements associated with balancers suppress recombination in defined selections of the genomes of balancer heterozygotes. In the work reported here, I used the complex balancer combination "A1" (WALLACE 1966; WALLACE, ZOUROS and KRIMBAS 1966) to suppress autosomal recombination in certain selected lines. The "A1" combination carries the dom-

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inant mutations *Cy*, *L*, and *Ubx* associated with rearranged and reciprocally translocated second and third chromosomes. It segregates as a unit, suppresses recombination almost completely in 75% of the total genome (see below), and it is lethal in homozygous condition.

I bred the *Cy L Ubx* rearrangement with flies decended from a wild population, so that it segregated against an otherwise wild-derived genetic background. From the resulting population, I drew individuals to initiate sixteen parallel selected lines, divided four each among four different recombination treatments (A, B, C, and D). Each recombination treatment was defined by a different repetitive cross which determined the nature of recombination in a given line (Figure 1). The C and D lines were effectively nonrecombinant for autosomal loci, while the A and B lines were free to recombine (though note that autosomal assortment was blocked in B line males). Accordingly, the C lines measure the effects of suppressing recombination, the A lines serve as "wild type" controls, and the B lines control for nonrecombinational effects associated with the *Cy L Ubx* rearrangement. The D lines were intended to measure response in the absence of selection on phenotypically "wild type" individuals. For technical reasons they proved difficult to handle and interpret, and I will omit discussing their responses in any detail.

Using mazes of HADLER's design (1964a,b) I selected each of the 16 selection lines for positive phototaxis for 21 generations. Phototaxis mazes sort flies according to their tendency to turn towards light or dark, given a succession of light-dark choices. Phototaxis maze behavior appears to be a polygenic trait, determined by many scattered polymorphic loci of small and more-or-less equal effect (HADLER 1964b; DOBZHANSKY and SPASSKY 1967; WALTON 1970; WOOLF 1972; MARKOW 1975), though the degree of polygenicity has yet to be rigorously demonstrated (see below).

Descendants of 48 wild-inseminated *D. melanogaster* females, sampled October 17, 1971, from IVES' (1970) South Amherst, Massachusetts, "Markert Site," formed the base population for these experiments. I maintained them three months in the laboratory as single-female lines prior to the first crosses. Detailed descriptions of the initial crosses, maze construction, and operating conditions during the experiments appear in THOMPSON (1974). Several points merit attention here.

The mazes presented 16 consecutive choice points to each traversing fly. I ran males and females separately as virgins. The major phenotypes ("wild type" and *Cy L Ubx*) were also run separately. Between runs, I opened and cleared mazes to avoid accidental cross-contamination of lines, and I systematically rotated lines among four mazes to randomize potential environmental effects on scores. Flies were reared and run at an average temperature of 24°C. Illumination intensity on the mazes averaged about 150 lux. I took precautions to run flies in an undisturbed state, usually for 18–24 hours, starting in the afternoon. Controls indicated that variation in run size and average age did not seriously influence mean score.

In the initial generation, selection intensity averaged about 17%. In later generations it tended to decline. All the *Cy L Ubx* rearrangements used in these experiments descended from a single male fly. Invariant marker chromosomes reduced effective population size (MCPHEE and ROBERTSON 1970). To equalize effective population size for autosomal loci, I varied the

A			B		
$X^a\ 2^a\ 3^a$		$X^a\ 2^a\ 3^a$	X^a Cy L Ubx		$X^a\ 2^a\ 3^a$
$Y^a\ 2^a\ 3^a$	×	$X^a\ 2^a\ 3^a$	$Y^a\ 2^a\ 3^a$	×	$X^a\ 2^a\ 3^a$
C			**D**		
$X^a\ 2^a\ 3^a$		X^a Cy L Ubx	X^a Cy L Ubx		X^a Cy L Ubx
$Y^a\ 2^a\ 3^a$	×	$X^a\ 2^a\ 3^a$	$Y^a\ 2^a\ 3^a$	×	$X^a\ 2^a\ 3^a$

FIGURE 1.—Repetitive crosses used to control recombination in the selection lines. The superscript "a" represents the Amherst origin of the background *X*, second, and third chromosomes. The letters A, B, C, and D signify different recombination treatments. For their significance, see text.

number of parents selected in the different recombination treatments, perpetuating each A line with 38 individuals of each sex each generation, each B and C line with 50 of each sex, and each D line with 75. Following the arguments of McPHEE and ROBERTSON (1970), effective population size for autosomal loci approximated 50 under these conditions. Run sizes ranged from about 50 to 500. In each line in each generation I selected the 38, 50 or 75 most photopositively scoring individuals of the appropriate genotype for each sex to serve as parents of the next generation. Selection and various controls required over 1000 separate maze runs involving at least 200,000 flies.

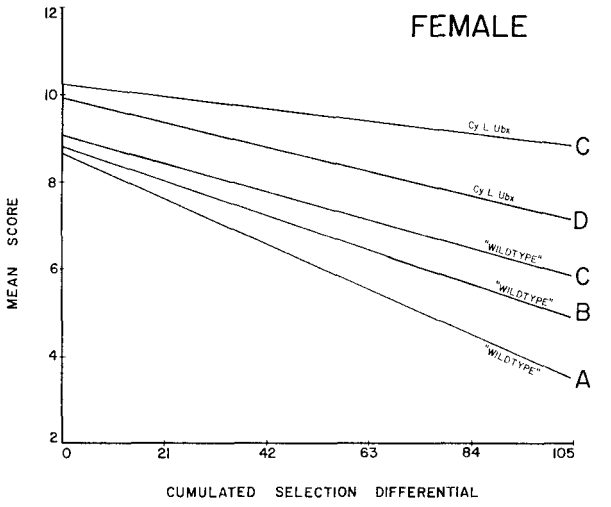
RESULTS

Most or all of the selected lines responded to selection, but the degree of response varied widely, both within and among recombination treatments, and between sexes and major phenotypes within individual lines. To facilitate comparisons among the different recombination treatments I will use "realized heritability" (h^2) as a measure of rate of response to selection. Realized heritability is the absolute value of the slope of the regression of phototaxis score on cumulated selection differential (FALCONER 1960). Cumulated selection differential represents a measure of the total selection to which a population has been exposed. It reflects selection intensity, the proportion of individuals selected in each generation, and the variability of the population from which they are drawn. By using cumulated selection differentials in conjunction with realized heritability, we can make direct comparisons among selection lines that have not been exposed to identical regimes of selection. In these experiments h^2 ranged from about .005 to .085 among the different selection lines, results comparable to those of HADLER (1964b) and MARKOW (1975).

When selection response remains directly proportional to cumulated selection differential over the course of selection, h^2 is a good measure of relative rates of response. In the work reported here this assumption held reasonably well. Phototaxis score and cumulated selection differential showed an approximately linear relationship. But two exceptions should be noted. Response was consistently greater at the beginning of selection than in later generations, and the response of the D line *Cy L Ubx* flies tended to level off in later generations. Neither exception seriously compromises the utility of h^2 as a measure of rate of response in the analysis that follows.

Figures 2 and 3 summarize response to 21 generations of selection. They illustrate the characteristic response of each recombination treatment by a line drawn with the mean slope (mean h^2) and mean score axis intercept of its four component selection lines (based on data in Tables 1 and 2). Because sex and major phenotype significantly influenced phototaxis score, responses are plotted separately by sex and by major phenotype for each recombination treatment. THOMPSON (1974) gives the detailed data on which Tables 1 and 2 are based.

Looking at Figures 2 and 3, three points stand out: (1) *Cy L Ubx* flies show less response to selection than their "wild type" counterparts. Strong evidence suggests that the eye mutation *L* (Lobe) directly suppresses photopositive maze behavior (THOMPSON 1974). (2) B and C line "wild type" flies register sub-



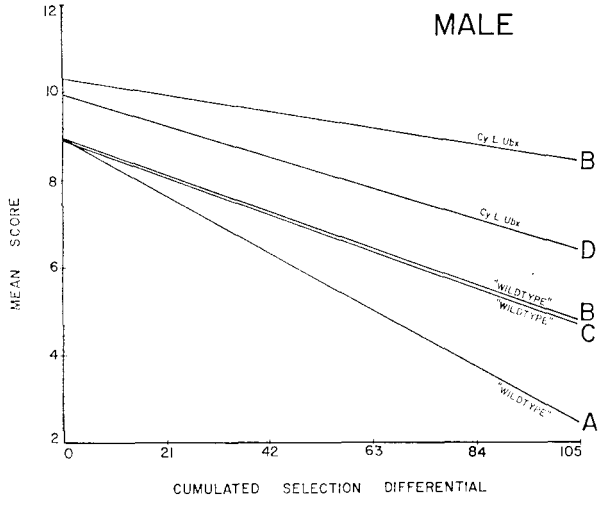


FIGURE 2 (top) and FIGURE 3 (bottom).—Mean response to selection among the different recombination treatments. Each response curve represents the mean regression line of score on cumulated selection differential for four replicates, calculated separately for each sex and major phenotype. To facilitate comparison, the regression lines are drawn as though all four recombination treatments reached the same mean cumulated selection differential at the end of selection. The slopes of the mean regression lines estimate relative rates of response to selection (h^2 or "realized heritability"). Phototaxis score is the mean tendency of flies to take the darker path given a succession of 16 light-dark choices. Zero is the most photopositive possible score (zero dark turns), 16 of the most photonegative. Eight represents phototactic "neutrality", the tendency to turn toward light and dark equal numbers of times.

TABLE 1

Response to selection—males

Recombination treatment and phenotype	Replicate no.	$h^2 \pm$ std. error	Score axis intercept $\pm$ std. error	Variance in h^2 among replicates ($\times 10^{-4}$)
A. "wild-type"	1	.0675 ± .0040	9.08 ± .23	
	2	.0469 ± .0042	8.76 ± .22	
	3	.0475 ± .0056	8.59 ± .30	
	4	.0864 ± .0054	9.32 ± .27	
	mean	.0621	8.94	3.55
B. "wild type"	1	.0442 ± .0048	8.72 ± .27	
	2	.0432 ± .0045	9.13 ± .25	
	3	.0421 ± .0050	9.24 ± .26	
	4	.0293 ± .0076	8.86 ± .37	
	mean	.0397	8.99	0.49
C. "wild type"	1	.0169 ± .0054	8.42 ± .28	
	2	.0440 ± .0056	9.56 ± .29	
	3	.0448 ± .0074	8.51 ± .37	
	4	.0539 ± .0062	9.04 ± .28	
	mean	.0399	8.88	2.55
B. *Cy L Ubx*	1	.0215 ± .0039	10.29 ± .23	
	2	.0195 ± .0035	10.39 ± .20	
	3	.0166 ± .0026	10.24 ± .15	
	4	.0134 ± .0030	10.49 ± .16	
	mean	.0178	10.35	0.12
D. *Cy L Ubx*	1	.0239 ± .0032	9.78 ± .15	
	2	.0394 ± .0055	10.05 ± .25	
	3	.0354 ± .0062	9.95 ± .27	
	4	.0350 ± .0039	9.98 ± .18	
	mean	.0334	9.94	0.44

stantially less rapid response to selection than the A line "wild type" controls. Not only do *Cy L Ubx* flies show poor response to selection, but selection on *Cy L Ubx* flies themselves must be less effective than selection on equivalent "wild type" flies. (3) Measuring response in "wild type" individuals, the mean responses of B and C line males are virtually identical, but C line females lag distinctly behind their B line counterparts. Taken at face value, the last result suggests that suppressing recombination did slow response to selection somewhat but, for some reason, only in females. However, this is not the case.

The cumulated selection differentials used to produce Tables 1 and 2 and Figures 2 and 3 were calculated on the assumption that parental sexes contributed equally to selection differential. If selection on *Cy L Ubx* flies is less effective than selection on "wild type" flies (point 2, above), this assumption does not hold for the B and C lines. Estimating from the data in Tables 1 and 2 on relative A and B and C line response, selection on *Cy L Ubx* individuals must be only about one third as effective as selection on "wild type" individuals in producing response in "wild type" flies. Applied to the data for mean cumulated selection differentials in Table 3, this estimate suggests that the mean *effective* B and C line selection

TABLE 2

Response to selection—females

Recombination treatment and phenotype	Replicate no.	$h^2 \pm$ std. error	Score axis intercept $\pm$ std. error	Variance in h^2 among replicates ($\times 10^{-4}$)
A. "wild type"	1	.0541 ± .0055	9.04 ± .31	
	2	.0407 ± .0062	8.61 ± .32	
	3	.0402 ± .0066	8.36 ± .36	
	4	.0653 ± .0066	8.89 ± .33	
	mean	.0501	8.73	1.44
B. "wild type"	1	.0376 ± .0054	8.66 ± .31	
	2	.0414 ± .0048	9.05 ± .27	
	3	.0328 ± .0035	8.69 ± .19	
	4	.0372 ± .0048	9.07 ± .25	
	mean	.0373	8.87	0.12
C. "wild type"	1	.0198 ± .0113	8.96 ± .61	
	2	.0367 ± .0076	9.22 ± .38	
	3	.0248 ± .0129	8.97 ± .61	
	4	.0416 ± .0111	9.31 ± .49	
	mean	.0307	9.12	1.03
C. *Cy L Ubx*	1	.0107 ± .0033	10.28 ± .18	
	2	.0146 ± .0024	10.09 ± .13	
	3	.0066 ± .0038	10.20 ± .19	
	4	.0162 ± .0028	10.25 ± .13	
	mean	.0120	10.21	0.18
D. *Cy L Ubx*	1	.0160 ± .0045	9.78 ± .21	
	2	.0268 ± .0040	10.03 ± .19	
	3	.0332 ± .0057	10.04 ± .25	
	4	.0283 ± .0039	9.91 ± .18	
	mean	.0261	9.94	0.53

differentials should be proportional to those shown in Table 4. These figures are only approximate and depend again on the assumption that change in score is directly proportional to increase in cumulated selection differential.

Table 4 predicts that the mean B line and C line responses should equal, respectively, about 74% (66.20/89.25) and 66% (59.30/89.25) of the mean A line response, estimates close to but a bit above the actual mean response ratios of 69% and 63%. Evidently, selection on *Cy L Ubx* flies is not quite as effective as the original estimate suggests, but the method is generally sound. Moving on

TABLE 3

Generation 21 cumulated selection differentials, averaged over replicates

Recombination treatment	Males	Females	Mean for both sexes
A	86.40	92.10	89.25
B	89.70	104.00	96.85
C	92.70	81.00	88.85
D	83.00	80.70	81.85

to the main point, Table 4 also predicts that the C lines will show only nine-tenths the realized response of the B lines (59.30/66.20 = .896), an estimate very close to the actual mean response ratio of .91 (1.01 in males, .82 in females). Ignoring sexual dimorphism, mean B line and C line responses were virtually the same. Recombination made no difference at all.

But what about the notable sexual dimorphism in relative response? By averaging over sexes, have I obscured otherwise important effects of recombination on response? Apparently not. Note that over the course of selection the A line "wild type" controls developed a strong and consistent sexual dimorphism in maze phototaxis behavior. Males registered a stronger response to selection. They became distinctly more photopositive than females. The suppressed recombination C lines show the same pattern (with the exception of line C1, the least responsive replicate), but the B lines developed no strong sexual dimorphism. These facts fall into place if it is assumed that selection on females most effectively changes female phenotype; selection on males, male phenotype. FRANKHAM (1968a,b) demonstrates this proposition for single-sex selection on *D. melanogaster* abdominal bristle number. In my experiments, it appears, B line selection acted primarily through "wild type" females, countering the trend toward dimorphism evident in the A line "wild type" controls. C line selection acted primarily through males, reinforcing the trend towards dimorphism. Thus close examination of sexual dimorphism in response upholds the basic conclusion: recombination had no effect on mean response to selection.

In nature the variability of a trait under selection must often be as important as its mean. Taking "wild type" responses sex by sex, the suppressed recombination C lines are more variable in response than the B lines, and the A lines surpass both in interreplicate variability (Tables 1 and 2). The difference in variance in response between the B and C lines is not quite significant in either sex, but it is suggestive. Though suppression of recombination had no demonstrable effect on mean response, it may have led to increased variation in response among replicate populations. If this is so, why are the variances of the freely recombining A lines even larger than those of the C lines? I suggest two contributing factors. First, the A line h^2's are larger than those of the B and C lines, and larger measurements tend to have larger variances (SIMPSON, ROE and LEWONTIN 1960). Second and more importantly, the A lines were, as noted above, really

TABLE 4

Estimated male and female contributions to effective cumulated selection differential in the B and C lines, averaged over replicates

	Contribution to effective cumulated selection differential	
	B	C
Male	14.20	46.35
Female	52.00	12.95
Total	66.20	59.30

more intensively selected than the B and C lines. For a given cumulated selection differential they represent a later stage in selection, and in later stages of selection variability among replicates commonly increases (FALCONER 1960).

After the 21st generation, I relaxed selection and monitored maze phototaxis behavior in the A, B and C lines every few generations up to generation 32. DOBZHANSKY and his co-workers (DOBZHANSKY and SPASSKY 1969; DOBZHANSKY, LEVENE and SPASSKY 1972) have demonstrated that *D. pseudoobscura* populations selected for phototaxis exhibit genetic homeostasis (LERNER 1954). When selection ceases, phototaxis scores tend to return over time towards the score prior to selection. MATHER (1953) suggests that genetic homeostasis results from linkage of favored alleles with alleles of ill effect at other loci. Should this be true, recombination would influence homeostatic behavior. But in these experiments it apparently had little effect. Figure 4 illustrates post-selection trends in phototaxis score. To facilitate comparison, changes in score have been averaged over replicates and sexes, and have been expressed in terms of deviation from generation 21 scores (following RATHIE and BARKER 1968). Only the A lines show a pronounced trend back toward more photonegative scores. The B and C lines show little change over the period monitored. The suffixes Y and N in Figure 4 indicate the presence or absence of autosomal recombination during relaxed selection. I split each post-generation 21 B and C line into two separate lines,

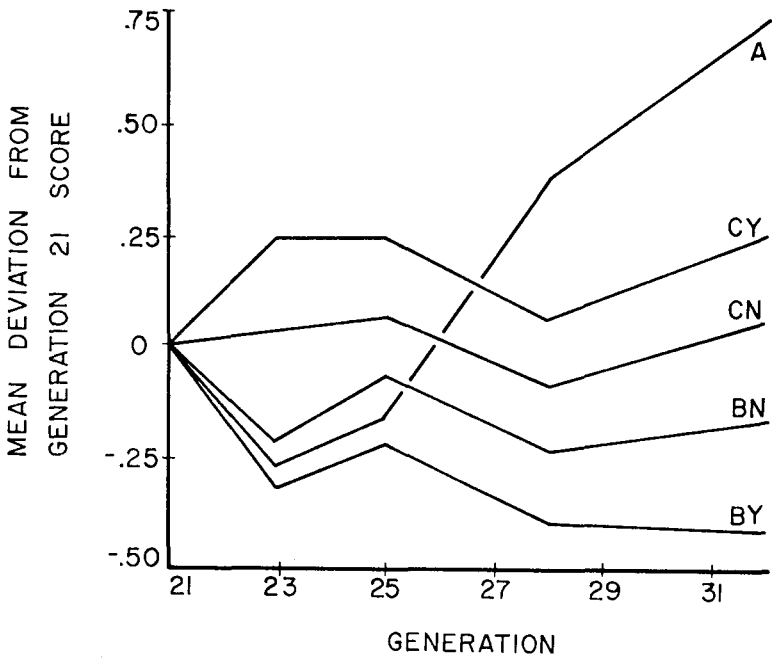


FIGURE 4.—Change in phototaxis scores after selection ceased, expressed as mean absolute deviation from scores in generation 21. Trends in deviation between generations 23 and 32 are the best measure of postselection response. An upward trend represents reversion toward more photonegative behavior. For an explanation of the suffixes Y and N, see text.

TABLE 5

Third chromosome crossing over in female Cy L Ubx *heterozygotes*

Map position	0.0	26.5	43.2	44.0	50.0	58.8	62.0	100.7
Marker	*ru*	*h*	*th*	*st*	*cu*	*Ubx*	*sr*	*ca*
% crossing over		0	0	0	1.3*	1.3*	0	13.4
No. crossovers/ No. flies counted		0/641	0/1551	0/1551	8/619	8/619	0/1551	86/641

* The crossovers in the *st-cu-Ubx* region were all double crossovers involving one crossover in each segment.

permitting free recombination in one (Y), suppressing autosomal recombination in the other (N). The BN lines and the CY lines, sexual lines made asexual and *vice versa*, scored more photopositively, on the average, than their reciprocally mated counterparts. In this limited sense, response in relaxed lines depended not on recombination *per se*, but on whether post-selection recombination differed from that of the parent selection lines.

Recombination suppression and X *chromosome response*

Are the results described above artefacts of experimental technique? Two potentially complicating factors deserve special consideration: the efficacy of the *Cy L Ubx* rearrangement and the selection response of the nonsuppressed *X* chromosomes. MacIntyre and Wright (1966) and McPhee and Robertson (1970) call into question the utility of the Ubx^{130} third chromosome which forms part of the *Cy L Ubx* rearrangement. They indicate that it may not suppress recombination effectively in the presence of other balancer chromosomes. I tested the properties of the *Cy L Ubx* rearrangement directly, using multiply marked *X*, second, and third chromosomes. The *Cy L Ubx* rearrangement suppresses autosomal recombination very effectively, with two exceptions. It permits virtually free recombination in the right tip of the third chromosome (Table 5) and it permits about 4% recombination between the right and left arms of the second chromosome (Table 6). The latter recombinants were easily eliminated by culling *Cy Ubx* and *L* phenotype individuals, so that second chromosome recombination suppression was virtually complete. All in all, the *Cy L Ubx* rearrangement renders about 75% of the total *D. melanogaster* genome nonrecombinant.

TABLE 6

Second chromosome crossing over in female Cy L Ubx *heterozygotes*

Map position	4.0	6.1	54.5	72.0	104.5
Marker	*ho*	*Cy*	*pr*	*L*	*bw*
% crossing over		0.2(?)*	1.7	2.3	0.6
No. crossovers/No. flies counted		2/993	24/1415	32/1415	7/1201

* The apparent crossovers between *ho* and *Cy* are based on phenotypically ambiguous individuals.

As expected, *X* chromosome recombination increases significantly in *Cy L Ubx* rearrangement heterozygotes, almost doubling along some parts of the chromosome (Table 7).

Wild *D. melanogaster* populations carry a range of natural inversions (WARTERS 1944; MOURAD and MALLAH 1960; WATANABE 1967; GROSSMAN 1967). Like the inversions associated with many balancer chromosomes, natural inversions may strongly affect recombination. Accordingly, I investigated the distribution of inversions in my wild-derived Amherst chromosomes before and after selection. Five inversions were tentatively identified. One, a small paracentric with breakpoints in regions 26 and 30 of the left arm of the second chromosome, has apparently not been formally described. The others appeared to be well known cosmopolitan forms: *In(2R)*Nova Scotia, *In(3R)C*, *In(3R)*Missouri, and *In(3R)*Payne (LINDSLEY and GRELL 1968). All five inversions were present before and after selection and none were especially common (frequencies ranged from about 1% to 12%). Two, *In(3R)C* and *In(3R)*Missouri, appeared to increase in overall frequency during selection. Inversions were dispersed unevenly among selection lines (cf. DOBZHANSKY and SPASSKY 1962, 1967), but there seemed to be no systematic differences in the distribution of inversions among recombination treatments. Evidently, hidden differences in overall inversion heterozygosity played little or no role in selection response.

There is also no evidence that Amherst natural inversions impaired recombination suppression by the *Cy L Ubx* rearrangement. The Amherst *X* chromosomes were structurally monomorphic. They could not interfere with autosomal recombination. The Amherst autosomal inversions neither coincided with *Cy L Ubx* inversions, nor overlapped them to form exchange triads (WALLACE 1953). It is unlikely that they seriously reduced the effectiveness of the *Cy L Ubx* rearrangement. I should note that I found no trace of *In(2L)t* in the Amherst population, though IVES (1947) has reported its presence in the past. This inversion, had it been present, could conceivably have interfered with recombination suppression in the left arm of the second chromosome.

Structural heterozygosity in one part of a Drosophila genome often increases recombination in other parts of the same genome (SCHULTZ and REDFIELD 1951; LUCCHESI and SUZUKI 1968). Some people suggest that this secondary recombination increase in nonhomologous parts of the genome may compensate all or in part for recombination suppression in the structurally heterozygous sections (STURTEVANT and MATHER 1938; CARSON 1953; BODMER and PARSONS 1962;

TABLE 7

X *chromosome crossing over in female* Cy L Ubx *heterozygotes*

Map position	0.0		13.7		21.0		33.0		56.7
Marker	*sc*		*cv*		*sn*^{3}		*v*		*f*
Map distance		13.7		7.3		12.3		23.4	
% crossovers		24.3		13.2		14.9		34.1	
No. crossovers/No. flies counted		114/470		62/470		70/470		91/267	

PARSONS 1973). Organisms, it is argued, preserve their evolutionary flexibility by shifting selection response to freely recombining parts of the genome, and the more freely these recombine, the more effective is response to selection. Note the underlying assumption, that more recombination permits speedier response to selection, which is the assumption that the experiments reported here set out to test. As noted above (Table 7), *X* chromosome recombination does increase substantially in *Cy L Ubx* heterozygotes. Could it be that the C lines shifted a substantial part of their selection response to the *X* chromosomes, negating the otherwise large effects of suppressing autosomal recombination?

To test this possibility I measured the independent effects of sex chromosomes and autosomes crossed from the generation 21 selected lines into an unselected Amherst genetic background. The crosses involved are detailed in THOMPSON (1974). The results appear in columns B, C, and D of Tables 8 and 9. To make comparisons between lines, it is necessary to normalize scores as proportions of parental selection line response, and to measure all responses against the scores of the unselected population into which the selected chromosomes were crossed.

TABLE 8

Chromosome substitution lines—males

A	B	C	D	E	F	G
	Substitution line scores*					
Original selection line	$\frac{S}{S}$	$\frac{U}{S}$	$\frac{S}{U}$	†Relative response attributable to *X* chromosome	‡Relative response attributable to autosomes	Total response attributable to independent effects
A1	4.39	4.36	8.91	.052	1.001	
2	4.78	6.52	9.49	—.075	.603	
3	6.33	6.45	7.70	.516	.958	
4	2.53	4.39	8.51	.098	.719	
mean	4.51	5.43	8.65	.148	.820	.968
B1	4.47	5.70	8.84	.068	.738	
2	5.21	5.87	7.81	.342	.833	
3	4.80	6.14	8.58	.133	.693	
4	5.64	6.78	9.19	—.009	.676	
mean	5.03	6.12	8.61	.134	.735	.869
C1	8.03	7.25	8.56	.531	1.690	
2	8.12	7.53	9.24	—.008	1.567	
3	4.10	5.97	6.83	.460	.630	
4	4.38	7.21	8.20	.201	.408	
mean	6.16	6.99	8.21	.296	1.074	1.370
Grand mean				.189	.877	1.066

* S = chromosomes from selected population, U = chromosomes from unselected population, arranged in each case as: $\frac{X \text{ chromosome}}{\text{autosomes}}$.

† $\frac{U}{U} - \frac{S}{U}$ $\frac{U}{U} - \frac{S}{S}$ $\frac{U}{U} - \frac{S}{S}$

‡ $\frac{U}{U} - \frac{U}{S}$ $\frac{U}{U} - \frac{S}{S}$ $\frac{U}{U} = 9.16$ (The mean male score in the unselected population)

TABLE 9

Chromosome substitution lines—females

A	B	C	D	E	F	G
	Substitution line scores*					
Original selection line	$\frac{S}{S}$	$\frac{U}{S}$	$\frac{S}{U}$	†Relative response attributable to *X* chromosome	‡Relative response attributable to autosomes	Total response attributable to independent effects
A1	4.19	6.05	7.67	.319	.636	
2	5.88	7.40	9.46	—.048	.556	
3	5.71	7.71	8.62	.192	.449	
4	3.22	6.13	8.11	.196	.521	
mean	4.75	6.82	8.47	.167	.541	.708
B1	3.01	5.12	7.52	.283	.665	
2	4.88	6.23	8.13	.265	.706	
3	6.03	5.28	9.04	.080	1.129	
4	5.86	6.05	9.83	—.125	.945	
mean	4.95	5.67	8.63	.126	.886	1.012
C1	6.48	7.00	8.22	.383	.816	
2	6.98	7.38	9.63	—.142	.828	
3	4.87	5.70	7.51	.404	.813	
4	4.80	7.85	8.29	.224	.322	
mean	5.78	6.98	8.41	.217	.695	.912
Grand mean				.170	.708	.880

*†‡ Footnotes as in Table 5 except $\frac{U}{U} = 9.30$ (The mean female score in the unselected population).

The transformations involved appear as footnotes to Tables 8 and 9, the transformed data appearing in columns E and F. The results for individual selection lines are quite variable, a consequence of large intrinsic nongenetic variation among phototaxis maze runs, but the drift of the data is fairly consistent and clear.

Taking the A, B, and C recombination treatments one by one separately for each sex, mean *X* chromosome contribution to total response ranges from 13% to 30%. *X* chromosome contribution may be a little greater in the C lines than in the others, but this difference has little statistical force ($p > .25$ in each sex that the difference in mean response between the C lines and the A and B lines is due to chance alone). Mean autosome contribution to total response ranges from 54% to 107% with no apparent trend towards differences among the recombination treatments. Overall, the chromosome substitution data provide little or no support for the hypothesis that inversion heterozygotes shift selection response to freely recombining sections of the genome, even when recombination in these latter sections is considerably boosted by the presence of nonhomologous inversions. McPHEE and ROBERTSON (1970) report similar results for selection on *D. melanogaster* sternopleural bristles. The primary experimental observation stands. Suppression of recombination had no measurable effect, hidden or overt, on mean response to selection. Note especially that the chromosome substitution

experiment constitutes an internal control for the more general result. If increased recombination could speed selection response, the C line *X* chromosomes should have exceeded those of the A and B lines in mean response, regardless of selection response among the autosomes. But this was apparently not the case.

The chromosome substitution data serve another purpose. They tell something about the genetic architecture underlying phototaxis selection response. Averaging over sexes and recombination treatments, the *X* chromosome and the autosomes account, respectively, for about 18% and 79% of total selection response. Were their action on phototaxis behavior completely independent, they should account together for about 99% of total response. Evidently, epistatic interactions between the *X* chromosome and the autosomes played little or no role in selection response. Furthermore, the *X* chromosome and the autosomes comprise, respectively, about 20% and 80% of the total haploid *D. melanogaster* genome, figures very close to their relative contributions to selection response. This suggests, but by no means proves, that positive phototaxis maze behavior may in fact approach the assumptions of polygenicity (many loci of small effect dispersed more or less evenly throughout the genome). SPICKETT and THODAY (1966), for example, demonstrate that polygenic traits may depend on very few effective loci, and the above results could all be explained by invoking just five equal loci distributed one to each major chromosome arm. To the degree that positive phototaxis maze behavior meets the criteria for polygenicity, the generality of the present results is strengthened.

DISCUSSION

The experiments suggest that recombination has no effect on mean response to selection and that in this limited sense, sex does not affect the rate of evolution. Certain earlier experiments, comparable in greater or lesser degree to my own, purport to demonstrate the opposite result. RASMUSON (1954, 1955) and CARSON (1958) claim to show that increased recombination increases response to selection, and MCPHEE and ROBERTSON (1970) and MARKOW (1975) argue that suppressed recombination suppresses response to selection. With the exception of MCPHEE and ROBERTSON's work, these experiments suffer serious technical flaws (THOMPSON 1976). But the greatest problem in interpreting work of this nature stems from the fact that sex may have very different consequences on response to selection in populations of different size. RASMUSON, CARSON and MCPHEE and ROBERTSON all selected on tiny populations, with effective sizes of ten or less. By population genetics standards, I selected on populations of intermediate size. A large part of the apparent contradiction in results may hang on this difference.

Given certain reasonable assumptions, increased recombination should accelerate selection response in very small populations. But as population size increases to intermediate magnitude, this effect should become smaller and smaller (FRASER 1957; MARTIN and COCKERHAM 1960). At some size the accelerating effect of recombination on response should vanish altogether (YOUNG 1966; QURESHI,

KEMPTHORNE and HAZEL 1968; QURESHI 1968). More abstract arguments suggest that in very large populations, recombination may often slow response to selection, the larger the population the greater the effect (see DISCUSSION in THOMPSON 1976). Seen in this light, my experimental results may stem directly from selection on populations of fortuitously moderate size. This does not settle the question. The precise nature of genetic variation in natural populations must deeply influence interactions among sex, population size, and response to selection. But regarding the nature of genetic variation, population geneticists do not agree (LEWONTIN 1974). Until the nature of variation is better understood, hypotheses concerning the role of sex in evolution must remain highly speculative. The simple notion that recombination speeds response to selection does not hold up under scrutiny.

More recombination-selection experiments should be performed, particularly experiments transcending Drosophila and encompassing populations of very large size. Even then a more profound question remains to be resolved (WILLIAMS 1975). Sex itself is a selectable trait. Species may be polymorphic for sex, or for increased and decreased recombination. In the last analysis, what maintains sex in local populations? In species? And do the experiments above remotely address this question?

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