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Interaction of selection and recombination.

The character of DDT-resistance of Drosophila melanogaster was used to investigate the interactions of selection and recombination. Our experiments started with two lines, Iso Null and Plus Null, out of our stock Berlin wild + K. Iso Null was isogenic and Plus Null heterogenic in the second chromosome. Each line was subjected to a high (5% survival rate; Iso S, Plus S) and a low (41.7% survival rate; Iso L, Plus L) selection pressure. In the 11th generation of selection, we started two new lines, Iso LS and Plus LS, out of the low selected ones by decreasing their survival rate to 5%. The influences of the 1st and 3rd chromosomes should be pointed out in the Iso-lines, and if recombination would have an important effect on selection response it should be seen in the Plus-lines. To compensate the environmental variation within and between generations of selection we corrected the average survival rates (LD_{50}) of each line by their corresponding controls (adding 10 for arithmetical convenience). Therefore the selection curves in Figure 1 represent the gain of selection in each line in relation to their respective controls. Furthermore we smoothed data five times by the three point formula and these data are shown in Figure 2.

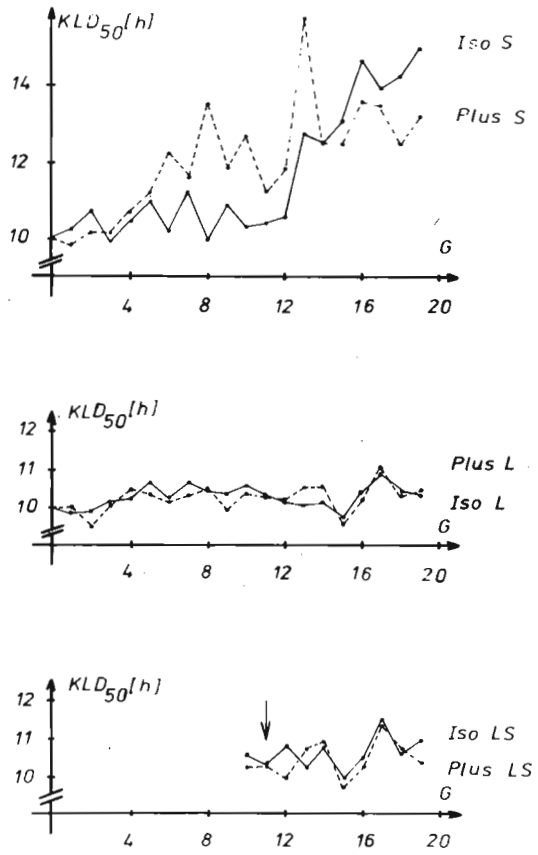

Fig. 1. Corrected average survival rates $KLD_{50}(h)$ of the selected lines plotted against generations (G). The arrow indicates increasing of selection pressure in the low lines.

In order to analyse our experimental data we started computer simulation. First, we used the deterministic model of Lush assuming two unlinked autosomal loci responsible for DDT-resistance with an intermediary and a recessive mode of inheritance. In this case, we got the best fit with the assumption of 5% alleles at each locus responsible for resistance in the initial populations and a coefficient of selection of about 0.70. Secondly, we started another model with 15 loci and three linkage groups using the Monte-Carlo-technique. The values of resistance were simulated by 0 (normal) and 1 (resistant), and between the five loci in each of the linkage groups we fixed the frequencies of recombination at 5, 10, 15 and 20 per cent. The mode of inheritance of the alleles for resistance was defined as recessive in the 1st and 3rd linkage groups and intermediary in the 2nd one without deviation from additivity. In the initial population we started with 5% alleles for resistance and 95% normal alleles at each locus, which were distributed at random. In this model the influence of recombination was strong, especially with respect to the level of resistance and the number of alleles which were fixed, but the effects were suspended in case of low survival rates (10% and 20%, resp.) and by recessive inheritance of the alleles for resistance. It could be pointed out that in our simulated populations the two plateaus observed during selection were not due to recombination, but to the effect of increasing percentages of the recessive alleles for resistance. On the other hand, recombination smoothed the selection curves just to a sigmoid.

Our experimental data and their analysis by computer simulation lead to the conclusion that recombination did not determine the twofold increase of the selection curve of Plus S. This must be much more due to the selection of each an intermediary and a recessive system of alleles responsible for DDT-resistance which may be unlinked. On the basis of our results we

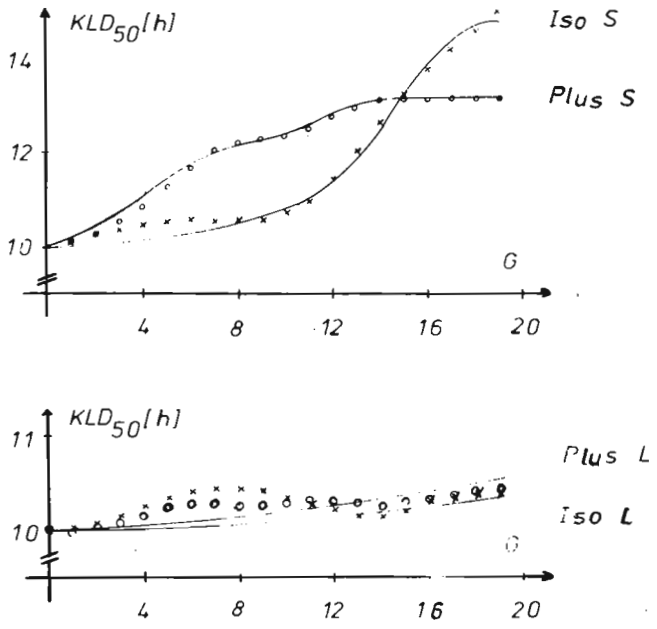

Fig. 2. Circles (o) and crosses (x) indicate the corrected average survival rates of high (uppermost) and low (bottom) selected tines after smoothing. According to the model of Lush the selection curves are the best fit to the experimental data.

can support Robertson's assumption about the importance of recombination or linkage, "that the effects of linkage are much less than we (I) had expected and less than might be assumed for discussions of linkage in literature".

References: Crow, J.F. and M. Kimura 1970, Harper and Row, New Nork; Köhler, W. 1973, Dissertation der Freien Universität Berlin; Robertson, A. 1970, Biomathematics 1:246-288.

Lamb, M.J. and L.J. Lilly. Birkbeck College, University of London, and Middlesex Hospital Medical School, London, England. No detectable increase in sex-linked recessive lethal frequency after feeding male D. melanogaster with the fungicide Benlate.

Benomyl (trade name "Benlate") is a widely used systemic fungicide. Hastie (1970) has reported benomyl-induced instability in Aspergillus nidulans diploids and Boyle (1973) has found cytogenetic effects in Allium cepa and Secale cereale; Dassenoy and Meyer (1973) showed that benomyl induced forward mutations in Fusarium oxysporum. In view of this evidence that benomyl may cause genetic damage, we decided to investigate possible mutagenic effects in D. melanogaster and report here the results of an experiment in which the M-5 technique was used to test for the induction of sex-linked recessive lethals.

Number of chromosomes tested and lethals found after feeding Benlate.

Brood	Fed Benlate		Fed DMSO	
	tests	lethals	tests	lethals
I	340	0	340	0
II	340	1	339	0
III	335	0	339	0
IV	310	0	338	0
V	300	0	320	0
VI	270	0	317	0
Total	1895	1	1993	0

A freshly prepared solution of 0.1% Benlate in 0.5% DMSO was fed to starved 3-day-old Or-R males; each male took approximately 0.14 mg of solution. Control males were fed with 0.5% DMSO. Each treated male was mated with 2 M-5 females in each of six 3-day broods. Approximately 10 chromosomes from each male in each brood were tested for the presence of sex-linked recessive lethals. The results obtained are given in the table. The data provide no evidence of a mutagenic effect of Benlate in Drosophila. It should be stressed, however, that the data are small, only one concentration has been used, only one type of mutation has been investigated, and tests of the mutagenicity of BCM, the breakdown product of benomyl which is formed in aqueous solution, have not yet been made. Further experiments to investigate possible mutagenic effects of benomyl and BCM are in progress.

References: Boyle, W.S. 1973, J. Heredity 64:49-50; Dassenoy, B. and J.A. Meyer 1973, Mutation Res. 21:119-120; Hastie, A.C. 1970, Nature 226:771.