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Effects of allozyme variation on fitness components in *Drosophila melanogaster*

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Abstract

We have explored in *Drosophila melanogaster* the fitness effects of allelic variation at three enzyme loci: α Gdh, Adh, and Acph. Viability and rate of development are studied at two densities, near-optimal and competitive. No genotypic effects could be demonstrated on rate of development at either density or on viability under optimal conditions. Small but significant effects on viability appear under competitive conditions. Fecundity is measured for all nine possible mating combinations between the three female and the three male genotypes at each locus. Female genotype has important fitness consequences; heterosis exists at every locus. Male genotype also contributes to fitness, but without heterosis. There are significant interactions between female and male genotypes, so that the fecundity of a mating combination cannot be determined from the average fitnesses of the female genotype and the male genotype involved.

Introduction

The neutrality theory of protein evolution propounds that most protein variants (allozymes) found in natural populations at nontrivial frequencies are adaptively equivalent. When investigating a particular protein polymorphism, the neutrality theory may be taken as the null hypothesis. Experiments are, then, designed to test whether the allozyme variants are selectively identical. If selection is demonstrated, the neutrality hypothesis is rejected; otherwise, neutrality remains the standing hypothesis for the investigated polymorphism.

The selective effects of allozyme variants have been investigated in very many ways, both in nature and in the laboratory. A particularly informative

approach consists in examining whether allozyme variation affects one or other component of the life cycle. In the present paper we report the results of a study of three allozyme polymorphisms in *Drosophila melanogaster*. Three fitness components are examined for each gene locus: viability, rate of development, and fecundity. The experiment is designed so that the fecundity of a female can be measured not only as a function of its own genotype but also as a function of the genotype of the male with which it mates.

Material and methods

The experimental strains were derived from a sample of 1 500 *Drosophila melanogaster* females collected in the fields of the University of California, at Davis. Each female was placed in a half-pint bottle with medium and the progeny maintained by mass culture. Very numerous single-pair crosses were made with the progenies of the wild flies, in

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such a way that the two parents in a cross always descended from different wild females. Five days after a single-pair cross was made, the two parents were assayed by electrophoresis (procedures of Ayala *et al.*, 1972). If both parents were identical homozygotes with the desired genotype, their progeny was identified as one of the experimental lines and maintained by mass culture. In order to obtain the desired number of strains with each genotype, single-pair crosses had to be made in some cases for more than one generation; but in every case, the two parents were derived from different wild females so that no inbreeding was involved.

The three loci studied are $\alpha Gpdh$ (chromosome II, 17.8, coding for alpha-glycerophosphate dehydrogenase), *Adh* (II, 50.1, coding for alcohol dehydrogenase), and *Acph* (III, 101.1, coding for acid phosphatase). Each locus is polymorphic for two alleles, *F* and *S*, with some other alleles found in low frequencies. At all three loci, the rarer of the two common alleles is *S*, which in the Davis natural population has a frequency of 0.18 for $\alpha Gpdh$, 0.40 for *Adh*, and 0.11 for *Acph*.

Four sets of experimental lines were obtained, each consisting of 30 independent strains homozygous at all three loci under investigation. The genetic constitution of the four sets is, as follows:

	(1)	(2)	(3)	(4)
$\alpha Gpdh$:	F/F	S/S	F/F	F/F
<i>Adh</i> :	F/F	F/F	S/S	F/F
<i>Acph</i> :	F/F	F/F	F/F	S/S

The effect of genotypic variation at $\alpha Gpdh$ was studied by using sets (1) and (2); sets (1) and (3) were used for *Adh*; and sets (1) and (4) for *Acph*. It can be seen that the experimental flies are homozygous for the *F* allele at the two loci other than the one under investigation. In order to obtain heterozygous flies for $\alpha Gpdh$, crosses were made between strains of set (1) and strains of set (2). Similarly, crosses between sets (1) and (3) produced the *Adh* heterozygotes, and between sets (1) and (4) the *Acph* heterozygotes.

Three fitness components were studied for each gene locus: fecundity (number of eggs laid by three females during four days), viability (percent of eggs developing into imagoes), and rate of development (number of days from egg to imago). Viability and rate of development were measured in two density

conditions: quasi-optimal (40 eggs placed in a half-pint culture with 40 cc of food medium) and competitive (200 eggs in a 2 × 8 cm vial with 8 cc of medium). The eggs counted in the fecundity experiment, placed in the appropriate cultures and densities, served for measuring viability and rate of development.

Fecundity was measured by placing in a vial three virgin females of a given genotype with three males of the same or different genotype. A paper teaspoon with medium was replaced daily in the vial. The eggs laid between days 6–9 were counted. The flies introduced in the vials were obtained as follows. For each homozygous genotype, 15 crosses were made, each involving two of the original 30 experimental strains; for each heterozygous genotype, 15 crosses also were made, involving 15 randomly chosen strains with a given homozygous genotype and 15 strains with the other genotype. The three females placed in a vial for measuring fecundity always came from strains different from those producing the males introduced in the same vial. Thus, the genetic background was thoroughly randomized not only for the flies mating in the experimental vials, but for their progeny as well. All nine possible combinations between the three female genotypes and the three male genotypes (♀ FF × ♂ FF, ♀ FF × ♂ FS, ♀ FF × ♂ SS, ♀ FS × ♂ FF, etc.) were made for each locus. Each combination was replicated 60 times. Hence, the total number of experimental vials for the fecundity experiment was: 3 loci × 9 genotypic combinations × 60 replicates = 1 620, in each of which the eggs were counted on four consecutive days.

Eggs of the same age and genotype, counted in the fecundity experiment, were placed in groups of 40 in half-pint cultures ('optimal' conditions), or in groups of 200 in vials (competitive conditions). The flies emerging in these cultures were counted daily. Viability was measured simply by the percent of flies emerging. Rate of development was measured in days by the formula $\sum n_i d_i / \sum n_i$, where n_i is the number of flies emerging on the d_i day after the eggs were placed in the cultures. Each genotype was studied in 30 replicates at each density, so that the number of cultures counted was 7 genotypes × 2 densities × 30 replicates = 420.

All experiments were performed in a controlled-temperature incubator at $25 \pm 0.5^\circ \text{C}$ and ca. 70% relative humidity.

The effects of genotype and density on viability and rate of development were first tested by a factorial analysis of variance. The percent viabilities were transformed into $\arcsine\sqrt{\text{percent}}$ for the analysis of variance. The genotypic effects on viability were significant and, hence, nested analyses of variance were made separately for each of the two densities. *A posteriori* comparisons between means were made by a Student-Newman-Keul test for least significant range (LSR) (Sokal & Rohlf, 1969, pp. 294–295).

The fecundity data were tested, first, by a factorial analysis of variance. Nested analyses of variance were also performed for the average fecundity of the three genotypes at each locus, separately for females and for males. (Nested analyses of variance were made also for testing the effects of each male genotype on a given female genotype, and viceversa; but these will not be given here, although they are taken into account in the evaluation of the results.) LSR tests also were carried out for comparisons between fecundity means.

Results

Table 1 gives the mean viability and rate of development for each genotype at each density. The table also shows by means of *t*-test comparisons that the viability is significantly lower, and the rate of development longer, under competitive conditions than when density is near optimal. The experiments were made at two densities not in order to test whether a reduction in fitness would occur at competitive densities, but rather in order to see whether the relative fitnesses of genotypes would change from one to another density, as it has been shown in other instances (e.g. Tosić & Ayala, 1981).

The analyses of variance shown in Table 2 manifest the large differences between the two densities but no significant interactions between density and genotype for either viability or rate of development. Genotypic differences have no significant effects on rate of development, but they are marginally significant for viability. Nested analyses of variance (Table 3) show that the genotypic effects on viability

Table 1. Viability (in percent) and rate of development (in days) of individuals with different genotype at one of three loci coding for enzymes. The *FF* individuals have this genotype at all three loci; individuals shown with the *FS* or *SS* genotype at a given locus, have the *FF* genotype at the other two loci. The mean and standard error are based on 30 replicates. Student's *t* is for pairwise comparisons between optimal and competitive densities.

Fitness component	<i>FF</i>	<i>αGpdh</i> <i>FS</i>	<i>SS</i>	<i>Adh</i> <i>FS</i>	<i>SS</i>	<i>Acph</i> <i>FS</i>	<i>SS</i>
Viability:							
optimal density	79.7 ± 3.6	76.1 ± 3.6	75.1 ± 3.7	86.1 ± 2.6	84.5 ± 2.8	85.6 ± 2.8	82.2 ± 3.2
competitive	69.0 ± 2.0	68.4 ± 2.0	71.9 ± 2.6	66.5 ± 2.0	76.0 ± 1.4	71.5 ± 1.9	68.1 ± 1.5
<i>t</i>	3.4**	2.6*	1.0	5.6**	3.1**	4.4**	4.0**
Rate of development:							
optimal density	8.96 ± 0.04	9.10 ± 0.05	8.96 ± 0.06	9.03 ± 0.04	8.95 ± 0.07	9.00 ± 0.05	9.07 ± 0.06
competitive	9.64 ± 0.06	9.65 ± 0.10	9.67 ± 0.08	9.95 ± 0.09	9.82 ± 0.08	9.71 ± 0.09	9.80 ± 0.10
<i>t</i>	6.8**	5.5**	7.1**	9.2**	8.7**	7.1**	7.3**

* Statistically significant, $P < 0.05$; ** $P < 0.01$

Table 2. Factorial analysis of variance for viability and for rate of development.

Source of variation	Degrees of freedom	Viability ^a Mean square	F	Rate of development Mean square	F
Genotype	6	286	2.11*	0.27	1.80
Density	1	11 252	82.97**	57.69	384.60**
Interaction	6	277	2.04	0.22	1.47
Error	406	136		0.15	

* Statistically significant, $P < 0.05$; ** $P < 0.01$

^a For the analysis of variance the percent viabilities have been transformed into $\arcsine\sqrt{\text{percent}}$

Table 3. Nested analysis of variance for viability under quasi-optimal density and under competitive conditions.

Source of variation	Degrees of freedom	Optimal density Mean square	F	Competitive density Mean square	F
Genotype	6	432	1.03	140	2.47*
Strain	28	412	2.25**	57	1.00
Error	175	183		57	

* Statistically significant, $P < 0.05$; ** $P < 0.01$

are not significantly heterogeneous at optimal density, but they are at the competitive density. It can be seen in Table 1 that the greatest differences occur at the *Adh* locus. Indeed, the LSR test shows that only one significant difference exists between the means, namely that between the viability of the SS and FS genotypes of *Adh* at high density.

Tables 4–6 give the mean number of eggs laid by each mating combination. Clearly, genotype has a substantial effect on fecundity. This is examined by factorial analyses of variance (Table 7) showing that both the female and the male genotype affect fecundity, and that there is significant interaction between the two. The contributions of the two sexes are examined by separate nested analyses of variance (Tables 8–10). The female genotypes are (highly) significantly heterogeneous at all three loci. The male genotypes are significantly heterogeneous only for *Acph*. Yet there are significant interactions between female and male genotype in all cases. That is, the performance of a given female genotype depends on the male with which she mates; and the performance of a given male genotype depends on the female with which he mates. In spite of these interactions, it appears that for *αGpdh* and *Adh* a male genotype that increases the fecundity of a given female genotype must have compensatory effects on the other female genotypes, since the male genotypes do not differ significantly in mean performance.

The most striking effect when the individual gen-

Table 4. Fecundity of the *αGpdh* genotypes (mean number of eggs with standard error). The number of replicates is 60 for each individual mating type; 180 for each average.

Female genotype	Male genotype			Female average
	F/F	F/S	S/S	
F/F	88.8 ± 5.3	74.1 ± 3.1	141.9 ± 7.4	101.6 ± 3.9
F/S	149.9 ± 5.4	140.8 ± 6.8	126.0 ± 5.1	138.9 ± 3.4
S/S	114.5 ± 7.0	84.4 ± 4.1	106.6 ± 7.0	101.8 ± 3.7
Male average	117.7 ± 3.9	99.8 ± 3.6	124.8 ± 3.9	

Table 5. Fecundity of the *Adh* genotypes (mean number of eggs with standard error). The number of replicates is 60 for each individual mating type; 180 for each average.

Female genotype	Male genotype			Female average
	F/F	F/S	S/S	
F/F	88.8 ± 5.3	83.0 ± 4.1	128.3 ± 6.5	100.0 ± 3.4
F/S	162.1 ± 6.1	136.1 ± 7.5	149.9 ± 6.3	149.3 ± 2.5
S/S	111.5 ± 5.1	93.7 ± 3.8	102.3 ± 6.0	102.5 ± 3.0
Male average	120.8 ± 3.9	104.3 ± 3.5	126.8 ± 4.2	–

Table 6. Fecundity of the *Acph* genotypes (mean number of eggs with standard error). The number of replicates is 60 for each individual mating type; 180 for each average.

Female genotype	Male genotype			Female average
	F/F	F/S	S/S	
F/F	88.8 ± 5.3	96.6 ± 4.7	121.9 ± 6.3	102.4 ± 3.3
F/S	164.8 ± 6.2	142.5 ± 6.3	174.8 ± 7.2	160.7 ± 3.8
S/S	94.4 ± 6.5	117.4 ± 5.5	142.4 ± 6.2	118.0 ± 3.8
Male average	116.0 ± 4.3	118.8 ± 3.4	146.4 ± 4.2	–

Table 7. Factorial analysis of variance for fecundity.

Source of variation	Degrees of freedom	<i>αGpdh</i> Mean square	F	<i>Adh</i> Mean square	F	<i>Acph</i> Mean square	F
Female genotype	2	84 234	41**	142 321	67**	167 225	77**
Male genotype	2	31 217	15**	25 578	12**	49 809	23**
Interaction	4	35 361	17**	12 905	6**	8 671	4**
Error	531	2 066		2 127		2 162	

** Statistically significant, $P < 0.01$

Table 8. Fecundity. Nested analysis of variance for the genotype averages at the *αGpdh* locus.

Source of variation	Degrees of freedom	Females Mean square	F	Males Mean square	F
Genotype	2	84 234	9.94**	31 217	2.83
Strain	42	8 470	4.44**	10 995	5.76**
Error	495	1 909		1 909	

** Statistically significant, $P < 0.01$ Table 9. Fecundity. Nested analysis of variance for the genotype averages at the *Adh* locus.

Source of variation	Degrees of freedom	Females Mean square	F	Males Mean square	F
Genotype	2	134 217	19.37**	25 578	2.02
Strain	42	6 927	3.57**	12 662	6.70**
Error	495	1 962		1 886	

** Statistically significant, $P < 0.01$ Table 10. Fecundity. Nested analysis of variance for the genotype averages at the *Acph* locus.

Source of variation	Degrees of freedom	Females Mean square	F	Males Mean square	F
Genotype	2	167 224	24.40**	44 162	3.57*
Strain	42	6 861	3.42**	12 368	5.92**
Error	495	2 008		2 089	

** Statistically significant, $P < 0.05$; * $P < 0.01$

Table 11. Deviations from expected number of eggs produced by each type of mating. The expected value is calculated from the data in Tables 4-6 by multiplying the corresponding female and male averages and dividing this product by the average of all nine genotypes.

Locus and female genotype	Male genotype		SS
	FF	FS	
<i>αGpdh</i> :			
FF	16	15	+31
FS	+7	+19	-26
SS	+9	4	-5
<i>Adh</i> :			
FF	14	6	+20
FS	+8	+3	11
SS	+6	+3	-9
<i>Acph</i> :			
FF	5	-1	+4
FS	+18	-8	10
SS	-13	+7	+6

otypes are considered is the strong overdominance of female fecundity. At each of all three loci, heterozygous females lay significantly more eggs than either one of the two homozygotes, as shown by the LSR test. This heterosis is quite remarkable in that it represents an apparent increment of 35-50 percent over the fecundity of the homozygous females. And it cannot be attributed to an increase in overall heterozygosity, because all experimental flies are fully outbred.

There is no overdominance among male genotypes. Indeed, at two loci, *αGpdh* and *Adh*, the heterozygous males have the worst performance of the three genotypes. However, LSR tests show significant differences in mean performance among the male genotypes only for the *Acph* locus, where the SS homozygotes induce significantly higher fecundity than either one of the other two genotypes, which do not differ from each other.

The interactions between female and male genotype are explored in Table 11, where the difference between the observed and the expected fecundity is given for each mating combination. The expected fecundity of a mating type is simply estimated by the product of the average fecundity of the two genotypes involved divided by the grand average. One discernable pattern appears at the $\alpha Gpdh$ and Adh loci; namely the fecundity of a homozygote is greater when it mates with a homozygote for the alternative allele than when it mates with a like homozygote. The departures from expectation for these combinations are highlighted in italics in Table 11. The evolutionary significance of this pattern of interactions is that it will contribute to maintaining genetic polymorphism (Serradilla & Ayala, 1983; Hader & Liberman, 1975; Cockeram *et al.*, 1972).

Figure 1 summarizes the fecundity data. It clearly shows the overdominance of the female heterozygotes as well as the complexity of the female-male interaction (e.g., the SS male genotype at the $\alpha Gpdh$ locus yields the highest fecundity of the three male genotypes when mating with the FF females, but the lowest when mating with the FS females).

Fig. 1. Fecundity of the various genotypes at three gene loci coding for enzymes. A, $\alpha Gpdh$; B, Adh ; C, $Acph$. The three bars in the middle represent the fecundity of the heterozygous female; the two sets on the sides show the fecundity of the homozygous females. For each female genotype, the middle bar represents the contribution of the heterozygous males; the two bars on each side, the contribution of the homozygous males. The heterozygous females exhibit overdominance at all three loci, but there is no male heterosis. Large interactions between female and male genotype are manifest.

Discussion

Genotypic variation associated with the three loci examined ($\alpha Gpdh$, Adh , and $Acph$) in this experiment is effectively neutral with respect to rate of development and with respect to viability under nearly optimal conditions; when there is competition for resources, the Adh genotype has small effects on viability. All three gene loci have significant effects on fecundity. These effects include overdominance of the heterozygous female at each locus, as well as increased fecundity when different homozygotes mate - both of which are processes that will contribute to maintain genetic polymorphism. Whether the allozyme polymorphisms found at these loci are maintained in nature by these or other processes cannot be decided by our experiments. These experiments, however, establish that, at least under certain conditions, fitness differences exist among individuals having different genotypes at the three loci.

The question arises whether the fitness differences observed are due to the loci examined or to other loci that might be associated in linkage disequilibrium with the target loci. The following observations are relevant. The two genomes of each fly used in the fecundity experiments come from separate wild-collected flies; thus, the experimental flies have levels of heterozygosity comparable to wild flies. The same can be said of the parents of the experimental flies (which come from the four sets of 30 experimental strains), all containing two genomes descended from two separate wild-collected flies. And the same is also the case for the zygotes in which viability and rate of development are measured, because their male and female parents derive in each case from different sets of wild-collected flies. The sampling variance of D_{ij} , the measure of linkage disequilibrium between loci i and j , can be calculated using the equation given by Laurie-Ahlberg and Wier (1979, p. 1309). Following the reasoning of Tosić and Ayala (1981), the sampling variance of D_{ij} in the present experiments can be calculated as, approximately, $1/240$ (four sets of 30 experimental strains are used, each strain having no less than two wild genomes). The very small sampling variance justifies the conclusion that no linkage disequilibrium has been created in the laboratory experiments. In addition, the use of considerably more than 100 different wild chromosomes in the

experiments ensures that more than 95% of the genetic variation present in the natural population is represented in the experiments.

That no linkage disequilibrium associations have been created in the laboratory, and that most of the natural genetic variation is represented in the experiment, do not warrant that the fecundity (and viability) variation observed in the experiments is due to the loci examined alone. If alleles at the experimental loci are nonrandomly associated in nature with alleles at other tightly linked loci, then the genetic effects observed might be due to such linked loci and/or to the target loci. But it should be noticed that if linkage disequilibrium exists in the experiments because it exists in nature, the observed effects would also affect the target loci in nature and thus contribute to maintaining the polymorphisms.

Our results are consistent with a number of studies in the last two decades showing that fertility may be a major contributor to fitness differences, often substantially more important than viability or rate of development. Sved and Ayala (1970) found that, in population cages, fertility ('late' fitness component) had greater importance than viability ('early' fitness component) in determining the fitness of *D. melanogaster* flies homozygous for full chromosomes. Anderson and Watanabe (1974) suggested that female fecundity and male mating capacity might be main contributors to the maintenance of chromosomal polymorphisms in *D. pseudoobscura*. Allozymes have been associated with fertility differences in a number of cases (e.g., Gilbert *et al.*, 1982; Marinković & Ayala, 1975a, b).

It has become increasingly apparent that male genotype contributes significantly to fitness. It has been known for a number of years that the mating success of a male is affected by its genotype (Petit & Ehrman, 1969; for allozyme genotypes see Knopien *et al.*, 1980; McKenzie & Fegent, 1980; Pot *et al.*, 1980). Maynard Smith (1956) has shown that females inseminated by outbred males produce more offspring than those inseminated by inbred males. Brittnacher (1981) has found in *D. melanogaster* that male mating capacity ('virility') has a very large effect on fitness variation, it being perhaps the single most important fitness component. Gilbert *et al.* (1981) and Gilbert and Richmond (1982) have demonstrated in *D. melanogaster* that the enzyme esterase-6 is transmitted to the females

in the male ejaculate during insemination. Genetically determined variation in this enzyme affects the time that a female will take from one to another mating and the number of progeny produced by the female.

The role of heterozygous superiority ('overdominance') in maintaining genetic polymorphisms has been discussed in a number of places and needs not be reviewed here. The particular case of allozyme overdominance has also been extensively considered (e.g., Milkman, 1966; Marinković & Ayala, 1975a, b; Berger, 1976; Kirpichnikov & Muske, 1980). A more distinctive phenomenon is that matings between dissimilar homozygotes have greater fecundity than matings between like homozygotes. This mode of selection in which association of opposites increases their fitness has been named *alloprocoptic* selection (Serradilla & Ayala, 1983). How large a role it may play in preserving allozyme and other polymorphisms must wait further investigation. The generality of the interactions between male and female genotypes with respect to fecundity warrants in any case the design of experiments such as those reported in Tables 3-6, in which the fitness of all possible male/female genotypic combinations can be evaluated. Few like experiments have been made (Bundgaard & Christiansen, 1972).

References

- Anderson, W. W. & Watanabe, T. K., 1974. Selection by fertility in *Drosophila pseudoobscura*. *Genetics* 77: 559-564.
- Ayala, F. J., Powell, J. R., Tracey, M. L., Mourao, C. A. & Pérez-Salas, S., 1972. Enzyme variability in the *Drosophila willistoni* group. IV. Genic variation in natural populations of *Drosophila willistoni*. *Genetics* 70: 113-139.
- Berger, E. M., 1976. Heterosis and the maintenance of enzyme polymorphism. *Am. Nat.* 110: 823-839.
- Brittnacher, J. G., 1981. Genetic variation and genetic load due to the male reproductive component of fitness in *Drosophila*. *Genetics* 97: 719-730.
- Bundgaard, J. & Christiansen, F. B., 1972. Dynamics of polymorphisms: I. Selection components in an experimental population of *Drosophila melanogaster*. *Genetics* 71: 439-460.
- Cockerham, C. C., Burrows, P. M., Young, S. S. & Prout, T., 1972. Frequency-dependent selection in random-mating populations. *Am. Nat.* 106: 493-515.
- Gilbert, D. G., Richmond, R. C. & Sheehan, K. B., 1981. Studies of esterase 6 in *Drosophila melanogaster*. VII. The timing of remating in females inseminated by males having active or null alleles. *Behav. Genet.* 11: 195-208.

- Gilbert, D. G. & Richmond, A. C., 1982. Esterase 6 in *Drosophila melanogaster*. Reproductive function of active and null males at low temperature. *Proc. natn. Acad. Sci. U.S.A.* 79: 2962-2966.
- Hadeler, K. P. & Liberman, U., 1975. Selection models with fertility differences. *J. Math. Biol.* 2: 19-32.
- Kirpichnikov, V. S. & Muske, G. A., 1980. The adaptive value of biochemical polymorphisms in animal and plant populations. *Genetica* 52/53: 183-193.
- Knoppien, P., Pot, W. & Van Delden, W., 1980. Effects of rearing conditions and age on the difference in mating success between alcohol dehydrogenase genotypes of *Drosophila melanogaster*. *Genetica* 51(3): 197-202.
- Laurie-Ahlberg, C. C. & Weir, B. S., 1979. Allozymic variation and linkage disequilibrium in some laboratory populations of *Drosophila melanogaster*. *Genetics* 92: 1295-1314.
- Marinković, D. & Ayala, F. J., 1975a. Fitness of allozyme variants in *Drosophila pseudoobscura*. I. Selection at the Pgm-1 and MC-2 loci. *Genetics* 79: 85-95.
- Marinković, D. & Ayala, F. J., 1975b. Fitness of allozyme variants of *Drosophila pseudoobscura*. II. Selection at the Est-5, Odh and Mdh-2 loci. *Genet. Res. Camb.* 24: 137-149.
- Maynard Smith, J., 1956. Fertility, mating behavior and sexual selection in *Drosophila subobscura*. *J. Genet.* 54: 261-279.
- McKenzie, J. A. & Fcgent, J. C., 1980. Mating patterns of virgin and inseminated *Drosophila melanogaster* of different alcohol dehydrogenase (Adh) genotypes. *Experientia* 36: 1160-1161.
- Milkman, R. D., 1966. Heterosis as a major cause of heterozygosity in nature. *Genetics* 55: 493-495.
- Petit, C. & Ehrman, L., 1969. Sexual selection in *Drosophila*. In: *Evolutionary Biology*, Vol. 3, ed. by Dobzhansky, Hecht & Steere, pp. 177-223. Meredith Corp.
- Petit, C., Kitagawa, O., Takanashi, E. & Mouaud, D., 1980. The failure to obtain sexual isolation by artificial selection. *Genetica* 54: 213-219.
- Pot, W., Van Delden, W. & Kruijt, J. P., 1980. Genotypic differences in mating success and the maintenance of the alcohol dehydrogenase polymorphism in *Drosophila melanogaster*: No evidence for overdominance or rare genotype mating advantage. *Behav. Genet.* 10(1): 43-58.
- Serradilla, J. M. & Ayala, F. J., 1983. Alloproceptive selection: A mode of natural selection promoting polymorphism. *Proc. natn. Acad. Sci. U.S.A.* 80: 2022-2025.
- Sokal, R. R. & Rohlf, F. J., 1969. *Biometry*. Freeman & Co., San Francisco.
- Sved, J. & Ayala, F. J., 1970. A population cage test for heterosis in *Drosophila pseudoobscura*. *Genetics* 66: 97-113.
- Tosić, M. & Ayala, F. J., 1981. Density- and frequency-dependent selection at the Mdh-2 locus in *Drosophila pseudoobscura*. *Genetics* 97: 679-701.

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