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Experiments designed to obtain evidence on selection for alpha-esterase haplotypes in laboratory populations of D.montana.

The gametic disequilibrium in natural populations among the 16 possible haplotypes of active (A) and null (0) alleles at the four closely-linked alpha-esterase loci in D.montana is attributed to natural selection (Baker & Kaeding 1981). We devised experiments to see if this selection could be demonstrated in laboratory populations.

In 1980 a control population cage was established from five ♂♂ and five ♀♀ from each of 75 isofemale strains collected in Utah the previous summer (1979). One year later (1981) this cage was sampled by crossing individual flies to homozygous null flies and electrophoresing their offspring to determine the two haplotypes in each sampled fly. Table 1 shows the observed numbers of the 16 different haplotypes in a sample taken from the Utah natural population in the summer of 1979 (data from Baker & Kaeding 1981), and the observed numbers in the sample taken from the control population cage after one year. The expected numbers (based on gametic equilibrium) of each haplotype and the ratio of observed/expected is also given. Table 2 provides the frequencies of the active alleles at the four loci observed in the natural population as well as the frequencies in the control population cage after a year. It can be seen from these two tables that gametic disequilibrium was observed in both the natural population and the control cage population although it was not as striking in the latter as in the former. It is also evident that the allelic frequencies were almost the same in the natural and laboratory populations although the frequency of active alleles at locus four was significantly lower in the population cage.

Now that we knew what the equilibrium allelic frequencies were in the laboratory and which haplotypes were favored and which disfavored under these conditions, it was possible to establish an experimental population cage in which the allelic frequencies were approximately the same as in the control cage population but in which the frequency of haplotypes in disfavor were in the majority. If selection were operating, the frequency of disfavored haplotypes should rapidly decrease with time in this experimental cage.

Table 1. (N = number of chromosomes)

Haplotype				Utah Nat. Population			Control Cage Pop.			Exper. Pop.	
1	3	2	4	Obs.	Exp.	Obs./Exp.	Obs.	Exp.	Obs./Exp.	N	Freq.
0	0	0	0	0	9.4	0	1	9.6	.10	480	.3125*
A	0	0	0	2	4.0	.50	1	3.8	.26		
0	A	0	0	13	11.1	1.2	13	15.1	.86		
0	0	A	0	8	11.5	.70	15	11.3	1.3		
0	0	0	A	5	5.1	.98	7	2.0	3.5		
A	A	0	0	18	4.7	3.8	7	5.9	1.2	96	.0625
0	0	A	A	22	6.2	3.5	1	2.3	.43	96	.0625
A	0	0	A	4	2.2	1.8	2	0.8	2.5		
0	A	A	0	18	13.5	1.3	23	17.7	1.3	288	.1875
A	0	A	0	5	4.9	1.0	14	4.4	3.2		
0	A	0	A	4	6.0	.67	4	3.1	1.3	192	.1250
0	A	A	A	0	7.3	0	1	3.6	.28		
A	0	A	A	0	2.7	0	0	0.9	0		
A	A	0	A	0	2.6	0	0	1.2	0		
A	A	A	0	1	5.8	.17	1	6.7	.15	384	.2500*
A	A	A	A	0	3.1	0	0	1.4	0		
				100	100.1		90	89.8		1536	1.0000

Table 2. (N = number of chromosomes sampled)

Date	Population	Frequency of Active Allele 1	3	2	4	N
1979	Utah, Natural	.30	.54	.55	.35	100
1980	Control Cage	.28	.61	.54	.17	90
Jun 82	Initial Exp. Cage	.31	.62	.50	.19	1536
Jul 82	Exp. 1st sample	.32	.74	.46	.17	100
Sep 82	Exp. 2nd sample	.41	.81	.64	.16	210
Dec 82	Exp. 3rd sample	.29	.58	.51	.24	252
Mar 83	Exp. 4th sample	.38	.62	.64	.27	254

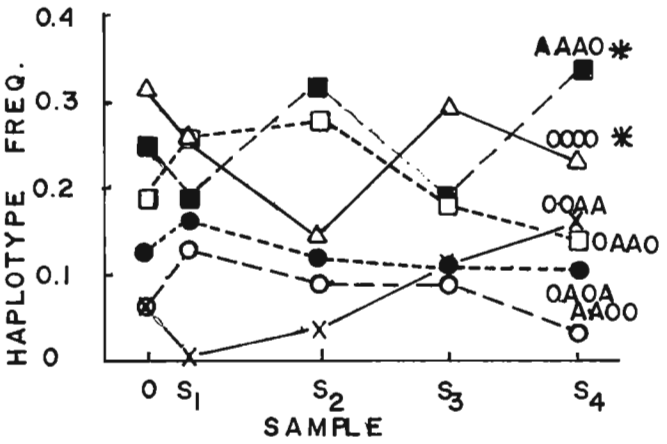

FIGURE 1.

The experimental cage was started with 768 flies comprised of equal numbers of ♂♂ and ♀♀ of the following genotypes: 96 00AA/AA00 + 192 0AA0/0A0A + 96 0AA0/0000 + 384 AAA0/0000. (The four genes are given in their order on the chromosome: 1, 3, 2, 4.) Table 1 gives the numbers of the six haplotypes used to initiate the experimental cage and an asterisk denotes a haplotype in extreme disfavor in both the control cage and the natural population. Thus, at a minimum, over 50% of the chromosomes in the experimental cage were putatively detrimental. The ratio of the six haplotypes initiating the experimental cage was arranged so that the allelic frequencies of active and null alleles at the four loci matched those of the control cage (see Table 2). Note that the cage was started from heterozygotes made from crosses of homozygous lines of the six haplotypes established previously from a population collected in Gothic, Colorado.

The experimental cage was set up in June 1982 as previously described, and in July F_1 larvae were removed, reared, and testcrossed to determine the relative frequencies of the six haplotypes among a sample of 100 chromosomes. Subsequent samples were taken in September 1982, December 1982, and March 1983. The haplotype frequencies observed in these samples are pictured in Figure 1, and the allelic frequencies observed are given in Table 2. There is no indication in these data of any consistent change in either haplotype frequencies or allelic frequencies over the period of experimentation. Certainly the detrimental haploytypes (asterisks) did not decrease in frequency. We estimate that the generation time in the cages was about a month, making a total of around nine generations between the first and the last sample. It was planned to take a last sample after an additional six months but a laboratory accident eliminated the population. These data provide no evidence of selection acting over the limited period of this experiment.

Supported by NSF Grant DEB-79-12336. References: Baker, W.K. & E.A.Kaeding 1981, Amer.Nat. 117:804-809.

Banerjee, I. & A.S.Mukherjee. University of Calcutta, India. Activation of potential initiation sites of DNA replication by Puromycin: evidence from fibre autoradiography.

In eukaryotes replicons occur in clusters (Edenburg & Huberman 1975). Such clusters are organized into replicon families and replicate at a given rate during DNA synthesis (S) phase (Van't Hof & Bjerknes 1978).

In the present study, DNA fibre autoradiography was used to determine the replication properties of polytene DNA at the level of initiation and chain elongation. We have used Puromycin which is a potent inhibitor of protein synthesis for monitoring the property. Our work is based on previous evidence supporting the idea that there are two different protein pools, one responsible for the control of DNA synthesis at the level of initiation and the