# SELECTION IN EXPERIMENTAL POPULATIONS OF DROSOPHILA MELANOGASTER WITH DIFFERENT GENETIC BACKGROUNDS[1]

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THE heterozygous carriers of some genetic variants are superior in fitness to the corresponding homozygotes. The causation of this hybrid vigor or heterosis (euheterosis according to DOBZHANSKY 1952) constitutes an important but as yet little understood problem. One possibility is that some gene alleles are "overdominant" with respect to fitness, and thus give heterosis in heterozygous conditions with certain other alleles at the same locus. On the other hand heterosis may be caused by interaction of nonallelic, but usually more or less closely linked gene complexes sometimes referred to as "supergenes" (DARLINGTON and MATHER 1949). Experimental discrimination between overdominance at a single locus and supergene interactions is a difficult matter. TEISSIER (1943), BUZZATI-TRAVERSO (1952), GUSTAFSSON and NYBOM (1950), and MUKAI and BURDICK (1959, 1960, 1961) described instances of heterotic effects of spontaneously arisen or induced mutants, which seem to represent clear cases of overdominance. JINKS (1955), using the method of diallel crosses, was, however, able to detect interaction of epistatic nature in some previously reported instances of overdominance. ROBINSON and COMSTOCK (1955) attempted to evaluate the relative importance of dominant and overdominant genes in the production of heterosis in corn; the available data, however, do not permit a definite answer to be given. The experiments described in the present article were designed to study the possible occurrence of heterosis following hybridization of mutant and wild-type strains of *Drosophila melanogaster*. The results of these experiments were reported at the 11th International Congress of Genetics (POLIVANOV 1963).

## MATERIALS AND METHODS

Five mutant and 42 wild-type strains of *D. melanogaster* were used. The mutant strains were: ebony, Stubble (hi60Sb), brown$^{75}$, scarlet, polished, and forked (b-7f). The wild-type strains were Blacksburg (Virginia), Capetown (South Africa), Formosa, Riverside (California), Santiago (Chile), SYSP (a strain inbred by brother-sister matings for 219 generations, obtained from DR. J. C. KING), and 36 strains obtained from single wild females collected by the author at Canandaigua (Ontario County), Ellenville (Ulster County), Monroe (Orange County) and Commack (Suffolk County), New York State.

Twenty experimental populations were set up in plastic polyethylene cages made from refrigerator boxes of the size $11\frac{1}{2} \times 8 \times 4\frac{1}{2}$ inches. Some of the cages contained eight and

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others 12 food cups. One new cup was inserted, and one old removed, twice a week in the cages with eight, and three times a week in the ones with 12 cups, so that each cup remained in the cage for about four weeks. The populations were fed on Spassky's medium to which brewer's yeast and six drops of Fleischmann's yeast suspension were added. All populations were kept in a dark constant temperature room at 25 ± ½°C.

The 20 populations were divided into five series according to the mutant used. All the populations of a given series were started at the same time and with the same number of flies. Each series consisted of two pairs of populations. Two were monochromosomal populations in which the nonmutant chromosomes came from a single or a few homozygous flies from the SYSP inbred strain. The other two were polychromosomal populations in which the nonmutant chromosomes were derived from many (12 to 31) wild strains. The gene frequencies were estimated from egg samples taken one or several generations apart. In order to take the egg samples, two fresh food cups were inserted into a population cage for about 3 or 4 hours. Then these cups were removed and the surface of the food medium with the eggs deposited on it was sliced off and transferred into culture bottles where flies were raised. One subsample was taken from each cup, and the third was combined from the remainder of both cups. Approximately 150 eggs were used per bottle. The frequency of heterozygotes was estimated by progeny tests of 50 to 100 flies (both males and females) taken from all three subsamples.

The third-chromosome mutant ebony (*e*) was used in the first series. The strain was obtained from the University of Rochester; however its exact origin is unknown. All populations were started with $F_1$ ebony × wild-type hybrids. For polychromosomal population, hybrids between ebony and 12 wild-type strains (six laboratory and six Canandaigua) were used. The hybrids were obtained from 24 reciprocal crosses. Equal number of flies were introduced in the two polychromosomal populations from each cross. However, flies from different crosses were not represented in equal proportions. (Ten strains were represented by 34 flies each, one by 44, and one by 16.) The monochromosomal populations were set up with hybrids between ebony and SYSP. The term "oligochromosomal" populations would be more correct in this case since several SYSP third chromosomes were introduced into both populations. However, since "oligochromosomal" populations approached the "monochromosomal" condition because SYSP strain was highly inbred, the term "monochromosomal" population will be used. In order to obtain a desirable number of flies, several reciprocal crosses were made. Altogether 9 SYSP females, 14 SYSP males, 8 ebony females and 11 ebony males were used. The populations of this series were started with 400 flies in each.

For the second series, the third-chromosome mutant Stubble was used. The polychromosomal populations were started with hybrids between Stubble and 31 different wild-type strains. The hybrids were obtained from the crosses of two *Sb/In(3LR)Ubx*$^{101}$ females and four wild-type males. The two polychromosomal populations received equal number of flies from each cross, but again flies from different crosses were not represented in equal proportion. The wild-type strains were derived from several local populations. Twenty Monroe, nine Commack, one Ellenville, and one SYSP strains were used. Monochromosomal populations were started with +$^{SYSP}$/*Sb* hybrids. To obtain them six *Sb/In(3LR)Ubx*$^{101}$ females were crossed to a single SYSP male. However, since too few offspring were obtained from this cross, some +/*Sb* flies were mated together, and the populations were started with the mixture of $F_1$ and $F_2$. All the founders of the monochromosomal populations were, however, descendants of a single SYSP male, and, therefore, only two SYSP chromosomes were introduced in these populations. All populations of the second series were started with 192 flies in each. Since Stubble is a recessive lethal with a dominant phenotypic effect, the percentages of the heterozygotes were estimated by direct scoring of flies hatching from the egg samples.

Series 3 was started with flies doubly heterozygous for the second chromosome mutant brown$^{75}$ and the third chromosome mutant scarlet. The offspring of a single double heterozygous (+$^{SYSP}$/*bw*$^{75}$; +$^{SYSP}$/*st*) male and 16 *bw*$^{75}$;*st* females were used as the founders for monochromosomal populations. Each population was started with 25 pairs of flies. The polychromosomal populations were also started with 25 pairs. Each pair was derived from a different cross of two *bw*$^{75}$;*st* females with four wild-type males: 14 Monroe, nine Commack, one Ellenville and one SYSP strains were used.

Since forked is a sex-linked gene, the populations of the fourth series were started with hybrid females and mutant males. All flies were $F_1$ offspring of wild-type males and mutant females. Each population was started with 58 flies. Polychromosomal populations contained flies derived from the crosses of forked females to males from 16 Monroe, nine Commack, one Ellenville, and one SYSP strains. Only a single pair from each cross was used (except for Ellenville and SYSP, from which two pairs were taken). The monochromosomal populations were started with the offspring of a single SYSP male and seven forked females.

Finally, the fourth-chromosome mutant polished (*pol*) was used in Series 5. All the populations were started with $F_1$ hybrids, 31 wild-type strains being used in the polychromosomal populations. These strains were the same as in Series 2. Mutant females were crossed to wild-type males, and in most cases only one pair from each cross was included among the founders. The monochromosomal populations were started with offspring of a single $+^{SYSP}/pol$ male and two mutant females. All populations were started with 77 flies in each.

## RESULTS

The results are reported in Tables 1 to 6. The first number in each column indictates the percentages, while the second, separated from the first by a dash, gives the number of flies from which the percentages are calculated. The percentages of homozygous mutants were estimated on the basis of the whole samples, but percentages of heterozygotes were estimated from smaller sub-

TABLE 1

*Frequencies in percent of ebony homozygotes and heterozygotes in mono- and polychromosomal populations*

| | Mono-1 | | Mono-2 | | Poly-1 | | Poly-2 | |
|---|---|---|---|---|---|---|---|---|
| | Hom. | Het. | Hom. | Het. | Hom. | Het. | Hom. | Het. |
| Days | % N | % n | % N | % n | % N | % n | % N | % n |
| 21 | 29.5–183 | 75– 48 | 27.2–147 | 67– 48 | 22.5–160 | 54– 46 | 21.4– 94 | 68– 50 |
| 42 | 24.8–113 | 72– 50 | 23.3–116 | 70– 47 | 11.8–212 | 68– 50 | 20.3–172 | 52– 50 |
| 63 | 28.0–275 | 66– 50 | 30.0–253 | 48– 50 | 7.9–253 | 48– 50 | 7.6–276 | 42– 50 |
| 84 | 23.9–322 | 68–100 | 28.2–316 | 75–100 | 12.3–287 | 49–100 | 12.1–239 | 42–100 |
| 105 | 17.7–419 | 52–100 | 23.8–524 | 57–100 | 8.5–494 | 42–100 | 9.8–356 | 43–100 |
| 127 | 17.0–259 | 55–100 | 19.7–309 | 63–100 | 4.7–339 | 41–100 | 2.4–249 | 47–100 |
| 148 | 21.9–501 | 64–100 | 20.5–469 | 55–100 | 5.0–597 | 32–100 | 6.1–559 | 30–100 |
| 169 | 10.0–498 | 71–100 | 12.5–465 | 60–100 | 4.5–599 | 38–100 | 5.1–586 | 22–100 |
| 191 | 16.7–471 | 58– 92 | 11.6–355 | 50–100 | 4.8–462 | 23–100 | 3.3–461 | 28–100 |
| 213 | 17.3–387 | 49–100 | 14 –477 | 51–100 | 3.8–397 | 27–100 | 2.2–501 | 21–100 |
| 234 | 8.1–285 | 49–100 | 14.4–270 | 65– 97 | 3.9–260 | 28–100 | 3.0–330 | 31–100 |
| 255 | 10.0–438 | 51–100 | 13.0–339 | 57–100 | 2.8–467 | 33–100 | 1.9–474 | 31–100 |
| 276 | 11.1–342 | 46–100 | 10.3–474 | 46–100 | 1.9–410 | 24–100 | 1.2–491 | 20–100 |
| 308 | 7.2–388 | 41– 99 | 7.7–259 | 51–100 | 0 –239 | 23–100 | 0.8–246 | 24–100 |
| 329 | 7.5–161 | 51– 74 | 9.7–236 | 46– 97 | 0.7–419 | 24– 96 | 1.5–551 | 23– 97 |
| 365 | 6.0–331 | 36– 81 | 10.7–232 | 59– 44 | 1.3–238 | 13– 92 | 0.8–357 | 16– 74 |
| 421 | 3.7–456 | 24–100 | 11.2–589 | 53– 99 | 0.2–591 | 16– 99 | 0.6–515 | 19– 97 |
| 483 | 1.8–441 | 19–100 | 7.1–438 | 43–100 | 2.0–443 | 18–100 | 0.7–413 | 13–100 |
| 554 | 2.9–381 | 26–100 | 5.7–405 | 41– 98 | 0 –352 | 13–100 | 0.8–400 | 18–100 |
| 632 | 0.3–373 | 17–100 | 3.8–400 | 38–100 | 0.5–430 | 22–100 | 0.5–375 | 14–100 |
| 702 | 0.6–348 | 13–100 | 0.8–388 | 27–100 | 0.2–418 | 9–100 | 0 –454 | 12–100 |
| 772 | 0.5–446 | 16–100 | 1.9–420 | 28–100 | 0.2–477 | 9–100 | 0.9–455 | 13–100 |
| 844 | 0.2–429 | 8–100 | 2.9–343 | 22–100 | 0.2–429 | 3–100 | 0.5–401 | 13–100 |
| 936 | 0 –417 | 6–100 | 1.3–379 | 21–100 | 0.2–423 | 6–100 | 0 –441 | 7–100 |

TABLE 2

*Frequencies in percent of Stubble heterozygotes in monochromosomal and polychromosomal populations*

| Days | Mono-1 % N | Mono-2 % N | Poly-1 % N | Poly-2 % N |
|---|---|---|---|---|
| 26 | 60.1–351 | 64.2–346 | 54.8–396 | 48.8–416 |
| 49 | 58.1–310 | 58.4–344 | 47.2–405 | 43.9–394 |
| 72 | 62.0–355 | 61.2–307 | 36.2–384 | 25.2–413 |
| 95 | 54.7–362 | 58.6–343 | 25.3–363 | 28.6–371 |
| 119 | 56.5–230 | 58.2–294 | 15.1–371 | 16.2–370 |
| 144 | 50.9–281 | 58.3–187 | 17.2–377 | 11.3–409 |
| 165 | 55.0–338 | 57.1–336 | 5.4–387 | 6.2–422 |
| 187 | 51.6–341 | 46.9–339 | 6.5–449 | 3.7–406 |
| 210 | 42.9–317 | 46.6–290 | 5.6–428 | 2.1–281 |
| 234 | 40.2–336 | 35.5–276 | 3.3–456 | 2.5–447 |
| 279 | 42.1–216 | 19.7–233 | 0.5–432 | 0.7–428 |
| 325 | 17.6–370 | 9.7–371 | 0.5–418 | 0.2–403 |
| 371 | 10.5–391 | 5.3–243 | 0 –426 | 0 –386 |
| 417 | 5.1–333 | 3.3–306 | 0 –437 | 0 –427 |
| 463 | 7.7–326 | 3.6–345 | 0.038–5170 | ..... |
| 548 | 2.4–338 | 0 –318 | ..... | ..... |

samples (taken from the total samples) of wild-type flies which were tested for heterozygosity. The percentages of Stubble heterozygotes were calculated from the whole samples since this gene is a recessive lethal with a dominant phenotypic effect. Gene frequencies were estimated using the method of maximum likelihood. Let the number of flies in a sample be $N$, and the number of flies tested for heterozygosity be $n$. The number of homozygous wild-type flies among the tested ones is $X_1$, the number of heterozygotes (also among the tested flies) $X_2$, and the

TABLE 3

*Frequencies in percent of scarlet homozygotes and heterozygotes in monochromosomal and polychromosomal populations*

| Days | Mono-1 Hom. % N | Mono-1 Het. % n | Mono-2 Hom. % N | Mono-2 Het. % n | Poly-1 Hom. % N | Poly-1 Het. % n | Poly-2 Hom. % N | Poly-2 Het. % n |
|---|---|---|---|---|---|---|---|---|
| 23 | 28.1–377 | 71– 51 | 24.4–360 | 70– 61 | 19.3–388 | 56– 81 | 18.7–265 | 56– 54 |
| 46 | 26.5–396 | 87– 69 | 26.2–367 | 80– 69 | 9.1–275 | 42–100 | 12.0–396 | 53– 97 |
| 69 | 25.1–370 | 81– 75 | 22.1–294 | 76– 86 | 6.0–331 | 38–100 | 9.8–418 | 56–100 |
| 92 | 23.1–324 | 72–100 | 21.5–307 | 84– 82 | 5.5–307 | 41–100 | 13.1–359 | 54–100 |
| 115 | 21.1–256 | 72–100 | 21.7–313 | 71–100 | 5.8–382 | 33–100 | 8.1–384 | 44–100 |
| 138 | 16.5–297 | 68– 37 | 21.7–337 | 68– 57 | 1.6–318 | 28– 71 | 3.1–420 | 52– 73 |
| 184 | 12.4–387 | 63–100 | 15.6–353 | 61–100 | 1.6–379 | 25–100 | 5.5–421 | 39–100 |
| 230 | 17.1–380 | 58–100 | 23.9–330 | 74–100 | 2.4–290 | 23–100 | 6.4–435 | 35–100 |
| 276 | 13.2–363 | 41–100 | 21.4–267 | 68–100 | 1.7–347 | 20–100 | 6.7–416 | 43–100 |
| 343 | 14.2–423 | 56–100 | 14.5–303 | 64–100 | 1.5–410 | 12–100 | 5.5–434 | 30–100 |
| 415 | 9.7–268 | 56–100 | 12.2–328 | 60–100 | 0 –414 | 7–100 | 3.6–420 | 33–100 |
| 489 | 9.6–416 | 41–100 | 6.0–399 | 50–100 | 0 –490 | 8–100 | 3.2–371 | 36–100 |

TABLE 4

*Frequencies in percent of brown$^{75}$ homozygotes and heterozygotes in mono- and polychromosomal populations*

| Days | Mono-1 Hom. % N | Mono-1 Het. % n | Mono-2 Hom. % N | Mono-2 Het. % n | Poly-1 Hom. % N | Poly-1 Het. % n | Poly-2 Hom. % N | Poly-2 Het. % n |
|---|---|---|---|---|---|---|---|---|
| 23 | 23.6–377 | 54– 52 | 19.4–360 | 63– 59 | 15.7–388 | 54– 80 | 9.8–265 | 53– 58 |
| 46 | 13.9–396 | 67– 82 | 22.3–367 | 61– 69 | 8.4–275 | 37–100 | 8.6–396 | 37– 94 |
| 69 | 11.9–370 | 64– 73 | 21.1–294 | 64– 80 | 13.9–331 | 46–100 | 14.6–418 | 53–100 |
| 92 | 14.2–324 | 58–100 | 17.6–307 | 65– 89 | 7.8–307 | 58–100 | 8.3–359 | 45–100 |
| 115 | 14.8–286 | 54–100 | 16.6–313 | 58–100 | 8.9–382 | 40–100 | 4.2–384 | 45–100 |
| 138 | 16.2–297 | 63– 35 | 15.1–337 | 75– 68 | 10.4–318 | 52– 65 | 5.9–420 | 29– 70 |
| 184 | 8.3–387 | 52–100 | 15.3–353 | 65–100 | 9.8–379 | 52–100 | 3.1–421 | 29–100 |
| 230 | 8.9–380 | 45–100 | 14.5–330 | 55–100 | 6.9–290 | 34–100 | 4.1–435 | 31–100 |
| 276 | 4.7–363 | 42–100 | 15.4–267 | 56–100 | 8.4–347 | 49–100 | 3.8–416 | 40–100 |
| 343 | 4.5–423 | 37–100 | 5.6–303 | 54–100 | 5.6–410 | 31–100 | 2.5–434 | 18–100 |
| 415 | 3.4–268 | 29–100 | 10.1–328 | 62–100 | 3.6–414 | 33–100 | 1.9–420 | 18–100 |
| 489 | 2.6–416 | 34–100 | 13.5–399 | 47–100 | 3.4–409 | 27–100 | 2.7–371 | 24–100 |

number of homozygous mutants in the whole sample $X_3$. The proportions of each genotype, $\alpha$, $\beta$, and $\gamma$, are estimated as follows:
Proportion of wild-type homozygotes:

$$\hat{\alpha} = \frac{N - X_3}{N} \frac{X_1}{n}$$

Proportion of heterozygotes:

$$\hat{\beta} = \frac{N - X_3}{N} \frac{X_2}{n}$$

TABLE 5

*Frequencies in percent of polished homozygotes and heterozygotes in mono- and polychromosomal populations*

| Days | Mono-1 Hom. % N | Mono-1 Het. % n | Mono-2 Hom. % N | Mono-2 Het. % n | Poly-1 Hom. % N | Poly-1 Het. % n | Poly-2 Hom. % N | Poly-2 Het. % n |
|---|---|---|---|---|---|---|---|---|
| 25 | 25.8–414 | 67–100 | 24.7–426 | 69–100 | 19.0–437 | 51–100 | 17.5–423 | 57–100 |
| 50 | 18.6–409 | 58– 93 | 18.8–420 | 55– 95 | 20.7–391 | 58– 98 | 16.4–391 | 59–100 |
| 72 | 20.2–376 | 54– 89 | 15.9–372 | 60–100 | 14.5–401 | 61– 95 | 15.3–392 | 57– 95 |
| 95 | 13.3–422 | 58– 95 | 14.7–360 | 59– 91 | 11.6–424 | 57– 94 | 17.8–421 | 57– 95 |
| 141 | 9.7–350 | 48–100 | 7.2–377 | 50–100 | 9.3–193 | 59–100 | 9.3–322 | 50–100 |
| 187 | 4.9–366 | 44–100 | 6.7–385 | 36–100 | 9.9–363 | 43–100 | 8.5–318 | 42–100 |
| 239 | 4.1–342 | 40–100 | 6.7–373 | 34–100 | 5.2–346 | 45–100 | 6.4–313 | 46–100 |
| 281 | 4.3–369 | 34–100 | 3.2–377 | 39–100 | 4.2–337 | 41–100 | 4.5–402 | 39–100 |
| 326 | 4.9–391 | 33–100 | 2.1–391 | 26–100 | 0.8–378 | 26–100 | 6.8–382 | 32–100 |
| 373 | 4.8–336 | 26–100 | 0.8–402 | 22–100 | 3.2–247 | 23–100 | 1.8–381 | 24–100 |
| 442 | 3.0–267 | 14–100 | 2.9–384 | 17–100 | 1.6–377 | 27–100 | 1.1–372 | 20–100 |

TABLE 6

*Frequencies in percent of forked males in monochromosomal and polychromosomal populations*

| Days | Mono-1 % N | Mono-2 % N | Poly-1 % N | Poly-2 % N |
|---|---|---|---|---|
| 22 | 66.8–217 | 64.3–216 | 72.8–217 | 66.8–207 |
| 45 | 56.0–191 | 59.6–203 | 55.7–185 | 59.3–118 |
| 68 | 49.1–232 | 50.8–238 | 51.0–143 | 54.4–125 |
| 92 | 55.8–226 | 45.2–217 | 46.8–188 | 51.9–183 |
| 115 | 47.7–218 | 42.6–195 | 54.2–144 | 43.9–132 |
| 139 | 42.5–228 | 43.8–208 | 48.2–191 | 41.9–148 |
| 161 | 38.1–202 | 35.2–125 | 46.9–192 | 30.2–205 |
| 186 | 45.6–206 | 38.7–217 | 47.8–178 | 42.6–169 |
| 208 | 38.5–218 | 33.0–191 | 37.6–189 | 38.0–129 |
| 233 | 34.2–170 | 39.9–178 | 31.7–161 | 19.0–168 |
| 257 | 39.8–201 | 30.3–217 | 31.9–185 | 17.2–151 |
| 303 | 33.5–203 | 25.3–170 | 17.5–211 | 12.4–185 |
| 346 | 27.6–185 | 26.1–207 | 17.6–222 | 13.4–194 |
| 393 | 23.5–213 | 27.9–201 | 10.1–188 | 2.9–170 |
| 438 | 27.9–190 | 24.4–172 | 5.3–207 | 3.8–157 |
| 509 | 21.9–205 | 26.8–194 | 1.6–185 | 1.2–173 |
| 576 | 14.2–212 | 20.1–174 | 0.5–200 | 1.6–186 |

Proportion of mutant homozygotes:

$$\hat{\gamma} = \frac{X_3}{N}$$

The knowledge of the frequency of each genotype permits the estimation of the gene frequency directly without assumptions of equilibrium or Hardy-Weinberg distribution. The gene frequencies $p$ and $q$ are then simply the proportions of homozygotes plus one half of the proportion of heterozygotes.

$$\hat{p} = \hat{\alpha} + \tfrac{1}{2}\hat{\beta} \quad \text{and} \quad \hat{q} = \hat{\gamma} + \tfrac{1}{2}\hat{\beta}$$

where $p$ is for the wild-type and $q$ for the mutant allele. The gene frequencies estimated in such a way are presented graphically in Figures 1 to 5.

Further, if we denote $X_1/n$ as $f$, and $X_2/n$ as $1-f$, we can express the variance of $q$, as:

$$\sigma_{\hat{q}}^2 = \frac{(1+f)^2\gamma(1-\gamma)}{4N} + \frac{f(1-f)(1-\gamma)^2}{4n} + \frac{f(1-f)\gamma(1-\gamma)}{4Nn}$$

Deviations of the genotypic frequencies in a given generation from the expected values based on data of the preceding generation were utilized to estimate the relative adaptive value of each genotype. If the relative adaptive value of the heterozygote is 1, then the adaptive value of the wild-type homozygote is

$$1-\hat{t} = \hat{W}_1 = 2\frac{\hat{\alpha}_1}{\hat{\beta}_1}\frac{\hat{q}_0}{\hat{p}_0}$$

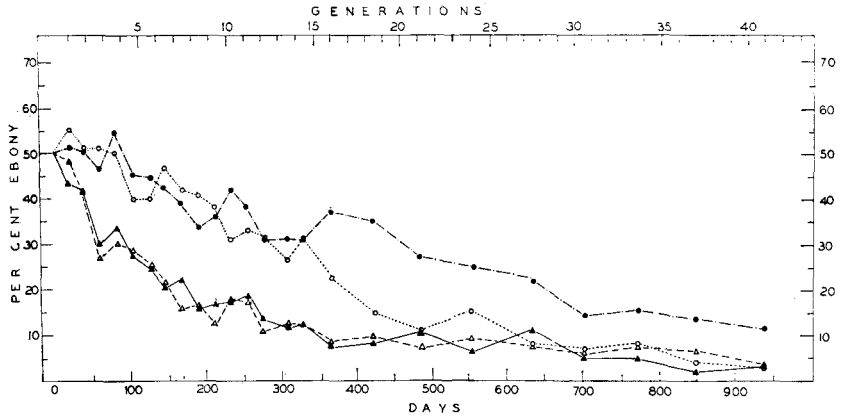


FIGURE 1.—Changes in frequencies of ebony in mono- and polychromosomal populations. Open circles: mono-1. Solid circles: mono-2. Solid triangles: poly-1. Open triangles: poly-2.

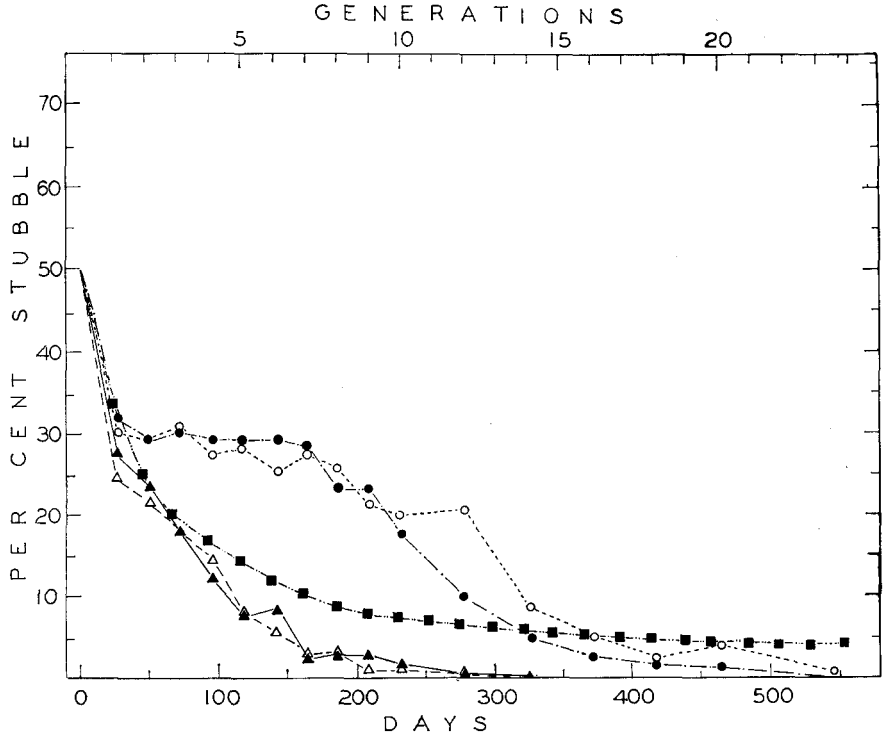


FIGURE 2.—Changes in frequencies of Stubble in mono- and polychromosomal populations. Open circles: mono-1. Solid circles: mono-2. Solid triangles: poly-1. Open triangles: poly-2. Solid squares: expected curve of elimination of recessive lethal.

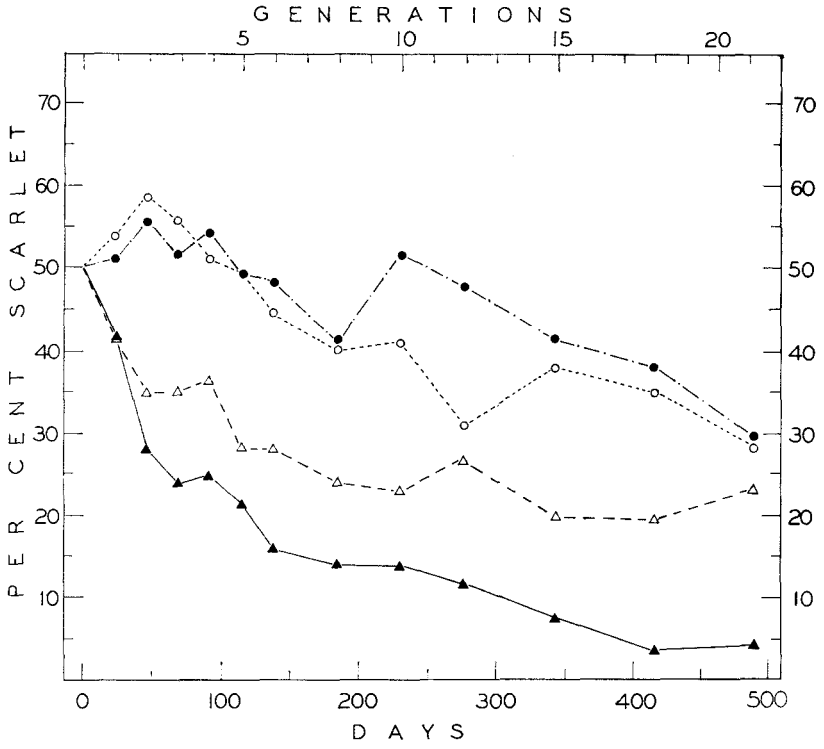


FIGURE 3.—Changes in frequencies of scarlet in mono- and polychromosomal populations. Open circles: mono-1. Solid circles: mono-2. Solid triangles: poly-1. Open triangles: poly-2.

and the adaptive value of mutant homozygotes is

$$1-\hat{s}=\hat{W}_2=2\frac{\hat{\gamma}_1}{\hat{\beta}_1}\frac{\hat{p}_0}{\hat{q}_0}$$

where the subscripts 1 and zero indicate given and preceding generations. The estimate of the generation length of 22 to 24 days given by BARKER (1962) has been used in the calculations.

In the case of heterosis or overdominance both selection coefficients $s$ and $t$ should be positive. In all other cases at least one of them should be negative.

The variances of the adaptive value are:

$$\sigma^2_{\hat{w}_1}=4\left[\left(\frac{f_1}{1-f_1}\right)^2\left(\frac{1}{p_0}\right)^4\sigma^2_{q_0}+\left(\frac{q_0}{p_0}\right)^2\left(\frac{1}{1-f_1}\right)^4\sigma^2_{f_1}\right]$$

$$\sigma^2_{\hat{w}_2}=4\left[\frac{1}{(1-\gamma_1)^4}\frac{1}{f_1^2}\left(\frac{p_0}{q_0}\right)^2\sigma^2_{\gamma_1}+\left(\frac{\gamma_1}{1-\gamma_1}\right)^2\frac{1}{f_1^4}\left(\frac{p_0}{q_0}\right)^2\sigma^2_{f_1}+\left(\frac{\gamma_1}{1-\gamma_1}\right)\frac{1}{f_1^2}\frac{1}{q_0^4}\sigma^2_{q_0}\right]$$

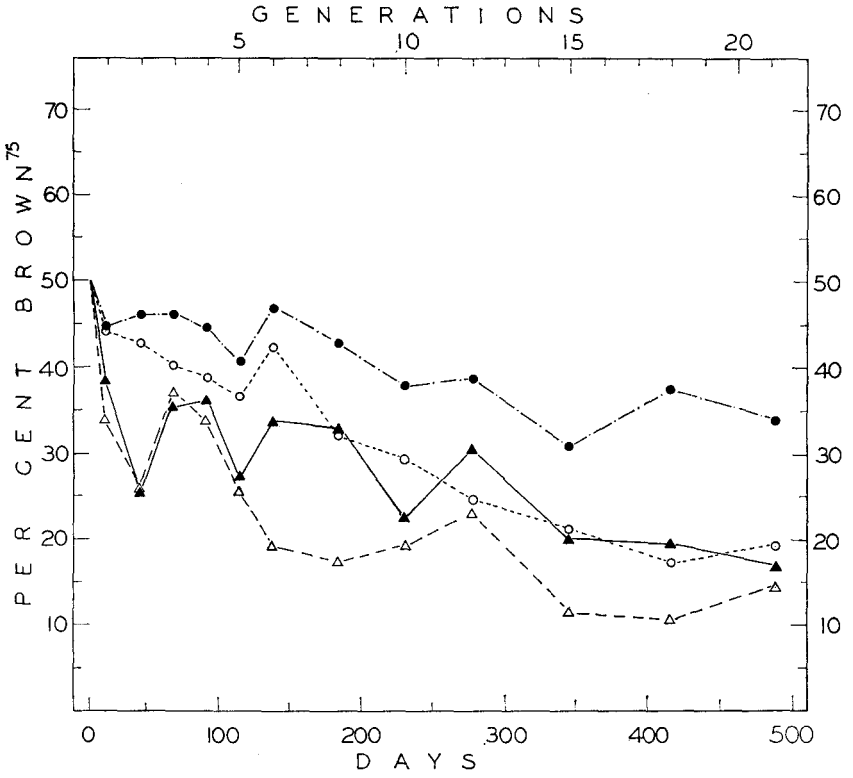


FIGURE 4.—Changes in frequencies of brown[75] in mono- and polychromosomal populations. Open circles: mono-1. Solid circles: mono-2. Solid triangles: poly-1. Open triangles: poly-2.

where $\sigma_f^2 = \frac{f(1-f)}{n}$

and $\sigma_\gamma^2 = \frac{\gamma(1-\gamma)}{N}$

Estimation of the variances of $W_1$ and $W_2$ from the combined data of the two ebony polychromosomal populations gave values of $\sigma^2_{\hat{w}_1}$ ranging from .01 to .19 and of $\sigma^2_{\hat{w}_2}$ from .003 to .06, the average values being .05 and .016 respectively. In those populations where the data were not combined these variances must be even larger. In the populations with Stubble where the percentage of heterozygotes was estimated on the basis of the whole samples, values of the variances for individual populations ranged from .006 to .145, with average being 0.041. The instances of large variances indicate a considerable error in the estimation of adaptive values for the intervals between two successive generations. But since deviations from the true value were more or less at random, the arithmetical means between individual estimates should give more reliable results.

If the adaptive values remain constant through the whole course of the experi-

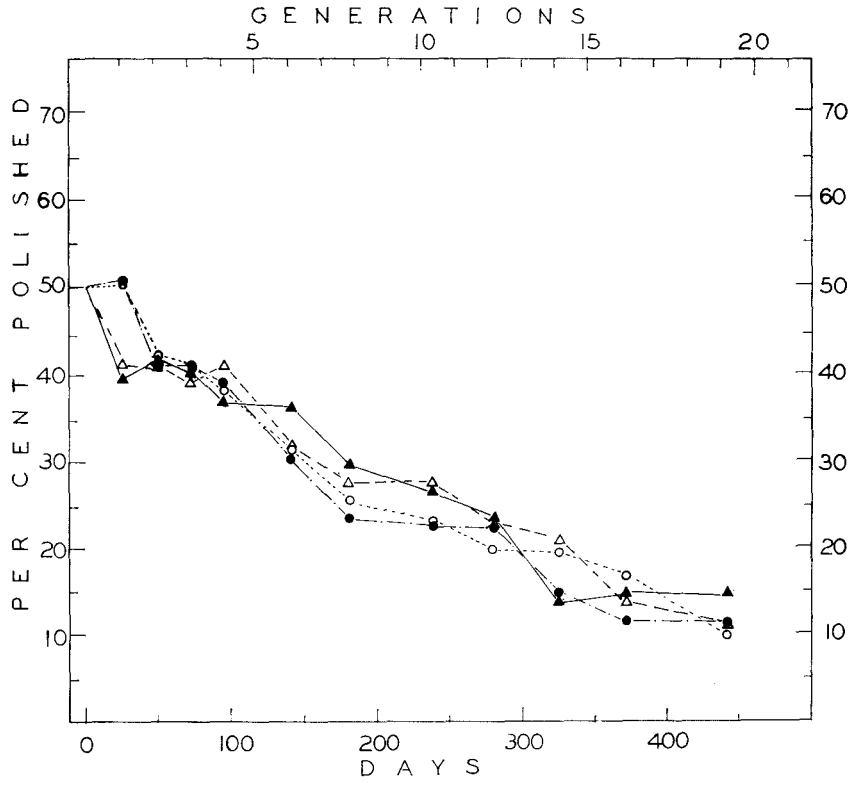


FIGURE 5.—Changes in frequencies of polished in mono- and polychromsomal populations. Open circles: mono-1. Solid circles: mono-2. Solid triangles: poly-1. Open triangles: poly-2.

ment, then the mean over all the data should give us satisfactory results. This was actually observed in the cases of all populations with the gene polished, and in monochromosomal populations with brown[75]. On the other hand, if the adaptive values were changing during the course of the experiment, then the mean of all data should not give satisfactory results. The experimental curves were then divided into segments in which the adaptive values seemed to remain more or less constant, and then the calculations were made for each segment separately. Of course, different estimates of adaptive value are obtained depending on where we divide the observed curve. The goodness of fit was estimated by calculating the expected gene frequency based on a given set of selection coefficients and by comparing these values with the experimental results. If the expected gene frequencies fall inside the confidence intervals of the experimental data, the values of the selection coefficients may be accepted as close to the true ones. On the other hand, if the expected values of the gene frequencies fall outside of the confidence intervals in many points, the estimates of the selection coefficients must be rejected.

As an example of the use of the method described above, the estimates of gene frequency of ebony are summarized in Table 7. The left column for each popu-

TABLE 7

*Frequencies (in percent) of the gene ebony*

| Generations | MONO-1 OBSERVED ± 2σ | MONO-1 EXPECTED s=.01 t=-.11 | .02 -.08 | -.25 -.43 | .06 -.08 | MONO-2 OBSERVED ± 2σ | MONO-2 EXPECTED s=.09 t=-.06 | .02 -.08 | .14 -.01 | POLY-1 OBSERVED ± 2σ | POLY-1 EXPECTED s=-.02 t=-.11 | .03 -.36 | -.05 -.14 | -.05 -.05 | POLY-2 OBSERVED ± 2σ |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 0 | 50.0 | 50.0 | 50.0 | | | 50.0 | 50.0 | 50.0 | 54.6 | 50.0 | 50.0 | 50.0 | 31.6 | | 50.0 |
| 1 | 55.9±6.1 | 48.8 | 48.8 | | | 51.5±7.0 | 48.0 | 48.8 | 52.5 | 43.6±7.5 | 49.1 | 45.5 | 29.9 | | 48.7±7.6 |
| 2 | 51.8±7.1 | 47.6 | 47.6 | | | 50.5±7.2 | 46.0 | 47.6 | 50.5 | 41.8±6.5 | 48.0 | 40.8 | 28.3 | | 41.0±7.2 |
| 3 | 51.8±6.0 | 46.3 | 46.3 | | | 46.8±6.6 | 44.2 | 46.3 | 48.5 | 30.0±7.0 | 46.8 | 35.9 | 26.7 | | 27.0±6.9 |
| 4 | 49.7±4.7 | 45.0 | 45.0 | | | 55.1±4.4 | 42.4 | 45.0 | 46.7 | 33.7±5.3 | 45.7 | 31.0 | 25.1 | | 30.6±5.5 |
| 5 | 39.1±5.0 | 43.6 | 43.8 | | | 45.6±4.6 | 40.4 | 43.8 | 44.9 | 27.7±4.5 | 44.3 | 26.3 | 23.5 | | 29.2±5.1 |
| 6 | 39.8±5.3 | 42.3 | 42.5 | | | 45.2±5.0 | 38.7 | 42.5 | 43.2 | 24.3±5.0 | 42.8 | 21.9 | 21.9 | 12.7 | 25.6±5.1 |
| 7 | 46.9±4.5 | 40.9 | 41.2 | | | 42.3±4.8 | 37.0 | 41.2 | 41.5 | 20.2±4.7 | 41.3 | 17.8 | 20.4 | 12.3 | 20.2±4.6 |
| 8 | 42.0±4.4 | 39.5 | 40.0 | | | 38.7±4.8 | 35.4 | 40.0 | 40.0 | 22.7±4.8 | 39.9 | 14.3 | 18.9 | 12.0 | 15.6±4.3 |
| 9 | 40.6±5.0 | 38.0 | 38.6 | | | 33.7±5.1 | 33.8 | 38.6 | 38.5 | 15.7±4.4 | | 11.3 | 17.5 | 11.6 | 16.8±4.6 |
| 10 | 37.6±5.0 | 36.7 | 37.4 | | | 36.0±4.9 | 32.2 | 37.4 | 37.0 | 16.8±4.6 | | 8.7 | 16.1 | 11.2 | 12.5±4.2 |
| 11 | 30.6±5.2 | 35.1 | 36.0 | 44.2 | | 42.2±5.1 | 30.7 | 36.0 | 35.7 | 17.3±4.8 | | 6.7 | 14.8 | 10.8 | 18.1±4.8 |
| 12 | 33 ±5.0 | 33.6 | 34.7 | 41.3 | | 37.8±5.0 | 29.3 | 34.7 | 34.4 | 18.7±5.0 | | 5.1 | 13.5 | 10.5 | 17.1±4.7 |
| 13 | 31.6±5.1 | 32.1 | 33.5 | 38.0 | | 31.0±5.0 | 27.9 | 33.5 | 33.2 | 13.7±4.4 | | | 12.4 | 10.1 | 11.1±4.1 |
| 14 | 26.4±5.0 | 30.6 | 32.2 | 34.5 | | 31.2±5.2 | 26.6 | 32.2 | 32.0 | 11.5±4.2 | | | 11.3 | 9.8 | 12.7±4.4 |
| 15 | 31.2±6.2 | 29.2 | 30.9 | 30.9 | 17.1 | 30.7±5.4 | 25.3 | 30.9 | 30.9 | 12.6±4.4 | | | 10.1 | 9.4 | 12.6±4.3 |
| 16 | 22.9±5.4 | 27.7 | 29.6 | 27.1 | 16.1 | 37.1±7.2 | 24.0 | 29.6 | 29.7 | 7.7±8.3 | | | 9.1 | 9.1 | 8.9±4.3 |
| 17 | | 26.3 | 28.4 | 23.1 | 15.1 | | 22.8 | 28.4 | 28.7 | | | | 8.2 | 8.8 | |
| 18 | 15.3±4.4 | 24.8 | 27.1 | 19.3 | 14.2 | 35.0±5.0 | 21.7 | 27.1 | 27.6 | 8.2±3.7 | | | 7.4 | 8.5 | 9.8±4.0 |
| 19 | | | 25.3 | 15.1 | 13.3 | | 20.6 | 25.3 | 26.6 | | | | 6.6 | 8.1 | |
| 20 | | | 24.7 | 12.4 | 12.4 | | 19.6 | 24.7 | 25.7 | | | | 5.9 | 7.8 | |
| 21 | 11.1±4.0 | | 23.6 | 9.6 | 11.6 | 27.1±5.1 | 18.6 | 23.6 | 24.8 | 10.8±4.0 | | | 5.3 | 7.5 | 7.2±3.4 |
| 22 | | | 22.5 | 7.3 | 10.9 | | | 22.5 | 23.9 | | | | 4.7 | 7.2 | |
| 23 | | | 21.4 | 5.4 | 10.2 | | | 21.4 | 23.1 | | | | 4.2 | 7.0 | |
| 24 | 15.5±4.5 | | 20.3 | 3.9 | 9.5 | 24.9±5.0 | | 20.3 | 22.4 | 6.5±3.4 | | | 3.7 | 6.7 | 9.7±4.0 |
| 25 | | | 19.2 | 2.0 | 8.9 | | | 19.2 | 21.6 | | | | 3.3 | 6.4 | |
| 26 | | | 18.2 | 1.6 | 8.3 | | | 18.2 | 20.9 | | | | 2.9 | 6.2 | |
| 27 | 8.7±3.8 | | 17.2 | 1.0 | 7.7 | 22.0±4.9 | | 17.2 | 20.2 | 11.4±4.2 | | | 2.7 | 5.9 | 7.5±3.5 |
| 28 | | | 16.3 | | 7.1 | | | 16.3 | 19.6 | | | | 2.3 | 5.7 | |
| 29 | | | 15.4 | | 6.6 | | | 15.4 | 19.0 | | | | 2.0 | 5.4 | |
| 30 | 7.0±3.4 | | 14.5 | | 6.2 | 14.2±4.5 | | 14.5 | 18.4 | 4.7±2.9 | | | 1.8 | 5.2 | 6.0±3.2 |
| 31 | | | 13.7 | | 5.7 | | | 13.7 | 17.8 | | | | | 5.0 | |
| 32 | | | 13.0 | | 5.3 | | | 13.0 | 17.3 | | | | | 4.8 | |
| 33 | 8.4±3.7 | | 12.2 | | 5.0 | 15.6±4.6 | | 12.2 | 16.8 | 4.7±2.9 | | | | 4.5 | 7.3±3.4 |
| 34 | | | 11.5 | | 4.6 | | | 11.5 | 16.3 | | | | | 4.3 | |
| 35 | | | 10.6 | | 4.3 | | | 10.6 | 15.8 | | | | | 4.1 | |
| 36 | 4.2±2.7 | | 10.0 | | 4.0 | 13.6±4.3 | | 10.0 | 15.3 | 1.7±1.6 | | | | 4.0 | 7.0±3.4 |
| 37 | | | 9.3 | | 3.7 | | | 9.3 | 14.9 | | | | | 3.8 | |
| 38 | | | 8.7 | | 3.4 | | | 8.7 | 14.5 | | | | | 3.6 | |
| 39 | | | 8.1 | | 3.2 | | | 8.1 | 14.1 | | | | | 3.4 | |
| 40 | 3.0±2.4 | | 7.6 | | 3.0 | 11.7±4.2 | | 7.6 | 13.7 | 3.2±2.4 | | | | 3.3 | 3.5±2.6 |

lation gives observed gene frequencies and twice their standard errors, giving confidence intervals at the 95 percent level.

The next column shows the expected gene frequencies, estimated on the assumption that selection coefficients are constant. Since, however, these coefficients did not give a satisfactory fit, the other sets of coefficients and the corresponding

gene frequencies are given in the following columns. Each of these last sets of selection coefficients fits the frequencies observed only during a particular interval of time. However, the expected gene frequencies are calculated beyond the limits of the corresponding period of time in order to show how the expected values would deviate from the observed ones. The data from both monochromosomal populations were combined together for the interval of the first 15 generations, and common selection coefficients were estimated for this period. However, for the rest of the experiment they were estimated separately for the two populations.

Since no statistically significant difference was observed between the two polychromosomal populations, the data were combined and the same sets of selection coefficients were estimated for these two populations. The results of the experiment with ebony shows that the initial intensity of selection against this mutant gene was considerably higher in polychromosomal than in monochromosomal

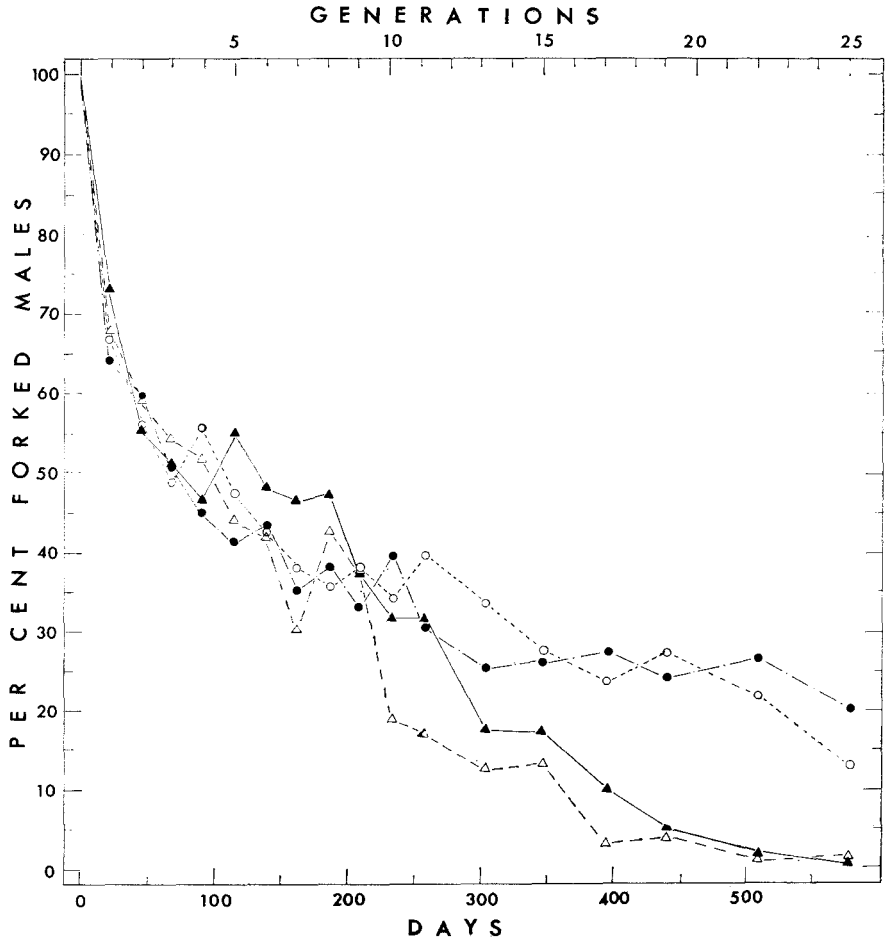


FIGURE 6.—Changes in frequencies of forked males in mono- and polychromosomal populations. Open circles: mono-1. Solid circles: mono-2. Solid triangles: poly-1. Open triangles: poly-2.

populations. In the later part of the experiment this difference tended, however, to disappear. The results thus show that ebony does not exhibit heterotic effects; this seems to contradict the findings of KALMUS (1945), TEISSIER (1947), JACOBS (1961) and MOREE and KING (1961). However, the results of the present experiment are in rather good agreement with those reported by GORDON (1935) on the release of ebony mutants in nature.

The selection coefficients for the other genes were estimated in the similar way. The expected gene frequencies are not given, and only the selection coefficients are indicated in Figure 7.

As could be seen from Figure 7, Stubble exhibited a temporary heterosis in monochromosomal populations, but this heterosis was soon lost. In polychromosomal populations, on the contrary, selection seems to act not only against the homozygotes, but against the heterozygotes as well. The adaptive value of mutant heterozygotes seems to be much lower than that of wild-type homozygotes. However, if the generation length is not correctly estimated, then disadvantage of Stubble heterozygotes might be considerably smaller than it was calculated. But even if we assume that the true length of generation was 11 instead of 23 days (a quite unrealistic assumption), the expected gene frequencies of a completely recessive lethal after 25 generations (275 days) would be approximately 3.6 percent, while the observed values at the corresponding point were .5 percent and .7 percent, showing that even under this assumption Stubble heterozygotes are at slight disadvantage.

The mutant scarlet gave results similar to the above. As in the case of ebony and Stubble, the initial rate of selection was higher in the polychromosomal than in the monochromosomal populations, and selection coefficients estimated on the assumption of constant adaptive values are not acceptable. Since the populations were started with only 50 flies each, it is perhaps not surprising that there was a consistent difference between the polychromosomal populations. The monochro-

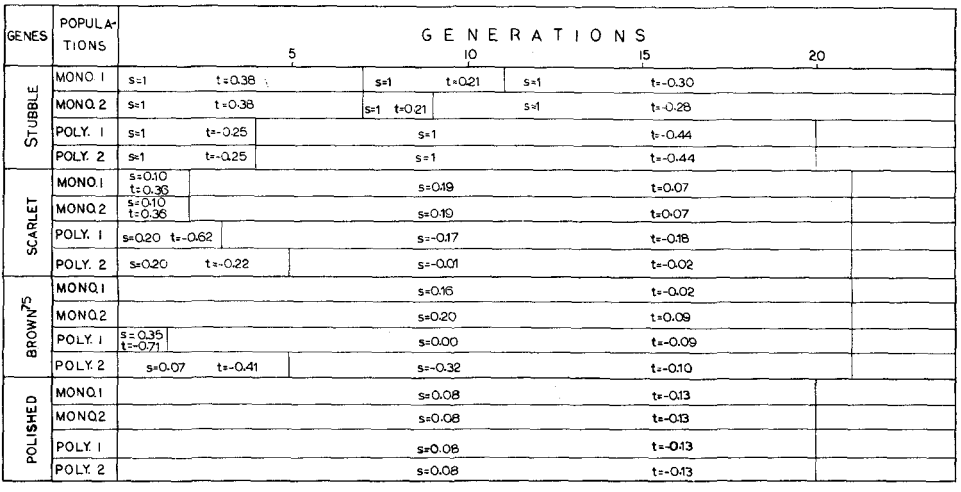


FIGURE 7.—Selection coefficients in the experimental populations.

mosomal populations, except for the interval between the 10th and 12th generations, when the difference between those populations was statistically significant, behave so similarly that they were treated together as one set. The polychromosomal populations, on the other hand, produced different results and were treated separately.

The experiment with scarlet indicates that the adaptive value of heterozygotes might be superior, almost equal, or inferior to that of both classes of homozygotes, depending on the rest of the genome (see Figure 7).

A similar range in adaptive values of heterozygotes was observed also in the case of brown $^{75}$, which was introduced in the same populations with scarlet. But in contrast with the behavior of scarlet, the two monochromosomal populations produced different results for brown$^{75}$. The selection coefficients in monochromosomal populations apparently remained constant throughout the whole course of the experiment (see Figure 7).

In both polychromosomal populations no single pair of $s$ and $t$ values satisfies the experimental data, but a division of the experimental curve in two or even in three segments, and estimation of $s$ and $t$ for each of them separately, does not produce very satisfactory results. In both populations, and especially in poly-1, the frequency of brown$^{75}$ fluctuated more than would be expected on the basis of experimental errors. It seems that two explanations of this phenomenon are possible: either fluctuation is caused by some uncontrolled environmental disturbances, or they were due to the extremely rapid changes in the adaptive values of different genotypes. The first explanation seems to be more probable since we have a similar rise in frequency of brown$^{75}$ in three out of four populations in the interval between the fifth and sixth generations. It is strange, however, that the polychromosomal populations should be more sensitive to the environmental fluctuations than the monochromosomal ones.

The fourth-chromosome mutant polished gives most uniform results. Except for the first generation there is no statistically significant difference between the four populations—two monochromosomal and two polychromosomal. They were treated as one set. There was no evidence of change of selection coefficients, and the single pair $s = .8$ and $t = -.13$ satisfies reasonably well all the experimental data.

In all previous estimations, the adaptive value of the heterozygotes was taken to be 1. However, if we assign the value 1 to the wild-type homozygotes, and let $h$ be the degree of dominance and $s$ the selection coefficient against the mutant homozygotes, then $s$ and $h$ can be easily calculated. When this is done, no significant correlation between $h$ and $s$ is found. The estimated value of the sample correlation coefficient, $r$, equals —.02, which for the sample size of 27 gives $-.39 \leq \rho \leq +.36$ as an approximate confidence interval at the 95 percent level. However, if certain doubtful cases were omitted, a more definite negative correlation could have been obtained.

In the experiment with forked, only mutant males were scored, and, therefore, it is impossible to estimate the adaptive values. It can only be said that the selection against forked proceeded similarly in all the populations for approximately

ten generations. Thereafter the monochromosomal and polychromosomal populations started to diverge more and more. At the 25th generation the frequencies of forked males in the monochromosomal populations were .142 and .201, while in the polychromosomal they were .005 and .016 (see Figure 6). The selection coefficients have changed at least in the polychromosomal populations. It is interesting to note that in the case of the third-chromosome mutants the difference between the polychromosomal and monochromosomal populations were strongly expressed at the beginning, while in later generations they tended to be less pronounced. In the case of forked we have just the opposite results.

## DISCUSSION

Although the utilization of hybrid vigor or heterosis in maize and in some other cultivated plants and animals is one of the most important practical applications of genetics to date, the nature and origin of this phenomenon remain insufficiently understood. It is differently interpreted by the classical and the balance theories of population structure, and the diverging interpretations seem to reflect two different biological philosophies (DOBZHANSKY 1955, 1963; CROW 1958; MULLER 1950, 1956). Even what would seem to be the simplest possible instance of heterosis, such as the apparent advantage of heterozygotes for certain mutant genes in experimental populations of Drosophila, is subject to different interpretations. This advantage may be due to overdominance, i.e., to a superior fitness produced by interaction of the mutant and the wild-type alleles of a single locus; or else the advantage may arise from the interactions of many linked loci in the chromosomes carrying the mutant and wild-type alleles.

The seemingly contradictory results of different competition experiments indicate that the effects of certain mutant genes on the adaptive value of their carriers depend probably on the effects of polygenic complexes linked with them (MERRELL 1953; MERRELL and UNDERHILL 1956; YANAGISHIMA 1953a,b; LUDWIN 1951; REED and REED 1950; BURI 1956; THOMPSON 1961; BOESIGER 1962).

DARLINGTON (1932) and MATHER (1943) argued that the antagonistic requirements of immediate fitness and future flexibility may be at least partially resolved by natural selection linking together favorably interacting genes. PARSONS and BODMER (1961) argued that overdominance is often a result of interaction of balanced polygenic complexes or supergenes, formed by natural selection. The evidence of the existence of such supergenes in natural populations can be found in the works of DOBZHANSKY and his collaborators (DOBZHANSKY 1950; DOBZHANSKY and LEVENE 1948; DOBZHANSKY, LEVENE, SPASSKY and SPASSKY 1959), who have shown that some chromosomal inversions in *D. pseudoobscura* act as such supergenes with heterotic properties. Evidence of the existence of supergenes has also been shown by BREESE and MATHER (1957, 1960), CLARKE and SHEPPARD (1960a,b,c), THODAY (1961), and others.

The question then arises whether the previously reported cases of heterosis for a specific mutant are due to overdominance at the single locus or to interaction of supergenes associated with this locus. SMATHERS (1961) came to the conclusion

that the heterosis observed by CARSON (1958) was due to interaction of polygenic blocks, and only in small degree due to heterozygosity at the loci of sepia, spineless and rough. Recently MERRELL (1963) has reported an interesting case of an apparent heterosis in *D. melanogaster*. The excess of $L^2/+$ heterozygotes in his case was due to the presence of recessive lethals in the wild-type chromosomes. This situation is very similar to that observed by the present author in Stubble monochromosomal populations.

In the present experiments it was expected that if heterosis is caused by overdominance at the locus in question, then it should manifest itself equally well in both monochromosomal and polychromosomal populations. If on the other hand it is due to the interaction of supergenes, it may be less pronounced in polychromosomal than in monochromosomal populations. A heterotic system established between different wild-type chromosomes may mask the heterotic effects of mutant genes.

The expectation based on the hypothesis of single-locus heterosis was fulfilled in no instance in the present experiments. Even in the cases where an apparent superiority of mutant heterozygotes was observed, as, for example, in the scarlet monochromosomal populations, the adaptive value of the heterozygotes was changing during the course of the experiment. No heterozygote superiority was observed in scarlet polychromosomal populations. In the case of brown$^{75}$, there was heterozygote superiority in mono-2 but not in mono-1 or in the polychromosomal populations, and hence we cannot assume that this superiority in mono-2 was an innate property of brown$^{75}$. These results are in better agreement with MATHER's hypothesis of balanced polygenes (MATHER 1955).

If supergenes found in natural populations produce heterotic interactions among each other, then it would be expected that the probability of the establishment of heterotic systems among different nonmutant chromosomes would be much higher in polychromosomal than in monochromosomal populations. This would increase the overall fitness of wild type and would be expected to result in a more rapid selection against the mutant chromosome in polychromosomal populations. This was actually observed in the experiments with the mutant genes on the second and third chromosomes.

The construction of laboratory populations always produces changes in the selective forces in comparison with those acting in the original founder populations. Now if the adaptive polygene complexes or supergenes were maintained in the founder populations by natural selection, then under new conditions new adaptive complexes might be formed by recombination between different chromosomes. It is likely then that the adaptive values of different mutant genotypes in the experimental populations would not remain constant or change gradually, but would change in a rather erratic way, since they would depend on the occurrence of particular crossing-over events. In other words, it would be expected that even the microevolutionary process in the experimental populations would proceed in a stochastic rather than deterministic way.

The results agree with these expectations. The changes in adaptive values of the different genotypes in the ebony mono-2 population were small, while in mono-1 at least one striking change took place. Abrupt changes in adaptive values,

attributable to single crossing-over events, were also observed in both Stubble monochromosomal populations, but the changes did not occur simultaneously. Changes of adaptive values were observed also in all polychromosomal populations, except in those with the gene polished. However, it is difficult to attribute the changes in most polychromosomal populations to a single crossing-over event, or to pin-point the exact time when they occurred.

The recombination between polygenic complexes found in different nonmutant and mutant chromosomes might lead to the formation of new adaptive supergenes not only in wild-type but also in mutant chromosomes. If the last event occurred, then a decrease in the intensity of selection against a mutant gene would be expected. The greater the number of different nonmutant chromosomes present in a population, the more probable is the increase in fitness of a mutant chromosome. And indeed, a marked decrease in the intensity of selection against mutant genes was observed in polychromosomal populations in the cases of brown, ebony and scarlet. But, on the other hand, in the Stubble polychromosomal populations the relative fitness of mutant chromosomes decreased with the course of time. Apparently crossing over in this case did not produce any improvements in mutant chromosomes.

Even more striking improvements in fitness of wild-type but not of mutant chromosomes were observed in the cases of the Stubble monochromosomal populations and in the ebony mono-1. The last results were obtained probably due to low viability of homozygotes for the original SYSP third chromosome. It is very likely that crossing over between mutant and SYSP chromosomes would increase the viability of SYSP but not of mutant chromosomes. The course of selection in laboratory populations may also be influenced if the viabilities of different genotypes depend upon the presence of other genotypes (TEISSIER and L'HERITIER 1937; LEWONTIN 1955; BEARDMORE 1963). According to WEISSBROT (personal communication), this differential facilitation or inhibition occurs owing to some chemical substances liberated by dead larvae. This phenomenon may explain the apparent negative heterosis observed in the scarlet, brown$^{75}$, and ebony polychromosomal populations. PARSONS and KROMAN (1960), by adjusting the concentrations of two melanine inhibitors—silver nitrate and phenyl-thiocarbamide—were able to create conditions lethal for the heterozygotes, but not for homozygous ebony and wild-type flies, so that both classes of homozygotes co-existed without forming viable hybrids. A differential facilitation or inhibition may explain also another unexpected result, the apparently higher adaptive value of brown$^{75}$ homozygotes in comparison with both homo and heterozygous wild-type flies in the poly-2 population. The higher viability of brown homozygotes in comparison with wild type was reported by NOZAWA (1958). According to him the advantage of brown homozygotes was due to the higher viability of brown larvae under crowded conditions.

It seems, however, that in the populations here described the recombinations between supergenes by crossing over played a more important role in determining the course of selection than did any differential inhibition or facilitation of carriers of different genotypes. The behavior of the fourth-chromosome mutant polished furnishes good evidence in support of the above conclusion. Crossing

over in the fourth chromosome must be an extremely rare event so that the gene complex in this chromosome is practically inherited as a unit. The populations with polished have shown no changes in the adaptive values of the genotypes involved, and no difference in the behavior of mono- and polychromosomal populations.

The behavior of sex-linked mutant forked suggests another possible mechanism. The presence of heterotic polygenic complexes is not expected in the X chromosome, since they would be exposed in males to hemizygous conditions, and in our experiment we do not see any significant difference in the initial rate of selection against this mutant between mono- and polychromosomal populations. On the other hand, recombination between different chromosomes may produce new types of nonmutant X chromosomes, better adapted to the cage conditions than the original ones. The probability of such an improvement of nonmutant chromosomes would be greater in polychromosomal than in monochromosomal populations. This may explain why we have a higher selection rate against forked in polychromosomal populations in late but not in earlier generations (see Figure 6). However, since only males were scored in this experiment, it is difficult to say something more definite about the mechanisms which operated in these populations. One can only say that the adaptive value even of a sex-linked mutant had changed in the course of time.

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## SUMMARY

The behavior of six mutant genes—brown$^{75}$, ebony, Stubble, scarlet, polished and forked—has been studied in experimental populations with different genetic backgrounds. All the populations (except those with forked) were started with equal frequencies (0.5) of the mutant and the corresponding wild-type alleles. The foundation stocks of one type of population (monochromosomal) contained only one or a few nonmutant chromosomes. The foundation stocks of the second type, polychromosomal populations, contained many wild-type chromosomes.

Natural selection led to decreases of the relative frequencies of the mutant genes in all cases. The initial rate of selection was, however, higher in polychromosomal than in monochromosomal populations in the cases of brown$^{75}$, ebony, Stubble, and scarlet. In the case of polished, no difference in the rate of selection between mono- and polychromosomal populations has been observed. In the case of forked, the difference in the rate of selection between two types of populations was noticeable only after the tenth generation. Estimation of the adaptive values of different genotypes indicates a superiority of mutant heterozygotes only in brown$^{75}$ and scarlet monochromosomal populations. A temporary superiority of

the heterozygotes has also been observed in Stubble monochromosomal populations. The relative adaptive values of the different genotypes have changed during the course of the experiments in all cases except those of polished, and brown[75] monochromosomal populations.

The results on the whole support the assumption that the adaptive values of the carriers of certain mutant factors depend not only on the effects of these mutants themselves but also on polygenic complexes associated with them.

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