THE EFFECT OF AN EXPERIMENTAL BOTTLENECK UPON QUANTITATIVE GENETIC VARIATION IN THE HOUSEFLY

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ABSTRACT

Effects of a population bottleneck (founder-flush cycle) upon quantitative genetic variation of morphometric traits were examined in replicated experimental lines of the housefly founded with one, four or 16 pairs of flies. Heritability and additive genetic variances for eight morphometric traits generally increased as a result of the bottleneck, but the pattern of increase among bottleneck sizes differed among traits. Principal axes of the additive genetic correlation matrix for the control line yielded two suites of traits, one associated with general body size and another set largely independent of body size. In the former set containing five of the traits, additive genetic variance was greatest in the bottleneck size of four pairs, whereas in the latter set of two traits the largest additive genetic variance occurred in the smallest bottleneck size of one pair. One trait exhibited changes in additive genetic variance intermediate between these two major responses. These results were inconsistent with models of additive effects of alleles within loci or of additive effects among loci. An observed decline in viability measures and body size in the bottleneck lines also indicated that there was nonadditivity of allelic effects for these traits. Several possible nonadditive models were explored that increased additive genetic variance as a result of a bottleneck. These included a model with complete dominance, a model with overdominance and a model incorporating multiplicative epistasis.

In the Modern Synthesis, species are regarded as integrated units of multilocus balance that function to guard their genetic and developmental integrity (DOBZHANSKY 1951; MAYR 1963). Under such a framework one is faced with the problem of how new species are formed, since this would involve the breakdown and reassembly of such units. Major theories of speciation have invoked small population size to varying extents to effect these changes. Genetic drift was integral to WRIGHT's shifting-balance theory of speciation (WRIGHT 1931, 1932, 1940). Bottlenecks were invoked more explicitly in the founder-flush theories of MAYR (1954, 1970, 1982) and CARSON (1968, 1975, 1982) and in the genetic transilience speciation model of TEMPLETON (1980a,b).

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The exact way that bottlenecks are supposed to effect genetic differentiation is unclear. Theories of speciation via bottlenecks, founder events, and/or drift rely on multilocus epistasis. Unfortunately, the dynamics of most multilocus systems with epistasis are intractable mathematically (see Hedrick, Jain and Holden (1978) for a recent review), so whether or not such selection can alter the balance in favor of speciation remains unknown. For example, Charlesworth and Smith (1982), using a simple two-locus model with epistasis, showed that bottlenecks can actually lower the chance of a peak shift, this being the exact opposite of the effect predicted by the founder-flush theory [see also the critical review of the founder-flush speciation theory by Barton and Charlesworth (1984)].

On the other hand, it seems clearly established that bottlenecks will deprecate genetic variation within populations. This will occur in the same way for variation based on major genes or for continuous variation based on polygenes (Lande 1980; Lewontin 1965; Nei, Maruyama and Chakraborty 1975; Maruyama and Fuerst 1984, 1985; Watterson 1984). But these results apply to single (or independent) loci with additive genetic effects. The theory does not accommodate either interallelic interactions (dominance, overdominance) or interlocus interactions (linkage, epistasis). Robertson (1952) demonstrated over 30 years ago that genetic variation due to recessive alleles, for example, may increase at least transiently as a result of inbreeding. In addition, bottlenecks would create and amplify linkage, making it unlikely that changes in variation at one locus, whether neutral or not, would be independent of changes at other loci (e.g., Hill 1977; Hill and Robertson 1968). Selection acting on only one locus within a correlated block of loci would alter the course of allele frequency change at other linked loci (e.g., Hill 1977; Maynard Smith and Haigh 1974; Kimura and Ohta 1971). Dobzhansky and Pavolovsky (1975) and Powell and Richmond (1974), for example, demonstrated that linkage effects altered the course of gene frequency change in small experimental populations of Drosophila pseudoobscura and D. palustorum, respectively. Thus, events during and immediately following a bottleneck or founder event (i.e., inbreeding or selection on the linked loci) could change predictions of how bottlenecks should affect variation.

It may be difficult to make general predictions as to how bottlenecks may affect genetic variation within and among populations for all but the simplest of circumstances. The more interesting circumstances of epistatic interactions and linkage would be more difficult to model. This being the case, it seems worthwhile to investigate the effects of bottlenecks through laboratory manipulations to indicate, at least, how such events potentially affect speciation (e.g., see Carson 1971). There have been few attempts to do so. Powell (1978) was able to generate significant premating isolation among experimental lines of D. pseudoobscura after eight founder-flush cycles. Ringo et al. (1985) were able to detect mating isolation after six founder flush cycles in D. simulans, but this occurred principally between the bottleneck lines and the base (control) line, not among the bottleneck lines. Templeton (1979a,b) demonstrated how bottlenecks could affect the genetic architecture of a species. By selecting for
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parthenogenetic lines of *D. mercatorum*, he was able to isolate rare genomes present in the original stocks that were viable in the homozygous state. FRANKHAM (1980) found that bottlenecks reduced the response to selection for abdominal bristle number in *D. melanogaster* in accordance with the predictions of ROBERTSON (1960) and JAMES (1971). But LINTS and BOURGOIS (1982) reported elevated genetic variation for sternopleural bristle number in a *D. melanogaster* line that had passed through an accidental bottleneck in the laboratory. This would theoretically allow an increased response in this line if selection were applied, the exact opposite of the results of FRANKHAM (1980). Clearly, more studies are needed to assess the potential genetic effects of bottlenecks upon populations, and this need seems to be particularly acute for polygenic variation, for which contradictory results have emerged.

The purpose of this study was to explore the effects of single bottleneck episodes of varying severity upon quantitative genetic variation of morphometric traits within experimental populations of the housefly, *Musca domestica* L. By varying the severity of the bottleneck (i.e., one, four or 16 pairs of flies) we hoped to quantify the effects of such bottlenecks upon genetic variation and to compare our results with theoretical predictions. Our analysis here focuses on changes in genetic variation within our experimental bottleneck lines. A companion paper focuses on differentiation among these same experimental bottleneck lines (BRANT, COMBS and McCOMMAS 1986).

MATERIALS AND METHODS

In August, 1980, a large sample of flies (>100 females) was taken from a landfill near Alvin, Texas, to establish an initially outbred laboratory population. In the *F*₂ generation, bottleneck lines were established in the following way. The offspring of isolated male-female pairs were reared in separate culture jars at optimal density (0.225 g CSMA larval medium per egg; BRYANT 1969). Bottleneck lines were initiated with offspring from exactly one, four or 16 pairs of flies; four such lines were established for each bottleneck size. The use of separate families ensured that the intended bottleneck size was achieved. All bottleneck lines were allowed to flush to normal population size (about 1000 pairs), which took about five generations. Thereafter, they were kept near the 1000-pair level by random culling of eggs during normal laboratory husbandry. The control line of outbred flies was maintained with this same husbandry procedure throughout the experiment. All larvae were reared at optimal densities *en masse* in 1-quart jars containing CSMA larval medium [see BRYANT (1969) for medium preparation]. Adults were fed daily with evaporated milk diluted 1:5 with water.

In generations *F*₆ and *F*₇, heritability tests were carried out on all lines as well as on the outbred control line. Virgin flies reared at standard densities were separated by sex and line within 12 hr of emergence. After 5–7 days, single male-female pairs were isolated, and their offspring were reared for parent-offspring tests (see below). The amount of CSMA larval medium provided to individual egg clutches was varied to keep the amount of medium at a constant (optimal) density of 0.225 g per egg. Upon emergence, ten offspring (five females and five males) were killed with cyanide and then pinned along with their parents for later measurement of morphological traits. Male offspring were excluded from the present analyses because of complications from two types of male-determining mechanisms initially present in the control population of flies, one being the standard *XY* system and the other a system in which the male-determining factor is present on an autosome (*A*₂ male-determining system of HIROY-
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Since females were all standard XX types, such chromosomal variation should not affect the heritability estimates for them.

Decreased viability, particularly in the single- and four-pair lines, made an initial goal of 50 families per bottleneck line unrealistic. In addition, only those parental flies with a complete record of all traits were used in the analyses (i.e., damaged wings on one of the parents would bar measurement). Hence, genetic parameters were estimated by pooling over replicate bottleneck lines (within a given bottleneck size), resulting in 110, 104, 156 and 48 families for lines of one, four and 16 pairs and the control line, respectively.

Eight morphological traits were measured for each fly [for a full description of these traits see table 1 of BRYANT (1977)]: wing length, wing width, head width (outer distance across eyes), inner eye separation (minimum distance between compound eyes), scutellum length along midline, scutellum width at base, length of thoracic suture and length of metafemur. For bilateral traits, the left side was analyzed here unless damage necessitated measurements from the right side. All traits were measured with an ocular micrometer and were converted to a common scale using natural logarithms before analysis.

Heritabilities were estimated by regressions of mean offspring values onto midparental values for each (log-transformed) trait (FALCONER 1981). This method yields the smallest statistical errors for a given sample size (KLEIN 1974; KLEIN, DE FRIES and FINKBEINER 1973). Since little additional precision is obtained by increasing family size, only three of the five females pinned per family were measured. Additive genetic variances were estimated as the product of these heritabilities and twice the phenotypic variance of midparental values. To the extent that phenotypic variances differ between males and females, this additive genetic variance may either overestimate or underestimate the actual values within a sex (FALCONER 1981), but it does provide an adequate basis for comparison among lines.

In addition to these heritability tests, viability components of fitness within lines were estimated by standard methods of rearing larvae to adulthood at near-optimal densities (e.g., BRYANT 1969; BRYANT and TURNER 1972). Eggs for these tests were collected from population cages, randomized to break up individual egg clutches and counted into batches of 80 eggs. These batches were placed into small cellcotton sacks within 60-ml bottles, each containing 18 g of CSMA larval medium; five such jars were set up per replicate bottleneck line (=20 per bottleneck size). After 48 hr, egg sacks were removed for determining egg hatching. Emerging adults were counted daily, and after all adults had emerged, the flies were dried at 100° for at least 24 hr and were weighed as a group per culture to determine mean dry weight per fly and total biomass produced per 80 initial eggs. These results allowed us to assay how the bottlenecks affected viability components of fitness.

RESULTS

Regression estimates of (narrow sense) heritabilities for the eight morphometric traits, using a weighted average over replicate lines within a bottleneck size, are given in Figure 1. Standard errors for these heritability estimates were also based on pooled regression estimates and were used to test for a significant difference in heritability for each trait between the control line and a given bottleneck size.

Two traits (scutellum length and metafemur length) did not show any significant differences in heritability between the bottleneck lines and the control. Four traits (wing width, head width, inner eye and thoracic suture) exhibited significantly greater heritabilities for the intermediate bottleneck sizes of four
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FIGURE 1.—Narrow-sense heritabilities for eight morphometric traits determined by offspring-midparent regressions for the bottleneck lines of one, four and 16 founding pairs and the control (C) line. Standard errors of heritabilities were computed from the regressions pooled over the four replicate lines within each bottleneck size. An underscore in each panel indicates that the average heritability for each bottleneck size was not significantly different from that for the control (if no underscore is present, the two heritabilities were significantly different at $P < 0.05$).

and 16 pairs. For the remaining two traits, one (scutellum width) showed greater heritabilities for all bottleneck lines compared with the control, and the other (wing length) had significantly lowered heritability than the control
only for the single-pair lines. Hence, no trait exhibited a consistent decline in heritability in relation to these bottleneck episodes. Heritabilities are ratios of additive to total phenotypic variance and are affected by changes in one or both of these variance components (for example, increased heritabilities may be entirely due to lowered phenotypic variance without any increase in additive genetic variance).

Additive genetic variance for each trait can be estimated directly from the covariance between offspring and parent (or midparent) values or from the product of heritability and total phenotypic variance (Falconer 1981). The additive genetic variances for all traits (pooled over replicate lines) are given in Figure 2. The expected loss in additive genetic variation is $1/2N$, where $N$ is the bottleneck size of two, eight and 32 flies (Lande 1980), resulting in additive genetic variance for these lines of 75, 94 and 98% of the control level, respectively. Clearly, our results are discordant with these expectations. In one bottleneck size, every trait exhibited increased additive genetic variance compared to the control line. Additive genetic variance in two traits (inner eye and scutellum width) was significantly greater than in the control line for all bottleneck sizes.

These traits are unlikely to be independent of each other but, rather, should exhibit patterns of intercorrelations indicating genetic and developmental affinities. To determine these, we computed an additive genetic correlation matrix among traits for the control line from cross-covariances of parents and their offspring for all pairwise combinations of traits (Falconer 1981). This matrix was then subjected to a principal axis rotation to determine the major interdependencies among the traits. The first four axes, summarizing 97% of the trait intercorrelations, are given in Table 1. The first axis, accounting for 55% of the standardized variation, represents genetic associations among five traits: wing length, wing width, head width, metafemur length and thoracic suture. This axis seems to best represent a “general body size” trait. The remaining three traits have lower genetic correlations with these five traits and with each other. Inner eye and scutellum width are associated with separate axes (II and III, respectively), whereas scutellum length is nearly equally associated with all four axes.

These trait associations can then be related to the changes in additive genetic variances for the bottleneck lines in Figure 2. Those five traits associated with the first principal axis in Table 1 all exhibit a pattern of increased additive genetic variances for intermediate bottleneck sizes (four and 16 pairs). On the other hand, inner eye and scutellum width exhibit greatest additive genetic variance for the single-pair bottleneck lines. Thus, there appear to be two major patterns of change in additive genetic variance for these traits; one pattern (greatest variances for intermediate bottleneck sizes) reflects concordant changes in five genetically intercorrelated traits identified by principal axis I, and another pattern (greatest variance in single-pair bottleneck lines) is associated with two independent genetic traits (inner eye and scutellum width). The remaining trait, scutellum length, is intermediate in response between
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Figure 2.—Additive genetic variances for the eight morphometric traits averaged over the four replicate lines within each bottleneck size. Each trait variance was tested against the control (C) by an F-ratio of the respective additive genetic variances. An underscore indicates that the two variances were not significantly different at $P > 0.05$. All variances have been multiplied by $10^4$ for convenience.

These two patterns (Figure 2) and is also evenly dispersed among the four principal axes of Table 1.

The results of the viability tests are summarized in Figure 3 as mean dry weight per fly (in milligrams), percent adult emergence per 80 initial eggs, and
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**TABLE 1**

Principal axes of the additive genetic correlation matrix for the control line

<table>
<thead>
<tr>
<th>Morphometric trait</th>
<th>Principal axis</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>I</td>
<td>II</td>
<td>III</td>
</tr>
<tr>
<td>Wing length</td>
<td>0.82</td>
<td>-0.28</td>
<td>0.23</td>
</tr>
<tr>
<td>Wing width</td>
<td>0.83</td>
<td>-0.21</td>
<td>0.29</td>
</tr>
<tr>
<td>Head width</td>
<td>0.89</td>
<td>0.46</td>
<td>-0.02</td>
</tr>
<tr>
<td>Inner eye</td>
<td>-0.04</td>
<td>-0.83</td>
<td>-0.43</td>
</tr>
<tr>
<td>Scutellum length</td>
<td>0.52</td>
<td>0.41</td>
<td>0.35</td>
</tr>
<tr>
<td>Scutellum width</td>
<td>0.47</td>
<td>0.13</td>
<td>-0.81</td>
</tr>
<tr>
<td>Metafemur length</td>
<td>0.89</td>
<td>0.09</td>
<td>-0.09</td>
</tr>
<tr>
<td>Thoracic suture</td>
<td>0.94</td>
<td>0.02</td>
<td>0.15</td>
</tr>
<tr>
<td>Percent explained variance</td>
<td>54.7</td>
<td>17.1</td>
<td>14.2</td>
</tr>
</tbody>
</table>

Entries are correlations of variables and factors; the largest correlation for each variable with a factor is underscored.

Total dry weight of biomass produced per culture. Adult size in houseflies (as well as most cyclorrhaphous diptera) is greatly affected by larval density (e.g., SULLIVAN and SOKAL 1963). Since the numbers of larvae per constant 18 g of medium differed among bottleneck lines due to differential hatching of eggs and survival of larvae, mean dry weight per fly was adjusted by regressing adult weight onto number of emerging adults per culture. Thus, the mean dry weights in Figure 3 have been adjusted to a constant density equivalent to that of the control so that the effects of inbreeding per se can be compared among bottleneck sizes.

Egg-to-adult viability decreased significantly among bottleneck sizes, as did (adjusted) mean dry weight per fly and total biomass per culture. These results suggest an increased homozygosity of recessive deleterious alleles, leading to inbreeding depression as a result of the bottleneck (CROW and KIMURA 1970; FALCONER 1981). Inbreeding depression is commonly observed in previously outcrossed species (FALCONER 1981; LATTER and ROBERTSON 1962; LERNER 1954; MAYNARD SMITH, CLARKE and HOLLINGSWORTH 1955). Fly size is positively correlated with the number of eggs in female flies (e.g., BLACK and KRASFUR 1986; ROBERTSON and SANG 1944; ROBERTSON 1957; WEBBER 1955) and with mating success in males (BALDWIN and BRYANT 1982). So size alone is likely to be correlated with overall fitness, leading to the expectation that the traits used here would reflect inbreeding depression by changes in their mean values.

**THEORETICAL CONSIDERATIONS**

If genetic variation is based on additivity of allelic effects, it would decrease as a function of $1/2N$ after a bottleneck of size $N$ individuals (LANDE 1980). Clearly, the variation in our lines did not follow this expectations, so we must seek possible explanations for our results in less simplistic models. Dominance (or overdominance) among alleles is necessary for inbreeding depression to occur, and epistasis can further impose nonlinearity to the inbreeding response.
The inbreeding depression we observed in Figure 3, therefore, indicates there was at least dominance (or overdominance) among alleles affecting body size and, hence, among alleles affecting individual traits correlated with body size. In addition, epistasis among loci controlling components of fitness is to be expected (e.g., Dobzhansky 1946, 1955; Spieß 1959; Charlesworth and Charlesworth 1973; Rose and Charlesworth 1980), so epistasis among loci contributing to body size (fitness) could also be affecting our results. Possible explanations for our anomalous results could therefore reside in nonadditive effects within and among loci. Little is known about how inbreeding and/or bottlenecks per se would affect genetic variation based on such nonadditive effects, although Robertson (1952) showed that inbreeding can increase genetic variation based on recessive alleles. We investigate three models of increasing levels of non-
additivity in relation to a bottleneck: dominance, overdominance and epistasis. As shown below, all three types of nonadditivity can lead to increased additive genetic variance as a result of a bottleneck.

**Complete dominance model:** With two alleles at one locus, the additive genetic variance \( V_A \) is given by Falconer (1981) as

\[
V_A = 2pq[a + d(q - p)],
\]

where \( q \) is the frequency of the rare recessive allele, \( a \) is an arbitrarily assigned genotypic deviation from the midpoint between the trait values of the two homozygotes, and \( d \) is the (residual) deviation from this linearity (additivity) due to dominance (Falconer 1981, pp. 109–110). When \( d = 0 \) there is complete additivity, and when \( d = a \) there is complete dominance of allelic effects.

The changes in additive genetic variance as a result of a bottleneck can be derived using the binomial probabilities of obtaining various allele frequencies of the recessive allele in the bottleneck lines given an initial frequency in the parental population. The expected additive genetic variance after a bottleneck is then the average of additive genetic variances for all possible frequencies of the recessive allele weighted by these probabilities of occurrence. Taking \( d = a \) for simplicity (complete dominance), the expected additive genetic variance, \( V'_A \), after a bottleneck of size \( 2N \) genes (\( N \) individuals) would be

\[
V'_A = \sum_{i=0}^{2N} \left( \binom{2N}{i} \right) \hat{p}^{2N-i} \hat{q}^i 8 \left( 1 - \frac{i}{2N} \right) \left( \frac{i}{2N} \right)^3 a^2,
\]

where \( \hat{q} (=1 - \hat{p}) \) is the frequency of the recessive allele in the base population, and \( i/2N \) is its frequency in a particular bottleneck line.

The ratio of additive genetic variance in a bottleneck line to the initial additive genetic variance in the base population for various initial values of the recessive allele and for bottleneck sizes of two (single pair) to 16 (eight pairs) is given in Figure 4. For completely additive effects (\( d = 0 \)) we note that this ratio would be equal to \( 1 - 1/2N \) for all initial allele frequencies, resulting in a general decrease in additive genetic variance. In contrast, the additive genetic variance of a trait influenced by a recessive allele will increase as a result of a bottleneck, and more so the rarer the allele in the base population. For example, when the initial allele frequency is 0.05, the average additive genetic variance after a bottleneck of size two (one pair) would be 23 times larger than that in the base population. Since alleles associated with decreased fitness (e.g., deleterious recessive alleles) should be low in frequency in the base population, the average effect of a bottleneck would be to increase rather than decrease the additive genetic variance.

One possible explanation for our observed increase in additive genetic variance after a bottleneck could reside with variation based on recessive alleles in low frequency in the base population. The decrease in fitness traits in Figure 3 indicates this may be so, because only dominance (or overdominance) among alleles results in inbreeding depression. This explanation may be adequate for the inner eye and scutellum width, which showed the greatest increases in additive genetic variance (over the control) for the smallest bottleneck sizes.
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The remaining traits, which define principal axis I in Table 1, exhibited greatest increases in additive genetic variance for an intermediate bottleneck size which contradicts this model. The model does not incorporate fitness per se although fitness is implied by an initial low frequency of recessive alleles. To see if explicit consideration of selection in the model could alter the balance in favor of higher variance for intermediate bottleneck sizes, we assumed a linear decline of fitness with frequency of the recessive allele so \( s = q \), where \( s \) is the selection coefficient against the recessive allele of frequency \( q \). This model lowered the average gain in additive genetic variance as a result of a bottleneck, but did not alter the ordering of such increases in additive genetic variance among bottleneck lines. A model based on sampling of rare recessive alleles (with or without selection) seems to explain our results inadequately, at least for those traits correlated with general body size (e.g., principal axis I of Table 1).

**Overdominance model:** Assume two alleles at a single locus such that the relative fitnesses of the two homozygotes are \( 1 - s_1 \) and \( 1 - s_2 \) compared to 1.0 for the heterozygote. The additive genetic variance for fitness can be
FIGURE 5.—Additive genetic variance in a bottleneck line over a range of initial equilibrium frequencies in the base population for the model of overdominance. For these results we scaled the selection coefficients to $s_1 + s_2 = 1.0$ (results based on equation 3). Results for bottleneck sizes of two, four, eight and 16 flies are shown. See text for further discussion.

obtained as the covariance of fitness with breeding values of genotypes (Falconer 1981, p. 107), where breeding value is in terms of the average effect of a gene substitution on a gene substitution on fitness:

$$V_A = 2 \hat{p} q (s_2 q - s_1 \hat{p})^2.$$  \hspace{1cm} (3)

The equilibrium value for $q$ is

$$\hat{q} = s_1/(s_1 + s_2).$$

Scaling $s_1 + s_2 = 1.0$ for convenience, the expected additive genetic variance as a result of a bottleneck would be the average of these additive genetic variances weighted by their (binomial) probabilities of occurrence as before.

$$V_A' = \sum_{i=0}^{2N} \binom{2N}{i} \hat{p}^{2N-i} \hat{q}^i \left[\left(1 - \frac{i}{2N}\right) \left(\frac{i}{2N}\right) \left(\frac{i}{2N} - s_2\right)\right]^2.$$  \hspace{1cm} (4)

We assume the base population to be in equilibrium, so initial additive genetic variance is zero. In Figure 5 we plot the average additive genetic variance for the overdominance model for various initial frequencies in the base population. As in the dominance model, there is an average increase in additive genetic variance as a result of a bottleneck. In contrast to the previous model the greatest gains in additive genetic variance occur for larger bottleneck sizes. This model does not seem to fit the pattern of increase in additive genetic variance for any of the traits (Figure 2).
**Epistasis model**: To investigate possible effects of nonadditivity among loci, we adopt a simple epistasis model of multiplicative decline in trait value (fitness) with increased homozygosity of those loci affecting the trait (FRANKLIN 1977). Suppose that making one such locus homozygous reduces fitness (and the trait value) by 50%; making two such loci homozygous reduces fitness by 75%, and so on. We cannot apply this model unless we know the number of loci affecting a trait. The number of loci involved is probably quite high, but a bottleneck will generate interlocus correlations that would persist for some time (AVERY and HILL 1979), particularly as there is no crossing over in male houseflies. Therefore, the units of selection after a bottleneck would be large blocks of genes rather than individual loci. The number of these linkage groups cannot be less than the number of haploid chromosomes, so we take this as a minimal estimate of such groups. Letting $F$ be the average homozygosity per individual, the exponential decline in fitness (trait value), $X$, in this model is then

$$X = e^{-4.158F},$$

so that the variance, $V'(X)$, for a trait within a bottleneck line would be

$$V'(X) = \sum_{i=0}^{6} \binom{6}{i} F^i(1 - F)^6-(\frac{1}{2})^{2i} - X^2. \quad (5)$$

The mean declines with homozygosity, but the variance increases for intermediate values of homozygosity before declining at higher values (Figure 6). Thus, in contrast to previous models the multiplicative epistasis model yields greatest additive genetic variance for intermediate levels of heterozygosity.

To see how this corresponds to our results we need estimates of average heterozygosity per individual for the bottleneck lines which cannot be taken directly from the results. From a companion study of electrophoretic variation on these same bottleneck lines (McCOMMAS and BRYANT, unpublished results), the average rates of fixation for these alleles were approximately 7, 13 and 38% for bottleneck sizes of 16, four and one pair, respectively. Changes in electrophoretic variation within and among populations does not necessarily correspond to changes in genetic variation of morphometric traits (e.g., GILES 1984; McCOMMAS and BRYANT, unpublished results). We recorded the number of single pairs in the heritability tests that did not produce offspring: the percentage of inviable pairs were 4, 11, 16 and 33% for the control, 16-pair, four-pair and single-pair lines, respectively. Assuming that inviability was related to homozygosity of lethal or moderately deleterious alleles present in the base population, their rates of fixation resulting in inviability largely correspond to the fixation rates observed for electrophoretic variants. The fixation rates for electrophoretic variants in this case seem to give reasonable estimates of fixation (homozygosity) in the bottleneck lines. When these homozygosity values are superimposed on the variance curve in Figure 6, the resultant pattern of genetic variance among bottleneck sizes is much like that observed for the five traits correlated with general body size (principal axis I in Table 1). The model of epistasis with multiplicative effects on fitness has the potential...
for explaining our results for these traits, whereas the previous models did not.

DISCUSSION

The accepted dogma concerning bottlenecks is that they will decrease genetic variability and more so the smaller the bottleneck size (LEWONTIN 1965; LANDE 1980; MARUYAMA and FUERST 1985). This is based on additivity of allelic effects within loci and on polygenic variation of additive effects among loci. But our results indicate that nonadditivity at either of these levels will increase additive genetic variation after a bottleneck. Depending on the basis of the variation, this increase can be greater for smaller bottleneck sizes. CARSON and TEMPLETON (1984) anticipated some of these results when they suggested that the antagonistic pleiotropy model of ROSE (1982, 1983) could provide for increased variance as a result of a bottleneck. ROSE's model relies on alleles at a single locus having opposite pleiotropic effects on two components of fitness (e.g., larval survival vs. adult fecundity) and results in overdominance in total fitness. Here, we have provided a simpler model of overdominance that confirms the prediction of CARSON and TEMPLETON [note that when the dominance parameters $h_1$ and $h_2$ are set to zero in ROSE's (1982) model, his formula 9 for additive genetic variance in fitness is identical to our formula 3].

Based on a correspondence of our results to those of the models investigated,
we felt the dominance model could explain the results for the two traits independent of body size that showed the greatest increases in variance for the smallest bottleneck size (inner eye and scutellum width). For the five traits associated with body size (principal axis I in Table 1) we felt the model incorporating multiplicative interaction of correlated blocks of genes best explained our data. Such a correspondence of theoretical results to data may say little about the actual processes involved. The observed decline in viability after a bottleneck in Figure 3 does indicate that deleterious recessive alleles were present in the base population. A decline in fitness with chromosomal homozygosity has been reported for *D. pseudoobscura* as well as *D. melanogaster* (Spassky, Dobzhansky and Anderson 1965; Temin et al. 1969; Kosuda 1971), but we are unaware of such data for the housefly. It may be difficult to discriminate among the models with our data because either dominance or overdominance (with or without epistasis) results in a decline in fitness (Crow and Kimura 1970). The way the total genetic variance is partitioned into additive and nonadditive components (dominance) does differ for dominance and overdominance models. In the model of overdominance, total genetic variance remains fairly constant over a wide range of allele frequencies about the equilibrium (Falconer 1981, pp. 117–118; note that Falconer’s model of pure overdominance is equivalent to our model when $s_1 = s_2 = 1.0$). But at equilibrium, additive genetic variance is zero, so the total genetic variance is composed solely of dominance variance. In this model a bottleneck would redistribute variance into the additive component, whereas total genetic variance would remain nearly constant. In contrast, in the dominance and epistatic models, total genetic variance would respond to increases in additive genetic variance. In our data the increases in additive genetic variance were accompanied by increases in total phenotypic variance (residual variances remained more or less constant in relation to the control). Hence, it is unlikely that our results were based on an initial equilibrium of overdominant effects in the base population, but were more likely based on dominance alone.

Whether or not our results are peculiar to the housefly remains problematic. Our models of trait variation presume a correlation between components of fitness and the traits via a general body-size factor. A general body-size factor is commonly found in studies of morphometric variation (e.g., Blackith and Reyment 1971), and such size is often correlated with components of fitness, particularly fecundity in insects [e.g., see Black and Krasfur (1986) for the housefly and Robertson and Sang (1944) for Drosophila]. Robertson and Reeve (1955) demonstrated an inbreeding depression for body size in *D. melanogaster*, and Tantawy (1957) and Tantawy and Reeve (1956) further related this to wing length and thorax size in this species. Hence, our results could well extend to other organisms. It is notable that Lints and Bourgeois (1982) reported an increase in heritability for sternopleural bristle number in a laboratory line of *D. melanogaster* that had undergone a population crash. Other evidence comes from studies on the Hawaiian Drosophila. Although this group emanated from a few founders representing a single lineage (Throckmorton 1966), and successive island colonization likely occurred via single
founder events [see CARSON and TEMPLETON (1984) for a recent discussion], there is little evidence that many of these founder-derived species have less genetic variation than their ancestral taxa (CRADDOCK and JOHNSON 1979; SENE and CARSON 1977).

The main interest in bottlenecks by evolutionary biologists is in their putative enhancement of speciation. If bottlenecks serve to increase additive genetic variance, if only transiently, the rate of evolution in a new environment may be accelerated compared to colonization without a population bottleneck. But this depends on the source of such increased variation. In our models increased variance is due to the action of deleterious alleles (or groups of such alleles) which would lead to decreased fitness. For an average level of homozygosity in a population, not all individuals would be homozygous to the same degree (FRANKLIN 1977; WEIR, AVERY and HILL 1980; AVERY and HILL 1977, 1979; CHAKRABORTY 1981). Selection against the more homozygous (less fit) individuals would retard evolutionary divergence of a founding population from the parental population (e.g., HAYMAN and MATHER 1953; CONNOR and BELLUCCI 1979). This would also be true for a disturbance from an overdominant equilibrium: a bottleneck would move the population away from the equilibrium, and subsequent selection would tend to move it back. In WRIGHT's terminology (WRIGHT 1931, 1932) a bottleneck would push a population off its local adaptive peak, and selection would attempt to restore it to the same peak. But if a founder population were in a sufficiently novel environment in which these alleles were no longer deleterious, a peak shift would be likely. This latter effect may be similar to that envisioned by CARSON (1968, 1971) in his founder-flush theory of speciation. In the flush phase following a founder event, many genotypes previously selected against are able to flourish in the novel environment. When selection recurs, the genotypes previously successful may no longer enjoy this benefit in the new environment, so selection would move the derived population to a new genetic equilibrium. As a consequence, a Wrightian peak shift may occur and lead to speciation.

A major question in evolutionary biology is whether genetic differences among populations and species are simply extensions of variation present in the ancestral (base) population or whether higher order macroevolutionary processes prevail (e.g., see GOULD 1980; CHARLESWORTH, LANDE and SLATKIN 1982). There are few data to resolve this issue, because a proper genetic analysis necessitates crossing of the species under study. COYNE (1983), in carrying out one such analysis, found that morphological differences among three sibling species of Drosophila could be easily extrapolated from genetic variation present among individuals within these species. In this context it is interesting to look at the type of morphological variation that taxonomically separates species of Musca, particularly within the domestica complex. One recurring trait separating members of this complex is the ratio of outer-head width to inner-eye separation (i.e., the “frons” ratio; see SACCÁ, 1964). There was little additive genetic variance for inner-eye in our base population. Yet, after a bottleneck this trait exhibited one of the largest increases in additive genetic variance of all traits. Hence, we observed not only an increase in
additive genetic variance as a result of a bottleneck but also that this increase in variation was greatest for the trait most frequently used in differentiating taxa within the *Musca domestica* complex. Taxonomic differentiation in this group could be based on variation potentially available to the speciation process after a bottleneck, but not evident within outbred populations. Bottlenecks could then well provide the catalyst critical for speciation in this group. Bottleneck effects may be more pervasive that we have previously thought, and speciation in other groups may occur much in the same way envisioned for the Hawaiian Drosophila.

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BOTTLENECK EFFECT ON VARIATION


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