Ecological Aspects of the Heritability of Body Size in Drosophila buzzatii

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ABSTRACT

The heritability of thorax length in the cactophilic Drosophila buzzatii was determined for flies from each of 10 rotting cactus cladodes. For each rot, emerging flies were used as parents of progeny reared in the laboratory. The methods used were full sib analysis with the parents mated assortatively and also offspring-parent regression. From this, heritabilities were measured for the laboratory environment and for the natural environment of the rotting cladode. For the laboratory environment, \( h^2 = 0.3770 \pm 0.0203 \) and for the natural environment \( h^2 = 0.0936 \pm 0.0087 \) within rots and \( h^2 = 0.0595 \pm 0.0123 \) for a population drawn randomly from different rots. Because of the possibility of genotype-environment interaction between the laboratory and rot environments, the methods of B. Riska, T. Prout and M. Turelli were used to show it is possible that there is no such interaction, but if there is, the above natural heritabilities are approximate lower bounds. These results are related to the general problem of determining heritabilities in nature where it is impractical to measure both parents and progeny in nature. Determining heritability not only in nature but in relation to subdivision into ephemeral patches (cladodes in this case) has an important bearing on natural selection response and to general theories of stabilizing selection proposed to explain the existence of genetic variation. Attempts were made to detect selection by using the size of emerging adults as an indicator of various levels of larval stress. No selection was detected, but the power to do so was very weak. Differences between progeny means from different rots indicated some genetic differences between rots which can be adequately explained by small numbers of founders. This suggests a random fine scale subdivision amounting to \( F_{ST} = 0.1483 \pm 0.0462 \).

This paper reports the results of an investigation of the heritability of body size in Drosophila buzzatii when subject to the environmental variation experienced in nature. The extensive data now accumulating on genetic variation for quantitative characters in natural populations of animals and plants report heritability estimated mostly in the uniform environmental conditions of the laboratory, greenhouse or garden, although there are notable exceptions which report natural heritability such as the work on song sparrows (Smith and Dhondt 1980), Darwin finches (Boag and Grant 1978; Gibbs 1988), great tits (Van Noordwijk, Van Belan and Scharloo 1980), jewelweed (Mitchell-Olds 1986) and sage (Shaw 1986).

Heritability under natural conditions as opposed to an experimental environment has an important bearing on the maintenance of polygenic variation in natural populations (Barker and Thomas 1987). Unlike the study of variation of the primary protein products of genes, or allozymes, where selection vs. neutrality is still debated, in the case of polygenic variation the ultimate phenotypes are known and frequently involve life history traits, or characters correlated with such traits, such as body size, which are phenotypes almost certainly subject to natural selection. However, if, for example, in plants because of their plasticity, environmental effects on some character in nature result in a heritability of essentially zero, as shown for plant size by Gottlieb (1977) in Stephanomeria exigua, then the genetic variation must be selectively neutral. Such an extreme might be uncommon, but low natural heritabilities would diminish the response to selection, and in the case of stabilizing selection-mutation balance, Turelli (1988) and Bulmer (1989) have shown that there is an inverse relation between the heritability and the amount of genetic variance maintained. This theory essentially proposes that the polygenic variation constitutes a mutational "load," although the load effects, if important, are diminished by the low heritability. On the other hand, when natural heritabilities are high, then one might be inclined to seek some form of balancing selection to explain the variation. Natural heritability, then, has an important bearing on the application of old theories to new data.

Coyne and Beecham (1987) studied the natural heritability of body size in Drosophila melanogaster. They found substantial natural heritability (contrary to an early report of one of us (Prout 1958a) sug-

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gesting a natural heritability of body size of zero in a

citrus grove). They also detected and measured a

substantial heritability of differences between popu-

lations. Here we take advantage of the well defined

ecology of the cactophilic *D. buzzatii* (Barker 1982)

to analyze the heritability of body size within and

between subdivisions of a single natural population.

**MATERIALS AND METHODS**

The flies used were derived from a natural population

breeding in *Opuntia stricta* at Trinkey State Forest (31°22'S,

149°27'E) near the town of Tambar Springs, N.S.W., Aus-

tralia. Individual rotting cactus cladodes, hereafter referred to

as "rots," were collected in the field, and flies raised from

these in the laboratory. Two such collections were made,

and, at each time, a sample of wild adults also was collected

by netting over fermenting banana baits.

At the first collection (August 12, 1987), 33 rots were

taken. Each was placed immediately on collection into a

plastic bag, and later that evening, set in a plastic container

on moist, sterile sand, and kept at 25°C. Emerging adults

were collected daily, sexed and stored in vials on live yeast-

ed (Saccharomyces cerevisiae) cactus-sucrose-yeast medium

(Stammer and Barker 1986) at 12°. By 3 weeks after

collection, the emergence distribution from each rot was

well-defined, and it was decided that flies emerging during

the period August 27–31, which would have been eggs or

very young larvae at the time of rot collection, would be

used as parents in the experiment. Sufficient flies thus were

available from 12 rots. Females from these collection days

were transferred to individual vials, and put at 25°C to check

virginity.

During the period of adult emergence from the rots, the

wild caught flies also were held (sexes separate) at 12°. The

females then were despermaphorized by placing them at −12° for

15 min (R. Frankham, personal communication). After

removal from the freezer, females were transferred immedi-

ately to an empty vial at 20°C to recover, and then to a

medium vial to check sterility over the next few days.

The second field collection was made on September 20,

1987, and procedures similar to those above were followed

except that for three rots, sets of early and of late emerging

flies were taken, on the basis that the early emerging flies

had completed more of their development in the field and

might have been subjected to less environmental stress due

to lower larval density and possibly better nutrition. The

late emerging flies were equivalent to those from the first

collection (i.e., emerging on average about 17 days after

collection), while the early emerging flies emerged on

average about 9 days after collection.

The trait studied was thorax length, measured from the

anterior margin of the thorax to the posterior tip of the

scutellum, with the fly dorsal side up. For measurement, the

flies were held on a stage as described by Robertson and

and four from the second, including the early and late

emergence pairs, were used for the heritability estimation

as follows: the potential parents were measured for thorax

length and then paired assortatively. From each pair, ten

newly hatched larvae (0–6 hr old) were put individually into

4.5 cm × 1.5 cm vials with 2 ml cactus-sucrose-yeast medium.

All eclosing progeny were measured for thorax length.

Also, for nine rots from the first collection, samples of

emerging females were measured, and also their offspring

were measured, grown individually in vials as just described.

The number of mates and size of the mates for these

parental females were unknown, so these data were not used

for heritability estimation, but they were used to provide

supplementary statistics.

The original measurements were in units of mm × 100.

For analysis a log transformation was used, \( Y = \log X \). Means

are reported in \( Y \) units and variances in \( Y^2 \times 10^3 \). The log

transformation removes the dependence of variance on the

mean (Robertson 1987). Also following Robertson, in

order to facilitate the sib analysis, individual male progeny

in each rot were transformed so that the mean of the males

equalled the mean of the females for each rot.

**RESULTS**

**Heritability:** Implicit in the following discussion of

heritabilities and genetic variance of body size is the

assumption that *D. buzzatii* is like *D. melanogaster* in

that maternal effects and dominance variance are of little

importance (Robertson and Reeve 1955; Robertson 1987).

Also, it will be assumed for the initial treatment of

the data that there are no genotype-environment in-

teractions between the laboratory environment of the

offspring and the natural environment of the parents.

After this, consideration will be given as to how the

interpretation of these initial results might be re-

stricted if there were such interactions.

Table 1 gives some basic statistics and heritability

estimates for each of the ten populations. The early

and late emergences from the same rot are identified by

the sequence "E" and then "L." No consistent differences nor statistically significant differences

were found in comparing early versus late emerging

flies nor their offspring with respect to means and

variances, nor for derived statistics such as heritability,

so that these three early and late pairs can be treated

as six independent rots (the "E" and "L" are omitted

from later tables). Biologically this is reasonable be-

cause as the rotting cladode ages this constitutes a new

environment and also the emerging flies probably had

different but overlapping sets of parents. Similarly,

there were no differences between rots from the first

collection ( #1–#3 ) and the second collection ( #4–

#10 ).

The first two columns of the table give the number of

full sib families and total number of offspring. The

next four columns provide the statistics necessary for

estimating the heritability in the two environments

(see below), namely, the variance of the parents \( V_p \),

the correlation between parents due to assortative

mating, \( r \), the component of variance between full sib

families \( V_s \) and \( t_e \) the intraclass correlation of full sibs.

From these the following estimates are shown: \( h^2_p \)

estimates heritability in the parental environment and

\( h^2_s \) estimates the heritability in the laboratory environ-
Heritability in the offspring environment is derived in Table A. The standard errors of these heritability estimates are approximations explained in APPENDIX B. The statistical significance levels associated with $2t_e$ indicate whether or not $V_e$ is greater than zero. All cases were significant at the 1% level or less.

The standard estimate of heritability from full sib families with assortative mating gives the heritability in the parental environment. The assumption at this point is that the genetic variance observed in the progeny environment would be unchanged in the environment in which the parents were raised. This heritability, denoted by $h_e^2$, is obtained as follows (Falconer 1981, p. 164).

$$h_e^2 = \frac{-1 + \sqrt{1 + 8rt_p}}{2r},\tag{1}$$

where,

$$t_p = V_{s}/V_p,$$

$t_p$ is a little larger than the intraclass correlation that would be observed in the parental environment. This is because $V_e$ is divided by $V_p$ which is not inflated due to the assortative mating. The heritability in the offspring environment is derived in APPENDIX A. It is as follows:

$$h_o^2 = \frac{2t_e}{1 + rh_e^2(1 - t_e)}\tag{2}$$

$t_e$ is the observed intraclass correlation and $2t_e$ would be the heritability were there no assortative mating. For each of the two heritability estimates, the empirical variance of the estimates across rots was similar to the mean of the approximate error variance, $(SE)^2$. For this reason they were assumed to be homogeneous and were pooled by weighting each value by the

"information," $I = (SE)^2$. These pooled values are designated $h_e^2$ and $h_o^2$ and their standard errors are $\sqrt{1/(SE)^2}$.

Table 2 gives the results of regression of offspring on parents. This provides another estimate of heritability in the parental environment, $h_p^2$. The individual regressions were heterogeneous across rots so arithmetic means, $\bar{b}$, are given at the bottom with their empirical standard errors. Because of the high assortative mating, $r = 0.9692$, the regressions on single parents are essentially the same as the regression on the midparent which is $\bar{b} = 0.0789$ ($SE = 0.0347$) and this agrees well with the results of sib analysis where $h_p^2 = 0.0699$ ($SE = 0.0079$). The difference between them gives $t = 0.253$ (d.f. = 18, $P = 0.80$).

The two collections of flies coming to banana bait were expected to yield lower heritabilities since the individuals most likely emerged from different rots. Combining the two gives $h_p^2 = 0.0927$, $SE = 0.0407$ and the regression $\bar{b} = -0.0579$, $SE = 0.0398$. The former is larger and the latter smaller than the values from Table 1 for $h_o^2$ and Table 2 for $\bar{b}$, respectively. However, the errors are so large and resulting confidence interval so wide that the expected reduction in heritability was not detectable. In fact, it will be seen presently that the expected reduction is not very great, so that much more data would be required to test this prediction.

**Means, variances and variance components:** Table 3 provides additional statistics for the ten experimental rots. The means are for females. It can be seen that the means of the parents, $M_p$, are lower and more variable than the means of the offspring, $M_o$. A statistical analysis will be given presently. The additive genetic variance, $V_{a}$, is obtained by the relation, $V_{a} = 2V_s/(1 + rh_e^2)$, derived in APPENDIX A, Equation 4. $V_e$

### Table 1

<table>
<thead>
<tr>
<th>Population</th>
<th>$N_p$</th>
<th>$N_o$</th>
<th>$V_p$</th>
<th>$r$</th>
<th>$V_e$</th>
<th>$2t_e$</th>
<th>se</th>
<th>$h_o^2$</th>
<th>se</th>
<th>$h_e^2$</th>
<th>se</th>
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</thead>
<tbody>
<tr>
<td>1</td>
<td>24</td>
<td>177</td>
<td>3.448</td>
<td>0.972</td>
<td>0.2624</td>
<td>0.361***</td>
<td>0.151</td>
<td>0.1346</td>
<td>0.0649</td>
<td>0.3259</td>
<td>0.1218</td>
</tr>
<tr>
<td>2</td>
<td>12</td>
<td>98</td>
<td>2.259</td>
<td>0.970</td>
<td>0.3119</td>
<td>0.645***</td>
<td>0.248</td>
<td>0.2264</td>
<td>0.1188</td>
<td>0.5615</td>
<td>0.1725</td>
</tr>
<tr>
<td>3</td>
<td>18</td>
<td>112</td>
<td>14.452</td>
<td>0.983</td>
<td>0.2488</td>
<td>0.349***</td>
<td>0.190</td>
<td>0.3354</td>
<td>0.0221</td>
<td>0.3997</td>
<td>0.1738</td>
</tr>
<tr>
<td>4$E$</td>
<td>26</td>
<td>192</td>
<td>2.512</td>
<td>0.978</td>
<td>0.1507</td>
<td>0.311***</td>
<td>0.139</td>
<td>0.1085</td>
<td>0.0546</td>
<td>0.2859</td>
<td>0.1139</td>
</tr>
<tr>
<td>5$L$</td>
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<td>250</td>
<td>2.515</td>
<td>0.969</td>
<td>0.1758</td>
<td>0.397***</td>
<td>0.152</td>
<td>0.1248</td>
<td>0.0562</td>
<td>0.5615</td>
<td>0.1086</td>
</tr>
<tr>
<td>6$E$</td>
<td>31</td>
<td>244</td>
<td>2.646</td>
<td>0.959</td>
<td>0.2290</td>
<td>0.587***</td>
<td>0.149</td>
<td>0.1512</td>
<td>0.0525</td>
<td>0.5323</td>
<td>0.1167</td>
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<tr>
<td>7$L$</td>
<td>33</td>
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<td>3.454</td>
<td>0.977</td>
<td>0.1848</td>
<td>0.483***</td>
<td>0.133</td>
<td>0.0985</td>
<td>0.0351</td>
<td>0.4505</td>
<td>0.1115</td>
</tr>
<tr>
<td>8$E$</td>
<td>27</td>
<td>178</td>
<td>0.877</td>
<td>0.944</td>
<td>0.2275</td>
<td>0.516***</td>
<td>0.165</td>
<td>0.3815</td>
<td>0.1379</td>
<td>0.4075</td>
<td>0.1058</td>
</tr>
<tr>
<td>9$L$</td>
<td>36</td>
<td>314</td>
<td>9.326</td>
<td>0.993</td>
<td>0.2314</td>
<td>0.512***</td>
<td>0.129</td>
<td>0.0474</td>
<td>0.0167</td>
<td>0.4947</td>
<td>0.1154</td>
</tr>
<tr>
<td>10</td>
<td>32</td>
<td>251</td>
<td>1.261</td>
<td>0.947</td>
<td>0.0911</td>
<td>0.187***</td>
<td>0.103</td>
<td>0.1288</td>
<td>0.0731</td>
<td>0.1681</td>
<td>0.0845</td>
</tr>
</tbody>
</table>

$N_p$ = number of parent pairs; $V_p$ = variance of the parents; $N_o$ = number of offspring; $r$ = correlation between parents; $V_e$ = component of variance due to family means; $h_p^2$ = heritability in parental environment; $SE$ = standard error; $t_e$ = intraclass correlation of siblings; $h_o^2$ = heritability in the offspring environment. **, *** = $0.01 < P < 0.001$ and $P < 0.001$, respectively, for tests against $t_e = 0$. $h_o^2$ and $h_p^2$ are means weighted by $1 = (SE)^2$ and $SE = \sqrt{\sum/(SE)^2}$. 

**Notes:**

- $N_p$ = number of parent pairs; $V_p$ = variance of the parents; $N_o$ = number of offspring; $r$ = correlation between parents; $V_e$ = component of variance due to family means; $h_p^2$ = heritability in parental environment; $SE$ = standard error; $t_e$ = intraclass correlation of siblings; $h_o^2$ = heritability in the offspring environment. **, *** = $0.01 < P < 0.001$ and $P < 0.001$, respectively, for tests against $t_e = 0$. $h_o^2$ and $h_p^2$ are means weighted by $1 = (SE)^2$ and $SE = \sqrt{\sum/(SE)^2}$. 

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**Equation:**

$$h_e^2 = \frac{-1 + \sqrt{1 + 8rt_p}}{2r},\tag{1}$$

**Table A:**

The individual regressions were heterogeneous across rots so arithmetic means, $\bar{b}$, are given at the bottom with their empirical standard errors. Because of the high assortative mating, $r = 0.9692$, the regressions on single parents are essentially the same as the regression on the midparent which is $\bar{b} = 0.0789$ ($SE = 0.0347$) and this agrees well with the results of sib analysis where $h_p^2 = 0.0699$ ($SE = 0.0079$). The difference between them gives $t = 0.253$ (d.f. = 18, $P = 0.80$).

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Effsering ccrrected for assortative mating as in VPE

VA, is the total variance corrected for assortative mating and offspring were measured but heritability analysis is the total observed variance in the offspring, and $\bar{h}$ are arithmetic means of the regression coefficients and se are empirical standard errors.

**TABLE 2**

Offspring parent regression coefficients

<table>
<thead>
<tr>
<th>Population</th>
<th>$N_p$</th>
<th>$N_o$</th>
<th>$O-D$</th>
<th>$O-S$</th>
<th>$O-MP$</th>
<th>se</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>24</td>
<td>177</td>
<td>0.038</td>
<td>0.050</td>
<td>0.043</td>
<td>0.0486</td>
</tr>
<tr>
<td>2</td>
<td>12</td>
<td>98</td>
<td>-0.064</td>
<td>-0.084</td>
<td>-0.074</td>
<td>0.0674</td>
</tr>
<tr>
<td>3</td>
<td>18</td>
<td>112</td>
<td>0.085</td>
<td>0.094</td>
<td>0.090</td>
<td>0.0260</td>
</tr>
<tr>
<td>4</td>
<td>26</td>
<td>192</td>
<td>0.145</td>
<td>0.115</td>
<td>0.130</td>
<td>0.0447</td>
</tr>
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<td>5</td>
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<td>250</td>
<td>0.176</td>
<td>0.140</td>
<td>0.160</td>
<td>0.0351</td>
</tr>
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<td>6</td>
<td>31</td>
<td>244</td>
<td>0.106</td>
<td>0.090</td>
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<tr>
<td>7</td>
<td>33</td>
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<td>-0.009</td>
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<td>0.406</td>
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<td>-0.007</td>
<td>-0.006</td>
<td>0.0177</td>
</tr>
<tr>
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<td>251</td>
<td>0.019</td>
<td>0.047</td>
<td>0.031</td>
<td>0.0568</td>
</tr>
</tbody>
</table>

Regressions: $O-D = \text{offspring on dam}; O-S = \text{offspring on sire}; O-MP = \text{offspring on midparent}; \bar{h}$ are arithmetic means of the regression coefficients and se are empirical standard errors.

**TABLE 3**

Means and variances

<table>
<thead>
<tr>
<th>$M_p$</th>
<th>$V_p$</th>
<th>$V_a$</th>
<th>$V_i$</th>
<th>$M_o$</th>
<th>$V_i$</th>
<th>$V_o$</th>
<th>$V_r$</th>
<th>$V_e$</th>
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<tbody>
<tr>
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<td>3.448</td>
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<td>2.9859</td>
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<td>2.2350</td>
<td>2.073</td>
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<td>2.2913</td>
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<td>0.8584</td>
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<td>0.7566</td>
<td>0.7404</td>
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<td>0.5425</td>
<td>2.071</td>
<td>0.8675</td>
<td>0.8073</td>
<td>0.4728</td>
</tr>
<tr>
<td>9</td>
<td>1.995</td>
<td>9.326</td>
<td>0.4420</td>
<td>8.8840</td>
<td>2.067</td>
<td>0.8942</td>
<td>0.8838</td>
<td>0.4418</td>
</tr>
<tr>
<td>10</td>
<td>2.048</td>
<td>1.261</td>
<td>0.1624</td>
<td>1.0986</td>
<td>2.070</td>
<td>0.9694</td>
<td>0.9595</td>
<td>0.7971</td>
</tr>
</tbody>
</table>

$\bar{V}_a = 0.3720$ $\bar{V}_e = 5.9027$ $\bar{V}_o = 0.9601$ $\bar{V}_r = 0.5881$

$SE = 0.0342$ $SE = 1.3327$ $SE = 0.0764$ $SE = 0.0715$

is the total observed variance in the offspring, and $V_o$ is the total variance corrected for assortative mating as explained in APPENDIX A, Equation 7. The environmental components of variance for offspring and parents, $V_{OE}$ and $V_{PE}$ are obtained by subtracting $V_A$ from the respective total variances, $V_o$ and $V_p$. Some of the statistics of Table 3 can be combined with the nine additional rots where female parents and offspring were measured but heritability analysis was not done (in one case the parents were not measured). There were no statistical differences between these and the ten experimental rots. Figure 1 shows the distribution of means of the female parents in all 18 rots and of the progeny means of all 19 rots. The hatched areas indicate the ten experimental rots. The means and variances are very different comparing parents and progeny, and in a direction indicating that the natural environment with lower means is less favorable than the laboratory environment, and also indicating the the natural environment is much more variable than the laboratory environment.

Table 4 is a one way analysis of variance of the data shown in Figure 1 for parents and for progeny producing a statistical evaluation of these differences in variance. Not only are the parental means significantly heterogeneous, but so also are the offspring means (parents $F_{17,1066} = 41.2, P < 5 \times 10^{-4}$; offspring $F_{18,2445} = 9.286, P < 5 \times 10^{-4}$). The parental means are more variable than the offspring means; the approximate lower 95% confidence limit of the component due to the means, $V_M$, for the parents is 1.49, while the upper 95% confidence limit of the same component for offspring is .216. The variance within rots is larger for parents than offspring, $F_{1066,2445} = 4.03, P < 10^{-4}$.

Table 5 compiles the various components of variance for parents and offspring. For the parents, the components are taken from the analysis of variance...
of all rots in Table 4. The offspring can be regarded as a two level hierarchy. First, the between and within rot components are for all rots, Table 4. Then, the within rot components are taken from the ten experimental rots of Table 3 and divided into additive genetic component, $V_A$, and environmental component $V_{OE}$. The sum of these latter components, $V_O = 0.9601$ agrees well with the within component for all rots, $V_{ow} = 0.9868$. The latter is expected to be slightly inflated because the ten experimental rots contributed the slightly inflated $V_O$.

These components are rearranged and further partitioned in Table 6 which organizes the components by parental vs. offspring environment, genetic vs. environmental cause, and within vs. between rot. The environmental components within rots are obtained by subtracting $V_A$ (from ten experimental rots) from the within component from all rots. For the parental environment this is $V_{pw} - V_A = V_{PEW} = 3.6033$ which agrees well with $V_{PE} = 3.9027$ obtained for the ten experimental rots. For the offspring environment this

<table>
<thead>
<tr>
<th>TABLE 4</th>
</tr>
</thead>
<tbody>
<tr>
<td>One way analysis of variance of thorax length within and between rots for parents and for offspring</td>
</tr>
<tr>
<td>ss</td>
</tr>
<tr>
<td>Parents</td>
</tr>
<tr>
<td>Between rot means</td>
</tr>
<tr>
<td>Within rots</td>
</tr>
<tr>
<td>$V_b = 4.4492$</td>
</tr>
<tr>
<td>Offspring</td>
</tr>
<tr>
<td>Between rot means</td>
</tr>
<tr>
<td>Within rots</td>
</tr>
<tr>
<td>$V_b = 0.1295$</td>
</tr>
</tbody>
</table>

The effects of rot means are highly significant. For parents $F_{17,1066} = 41.2$ and for offspring $F_{18,2445} = 9.286$. $V_b$ is the component of variance due to means. Is $V_{ow} - V_A = V_{OEw} = 0.6148$ which also agrees with the value of $V_{OE} = 0.5881$ from the ten experimental rots. It is assumed that the variance between progeny means from different rots reared in the common laboratory environment, $V_{oB}$ of Table 5, is genetic and is designated $V_G$ in Table 6. It follows from this that subtracting $V_G$ from the between rot component in the parental environment, $V_{pB}$ of Table 5, the environmental component of the between rot differences can be obtained, $V_E$.

These components lead to five different kinds of heritability which are shown in Table 7. The within rot heritabilities in the offspring environment, $h^2_{oe}$ and parental environment, $h^2_{pw}$, agree reasonably well with the estimates in Table 1 which were obtained by computing the heritabilities for the ten experimental rots first and then pooling them. The "across rot" (AR) heritabilities refer to a population of individuals drawn from different rots independent of the genetic
Genotype-environment interaction: The previous sections assumed no genotype-environment interactions. This section will consider the constraints placed on these conclusions when allowing for the possibility of interactions between the offspring laboratory environment and the parental environment.

Because of the need to consider such interactions, especially for these experiments with assortative mating, Riska, Prout and Truelli (1989) have developed general statistical procedures for this kind of laboratory-nature experiment. They give procedures for estimating a lower bound on the heritability in nature which is the most information one can obtain from such an experimental design. Their paper should be consulted for derivations of the results to be used here.

Adapting their findings to the particular experiments reported here, it turns out that the lower bound on $h^2_p$ can be obtained by combining information from the variance of sibships with the offspring parent regression. (In the Riska, Prout and Truelli notation their "O" is their "L" for laboratory, and our "p" is their "N" for nature.)

For the sibling analysis, the Riska, Prout and Truelli result is,

$$2 t_p = k h^2_p (1 + r \gamma^2 h^2_p).$$

where,

\begin{align*}
\gamma &= \text{additive genetic correlation of the trait in the two environments.}
\end{align*}

The absence of genotype-environment interaction is defined by no difference in variance in the two environments, $k = 1$, and perfect correlation in the expression of the genotypes in the two environments, $\gamma = 1$. In this case (3) becomes the standard relationship for assortative mating given as Equation 1.

For regression of offspring on parent with genotype-environment interaction the relation is as follows:

$$\beta = \gamma \sqrt{k} h^2_p.$$  \hspace{1cm} (4)

This is a modification of the relationship pointed out by Lande in an appendix to Coyne and Beecham (1987).

Equations 3 and 4 can be combined to obtain an estimate of $\gamma^2 h^2_p$ as follows

$$\frac{2 t_p}{\beta^2} = \frac{1 + \gamma^2 h^2_p}{\gamma^2 h^2_p},$$

so that

$$\gamma^2 h^2_p = \frac{\beta^2}{2 t_p - \beta^2 r}. $$  \hspace{1cm} (5)

since $0 \leq \gamma^2 \leq 1$, then $\gamma^2 h^2_p$ gives a lower bound for $h^2_p$.

Using the means of $V_e$ and $V_p$ in Table 1, $t_p = 0.0494$ and the mean $r$ is 0.9692. The estimate of $\beta$ is $b = 0.072 \pm 0.041$ and not significantly different from zero ($P = 0.10$). This lack of significance is not surprising since its predicted value is $h^2_p = 0.0291$ (which does differ from zero because the components used to estimate it were statistically greater than zero).

Additional components of variance

<table>
<thead>
<tr>
<th></th>
<th>Parental environment</th>
<th>Offspring environment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Genetic</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Within rots</td>
<td>$V_a$</td>
<td>0.3720</td>
</tr>
<tr>
<td>Between rots</td>
<td>$V_C$</td>
<td>0.1295</td>
</tr>
<tr>
<td>Environmental</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Within rots</td>
<td>$V_{PEW}$</td>
<td>3.6033</td>
</tr>
<tr>
<td>Between rots</td>
<td>$V_E$</td>
<td>4.3197</td>
</tr>
</tbody>
</table>

All derived from Table 5. $V_C = V_{ob}$ of Table 5; $V_{PEW} = V_{pe} - V_a$ of Table 5; $V_E = V_{pe} - V_{ob}$ of Table 5; $V_{ob} = V_{ob} - V_a$ of Table 5.
TABLE 7
Heritabilities

<table>
<thead>
<tr>
<th>Offspring environment</th>
<th>$h^2$</th>
<th>SE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Within rots</td>
<td>$h^2_{ow} = \frac{\bar{V}}{V_A + V_{eo}}$</td>
<td>0.3770</td>
</tr>
<tr>
<td>Across rots</td>
<td>$h^2_{AR} = \frac{\bar{V}}{V_A + V_C + V_{eo}}$</td>
<td>0.4493</td>
</tr>
<tr>
<td>Parental environment</td>
<td>$h^2_{pw} = \frac{\bar{V}}{V_A + V_{ew}}$</td>
<td>0.0936</td>
</tr>
<tr>
<td>Across rots</td>
<td>$h^2_{AR} = \frac{\bar{V}}{V_A + V_C + V_{ew} + V_e}$</td>
<td>0.0595</td>
</tr>
<tr>
<td>Between rots</td>
<td>$h^2 = \frac{V_C}{V_G + V_e}$</td>
<td>0.0291</td>
</tr>
</tbody>
</table>

All variance components from Table 6.

$h \equiv 1$ and $\gamma \equiv 1$ which suggests that there may be no substantial genotype-environment interaction. Nevertheless, assuming there is such interaction, these results provide a lower bound on $V_A$ because $V_A \gamma^2 h^2 = \gamma^2 V_A$. Using $V_{pw} = 3.9753$ (Table 5) the result is $V_A \geq 0.2667$. This is a little smaller than the value used of $\bar{V}_A = 0.3720$ meaning that the values given as heritabilities in Table 7 would be somewhat reduced, and now become lower bounds.

In the previous section, Equation 2 shows that the computation of the heritability in the offspring environment, $h^2$, depends on first determining $h^2$. In this case with the possibility of genotype-environment interactions the situation is the same except that the determination of $h^2$ depends first on calculating $\gamma^2 h^2$ instead of $h^2$. Multiplying both sides of Equation 3 by $V_s$ results in the following relationship:

$$2V_s = V_{AO}(1 + \gamma^2 h^2), \quad (6)$$

where

$V_s = \text{variance of siblings as before}$

$V_{AO} = \text{additive genetic variance in the offspring environment or } V_A$.

From this point it is only necessary to proceed as in APPENDIX A, substituting $(1 + \gamma^2 h^2)$ for $(1 + rh^2)$, with the result,

$$h^2 = \frac{2t_c}{1 + \gamma^2 h^2(1 - t_o)}. \quad (7)$$

This is the same as Equation 2 with $\gamma^2 h^2$ substituted for $h^2$.

It is important to note this estimate of $h^2$ is a point estimate, and not a “bound” as is $\gamma^2 h^2$. Using mean values from Table 1, $h^2 = 0.4022$ which is close to $h^2 = 0.3592$ so that the within and between rot heritabilities in the offspring environment of Table 1 remain essentially the same.

It is not known how genotype environment interaction would affect the estimate of population heritability, $h^2_b$, because the offspring-parent regression of rot means does not differ statistically from zero, meaning that there is no regression information to be employed in procedures similar to the above.

**Tests for selection:** Although this experiment was designed for estimating heritability, the possibility was anticipated of detecting selection on larvae which might affect adult body size. The results are negative, but worth reporting. The conjecture is that if this genetic variation is subject to selection then this selection will be more intense in a stress environment than in an optimal one.

There were two opportunities for identifying variation in the nutritional environment of the larvae. Differences between flies emerging early versus late from the rots is one of them, and the other uses the average phenotypic size from different rots as an indicator of stress on the larvae, the smaller the size the greater the stress. As already reported we found no differences in any statistic between early and late flies.

Concerning fly size, most of the difference between rot means is environmental (Table 6). There is direct evidence that at least some of this could be due to crowding. The number of flies emerging from different rots ranged from 89 to 672, and there is a significant negative regression of body size on rot yield, $b = -0.063$ ($t = 2.49, d.f. = 15, P = 0.03$) suggesting a phenotypic effect on adults due to larval crowding (the across rot heritability may, thus, be oversimplified).

There are three possibilities for the effect of selection: greater stress might intensify directional selection for genetically larger size, for smaller size or for an optimum size. The first hypothesis proposes that
the phenotypically smaller parents were subject to intense selection as larvae of a kind which would result in larger progeny in the optimal laboratory environment as compared to the progeny of the larger parents who as larvae were not subject to such selection. This predicts a negative regression of offspring mean and parental mean. The second hypothesis proposes the opposite kind of selection, predicting a positive regression of offspring mean on parental mean, and the third hypothesis is that selection is of the kind observed by LINNEY, BARNES and KEARSEY (1971) where larval crowding imposed stabilizing selection on adult bristle count which, in this case with adult parental size as an indicator of stress as larvae, predicts a positive regression of additive genetic variance on parental mean. None of these regressions were significant. The one case that was suggestive has already been noted: the positive but not significant regression of phenotypic offspring mean on parental mean where larval stress results in genetically smaller adults. Perhaps a more extensive survey would confirm this relationship. This is of some interest because this particular pattern would be consistent with the conjecture of WILKINSON and others (ROFF 1981; WILKINSON 1987) that stress conditions favor early developing larvae and small adult body size, although this was not observed by BIERBAUM, MUELLER and AYALA (1989). Such an effect could provide the counter selection to the positive selection for body size in adult females (ROBERTSON 1957) and males (WILKINSON 1987; PARTRIDGE, HOFFMAN and JONES 1987). A complication of this pattern of a positive correlation of parent and offspring size across rots is that this is also predicted from the simple population heritability because they use for parents either flies from different rots or reared from apples and the latter were pooled. Individual apples are comparable to our groups at this level of grouping. This is in contrast to D. melanogaster where apples, for instance, certainly constitute patches but there must be many other unknown groupings of resources which could be just as important as or even more important than apples. That rots are relevant patches for body size is indicated by the fact that about half of the environmental differences in individual body size is due to differences between rots (Table 6). Also, there appear to be genetic differences between offspring of flies from different rots.

The importance of studying the ecology of heritability in relation to natural selection on the trait (BARKER and THOMAS 1987). We chose to study body size, because it is probably subject to natural selection. As already mentioned large males have an advantage in mating, large females have a higher fertility and, as noted above, it has been conjectured that there is counter selection for small adult body size in the larvae.

The response to such selection depends on heritability, and our emphasis is that heritability depends on ecology. If males emerging from a given rot mate locally, then the component of selection response due to male mating success would be determined by the within rot heritability, while if males first disperse from their rot of origin and subsequently aggregate when competing for females, the selection response due to mating would be determined by the lower across rot heritability. Similarly, if the cohort of females from each rot laid their eggs in isolation, then the within rot heritability would apply, while if females from different rots were aggregated by being attracted to the same egg laying site (new rots), which almost certainly happens, then the across rot heritability would apply. The actual behavior in this regard in D. buzzatii is not known. The point is that the heritability appropriate for selection response could be different between the two sexes for ecological and behavioral reasons, although in this case the difference is not great.

As stated in the introduction, the accumulating data on heritability of life history traits in animals and plants is mostly the heritability in the laboratory, greenhouse or garden. We suggest that these studies need to be extended not only to field conditions in...
Heritability in Nature

...general but to the relationship between the particular life history trait and the relevant ecological circumstances, because evidently environments are more often patchy than not. As noted by van Noordwijk and Gehhardt (1987, p. 74), in discussion of still other ecological problems, the appropriate heritability for selection on a particular trait could be quite different from that indicated by a gross estimate of the wild heritability such as that of Coyne and Beecham (1987).

For the case at hand, if it turns out that there is net stabilizing selection on body size in Drosophila, this will explain why flies are not larger, but will not explain the existence of the genetic variation.

As noted in the introduction, the Bulmer (1989) and Turelli (1988) reviews of stabilizing selection with pleiotropy identify heritability as an important parameter. Also, in both the Gaussian (Lande 1975) and the rare allele (Barton and Turelli 1987) analysis of mutation-selection balance, the amount of genetic variance maintained is an increasing function of the environmental variance. Therefore, determining the relevant heritability and associated environmental variance will play an important role in deciding whether the observed genetic variance can be explained by simple stabilizing selection or whether some form of balancing selection must be sought, such as proposed by Gillespie and Turelli (1989).

Finally, our study suggests some kind of genetic differentiation between patches. The laboratory offspring showed average differences between rots. It has already been speculated that this could be due to selection. It also could be due to genetic drift due to a finite number of females founding each rot. Such an effect can be evaluated by considering the effect of genetic drift on partitioning the additive genetic variance within and between rots. The value of $F_{ST}$ is related to the ratio of between population ($V_G$) and within population ($\bar{V}_A$) genetic variance as follows (Falconer 1981, p. 241).

$$\frac{V_G}{\bar{V}_A} = \frac{2F}{1 - F}$$

Solving for $F$,

$$F = \frac{V_G}{V_G + 2\bar{V}_A}.$$  

Using $V_G$ and $\bar{V}_A$ from Table 6, $F = 0.1483$ with approximate standard error, $SE \approx 0.0462$. For one round of drift $F = 1/(2N)$ or $N = 1/(2F)$ giving $N = 3.4$, $SE \approx 1.05$. For two rounds of drift $F = 1/N - 1/4N^2$ or $N = (1 + \sqrt{1 - F})/2F$ giving $N = 6.5$, $SE \approx 2.03$. Santos, Ruiz and Fontdevila (1989) state that rotting cladodes could support "at most, two generations." However, the addition of external recruits to the second generation means that $N$ must be smaller than 6.5 to account for the observed $F$.

The above authors, using $F$ statistics from inversions, estimate $N = 10$, and the Barker and Fredline (1985) study of the productivity of D. buzzatii indicates that $(1/2)N = 5$ females would be capable of producing offspring up to the 672/rot which we observed. The upper 95% confidence limit on $N$ for one round of drift is 5.5 and for two rounds is $N = 10.5$. When considering the unaccounted for sources of error this result would appear to be consistent with the estimate of Santos, Ruiz and Fontdevila (1989) and the biological considerations of Barker and Fredline. The simplest conclusion, then, is that the observed $F_{ST}$ is due to genetic drift, and this would be an equilibrium value because with ephemeral patches, the drift cannot accumulate. More studies of the ecology of egg deposition by females and also of differences between laboratory progeny of flies from different rots would be required to confirm this conclusion as opposed to selection being the cause of the differences as discussed earlier.

We therefore conclude that when the net selection on body size is understood, any theory about the maintenance of this variation will have to take into account not only the possibility that the heritability governing selection response could be as low as 5% or 10%, as already noted, but also that the subdivision by these ephemeral patches results, by genetic drift alone, in a positive contribution to the genetic variance, although this appears not to be very large, amounting to a factor of 1.148 (1 + $F$ as in Falconer, 1981, p. 241).

It seems evident to us that a full understanding of polygenic variation in natural populations, including explanations for its existence, depends in a most important way on knowing the heritability in nature, for reasons discussed. In many cases heritability in the wild can be determined directly—it is empirical. In many other cases this is difficult or impossible as it would be for D. buzzatii and many other species of Drosophila; and it is easy to think of other organisms where the reproductive ecology precludes direct estimates of natural heritability. In these cases experiments involving some mix of artificial and natural environments need to be devised, exploiting the ecology peculiar to the organism as we have tried to do with D. buzzatii.

The work done was done while T.P. held a University of New England Visiting Research Fellowship, and was supported by a grant to J.S.F.B. from the Australian Research Grants Scheme. We are grateful to Annette Edmonds and Chris Leger for technical assistance and to Bruce Riska and Michael Turelli for helpful suggestions with the manuscript.

LITERATURE CITED

Barker, J. S. F., 1982 Population genetics of Opuntia breeding Drosophila in Australia, pp. 209–224 in Ecological Genetics and


Communicating editor: A. G. Clark

APPENDIX A

Estimates of heritability with assortative mating: The following relationship for the full sib variance we take as given (see Falconer, 1981, p. 165).

\[ V_r = 1/4 h_p^2 V_p (1 + r h_p^2) \]  

or

\[ V_r = V_p = 1/4 h_p^2 (1 + r h_p^2) \]  

where,

- \( V_r \) = component of variance due to family means,
- \( r \) = correlation between parents due to assortative mating,
- \( h_p^2 \) = heritability in the parental environment,
- \( V_p \) = phenotypic variance of parents.

For a derivation of (1) showing that \( h_p^2 \) and \( V_p \) refer specifically to the parental environment, see Prout (1958b). Solving (1B) for \( h_p^2 \) gives Equation 1 in the text (and Falconer, 1981, p. 164, line -1).

The offspring heritability \( h_p^2 \) is different from the parental heritability because of the difference in the environmental component of variance.

The object is to compute

\[ h_p^2 = \frac{V_A}{V_A + V_{so}} \]  

(2)
Assuming that the assortative mating does not change the segregation variance within families, $V_w$, then

$$V_w = \frac{1}{2} V_A + V_{so}.$$  

(3)

Letting $h_s^2 V_p = V_A$ in Equation 1A, and solving (1A) for $V_A$ gives

$$\frac{2V_s}{1 + rh_s^2} = V_A,$$  

(4)

and

$$\frac{V_s}{1 + rh_s^2} = \frac{1}{2} V_A.$$  

(5)

The numerator of (2) is (4) and the denominator of (2) is obtained by adding (3) and (5) giving

$$h_s^2 = \frac{2V_s(1 + rh_s^2)^{-1}}{V_w + V_s(1 + rh_s^2)^{-1}}.$$  

(6)

The intraclass correlation, $t_s$, observed in the offspring is defined,

$$t_s = \frac{V_s}{V_s'},$$

and

$$1 - t_s = \frac{V_w}{V_s'}. $$

Solving the above for $V_s$ and $V_s'$ and substituting for the latter in (6) gives equation (2) in the text. Note that $V_s'$ is the observed offspring variance which is inflated due to assortative mating. The corrected offspring variance, $V_s$, recorded in Table 3, is the denominator of (6) or

$$V_s = V_w + V_s(1 + rh_s^2)^{-1},$$

or, since

$$V_s' = V_w + V_s,$$

$$V_s = V_s' - V_s rh_s^2(1 + rh_s^2)^{-1},$$

(7)

APPENDIX B

Approximate variances of $h_s^2$ and $h_t^2$: Both variances were obtained using the "delta" method (KENDALL and STUART, 1976, Ch 10.6), where the variable whose variance is desired is a function of one or more variables whose variances are known.

$$Y = f(X_i)$$

$$\text{Var}(Y) = \sum \left( \frac{\partial Y}{\partial X_i} \right)^2 \text{Var}(X_i) + 2 \sum_{i<j} \frac{\partial Y}{\partial X_i} \frac{\partial Y}{\partial X_j} \text{Cov}(X_i X_j),$$

where

$$\text{Var}(V_s) = \frac{2 V_s^2}{df + 2},$$

$$\frac{1}{n^2} (\text{Var}(V_{bs}) + \text{Var}(V_o)),$$

$V_{bs} =$ variance between sib means obtained as for

$$\text{Var}(V_s)$$

$V_o =$ variance within sibships obtained as for

$$\text{Var}(V_s)$$

$n =$ adjusted number of individuals per sibship.

Offspring heritability, $h_s^2$

$$\text{Var}(h_s^2) = \frac{4}{((2r h_s^2 + 1)V_p)^2} (\text{Var}(V_o) + t_s^2 \text{var}(V_s))$$

where

$$\text{Var}(V_o) = \frac{2 V_o^2}{df + 2},$$

$$\frac{1}{n^2} (\text{Var}(V_{bs}) + \text{Var}(V_o)),$$

$V_{bs} =$ variance between sib means obtained as for

$$\text{Var}(V_s)$$

$V_o =$ variance within sibships obtained as for

$$\text{Var}(V_s)$$

$n =$ adjusted number of individuals per sibship.