The Evolution of Dimorphic Traits: Predicting the Genetic Correlation Between Environments

Derek A. Roff

Department of Biology, McGill University, Montréal, Québec, Canada H3A 1B1

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ABSTRACT

Many traits vary in a dichotomous manner, although the underlying genetic determination is polygenic. The genetic basis of such dimorphic traits can be analyzed using the threshold model, in which it is assumed that there is a continuously distributed underlying character and the phenotype is determined by whether the character is above or below a threshold. Threshold traits frequently vary with environmental variables such as photoperiod, temperature and density. This effect can be accounted for using a threshold model in which (1) there is a critical value of the environmental variable at which a genotype switches to the alternate morph, and (2) switch (threshold) points are normally distributed in the population. I term this the environmental threshold (ET) model. I show that the ET model predicts that across environments differing in only one factor the genetic correlation will be 1. This prediction is supported by data from three wing dimorphic insects. Evidence is presented that the genetic correlation between environments differing in two components (temperature and photoperiod) is less than 1.

MANY traits occur as dimorphic, rather than continuously distributed characters: for example, pupal color in swallowtail butterflies (HAZEL 1977), shell shape in acorn barnacles (LIVELY 1986), cyclomorphosis in zooplankters (DODSON 1989), pedomorphosis in amphibia (SEMLITSCH 1985), dental dimorphism in some species of fish (SKÚLASON, NOAKES and SNORRASON 1989; MEYER 1990), wing dimorphism in insects (HARRISON 1980; ROFF 1986a), sex ratio in turtles (BULL, VOGT and BULMER 1982) and diapause in insects (MOUSSEAU and ROFF 1989).

Although only two phenotypes are discernable, dimorphic variation may be due to the additive effect of many loci, the particular manifestation of the trait being a function of a threshold of sensitivity. According to the threshold model, a continuously varying character underlies the expression of the trait, individuals with values lying above the threshold developing into one morph, individuals lying below the threshold developing into the other morph (FALCONER 1989).

The threshold model was developed specifically to address the question of discrete states in a fixed environment. However, the proportion of each morph in a population frequently varies both with genotype and environment. For example, in wing dimorphic insects both temperature and genotype determine the proportion of macropterous (fully winged, flight capable) individuals (HARRISON 1980; ROFF 1986a). Similarly, the proportion of males in some reptile species is determined by incubation temperature of the eggs as well as genotype (BULL, VOGT and BULMER 1982; JANZEN 1992). A character expressed in two environments can be viewed as two characters that are genetically correlated (FALCONER 1952). Such an approach becomes mathematically intractable if there are a large number of environments. In the case of variation in a single environmental factor, we can approach the problem by mathematically describing the relationship between the character and the environment (the reaction norm), and then viewing the model parameters as themselves inherited characters. In a recent paper, HAZEL, SMOCK and JOHNSON (1990) proposed an alternate model for reaction norms of dimorphic traits. This model is a special case of the more general approach outlined above and is notable in that, as shown below, it predicts the genetic correlation between environments differing in a single factor (*e.g.*, two different temperatures) to be 1. This prediction is tested using data from three wing dimorphic insects, the sand cricket, *Gryllus firmus*, the ground cricket, *Dianemobius fascipes*, and the small brown planthopper, *Laodelphax striatellus*.

THE ENVIRONMENTAL THRESHOLD MODEL

The threshold model, as typically presented, assumes a fixed threshold and a continuously distributed underlying trait. For example, in the case of wing dimorphism, it has been hypothesized that wing production is governed by the titer of juvenile hormone (JH) at a particular stage of larval development (SOUTHWOOD 1961; WIGGLESWORTH 1961): larvae in which JH exceeds a critical threshold develop into the

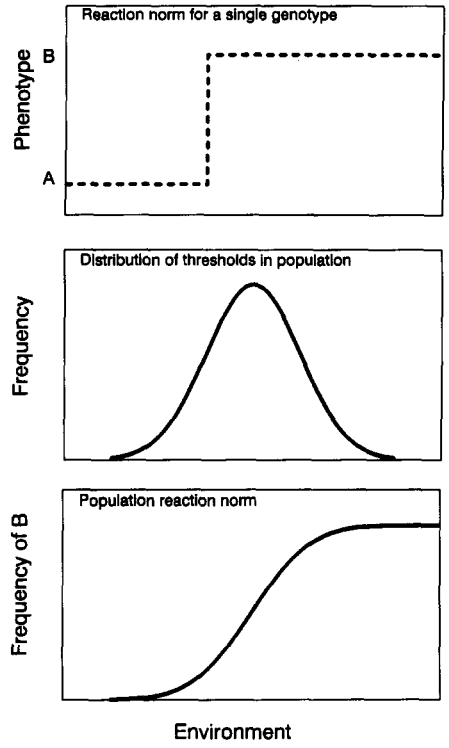

FIGURE 1.—Schematic illustration of the environmental threshold model for a dimorphic trait that varies with genotype and environment. For each genotype there is a fixed threshold of the environmental variable that switches the phenotype to the alternate morph (A, B, upper panel). These thresholds are normally distributed in the population (middle panel), giving rise to a population reaction norm that is a cumulative normal (bottom panel).

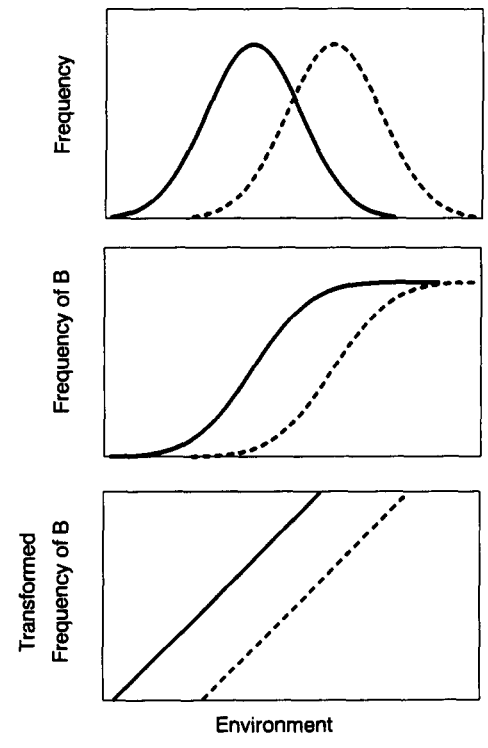

FIGURE 2.—Schematic illustration of the effect of selection on the norm of reaction under the ET model. Selection for an increased frequency of one morph shifts the distribution of threshold values at which the switch between morphs occurs (dashed curve in top panel), causing a concomitant shift in the reaction norm (middle panel). By appropriate transformation (*e.g.*, probit) the two curves can be linearized, the curve after selection remaining parallel to the original.

micropterous (short winged and flightless) morph while larva with JH titers below this threshold develop into macropters (long winged and flight capable). Genetic variation in wing dimorphism is then a consequence of genetic variation in JH titers (ROFF 1986a). Experimental support for this model comes from analysis of the two cricket species *Gryllus rubens* and *G. firmus* (ZERA and TIEBEL 1988, 1989; YADLOWSKI and FAIRBAIRN, unpublished data).

In contrast to the above, the model proposed by HAZEL, SMOCK and JOHNSON (1990) assumes that it is the threshold that is variable and the underlying trait distribution that is fixed. It is assumed that each genotype has a switch point, or threshold, along the environmental gradient at which the phenotypic expression of the genotype abruptly shifts from one morph to the other (Figure 1). I shall refer to this model as the environmental threshold (ET) model. To apply standard quantitative genetic theory to the ET model, we make the usual assumption that the character, in this case the switch point, is normally distributed in the population. As a consequence, the norm of reaction for the population (the relationship between proportion of a particular morph and the value of the environmental variable, as opposed to the relationship for a particular genotype as shown in the top panel of Figure 1) will be a cumulative normal (Figure 1). Since the distribution of thresholds in each environment along the gradient is, by definition, the same, it follows that while the proportion of a morph will vary across environments, the heritability of the trait will not. Thus, if we designate one environment as x and the other as y, we have $h_x^2 = h_y^2$, and for the phenotypic variances, $\sigma_x^2 = \sigma_y^2$. The genetic correlation across environments along the gradient will be +1. This can be demonstrated in two ways. First, by changing the environment we do not change the underlying character, only its expression on the 0–1 scale: since the method of estimation corrects for this (FALCONER 1989), we are in fact measuring the same trait independently of the environment, and therefore, the genetic correlation must by definition be +1. The second approach, relevant to the later analysis, considers the effect of selection on the reaction norm. Selection will shift the distribution of switch points, thereby shifting the reaction norm by the same amount (Figure 2). Since the reaction norms are parallel there is no G × E interaction and the genetic correlation is 1. This can be shown formally as follows: the genetic

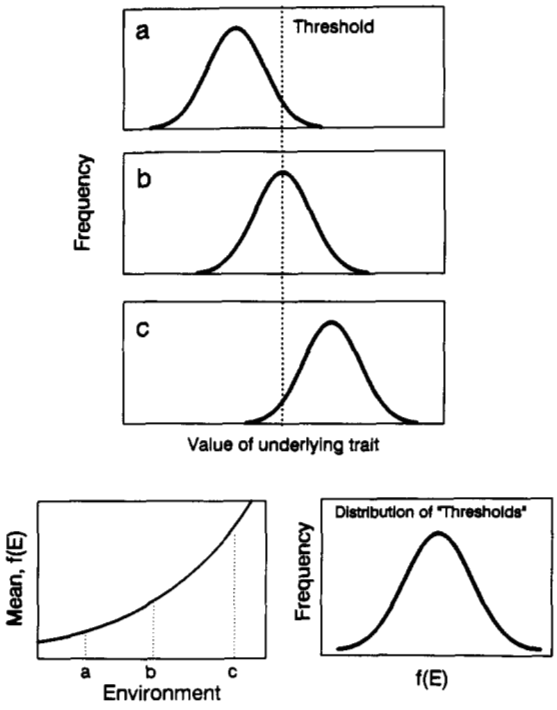

FIGURE 3.—Top three panels (a, b, c) show hypothetical shift in the distribution of the continuously distributed trait underlying the expression of a dimorphic character with variation in a single environmental factor (a < b < c). Individuals lying to the right of the fixed threshold develop into one morph, individuals to the left into the alternate. The change in the value of the mean can be expressed as a monotonic function, $f(E)$, of the environment (bottom left panel). By transforming the environmental value using $f(E)$, the trait can be mathematically described as a normal distribution of thresholds as in the ET model.

correlation between x and y, r_A, is given by (FALCONER 1989, p. 318),

$$r_A = \frac{R_y}{ih_x h_y \sigma_y} = \frac{R_y}{\frac{R_x}{h_x^2 \sigma_x} h_x h_y \sigma_y} \quad (1)$$

where R_x is the direct response to selection on x, R_y is the correlated response of y to selection on x, and i is the selection intensity. From the considerations above, $\sigma_y = \sigma_x$, $h_x = h_y$, and the correlated response of y is the same as the response of x, $R_x = R_y$. Substituting in Equation 1 gives $r_A = 1$.

To relate the ET model to the more usual model in which the threshold remains fixed and the underlying character (*e.g.*, JH titer) varies along the environmental gradient we proceed as follows. First, we assume that the only action of the environmental factor is to shift the distribution of the underlying character (Figure 3). Thus, we can define a function $f(E)$ that describes the relationship between the mean and the environmental value, E. We can now rescale the character value such that the underlying character remains fixed and the threshold varies: the "threshold" when the environment has the value E is $T - f(E)$,

TABLE 1

Mean proportion of macropterous adult *G. firmus* produced under three different photoperiod/temperature combinations

	17/30	12/30	15/25
Female	0.42	0.21	0.13
Male	0.21	0.19	0.05

where T is the value of the threshold on the original scale. The distribution of "thresholds" will be normal on the transformed environmental scale $f(E)$. In this model the genetic correlation of 1 arises from the assumption that the same ranking applies along the environmental gradient (*i.e.*, a simple shifting of the distribution), which is equivalent to assuming that the function $f(E)$ applies to all genotypes. If this is not the case then the genetic correlation will be less than 1. In many cases traits vary in response to more than one variable: for example, wing dimorphism in insects is frequently responsive to both photoperiod and temperature (HONEK 1976). The above argument can be extended to two environments, a genetic correlation of 1 then requiring that a common function, say $f(E_1, E_2)$ where the two environmental variables are E_1 and E_2, applies to all genotypes.

EMPIRICAL TESTS

Test 1. *G. firmus*: Crickets were raised in full sib groups of 60 hatchlings in mouse cages (29 cm long × 19cm wide × 13 cm high), and fed *ad libitum* with rabbit chow. Full details of the rearing regime and history of the stock are given in ROFF (1986b). Twenty-two families were each split upon hatching into four groups (cages) and raised under four photoperiod/temperature combinations (hr of light/temp): 17/30; 12/30; 15/25; 12/25. Only two families produced macropterous adults under the last combination (12/25), and therefore the data for this combination could not be used. The proportion macropterous produced under the other three combinations varied widely, from 0.05 to 0.42 (Table 1).

The value of the genetic correlation between environments can be estimated by the family mean correlation (VIA 1984), where in the present case, the family mean is the proportion of macropterous adults per family. The observed genetic correlation will be less than the true value because the numerator in the formula for the correlation contains sampling error in addition to genetic covariance, and the denominator may be inflated by within-family error (VIA 1984). Because Fisher's z-transformation goes to infinity as the correlation approaches 1, it is not possible to test an estimated proportion against an expected value of 1 (SOKAL and ROHLF 1981, pp. 583–591). Therefore, I estimated the effect of sampling variation on the expected correlation by Monte Carlo simulation using

a modification of the model of OLAUSSON and RÖNNINGEN (1975). The phenotypic value of the underlying continuous variable for the ith individual from full sib family j, $P_{i,j}$ is equal to

$$P_{i,j} = X_j \sqrt{\frac{1}{2} h^2} + Y_{i,j} \sqrt{1 - \frac{1}{2} h^2} \qquad (2)$$

where X_j is a random normal variate with zero mean and unit variance common to family j, $Y_{i,j}$ is a random normal variate with zero mean and unit variance unique to individual i,j, and h^2 is the heritability of the trait. The phenotypic value of the individual on the expressed dimorphic scale is determined from the value of the underlying variable relative to the threshold value. The latter, z, is the abscissa on the standardized normal curve corresponding to the proportion macropterous, p, in the population. The formula given by HAMAKER (1978) was used to derive z from p. A genetic correlation of +1 was simulated by generating a full sib family of size N, and then dividing the group into two environments specified by two different values of p. To conform approximately to the regimes 17/30 and 12/30, the two values of p were set at 0.42 and 0.21, respectively (Table 1). The number per environment, $N/2$, was set at 16, the average number of individuals per sex per cage. The proportion per cage was transformed (arcsine-square root) prior to the estimation of the correlation between cages. The mean correlation, r, based on 100 replicates is 0.744 (SD = 0.111), and even when $N/2$ is increased to 100 the mean value of r is still only 0.942 (SD = 0.024). These results demonstrate that even when the genetic correlation is +1 the method of family mean estimation may be seriously biased downward.

In addition to the present experiment, data from split family rearings are available from rearings reported in ROFF (1986b). There are significant correlations across all environments (Table 2), confirming the existence of positive genetic correlations. The correlations across the two environments differing in only a single factor (17/30 *vs.* 12/30) are the highest, r ranging from 0.79–0.87, and within the 95% confidence region obtained from the simulation. The genetic correlation between environments differing in only a single factor (17/30 *vs.* 12/30) is significantly higher (one-tailed test, z-transformation method, SOKAL and ROHLF 1981) than those between environments differing in both photoperiod and temperature (17/30 *vs.* 15/25, and 12/30 *vs.* 15/25, comparisons a-b and b-c, Table 2). There is no significant difference between the two environments differing in both photoperiod and temperature (comparison b-c, Table 2).

Test 2. *D. fascipes:* According to the ET model, after several generations of selection the norms of reaction curves should be shifted but not changed in shape. Because the norm of reaction curve is predicted to be cumulative normal, it should be linearized by transformation to z, where z is the abscissa on the unit normal corresponding to the proportion macropterous (Figure 1). Selection for, say, increased macroptery is predicted to shift the reaction curve to the left, but it should remain parallel to the reaction curve of the unselected population.

MASAKI and SENO (1990) selected for increased proportion macropterous in a photoperiod of 12L:12D (LW line), and for increased proportion micropterous in a photoperiod of 13L:11D (SW line). In each generation a subsample of each selected line was reared in the photoperiodic regime under which the line was not being selected. The heritability of wing dimorphism in *D. fascipes* is moderate ($h^2 = 0.30$), and a significant change in proportion macroptery was achieved within 14 generations (Figure 4). The correlated response to selection can be predicted using data from the base population (generation zero) and the response of the selected lines. We first transform the environmental variable, x, by arbitrarily setting $x = 0$ for a photoperiod of 12L:12D, and $x = 1$ for 13L:11D. The estimated relationship between the transformed proportion macropterous and x in the base population is $z = -0.89 + 1.13x$. Assuming a genetic correlation of 1, the predicted correlated response of the LW line is given by $z_C = z_{LW} + 1.13$, where z_C is the transformed predicted proportion macropterous, and z_{LW} is the transformed observed proportion macropterous in the LW line. Similarly, the predicted correlated response in the SW line is given by $z_C = z_{SW} - 1.13$. There is excellent agreement

TABLE 2

Family mean correlations across environments in the incidence of macroptery

	17/30	12/30	15/25
17/30		0.87***[a] 0.83***[1]	0.63**[b]
12/30	0.79***[1] 0.81***[a]		0.65**[c]
15/25	0.49*[b]	0.58**[c]	

* $P < 0.05$; ** $P < 0.01$; *** $P < 0.001$.
[1] Data from ROFF (1986b).

Comparison	Females		Males	
	t	P(one-tailed)	t	P(one-tailed)
a,b	1.76	0.04	2.38	0.01
a,c	1.63	0.05	1.99	0.02
b,c	0.11	0.46	1.37	0.09

Results for females are shown in the upper portion, males in the lower. Comparisons among environments restricted to data from the experiment outlined in this paper, shown by superscripts a, b, c. All statistical tests done using Fisher's z-transformation (SOKAL and ROHLF 1981).

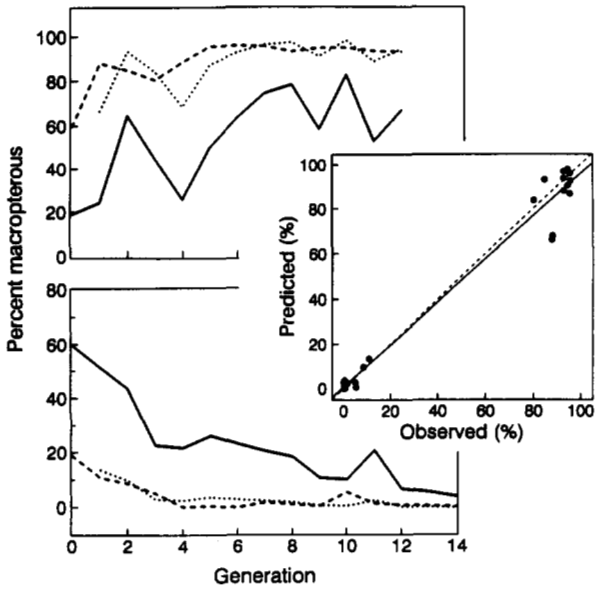

FIGURE 4.—Direct (solid lines) and correlated (dashed lines) responses to selection for increased (upper panel) and decreased (lower panel) macroptery in the ground cricket, *D. fascipes*. Selection for increased macroptery carried out at a photoperiod of 12L:12D, and the correlated response measured at 13L:11D. Selection for decreased macroptery carried out at 13L:11D and the correlated response measured at 12L:12D. Dotted lines show the predicted correlated response. Middle panel shows the regression of predicted correlated response on observed ($r = 0.99$, $n = 26$, $P < 0.001$). Data from MASAKI and SENO (1990).

between observed and predicted correlated responses ($r = 0.99$, $n = 26$, $P < 0.001$, Figure 4).

Test 3. *L. striatellus:* Selection for decreased macroptery in the small brown planthopper, *L. striatellus*, produced a highly significant response, the incidence declining from about 80% in the initial population to about 10% after 13 generations of selection (MORI and NAKASUJI 1990). The effect of density on the incidence of macroptery was measured in generations 1, 5 and 11. In this case density can be considered an environmental variable in the same manner as temperature or photoperiod. The ET model predicts that the transformed reaction norms will be parallel, with that from the initial generation being to the left of that from generation 5, which itself will be to the left of that from generation 11. These predictions are upheld (Figure 5), there being highly significant effects due to density ($P < 0.001$) and generation ($P < 0.001$), but no significant interaction ($P = 0.85$).

DISCUSSION

A corollary of the model proposed by HAZEL, SMOCK and JOHNSON (1990) for the inheritance of dimorphic traits is that the genetic correlation between two environments differing in only a single factor such as photoperiod, temperature or density will be exactly 1. The three tests presented in this paper all utilize the phenomenon of wing dimorphism

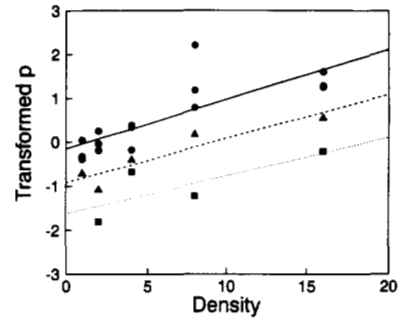

FIGURE 5.—Reaction norms for the small brown planthopper, *L. striatellus*, in the initial population (circles), after 5 (triangles) and 11 (squares) generations of selection. Lines show regressions fitted to each generation separately. Data from MORI AND NAKASUJI (1990).

in insects. For *G. firmus* the estimated genetic correlation between two environments differing in photoperiod (17 hr and 12 hr) was estimated using family mean correlation. In his analysis of the inheritance of sex ratio in the snapping turtle, *Chelydra serpentina*, JANZEN (1992) used the same approach to estimate the genetic correlation between environments differing in temperature. The usual statistical test for correlation, Fisher's z-transformation, precludes the testing of the hypothesis of an expected correlation of 1 because z approaches infinity as the correlation approaches 1. JANZEN (1992) attempted to overcome this problem by use of the jackknife to estimate confidence limits. This approach is inadequate for two reasons: first, the jackknife should not be used unless it can be shown either theoretically or by simulation that the estimated confidence limits are correct (POTVIN and ROFF 1993). The second reason is that because of sampling variation the expected correlation using the family mean correlation will be less than 1 even when the hypothesis $r_A = 1$ is correct. This arises because the proportion estimated for each family will itself have sampling error. The deletion of single families in the jackknife method in no way corrects for this bias. The expected family mean correlation and its distribution can be estimated using Monte Carlo simulation. For *G. firmus* the genetic correlation between 17/30 and 12/30 is consistent with the correlation estimated by simulation under the hypothesis $r_A = 1$. JANZEN's analysis also failed to find any G × E interaction. However, these tests cannot exclude the possibility of a genetic correlation that is substantially less than 1, and the simulation suggests that very large sample size (approximately 100 individuals per family) may be required for the expected correlation to be close to 1 and the plausible range of values to be small.

MASAKI and SENO (1990) selected for increased and decreased macroptery in the cricket, *D. fascipes*, raising the LW line in one photoperiod (12L:12D) and the SW line in another (13L:11D). Each generation a subsample of crickets were raised in the alternate

regime: the ET model can be used to predict the correlated response in this regime. The predicted responses closely match the observed (Figure 4), and thus the hypothesis $r_A = 1$ cannot be rejected. This test does not directly answer the question of whether a genetic correlation substantially less than 1 would also be consistent with the data. To predict the correlated response for a genetic correlation less than 1 requires estimates of the direct response, the heritabilities and the phenotypic variances (see Equation 1). Such data are not available in the present case. However, the fact that the predicted and observed responses are so highly correlated ($r = 0.99$, $n = 26$) suggests that alternate models are unlikely to fit the data more satisfactorily.

Of the three tests, that using the selection experiment of MORI and NAKASUJI (1990) on the planthopper, *L. striatellus*, is the most convincing. Utilizing the general model outlined in the introduction, we can write the norm of reaction as some function of density such as $g(d, p_1, p_2 \ldots)$, where d is density and the ps are parameter values. Thus, for example, the function might be $P = p_1 + p_2 d$, or $P = p_1 + p_2 d + p_3 d^2$, where P is the proportion macroptererous or some transformation thereof. The parameter values are then considered to be inherited traits, which themselves could be correlated with each other. Selection on proportion macropterous will act to change the parameter values. The ET model postulates $P = p_1 + p_2 d$, where P is the transformed proportion (=the abscissa on the unit normal corresponding to the proportion macropterous), with $p_1 = -\mu/\sigma^2$ and $p_2 = 1/\sigma^2$, where μ is the density at which 50% of adults are macropterous, and σ^2 is the phenotypic variance. Selection shifts μ but does not change σ^2; accordingly, the norm of reaction moves under selection but does not change its slope. The aforementioned transformation of P clearly linearizes the reaction norms and there is no statistical evidence for an interaction between the lines (Figure 5). Even when the lines are fitted independently, as shown in Figure 5, there is no substantial variation in slope.

The critical assumption of the ET model is that there is a single and unique switch point for each genotype: that is, when raised in a particular environment, a given genotype will always develop into the same morph. An alternative hypothesis, called "adaptive coin-flipping" (KAPLAN and COOPER 1984), is that even in an invariant environment individuals of the same genotype may differ in phenotype. This hypothesis requires that there are some probabilistic processes happening during development, evidence for which is scant (KAPLAN and COOPER 1984). Such a model is not required to adequately account for the data reported in this paper.

All three tests reported here are based upon the population consequences of the ET model. Obviously, the most stringent test is to show that the basic assumption of a step function for each genotype is correct. This is not possible with sexually reproducing animals but might be feasible using a parthenogenetic species. In the case of wing dimorphism a possible candidate is aphids, which are wing dimorphic and at some stage in their life cycle are parthenogenetic.

The data from *G. firmus* suggests that the genetic correlation between environments differing along two axes (temperature and photoperiod) is less than 1 (Table 1). Since separate sensory systems are involved and possibly different biochemical pathways, this result is perhaps not surprising. Further experiments are required to determine the correlation structure between the several environmental factors that can influence wing morph.

The three tests reported in this paper jointly provide support for the ET model. However, the difficulties of establishing narrow bounds on the genetic correlation necessitate more experimental support; experiments utilizing the family mean correlation require larger family sizes, while experiments based on the correlated response to selection need to be designed to measure both the genetic parameters required to estimate r_A and to examine the shift in the norm of reaction during the course of selection. Finally, utilization of clonal organisms may permit a direct test of the critical assumptions of the ET model that there is a single switch point for each genotype.

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