Cryptic Evolution: does Environmental Deterioration have a Genetic Basis?

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Keywords: Quantitative Genetics, Indirect Genetic Effects, Cryptic Evolution & Competition

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Abstract

Cryptic evolution has been defined as adaptive evolutionary change being masked by concurrent environmental change. Empirical studies of cryptic evolution have usually invoked a changing climate and/or increasing population density as the form of detrimental environmental change experienced by a population undergoing cryptic evolution. However, Fisher (1958) emphasized that evolutionary change in itself is likely to be an important component of ‘environmental deterioration’, a point restated by Cooke et al. (1990) in the context of intra-specific competition. In this form, environmental deterioration arises because a winning lineage has to compete against more winners in successive generations as the population evolves. This ‘evolutionary environmental deterioration’ has different implications for the selection and evolution of traits influenced by resource competition than general environmental change. We reformulate Cooke’s model as a quantitative genetic model to show that it is identical in form to more recent developments proposed by quantitative geneticists. This provides a statistical framework for discriminating between the alternative hypotheses of environmental change and environmental deterioration caused by evolutionary change. We also demonstrate that in systems where no phenotypic change has occurred, there are many reasonable biological process that will generate patterns in predicted breeding values which are consistent with what has been interpreted as cryptic evolution, and care needs to be taken when interpreting these patterns. These processes include mutation, sib-competition and invisible fractions.
The absence of micro-evolutionary change where it is expected is a long-standing problem in evolutionary biology (Merila et al., 2001b). The breeders’ equation is the simplest model of evolutionary change for continuous characters, and predicts evolutionary change when a heritable trait is under directional selection (Falconer, 1983). Given that most traits are heritable (Lynch and Walsh, 1998) and estimates indicate that selection is often directional (Kingsolver et al., 2001; Hereford et al., 2004; Perez and Munch, 2010), we should expect evolutionary change to be observed much more often if the breeders’ equation adequately captures the evolutionary process and estimates of selection and heritability are correct (Kinnison and Hendry, 2001). Proposed solutions to this problem have usually invoked some correlated aspect of an individual’s phenotype (Lande, 1979; Price et al., 1988; Blows and Higgie, 2003; Blows et al., 2004), a relative’s phenotype (Kirkpatrick and Lande, 1989) or the environment (Mitchell-Olds and Shaw, 1987; Rausher, 1992). Once these correlated ‘traits’ are taken into account the predicted genetic covariance between the trait and fitness may be reduced. Under a more general quantitative genetic model of evolutionary change (Robertson, 1966) this genetic covariance is equal to the evolutionary response to selection. However, in long-term studies of wild systems it has been hard to identify correlated traits that can adequately explain the lack of evolutionary change often observed (Merila et al., 2001b).

A related hypothesis was proposed by Cooke et al. (1990), who suggested that evolutionary change may be occurring, but that its phenotypic consequences could be masked by concurrent environmental change. In their original model Cooke et al. (1990) suggested that a major component of this environmental change could be caused by the evolution of competitive ability in the population itself, resulting in ‘red queen’ dynamics (Van Valen, 1973). Although Cooke et al. (1990) suggested their results were an extension to Fisher’s (1958) fundamental theorem, Frank and Slatkin (1992) convincingly argued that this process was subsumed within Fisher’s concept of ‘environmental deterioration’, which had been widely misinterpreted. In this context, ‘environmental deterioration’ arises because as the population evolves, a winning lineage has to compete against more winners in successive generations resulting in a drop in relative fitness, a process that we call ‘evolutionary environmental deterioration’. However, Cooke et al. (1990) noted that they were not able to distinguish between evolutionary environmental deterioration and other forms of environmental change in their long-term data set on lesser snow geese (Anser caerulescens), because population density had also increased over the study period. Subsequently, Merila et al. (2001a) using a quantitative genetic approach on a population of collared flycatchers (Ficedula albicolis), demonstrated that although the genetic component of ‘condition’ had increased over the study period the environmental component had decreased. Similar findings have been shown in other long-term vertebrate studies (Garant et al., 2004; Wilson et al., 2007), and have been called ‘cryptic evolution’. Although these studies often cite, and even explicitly test
Cooke et al.’s (1990) original model, they have invoked either changes in population density and/or changes in climate as the source of environmental change, without considering the process that both Cooke et al. (1990) and Fisher (1958) emphasized as important: environmental deterioration as a result of evolutionary change. In addition, all of these studies have been on body-size related traits which appear to be under consistent positive directional selection (Kingsolver and Pfennig, 2004; Perez and Munch, 2010). Whereas general environmental change would only reduce body size under certain scenarios, environmental change caused by the evolution of competitive ability would always act to reduce body-size and may therefore likely to be a more general explanation for why the body-size of most species seems to be sub-optimal despite being heritable (Blanckenhorn, 2000).

In this paper, we cast Cooke et al.’s (1990) model as a quantitative genetic model and clarify its meaning in terms of the multivariate breeders’ equation. Using recent advances in statistical theory (Gianola and Sorensen, 2004) we then show how evolutionary environmental deterioration can be distinguished from general environmental change in real data. We also show that in its most basic form Cooke et al.’s (1990) model is virtually identical to indirect genetic models derived in plant breeding Griffing (1967) and more recently applied in animal breeding (Bjima et al., 2007a). However, since basic animal models have been used to test for cryptic evolution (Merila et al., 2001a; Garant et al., 2004; Wilson et al., 2007) we determine the conditions under which predicted breeding values from a basic animal model increase over time, but environmental deviations decrease over time - a pattern that has generally be considered as conclusive evidence of cryptic evolution (Postma, 2006). We show that although these patterns are consistent with environmental deterioration, there are alternative biological processes that can give rise to such trends.

Results

Evolutionary Environmental Deterioration and Quantitative Genetics

Without loss of generality we use a simplified version of Cooke et al.’s (1990) model where the phenotype \( (v_1) \) follows the model:

\[
v_1 = R\epsilon_1 \alpha_1
\]  

(1)

where \( \epsilon_1 \) and \( \alpha_1 \) are, respectively, an environmental effect and a genetic effect that determine the conversion of resource \( R \) into \( v_1 \). In Cooke et al.’s (1990) original model a second environmental deviation is included in the product, but is redundant for our purposes.
The amount of resource available to an individual is determined by

$$R = \frac{A}{N} \bar{\nu}_2$$

(2)

Here the fraction $\frac{A}{N}$ is the amount of the total resource ($A$) available to an individual when the resource is shared evenly over all individuals ($N$ is the population size). The second fraction $\frac{\bar{\nu}_2}{\nu_2}$ determines the ability of an individual to obtain more or less than its fair share, where the trait $\nu_2$ can be viewed as competitive ability and the overbar denotes the expectation. When there is no variation in competitive ability then this fraction is unity and the resource is evenly split.

As with the first trait, the competitive ability is assumed to follow a multiplicative quantitative genetic model:

$$\nu_2 = \epsilon_2 \alpha_2$$

(3)

The model can be transformed into the more standard additive quantitative genetic model by working on the log scale ($y_1 = \log(\nu_1)$):

$$y_2 = \mu_2 + a_2 + e_2$$

$$y_1 \approx \mu_1 + a_1 + e_1 + \log(A) - \log(N) + \mu_2 + a_2 + e_2 - \left[ \bar{y}_2 + \frac{\sigma^2_2}{2} \right]$$

(4)

where $\mu_1$ and $\mu_2$ are trait specific intercepts. The approximation is due to $\log(\bar{\nu}_2) = \log(e^{\bar{y}_2}) \approx \bar{y}_2 + \frac{\sigma^2_2}{2}$.

We can get an expression for the total change in the mean of the two traits by combining the genetic response to selection based on the multivariate breeders’ equation (Lande, 1979) with the expression for environmental change (see Iwasa and Pomiankowski (1991) also):

$$\begin{bmatrix} \Delta \bar{y}_1 \\ \Delta \bar{y}_2 \end{bmatrix} = \begin{bmatrix} \sigma^2_{a_1} + \sigma^2_{a_2} + 2\sigma_{a_1,a_2} & \sigma^2_{a_2} + \sigma_{a_1,a_2} \\ \sigma^2_{a_2} + \sigma_{a_1,a_2} & \sigma^2_{a_2} \end{bmatrix} \begin{bmatrix} 0 \\ \beta_{y_2} \end{bmatrix} + \begin{bmatrix} \Delta \log(A) - \Delta \log(N) - \Delta \left[ \bar{y}_2 + \frac{\sigma^2_2}{2} \right] \\ 0 \end{bmatrix}$$

(5)

The first term on the RHS is the familiar multivariate breeders’ equation (Lande, 1979) where the matrix is the genetic covariance matrix for the two traits, and the vector is a vector of directional selection gradients.

We assume that although there is direct selection to acquire more resources ($\beta_{y_2} > 0$) the allocation of those resources is already optimized and not under directional selection ($\beta_{y_1} = 0$). The second term on the RHS
captures ‘environmental’ change, although it should be understood that environmental change in $y_1$ includes change induced by change in the mean value of $y_2$, which itself may be due to a genetic response to selection.

If we assume that the population size is large enough that the covariance between population level properties of $y_2$ (e.g. $e^{y_2}$) and an individual’s breeding value or environmental deviation are negligible, and that change in the higher order moments of $y_2$ are also small (i.e. $\Delta \sigma_{y_2}^2 \approx 0$), Equation 5 can be simplified:

$$
\begin{bmatrix}
\Delta \bar{y}_1 \\
\Delta \bar{y}_2
\end{bmatrix}
\approx
\begin{bmatrix}
\beta_{y_2}(\sigma_{a_2}^2 + \sigma_{a_1,a_2}) + \Delta \log(A) - \Delta \log(N) - \Delta \bar{y}_2 - \Delta \sigma_{y_2}^2 \\
\beta_{y_2}\sigma_{a_2}^2 \\
\beta_{y_2}(\sigma_{a_2}^2 + \sigma_{a_1,a_2}) + \Delta \log(A) - \Delta \log(N) - \beta_{y_2}\sigma_{a_2}^2 \\
\beta_{y_2}\sigma_{a_2}^2
\end{bmatrix}
$$

Equation 6

The term $\beta_{y_2}(\sigma_{a_2}^2 + \sigma_{a_1,a_2})$ can be interpreted as ROBERTSON’s (1966) genetic covariance between a trait and fitness. To see how this interpretation can be given, decompose fitness ($W$) into a component predicted by $y_2$ and a residual $e_W$:

$$
W = \beta_{y_2}(\mu_2 + a_2 + e_2) + e_W
$$

Equation 7

The covariance between $y_1$ and fitness within a generation can then be decomposed into a genetic and environmental covariance assuming residuals are uncorrelated with breeding values:

$$
\text{COV}(y_1, W) = \text{COV}(a_2 + e_2 + a_1 + e_1, \beta_{y_2}(\mu_2 + a_2 + e_2) + e_W)
= \text{COV}(a_2 + a_1, \beta_{y_2}a_2) + \text{COV}(e_2 + e_1, \beta_{y_2}e_2)
= \beta_{y_2}(\sigma_{a_2}^2 + \sigma_{a_1,a_2}) + \beta_{y_2}(\sigma_{e_2}^2 + \sigma_{e_1,e_2})
$$

Equation 8

Designating ROBERTSON’s (1966) genetic covariance as $S_{G}$ we have:

$$
\Delta \bar{y}_1 = S_{G} + \Delta \log(A) - \Delta \log(N) - \beta_{y_2}\sigma_{a_2}^2
$$

Equation 9

where $S_{G}$ is equal to evolutionary change in the absence of environmental deterioration (ROBERTSON, 1966), and $\Delta \log(A)$ and $\Delta \log(N)$ are changes in environmental quality and population density respectively. COOKE et al. (1990) put special emphasis on $-\beta_{y_2}\sigma_{a_2}^2$ as a source of environmental deterioration, and FRANK and SLATKIN (1992) showed that this quantity is closely linked to FISHER’s (1958) concept of environmental
deterioration (see also Price, 1972b; Ewens, 1989). However, Cooke et al. (1990) indicated that \( \Delta \log(N) \) was also positive during the course of their study and so the effect of changes in population size could not be distinguished from environmental deterioration caused by the evolution of competitive ability. More recent studies have considered only the role of environmental change in terms of changes in population density \( (N) \) (Larsson et al., 1998; Wilson et al., 2007) or changes in environmental quality \( (A) \) (Merila et al., 2001a) or both (Garant et al., 2004), without considering the final term in Equation 9 - evolutionary environmental deterioration.

\[ A \text{ test for evolutionary environmental deterioration - a recursive quantitative genetic model} \]

The ability to acquire resources \( (y_2) \) or the total amount of resources available \( (A) \) are usually not measured, so we ask whether it is possible to distinguish between environmental change and evolutionary environmental deterioration given information on \( y_1 \) only. To illustrate the point we assume discrete annual generations in which all individuals interact equally, but emphasize that the model generalizes to more complex scenarios.

The model presented above can be expressed as a recursive quantitative genetic model (Gianola and Sorensen, 2004) since one of the response variables \( (y_2) \) directly affects the other \( (y_1) \). Following Gianola and Sorensen (2004) we can rearrange Equation 4 such that the traits appear on the LHS and the unknown parameters appear on the RHS:

\[
y_1,i - y_2,i + \frac{1}{N_{k_i}} \sum_{j \in \mathcal{I}_i} y_{2,j} \approx \mu_1 + a_{1,i} + e_{1,i} + b_{k_i} \\
y_2,i = \mu_2 + a_{2,i} + e_{2,i} \tag{10}
\]

where \( b \) are year effects that include the effects of between-year variation in \( A \) on the expression of \( y_1 \). \( k_i \) indexes the year in which individual \( i \) was present and \( \mathcal{I}_i \) the set of all individuals present in that year (including \( i \) itself). The LHS of equation 10 for all individuals, nesting individuals within traits, can be represented in a more manageable matrix form, where \( X \) is an incidence matrix relating individuals to years, and \( D \) is a diagonal matrix with reciprocal annual population sizes along the diagonal:
where \( m \) is the total number of individuals across all years. The matrix on the RHS of Equation 11 is the structural coefficient matrix \((\Lambda)\) of Gianola and Sorensen (2004), and it can be shown (Appendix 1) that the marginal distribution of \( y_1 \) (after marginalizing \( y_2 \)) is given by:

\[
y_1 \sim N (X\beta, \sigma^2 + 2\sigma_{e_1,e_2} + \sigma^2_{a_1} + \sigma^2_{a_2} + 2\sigma_{a_1,a_2} + \sigma^2_{a_2}) (BA + \sigma^2_{a_2} BAB)
\]

When there is no variance in the ability to acquire resources \((\sigma^2_{e_2} = \sigma^2_{a_2} = 0)\) the model coincides with the standard animal model which has been used to test for cryptic evolution (see below). The marginal distribution of the full model, however, has a similar form to that under Willham’s (1972) maternal effect model (see p573 Sorensen and Gianola, 2002), as was noted by Bijma et al. (2007a) for their model of indirect genetic effects. In fact this model coincides with Bijma et al.’s (2007a; 2007b) model under the null hypothesis proposed by Hadfield and Wilson (2007): that the more individuals you interact with the less effect you can have on any one individual, hence the matrix of reciprocal population sizes \(D\). The only slight difference is that an individual also affects itself (by using up its own resources) whereas in Bijma et al.’s (2007a; 2007b) model the indirect effect (called associative effect following Griffing (1967)) caused by expression of \( y_2 \) is only felt by other individuals.

Each element of the matrix \( \sigma^2_{a_2} BAB \) has the form \( \sigma^2_{a_2} E[r_{I,J}] \) where the expectation is the mean relat-
edness between individuals in \( i \)'s year and \( j \)'s year. This implies that two individuals should be more similar if their respective competitors are more closely related and therefore similar in competitive ability.

The term \(- (\sigma_{a_1,a_2} + \sigma_{a_2}^2)(BA + AB)\) is harder to understand and implies that the expected covariance between individual \( i \) and individual \( j \)'s phenotype is proportional to the mean relatedness of \( i \) to the individuals alive in \( j \)'s year + the mean relatedness of \( j \) to the individuals alive in \( i \)'s year \((E[r_{i,j}] + E[r_{j,i}])\). If the genetic covariance between competitive ability and the allocation of resources to the focal trait \((y_1)\) is zero then this implies that two individuals should be less similar when they interact with each other’s relatives.

To see how this arises, imagine the extreme situation where individual \( i \) only interacts with \( j \)'s relatives and individual \( j \) only interacts with \( i \)'s relatives. If individual \( i \) is a better competitor than the relatives of \( j \) then it will have a large phenotype. If competitive ability is heritable then on average relatives of \( i \) are likely to out compete individual \( j \) and cause its phenotype to be small.

A similar phenomenon arises at the environmental level when the environmental covariance between competitive ability and the allocation of resources to the focal trait is zero. The term \(- (2\sigma_{e_1,e_2} + \sigma_{e_2}^2)B\) then implies that individuals who interact should resemble each other less because if individual \( i \) takes more resource this leaves less for \( j \). This effect decreases as populations become large because the effect of individual \( i \) on \( j \)'s phenotypes will become diluted. More flexible relationships between population density and the strength of competition could be entertained (Hadfield and Wilson, 2007; Bijma, 2010c).

Based on these results we suggest that a test for evolutionary environmental deterioration should first involve estimating the indirect genetic (co)variances determined above (i.e. \( \sigma_{a_2}^2 \) and \( \sigma_{a_1,a_2} \)). If these are found to be different from zero, then showing that the breeding value for competitive ability has increased would be consistent with evolutionary environmental deterioration.

**The conditions for the pattern of cryptic evolution**

Above we have argued that general environmental change, and evolutionary environmental deterioration - as envisaged by Cooke et al. (1990) (Frank and Slatkin, 1992) - are separate and distinct processes. Evolutionary environmental deterioration depends on the presence of indirect effects, both genetic (Wolf et al., 1998) and environmental, arising from competition. To date, these indirect effects have not been included in models used to test for cryptic evolution. Rather models of the form
\[
y_1 \sim N \left( X \beta, \sigma^2_I + \sigma^2_A \right)
\]

have generally been used, and individual predictions of breeding values and environmental deviations obtained using best linear unbiased prediction (BLUP). When the BLUP breeding values and BLUP environmental deviations from these models change in different directions over time it is generally assumed that this is good evidence of cryptic evolution (Postma, 2006). If the model defined by Equation 13 is the correct model, then the trends in BLUP breeding values and BLUP environmental deviations are unbiased estimators of the trends in actual breeding values and actual environmental deviations, although care has to be taken with hypothesis testing (Hadfield et al., 2010). However, if the model defined by Equation 13 is not the correct model then predicted and actual trends may not coincide. In this section we determine the conditions under which fitting the basic animal model (Equation 13) would result in predicted trends that are consistent with what has been interpreted as cryptic evolution. We use these results to show that evolutionary environmental change does give rise to predicted trends that are consistent with the process, despite the model being wrong (i.e. Equation 13 is different from Equation 12). However, we also show that there are other biological processes not explicitly modeled that would also result in similar patterns.

In order to understand the statistical properties of estimators of evolutionary genetic change it is necessary to explicitly consider the process of selection, and Gianola et al. (1988) pointed out that to understand selection adequately the pedigree must be treated as a random variable rather than a fixed quantity. To be able to do this we consider the joint distribution of pedigrees and phenotypes when pedigrees consist of parents and their full-sib families, and let the number of sibs in each family depend on the phenotypes of the parents. We then ask what properties of this joint distribution would give rise to predictions that have been treated as evidence of cryptic evolution.

Under the assumption that the mean phenotype has not changed between the parental and offspring generation, we find that a positive trend in predicted breeding values and a negative trend in predicted environmental effects will result when (see Appendix for further details):

\[
\text{COV} (\Delta z, f_1(n)) + E[f_1(n)] E[\Delta z] < \text{COV} (\bar{y}, f_2(n))
\]

where \(\Delta z\) is the deviation, mid-offspring phenotype minus mid-parent phenotype (\(\bar{y}\)). Expectations are taken over families (as in Price’s (1972a) Equation), each of which has two parents but a variable number of \(n\) offspring. The functions \(f_1(n)\) and \(f_2(n)\) are monotonic positive functions of parental fitness:
\[ f_1(n) = \frac{n(1+r+rn)}{(1+r+rn)+2r(1+r)} \]
\[ f_2(n) = \frac{n(1+3r+rn)}{(1+r+rn)+2r(1+r)} \]  

(15)

where \( r \) is the ratio \( \frac{\sigma^2_e}{\sigma^2_a} \). We had little success in simplifying the analysis or extending it to more general scenarios, but some insights can be gained from inequality 14 which we illustrate and validate with simulations of more complex pedigrees.

Because the functions are monotonic functions of parental fitness the sign of the covariances will be the same as if parental fitness was used directly. If we assume directional selection on the trait is either absent or positive (i.e. the RHS is equal to or greater than zero) then any process that either a) reduces \( E[\Delta z] \) or b) reduces the covariance between \( \Delta z \) and parental fitness, could give rise to patterns in predicted breeding values that have been interpreted as evidence of cryptic evolution.

**Example 1: Sib-effects**

General changes within families (\( E[\Delta z] \)) may be the result of between generation changes in the environment such as environmental deterioration (Frank and Slatkin, 1992), but there are also other mechanisms. For example, under a neutral model most parents have more sibs than their offspring (see Appendix) and so any effect of family size on offspring phenotype can cause a general difference between parent and offspring phenotype. To illustrate, we made a simple simulation with discrete generations (30), a constant population size (100) and no selection on phenotype. Individuals formed monogamous pairs at random, and family sizes were generated from a Poisson distribution with a mean of \( \exp(2) = 7.4 \). The resulting offspring were sampled at random to form the following generation, and their phenotypes were simulated according to:

\[ y_i = a_i + \beta_s n_{p_i} + e_i \]  

(16)

where \( n_{p_i} \) is offspring \( i \)'s parental fitness (i.e. the number of sibs individual \( i \) has). The residual (\( e_i \)) was simulated from a standard normal distribution, and the breeding value (\( a_i \)) from a normal distribution with a mean equal to the average breeding value of \( i \)'s parents and a variance equal to half the additive genetic variance, which was also set to 1 (i.e. \( r = \frac{\sigma^2_e}{\sigma^2_a} = 1 \)). \( \beta_s \) is the effect that an increase in sibship size has on an individual's phenotype which we set to 0.5. A simple animal model was fitted using the resulting pedigree and phenotype data, and breeding values predicted. Across 100 replications the average change in
the true breeding values was small and non-significant ($0.017 \pm 0.060, p = 0.775$) but the change in predicted breeding value was large ($2.229 \pm 0.042, p < 0.001$). Figure 1 shows the actual trend in breeding values (and environmental deviations over time) in gray, and the predicted trends in black, for a typical simulated run. There are some traits for which positive values of $\beta_{sa}$ are likely, for example chicks from larger broods often beg more (e.g. Neuenschwander et al., 2003), but there are as yet no published studies of cryptic evolution for this type of trait. For traits that are negatively affected by sib-competition, such as body size, $\beta_{sa}$ is likely to be negative and the primary motivation for using a positive coefficient is to be consistent (i.e. to obtain positive rather than negative trends in predicted breeding values). Reversing the sign of the coefficient results in a decrease in predicted breeding values and an increase in environmental deviations which could potentially mask the signature of cryptic evolution.

![](image1.png)

Figure 1: Simulated breeding values (solid gray line) and environmental deviations (dashed gray line) when interacting with more sibs increases phenotypic value. Predicted breeding values (solid black line) and predicted environmental deviations (dashed black line) for the same data using the basic animal model.

**Example 2: The invisible fraction**

In addition to general changes within families ($E[\Delta z]$) as exemplified above, any process that reduces the covariance between $\Delta z$ and parental fitness more than that expected under a standard quantitative genetic model, could also give rise to patterns in predicted breeding values that have been interpreted as cryptic evolution. In the above example, this covariance will be positive and will in part counteract the effects of the negative $E[\Delta z]$. However, other mechanisms can generate negative covariances. For example, mutations affecting body size in *Caenorhabditis elegans* tend to reduce rather than increase size (Azevedo et al., 2002).
If these mutations act proportionally rather than additively then the same mutation affecting offspring of large worms will tend to reduce body size more than in offspring of small worms. If body size is under positive selection then these mutations would give rise to negative covariances between parental fitness and $\Delta z$.

It is also important to realize that parental fitnesses and parental/offspring phenotypes are not necessarily the real values, but the values that went into the analysis. If small parents tend not to be observed (for example sneaky males are not identified) then the offspring of single parents tend to be smaller than expected under the assumption of random mating, which is assumed in the basic model. Likewise, Mojica and Kelly (2010) show that although large flowers confer a fecundity advantage in Mimulus guttatus, larger-flowered genotypes have greater mortality prior to trait expression and so their flower size goes unmeasured. Under this scenario, correlative studies will tend to measure a smaller sized subset of offspring from large parents resulting in $\Delta z$ values which negatively covary with measured parental fitness (fecundity). The sub-set of unmeasured dead individuals has been termed the invisible fraction (Grafen, 1988) and is known to cause problems for the estimation of quantitative genetic parameters (Im et al., 1989). Here we show that this form of missing data is sufficient to generate trends in predicted breeding values that resemble patterns that would be obtained from cryptic evolution.

Again, we made a simple simulation with discrete generations (30) and a constant population size (100) in which individuals with a propensity to produce large flowers ($y$) have a reduced chance of surviving to maturity (and therefore being measured) but conditional on flowering have higher fecundity. Family sizes were generated from a Poisson distribution:

$$n_i \sim \text{Pois}(\exp(2 + \beta_f y_i + y_j + y_i))$$

where individual $j$ is $i$’s mate, and $\beta_f$ is the fecundity selection gradient which was set to 0.2. The phenotypes of individuals were generated according to the basic quantitative genetic model:

$$y_i = a_i + e_i$$

However, the probability of an individual surviving to adulthood (and therefore being measured) was proportional to the density of $y$ in a normal distribution with mean equal to $\bar{y} + S$ and a variance of $\gamma$. $S$ is the deviation from the optima of the population mean in that year ($\bar{y}$) and was set to -0.2, and $\gamma$ is equivalent
to the strength of stabilizing selection around the optima which was set to 1.75. These parameters result in a selection regime where fecundity selection and viability selection are approximately equal.

Again, a simple animal model was fitted using the resulting pedigree and phenotype data, and breeding values predicted. Across 100 replications the average change in the true breeding values was small and non-significant ($-0.006 \pm 0.046, p = 0.893$) but the change in predicted breeding value was large ($0.953 \pm 0.018, p < 0.001$). Figure 2 shows the actual trend in breeding values (and environmental deviations over time) in gray, and the predicted trends in black, for a typical simulated run.

![Figure 2: Simulated breeding values (solid gray line) and environmental deviations (dashed gray line) when survival and fecundity selection balance. Predicted breeding values (solid black line) and predicted environmental deviations (dashed black line) for the same data using the basic animal model. In both cases, individuals that failed to survive were not included in the analysis.](image)

**Discussion**

In this paper we clarify the original meaning of environmental deterioration (Fisher, 1958; Cooke et al., 1990; Frank and Slatkin, 1992) by placing it in the context of quantitative genetics. By doing this, we show that previous quantitative genetic studies in wild systems (Merila et al., 2001a; Garant et al., 2004; Wilson et al., 2007) had ignored the process of evolutionary environmental deterioration that Fisher (1958) and particularly Cooke et al. (1990) had emphasized as important (Frank and Slatkin, 1992).

Evolutionary environmental deterioration in the sense used by Cooke et al. (1990) arises because as genotypes that confer greater competitive ability spread, the amount of resources available to other geno-
types diminishes. When the amount of resource is fixed this leads to a zero-sum game whereby the mean resource acquired remains constant, and the mean trait value does not change despite underlying evolutionary change (Dickerson, 1955; Griffing, 1967; Wolf, 2003). Moreover, the share of the resource acquired by the superior genotypes diminishes as they spread because they end up competing with themselves, making it more difficult to observe the phenotypic effects of superior genotypes. Because it is this information that is used to detect evolutionary environmental deterioration in the statistical procedure outlined in this paper, we acknowledge that power may be low (Bijma, 2010a). Greater success may be had with lab or field based time-shift experiments that are able to measure individuals in environments characteristic of earlier or later generations, as is done in studies of host-parasite coevolution (Gaba and Ebert, 2009).

In a recent paper, Hadfield et al. (2010) showed that current methods for detecting cryptic evolution are highly anti-conservative and that the evidence for cryptic evolution in two of the published examples (Garant et al., 2004; Wilson et al., 2007) was weak. However, it should be understood that the estimates of evolutionary change are conditional on the model used, and that by obtaining the marginal distribution of the data under Cooke’s model, we show that models previously used to demonstrate cryptic evolution are the wrong models if evolutionary environmental change is the main cause of any environmental deterioration. However, it would be possible to obtain estimates of evolutionary change under a model of evolutionary environmental deterioration, and test them appropriately. In this manuscript we took, as Cooke et al. (1990) did, a simplified pedagogical model where all individuals in a year interact to the same degree. We acknowledge that this is ecologically naïve, and that to obtain more accurate and more powerful estimates of this evolutionary process it is undoubtedly necessary to work with systems where groups of interacting individuals can be defined, or the level of interaction between different individuals quantified. In many systems, interacting individuals will be relatives and we stress that in obtaining Equation 6 the simplifying assumption that the covariance between an individual’s breeding value and the mean breeding value of the group is zero precludes a response to kin selection that can and should be included (Bijma, 2010b; McGlothlin et al., 2010). In addition, Cooke et al.’s (1990) model assumes that competitive abilities are transitive in the sense that if individual A outcompetes B, and individual B outcompetes C, then individual A must outcompete individual C (Harris et al., 2008). Such a model predicts that individuals in later generations would on average outcompete individuals in previous generations, which may hold over short time-scales but is unlikely to be a general property of competitive interactions, as evidenced in yeast (Paquin and Adams, 1983). Furthermore, it is important to recognize that even if patterns consistent with cryptic evolution are found, there are other biological processes that may be responsible for them. For example, because groups that share more relatives are likely to be close in time any temporal autocorrelation in environmental effects
may be wrongly interpreted as indirect genetic effects. Here, using a simplified full-sib model we have derived conditions under which a conclusion of cryptic evolution might be drawn from the basic animal model and find that sibling competition, selection bias and mutation could all cause patterns that are equally consistent with this interpretation.

However, as noted above, our formulation of Cooke et al.’s (1990) model is virtually identical to indirect genetic effect models (Griffing, 1967) recently applied to livestock data (Bijma et al., 2007a). These models have been used to demonstrate the influence of indirect genetic effects in poultry and pigs, where groups of interacting individuals can be readily defined as those animals sharing a cage or pen (e.g., Bijma et al., 2007b; Bergsma et al., 2008). Bijma et al. (2007b) have argued that negative covariance between direct and indirect genetic effects can be interpreted as arising from heritable variation in competitive ability and should constrain phenotypic responses to selection among individuals (Griffing, 1967). Selection experiments that have explicitly considered indirect genetic effects have yielded results supporting this prediction (e.g., Goodnight, 1985; Muir, 2005). This source of constraint, and the phenomenon of evolutionary environmental deterioration discussed here are one and the same. Given the potential importance of competition and resource limitation in natural populations we believe the conditions that would give rise to evolutionary environmental deterioration are widespread. However, it is currently difficult to assess the potential magnitude of such effects without a better idea of the amount of additive genetic variance in competitive ability that segregates under natural conditions in wild populations. Thus while we certainly expect unequivocal demonstration of cryptic evolution by evolutionary environmental deterioration to be difficult, the importance of resource dependent trait expression for many aspects of an organism’s phenotype makes this an interesting, if challenging, topic for further study in wild systems.

We thank Michael Morrissey, Josephine Pemberton, Ben Sheldon and Craig Walling for useful discussions regarding this work and Andy Gardner for saving us from one mistake. JDH was funded by NERC and a Leverhulme trust award to LEBK. AJW was funded by BBSRC and LEBK by the Royal Society.
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