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Commentary

On the heritability of developmental stability

J. P. Swaddle

Applied Ornithology Unit, Division of Environmental and Evolutionary Biology, Graham Kerr Building, Institute of Biomedical and Life Sciences, University of Glasgow, Glasgow G12 8QQ, UK, e-mail: gbza86@udcf.gla.ac.uk

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Introduction

Møller and Thornhill (1997) assessed the heritability of developmental stability by a meta-analysis of all published (and some unpublished) heritability estimates of fluctuating asymmetry in bilateral traits. From a limited number of these studies they also calculated the additive genetic coefficient of variation (CVA). The usefulness of CVA is that it may correct for covariance between phenotypic variation and trait size (Houle, 1992). In eight out of the thirty-two studies that they reported, there was a statistically significant estimate of heritability. Twenty-eight of these studies, representing fifteen species, were entered into a meta-analysis. From this analysis, Møller and Thornhill (1997) concluded that there was a significant additive genetic component to development stability, but this component was relatively small. They also found that the CVA is approximately eight times larger for developmental stability than for trait size. In this manuscript I wish to highlight some of the benefits and limitations of this analysis, its implications and to suggest alternative approaches to the study of heritability of developmental stability that may prove useful.

Benefits and limitations of Møller and Thornhill's meta-analysis

Meta-analysis can be a very powerful statistical tool that can collate results from studies of varying methodology (review in Arnqvist and Wooster, 1995). Statistically, meta-analysis can provide a much more powerful test of a hypothesis than the individual tests that contribute the data. Also, within the analysis, data can be weighted so that poorly conducted studies do not contribute as much as well-constructed investigations. Therefore, identification of the variables that distinguish studies of varying quality is a crucial step in this type of analysis procedure. Møller

and Thornhill (1997) identified four criteria that could potentially sort studies in terms of their quality. (i) Whether the investigators tested for the statistical properties of fluctuating asymmetry (refer to Palmer and Strobeck 1986; Swaddle et al., 1994). (ii) The type of study performed (parent-offspring regression and sib analysis versus selection experiment). (iii) Internal validity of individual studies. This was scored as high if maternal and common environmental effects were minimised by standardised rearing conditions, or if sample sizes were large (>50). (iv) External validity, which was scored as high if the study population appeared to be stable and had been selected at random. These elements were entered into the meta-analysis except for external validity, as most studies were scored as high.

A relevant questions is, do these criteria adequately separate higher-quality from lower-quality studies? A test for the statistical properties of fluctuating asymmetry seems appropriate, as directional asymmetry and antisymmetry may not reflect developmental stability. The type of study performed is also highly relevant, but in this case perhaps Møller and Thornhill should have discriminated between full-sib and half-sib analysis studies. In full-sib estimates of heritability, the additive genetic variance is confounded with genetic variance due to dominance and common environmental conditions between sibs. Even if environmental conditions during development are standardised, the additive genetic variance will not be accurate and vary from the true value by an unknown amount. Whereas, in studies of paternal (and maternal) half-sibs these problems are negated (Willham, 1972; Falconer, 1989; review in Arnold, 1994). Møller and Thornhill (1997) do not distinguish between these two categories of sib studies, although full-sib figures are used in most cases (e.g., Mi and Rashad, 1977; Bener, 1979; Corrucini and Potter, 1981) and a mean between full-and half-sib values in others (e.g., Townsend and Brown, 1980). This lack of discrimination makes their analysis less accurate, although it may not alter the overall affect that they report.

The second point I would like to make relates to the determination of intrinsic validity. Møller and Thornhill (1997) scored a study as having high internal validity when rearing conditions were standardised, or if sample sizes were large (>50). Both of these conditions are important in obtaining accurate estimates of heritability. When heritability is low, as in this case, the sample sizes have to be large (>50) in order to detect a statistically significant level of heritability (review in Arnold, 1994). Additionally, standardising the environmental conditions of parents and offspring minimises the chances that the two main assumptions of parent-offspring analyses are violated. First, that environmental conditions of parent and offspring are not correlated; and second, that additive genetic values are not correlated with environmental values (Falconer, 1989). In the case of developmental stability, if environmental conditions are not standardised, this latter assumption could often be violated as symmetric individuals are known to outperform asymmetric individuals (Møller and Swaddle, 1997), and so may outcompete asymmetric individuals for the best environmental conditions during development. Both the criteria of large sample size and standardised conditions should be fulfilled for a study to be rated as possessing high internal validity. This would affect the status of several studies in the meta-analysis (e.g., Leary et al., 1985; Leamy, 1986; Tuinstra et al., 1990;

Scheiner et al., 1991; Leary et al., 1992). Møller and Thornhill's analysis indicated that studies with high internal validity, according to their classification, showed a larger effect of heritability than studies of low internal validity, again suggesting that the effect is genuine. However, these laboratory estimates are likely to over-estimate heritability in the field, as environmental conditions alter developmental stability and influence genotypes differentially (review in Møller and Swaddle, 1997). Therefore, these types of studies may be of minimal ecological relevance for inferring the outcome of selection in the field. One way around this problem may be to regress the developmental stability of laboratory-reared offspring on their wild caught parents (cf. Coyne and Beecham, 1987; Prout and Barker, 1989). Additionally, the effects of relevant environmental factors on the heritability of developmental stability could be studied in controlled conditions (cf. Hedrick, 1994).

Implications

The additive genetic component of developmental stability appears to be low. This is in accord with Fisher's fundamental theorem, which suggests that characters closely associated with fitness should have low heritability (Fisher, 1930). Although, more recent theory has suggested that heritability does not have to be low in traits associated with fitness, even if the heritability of overall fitness is low (Charlesworth, 1987; Price and Schluter, 1991). Recent theory has also suggested that the genetic variability of fitness traits should be high (Charlesworth, 1987; Houle, 1992; Pomiankowski and Møller, 1995), which is in accord with Møller and Thornhill's findings. Møller and Thornhill explain the maintenance of a high genetic variability for developmental stability through a nexus of interacting factors. In addition to these, there may be an intrinsic developmental cost in producing a developmentally stable, symmetric phenotype. This cost may be manifest in terms of a localised, or left-right, signalling system which monitors and regulates morphogenesis (review in Møller and Swaddle, 1997). It is also likely that the cost of producing the most stable symmetric form will rise exponentially as the phenotype approaches symmetry, as morphogenesis is increasingly tightly controlled. This intrinsic developmental cost may help to maintain a certain degree of instability in developmental processes and hence increase genetic variability.

Suggestions for future studies

I have already indicated some problems with inferring laboratory measured heritability into selection in the field and suggested alternative and additional approaches. Here, I would like to suggest two further ways in which the study of heritability of developmental stability may be modified and improved. First, as Møller and Thornhill (1997) remark, heritability obtained from linear regression analyses may not be valid, as fluctuating asymmetry will often violate the assumptions of linear regression due to its distinctive half-normal distribution (see Palmer

and Strobeck, 1986; Swaddle et al., 1994). Some fluctuating asymmetry data can be successfully transformed to fit the regression assumptions by Box-Cox transformations (Palmer and Strobeck, 1986; Swaddle et al., 1994). The use of such transformations, in future studies, may belay these statistical problems and reveal more accurate measures of heritability.

Second, asymmetry values, often, do not correlate between traits on the same individual (e.g., Efimov et al., 1987; review in Møller and Swaddle, 1997). This implies that developmental stability can not be seen as a single trait, and the present assessment of heritability may not be appropriate. The heritability of developmental stability could be approached from a multivariate perspective (review in Arnold, 1994). Construction of additive genetic variance-covariance matrices, that include measures of developmental stability from several traits on the same individuals, may provide a closer estimate to the heritability of overall developmental stability than heritability in single traits.

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