A simple model of the relationship between asymmetry and developmental stability

D. HOULE

Department of Zoology, University of Toronto, Toronto, Ontario M5S 3G5, CANADA, and Department of Biological Science, Florida State University, Tallahassee, FL 32306–1100, USA

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Abstract

The relationship between developmental stability and morphological asymmetry is derived under the standard view that structures on each side of an individual develop independently and are normally distributed. I use developmental variance of sizes of parts, V_D, as the converse of developmental stability, and assume that V_D follows a gamma distribution. Repeatability of asymmetry, a measure of how informative asymmetry is about V_D, is quite insensitive to the variance in V_D, for example only reaching 20% when the coefficient of variation of V_D is 100%. The coefficient of variation of asymmetry, CV_{FA}, also increases very slowly with increasing population variation in V_D. CV_{FA} values from empirical data are sometimes over 100%, implying that developmental stability is sometimes more variable than any previously studied type of trait. This result suggests that alternatives to this model may be needed.

Introduction

There has been considerable speculation about the degree of variation among individuals in their developmental stability, their ability predictably to complete development to an optimum state (Palmer, 1996; Møller & Swaddle, 1997). The principal hurdle to empirical studies of developmental stability is that, in most cases, we do not know what the optimum state of a trait is, so we cannot say how much of the variation is due to variation in the optimum and how much to a failure to develop to that optimum. When the same structure develops on either side of a symmetrical body however, we can assume that the optimum state is often one of perfect symmetry. Where this symmetry of the optimum can be assumed, asymmetry is referred to as fluctuating asymmetry (FA) (Palmer & Strobeck, 1986; Palmer, 1994). A large body of work now assumes that FA is a good indicator of developmental stability (Møller & Swaddle, 1997).

Correspondence: David Houle, Department of Biological Science, Florida State University, Tallahassee, FL 32306–1100, USA.
Tel.: +1 850 645 0388; fax: +1 850 644 9829;
e-mail: dhoule@bio.fsu.edu

Surprisingly, given these attractions of FA as an indicator of developmental stability, the precise relationship between the two has never been addressed analytically. Previous studies have, with few exceptions, relied on the negative relationship between FA and developmental stability that exists by definition. There are several barriers to studying this relationship. First, developmental stability is currently a hypothetical entity that has no unique relationship with observable properties of organisms. For example, FA is known to be affected by the environment an individual develops in (Parsons, 1990) as well as by variation in the developmental stability of individuals. It is also not clear that there is any single property of an individual that can be labelled developmental stability, as each trait of an individual may to some degree have different stability properties from every other trait (Møller & Swaddle, 1997, pp. 53–55; Leung & Forbes, 1997). For example, traits that develop at different times, or in different parts of the body, may differ in their developmental stability. Taken together, these complications have so far precluded empirical characterization of the relationship between developmental stability and FA, despite a number of demonstrations that variation in developmental stability

of some characters does exist (Parsons, 1990; Whitlock & Fowler, 1997; Gangestad & Thornhill, 1999).

In the absence of detailed empirical data, we must depend on models to develop our intuition. The majority of workers have adopted the following standard model of fluctuating asymmetry (Palmer & Strobeck, 1986; Palmer, 1996). Each individual is characterized by its developmental variance, the converse of developmental stability. Paired structures are assumed to develop independently towards the same expected size but to show some normally distributed variation around that expectation. The amount of variation around the expected size is determined by the developmental variance. In this model, the difference between paired structures is therefore also normally distributed, with variance twice that of the variance of each paired structure.

Several recent explorations of this standard model suggest that the expected relationship between FA and developmental stability is very weak. FA essentially measures developmental variance, the variance in size of body parts when they develop in the same environment, which is the converse of developmental stability. Variances are more difficult to estimate well than means, so we should expect that more sampling effort would be needed to study FA than a typical trait. Unfortunately, FA must usually be estimated from a single pair of measurements, yielding a poor estimate of the variance and therefore of the proportion of variation in FA that could be due to real variation in developmental stability (Whitlock, 1996; Houle, 1997).

The likelihood that only a small amount of information about individual developmental stability is gained from a single measure of FA has led to two efforts to quantify how much of the variation in FA could be due to variation in developmental stability, both assuming the simple model of the development of paired structures outlined above. One such effort used the relationship between the mean and variance of FA (Whitlock, 1996), whereas the other exploited the kurtosis in FA expected to arise from variation in developmental stability (Gangestad & Thornhill, 1999).

Whitlock (1996) observed that there is a simple relationship between the mean FA and the variance in FA for individuals with the same developmental stability. Therefore, one can calculate by subtraction the proportion of the total variance in FA that can be due to differences in developmental stability. This quantity is familiar from quantitative genetics as the repeatability. Although this insight is correct, the formulas given by Whitlock (1996) were incorrect; corrected formulas have now been published (Van Dongen, 1998b; Whitlock, 1998). The repeatability provides an intuitive measure of the reliability of individual measurements. More importantly, the repeatability sets an upper limit to the heritability, the proportion of the variance that can be due to genetic causes. It also sets an upper limit on the phenotypic correlation between the asymmetries of different pairs of traits on the same individual. Whitlock showed that the maximum repeatability of FA is 0.64 and that the coefficient of variation of FA is sometimes so large that the repeatability would be expected to approach this maximum value (Whitlock, 1996). Paradoxically, because FA is such a poor measure of variance, even traits with low repeatability and small correlations of FA among traits may reflect a great deal of variation in developmental stability.

Using simulations, Gangestad & Thornhill (1999) derived an empirical relationship between repeatability and kurtosis in signed FA, the difference in size between paired structures. They come to conclusions similar to those of Whitlock, arguing that, despite the low repeatability of many estimates of FA, the heritability of developmental stability itself may be high.

In this paper, I extend Whitlock's (1996, 1998) work to consider the relationship between the distribution of developmental stabilities and the variance of FA. Whitlock's approach leads to an estimate of the proportion of the variation in FA that can be due to variation in developmental stabilities, but it does not consider variation in developmental stability explicitly. The results presented here go the next step and allow inferences about the amount of variation in developmental stability from the repeatability of FA, based on the standard model. Previous work that has explicitly included variation in developmental stability has considered mixtures of individuals with two or three different stabilities (Houle, 1997; Van Dongen, 1998b), rather than more realistic continuous distributions. Other work has relied on simulations (Leung & Forbes, 1997; Van Dongen, 1998b; Gangestad & Thornhill, 1999), which are difficult to generalize. The principal result of this model is that, in order for measures of FA to have the substantial repeatabilities implied by some data, mean-standardized variation in developmental stability would have to be higher than for most previously studied traits.

In the next section I present an intuitive introduction to the model. The *Mathematical results* section then derives the relationship between developmental stability and measures of asymmetry based on this model. From these relationships, I then obtain *Numerical results*. The reader who wishes to obtain the main results without mathematical details may skip the *Mathematical results* section.

The model

I assume a population of organisms that are unable to regulate development perfectly. This imperfect development is studied by measuring a pair of structures on either side of an axis of symmetry, such as right and left limb lengths. I start with the commonly accepted model for the development of bilaterally paired traits, which imagines that each side of the organism develops independently of the other and that the variation in each side is normally distributed (Palmer & Strobeck, 1986; Palmer,

1996). This model of asymmetry has a pragmatic basis and is not directed at attempting to discern the causes of variation in development. It merely assumes that developmental variance exists and that developmental variance captures something about what we intuitively mean when we discuss developmental stability. If we understood the details of development of the morphological structures, we could make the relationship between asymmetry and development explicit. Clearly, the present state of knowledge does not allow this step, although a number of speculative efforts in this direction have been undertaken (Graham *et al*., 1993; Klingenberg & Nijhout, 1999).

Variation in developmental stability has often been shown to be caused by environmental variation (Parsons, 1990) and in some cases to have a genetic basis as well (Parsons, 1990; Whitlock & Fowler, 1997; Gangestad & Thornhill, 1999). Developmental stability can in principle be decomposed into developmental noise, factors that cause variation in development, and developmental homoeostasis, processes that damp out the effects of developmental noise (Palmer, 1996; Leung & Forbes, 1997). In practice these are usually indistinguishable, so they are considered together here.

One must model at least four kinds of variances to investigate the relationship between variation in developmental stability and variation in asymmetry. The most familiar of these is the variance of asymmetry itself, which I symbolize σ^2. The variance of asymmetry depends on the variance in the traits from which asymmetries are calculated, that is, on the developmental variance, V_D. V_D is the converse of developmental stability. In addition, the variance of sides may contain measurement error, V_e. The fourth sort of variance is variation in the developmental variance, which has not been explicitly included in previous analyses.

I assume that each individual offers two or more examples or realizations of the same trait, S, which I will refer to as 'sides', although their spatial arrangement is not important. On the ith individual, the S-values are drawn independently from the same normal distribution with variance V_D. The mean of this distribution must be much greater than $\sqrt{(V_D)}$ in order to preserve approximate normality but is otherwise free to vary. For simplicity, I assume that the mean of the distribution of sides is uncorrelated with V_D. The developmental variance of the ith individual will be represented as V_{Di}. It may consist of variation caused by both genotype and environment. I consider two statistics to measure asymmetry. First is the absolute value of the difference between sides $FA_i = |S_{i1} - S_{i2}|$. Second is the variance of sides

$$s_i^2 = \frac{\sum_{j=1}^{n} \left(S_{ij} - \bar{S}_i\right)^2}{n-1}, \qquad (1)$$

where n is the number of 'sides' measured. The variance of sides has statistical properties superior to those of FA, even when there are only two sides (Palmer & Strobeck, 1986).

My goal is to model the variance in asymmetry as a function of population variation in developmental variance, V_{V_D}, so we need to consider what the distribution of V_D would look like. The family of distributions I have chosen to represent this situation is shown in Fig. 1. Before I give the mathematical basis for these distributions, I give the following intuitive justification.

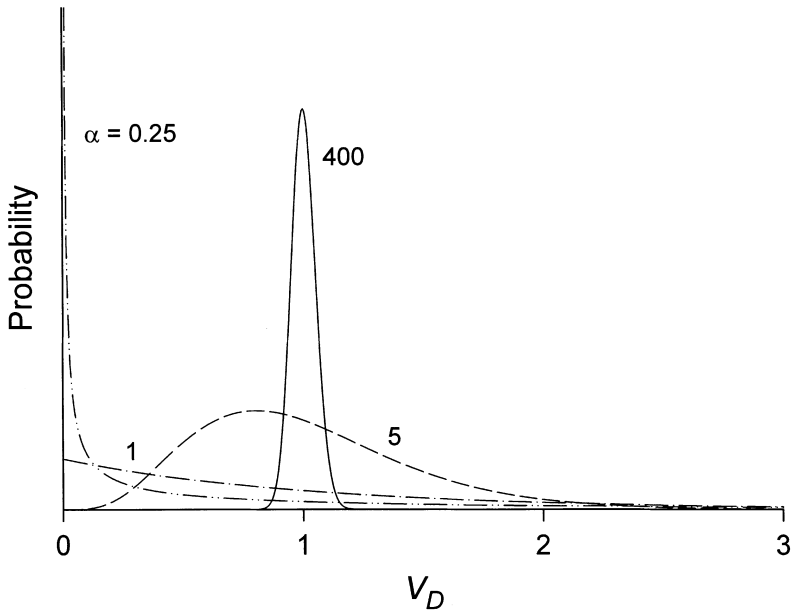

Fig. 1 Shape of the gamma distribution for different values of α, the shape parameter. All of these distributions have a mean of 1, which was accomplished by setting $\beta = 1/\alpha$, where β is the scale parameter.

The choice of a distribution for developmental variances, V_D, must take into account the fact that a variance cannot be negative. Consider a series of populations with the same mean developmental variance, $\bar{V}_D$, but that differ in the population variance of developmental variance, V_{V_D}. When V_{V_D} is small (as shown in the curve labelled $\alpha = 400$ in Fig. 1) the distribution can be nearly symmetrical, as it is very unlikely that a value will fall near $V_D = 0$. However, as the population variance of V_D increases, the fact that V_D cannot be negative has a larger and larger effect on the distribution. If the mean is to be held constant, the lack of negative values means that the likelihood that a value falls between zero and the mean must increase to compensate for the unconstrained tail of large values to the right of the mean. The result is that the distribution must become increasingly skewed as the variance goes up (represented by decreasing α values in Fig. 1), and the mode of the distribution must shift to the left. When V_{V_D} is very large relative to the mean, the mode is very close to 0 but balanced by an increasingly long tail of large V_D values.

A distribution that has these properties is the gamma distribution, which is only defined for values of $V_D > 0$. For the gamma, the probability that individual i has developmental variance V_D is

$$f(V_{D\cdot i}) = \frac{1}{\Gamma(\alpha)\beta^{\alpha}} V_{D\cdot i}^{\alpha-1} e^{-\frac{V_{D\cdot i}}{\beta}}, \qquad (2)$$

where $\Gamma()$ denotes the gamma function. The gamma distribution has two parameters, α and β. A principal attraction of the gamma distribution is the variety of shapes it can assume, depending on the value of the 'shape' parameter α. When α is large, the gamma approaches a normal distribution. Both the exponential and the χ^2 distributions are special cases of the gamma distribution. β is the 'scale' parameter. The mean of the gamma distribution is $\alpha\beta$, and the variance is $\alpha\beta^2 = V_{V_D}$. Note that the coefficient of variation of a gamma-distributed variable such as V_D is

$$CV_{V_D} = \frac{100}{\sqrt{\alpha}}. \qquad (3)$$

In addition to the developmental variance, V_D, I assume that the observed variance of S may also include measurement error, V_e. Measurement error is assumed to be constant over all individuals. To incorporate measurement error, the distribution $V_i = V_{D,i} + V_e$ can be modelled as a gamma distribution with a minimum value at V_e, rather than 0.

Most elements of this model are shown in Fig. 2, which shows the distribution of the developmental variance, V_D, sides S, and FA for two different distributions of V_D. The first row of the figure shows the distributions of V_D. On the left, $\alpha = 0.5$ resulting in a highly skewed distribution with a high CV; the column on the right shows $\alpha = 100$, a fairly symmetrical distribution with a low CV. Measurement error is assumed to be absent. The second row of the plots shows distributions of sides for representative values of V_D. The mean of the sides is always equal to 10, and the distributions are always normal. In each case, the upper right panel of each triplet gives the distribution for a value of V_D at the 95th percentile of the distribution, the lower left panel gives the distribution for a value of V_D at the 5th percentile of the distribution, and the middle panel shows the value at the median value of V_D. For the small α value, the difference in the distributions of S is immediately apparent; the distribution at the 5th percentile has such a small variance that the peak is off the scale chosen. Note that the 95th percentile for this distribution is at $V_D = 6.05$, emphasizing the presence of a long tail of large V_D values that is not otherwise apparent in the figure. For the large-α case, however, the distributions of S are so similar that no difference is apparent to the eye.

These distributions of S will not be observed directly, as each individual has only two sides. Instead, we directly observe the distribution of sides in a population of individuals, where each individual has a different developmental variance drawn from the distributions at the top of the figure. This distribution is shown in the third row of the figure. Note that the distribution with small α results in a kurtotic distribution of sides, whereas the large-α case has a nearly normal distribution of sides. This kurtosis is expected because the distribution is a combination of normal distributions with very different variances (Wright, 1968; Houle, 1997; Leung & Forbes, 1997; Gangestad & Thornhill, 1999). Finally, we calculate FA by taking the absolute value of the difference in two sides of the same individual, resulting in the peaky, long-tailed distribution of FA in the small-α case, and a distribution close to the half normal in the large-α case.

Given this model, we are interested in how informative these measures of asymmetry are concerning the developmental variance of an individual, when individuals vary in V_D. As pointed out by Whitlock (1996, 1998) a good measure for this purpose is the repeatability, symbolized $\Re$, the proportion of the variance in asymmetry that is due to real differences in developmental variance. The repeatability sets an upper limit both to the heritability of asymmetry and to the correlation of asymmetries of different structures on an individual. To calculate the repeatability of a measure of asymmetry, we need to know the total observed variance in asymmetry in the whole population, σ_T^2, and the realization variance, σ_R^2, the variance in observed asymmetry among individuals with the same V_D values. This σ_R^2 term includes any measurement error. The remaining variance, σ_I^2, is the true variance among individuals remaining after the realization variation is removed. By definition, $\sigma_T^2 = \sigma_R^2 + \sigma_I^2$.

Note that both Whitlock (1998) and Van Dongen (1998b) treat the parameter σ_I^2 as the variance in developmental stability. (Their notation differs from mine: σ_I^2 is V_{DS} in Whitlock; V_{ind} in Van Dongen.) I reserve the term developmental stability for the inverse of the

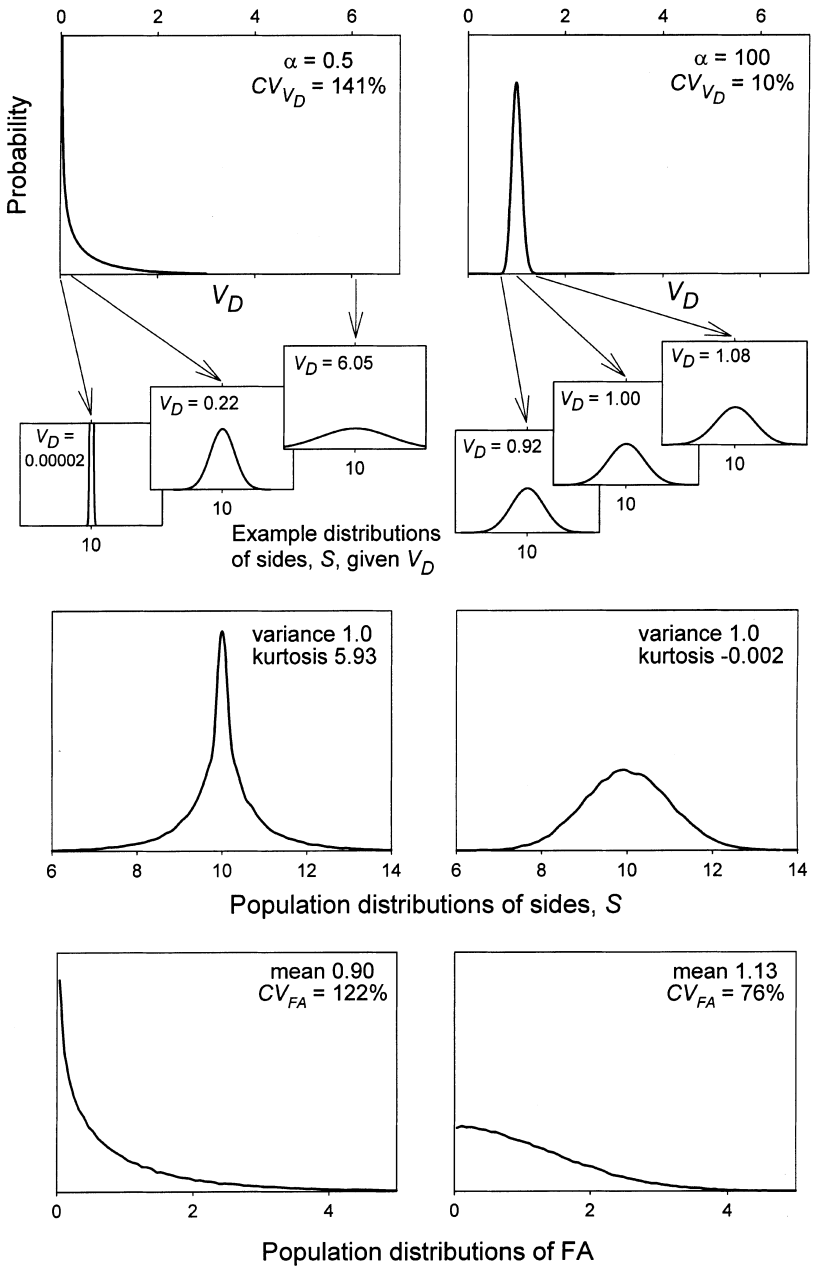

Fig. 2 Relationship between the distribution of the developmental variance, V_D, and the distribution of sides, S, and fluctuating asymmetry, FA. The top two rows give the theoretical distributions of V_D and S, while the last two rows are distributions of 100 000 simulated observations. The simulated distributions are splined to smooth the observed values. See text for additional explanation.

developmental variance of sides, V_D, and the term variance in developmental stability for the variance of developmental stability, $V_{V_D} = \alpha\beta^2$. Although in some cases (see below) $V_{V_D} = \sigma_I^2$, defining some aspect of the variance in symmetry as developmental stability risks losing track of the important distinction between developmental stability and the effects it has on a particular phenotype.

Mathematical results

The expected value of FA_i is

$$E(FA_i) = \overline{FA}_i = 2\sqrt{(V_{D\cdot i} + V_e)/\pi} \qquad (4)$$

and the variance is

$$Var(FA_i) = 2(V_{D\cdot i} + V_e)\frac{\pi - 2}{\pi}, \qquad (5)$$

where the expectations are over hypothetical replicate individuals with the same $V_{D,i}$ values. The realization variance is

$$\sigma^2_{R\cdot FA} = E\left[\frac{(\pi - 2)}{\pi}2(V_{D\cdot i} + V_e)\right] = \frac{2(\pi - 2)}{\pi}\left(V_e + \int_0^\infty V_{D\cdot i} f(V_{D\cdot i})\mathrm{d}V_{D\cdot i}\right)$$

$$= \frac{2(\pi - 2)}{\pi}(\alpha\beta + V_e). \quad (6)$$

The other variance components cannot be obtained in closed form when there is measurement error, so I first consider the case of no measurement error, $V_e = 0$. Then the mean FA is

$$\overline{FA} = E\left[2\sqrt{V_{D\cdot i}/\pi}\right] = \frac{2}{\sqrt{\pi}}\sqrt{\beta}G \quad (7)$$

where

$$G = \frac{\Gamma(\alpha + 1/2)}{\Gamma(\alpha)}. \quad (8)$$

The total variance in FA is

$$\begin{aligned}\sigma^2_{T\cdot FA} &= \int_0^\infty \left(Var(FA_{N\cdot i}) + (E[FA_i])^2\right) \\ &\quad \times f(V_{D\cdot i})\mathrm{d}V_{D\cdot i} - \overline{FA}^2 \\ &= 2\alpha\beta - \overline{FA}^2 \\ &= 2\beta\left(\alpha - \frac{2G^2}{\pi}\right). \end{aligned} \quad (9)$$

Finally, the true variance in FA among individuals can be obtained as the difference between eqns 9 and 6,

$$\sigma^2_{I\cdot FA} = \beta(\alpha - G^2)\frac{4}{\pi}. \quad (10)$$

The repeatability of FA when there is no measurement error is then

$$\Re_{FA} = \frac{\sigma^2_{I\cdot FA}}{\sigma^2_{T\cdot FA}} = \frac{\alpha/G^2 - 1}{\frac{\alpha\pi}{2G^2} - 1}. \quad (11)$$

The results in eqns 6–11 were checked by simulations in SAS (results not shown; SAS Institute, 1990). Although I was not able to obtain general analytical results for the gamma distribution with error variance, numerical results were obtained by numerical integration of eqns 6 and 9 in Maple V (Waterloo Maple, 1997), with $V_e + V_{D\cdot i}$ substituted for $V_{D\cdot i}$.

It is also useful to consider the coefficient of variation of FA itself, which is readily measured (e.g. Whitlock, 1996). For the case of no error variance,

$$CV_{FA} = 100\sqrt{\frac{\alpha\pi}{2G^2} - 1}. \quad (12)$$

Just as Whitlock showed that the maximum repeatability of *FA* measures is a function of CV_{FA}, eqn 12 can be solved iteratively to yield an estimate of α, and CV_{V_D} from CV_{FA}.

Because the alternative measure of asymmetry, s^2, is a variance, it follows a χ^2 distribution with $n - 1$ degrees of freedom, and therefore has expected value $V_{D\cdot i} + V_e$, and variance $2(V_{D\cdot i} + V_e)^2/(n - 1)$. The mean is therefore just $\bar{V}_D + V_e$, or $\alpha\beta + V_e$. The realization variance is

$$\sigma^2_{R\cdot S^2} = \frac{2}{n-1}\left(\alpha\beta^2(\alpha + 1) + 2\alpha\beta V_e + V_e^2\right), \quad (13)$$

and the total variance is

$$\sigma^2_{T\cdot S^2} = \frac{2}{n-1}\left(\alpha\beta^2(\alpha + 1 + (n-1)/2) + 2\alpha\beta V_e + V_e^2\right). \quad (14)$$

Taking the difference between eqns 13 and 14 gives the individual variance $\sigma^2_{I\cdot s^2} = \alpha\beta^2$. Thus, the true variance of sides among individuals is the variance in developmental variance. The repeatability is then readily calculated as $\Re_{s^2} = \sigma^2_{I\cdot s^2}/\sigma^2_{T\cdot s^2}$. These results were checked by simulations in SAS (results not shown; SAS Institute, 1990).

Numerical results

Figure 3 shows $\Re_{FA}$, the repeatability of FA as a function of CV_{V_D}, the coefficient of variation of developmental variance. In all cases, the mean developmental variance was held constant at 1. The small graphs along the top show the very wide range of distributions considered, from symmetrical distributions with small CVs on the left to highly skewed distributions with a very strong mode at 0 on the right. The chief result is that $\Re_{FA}$ increases rather slowly with CV_{V_D}. Only when CV_{V_D} is very large does the repeatability reach substantial values. The effect of measurement error, V_e, on repeatability is shown in the lower curves in Fig. 3. The range of measurement error considered is quite large; when $V_e = 1$ the variance due to measurement error is as large as the mean variance of sides. Increasing measurement error lowers the repeatability.

Figure 4 shows the coefficient of variation of fluctuating asymmetry, CV_{FA}, values calculated for the same parameter set used in Fig. 3. When α is large and the coefficient of variation of developmental variance, CV_{V_D}, is small, CV_{FA} is very close to the theoretical minimum of $\sqrt{[(\pi - 2)/2]} \approx 76\%$, which follows from eqns 4 and 5. Only when CV_{V_D} is very large does CV_{FA} increase substantially. Increasing measurement error lowers CV_{FA}, as it increases the mean fluctuating asymmetry, as well as the variance.

Figure 5 shows $\Re_{s^2}$, the repeatability of s^2, the alternative estimator of asymmetry, for the same parameter set used in Figs 3 and 4. The overall shape of these curves is similar to that for $\Re_{FA}$, in that the repeatabilities only become substantial when the coefficient of variation of developmental variance is quite large. $\Re_{s^2}$ is always lower than $\Re_{FA}$, although this difference becomes less marked when V_e is large. One advantage of s^2 as a measure of asymmetry is that it can incorporate information on more than two sides. Figure 6 shows how repeatability increases with n, the number of sides measured. This relationship emphasizes the usefulness of organisms with multiple realizations of traits per individual for the investigation of developmental stability (Leung *et al.*, 2000).

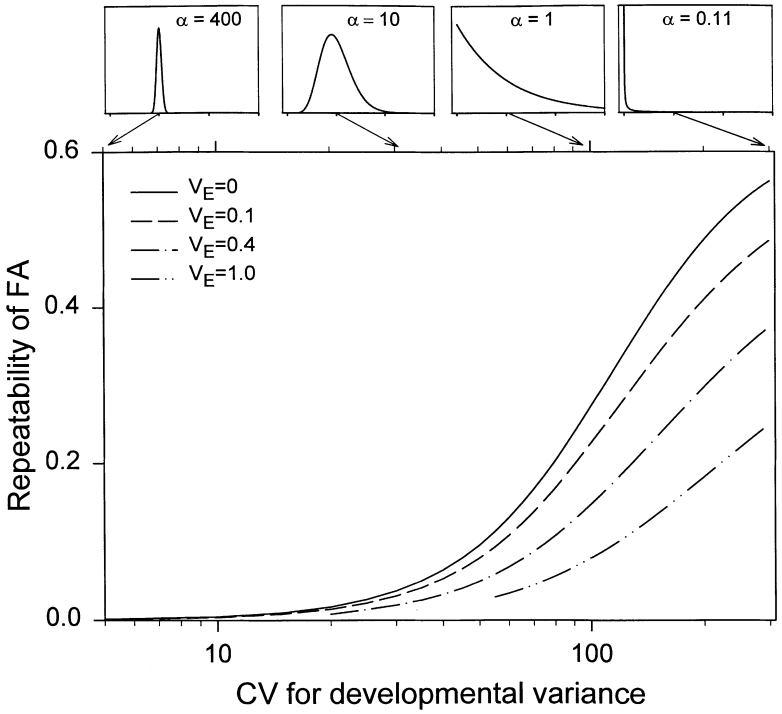

Fig. 3 Repeatability of FA measures as a function of the coefficient of variation of developmental variance, CV_{V_D}, and measurement error, V_e. The graphs along the top are probability density functions of developmental variance for representative distributions of CV_{V_D}, the shape of which is determined by α as $CV_{V_D} = 1/\sqrt{\alpha}$. Numerical integration could not be carried out for large values of α and V_e, so curves for these combinations are truncated in the figure.

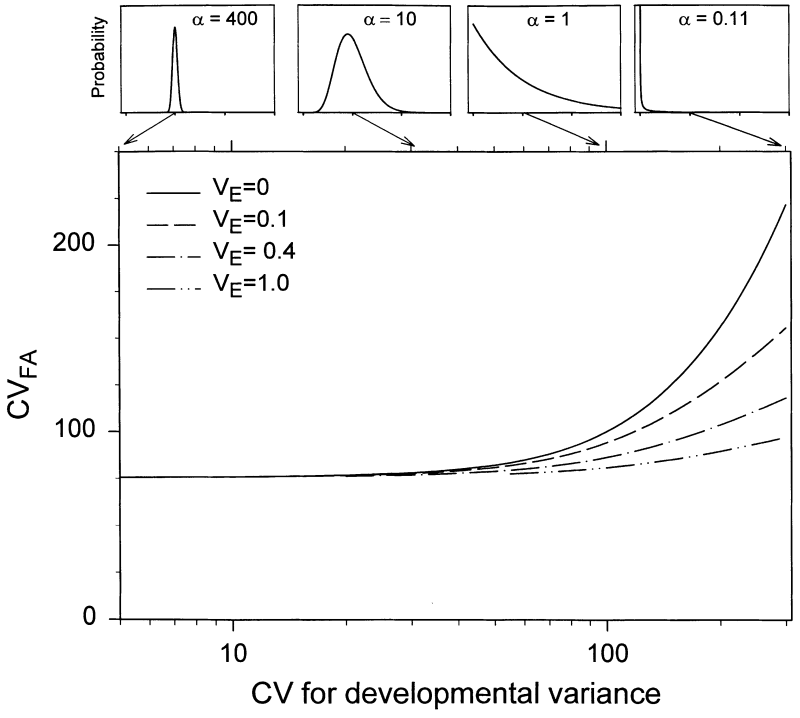

Fig. 4 The observed coefficient of variation of FA, CV_{FA}, as a function of the coefficient of variation of developmental variance and measurement error, V_e.

Discussion

In this paper I have extended a standard model of fluctuating asymmetry to explicitly include variance in the developmental stability parameter (here represented by its converse developmental variance) assumed to underlie variation in asymmetry. The main new result of the model is the very weak relationship between

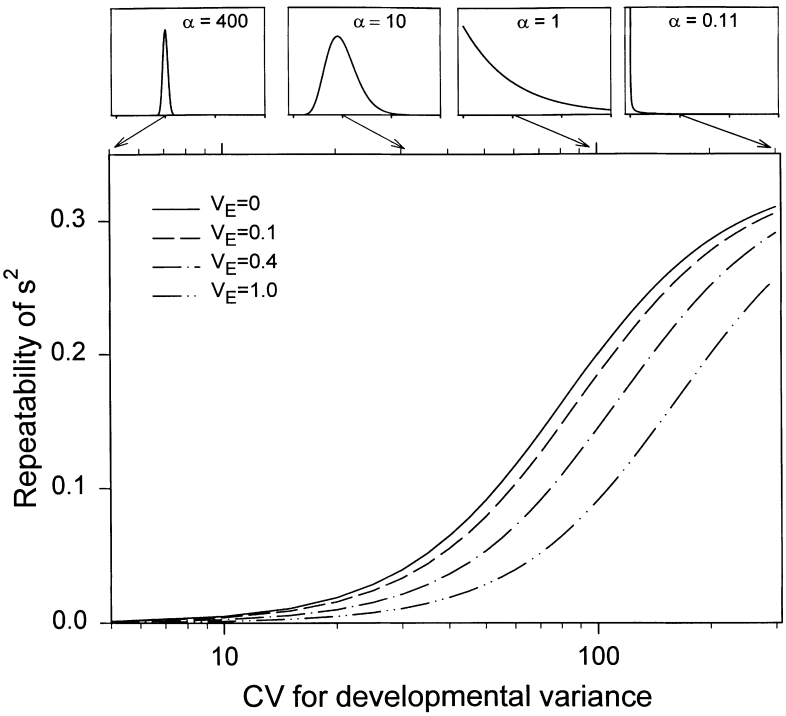

Fig. 5 The repeatability of asymmetry estimated as s^2 as a function of the coefficient of variation of developmental variance and measurement error, V_e. Parameter values are the same as Fig. 3.

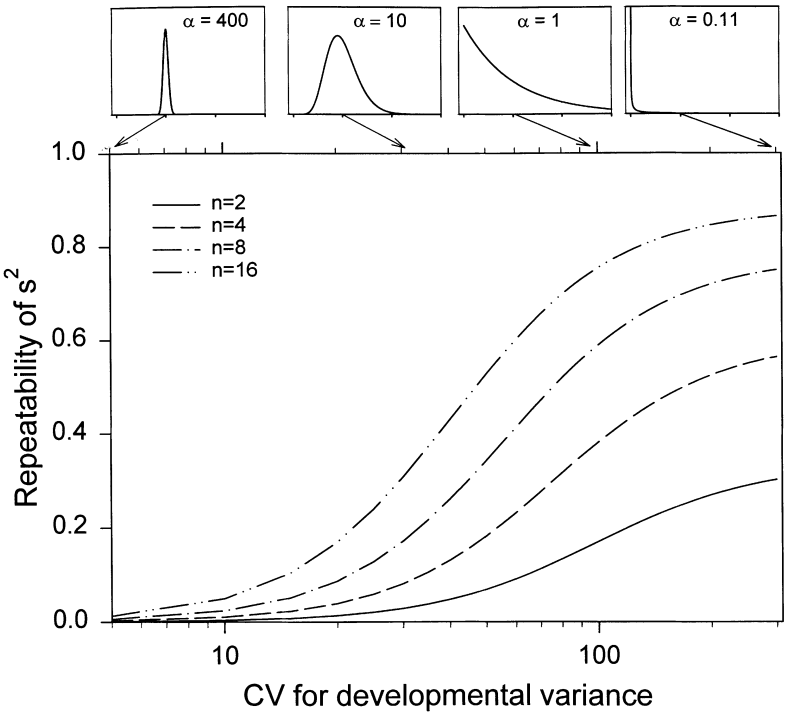

Fig. 6 The repeatability of asymmetry estimated as s^2 as a function of the coefficient of variation of developmental variance and the number of sides measured, n.

variance in developmental stability and fluctuating asymmetry. Only when the underlying variance in developmental stability is enormous does that variation become apparent in measures of asymmetry. To see this pattern, note (in Figs 3 and 5) that, in the best case when there is no error variance, the repeatability of each

asymmetry measure reaches the very modest value of 20% only when the coefficient of variation of developmental variance is about 100%. A coefficient of variation of 100% means that the standard deviation is equal to the mean. Since variances must be positive, this 100% coefficient of variation is accompanied by a highly skewed distribution of developmental variances. Perhaps even more striking is that the coefficient of variation of FA in the population is only about 30% higher than its minimum expected value when the coefficient of variation of developmental variance is 100%, as shown in Fig. 4.

One reason for presenting results in terms of repeatability is that it sets an upper limit to the heritability of asymmetry. If the heritability of developmental variance is 1, the heritability of asymmetry is its repeatability. In reality, there is ample evidence that individuals differ in developmental stability because of environmental factors as well as genetic ones (Parsons, 1990), and the heritability of asymmetry would certainly be lowered as a result, perhaps quite substantially. When the heritability of developmental variance is less than 1, we can be sure that the heritability of asymmetry will be less than its repeatability. One useful and simple result is that adding a given environmental variance to the developmental variance lowers the repeatability of asymmetry in exactly the same manner as does measurement error. For example, the line corresponding to $V_e = 1$ in Fig. 3 is equivalent to a heritability of asymmetry of 0.5. It is therefore not surprising that asymmetry seems to have very low heritability, usually less than 5% (Whitlock & Fowler, 1997; Gangestad & Thornhill, 1999).

Another very useful interpretation of the repeatability is as the correlation of asymmetry values for sets of traits with the same developmental variances. If the developmental variances of different traits are proportional – that is the variance of one trait is a multiple of the variance in some other trait – the correlation of asymmetries of these traits on the same individual will equal the repeatability. However, different traits on an individual may differ in their developmental stabilities for a variety of reasons (Møller & Swaddle, 1997, pp. 53–55; Leung & Forbes, 1997), so correlations may be substantially less than the repeatability. Gangestad & Thornhill (1999) reviewed a number of large data sets, however, and argued that correlations in FA among traits are close to those expected on the basis of kurtosis and average trait repeatabilities under their model. Future work comparing repeatabilities estimated from CVs and from kurtosis, trait correlations, and the heritability of FA in the same population could provide a means for testing models of FA, such as the one presented in this paper.

Given the general result that asymmetry rises slowly with the variation in developmental stability, it is interesting to observe that the coefficient of variation of fluctuating asymmetry is sometimes greater than 100% (see, e.g., Whitlock, 1996; Van Dongen, 1998a). Above $CV_{FA} = 100\%$, the CV of developmental stability rises extremely rapidly, as shown in Fig. 4. For example, $CV_{FA} = 170\%$ for a sample of 188 tarsus lengths in the olive sunbird, *Nectarina olivacea* (Van Dongen, 1998a). This result implies a CV of developmental stability of 220%. On the other hand, the available data sets with the largest sample sizes imply modest CVs for developmental stability (Gangestad & Thornhill, 1999).

To compare these inferred coefficients of variation for developmental stability with those for other traits, CV_{FA} should be divided by two, because they have units of the trait squared (Lande, 1977; Houle, 1992). The phenotypic coefficients of variation of morphological traits are generally between 2 and 20%, whereas fitness components have values generally between 10 and 100% (Houle, 1992). Thus, in cases such as the olive sunbird (Van Dongen, 1998a), where the predicted variation in developmental stability is extremely high, either the model is false, or developmental stability is sometimes more variable than for any previously studied traits. Asymmetry has attracted attention because it potentially captures information about developmental stability, which may be of fundamental importance to fitness. If developmental stability has higher variance than typical for fitness, this raises an interesting paradox.

The standard model makes a number of questionable assumptions to the standard model that could increase the CV_{FA} when violated. First, the model assumes that the distribution of sides is normal for a given level of developmental stability. The distribution of sides could instead be a mixture of different distributions, perhaps reflecting discrete events that deflect development into alternative pathways. For example, asymmetrical use, injury, starvation, or other traumas might have large effects on development, although not necessarily so large as to lead to the rejection of an individual as a statistical or biological outlier. Extreme individuals have a disproportionate impact on measures of variation. For example, Whitlock (1996) notes that exclusion of a single highly asymmetrical individual lowers CV_{FA} in his sample of wolf jaws from 145% to about half that, close to the minimum value expected if there were no variation in developmental stability. In correlational studies, extreme individuals can account for much of the apparent power of a model to explain the data (Leung & Forbes, 1997).

The developmental model of Klingenberg & Nijhout (1999) provides a very different explanation for how normality could be violated. These authors point out that developmental stability is likely to be an epiphenomenon of the parameters of the developmental system, rather than a single property of that system. They expect that variation in the fundamental developmental properties will have nonlinear effects on morphology, which could easily lead to non-normal distributions of sizes. Similarly, nonlinear interactions during development could result in distributions of developmental variances that are very different from the gamma distribution I assumed.

Another possibly incorrect assumption is that each side develops independently. Because sides usually develop simultaneously on the same organism, there are opportunities for interactions during development (Graham *et al.*, 1993; Klingenberg & Nijhout, 1998). The most plausible kind of interaction would be competition for resources during development (Klingenberg & Nijhout, 1998). This would tend to cause antisymmetry and lower CV_{FA} (Van Dongen, 1998a) and so cannot help to explain the high values that raise questions under the present model. The finding that some distributions of FA are consistent with condition-dependent antisymmetry (Rowe *et al.*, 1997) may indicate that models of this type should be taken seriously.

In addition to these assumptions common to the standard models of asymmetry, I also had to make an assumption about the actual distribution of developmental stabilities. I chose the gamma distribution, but other distributions, such as the log-normal, may be worth considering. My principal conclusion, that the usefulness of FA as an indicator of individual developmental stability is poor unless the variance of developmental stability is extremely large, does not depend on the choice of distribution. Previous numerical or simulation studies using mixtures of two developmental stabilities (Houle, 1997), three developmental stabilities (van Dongen, 1998b), and normal, half-normal and uniform distributions of developmental stabilities (Gangestad & Thornhill, 1999) yield similar conclusions.

In summary, this elaboration of a basic model in asymmetry studies is consistent with many of the largest experimental studies in suggesting that the proportion of the within-population variation in fluctuating asymmetry that can be explained by variation in developmental stability is small (Gangestad & Thornhill, 1999). If the variation in developmental stability is typical of that found for other sorts of traits, then the value of FA as an indicator of developmental stability is low. On the other hand, a number of smaller studies report distributions of FA that imply enormous variation in developmental stabilities (Whitlock, 1996; Van Dongen, 1998a; Lens & van Dongen, 1999; Lens *et al.*, 1999). In these cases, we need either to explain how the variance in developmental stability can be so high or to modify this standard model of the relationship between developmental stability and fluctuating asymmetry.

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