Evolution, 53(5), 1999, pp. 1396–1405

ANALYSIS OF GENOTYPIC DIFFERENCES IN DEVELOPMENTAL STABILITY IN *ANNONA CHERIMOLA*

FRANCISCO PERFECTTI[1] AND JUAN PEDRO M. CAMACHO
Departamento de Genetica, Facultad de Ciencias, Universidad de Granada, 18071 Granada, Spain
[1]*E-mail: fperfect@goliat.ugr.es*

Abstract.—The genetic basis of developmental stability, measured as asymmetry (fluctuating asymmetry in leaves), was analyzed in leaves and flowers of cherimoya (*Annona cherimola* Mill) and atemoya (*A. cherimola* × *A. squamosa*). The individuals analyzed belonged to a controlled collection of cultivars (clones) that had previously been characterized by means of isozymes. We used a nested design to analyze the differences in asymmetry at several sampling levels: individual leaves and flowers, individual trees, and genotypes. The clonal repeatability of developmental stability was not significantly different from zero, thus suggesting the absence of heritability of the asymmetry for leaves and flowers under these environmental conditions. No relationship between asymmetry and individual heterozygosity was found, but leaf fluctuating asymmetry was significantly related to particular isozymic genes. Petal and leaf size showed a phenotypically plastic response to the exposure zone of the tree (mainly due to light). Leaf fluctuating asymmetry also showed such a plastic response. No significant correlation was found between asymmetry and any pomological characters (some of these being fitness related). Finally, the hybrid species (atemoya) did not show larger developmental instability than did the parental species (cherimoya). All these data show that cherimoya asymmetry reveals the random nature of developmental noise, with developmental stability for leaves being possibly related to specific chromosome regions, but with weak evidence for genotypic differences in developmental stability.

Key words.—*Annona cherimola*, developmental stability, fluctuating asymmetry, heritability, heterozygosity, phenotypic quality, plant plasticity.

Received April 28, 1998. Accepted April 20, 1999.

Developmental precision, how a structure develops to fit an ideal pattern, is influenced by two opposing forces: developmental noise and developmental homeostasis (Palmer and Strobeck 1986; Palmer 1996). The former has a random nature and can arise from environmental and genetic causes, and, when acting during growth, tends to divert a structure from its ideal for a particular genotype and environment. Developmental homeostasis (or developmental stability) involves processes that buffer developmental noise, thereby producing higher stability during growth. These buffering processes may have a genetic basis, providing some genotypes with a superior developmental precision under particular environmental circumstances.

Fluctuating asymmetry (FA) is a pattern of random deviation with regard to perfect symmetry, involving a normal distribution of the right minus left values with mean zero (Mather 1953; Thoday 1958; Van Valen 1962). FA is a useful index of developmental precision (Markow 1994; Palmer 1996; Møller and Swaddle 1997). The effect of different levels of stress on developmental homeostasis may be assessed using FA (Leary and Allendorf 1989; Parson 1990; Clarke 1992). The stress may have an environmental cause, such as temperature, toxins, or parasites (Parson 1961; Valentine and Soulé 1973; Bagchi and Iyama 1983; Scheiner et al. 1991; Freeman et al. 1993), or may be caused by such genetic factors as new mutations or disruption of coadapted gene complexes produced in hybridation or inbreeding (Levin 1970; Leary et al. 1983; Clarke and McKenzie 1987; Graham 1992).

As a potential measurement of developmental homeostasis, FA has been related to heterozygosity (H). Since Lerner's (1954) hypothesis of increased homeostasis in multi-locus heterozygotes, many studies have addressed this relationship (reviewed by Mitton 1993). Positive associations between heterozygosity and developmental homeostasis have often been found, mainly at the population level (Mitton and Grant 1984; Mitton 1993; but for a different interpretation see Clarke 1993; Britten 1996), though less often at the individual level (Palmer and Strobeck 1986; Britten 1996). Several studies have proposed that developmental stability may be related to some individual loci instead of genome heterozygosity (e.g., Mitton 1978; McKenzie and Clarke 1988; Leary et al. 1993; Messier and Mitton 1996; Leamy et al. 1997).

For traits showing FA, differences in asymmetry have been considered to be a signal of individual quality in animals (Møller 1992; Swaddle and Cuthill 1994), and in plants (Møller 1996), a signal to pollinators (Møller 1995; Møller and Eriksson 1995) and a predictor of performance in long-lived cultivated species (Bagchi et al. 1989). The relationship between individual quality and FA has also been credited with importance in sexual selection (Møller and Pomiankowski 1993; Watson and Thornhill 1994).

The individual buffering capacity to cope with developmental noise presumably has a heritable basis (Gavrilets and Hasting 1994). The heritability of FA has been used to study this presumed heritable basis of developmental homeostasis. Møller and Thornhill (1997) have claimed a low but significant heritability for FA, although the validity of their conclusion has been widely questioned (Leamy 1997; Markow and Clarke 1997; Palmer and Strobeck 1997; Swaddle 1997; Whitlock and Fowler 1997).

Most FA studies have focused on animals, although plants, due to their modular construction and facility of cloning, are excellent material for studies of developmental stability (Freeman et al. 1993; Palmer 1996). However, the typical phenotypic plasticity of plants may obscure the relationships between asymmetry and developmental stability (Bradshaw 1965; Palmer 1996) because the phenotype variance of in-

dividuals of the same genotype may be produced by two general processes, plasticity and noise (Simons and Johnston 1997) and because some kinds of asymmetry may be caused by repeating environmental factors such as temperature or light gradients.

In the present paper, we address the genetic basis of developmental homeostasis in a controlled collection of a cultivated tree, the cherimoya (*Annona cherimola* Mill) and in its hybrid, the atemoya (*A. cherimola* × *A. squamosa*). Controlled tree collections have several advantages in relation to natural populations: Each cultivar is represented by several trees with the same genotype (enabling the partitioning of phenotypic variance at genotype, tree, and trait levels); collections are usually genetically characterized; and individuals have the same macroenvironment, which is usually favorable. We measured leaf-width and petal-length asymmetries as indexes of developmental stability, seeking to assess its possible genetic basis. Specifically, we addressed the following questions. Is individual asymmetry a signal of individual phenotypic quality? Is individual asymmetry related to individual heterozygosity or alleles at some loci? Is developmental stability heritable? Is developmental stability affected by a plant's plastic response to environmental factors (e.g., light)? Are hybrids more developmentally unstable than the original species?

Materials and Methods

Research Organism and Sampling Procedure

We analyzed the asymmetry of leaves and petals in cherimoya (*Annona cherimola* Mill, Annonaceae) and atemoya (*A. cherimola* × *A. squamosa*). The cherimoya tree is a semideciduous fruit tree of Andean origin and is cultivated in several subtropical zones in the world as a valuable commercial crop. The cherimoya tree seems to have scarcely changed morphologically and genetically during its domestication process (Popenoe 1921; Perfectti 1995). The cherimoya leaves are alternate, oval to slightly pointed, entire and pubescent, with a flat to slightly undulate blade and a short petiole (Schroeder 1945). The yellowish green flowers, which are inconspicuous but aromatic (Schroeder 1945), are generally pendulent on short peduncles. Ranging from 15 mm to 50 mm in length, the flower consists of three large fleshy petals subtended by three small sepals and three small undeveloped petals opposite the sepals. Occasionally these rudimentary petals develop into normal petals (Schroeder 1945). Flowers show protogyny (Thompson 1970). The pollinator in the cherimoya origin area (Peru and Ecuador) seems to be a nitidulid beetle (Kahn et al. 1991).

Twenty-two cultivars of cherimoya and three cultivars of atemoya were sampled. Each cultivar had an particular genotype that did not vary among trees belonging to the same cultivar (Perfectti 1995; Perfectti and Pascual 1998a). Therefore, each cultivar, propagated vegetatively by graft, was a clone. Each cultivar will be referred to as a genotype. These genotypes were grown in plot 29 of the subtropical tree collection of the CSIC Experimental Station La Mayora (Algarrobo-Costa, Malaga, Spain). We selected these genoypes to cover the entire range of heterozygosity of this collection (0.043–0.391 in cherimoya), and because at least two trees

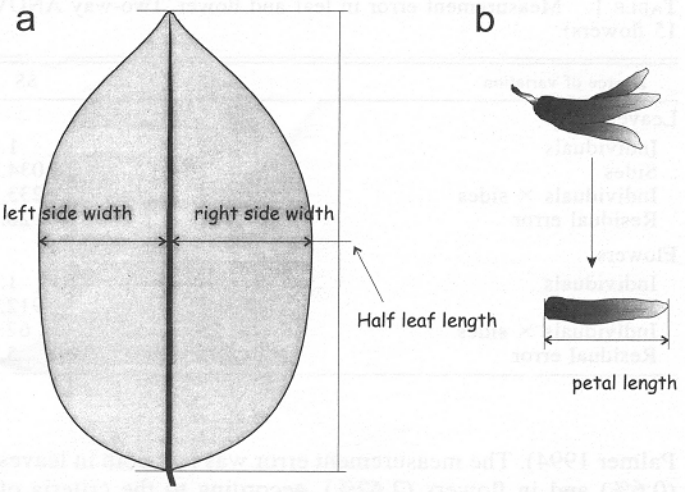

FIG. 1. Morphological characters measured in leaf (a) and flower (b).

of each genotype were grown in this plot. Trees were planted in a completely randomized design. Heterozygosity was assessed by the isozyme analysis of 23 loci (Perfectti 1995; Perfectti and Pascual 1998a). Only three atemoya genotypes were available for sampling in the same plot. *Annona squamosa*, the other parental species of the hybrid atemoya, was not available for sampling.

We used a nested design to study variation for asymmetry and size at several levels. From each genotype studied we sampled two trees. In each tree, we sampled two zones (north and south sides, the south side having more light exposure), and in each zone we collected five leaves and five flowers, that is, we collected 20 leaves and 20 flowers per cherimoya genotype. Only one tree was sampled for the cultivars Atemoya African Pride and Atemoya Pink Mammoth, because this collection had only one specimen of each.

Leaves were sampled among the first completely developed new spring leaves to standardize age and position on the branch. Flowers were collected in the female phase to standardize the developmental stage. Leaves and flowers were collected into bags and measured in the laboratory over a two-week period. During this time the material was refrigerated. The leaves, which show bilateral symmetry, were measured for width on the right and left side of the rachis at half the leaf length (see Fig. 1). The three radially symmetrical petals of the flower were detached and the length of each individual petal was measured with a caliper to the nearest 0.05 mm. Mean values of right and left leaf width were recorded as Lsize and mean petal length, Psize.

Measurement Error

Measurement error for each trait was calculated by repeating the measurements in a subsample of 36 leaves and 15 flowers (three petals/flower) of different cultivars and calculating a two-way ANOVA (sides × individuals, Table 1; Palmer 1994). In both cases the interaction between individual and side was significant ($P < 0.001$), implying that the measurement error was negligible both in leaves and in flowers compared to the variation between sides (asymmetry;

TABLE 1. Measurement error in leaf and flower. Two-way ANOVA of side (two in leaves, three in flowers) × individuals (36 leaves, 15 flowers).

Source of variation	df	SS	MS	*F*	*P*
Leaves					
Individuals	33	1.65	546.502	1414.487	< 0.001
Sides	1	18,034.56	1.654	0.234	0.631
Individuals × sides	33	233.53	7.077	18.316	< 0.001
Residual error	68	26.27	0.386		
Flowers					
Individuals	14	1.73	65.145	576.359	< 0.001
Sides	2	912.02	0.865	0.391	0.680
Individuals × sides	28	62.03	2.215	19.601	< 0.001
Residual error	45	5.09	0.113		

Palmer 1994). The measurement error was low both in leaves (0.6%) and in flowers (2.62%), according to the criteria of Yezerinac et al. (1992).

Asymmetry Indexes

For leaves, we calculated asymmetry as right-side minus left-side width of each leaf. The distribution of leaf signed asymmetry was normal (W-statistic = 0.982, n = 439, NS) after removing one outlier, and with a mean equal to zero (t-test = −1.63, df = 438, NS). No evidence for directional asymmetry was found in cherimoya leaves, as revealed by a paired t-test comparing left and right measurements (t = −1.417, df = 438, NS). Therefore, we concluded that cherimoya leaves show FA.

Because cherimoya flowers show radial symmetry, we calculated floral asymmetry as the difference between the length of the longest and the shortest petals (Møller and Eriksson 1994). This criterion provides an index similar to the unsigned asymmetry, but we did not make assumptions concerning FA in petals because we could not differentiate between types of asymmetry. After examining asymmetry values, we discarded seven outliers from 422 flowers. These values were found mainly when flowers varied in petal number, a common phenomenon in cherimoya (Schroeder 1945).

We found a positive and significant relationship (R = 0.286, $P < 0.001$) between unsigned leaf asymmetry and Lsize. Flowers also registered a positive correlation between Psize and asymmetry (R = 0.251, $P < 0.001$). Because the allometric relationship between unsigned asymmetry and trait size did not deviate significantly from isometry, we adjusted for scaling effects by dividing by mean trait size (index 2 in Palmer 1994). The transformation succeeded in correct the scaling effects (leaves: R = 0.063, NS; flowers: R = 0.066, NS). Henceforth, this asymmetry index (AI) will be called LAI for leaves and PAI for flower petals.

The AI was calculated for each individual leaf and flower, and as a mean for each genotype ($\overline{\text{LAI}}$ and $\overline{\text{PAI}}$). Because the AI did not show a normal distribution, we normalized it before statistical analyses by means of a Box-Cox transformation ($y' = [y + 0.001]^{0.4}$) following Swaddle et al. (1994). Mean asymmetry and mean size indexes were compared by paired t-tests. We also compared the coeficients of variance (CV) of size traits ($\overline{\text{Lsize}}$ and $\overline{\text{Psize}}$) and the CV of $\overline{\text{LAI}}$ and $\overline{\text{PAI}}$ by paired t-tests.

The relationship between the AI in flower and leaf traits of the same genotype were determined by Pearson product-moment correlations of the mean values for genotype. The relationship between several pomological characteristics and both the genotype AI and genotype mean trait size ($\overline{\text{Lsize}}$ and $\overline{\text{Psize}}$) were analyzed using Pearson correlations, applying the sequential Bonferroni's test to avoid Type I errors. These data, four-year means obtained from Perfectti (1995), included: fruit weight, percentage of skin, percentage of heart, percentage of flesh, percentage of seeds (w/w with respect to the total fruit weight), seed weight, seed index (number of seeds in 100 g of fruit), skin resistance, percentage of fruits parasited by insects of the genus *Ceratitis,* and Brix degrees of flesh. These variables were normalized where necessary.

Morphological Variation in Relation to Genotypes

The relationship between mean genotype asymmetry and heterozygosity (H) was investigated by means of Pearson's product moment correlation. Heterozygosity values for each genotype were calculated as the proportion of heterozygous loci of 23 isozyme loci (Perfectti 1995).

To find the individual genes that explain more variance of asymmetries and character sizes, we performed a multiple regression among allele frequencies (0, 0.5, or 1, i.e., not present, heterozygote, or homozygote) as independent variables and both mean genotype asymmetries and mean genotype trait sizes as dependent variables. Genotypes of 13 polymorphic genes (*Adh1, Got1, Got2, Idh2, Mdh1, Me1, Pgi1, Pgm1, Pgm2, Skd1, Tpi1, Tpi2,* and *Tpi3*) for these genotypes were taken from Perfectti and Pascual (1998a). To minimize the dependence between independent variables, we discarded the less frequent allele at each locus, following Xie and Knowles (1992). To avoid spurious regressions, tolerance was set at 0.01, the value of F to enter to 4.0, and the value of F to out to 3.9.

To analyze the effect of genotype and light exposure zone (north versus south) on AI and trait size, we performed a cross nested ANOVA using the GLM procedure (SAS Institute 1989). The ANOVA model included exposure zone and genotype as independent factors. Tree was nested within genotype, and flower and leaf measurements were nested within tree. Type III sums of squares were used. We estimated the variance components from the expected mean squares. Ex-

cluding the variance from exposure zone, we partioned the total variance (V_P) into the different sample levels (leaves and flowers, trees, genotypes), determining the contribution of these levels to the total variance. The bottom level gathers the variance within trees and reflects the contribution of the special environmental variance (temporary or localized circumstances) to the total variance (V_{ES}/V_P). The intermediate level represents the variance among trees within genotypes. This level reflects the general environmental component of the variance arising from permanent or nonlocalized circumstances (V_{EG}/V_P; Falconer 1989). The upper level, among genotypes, accounted for the variance induced by genetic differences. The percentage of the total variance at this level represents the clonal repeatability (V_G/V_P), an upper limit to the heritability (Falconer 1989).

Finally, atemoya asymmetry values were used only to compare the asymmetry performance of hybrids with respect to its cherimoya progenitor by means of one-way ANOVAs.

Results

Values for asymmetry and character size in each genotype ($\overline{\text{LAI}}$, $\overline{\text{PAI}}$, $\overline{\text{Lsize}}$, and $\overline{\text{Psize}}$) and mean values for cherimoya and atemoya are shown in Table 2. We will first report the data obtained for cherimoya. The $\overline{\text{PAI}}$ was higher than the $\overline{\text{LAI}}$ (paired t-test, $t = -5.658$, df = 21, $P < 0.001$). Asymmetry variance was higher in leaves. Coefficients of variation were higher in leaves than in flowers. Leaf widths were more variable than petal lengths ($t = -4.385$, df = 21, $P < 0.001$), and the leaf-asymmetry CV was also higher than the petal-asymmetry CV ($t = 3.946$, df = 21, $P < 0.001$).

Means for leaf and flower did not correlate with the asymmetry traits analyzed. Petal size ($\overline{\text{Psize}}$) did not correlate with leaf size ($\overline{\text{Lsize}}$; $r = 0.402$, $P = 0.064$) nor did petal asymmetry ($\overline{\text{PAI}}$) correlate with leaf asymmetry ($\overline{\text{LAI}}$; $r = -0.003$, $P = 0.990$) . The AI and size traits were not indicators of pomological quality, because (after applying the sequential Bonferroni's test to avoid Type I errors) pomological variables did not significantly correlate with asymmetry or character sizes (Table 3).

To test whether heterozygosity is related to developmental homeostasis, we analyzed the correlations between H and asymmetry. Heterozygosity of cherimoya genotypes ranged from 0.043 to 0.391 and did not significantly correlate with asymmetry or size measurements (Table 4). The possibility that individual loci, instead of the heterozygosity, were involved in the extent of asymmetry and character size was also investigated. We performed multiple lineal regressions among allele frequencies (independent variables) and both asymmetry indexes and character sizes (dependent variables; Table 4). In leaves, three alleles accounted for 53.3% of the variance for $\overline{\text{LAI}}$. Of the total variance of $\overline{\text{Lsize}}$, 37.6% was explained by one allele. In flowers, 21% of $\overline{\text{PAI}}$ variance was explained by only one allele, but, after inspection of the regression, it became evident that just one point was responsible for this slight significant P-value. $\overline{\text{Psize}}$ was not related to any allele studied.

To ascertain how the variance for asymmetry and character size was distributed at the several sampling levels and how exposure zone affected to these variables, we used a cross nested ANOVA (Table 5). Lsize appeared to be influenced by genotype and exposure zone. The interaction of these two factors was also significant. Mean Lsize was 40.73 mm on the north side and 43.14 mm on the south side. Genotype did not affect leaf asymmetry. However, LAI were affected by exposure zone, with larger values in the south-facing leaves. Psize was influenced by both genotype and exposure (Table 5). Psize mean was 29.72 mm on the north side and 30.73 mm on the south side. PAI, however, was not influenced either by genotype or by exposure, even though the differences among trees between genotypes seem to affect petal asymmetry.

Total phenotypic variance minus the variance associated with exposure zone was partitioned into components representing the variance at three levels (Table 5). At the botton level (within trees) we found 97% of the total variance of the leaf AI and more than 73% of the petal AI. The intermediate level represents the variance among trees within genotypes. LAI showed no variance at this level. In PAI, this level accounted for 26.6% of total variance. The upper level, among genotypes, accounted for the variance induced by genetic differences. This level gathered 3% of total variance in leaf AI, but with a nonsignificant P-value. The petal AI accounted for 0% of total variance. Therefore, genetic differences among genotypes did not seem to influence the AI of leaves or flowers, and thus no heritability of the AI should be expected in this environment. However, Lsize and Psize showed a different partitioning of the total phenotypic variance. Within-trees variance showed lower values: 13.3% in Lsize and 32.2% in Psize. The second level (among trees within genotypes) accounted for 54.2% in Lsize and 39.2% in Psize, with significant values in both cases. The upper level (among genotypes) explained 32.5% of total variance shown by Lsize and 28.6% of that shown by Psize. Therefore, clonal repeatability was significant for both variables (Table 5).

Finally, we studied atemoya individuals to compare developmental instability of these hybrids with that of their ancestor species, i.e. cherimoya. Atemoya leaves were narrower ($\overline{\text{Lsize}}$ mean=25.65 mm) than cherimoya leaves ($\overline{\text{Lsize}}$ mean = 41.93 mm; $F = 12.566$; df = 1,23; $P = 0.002$), but no significant differences were found for the $\overline{\text{LAI}}$ ($\overline{\text{LAI}}$ $F = 0.002$, df = 1,23, $P = 0.967$). Likewise, petal length was not significantly different in atemoya and cherimoya ($F = 0.478$, df = 1,23, $P = 0.497$), and the $\overline{\text{PAI}}$ did not differ between the two species ($\overline{\text{PAI}}$ $F = 1.937$, df = 1,23, $P = 0.177$). Levene's test failed to show significant differences for between-group variances for any variable analyzed.

Discussion

Fluctuating Asymmetry as an Indicator of Individual Quality

The AI values in cherimoya (mean $\overline{\text{LAI}} = 0.044$, mean $\overline{\text{PAI}} = 0.057$) fell within the range of values reported in previous studies (Møller and Eriksson 1994; Sherry and Lord 1996b). The general lower flower asymmetry in comparison to leaf asymmetry (Sherry and Lord 1996b; Evans and Marshall 1996) has been suggested to be a consequence of a more stable development of flowers (Sherry and Lord 1996b).

TABLE 2. Asymmetry in 22 cherimoya and three atemoya cultivars. cv, cultivars; H, heterozygosity; *n*, number of leaves or flowers; CV, coefficient of variation; LAI and PAI, unsigned asymmetry divided by trait mean size. Values in cherimoya means and atemoya means are means between cultivar values.

		Leaf half width					Petal length				
Cultivar[1]	H	*n*	Size ± SE ($\overline{Lsize}$)	CV ($\overline{Lsize}$)	$\overline{LAI}$ ± SE	CV ($\overline{LAI}$)	*n*	Size ± SE ($\overline{Psize}$)	CV ($\overline{Psize}$)	$\overline{PAI}$ ± SE	CV ($\overline{PAI}$)
BH	0.174	20	41.15 ± 0.84	9.09	0.0499 ± 0.0075	66.81	20	34.01 ± 0.78	10.30	0.0479 ± 0.0056	52.36
BO	0.261	20	47.75 ± 2.21	20.72	0.0552 ± 0.0078	63.49	17	27.28 ± 0.78	11.82	0.0449 ± 0.0048	43.77
BS	0.130	20	40.69 ± 1.74	19.13	0.0557 ± 0.0178	142.73	20	30.42 ± 0.70	10.24	0.0625 ± 0.0097	69.19
C3td	0.174	20	49.56 ± 1.43	12.88	0.0355 ± 0.0063	78.94	19	30.69 ± 0.85	12.11	0.0528 ± 0.0076	62.46
C3te	0.174	20	54.38 ± 1.40	11.52	0.0298 ± 0.0058	87.08	19	28.97 ± 0.69	10.40	0.0555 ± 0.0130	101.88
C4	0.304	20	46.99 ± 1.14	10.86	0.0526 ± 0.0099	84.34	20	29.36 ± 0.52	7.90	0.0524 ± 0.0048	41.10
CA	0.217	20	34.05 ± 0.81	10.70	0.0437 ± 0.0074	75.76	14	28.21 ± 0.72	9.56	0.0435 ± 0.0051	43.72
CN	0.043	20	41.45 ± 1.54	16.56	0.0602 ± 0.0100	74.62	18	34.08 ± 1.21	15.11	0.0604 ± 0.0063	44.50
CR	0.087	20	55.78 ± 1.29	10.32	0.0431 ± 0.0073	75.91	20	31.41 ± 0.89	12.67	0.0569 ± 0.0083	65.23
CT	0.304	20	38.86 ± 1.22	14.04	0.0494 ± 0.0082	74.56	20	27.86 ± 0.48	7.67	0.0527 ± 0.0069	58.67
CU	0.174	20	32.94 ± 1.76	23.88	0.0399 ± 0.0075	84.02	15	24.65 ± 0.81	12.68	0.0497 ± 0.0079	61.74
CH	0.217	19	45.59 ± 1.41	13.46	0.0568 ± 0.0082	62.94	20	30.02 ± 0.49	7.30	0.0784 ± 0.0115	65.43
FI	0.217	20	30.91 ± 1.31	19.02	0.0310 ± 0.0051	73.24	18	29.04 ± 0.57	8.27	0.0544 ± 0.0077	60.08
MA	0.217	20	39.04 ± 1.19	13.65	0.0560 ± 0.0103	82.65	19	30.46 ± 0.54	7.69	0.0573 ± 0.0073	55.44
NE	0.217	20	36.27 ± 1.00	12.38	0.0337 ± 0.0052	69.36	20	32.00 ± 1.10	15.31	0.0477 ± 0.0049	45.94
P410	0.217	20	35.64 ± 1.08	13.50	0.0326 ± 0.0048	65.52	19	26.21 ± 0.49	8.14	0.0584 ± 0.0105	78.62
P606	0.217	20	37.61 ± 1.36	16.19	0.0351 ± 0.0057	72.23	18	30.66 ± 0.59	8.17	0.0832 ± 0.0114	58.24
PC	0.391	20	46.39 ± 1.56	15.07	0.0541 ± 0.0106	87.71	20	29.79 ± 0.57	8.53	0.0487 ± 0.0061	56.00
PÑ	0.043	20	28.68 ± 0.84	13.03	0.0336 ± 0.0051	67.73	20	29.60 ± 0.65	9.85	0.0485 ± 0.0060	54.95
SP	0.261	20	50.81 ± 1.22	10.70	0.0330 ± 0.0055	74.90	20	37.93 ± 0.94	11.11	0.0764 ± 0.0083	48.60
SP78	0.304	20	39.11 ± 1.29	14.76	0.0296 ± 0.0053	80.40	20	26.82 ± 0.49	8.19	0.0551 ± 0.0098	79.31
WH	0.304	20	48.80 ± 1.22	11.17	0.0577 ± 0.0088	68.30	19	35.71 ± 0.97	11.84	0.0616 ± 0.0124	88.04
Cherimoya (mean)		22 cv	41.93 ± 1.60	14.21	0.0440 ± 0.0023	77.87	22 cv	30.24 ± 0.67	10.22	0.0568 ± 0.0023	60.69
AA	0.391	10	22.31 ± 0.48	6.77	0.0315 ± 0.0055	55.13	10	33.25 ± 0.93	8.87	0.0487 ± 0.0147	95.31
AG	0.435	20	20.91 ± 0.60	12.81	0.0387 ± 0.0068	78.92	20	26.52 ± 0.48	8.03	0.0567 ± 0.0100	78.62
AP	0.348	10	33.73 ± 0.91	8.54	0.0603 ± 0.0206	107.80	10	26.88 ± 0.88	10.34	0.0476 ± 0.0101	67.37
Atemoya (mean)		3 cv	25.65 ± 4.06	9.37	0.0435 ± 0.0087	80.62	3 cv	28.88 ± 3.78	9.08	0.0510 ± 0.0029	80.43

[1] Cultivars names: BH, Booth; BO, Bonita; BS, Bronce Suave; C3te, Chiuna 3 temprana; C3td, Chiuna 3 tardia; C4, Chiuna 4; CA, Campas; CN, Concha Lisa; CR, Corazon; CT, Ott; CU, Cumbe; CH, Chaffey Riverside; FI, Fino de Jete; MA, Manteca; NE, Negrito; P410, Peru 410-16; P606, Peru 606; PC, Pinchudo; PÑ, Piña; SP, Spain; SP78, Seleccion Peru 78; WH, White; AA, Atemoya African Pride; AG, Atemoya Gefner; AP, Atemoya Pink Mammoth.

TABLE 3. Pearson product-moment correlation coefficients between several pomological variables and both leaf and flower variables. *P*-values are shown in parentheses.

	Leaves		Flowers	
	$\overline{\text{Lsize}}$	$\overline{\text{LAI}}$	$\overline{\text{Psize}}$	$\overline{\text{PAI}}$
Fruit weight	0.236 (0.291)	0.259 (0.244)	0.267 (0.229)	0.092 (0.685)
% skin	0.093 (0.680)	−0.286 (0.198)	−0.285 (0.199)	0.047 (0.837)
% heart	−0.206 (0.357)	−0.423 (0.050)	−0.314 (0.154)	−0.177 (0.430)
% flesh	−0.122 (0.590)	0.283 (0.201)	0.229 (0.304)	−0.055 (0.807)
% seeds	0.112 (0.621)	−0.092 (0.683)	−0.078 (0.731)	0.065 (0.773)
Seed index	−0.075 (0.742)	−0.306 (0.165)	−0.229 (0.306)	−0.221 (0.324)
Mean seed weight	0.157 (0.484)	−0.023 (0.919)	−0.046 (0.838)	0.289 (0.191)
skin resistance	0.374 (0.086)	0.078 (0.731)	0.182 (0.416)	−0.009 (0.969)
% *Ceratitis* infection	0.276 (0.213)	0.068 (0.765)	0.087 (0.701)	0.091 (0.686)
Brix degrees	0.013 (0.955)	0.412 (0.057)	−0.028 (0.900)	0.025 (0.912)

Møller and Eriksson (1994) reported opposite patterns depending on the plant species. In cherimoya, leaves showed a greater size coefficient of variation than did flowers, fitting the general pattern of more variability in leaves than in flowers. However, we found that the leaf AI showed lower values than did the petal AI. The development of the cherimoya flower appears not to be very precisely controlled. In some flowers, rudimentary petals occasionally develop into large petals, producing flowers with an unusual number of large petals (Schroeder 1945). The higher petal AI could be due to imprecise flower development. Because the PAI was measured as the difference between the two most unequal petals, but LAI was the difference between the two leaf halves, the different measurement procedures could have maximized the petal AI values in relation to the leaf AI. Another possibility is that cherimoya flowers show some other type of asymmetry (antisymmetry or directional asymmetry) that cannot be deciphered by our analysis because radial symmetry makes it difficult to test this possibility.

Petal asymmetry did not correlate with leaf asymmetry as in other studies (Møller and Eriksson 1994; Evans and Marshall 1996; Sherry and Lord 1996b), suggesting the existence of trait- or organ-specific developmental stability processes (Leamy 1993). Nevertheless, a theoretical approach has recently shown that this lack of correlation is expected even assuming a general (nonspecific) developmental buffering capacity (Leung and Forbes 1997).

In addition to the pattern of leaves being more variable in size than flowers, Møller and Eriksson (1994) found a pattern of negative or nonsignificant correlation between petal length and FA and positive or nonsignificant correlation between leaf size and leaf FA. They assumed that high-quality individuals produced larger sexual traits with smaller FA levels. Later, Møller and Eriksson (1995) found nectar content to be positively correlated with petal length and negatively with petal FA in several plant species. These authors also found assortative mating between flowers with low levels of FA mediated by pollinator preference for more symmetrical flowers. Møller (1995) showed this pollinator preference experimentally, and Møller and Eriksson (1995) suggested that a sensory bias of the pollinator for a symmetric pattern or a reinforcement of pollinator behavior (the more symmetrical the flower, the greater the reward) may explain the evolution of these preferences. In addition, because FA may be an indicator of the general condition of the plant (e.g., Møller 1996), Møller and Eriksson (1995) hypothesized that the maintenance of this pollinator preference for larger and more symmetrical flowers may be explained by the good genes argument: Those plants with higher developmental quality will produce more symmetrical and larger flowers with a greater reward for pollinators and thus will be more visited by them.

In cherimoya, this scenario is not supported. The positive correlation between petal length and absolute petal asymmetry, the higher values of PAI compared to LAI, the low control over the number of large petals, the plasticity of petal size, and the absence of a clear relationship between asymmetry and genotype do not suggest that petal length asymmetry works as an honest signal of quality for pollinators. Present knowledge on pollination biology of cherimoya trees is scarce. In Peru and Ecuador, where cherimoya originated, a nitidulid beetle seems be the pollinator (Kahn et al. 1991). Because cherimoya flowers are inconspicuous but very fragrant (Schroeder 1945), chemical signals must be an important factor in flower localization by pollinators, reducing the importance of petal FA as a signal of quality.

TABLE 4. Pearson product-moment correlation coefficients between heterozygosity and leaf and flower variables. Multiple regression with allele frequencies as independent variables. Genes in the regression equation are shown. P-values are shown in parentheses.

	Heterozygosity	Alleles (multiple regression)	
Leaves			
$\overline{\text{Lsize}}$	0.162 (0.471)	*Tpi2*	$R = 0.614\ R^2 = 0.376\ (0.002)$
$\overline{\text{LAI}}$	0.103 (0.694)	*Pgm1, Got2, Idh2*	$R = 0.730\ R^2 = 0.533\ (0.003)$
Flowers			
$\overline{\text{Psize}}$	−0.018 (0.646)	No variables entered into the regression equation	
$\overline{\text{PAI}}$	−0.036 (0.875)	*Adh1*	$R = 0.458\ R^2 = 0.209\ (0.032)$

TABLE 5. Analysis of sun exposure zone and genotype. Cross-nested ANOVAs on asymmetry and character size of leaves and flowers. The total variance minus the contribution of exposure zone was partitioned into three levels: among genotypes, among trees within genotypes, and within trees. For each level, the percent of the total variance was calculated.

	Source of variation	df	MS	F	Variance	% Variance
Leaf						
LAI	Among genotypes	21	0.01335	1.54	3.184	3.05
	Exposure	1	0.03829	4.43*		
	Among trees within genotypes	22	0.00790	0.91	−4.341	0.00
	Genotype-exposure interaction	21	0.01266	1.46		
	Among trees within genotypes-exposure interaction	22	0.01197	1.38		
	Within trees	352	0.00864		101.157	96.95
Lsize	Among genotypes	21	1128.41	57.94***	47.526	32.51
	Exposure	1	651.27	33.44***		
	Among trees within genotypes	22	177.89	9.13***	79.207	54.17
	Genotype-exposure interaction	21	56.85	2.92***		
	Among trees within genotypes-exposure interaction	22	117.86	6.05***		
	Within trees	352	19.48		19.477	13.32
Flower						
PAI	Among genotypes	21	0.00776	1.35	−0.00007	0.00
	Exposure	1	0.00185	0.32		
	Among trees within genotypes	22	0.01016	1.77*	0.002	26.59
	Genotype-exposure interaction	21	0.00438	0.76		
	Among trees within genotypes-exposure interaction	20	0.00893	1.55		
	Within trees	329	0.00575		0.006	73.41
Psize	Among genotypes	21	188.92	22.07***	7.603	28.61
	Exposure	1	133.09	15.55***		
	Among trees within genotypes	22	30.64	3.58***	10.409	39.17
	Genotype-exposure interaction	21	14.96	1.75*		
	Among trees within genotypes-exposure interaction	20	12.74	1.49		
	Within trees	329	8.56		8.560	32.21

* $P < 0.05$; *** $P < 0.001$.

Some pomological variables (resistance to insects of the genus *Ceratitis*, seed weight, fruit weight) are clearly fitness-related traits, but were not correlated with the asymmetry indexes nor were any of the other pomological characters analyzed in the present study. Therefore, in cherimoya, FA is not a useful predictor of commercial or fitness genotype quality. Leung and Forbes (1997) concluded that FA is a very poor predictor of quality for low FA values, although high FA values can indicate low-quality individuals.

Developmental Homeostasis and Heterozygosity

Heterozygosity (H) has often been related to increased developmental homeostasis (Lerner 1954; Mitton and Grant 1984). Several studies, mainly in animals (reviewed by Mitton and Grant 1984), have reported a positive correlation between H and increased homeostasis, mainly at the population level (Mitton and Grant 1984; Palmer and Strobeck 1986; Mitton 1993). In plants, some of the earliest studies addressed this relationship (Mather 1953; Lerner 1954; Thoday 1955; Paxman 1956; Levin 1970). Recently, Sherry and Lord (1996a) found that a *Clarkia tembloriensis* selfing population with low H-values had a significantly higher FA than its neighboring outcrossed population, which had higher H values. However, another pair of selfing and outcrossed populations studied had no differences in FA. Strauss (1987) failed to find a direct relationship between H and developmental stability in *Pinus attenuata*.

We found no correlation between individual multilocus heterozygosity and petal or leaf asymmetries. In a meta-analysis of correlation coefficients between individual multilocus heterozygosity and FA, Britten (1996) found that the effect of H on developmental stability estimates were weak or nonexistent. Because a few polymorphisms may not be a good predictor of genome heterozygosity (Mitton 1993), multilocus heterozygosity may not be a good predictor of developmental stability.

Several studies (Mitton 1978; Coelho and Mitton 1988; McKenzie and Clarke 1988; Leary et al. 1993; Messier and Mitton 1996) have demonstrated that developmental stability may be related to some individual loci instead of genome heterozygosity. Particular loci can influence developmental stability via metabolic efficiency (Palmer 1996) or cell adhesion functions (Clarke 1997). In cherimoya, three genes (*Pgm1, Got2* and *Idh2*) explained 53.3% of the $\overline{\text{LAI}}$ variance. These three loci are not linked (Perfectti and Pascual 1998b). However, this relationship between $\overline{\text{LAI}}$ and some alleles could be an artifact of the statistical procedure. High numbers of independent variables (alleles) in relation to the dependent variables (genotypes) may produce significant regression coefficients, although these regressions will be unstable and are unlikely to be replicated. Because the cross-nested ANOVA analysis (see below) failed to show a significant genetic component of the variance of $\overline{\text{LAI}}$, it is difficult to accept the regression analysis as definitive evidence. More studies are necessary to test the relationship between specific alleles and leaf FA.

Genotypic Differences in Developmental Stability

In a meta-analysis of heritability estimates of developmental stability, Møller and Thornhill (1997) found low but significant heritability of developmental stability (mean value = 0.27). But this meta-analysis has received serious criticism (e.g., Leamy 1997; Markow and Clarke 1997; Palmer and Strobeck 1997; Swaddle 1997; Whitlock and Fowler 1997). We have not estimated FA heritability in cherimoya leaves and petals, but rather clonal repeatability, which yields an upper limit to heritability (Falconer 1989). From the nonsignificant values of FA clonal repeatability in cherimoya, we can infer that FA heritability did not significantly differ from zero for these characters, in this population, and in this environment. As Leamy (1997) emphasized, most studies using FA to estimate the heritability of developmental stability have not reported significant results. However, trait sizes in cherimoya (Lsize and Psize) showed high and significant values of clonal repeatability, thus implying high heritability values, as is usual for morphological traits (Mousseau and Roff 1987). Also, a recent study in *Drosophila melanogaster* has shown significant mutational heritability for sternopleural bristle number and wing length, but nonsignificant heritability for FA of these same traits, which strongly suggests that FA only reflects developmental noise in that population (Monedero et al. 1997). Our results in cherimoya leaves and flowers also suggest that FA reflects only developmental noise affecting the different genotypes similarly. Because of cultivation, it is plausible that these trees are growing under low stress levels, which could explain the similarity of FA levels among different genotypes. A more severe stress level could make evident differences in developmental homeostasis among different genotypes (Parson 1990). Alternatively, performance differences among genotypes are perhaps manifested only at certain developmental stages (Endler 1986) not analyzed in the present work.

Fluctuating Asymmetry and Plant Plasticity

Phenotypic plasticity involves developmental changes induced by environmental variables that produce microadaptations to the environment during development (Bradshaw 1965; Winn 1996). In cherimoya, both petal length and leaf width showed a plastic response to the different solar orientations (north and south sides) of the tree. Leaves and flowers on the south side were larger than those of the north side. The south side received more insolation than did the north side, and this fact implies increased photosynthetic productivity and thus more resources for growth. It is commonly assumed that flowers are less plastic than leaves (Sherry and Lord 1996b). Flowers, due to their reproductive function, are more subjected to stabilizing selection (Berg 1959; Bradshaw 1965). In cherimoya, both petal length and leaf width were affected by genotype and by exposure (north and south orientations; see Table 5). The interaction between genotype and exposure (i.e., genotype and plasticity) was significant in both petals and leaves, implying varying plastic responses of the different genotypes. A weak genetic basis for the plastic response to light gradients has been reported in some plants (Jasienski et al. 1997). The plastic response in leaves may have a microadaptive explanation, but the same response in petals is more difficult to explain. Perhaps flower plastic response is simply a by-product of the higher level of resources available to south-facing branches.

Several studies have reported no relationship between FA and plastic responses (e.g., Bagchi and Iyama 1983). However, phenotypic plasticity in cherimoya affected the degree of asymmetry in leaves but not in flowers. Leaves exposed to more insolation grew larger and showed more FA than expected due to the increase in size (LAI is a size-corrected index of asymmetry). The probably faster growth of the south-facing leaves may impose stressful conditions that increase the FA level. However, flowers exposed to the south side did not show greater asymmetry although they showed an increase in size. In addition, petal asymmetry was affected by differences among trees, but not among genotypes. This suggests that petal asymmetry can reveal developmental noise due to localized circumstances.

Fluctuating Asymmetry in Hybrids

The disruption of genomic coadaptation produced in hybridations may be reflected in higher levels of developmental instability (Zakharov 1989; Graham 1992), as several studies have reported (e.g. Manly and Ledig 1979; Graham and Felley 1985; Leary et al. 1985). However, other studies have reported no differences between hybrids and parental species (e.g., Felley 1980; Freeman et al. 1995). The genotypes of atemoya, the hybrid between *A. cherimoya* and *A. squamosa*, did not show an increased level of FA for any of the traits studied in comparison with *A. cherimola*. Taking into consideration that we have not sampled *A. squamosa*, the other parental species, several possibilities may explain these results. First, the two species have highly compatible genomes. In addition, these atemoya genotypes might be the result of artificial or natural selection for more coadapted genomes. Second, disruption of genomic coadaptation can occur without FA as an indicator. Third, hybrids show disruption of genomic coadaptation, but only within the context of their natural environment. The favorable breeding environment might decrease the level of stress and obscure a possible higher developmental instability of the hybrid. Given the high degree of hybridization among species of this genus (Venkataratnam and Satyanarayanaswamy 1958) this possibility could be tested by analyzing a collection of different hybrid species bred under different environmental conditions. Finally, the atemoya genotypes analyzed show greater heterozygosity that did cherimoya genotypes, and the possible buffering effect of increased heterozygosity (Palmer and Strobeck 1986) might balance the disruption of coadapted gene complexes.

In conclusion, cherimoya FA is a clear example of the random nature of developmental noise. Despite the weak evidence for genetically based developmental homeostasis in cherimoya (developmental stability for leaves being related to specific loci), we have found no general genetic influence in developmental stability (clonal repeatability was not significantly different from zero), perhaps because of the low level of stress obscured the relationship between genotype and developmental homeostasis.

ACKNOWLEDGMENTS

We thank J. M. Farré, J. M. Hermoso, M. Rámos, R. González, M. Bueno, and L. Martín for providing access to the cherimoya collection and the pomological data. We also thank J. M. Gómez, C. López-Fanjul, M. Polak, I. Reche, and an anonymous reviewer for providing comments and criticisms. FP was supported by a fellowship (Programa de Becas Puente) from the University of Granada.

LITERATURE CITED

Bagchi, S., and S. Iyama. 1983. Radiation induced developmental instability in *Arabidopsis thaliana*. Theor. Appl. Genet. 65:85–92.

Bagchi, S. K., V. P. Sharma, and P. K. Gupta. 1989. Developmental instability in leaves of *Tectona grandis*. Silvae Genet. 38:1–6.

Berg, R. L. 1959. A general evolutionary principle underlying the origin of developmental homeostasis. Am. Nat. 93:103–105.

Bradshaw, A. D. 1965. Evolutionary significance of phenotypic plasticity in plants. Adv. Genet. 13:115–155.

Britten, H. B. 1996. Meta-analyses of the association between multilocus heterozygosity and fitness. Evolution 50:2158–2164.

Clarke, G. M. 1992. Fluctuating asymmetry: a technique for measuring developmental stress of genetic and environmental origin. Acta Zool. Fenn. 191:31–35.

———. 1993. The genetic basis of developmental stability. I. Relationships between stability, heterozygosity and genomic coadaptation. Genetica 89:15–23.

———. 1997. The genetic and molecular basis of developmental stability: the *Lucilia* story. Trends Ecol. Evol. 12:89–91.

Clarke, G. M., and J. A. McKenzie. 1987. Developmental stability of insecticide resistant phenotypes in blowfly: a result of canalizing natural selection. Nature 325:345–346.

Coelho, J. R., and J. B. Mitton. 1988. Oxygen consumption during hovering is associated with genetic variation of enzymes in honey-bees. Funct. Ecol. 2:141–146.

Endler, J. A. 1986. Natural selection in the wild. Princeton Univ. Press, Princeton, N.J.

Evans, A. S., and M. Marshall. 1996. Developmental instability in *Brassica campestris* (Cruciferae): fluctuating asymmetry of foliar and floral traits. J. Evol. Biol. 9:717–736.

Falconer, D. S. 1989. Introduction to quantitative genetics. 3d ed. Longman, London.

Felley, J. 1980. Analysis of morphology and asymmetry in bluegill sunfish (*Lepomis macrochirus*) in the southeastern United States. Copeia 1980:18–29.

Freeman, D. C., J. H. Graham, and J. M. Emlen. 1993. Developmental stability in plants: symmetries, stress and epigenesis. Genetica 89:97–119.

Freeman, D. C., J. H. Graham, D. W. Byrd, E. D. McArthur, and T. W. Turner. 1995. Narrow hybrid zone between two subspecies of big sagebrush, *Artemisia tridentata* (Asteraceae). III. Developmental instability. Am. J. Bot. 82:1144–1152.

Gavrilets, S., and A. Hasting. 1994. A quantitative-genetic model for selection on developmental noise. Evolution 48:1478–1486.

Graham, J. H. 1992. Genomic coadaptation and developmental stability in hybrid zones. Acta Zool. Fenn. 191:121–131.

Graham, J. H., and J. D. Felley. 1985. Genomic coadapatation and developmental stability within introgressed population of *Enneacanthus gloriosus* and *E. obesus* (Pisces, Centrarchidae). Evolution 39:104–114.

Jasieski, M., F. J. Ayala, and F. A. Bazzaz. 1997. Phenotypic plasticity and similarity of DNA among genotypes of an annual plant. Heredity 78:176–181.

Kahn, T. L., N. C. Ellstrand, and M. L. Arpaia. 1991. Current research on cherimoya cultivars and flowering behavior in California. Fruit Gardener June:8–11.

Leamy, L. 1993. Morphological integration of fluctuating asymmetry in the mouse mandible. Genetica 89:139–153.

———. 1997. Is developmental stability heritable? J. Evol. Biol. 10:21–29.

Leamy, L. J., E. J. Routman, and J. M. Cheverud. 1997. A search for quantitative trait loci affecting assymmetry of mandibular characters in mice. Evolution 51:957–969.

Leary, R. F., and F. W. Allendorf. 1989. Fluctuating asymmetry as an indicator of stress in conservation biology. Trends Ecol. Evol. 4:214–217.

Leary, R. F., F. W. Allendorf, and K. L. Knudsen. 1983. Developmental stability and enzyme heterozygosity in rainbow trout. Nature 301:71–72.

———. 1985. Developmental instability as an indicator of reduced genetic variation in hatchery trout. Trans. Am. Fish. Soc. 114: 230–235.

———. 1993. Null alleles at two lactate dehydrogenase loci in rainbow trout are associated with decreased developmental stability. Genetica 89:3–13.

Lerner, I. M. 1954. Genetic homeostasis. Oliver and Boyd, Edinburgh.

Leung, B., and M. R. Forbes. 1997. Modelling fluctuating asymmetry in relation to stress and fitness. Oikos 78:397–405.

Levin, D. A. 1970. Developmental instability in species and hybrids of *Liatris*. Evolution 24:613–624.

Manly, S. A. M., and F. T. Ledig. 1979. Photosynthesis in black and red spruce and their hybrid derivatives: ecological isolation and hybrid adaptative inferiority. Can. J. Bot. 57:305–314.

Markow, T. A., ed. 1994. Developmental instability: its origins and evolutionary implications. Kluwer, Dordrecht, The Netherlands.

Markow, T. A., and G. M. Clarke. 1997. Meta-analysis of the heritability of developmental stability: a giant step backward. J. Evol. Biol. 10:31–37.

Mather, K. 1953. Genetic control of stability in development. Heredity 7:297–336.

McKenzie, J. A., and G. M. Clarke. 1988. Diazinon resistance, fluctuating asymmetry and fitness in the Australian sheep blowfly, *Lucilia cuprina*. Genetics 120:213–220.

Messier, S., and J. B. Mitton. 1996. Heterozygosity at the malate dehydrogenase locus and developmental homeostasis in *Apis melifera*. Heredity 76:616–622.

Mitton, J. B. 1978. Relationship between heterozygosity and variation of morphological characters in natural populations. Nature 273:661–662.

———. 1993. Enzyme heterozygosity, metabolism, and developmental stability. Genetica 89:47–65.

Mitton, J. B., and M. C. Grant. 1984. Associations among protein heterozygosity, growth rate, and developmental homeostasis. Annu. Rev. Ecol. Syst. 15:479–499.

Møller, A. P. 1992. Female preference for symmetrical male sexual ornaments. Nature 357:238–240.

———. 1995. Bumblebee preference for symmetrical flowers. Proc. Natl. Acad. Sci. USA 92:2288–2292.

———. 1996. Developmental stability of flowers, embryo abortion, and developmental selection in plants. Proc. R. Soc. Lond. B Biol. Sci. 263:53–56.

Møller, A. P., and M. Eriksson. 1994. Patterns of fluctuating asymmetry in flowers: implications for sexual selection in plants. J. Evol. Biol. 7:97–113.

———. 1995. Pollinator preference for symmetrical flowers and sexual selection in plants. Oikos 73:15–22.

Møller, A. P., and A. Pomiankowski. 1993. Fluctuating asymmetry and sexual selection. Genetica 89:267–279.

Møller, A. P., and J. P. Swaddle. 1997. Asymmetry, developmental stability, and evolution. Oxford Univ. Press, New York.

Møller, A. P., and R. Thornhill. 1997. A meta-analysis of the heritability of developmental stability. J. Evol. Biol. 10:1–16.

Monedero, J. L., D. Chavarrías, and C. López-Fanjul. 1997. The lack of mutational variance for fluctuating and directional asymmetry in *Drosophila melanogaster*. Proc. R. Soc. Lond. B Biol. Sci. 264:233–237.

Mousseau, T. A., and D. A. Roff. 1987. Natural selection and the heritability of fitness. Heredity 59:181–197.

Palmer, A. R. 1994. Fluctuating asymmetry analyses: a primer. Pp. 335–364 *in* T. A. Markow, (ed.) Developmental instability: its origins and evolutionary implications. Kluwer, Dordrecht, The Netherlands.

———. 1996. Waltzing with asymmetry. BioScience 46:518–530.
Palmer, A. R., and C. Strobeck. 1986. Fluctuating asymmetry: measurement, analysis, patterns. Annu. Rev. Ecol. Syst. 17:391–421.
———. 1997. Fluctuating asymmetry and developmental stability: heritability of observable variation vs. heritability of inferred cause. J. Evol. Biol. 10:39–49.
Parson, P. A. 1961. Fly size, emergence time and sternopleural chaeta number in *Drosophila*. Heredity 16:455–473.
———. 1990. Fluctuating asymmetry: an epigenetic measure of stress. Biol. Rev. Camb. Philos. Soc. 65:131–145.
Paxman, G. J. 1956. Differentiation and stability in the development of *Nicotiana rustica*. Ann. Bot. 20:331–347.
Perfectti, F. 1995. Estudio de marcadores genéticos en chirimoyo y su aplicación a la identificación varietal. Evaluación de los recursos genéticos. Ph.D. diss., University of Granada, Granada, Spain.
Perfectti, F., and L. Pascual. 1998a. Characterization of cherimoya germplasm by isozyme markers. Fruit Var. J. 52:53–62.
———. 1998b. Genetic linkage of isozyme loci in *Annona cherimola*. Hereditas 128:87–90.
Popenoe, W. 1921. The native home of the cherimoya. J. Hered. 12:331–337.
SAS Institute. 1989. SAS/STAT user's guide. Vers. 6. SAS Institute, Inc., Cary, NC.
Scheiner, S. M., R. L. Caplan, and R. F. Lyman. 1991. The genetics of phenotypic plasticity. III. Genetic correlations and fluctuating asymmetries. J. Evol. Biol. 4:51–68.
Schroeder, C. A. 1945. Cherimoya culture in California. Univ. of California Dept. of Horticulture, Mimeographed Circular S. 15: 1–6.
Sherry, R. A., and E. M. Lord. 1996a. Developmental stability in leaves of *Clarkia tembloriensis* (Onagraceae) as related to population outcrossing rates and heterozygosity. Evolution 50:80–91.
———. 1996b. Developmental stability in flowers of *Clarkia tembloriensis* (Onagraceae). J. Evol. Biol. 9:911–930.
Simons, A. M., and M. O. Johnston. 1997. Developmental instability as a bet-hedging strategy. Oikos 80:401–406.
Sokal, R. S., and F. J. Rolf. 1981. Biometry. 2d ed. Freeman, New York.
Strauss, S. H. 1987. Heterozygosity and developmental stability under inbreeding and crossbreeding in *Pinus attenuata*. Evolution 41:331–339.
Swaddle, J. P. 1997. On the heritability of developmental stability. J. Evol. Biol. 10:57–61.
Swaddle, J. P., and I. C. Cuthill. 1994. Preference for symmetric males by female zebra finches. Nature 367:165–166.
Swaddle, J. P., M. S. Witter, and I. C. Cuthill. (1994). The analysis of fluctuating asymmetry. Anim. Behav. 48:986–989.
Thoday, J. M. 1955. Balance, heterozygosity and developmental stability. Cold Spring Harbor Symp. Quant. Biol. 20:318–326.
———. 1958. Homeostasis in a selection experiment. Heredity 12: 401–414.
Thomson, P. H. 1970. The cherimoya in California. California Rare Fruit Growers Yearb 2:20–34.
Valentine, D. W., and M. E. Soulé. 1973. Effects of pp′-DDT on developmental stability of pectoral fin rays in the grunion, *Leuresthes tennuis*. Fish. Bull. 71:921–926.
Van Valen, L. 1962. A study of fluctuating asymmetry. Evolution 16:125–142.
Venkataratnam, L., and G. Satyanarayanaswamy. 1958. Studies on genetic variability in *Annona squamosa* L. Indian J. Hortic. 15: 228–238.
Watson, P. J., and R. Thornhill. 1994. Fluctuating asymmetry and sexual selection. Trends Ecol. Evol. 9:21–25.
Whitlock, M. C., and K. Fowler. 1997. The instability of studies of instability. J. Evol. Biol. 10:63–67.
Winn, A. A. 1996. The contribution of programmed developmental change and phenotypic plasticity to within-individual variation in leaf traits in *Dicerandra linearifolia*. J. Evol. Biol. 9:737–752.
Xie, C. Y., and P. Knowles. 1992. Association between allozyme phenotypes and soil nutrients in natural population of Jack pine (*Pinus banksiana*). Biochem. Syst. Ecol. 20:179–185.
Yezerinac, S. M., S. C. Lougheed, and P. Handford. 1992. Measurement error and morphometric studies: statistical power and observer experience. Syst. Biol. 41:471–482.
Zakharov, V. M. 1989. Future prospects for population phenogenetics. Sov. Sci. Rev. F. Physiol. Gen. Biol. 4:1–79.

Corresponding Editor: T. Markow