

A trade-off between early growth rate and fluctuating asymmetry in Brazilian boys

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Abstract

Background: Right–left discrepancies in normally symmetrical traits are assumed to result from inability of the individual to buffer environmental and genetic stresses. Fluctuating asymmetry (FA) may therefore signal the quality of an individual to potential mates, or to parents during early life. FA could signal the heritable ability to buffer stress—the ‘good genes’ hypothesis. Alternatively, FA could signal the degree of within-lifetime exposure to stress, in particular during specific sensitive periods of development—the ‘good development’ hypothesis.

Aim: We tested the hypotheses that FA at 9 years of age is positively related to (a) fetal growth rate, (b) early infant growth rate, and (c) total post-natal growth rate.

Methods: FA, weight and height were measured in a sample of 172 boys aged 9 years from Pelotas, Brazil, who had previous measurements of weight and height at birth and 6 months.

Results: Fetal growth was not related to FA, however FA was positively related to total weight gain after birth ($p < 0.05$). This association could be broadly attributed to weight gain in the first 6 months of post-natal life ($p = 0.075$). Those currently obese had significantly greater FA than those non-obese ($p < 0.05$).

Conclusions: Our results support the ‘good development’ hypothesis, and suggest that growth rate during an early post-natal critical window, previously linked to numerous health outcomes, also has long-term effects on FA.

Keywords: *Fluctuating asymmetry, signalling, growth, fitness*

Introduction

Fluctuating asymmetry (FA) comprises small random deviations from perfect bilateral symmetry (Van Valen 1962). Right–left discrepancies in normally symmetrical traits

have been assumed to result from inability of the individual to buffer environmental and/or genetic stresses (Palmer and Strobeck 1986; Parson 1990). From this theoretical perspective, FA might therefore act either as a signal of an individual's competence at buffering stress, or as a signal of the extent of exposure to stress, and hence reflect fitness. How such associations could develop remains poorly understood, and may differ between species.

The hypothesis that FA acts as a signal of organism viability and fitness is supported by a number of studies. In various species FA has been linked to differential male fecundity, with females preferring males with lower FA (McLachlan and Cant 1995; Simmons 1995; Møller and Thornhill 1998). Increased FA has been shown to reduce male fecundity either through the mechanism of intrasexual competition for mates, or through the mechanism of mate choice by the opposite sex (Liggett et al. 1993; Arcese 1994; Rintamaki et al. 1997). In humans, FA was found to be negatively associated with a proxy index of fecundity (Thornhill and Gangestad 1994). Male FA was associated with increased morbidity and a decreased number of offspring in Belize (Waynforth 1998). FA has also been linked to various indices of sexual functionality (Gangestad et al. 1994; Manning et al. 1998). These studies suggest that human sexual selection is influenced by FA, and in non-human species even small asymmetries may be associated with a reduction in mating opportunities (Thornhill 1992a, 1992b). However, the issue remains controversial and two recent studies of mammals failed to relate FA in male secondary sexual traits to fitness (Côté and Festa-Bianchet 2001; Kruuk et al. 2003). The evidence in favour of possible underlying mechanisms (see below) is also subject to continued debate.

Two hypotheses have been proposed to account for an association between FA and fitness. First, genetic factors are assumed to influence the ability to buffer against environmental stress. There is evidence that FA is partly heritable with relatives resembling each other in the magnitude of their asymmetries, rather than the direction (Van Valen 1962; Møller and Thornhill 1997). Hence, it has been proposed that FA may signal 'good genes', i.e. the heritable capacity to buffer environmental or genetic stress (Palmer and Strobeck 1986; Parson 1990). In humans, for example, FA is increased in Down's syndrome (Townsend 1983). However, several recent studies in animals have failed to show heritability in traits that are sexually selected (Côté and Festa-Bianchet 2001; Kruuk et al. 2003). More generally, a meta-analysis of published studies of animals and plants examining the correlation between indices of fitness and heterozygosity (assumed to benefit fitness) concluded that these traits tended to be only weakly related (Britten 1996), although few of the studies in this review addressed FA. Thus, the genetic basis of any association between fitness and FA remains uncertain.

Where traits do not have heritability of FA, an alternative mechanism linking FA and fitness may be relevant. In non-human animal species, a wide range of environmental stresses have been associated with FA, including unusual temperature, noise, high population density and nutritional stress (Gest et al. 1986; Kowner 2001). However, other studies have failed to find such associations (Bjorksten et al. 2000, 2001; Vollestad and Hindar 2001; Woods et al. 2002). In humans, the evidence is likewise inconsistent. FA has been linked to environmental stresses such as parasitism (Bailit et al. 1970) and exposure to alcohol or tobacco smoke *in utero* (Kieser 1992; Wilber et al. 1993; Kieser and Groeneveld 1994). However, whereas some studies have associated FA with poor living conditions (Doyle and Johnston 1977; Harris and Nweeia 1980), others have not (Black 1980; Flinn et al. 1999).

Increasingly, it is appreciated there are major methodological difficulties in researching the effect of stress on fitness, and also that the interaction between genetic and environmental factors affecting morphological development is complex (Lens et al. 2002).

For example, it has been suggested that high levels of environmental stress might lead to developmentally unstable individuals being selected out of the gene pool, thus reducing associations between fitness and stress (Hendrickx et al. 2003). Another problem is that even in laboratory experiments, it may be hard to control or predict environmental variability (Kristensen et al. 2003). A third problem concerns the use of variability in a biological trait, rather than direct measurement of an external environmental factor, as the marker of stress. It is possible that underlying factors might exert common effects on both the trait and FA, thus reducing the ability to detect an effect of stress on FA. This issue is addressed further in the discussion.

The notion that within-lifetime stress increases FA introduces the concept of trade-offs, which are a central feature in life history theory (Stearns 1992). Trade-offs are predicted where resources are limited, such that their allocation to one end precludes their allocation elsewhere. In many species of animal, for example, growth during development is subject to trade-offs between the quantity and quality of tissues, and rapid growth is often achieved at the expense of cellular differentiation (Arendt 1997). Between-species variation in intrinsic growth rate can therefore be attributed to the relative importance of size vs. maturation (Arendt 1997). Within species, energy allocation is also critical to growth rate, and developmental quality has been proposed to be compromised when the relative allocation of energy to growth is increased (Arendt 1997). Consistent with this hypothesis, several studies have associated energy stress with FA (e.g. Wilson and Manning 1996).

In humans, many studies appear to show stronger associations between FA and stress where the environmental exposure occurs in early life. Medical research is likewise increasingly focusing on the hypothesis that early-life exposure to environmental stresses is associated with subsequent adverse outcome. Such associations have been termed 'programming', whereby exposure to an environmental stress during a sensitive period or 'critical window' in early life exerts long-term effects on the organism's phenotype (Lucas 1991). FA may be one such outcome that is programmed during early life, and hence act as a signal of the organism's general experience during this period. Initial research on programming reported associations between reduced fetal growth and later diseases in both animals and humans (Barker 1998; Waterland and Garza 1999). However, more recent work suggests that compensatory growth *following* fetal growth retardation may be most strongly predictive of later phenotype. In a wide variety of organisms, compensatory growth has been linked to adverse phenotype in later life (Metcalf and Monaghan 2001). In humans, early post-natal growth appears to programme later outcome across the entire birth weight range, with the most deleterious effects occurring in those who have increased most in size between birth and follow-up (Lucas et al. 1999).

The 'good genes' hypothesis predicts a negative relationship between FA status and size. However, the extra energy requirements of compensatory growth are predicted to inflict a cost on symmetry, giving a positive relationship between growth rate and FA. This scenario generates the hypothesis that FA could signal phenotypic quality, 'good development' rather than genetic constitution, by reflecting whether or not deleterious compensatory growth has occurred during a critical period of physiological development.

We addressed the 'good development' model by testing the hypothesis that growth rate in fetal life, infancy, or total post-natal life is positively associated with FA at 9 years. Growth was selected as the stressor because it has previously been associated with diverse health outcomes. FA was selected as the index of developmental stability because it has previously been shown to vary in relation to growth in studies of humans (Wilson and Manning 1996). We addressed the 'good genes' model by testing the hypothesis that FA is negatively associated with current size.

Methods

Subjects

The investigation was carried out in Pelotas, southern Brazil, where all children born during 1993 ($n=5304$) participated in a longitudinal birth cohort study. In this cohort, mean maternal schooling was 6.7 (SD 3.6) years, with only 2.5% of all mothers never having attended school. Mean paternal schooling was 6.8 (SD 3.5) years, with only 2.6% of all fathers never having attended school. Mean family income at birth was 4.3 minimum wages, equivalent to US\$ 350 per month. However, median family income was only 2.5 minimum wages, equivalent to US\$ 200 per month, while 16.7% of all families earned less than 1 minimum wage per month, equivalent to less than US\$ 90. According to information on ethnicity obtained by maternal interview, 78% of children had two white parents, 17% two black parents, and 11% one white and one black parent. Measurements of infant weight were made at birth and 6 months.

From this cohort, a sub-sample of 172 children was investigated at 9 years. Four groups of children were selected. Initially, the sample was divided into those with or without low birth weight. In each of these two groups, children were further selected according to whether they had above or below the median rate of weight gain between birth and 4 years of age. The sample therefore embraced a wide range of both birth weight and weight gain in infancy. A total of 174 children were invited to participate. The refusal rate was 1% ($n=2$). Participants were visited at home or at school for a 20-min measurement session. Ethical approval was given by the Federal University of Pelotas. Written informed consent was provided by the parent, and verbal assent by the child.

Measurements

Measurements were made of height and weight, with precision of 0.23 cm and 0.07 kg, respectively. FA was measured at seven sites: the length of all four fingers, ear height, and length and width of the foot. Finger length was defined as the distance between the tip of the finger and the centre of the basal skin crease (ventral surface) proximal to the palm. Ear height was defined as the maximal distance from the anterior to the posterior tip of the pinna. Foot width was measured at the widest point. Foot length was measured from the back of the heel to the tip of the big toe. All measurements were made with electronic calipers reading to 0.01 mm, except for foot length where a metal ruler was used in combination with a sliding set-square, reading to 0.5 mm. All data on weight and height were converted into standard deviation score (SDS) format, using current UK reference data due to their extending back to 31 weeks' gestation that therefore take into account gestational age (Freeman et al. 1995).

Measurements of trait size were repeated if the first two measurements differed by more than 0.5 mm, until two readings within this range were obtained. The data on repeat measurements were used to verify whether there was a significant bias between first and second measurements. Repeatability was calculated using the intra-class correlation coefficient, and the ratio of between-subject to within-subject variance (measurement error) using repeated measures ANOVA tests (Trivers et al. 1999). Each trait was tested for normality and rejected if the distribution differed significantly from normal.

The average of the two readings was calculated for each side of the body. The difference between sides was then calculated for each trait, i.e. absolute FA. However, absolute FA does not take into account variability between individuals in body size, and hence trait size.

In deriving a composite index, the mean value will be disproportionately influenced by traits that are larger and hence have larger absolute asymmetries. However, if absolute FA is adjusted by dividing by the individual's trait size, there is a risk that the derived variable may control for the variability in condition that is being investigated (Palmer 1994).

In the present study, the following approach was adopted. First, absolute FA was calculated as the difference between sides, with all asymmetries converted to positive values. The relationship between absolute FA and trait size was then explored in each trait using correlation analysis. For a correlation between two traits of r , the proportion of variation in one trait that can be explained by the other is given by:

$$\text{Proportion of variability (\%)} = (1 - \sqrt{1 - r^2}) \times 100 \quad (1)$$

This equation was used to assess the extent to which trait size accounted for variation in absolute FA.

Second, relative FA was calculated as (difference/average trait size) \times 100, where the average trait size included both sides of the body for each trait, and again all values were converted to positive. Absolute and relative FA of individual traits then had half normal distributions. For those traits with signed FAs with normal distributions, the FA values were then summed to give composite indices, absolute and relative FA. These values were then transformed ($\log x + 1$).

Statistics

Multiple regression analyses were used to investigate the association between growth rate and FA. Models were constructed to investigate the effect of birthweight SDS alone, and then the effect of change in SDS birth to 9 years, or birth to 6 months, adjusting for birthweight SDS. Consistency between individual traits in relative FA was assessed by correlation analysis.

Results

A description of age, anthropometry and weight gain in the sample is shown in Table I. The sample was broadly representative of the entire 1993 cohort according to social criteria. There was a wide range of family income, with 16% of the boys from a family that earned less than 1 minimum wage per month, and 7.7% from a family earning ≥ 10 minimum wages per month. Maternal schooling was 6.8 (SD 3.3) years, similar to that of the whole cohort. Duration of maternal education was as follows: ≥ 9 years, 23.4%; 5–8 years, 57.0%; 1–4 years, 18.6%; never studied, 1.2%. Mean paternal schooling was 6.9 (SD 3.4) years, again representative of the cohort, with 2.5% of fathers never having studied. However, reflecting our study design, the sample of infants in the present study had lower mean (SD) birth weight than the total birth cohort (2.82 vs. 3.15 kg). The raw symmetry data are given in Table II.

The ratio of between-subject to within-subject variance was >100 ($p < 0.0001$) for all traits on both sides of the body, indicating that measurement error had negligible impact on subsequent analyses. Two traits (foot length and foot width) had mean FA significantly different from zero, suggesting directional asymmetry, and were therefore excluded

Table I. Description of the sample.

	Mean	SD	Range
<i>Infant</i>			
Birth weight (kg)	2.83	0.68	1.40–4.69
Birth length (cm)	47.5	3.1	38.0–54.0
Gestational age (weeks)	37.8	1.9	31.7–41.2
Post-natal weight gain (kg)	4.87	0.98	1.55–7.62
<i>Mother</i>			
Age (years)	26.5	6.7	14–44
Weight (kg)	56.6	10.9	35–113
Height (m)	1.59	0.07	1.41–1.80
BMI (kg m ⁻²)	22.3	3.9	14.5–39.6
<i>Child at 9 years</i>			
Age (years)	9.5	0.3	9.0–10.1
Weight (kg)	31.7	8.0	18.1–61.8
Height (m)	1.35	0.07	1.15–1.55
BMI (kg m ⁻²)	16.4	3.1	12.3–28.1
<i>Weight SDS</i>			
Birth	-0.64	1.21	-3.80–2.76
6 months	-0.21	1.45	-5.76–3.23
9 years	0.34	1.35	-3.69–3.38
Δ birth to 6 months	0.44	1.37	-5.11–3.68
Δ 6 months to 9 years	0.54	1.26	-2.53–4.94

Δ = change.

Table II. Fluctuating asymmetry (FA) measurements.

	Trait size		Average FA		Positive FA		Positive relative FA	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD
First finger	56.4	4.2	0.05	1.34	1.04	0.83	1.85	1.47
Second finger	63.4	4.4	0.12	1.00	0.77	0.64	1.22	1.01
Third finger	58.8	4.2	0.06	1.25	0.91	0.86	1.54	1.47
Fourth finger	46.1	3.8	-0.10	1.43	1.13	0.87	2.46	1.88
Ear	59.5	3.9	-0.04	1.38	1.06	0.88	1.79	1.48
Foot length	212.2	13.7	-0.50	2.80	2.15	1.76	1.01	0.83
Foot width	84.6	6.0	-0.64	1.73	1.44	1.15	1.70	1.36

Values for average trait size, FA and positive FA are given in millimetres. Relative FA calculated as (difference/population average) × 100%.

from analyses. The remaining five traits were combined to give composite indices, absolute and relative FA5.

Correlations between absolute FA and trait size were <±0.1 (NS) for five of the seven traits, with *r*-values of 0.18 (*p* < 0.02) for the fourth finger and 0.15 (*p* = 0.057) for foot width. Insertion of these *r*-values in Equation 1 showed that for all traits the percentage of variation in FA that could be attributed to variability in trait size was <2%.

There was minimal consistency in FA between traits. Values for the correlations of relative FA between different traits are presented in Table III. Only two such correlations achieved significance. Both these correlations were modest in magnitude (*r* ≤ 0.20) and significance (*p* < 0.05).

Table III. Correlation coefficients for the consistency of fluctuating asymmetry (FA) between traits.

	First finger	Second finger	Third finger	Fourth finger	Ear	Foot length
Second finger	0.04					
Third finger	-0.08	0.06				
Fourth finger	-0.07	-0.10	0.17*			
Ear	0.02	-0.20*	-0.03	-0.02		
Foot length	-0.06	-0.06	0.10	0.02	0.06	
Foot width	-0.00	0.04	-0.03	-0.01	0.05	0.11

* $p < 0.05$.

Table IV. Fetal, infant and childhood growth and later fluctuating asymmetry (FA5).

	Absolute FA5			Relative FA5		
	Coefficient	SE	p -value	Coefficient	SE	p -value
<i>Model 1</i>						
Constant	1.530	0.032	<0.001	2.118	0.033	<0.001
Birth weight SDS	0.006	0.023	0.78	0.008	0.024	0.74
<i>Model 2</i>						
Constant	1.505	0.034	<0.001	2.092	0.034	<0.001
Birth weight SDS	0.037	0.027	0.18	0.037	0.028	0.18
Δ weight SDS 0–9 years	0.048	0.022	0.032	0.047	0.023	0.038
<i>Model 3</i>						
Constant	1.520	0.032	<0.001	2.107	0.033	<0.001
Birth weight SDS	0.016	0.025	0.52	0.017	0.026	0.51
Δ weight SDS 0–6 months	0.039	0.022	0.075	0.040	0.022	0.072

SDS = standard deviation score, Δ = change.

Multiple regression models are given in Table IV. There was no significant relationship between birth weight SDS and either absolute or relative FA5. After taking into account birth weight SDS, change in weight SDS between birth and 9 years was significantly positively associated with relative FA5 (absolute FA5: $p = 0.032$; relative FA5: $p = 0.038$). After taking into account birth weight SDS, change in weight SDS between birth and 6 months was also positively associated with relative FA5, however the association was just outside the cut-off for statistical significance (absolute FA5: $p = 0.075$; relative FA5: $p = 0.072$). After adjusting for birth weight, those obese had significantly greater FA than those non-obese ($p < 0.05$), and significantly greater rates of weight gain from birth to 6 months ($p < 0.001$) and in childhood ($p < 0.001$).

Discussion

This study considered whether FA acts as a signal of differential growth in humans, focusing on two separate hypotheses, the ‘good genes’ and the ‘good development’ hypotheses. These hypotheses both relate FA to adaptive capacity. Whereas the ‘good genes’ hypothesis relates

to differential fitness acquired over generations, the ‘good development’ hypothesis relates to environmental effects experienced within the lifetime of the organism.

Our study showed that faster growth rate in post-natal life, but not in fetal life, was associated with increased asymmetry at 9 years, with this relationship broadly accounted for by growth occurring in the first 6 months after birth. In contrast, there was no significant correlation between FA and size at 9 years. Our results therefore favour the ‘good development’ hypothesis, and imply that symmetry status at 9 years acts as a signal of the rate of growth in an early window of development. This critical window has also previously been shown to be important for a wide range of diseases.

There were wide ranges of variability in size and growth rates. The fact that the overall association between FA and growth rate could be attributed to weight gain in early infancy may be due to the fact that growth consumes a large proportion of energy intake in early life (33% at 6 weeks of age), but a very small proportion (<4%) from late infancy onwards (Wells and Davies 1998). Thus, the slow mean rate of childhood growth has less chance of being costly for symmetry. However those currently obese, who have had the maximal weight gain over the entire period of growth, also had significantly higher FA than those non-obese. Thus, while FA at 9 years appears to signal early growth rate, FA also reflects growth rate in subsequent periods too. This is consistent with the notion of asymmetry accumulating in successive growth periods, but with the effect of infancy persisting because of the high cost of growth characteristic of this time period.

Our study did not find consistency within individuals in FA between traits. This may relate to the fact that traits themselves vary in their complexity. For example, each digit length reflects the contribution of several individual bones, each of which may differ in length between the two sides of the body (Livshits et al. 1998). From a statistical perspective, complex traits are predicted to have lower FA, since random left–right fluctuations in, for example, the individual bone lengths tend to cancel out when symmetry of a complete digit is considered (Livshits et al. 1998). These authors likewise found that within the human hand, the majority of FA measurements showed no within-individual consistency. Thus the choice of which traits to measure will influence the ability to detect FA, and to provide a more reliable index of FA it is conventional to measure a number of traits, as undertaken in the present study.

Although the notion that FA may reflect quality is intuitively attractive, such an association is not the only mechanism which might account for preference for symmetrical individuals. Using simple artificial neural networks, Johnstone (2002) demonstrated that preference for symmetry in specific traits might evolve as a by-product of selection for mate recognition. However, our study linking FA with early growth rate, a variable previously shown to be associated with fitness, is not consistent with that model. It remains to be seen whether the association we found applies to traits such as facial symmetry that are more fundamental to recognition in our species.

Instead, our findings are consistent with several empirical studies of other species. In birds, early nutritional stress has been shown both to increase FA in feather development (Swaddle and Witter 1994), and to reduce subsequent song quality (Nowicki et al. 2002; Buchanan et al. 2003). This effect on song could be attributed to alterations in the region of the brain associated with song repertoire (Nowicki et al. 2000). These associations appear to develop because asymmetry induced during sensitive developmental windows is preserved into later life. The rapid growth that occurs in specific organs or traits during such critical periods may reduce the opportunity for subsequent negative feedback or compensatory growth (Møller and Swaddle 1997). Thus, we suggest that FA may signal the quality

of development in specific critical windows if these windows are characterized by maximal growth rates, and species may vary in the mechanism by which symmetry, and the preference for it, develops.

Our results are also consistent with data from rats, in which growth rate was positively associated with FA. Leamy and Atchley (1985) predicted that rat strains selected for either fast or slow growth would have increased FA relative to controls, since increased homozygosity (resulting from the selection) is generally associated with increased FA (Arendt 1997). However, they found instead that fast growth increased FA and slow growth reduced it, clearly implicating growth rate as a stress on symmetry. More generally, our results are consistent with the concept of a trade-off between growth rate and developmental quality proposed by Arendt (1997). In general, trade-offs are difficult to demonstrate, as, whether due to common genetic effects or generic phenotypic quality, individuals with high quality in one trait often demonstrate high quality in other traits too. However, both symmetry and growth require energy, hence for any given quality of individual, an increased allocation of energy to one trait is predicted to exert a cost on the other.

Consistent with this approach, previous studies in humans have suggested the existence of a trade-off between energy requirements for growth and developmental stability. FA has been found to increase during periods when growth rate is higher (Wilson and Manning 1996), and in males to be positively correlated with resting metabolic rate (Manning et al. 1997). Significantly, most growth variability manifests in early life, when differences between individuals are large. Thus, our findings are consistent with other studies which likewise link poor health outcome with growth patterns: insulin resistance, for example, is associated with the accumulation of excess weight at any point in childhood, but is most strongly predicted by growth in the immediate post-natal period (Singhal et al. 2003). More generally, faster infant growth has been linked to an increased risk of the metabolic syndrome in later life (Singhal and Lucas 2004), especially in those born small (compensatory growth). In mice, faster infant growth has also been linked to reduced longevity (Ozanne and Hales 2004). Since early growth appears to be costly for symmetry as well as health, symmetry status may signal fundamental information about fitness and longevity to potential mates.

A relationship between early growth rate and later FA could be amplified during adolescence. Early deficits in growth rate may be partly recovered through a longer period of adolescent growth (Lazar et al. 2003). More rapid 'catch-up' growth during adolescence might lead to increased FA by the time adulthood is attained, and amplify the signal of 'poor development' during early critical windows to potential mates. Consistent with this hypothesis, FA has been found to increase during adolescence (Wilson and Manning 1996).

It could be argued that current body size in humans provides ample information about early growth rate, such that FA would be a superfluous signal. Larger children could be assumed to be healthier and hence fitter. However, it is clear from the longitudinal data in this study that boys can achieve the same size at 9 years through markedly different growth trajectories, with such differential growth patterns known to influence health status during adulthood and hence probable fitness. Early compensatory growth promotes short-term survival in those born small (Victoria et al. 2001), but FA subsequently signals the cost in fitness that has been paid to achieve this end. The fact that it is infant growth, rather than fetal growth, which is associated with increased FA suggests that it is not being small, but *compensating* for such small size (Ong et al. 2000), that increases both the risk of disease and FA.

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Résumé. *Arrière plan:* On assume que les différences droite-gauche des traits normalement symétriques, sont le résultat de l'inaptitude des individus à amortir les stress environnementaux et génétiques. L'asymétrie fluctuante (AF) peut alors signaler la qualité d'un individu à des conjoints potentiels ou à ses parents tôt dans la vie. L'AF pourrait indiquer l'aptitude héréditaire à gérer les stress – l'hypothèse des «bons gènes». Par ailleurs, l'AF pourrait indiquer le degré d'exposition au stress dans le courant de la vie, en particulier au cours de périodes particulièrement sensibles du développement – l'hypothèse du «bon développement».

But: On a testé l'hypothèse que l'AF à l'âge de neuf ans est positivement associée (a) au rythme de croissance foetale, (b) au rythme de croissance infantile et (c) au rythme de croissance post-natal.

Méthodes: L'AF, le poids et la stature ont été mesurés dans un échantillon de 172 garçons de Pelotas (Brésil) âgés de 9 ans, dont le poids et la stature avaient été préalablement mesurés à la naissance et à 6 mois.

Résultats: La croissance foetale n'est pas associée à l'AF bien que l'AF soit positivement liée au gain de poids total après la naissance ($p < 0,05$). Cette association pourrait en gros être attribuée au gain de poids au cours des six premiers mois de la vie post-natale ($p < 0,075$). L'AF est significativement plus élevée chez les obèses que chez les non obèses ($p < 0,05$).

Conclusion: Nos résultats sont en faveur de l'hypothèse du «bon développement» et suggèrent que le rythme de la croissance au cours d'une fenêtre critique du début de la période post-natale qui a antérieurement été mise en rapport avec de nombreuses questions de santé, aurait également un effet à long terme sur l'AF.

Zusammenfassung. *Hintergrund:* Es wird oft angenommen, dass Rechts-Links-Unterschiede bei normalerweise symmetrisch verteilten Merkmalen aus der Unfähigkeit des Individuums resultieren, umweltbedingte und genetische Belastungen abzufangen. Unterschiedliche Ausprägungen von Asymmetrie (fluktuiierende Asymmetrie, FA) könnten daher als Ausdruck für die Güte eines Individuums in den Augen potentieller Lebenspartner oder – während früher Lebensabschnitte – der Eltern angesehen werden. FA könnte ein Zeichen für die erbliche Fähigkeit sein, Belastungen abzufangen—die Hypothese von den 'guten Genen'. Andererseits könnte FA auch ein Zeichen für das Ausmaß von Belastungen sein, denen das Leben bisher ausgesetzt war, insbesondere während sensibler Entwicklungsphasen—die Hypothese von der 'guten Entwicklung'.

Ziel: Wir testeten die Hypothese, dass FA im Alter von 9 Jahren positiv mit (a) der fetalen Wachstumsrate, (b) mit der frühkindlichen Wachstumsrate, und (c) mit der gesamten postnatalen Wachstumsrate korreliert ist.

Methoden: FA, Gewicht und Körperhöhe wurden in einer Stichprobe von 172 9-jährigen Knaben aus Pelotas, Brasilien, bestimmt, von denen frühere Messungen von Gewicht und Körperlänge bei Geburt und im Alter von 6 Monaten vorlagen.

Ergebnisse: Fetales Wachstum war nicht mit FA korreliert, allerdings war FA positiv mit der Gewichtsentwicklung nach der Geburt korreliert ($p < 0,05$). Diese Beziehung könnte großenteils dem Gewichtszuwachs der ersten 6 Lebensmonate zugeordnet werden ($p = 0,075$). Diejenigen, die zum Zeitpunkt der Messung adipös waren, zeigten signifikant höhere FA als nicht-adipöse ($p < 0,05$).

Zusammenfassung: Unsere Ergebnisse stützen die Hypothese der 'guten Entwicklung', und legen nahe, dass die Wachstumsrate während eines frühen kritischen postnatalen Entwicklungsfensters, die schon früher mit zahlreichen Aspekten der gesundheitlichen Entwicklung in Beziehung gebracht wurde, auch einen Langzeiteffekt auf FA hat.

Resumen. *Antecedentes:* Se asume que las discrepancias derecha-izquierda en los rasgos normalmente simétricos son el resultado de la incapacidad de los individuos para tamponar el estrés ambiental y genético. La asimetría fluctuante (AF) puede por lo tanto indicar la calidad de un individuo a los cónyuges potenciales, o a los padres durante las etapas tempranas de la vida. La AF podía indicar la capacidad hereditaria para tamponar el estrés – la hipótesis de los “buenos genes”. Como alternativa, la AF podría indicar el grado de exposición al estrés a lo largo de la vida, en particular durante periodos especialmente sensibles del desarrollo – la hipótesis del “buen desarrollo”.

Objetivo: Comprobamos las hipótesis de que a los 9 años de edad la AF está positivamente relacionada con (a) la tasa de crecimiento fetal, (b) la tasa de crecimiento en la primera infancia, y (c) la tasa de crecimiento postnatal total.

Métodos: Se midieron la AF, el peso y la talla en una muestra de 172 chicos de 9 años de edad, residentes en Pelotas, Brasil, a los que se les había medido previamente el peso y la talla al nacimiento a los 6 meses de edad.

Resultados: El crecimiento fetal no estaba relacionado con la AF, sin embargo la AF estaba positivamente relacionada con el incremento total del peso después del nacimiento ($p < 0,05$). Esta asociación podría atribuirse globalmente al incremento del peso en los 6 primeros meses de vida postnatal ($p = 0,075$). Los niños actualmente obesos tenían una AF significativamente mayor que los no obesos ($p < 0,05$).

Conclusiones: Nuestros resultados apoyan la hipótesis del “buen desarrollo” y sugieren que la tasa de crecimiento durante una ventana crítica post-natal temprana, conectada previamente a los numerosos resultados de la salud, también tiene efectos a largo plazo en la AF.

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