Birth order and fluctuating asymmetry: a first look

Martin L. Lalumière^{1,2*}, Grant T. Harris¹ and Marnie E. Rice¹

¹*Mental Health Centre Penetanguishene, 500 Church Street, Penetanguishene, Ontario, Canada L9M 1G3*

²*Centre for Addiction and Mental Health, 250 College Street, Toronto, Ontario, Canada M5T 1R8*

We investigated the hypothesis that maternal immunoreactivity to male-specific features of the foetus can increase developmental instability. We predicted that the participants' number of older brothers would be positively related to the fluctuating asymmetry of ten bilateral morphological traits. The participants were 40 adult male psychiatric patients and 31 adult male hospital employees. Consistent with the hypothesis, the participants' number of older brothers—but not number of older sisters, younger brothers or younger sisters—was positively associated with fluctuating asymmetry. The patients had significantly larger fluctuating asymmetry scores and tended to have more older brothers than the employees, but the positive relationship between the number of older brothers and fluctuating asymmetry was observed in both groups.

Keywords: fluctuating asymmetry; birth order; maternal immunoreactivity

1. INTRODUCTION

Fluctuating asymmetry (FA) is a measure of the random fluctuations between the left and right sides of bilateral morphological traits. It represents 'imprecise expression of underlying developmental design due to developmental perturbations' (Gangestad & Thornhill 1997, p. 72) and is possibly the best measure of developmental instability (Møller & Swaddle 1997; Thornhill & Møller 1997). Low FA is associated with high phenotypic quality in several species, including humans (Møller 1990; Thornhill & Gangestad 1993; Malyon & Healy 1994; Manning *et al.* 1997).

In several species, low FA individuals grow faster and have higher fecundity and survival (Møller 1997). In humans, low FA men report more sexual partners, an earlier age at first intercourse and have more offspring than high FA men (Thornhill & Gangestad 1994; Waynforth 1998). Higher FA is related to schizophrenia (Mellor 1992), mental retardation, developmental delay (Naugler & Ludman 1996), birth prematurity (Livshits & Kobylansky 1991) and left-handedness (Yeo *et al.* 1993).

FA in non-human species is caused by environmental stressors such as pathogens, extreme temperature and pollutants and genomic stressors such as directional selection, mutations and homozygosity (reviewed by Parsons 1990; Møller & Swaddle 1997). Human asymmetry probably has similar causes (Livshits & Kobylansky 1991).

One class of environmental factors associated with higher FA are prenatal stressors. Smoking during pregnancy is associated with elevated levels of dental asymmetry in some groups of children (Kieser & Groeneveld 1994; Kieser *et al.* 1997). The presence of

infectious diseases in pregnant mothers is related to a higher body FA in newborns (Livshits *et al.* 1988). Patients with foetal alcohol syndrome have higher dermatoglyphic FA than controls (Wilber *et al.* 1993). A number of prenatal environmental stressors during pregnancy have been found to create dental asymmetry in the offspring of rats (e.g. Sciulli *et al.* 1979; Mooney *et al.* 1985).

Another prenatal factor that may increase FA is maternal immunoreactivity to male-specific features of the foetus. In an important paper, Gualtieri & Hicks (1985) argued the following ideas.

- (i) A mother is more likely to develop an immune response to a male foetus than to a female foetus because male foetuses are more antigenic to her.
- (ii) Maternal immunoreactivity to male foetuses can explain the observed male-biased sex ratio in foetal mortality, neurodevelopmental problems and obstetrical complications.
- (iii) Because of the 'memory' property of the immune system, each male foetus increases the probability and strength of such a maternal immune response and, therefore, the frequency and severity of selective male afflictions.
- (iv) Female foetuses, because they are much less likely to provoke a maternal immune response, have little effect on the probability and severity of selective afflictions of later offspring.

Evidence for male-specific maternal immunoreactivity has been reviewed by Gualtieri & Hicks (1985) and Blanchard & Klassen (1997). Of note, Vernier (1975) reported that the human placenta, which provides a functional barrier against maternal immunological reactions, is larger in male than in female foetuses. The placenta of the newborn male also weighs more if the preceding sibling is a male than if the preceding sibling is

*Author for correspondence (martin_lalumiere@camh.net).

a female. Finally, Vernier (1975) reported that the size of the placenta is positively related to the number of preceding male siblings (see also Warburton & Naylor 1971).

The male-specific maternal immunoreactivity theory is thus in part a theory of the relationship between birth order and deleterious developmental outcomes. Because FA is a measure of developmental instability, one could expect that increased maternal immunoreactivity (as a function of the number of older brothers) results in greater FA. Consistent with this notion, Bogle (1989) reported that parity (first, second or third pregnancy) was positively although non-significantly related to the FA of the a–b ridge count in a twin sample; however, she did not analyse the data according to the sex of proband or antecedent siblings.

In this paper, we investigate the relationship between the number of older brothers and FA in two different groups of adult males. It was predicted that the male FA scores would be positively and linearly related to their number of older brothers. The number of other types of siblings (older sisters, younger brothers and younger sisters) should not be related to FA. This is the first description and test of this hypothesis.

2. METHODS

(a) *Participants*

As part of a study on the neurodevelopmental stability of violent offenders and non-offenders, we gathered biographic and FA data from patients and employees at a maximum-security psychiatric institution. The patients were 38 men who had been charged with violent crimes (murder, sexual assault, assault and armed robbery) plus two who were civilly committed because of concerns about their potential for violence. Their mean age was 37.8 years (range 19–62 years). A review of their clinical files showed that 80% had not completed high school, 85% were single, 94% had no occupation or unskilled and semi-skilled occupations at the time of their arrest, 60% had a diagnosis of personality disorder, 58% a diagnosis of psychosis and 30% a diagnosis of substance abuse (patients could have more than one diagnosis). The employees were 31 men with a mean age of 38.8 years (range 14–65 years). Based on self-report, 6.5% had not completed high school and 29.0% were single.

(b) *Measures and procedures*

Sibling information was obtained from a standardized interview. The patients' clinical records were also reviewed. We collected information about the age, sex and genetic relatedness of each sibling. Only siblings genetically related to the participants through their mothers were included in the analyses. The Pearson correlation between the number of older brothers obtained from interviews and file reviews was 0.92. The corresponding values for older sisters, younger brothers and younger sisters were 0.94, 0.68 and 0.91, respectively. The results presented below are based on interview data but similar results were obtained from file data on the patients. All participants gave informed consent and the patients were paid \$5 for their participation.

FA was assessed with a digital caliper (Mitutoyo 500–138) measuring to the nearest 0.01 mm. Two trained researchers measured each participant. The left and right sides of ten bilateral traits (length of ears and third, fourth and fifth digits and

width of ears, hands, wrists, elbows, feet and ankles) were measured separately.

(c) *Manipulation of FA data*

The scores from both raters were first averaged. Signed and unsigned composite FA scores were then calculated for each participant, taking into account body size differences:

$$\text{signed FA} = \frac{\sum_{i=1}^{10} (L - R)/0.5(L + R)}{10}, \quad (1)$$

and

$$\text{unsigned FA} = \frac{\sum_{i=1}^{10} |L - R|/0.5(L + R)}{10}. \quad (2)$$

To avoid extreme scores due to a history of injury or large measurement error, an arbitrary maximum score of ($\pm$)0.10 for signed scores and 0.10 for unsigned scores was used for each trait (used for 6.1% of all values). Thus, the theoretical range of the FA composite scores was -0.10 to $+0.10$ for the signed scores and 0.00 to 0.10 for the unsigned scores.

The estimated reliability of the unsigned FA scores based on all ten features measured twice was 0.65 (Spearman–Brown formula; Anastasi 1988). This value is similar to those obtained by other investigators using similar measurement procedures (e.g. Furlow *et al.* 1997).

The mean of the signed composite scores across all participants was -0.00551 (95% confidence interval 0.00336). The minimum and maximum scores were -0.031 and $+0.032$. The skewness was 0.485 ($z=1.70$) and the kurtosis was -0.233 ($z=-0.41$). Thus, the signed composite scores were normally distributed but were significantly shifted to the left of the distribution, suggesting the presence of directional asymmetry (DA) favouring the right side.

Four of the ten features showed statistically significant right-biased DA (wrist, foot and ankle width and fourth finger length) and one showed statistically significant left-biased DA (elbow width). A signed composite FA score based on the five features that did not show DA (hand and ear width and ear, third and fifth finger length) was calculated for each participant. The mean was -0.00378 (95% CI ± 0.00493) and did not significantly deviate from zero; the distribution was normal. Its estimated reliability was 0.63.

The analyses were performed on both the unsigned FA composite score based on the ten features (FA₁₀) and on the unsigned FA composite score based on the five features that did not show DA (FA₅). The results presented below are for FA₅ and did not differ from the results obtained with FA₁₀.

3. RESULTS

A simultaneous multiple regression was conducted in which the dependent variable was FA₅ and the predictor variables were the number of older brothers, number of older sisters, number of younger brothers and number of younger sisters. This analysis allowed us to investigate the effect of the number of older brothers independent of the effect of the number of siblings of other categories and, indirectly, independent of family size. The results presented in table 1 show that only the number of older brothers significantly contributed to variance in FA₅. The

Table 1. Results of linear regression analysis and zero-order correlations for all participants (FA₅)

predictor	standardized beta (β)	Pearson r (parametric)	Spearman r (non-parametric)
number of older brothers	+ 0.276*	+ 0.305**	+ 0.267*
number of older sisters	+ 0.006	+ 0.125	+ 0.112
number of younger brothers	− 0.143	− 0.195	− 0.190
number of younger sisters	− 0.029	− 0.104	− 0.093

* $p < 0.05$ and ** $p < 0.01$ (two-tailed).

larger the number of older brothers a participant had, the larger his FA₅ value. The univariate parametric and non-parametric correlations shown in table 1 confirm these results.

The patients tended to have a larger number of older brothers (mean = 0.73 and 95% CI = ± 0.26) than the employees (mean = 0.58 and 95% CI = ± 0.32). The patients were also significantly more asymmetrical (mean = 0.0376 and 95% CI = ± 0.00035) than the employees (mean = 0.030 and 95% CI = ± 0.00034), raising the possibility that the relationship between the number of older brothers and FA is due to group membership. The multiple regression was repeated with group membership (patients versus employees) entered as a predictor. The results showed that the number of older brothers was still significantly related to FA₅ ($\beta = 0.246$ and $p < 0.05$). Group membership was the only other significant predictor ($\beta = 0.321$ and $p < 0.01$). The Pearson correlation between FA₅ and the number of older brothers was positive in both groups (patients = 0.309 and employees = 0.238). These results suggest that the relationship between the number of older brothers and FA₅ was present in both groups. Finally, the relationship between maternal age and FA₅ was -0.090 ($p > 0.45$).

4. DISCUSSION

FA, a measure of developmental instability, was related to the participants' number of older brothers (but not to the participants' number of siblings of other categories or to maternal age) in a group of psychiatric patients and in a group of 'ordinary' men. Although more studies are needed to confirm this result, the positive relationship between FA and number of older brothers in two different groups of men supports the hypothesis that maternal immunoreactivity to male foetuses increases developmental instability.

The male-specific maternal immunoreactivity hypothesis predicts that female FA should not be related (or much less related) to fraternal or sororal birth order. The male-specific maternal immunoreactivity hypothesis also suggests that, *ceteris paribus*, an unselected sample of males will show larger FA values than an unselected sample of females. Recent studies comparing male and female FA scores have produced mixed results (e.g. Micle & Kobylansky 1988; Bogle 1989; Thornhill & Gangestad 1994; Arrieta *et al.* 1995; Manning 1995; Wilson & Manning 1996; Yeo *et al.* 1997; Trivers *et al.* 1999).

Some measures of FA do not change with postnatal age (e.g. dermatoglyphics), while others (e.g. finger

length) seem to diminish with age in children and adolescents (Wilson & Manning 1996), perhaps reflecting the operation of feedback mechanisms during development (cf. Møller & Thornhill 1997). The measures of FA used in this study could potentially have been influenced by postnatal events such as injuries and infections. Can a postnatal mechanism account for the relationship between the number of older brothers and FA in males? Although it is difficult to imagine a mechanism that could explain birth order effects limited to older brothers, only studies using categories of siblings that differ in genetic relatedness (e.g. maternal versus paternal half brothers) will allow us to answer this question definitively. The study of dermatoglyphic ridges and patterns, which are formed *in utero* and are only influenced by factors operating prenatally, would allow a more direct test of the male-specific maternal immunoreactivity hypothesis.

It is interesting that a group of violent men with various psychiatric diagnoses scored higher on FA than ordinary men. Men with serious psychiatric disorders such as schizophrenia tend to show larger FA than controls (Markow & Wandler 1986; Mellor 1992). We and others have reported that men with a history of aggression or violence show signs of neurodevelopmental disturbances such as learning disability, neuromotor deficits, obstetrical complications and low IQ (Raine *et al.* 1994, 1996; Harris *et al.* 1999; reviewed in Moffitt 1993). It will be worthwhile investigating the relationship between a history of violence, serious psychiatric disorders and FA further.

We thank Ray Blanchard, James Cantor, Vern Quinsey and Michael Seto for useful suggestions on a previous version of this article.

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