

# Fluctuating asymmetry and general intelligence: No genetic or phenotypic association

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## Abstract

Fluctuating asymmetry (FA) is the non-pathological left–right asymmetry of body traits that are usually left–right symmetrical, such as eye breadths and elbow to wrist lengths in humans, but which can be affected by developmental stressors. It is generally considered throughout biology to be an indicator of developmental instability and thus of lack of overall biological fitness. Several investigators have proposed that deficiencies in general intelligence (*g*) may be indicators of the same kind of instability in human brain development. If so, FA and *g* should be negatively correlated. Moreover, because *g* shows substantial genetic influences, FA should also show genetic influences, and the two sets of genetic influences should be correlated. We investigated these propositions in a sample of 263 adults that included 88 pairs of twins. Results indicated genetic influences on FA, but FA and *g* were not correlated at either the observed or genetic levels.

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Beginning with conception, an organism develops so as to express a phenotype appropriate to its species and life conditions so that it can reproduce successfully. This task varies in difficulty depending on the particular genotype the organism carries as well as on environmental perturbations and chance events such as mutations, exposure to toxins, pathogens and parasites, injuries, and starvation, both pre- and postnatally (Moller & Swaddle, 1997; Yeo & Gangestad, 1998). Individuals also vary in the stability of their development in the face

of these perturbations: some individuals develop normally and adaptively under most conditions encountered by their species, while others show maladaptive or disordered development under even very slightly adverse circumstances (Gangestad & Thornhill, 1999). Some studies find genetic influences on these individual differences (Moller & Thornhill, 1997).

Body symmetry is one key manifestation of developmental stability at the morphological level. Far short of producing actual pathology, environmental perturbations and chance events can affect the precision of the developmental body design so that bilateral morphological traits such as wing size, toe length, and ear breadth show asymmetry. Thus one way of estimating the extent of developmental instability is to estimate fluctuating asymmetry

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(FA), or the non-pathological left–right asymmetry of individual bilateral body traits for which signed differences between right and left sides in the population have a mean of zero and are nearly normally distributed (Van Valen, 1962). In humans, examples of such traits would be eye breadths and elbow to wrist lengths. In other animals, examples would be wing spans and fin sizes.

It is known that FA increases across a wide variety of species with exposure to extreme conditions in development (Moller & Thornhill, 1997; Parsons, 1990). FA is thus an index of an individual's success in dealing with environmental stresses and genetic vulnerabilities. Moreover, FA appears to be a measure of general biological fitness, as it predicts evolutionarily relevant outcomes such as health, survival, fecundity, and mating success across many species, including humans (Gangestad & Simpson, 2000; Gangestad & Thornhill, 1999; Leung & Forbes, 1996; Mealey, Bridgstock, & Townsend, 1999; Moller & Thornhill, 1998; Rhodes et al., 2001; Thornhill & Moller, 1998). Thus, though FA is specifically morphological, it can be expected to be related to other forms of developmental integrity. In particular, in humans mental retardation has been related to high developmental instability (Thornhill & Moller, 1997). As many forms of mental retardation involve disruptions in neurological functional integrity, Furlow, Armijo-Prewitt, Gangestad, and Thornhill (1997) proposed that non-pathological individual differences in general intelligence ( $g$ ) may also reflect individual differences in developmental instability. If so, FA should be negatively correlated with measures of  $g$ .

In general, studies have supported this proposition, though sample sizes have been small, the resulting correlations have varied considerably, and there may be unpublished studies with null findings. Furlow et al. (1997) reported an average correlation of  $-.23$  across two studies of 112 and 128 undergraduate students. Luxen and Buunk (2006) reported a correlation of  $-.25$  in a population sample of 81. Prokosch, Yeo, and Miller (2005) found a correlation of  $-.39$  between their highest  $g$ -loaded test and FA in a sample of 78 young male undergraduates. Thoma, Yeo, Gangestad, Halgren, Sanchez, and Lewine (2005), using a sample of 19 males from a university community, reported a correlation of  $-.49$ . Bates (2007) reported correlations of  $-.41$  and  $-.29$  in samples of 98 university students and their family members and friends and 164 university students, respectively. In contrast, we know of one as-yet-unpublished study based on a sample of 65 men who took a 12-item version of the Raven that showed a (positive) correlation of  $.17$  (personal communication with Steve Gangestad, January 16, 2007). There is some

evidence that the association may be stronger in males than in females: several of the studies have used samples consisting only of males, and Rahman, Wilson, and Abrahams (2004) reported significant correlations involving several tests of different abilities on the order of  $-.30$  in men but not in women in a sample of 240 (120 of each sex). Of course, the as-yet-unpublished positive correlation of  $.17$  in men calls that into question.

Any proposed association between FA and  $g$  has to consider the fact that  $g$  shows substantial genetic influences. From 50% to 80% of the individual differences in  $g$  can be attributed to genetic influences, and many of the brain structural components that are related to  $g$  also show strong genetic influences (Deary, Spinath, & Bates, 2006). In addition, the genetic influences on these brain structural components appear to be strongly related to those on  $g$  (Posthuma et al., 2002; Toga & Thompson, 2005). Given the large body of evidence for these genetic influences and their strength, if there is any meaningful association between FA and  $g$ , then FA must also show genetic influences. Most people have tended to assume that the evidence for genetic influences on  $g$  means that there are genes coding specifically for intelligence. Another possibility, however, is that these genetic influences may result from fitness-related variation that affects neurological integrity and efficiency as well as morphological, immunological, metabolic, and other forms of developmental integrity (Furlow et al., 1997). To the extent that this is the case and that both FA and  $g$  index this overall developmental integrity, we should expect genetic influences common to both FA and  $g$ . There is, of course, no reason that there could not be both genetic influences specific to  $g$  and genetic influences on fitness-level variation that contribute to many traits including  $g$ .

The purpose of this study was to examine new data relevant to these issues. Specifically, we investigated a sample of 263 individuals for whom measures of both FA and  $g$  were available to assess the correlation between FA and  $g$ . This sample is relatively large for studies of human FA, but it is of even greater importance because it includes 88 pairs of twins. We used these pairs of twins to estimate the genetic influences on FA and the extent to which these genetic influences were associated with genetic influences on  $g$ .

## 1. Method

### 1.1. Research participants

Our sample came from the Minnesota Study of Twins Reared Apart (MISTRA). MISTRA was initiated to

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