AMERICAN JOURNAL OF HUMAN BIOLOGY 15:601–619 (2003)

Attention-Deficit Hyperactivity Disorder (AD/HD) and Fluctuating Asymmetry (FA) in a College Sample: An Exploratory Study

C. BURTON,[1] J.C. STEVENSON,[1]* D.C. WILLIAMS,[2] P.M. EVERSON,[1] E.R. MAHONEY,[4] AND J.E. TRIMBLE[3]
[1]*Department of Anthropology, Western Washington University, Bellingham, Washington 98225*
[2]*Department of Biology, Western Washington University, Bellingham, Washington 98225*
[3]*Department of Psychology, Western Washington University, Bellingham, Washington 98225*
[4]*Survey Research and Development, PeaceHealth Corp., Bellevue, Washington 98007*

ABSTRACT Departures from normal development can be partly assessed by measuring fluctuating asymmetry (FA), that is, differences from perfect symmetry in traits that display bilateral symmetry. Attention-deficit hyperactivity disorder (AD/HD), one of the most common psychiatric conditions, is diagnosed if there are developmentally inappropriate levels of inattention, hyperactivity, and impulsivity. The objective here is to measure whether AD/HD behaviors positively correlate with FA in head, hands, and fingerprints of a sample of college students (n = 176, 57 male, 119 female) not selected for AD/HD. FA was measured as the absolute value of the difference between right and left sides divided by group mean trait size. Average FAs (mean, SE) were lowest for finger lengths (e.g., male, 3rd, 0.011 ± 0.001; female, 3rd, 0.012 ± 0.001) and highest for digit ridge counts (e.g., male, 5th, 0.075 ± 0.007; female, 2nd, 0.069 ± 0.005). Average FAs were similar between the sexes and only one facial measure and the facial index (summed FAs) differed significantly between the sexes (F > M). The scores for measures of the adult AD/HD behavioral assessment instrument, the Wender Utah Rating Scale (WURS) were high overall in this sample and males exhibited higher rates of symptoms than females. A Rasch measurement model analysis of individual responses to the WURS produced a true interval score for each person that is a measure of individual "AD/HDness." FA indices were then regressed on Rasch scores. A univariate analysis of all the variables demonstrated a significant interaction of sex. Hand, Dermatoglyphic, Face, and Total Indices were then regressed by sex on the Rasch values of "AD/HDness." Only in males was there a trend for the Dermatoglyphic Index ($F_{1,55} = 3.627$, $P = 0.062$) and Total Index ($F_{1,55} = 3.811$, $P = 0.056$) to increase as AD/HDness increases. Am. J. Hum. Biol. 15:601–619, 2003. © 2003 Wiley-Liss, Inc.

Attention deficit/hyperactive disorder (AD/HD) is one of the most common psychiatric conditions and is diagnosed if there are developmentally inappropriate levels of inattention, hyperactivity, and impulsivity (APA, 1994; Anastopoulos and Shelton, 2001; Barkley, 1997b). Symptoms must be exhibited for at least 6 months and must have originated at or before 7 years of age. The problematic behaviors must lead to clinical impairments on the job, in school, or in social settings. Scahill and Schwab-Stone (2000) estimate prevalence at 5–10% in school-age children. Adult prevalence rates are lower, at 1–6% (Wender 1997), and adolescent levels are intermediate (Nolan et al., 2001). There is a cross-culturally consistent, uneven sex ratio, with significantly more males, 2:1 in epidemiological samples versus 10:1 in self-identified or professionally referred clinical samples (Gingerich et al., 1998). Many clinical studies indicate that the expression of AD/HD may be somewhat different in females, with lower rates of hyperactivity and aggression (Gaub and Carlson et al., 1997).

AD/HD has one of the highest heritabilities for a "trait," averaging 0.64–0.91 (Edelbrock et al., 1995; Gillis et al., 1992; Levy et al., 1997; Zahn-Waxler et al., 1996). The models that attempt to explain this disorder argue that AD/HD likely reflects reduced efficiency in executive functions, particularly self-regulation (impulse control) (Baird et al., 2000; Barkley, 1997a,b, 2001; Stevenson and Williams, 2000) and/or deficiencies in reward mechanisms (Blum et al., 2000), or both (Sonuga-Barke, 2002).

*Correspondence to: Joan C. Stevenson, Department of Anthropology, Western Washington University, Bellingham, WA 98225-9083. E-mail: jcstvnsn@cc.wwu.edu

Received 9 July 2002; Revision received 15 January 2003; Accepted 18 January 2003

Published online in Wiley InterScience (www.interscience.wiley.com). DOI: 10.1002/ajhb.10179

© 2003 Wiley-Liss, Inc.

Individuals with AD/HD exhibit biochemical and gross anatomical differences from controls. Irregularities in neurochemistry are implicated and include the dopamine system (e.g., Comings, 2001; Ernst et al., 1998; Faraone et al., 2001) and perhaps the serotonergic and adrenergic systems (e.g., Quist et al., 2000; Comings et al., 2000). Tannock (1998) reviewed the neuroimaging studies of AD/HD subjects, which document atypical cerebral lateralization and abnormalities in the prefrontal cortex, basal ganglia, and corpus callosum. Casey et al. (1997) found abnormal measures for the prefrontal cortex, caudate, and globus pallidus to correlate and predict poorer task performance (typically deficient in individuals with AD/HD) in a sample of boys with AD/HD. Performance improved with age, depending on the relative size of these structures, supporting the belief that AD/HD behaviors likely partly reflect a lag in development (that correlates with anatomical traits).

Individuals with AD/HD exhibit the delay through extreme and developmentally inappropriate levels of attentiveness, restlessness, and impulsivity, behaviors that vary "genetically throughout the entire population rather than as a disorder with discrete determinants" (Levy et al., 1997:737). The heritability of AD/HD symptoms is high for the general population (Rhee et al., 1999), but these symptoms are thought to represent an etiologically complex disorder representing the action of multiple genes affecting either the brain's reward system or executive functions (e.g., Auerbach et al., 2001; Comings et al., 2000; Fisher et al., 2002; Miller et al., 2001).

Polygenic traits are expressed as phenotypic continua such that ends of the distribution can include individuals who have increased levels of homozygosity and, usually, higher levels of developmental instability due to genetic and environmental stressors (Markow, 1992). The developmental instability associated with polygenic disorders such as AD/HD can be expressed in a variety of ways, e.g., as in deviations in brain laterality (Castellanos et al., 1994, 1996; Filipek et al., 1997; Hynd et al., 1993; Reid and Norvilitis, 2000), by increased rates of minor physical anomalies (Durfee, 1974; Firestone et al., 1978; Krouse and Kauffman, 1982; Quinn and Rapoport, 1974; Rapoport and Quinn, 1975; Waldrop and Halverson, 1971), or, untested to date in individuals with AD/HD, as increased fluctuating asymmetry (FA). FA describes departures from normal developmental trajectories that are measured as differences from perfect symmetry (0 symmetry) in traits that normally display bilateral symmetry (Møller and Swaddle, 1997). Low FA has been correlated with various aspects of good health (Thornhill and Møller, 1997). Shackelford and Larsen (1997) found that the more facially symmetric individuals were in better psychological, affective, and physiological health (including lower levels of impulsivity) than individuals with higher levels of facial asymmetry. They assessed this correlation in a healthy college population. We propose in this study also to examine a college sample. A "normal" population sample should represent a continuum of low to high AD/HD symptoms because of the polygenic nature of this disorder and the high heritability of symptoms in the general population, presumably including college populations where AD/HD symptoms have also been documented (e.g., Fossati et al., 2001; Lara-Muñoz et al., 1998; Weyandt et al., 1995). We predict that developmental instability (as measured through FA) will positively correlate with the degree of AD/HD disability (as measured here through a Rasch analysis of a behavioral scale assessing childhood AD/HD symptoms).

SUBJECTS AND METHODS

Data collection

There were 246 participants (18–32 years of age) who were recruited from introductory Biology and Biological Anthropology classes by giving them extra credit on the laboratory portions of their grade. The Human Subjects Review Committee of Western Washington University approved the project before data were collected. Five individuals were fully assessed but removed from further analysis due to injuries or reconstructive surgeries on the regions of the body relevant to the study. All of the 241 remaining individuals completed the Wender Utah Rating Scale (WURS) scale and anthropometric measures were taken, but the camera malfunctioned for the facial pictures for the first 58 individuals. Their responses to the WURS were included to generate the scale measuring "AD/HDness" but they were excluded from further analyses.

Thus, the completely measured sample consisted of 183 individuals including 125 females and 58 males. Each participant completed the WURS, a diagnostic instrument that was developed by Wender and colleagues (Wender et al., 1981; Ward et al., 1993; Wender, 1995) in order to assess AD/HD symptomology for adults retrospectively. The use of the Wender scale is not to diagnose but to retrospectively assess the severity of symptoms in childhood for adults who meet Utah criteria (Wender et al., 2000). (The Utah Criteria exclude individuals with major mood disorders, schizophrenia, and schizophrenic spectrum disorders and borderline and antisocial personality disorder.)

The WURS has been found to be internally reliable and discriminates well between adults with AD/HD versus normal adults, or versus depressed adults (Fossati et al., 2001; Rossini and O'Connor, 1995; Stein et al., 1995; Ward et al., 1993; Weyandt et al., 1995). It comprises a 61-item self-report form devised to evaluate the presence and severity of AD/HD symptoms when the individual was a child, presented in Table 1. Subjects rated themselves for each item as: not at all or very slightly (score = 0), mildly (score = 1), moderately (score = 2), quite a bit (score = 3), or very much (score = 4). One question asked whether the individual had "trouble with police, booked, convicted" and was replaced with "ever receive a traffic ticket? For what?"; college students seemed more likely to have police contact in conjunction with traffic violations, also an indicator of impulsivity (Cox et al., 2000). Subjects also provided information on age, sex, and whether they had prior physical injuries or surgeries that would interfere with assessment of FA. Ward et al. (1993) also created an abbreviated version 25-item subset of the WURS that also exhibits internal and temporal consistency (Rossini and O'Connor, 1995).

Faces were photographed with a Kodak DC90 (2.5 megapixel) Zoom Digital Camera in a naturally well-lit room. The camera and tripod were positioned securely 104 cm (included head and top of shoulders) from a taped line on which subjects were to position themselves with the center of their feet. The camera was adjusted vertically relative to the face. They were instructed to maintain a neutral expression. Hair and glasses were removed if in the way. The photographer positioned their heads so that they faced the camera square to the vertical plane. The top of the camera reticle (square with crosshairs) was positioned at the level of the eyebrows. All images were saved in 32-bit color at 96 dpi.

PaintShop Pro 6.0 enhanced the facial features and superimposed a grid with horizontal and vertical ruled scales that allowed measurement of facial features. All measurements were taken to the nearest 0.01 mm using PaintShop Pro 6.0. The sizes of six traits were measured on each side of the face so that the degree of asymmetry between right and left sides could be calculated. The sizes of five traits were measured in relation to the midsagittal plane situated by placing a vertical line on the center of the philtrum or nasal septum and lining it vertically with the midpoint between the eye pupils (see Fig. 1). The sixth trait, ear placement, was measured from the most superior point on the pinna to the horizontal plane tangential to the most inferior curve of the lower lip. Ear height was measured from the most superior point of the pinna to the most inferior point of the ear lobe using Paleotech spreading calipers accurate to 1 mm (Waynforth, 1998).

Photocopies were made of fully extended hands pressed on the glass and labeled by subject code. Ridge counts were made with good lighting and a magnifying glass (10×). Dermatoglyphic measurements of asymmetry were taken using the medial-digital ridge count, which involved counting the number of horizontal ridges cutting or touching a straight line drawn through the center of the medial portion of each digit, 2–5. This is not a typical dermatoglyphic measurement (e.g., Holt 1968) but required less interpretation of the complexity of the fingerprint patterns.

The ventral surface of digits 2–5 were measured from the basal crease of the digit to the furthest portion of the fleshy tip of the finger fully extended using Paleotech spreading calipers accurate to 1 mm. Wrist width measurements were taken by measuring the maximum wrist width from the midline crease of each wrist with the arm in the relaxed supine position using the same calipers.

Analysis

The reliability of the measurements was estimated by calculating repeatabilities (r_I, the intraclass correlation coefficient; Krebs, 1989)

TABLE 1. Key for Figures 2 and 3, Wender Utah Rating Scale (Ward et al., 1993)

AS A CHILD I WAS (OR HAD):
1. Active, restless, always on the go
2. Afraid of things
3. Concentration problems, easily distracted
4. Anxious, worrying
5. Nervous, fidgety
6. Inattentive, daydreaming
7. Hot- or short-tempered, low boiling point
8. Shy, sensitive
9. Temper outbursts, tantrums
10. Trouble with stick-to-it-iveness, not following through, failing to finish things started
11. Stubborn, strong-willed
12. Sad or blue, depressed, unhappy
13. Incautious, dare-devilish, involved in pranks
14. Not getting a kick out of things, dissatisfied with life
15. Disobedient with parents, rebellious, sassy
16. Low opinion of myself
17. Irritable
18. **Outgoing, friendly, enjoyed company of people**
19. Sloppy, disorganized
20. Moody, ups and downs
21. Angry
22. **Friends, popular**
23. **Well-organized, tidy, neat**
24. Acting without thinking, impulsive
25. Tendency to be immature
26. Guilty feelings, regretful
27. Losing control of myself
28. Tendency to be or act irrational
29. Unpopular with other children, didn't keep friends for long, didn't get along with other children
30. Poorly coordinated, did not participate in sports
31. Afraid of losing control of self
32. **Well-coordinated, picked first in games**
33. Tomboyish (for women only)
34. Running away from home
35. Getting into fights
36. Teasing other children
37. Leader, bossy
38. Difficulty getting awake
39. Follower, led around too much
40. Trouble seeing things from someone else's point of view
41. Trouble with authorities, trouble with school, visits to principal's office

MEDICAL PROBLEMS AS A CHILD

42. Headaches
43. Stomachaches
44. Constipation
45. Diarrhea
46. Food allergies
47. Other allergies
48. Bedwetting

AS A CHILD I WAS (OR HAD):

49. **Overall a good student, fast**
50. Overall a poor student, slow learner
51. Slow in *learning* to read
52. Slow reader
53. Trouble reversing letters
54. Problems with spelling
55. Trouble with mathematics or numbers
56. Bad handwriting
57. Able to read pretty well but never enjoyed reading
58. Not achieving up to potential
59. Repeating grades
60. Suspended or expelled
61. Ever receive a traffic ticket?

Bold items have reversed Likert scales from the other questions for the Rasch measurement model analysis.

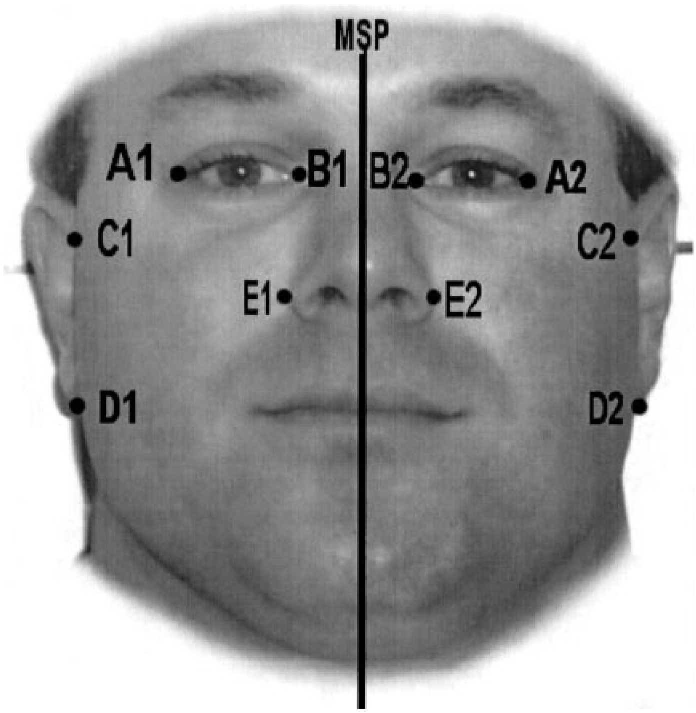


Fig. 1. Landmarks used for measurement of facial traits. Vertical solid line shows the midsagittal plane. Each trait (left, right) was measured as follows: Eye Width (A1-B1, A2-B2), Outer Eye Closure Width (A1-MSP, A2-MSP), Cheek Bone Width (C1-MSP, C2-MSP), Jaw Width (D1-MSP, D2-MSP), Nasal Width (E1-MSP, E2-MSP).

separately for each of the measures from the photographs and of ridge counts, finger lengths, and wrist widths. One person (CB) made all the measures twice. Each pair of identical measures was averaged.

Signed asymmetries for individual traits were calculated as the absolute value of R–L, where R and L are sizes of the right and left sides of a trait. Unsigned asymmetries were calculated as $|R–L|$. Fluctuating asymmetries (FAs) for each measure were calculated by dividing the unsigned asymmetries by the average of the trait, i.e., $(|R–L|)/\text{avg}(|R–L|)$. Composite indices were generated for hand, ridge counts, and face because they are a better reflection of an individual's developmental stability than any single trait (Dufour and Weatherhead, 1996; Gangestad and Thornhill, 1999; Hume and Montgomerie, 2001; Leung and Forbes, 1997; Livshits and Kobyliansky, 1987; Pechenkina et al., 2000). Leung et al. (2000) compared composite indices and recommended summing the formula above for unsigned symmetries across the traits. This composite index adjusts for the sample mean trait size such that all traits have equal weight in this measure of developmental stability (Hume and Montgomerie, 2001).

Descriptive statistics were calculated for each measure and index. Seven outliers were removed (one male and six females) following Tukey (1977). Descriptive statistics were then recalculated. Spearman rank correlations with Bonferroni correction were calculated among unsigned asymmetries of all traits for each subject because some distributions of traits were significantly different from normal. (FAs for males and females were compared using Levene's test for equality of variances and t-tests.)

The WURS symptoms were "summarized" both as an average of the summed responses to the short version of the Wender scale and by a Rasch measurement model analysis of the responses for each individual by using WINSTEPS (Linacre and Wright, 2000), which transforms ordered or unordered categorical data into an interval-level unidimensional measure of the construct "AD/HDness." Individuals are placed on a real number line that represents (with known accuracy and precision) their measured amounts of "AD/HDness" (DeRoos and Allen-Meares, 1998). The Rasch was the preferred summary measure of AD/HD behaviors (rather than a summed Likert) and was used in the regression and correlation analyses (Bond and Fox, 2001). The Rasch logit values positively correlated with the summed Likerts for the long (r = 0.786, $P = 0.000$) and short (r = 0.849, $P = 0.000$) versions of the WURS (and the short and long with each other, r = 0.926, $P = 0.000$), but see Massof (2002) for a history of the problems associated with using summed Likert scales and details about Likert's mistakes in his influential publication (1932).

Univariate analysis with interaction terms was run to assess the separate and combined contributions of the indices. Each composite index, face, ridge count, hand, and total indices were regressed separately on the Rasch measure of "AD/HDness" for males and females (Zar, 1996). Pearson's correlations were also calculated for FA indices and the Rasch logits.

RESULTS

Of the 241 participants, only 233 (169 F, 64 M) gave complete information on age. The subjects were primarily young adults (mean ± SE, 20.3 ± 0.26 and 19.9 ± 0.31 years, for females and males, respectively) and most were of European descent.

All of the measures of the traits had acceptably high and statistically significant repeatabilities (means = 0.94, range = 0.79–0.99, n = 16 traits) in each case ($F_{182,365} > 3.854$, $P < 0.01$).

Means, standard errors, skewness, kurtosis, and *t*-tests for difference from zero for signed asymmetries are presented in Table 2. Only six traits had signed asymmetries with a normal distribution and a mean of zero, thus exhibiting true FA, including wrist width, length of digit 3, ridge counts for digits 2, 4, and 5, and nose width. The signed asymmetries for four traits, ridge count for digit 3, ear height, ear placement, and jaw width, were not significantly different from zero but are leptokurtic. Three traits exhibited directional asymmetry (DA) and include lengths of digits 4 and 5 and eye width. The signed asymmetries for three traits, length of digit 2, chin width, and jaw width, exhibit both DA and leptokurtosis. Recent discussions indicate that traits showing significant DA and leptokurtosis are still appropriate to use as measures of developmental instability (Gangestad and Thornhill, 1999; Leamy, 1999; Leung and Forbes, 1997).

Organism-wide asymmetries were examined by looking at correlations among unsigned asymmetries of all traits for each subject. Spearman rank correlations were used because some distributions were significantly

TABLE 2. Signed asymmetries: descriptive statistics

Variable	Mean	Std. error	Skewness	Kurtosis	*t*	Sig.
Wrist width	−0.1236	0.1017	−0.017	−0.347	1.215	0.226
Digit 2 length	−0.4910	0.0972	−0.379	1.543	5.048	**0.000**
Digit 3 length	0.0935	0.0937	−0.263	0.746	0.998	0.320
Digit 4 length	0.4570	0.0879	−0.013	0.012	5.198	**0.000**
Digit 5 length	0.7210	0.1050	0.161	0.445	6.856	**0.000**
Ridge Count Digit 2	−0.1100	0.1500	−0.165	−0.114	0.722	0.471
Ridge Count Digit 3	0.2300	0.1800	0.549	4.285	1.305	0.193
Ridge Count Digit 4	0.0164	0.1300	0.211	0.397	0.124	0.901
Ridge Count Digit 5	0.0601	0.1200	0.182	0.001	0.516	0.607
Chin width	0.6700	0.3200	−0.126	2.054	2.114	**0.036**
Ear Height	−0.0540	0.1300	0.338	1.686	0.419	0.676
Ear Placement	−0.0857	0.3000	−0.611	3.320	0.289	0.773
Eye width	0.6400	0.1700	0.210	0.616	3.788	**0.000**
Jaw width	0.3900	0.3300	−0.119	2.242	1.186	0.237
Nose width	0.2300	0.1800	−0.123	0.977	1.254	0.212
Outer eye width	0.7700	0.2800	0.986	4.109	2.720	**0.007**

N = 176, df = 175.

different from normal. Pairwise Spearman rank correlations between unsigned asymmetries for all traits were more often significantly positive rather than negative (positive/negative = 63/57). However, only four of the 120 between-trait correlations were significant after sequential Bonferroni correction. There were significant positive correlations ($P < 0.0004$) within the face set of traits between chin width and jaw width (r = 0.36), chin width and nose width (r = 0.31), outer eye width and chin width (r = 0.28), and between the length of digit 4 and ridge count of digit 3 (r = 0.26).

Table 3 presents the means and standard errors for FAs for total, male, and female samples. Also presented are comparisons of males to females using Levene's test for inequality of variances and *t*-tests. FAs for Hand Traits ranged in males from 0.011 ± 0.001 for length of digit 3 to 0.021 ± 0.002 for length of digit 5 and in females from 0.012 ± 0.001 for length of digit 3 to 0.021 ± 0.002 for length of digit 5. FAs for Dermatoglyphic Traits ranged in males from 0.052 ± 0.005 for ridge counts for digit 4 to 0.075 ± 0.007 for ridge counts for digit 5 and in females from 0.056 ± 0.004 for ridge counts for digit 2 to 0.069 ± 0.005 for ridge counts for digit 2. FAs for Face Traits ranged in males from 0.022 ± 0.003 for ear height to 0.040 ± 0.009 for nose width and in females from 0.020 ± 0.002 for ear height to 0.061 ± 0.008 for nose width. Overall, males and females are similar for most traits. Levene's Test for Equality of Variances demonstrates that variances are not significantly different for hand or dermatoglyphic traits but are significantly different between male and female face FAs (F-value, probability) for eye width (5.827, 0.017), and outer eye width (4.425, 0.037), nose width (7.658, 0.006) and the Face Index (4.391, 0.038). The *t*-test only revealed two statistically significant differences for those four traits with unequal variances: outer eye width ($t = 2.565$, $P = 0.011$) and Facial Index ($t = 2.18$, $P = 0.031$).

Table 4 presents the means and standard deviations of responses to the shortened version of the WURS for total sample, both sexes, and comparative data from Wender (1995) including adults with AD/HD and "normal" comparison subjects. (Summed Likert scores for the complete Wender scale in this sample strongly positively correlated with the short form, r = 0.941, $P = 0.000$.) Males exhibit overall higher symptom rates, and variances differ statistically significantly according to the Levene's test (F-value, probability) for "Sad or blue, depressed, unhappy" (5.098, 0.025), "Losing control of myself" (13.416, 0.000), "Tendency to be or act irrational" (5.022, 0.025), "Unpopular with other children" (5.098, 0.025), "Trouble with authorities" (34.478, 0.000), and "Not achieving up

TABLE 3. FA traits and indices by sex: descriptive statistics, Levene's test of equality of variance, and t-test of independent means

Indices and traits	Total (n = 176) Mean	Total SE	Male (n = 57) Mean	Male SE	Female (n = 119) Mean	Female SE	Levene's F	Levene's Sig.	*t*	Sig.
Hand Index	0.080	0.002	0.079	0.004	0.081	0.003	0.420	0.518	0.368	0.714
Wrist width	0.019	0.001	0.020	0.002	0.019	0.001	0.167	0.683	0.330	0.742
Digit 2 length	0.015	0.001	0.015	0.002	0.016	0.001	0.227	0.634	0.492	0.624
Digit 3 length	0.012	0.001	0.011	0.001	0.012	0.001	0.153	0.696	0.426	0.671
Digit 4 length	0.013	0.001	0.013	0.001	0.014	0.001	0.228	0.634	0.686	0.494
Digit 5 length	0.021	0.001	0.021	0.002	0.021	0.002	0.032	0.858	0.172	0.864
Dermatoglyphic Index	0.252	0.009	0.263	0.016	0.246	0.010	0.157	0.692	0.905	0.367
Ridge Count Digit 2	0.068	0.004	0.067	0.006	0.069	0.005	2.295	0.132	0.172	0.864
Ridge Count Digit 3	0.062	0.004	0.069	0.010	0.059	0.005	1.823	0.179	1.060	0.290
Ridge Count Digit 4	0.054	0.003	0.052	0.005	0.056	0.004	0.584	0.446	0.574	0.566
Ridge Count Digit 5	0.068	0.004	0.075	0.007	0.063	0.005	1.684	0.196	1.424	0.156
Facial Index	0.226	0.001	0.189	0.019	0.244	0.015	**4.391**	**0.038**	**2.180**	**0.031**
Chin width	0.028	0.003	0.023	0.004	0.031	0.003	2.375	0.125	1.326	0.186
Ear height	0.021	0.002	0.022	0.003	0.020	0.002	0.001	0.980	0.497	0.620
Ear placement	0.025	0.003	0.025	0.005	0.026	0.003	0.012	0.914	0.058	0.954
Eye width	0.029	0.003	0.027	0.005	0.031	0.004	**5.827**	**0.017**	0.563	0.574
Jaw width	0.033	0.003	0.029	0.005	0.035	0.004	0.701	0.403	0.959	0.339
Nose width	0.054	0.006	0.040	0.009	0.061	0.008	**7.658**	**0.006**	1.670	0.097
Outer eye width	0.035	0.003	0.023	0.004	0.040	0.004	**4.425**	**0.037**	**2.565**	**0.011**
Total Index	0.558	0.014	0.531	0.025	0.570	0.017	0.411	0.522	1.300	0.195

*TABLE 4. WURS short form results by sex with comparison data**

WURS item	Adults with ADHD Subjects (n = 81)		Normal comparison Subjects (n = 100)		College students Total Subjects (n = 176)		College students Male Subjects (n = 57)		College students Female Subjects (n = 119)	
Individual Items	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Concentration problems, easily distracted	3.3	0.9	0.7	0.9	2.0	1.1	2.3	1.1	1.8	1.1
Anxious, worrying	2.8	1.1	1.1	1.0	2.1	1.2	2.27	1.2	2.0	1.2
Nervous, fidgety	3.1	0.9	0.6	0.9	1.9	1.1	2.0	1.2	1.8	1.0
Inattentive, daydreaming	3.2	1.0	0.6	0.8	2.2	1.1	2.5	1.2	2.1	1.0
Hot- or short-tempered, low boiling point	2.7	1.3	0.8	1.0	1.8	1.1	2.1	1.2	1.7	1.1
Temper outbursts, tantrums	2.4	1.2	0.6	0.9	1.1	1.1	1.8	1.0	1.8	1.1
Trouble with stick-to-it-iveness	3.0	1.1	0.7	0.9	1.7	0.9	1.9	1.0	1.6	0.9
Stubborn, strong-willed	3.1	1.1	1.4	1.2	2.9	1.3	2.9	1.2	2.9	1.3
Sad or blue, deprressed, unhappy	2.2	1.2	0.4	0.7	1.4	0.7	1.6	0.8	1.4	0.6
Disobedient, rebellious, sassy	2.4	1.4	0.5	0.7	1.8	0.9	1.8	0.8	1.7	0.9
Low opinion of myself	2.6	1.3	0.7	0.8	1.6	0.7	1.5	0.7	1.6	0.7
Irritable	2.4	1.1	0.4	0.6	1.7	0.9	1.8	0.8	1.7	0.9
Moody, ups and downs	2.8	1.0	0.8	0.8	1.8	0.9	2.0	1.0	1.8	0.9
Angry	2.5	1.2	0.6	0.8	1.4	0.8	1.6	0.7	1.4	0.8
Trouble seeing things from someone else's point of view	2.3	1.1	0.8	1.2	1.6	0.8	1.6	0.7	1.6	0.9
Acting without thinking, impulsive	2.9	1.1	0.8	0.9	2.2	1.1	2.5	1.1	2.1	1.0
Tendency to be immature	2.8	1.6	0.7	0.9	1.9	0.9	2.2	0.9	1.8	0.9
Guilty feelings, regretful	2.6	1.1	0.6	0.8	2.0	1.1	2.3	1.1	1.9	1.0
Losing control of myself	2.2	1.3	0.3	0.6	1.4	0.8	1.7	0.9	1.3	0.6
Tendency to be or act irrational	2.0	1.2	0.2	0.5	1.5	0.8	1.6	0.9	1.4	0.7
Unpopular with other children	1.8	1.3	0.2	0.5	1.2	0.4	1.2	0.5	1.2	0.4
Trouble with authorities, trouble with school, visits to principal's office	1.8	1.6	0.2	0.6	1.3	0.7	1.6	0.9	1.2	0.5
Overall a poor student, slow learner	1.4	1.4	0.1	0.3	1.29	0.67	1.4	0.7	1.26	0.7
Trouble with mathematics or numbers	2.1	1.5	0.5	1.0	1.9	1.2	1.9	1.3	1.9	1.2
Not achieving up to potential	3.2	1.0	1.1	1.2	1.7	1.0	2.2	1.3	1.4	0.8
Total Scores										
Women	65.8	14.3	15.0	8.5					42.1	11.7
Men	60.3	14.2	17.9	11.0			48.8	12.9		
All Subjects	62.2	14.6	16.1	10.6	44.0	12.4				

*Comparison data from Wender, 1995:247

to potential" (19.392, 0.000). Of the symptoms with unequal variances, three were also significantly different according to the *t*-test (T, probability) "Losing control of myself" (2.499, 0.015), "Trouble with authorities" (60.957, 0.002), and "Not achieving up to potential" (60.799, 0.000). Of those symptoms with equivalent variances, several symptoms were significantly different (T, probability): "Concentration problems" (2.09, 0.038), "Inattentive, daydreaming" (2.482, 0.0140, "Temper outbursts, tantrums" (2.118, 0.36), "Trouble with stick-to-it-iveness" (2.276, 0.024), "Acting without thinking, impulsive" (2.487, 0.014), "Tendency to be immature" (2.422, 0.017), "Guilty feelings, regretful" (2.411, 0.017).

The performance of both sexes on the WURS lies between the "normal" and AD/HD sample of Wender (1995:257). Overall, these college students exhibit a relatively high frequency of symptoms.

When the WURS was subjected to a Rasch rating scale analysis, 7 of the 58 items were found to have statistically significant misfit. See Figure 2 (WURS Item Fit) and Table 1 for more complete descriptions of the questions. The fit characteristics of these seven items indicated that the there is an inconsistency in measurement assumptions among these seven items and the balance of the items on the survey. The final measure thus consists of 51 items. Respondents did not use the middle three categories (mildly, moderately, quite a bit) of the original five response categories as distinct categories. Collapsing these three categories resulted in a useable measure. The fit values of all respondents were acceptable, indicating that the respondents cooperated in the measurement effort by giving thoughtful, straightforward answers to the questions. "AD/HDness" does appear to fit the Rasch measurement model well and represents a unidimensional construct.

Item difficulty by sex is presented in Figure 3. The higher the item difficulty score, the more affected by AD/HD an individual must be to endorse the behavior. There is a reasonably close match between the sexes.

Table 5 presents a univariate analysis with interaction terms. The only result of note is the significant interaction effect of sex. Thus, sexes were also examined separately by regression analyses. The scatterplots and

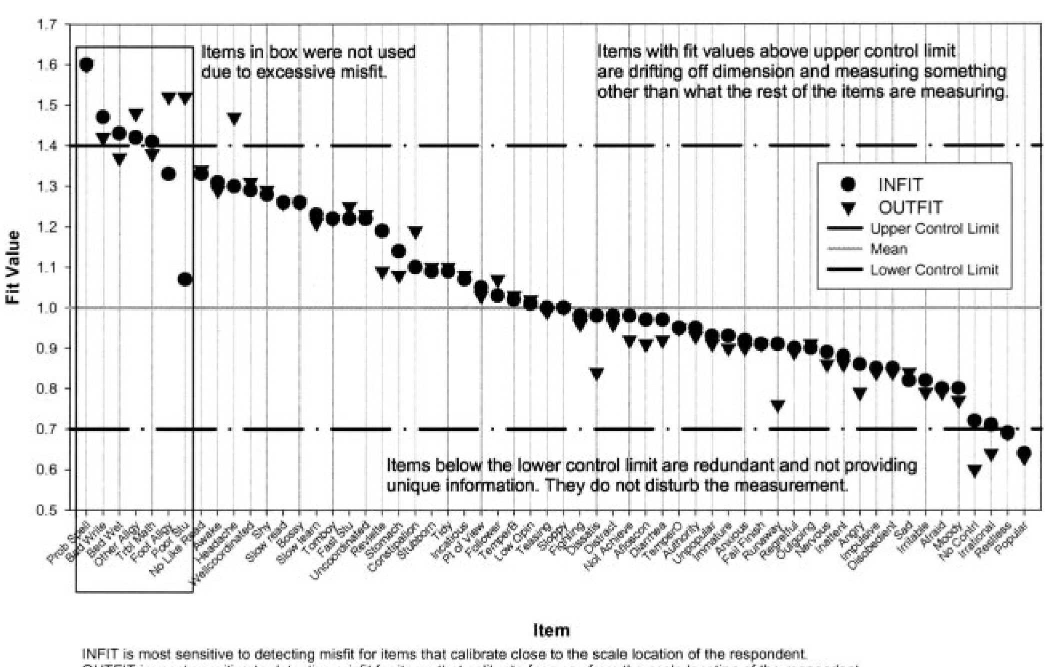


Fig. 2. WURS item fit: extent to which all items are measuring the latent construct (AD/HDness).

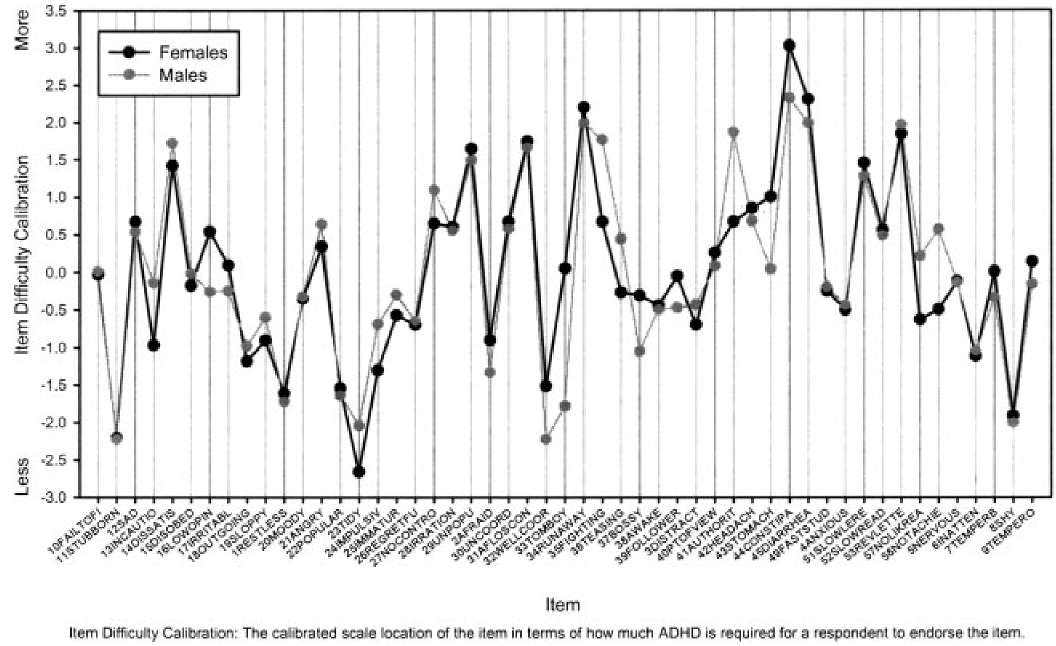


Fig. 3. Item difficulty by sex: how much of the latent construct (AD/HDness) a person must have to exhibit that factor.

TABLE 5. Univariate analysis of variance with interaction terms

Source	SS	df	MS	F	Sig.
Corrected model	20.888	8	2.611	3.366	**0.001**
Intercept	6.98	1	6.98	8.998	**0.003**
Sex	16.791	1	16.791	21.646	**0.000**
Hand Index	0.495	1	0.495	0.638	0.426
Dermatoglyphic Index	0.016	1	0.016	0.020	0.888
Face Index	0.738	1	0.738	0.952	0.331
Hand Index x Dermatoglyphic Index	0.063	1	0.063	0.081	0.776
Hand Index x Face Index	0.693	1	0.693	0.893	0.346
Dermatoglyphic Index x Face Index	0.502	1	0.502	0.647	0.422
Hand Index x Dermatoglyphic Index x Face Index	0.575	1	0.575	0.741	0.391
Error	129.547	167	0.776		
Total	912.422	176			
Corrected Total	150.434	175			

regression lines for the Hand, Dermatoglyphic, Face, and Total indices versus Rasch logit values (AD/HDness) for males and females, respectively, are presented in Figures 4A,B and 5A,B. The only statistically interesting correlations (Pearson's r's) were in males for Dermatoglyphic Index regressed on the Rasch logit ($F_{1,55} = 3.627$, $P = 0.062$), and Total Index regressed on the Rasch score ($F_{1,55} = 3.811$, $P = 0.056$).

The Pearson's r correlations (one tail) among the indices relative to each other and the Rasch scores are presented in Table 6. After Bonferroni correction the Total Index only correlates significantly with the Dermatoglyphic and Face indices for Males,

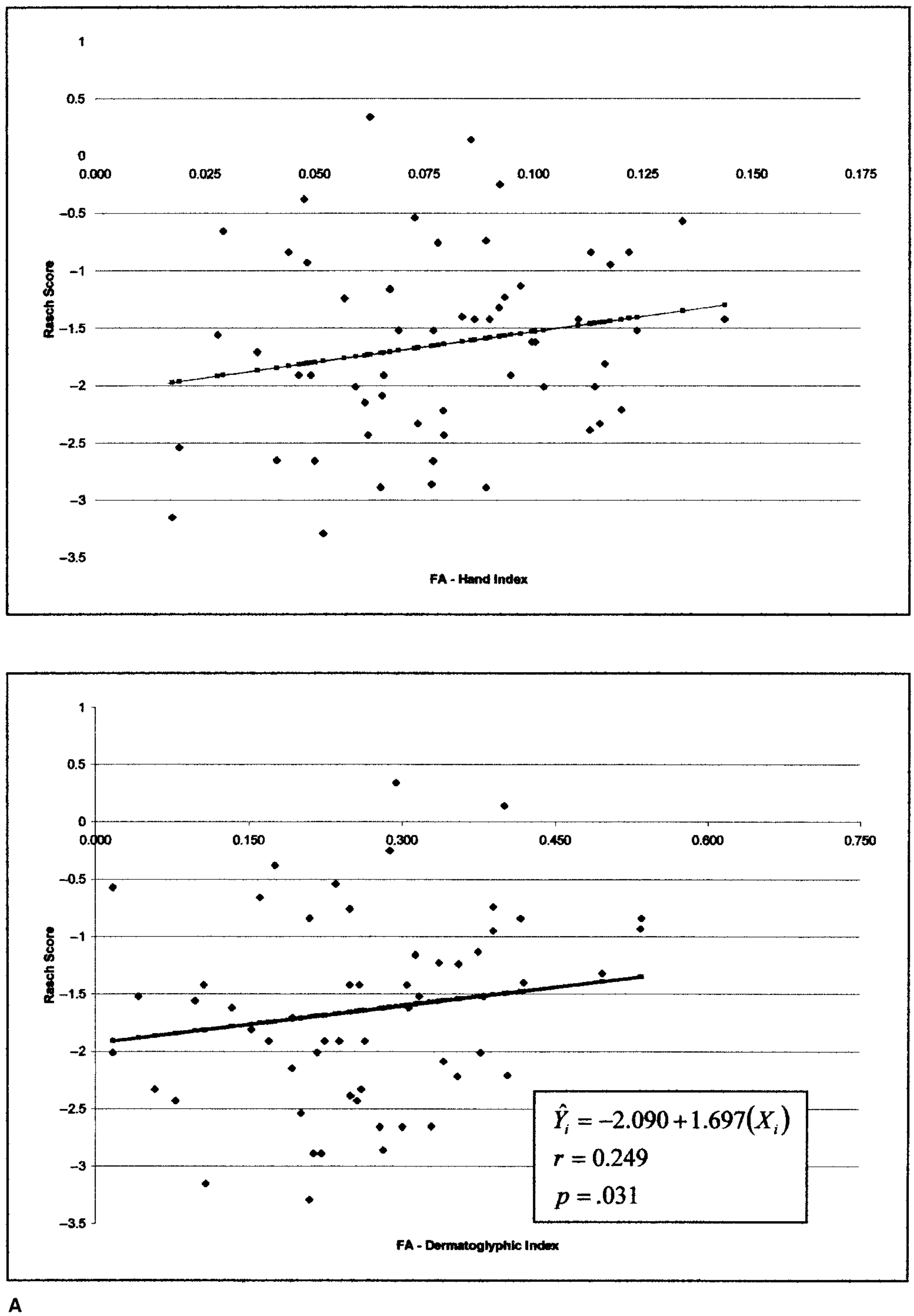
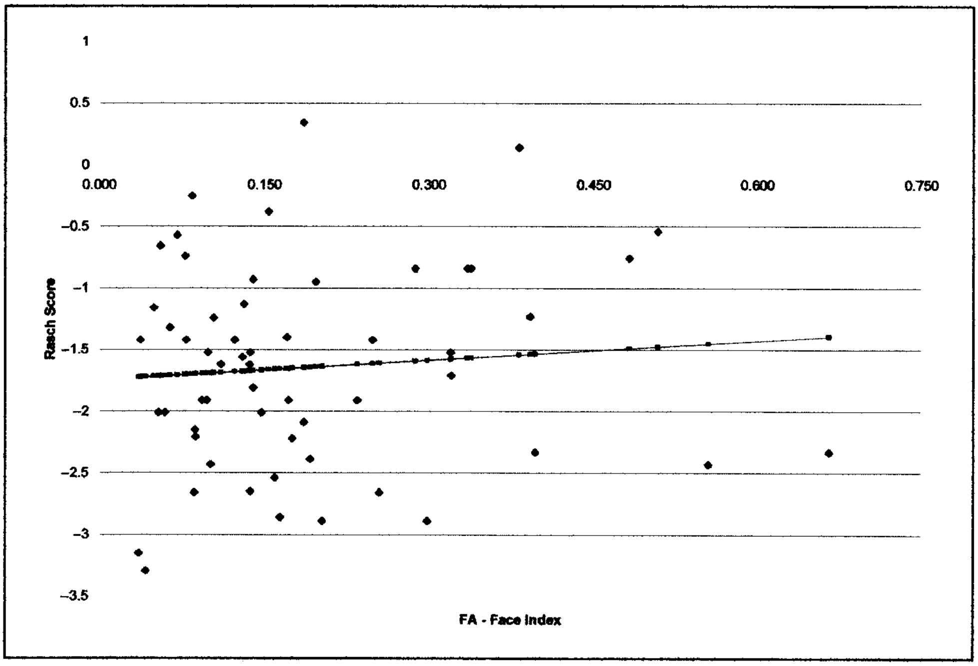
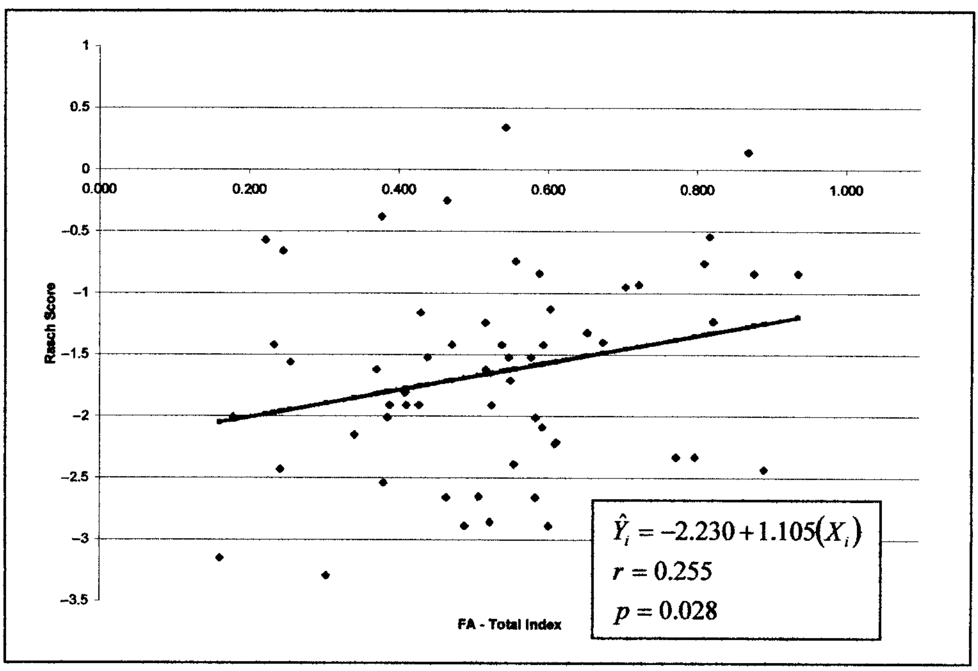


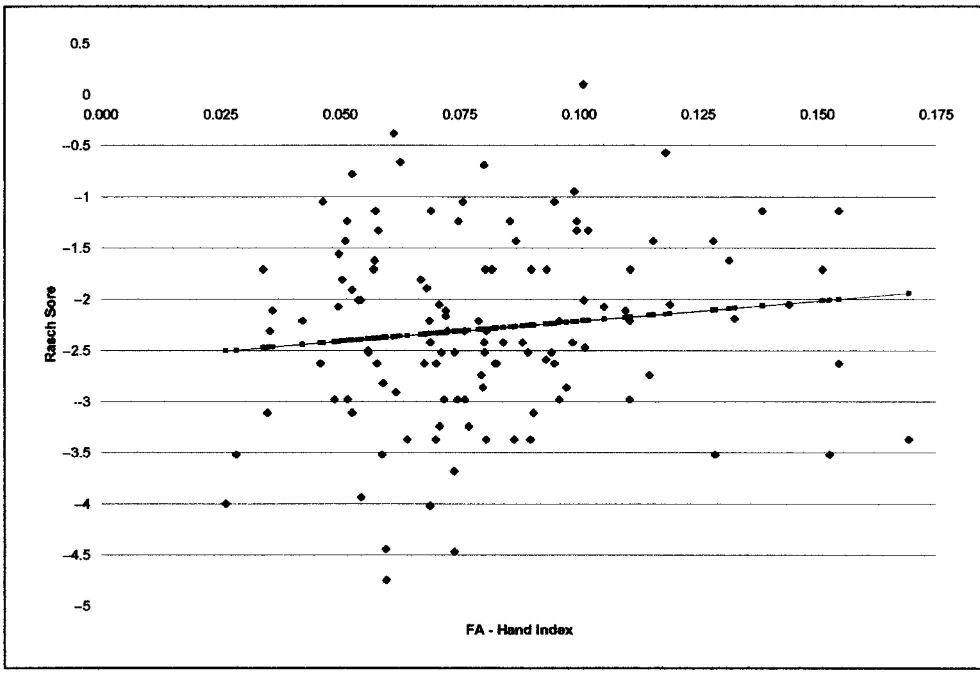
Fig. 4. **A:** Scatterplots of Rasch scores vs. Hand and Dermatoglyphic FA indices, respectively, for males. **B:** Scatterplots of Rasch scores vs. Facial and Total FA indices, respectively, for males.

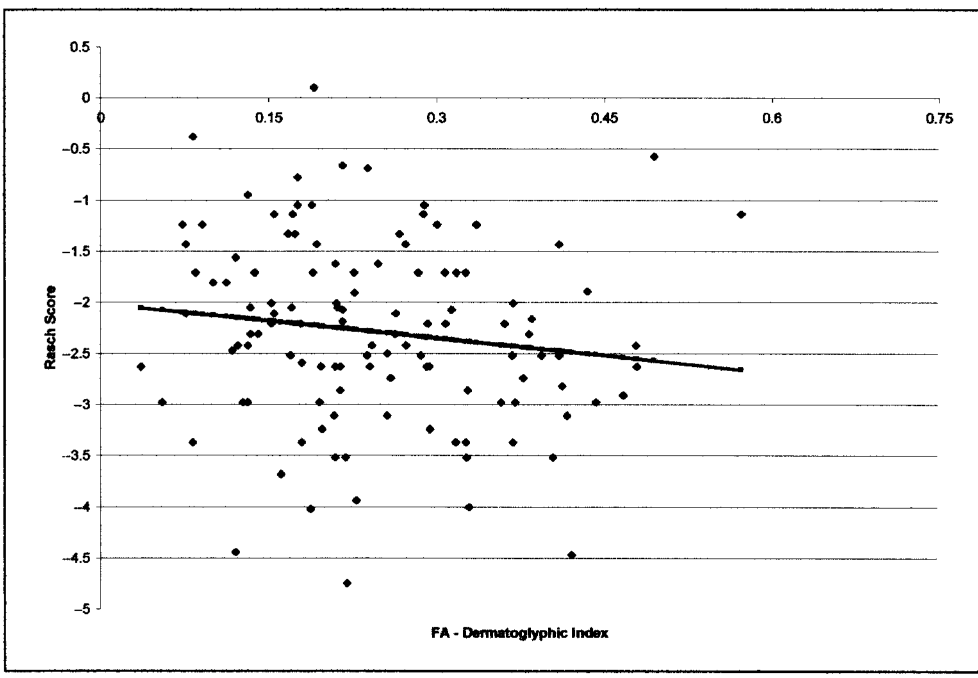




B

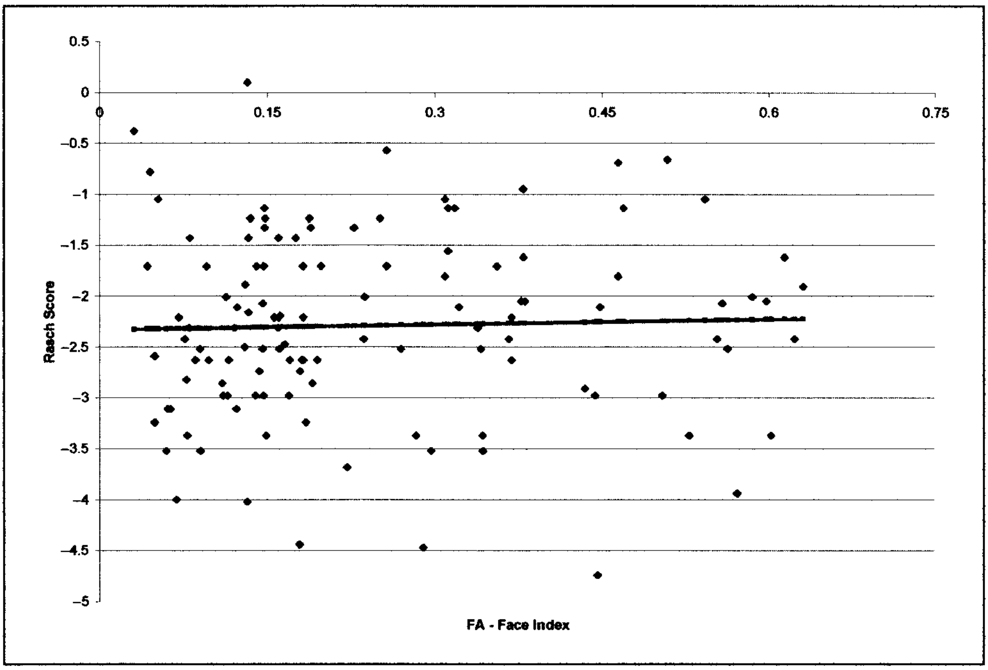
Fig. 4. (Continued.)

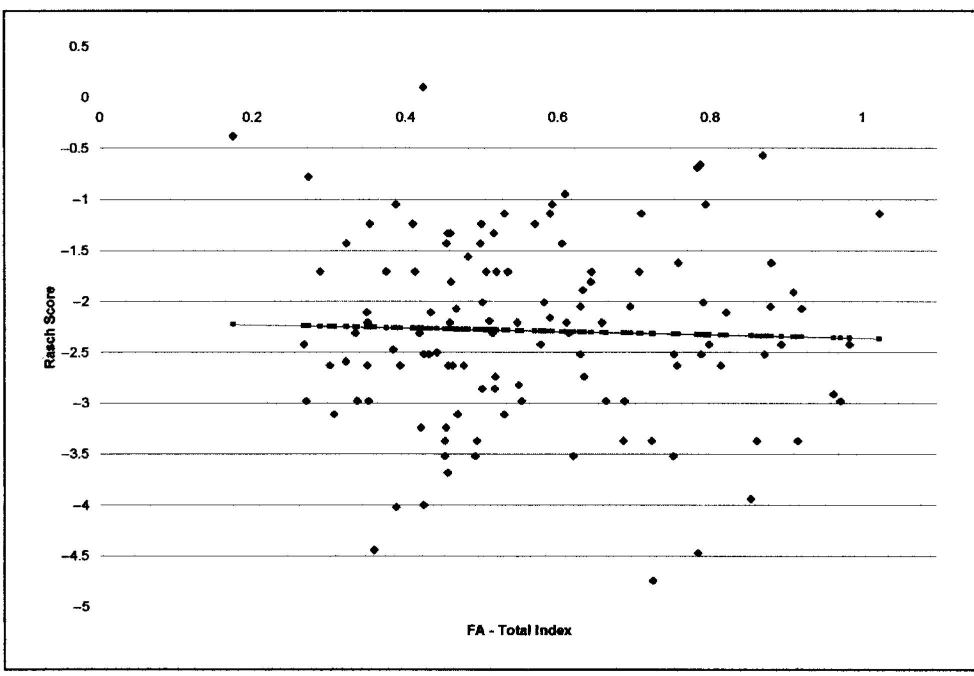




A

Fig. 5. **A:** Scatterplots of Rasch scores vs. Hand and Dermatoglyphic FA indices, respectively, for females. **B:** Scatterplots of Rasch scores vs. Facial and Total FA indices, respectively, for females.





B

Fig. 5. (Continued.)

TABLE 6. Correlation matrices: indices and Rasch score by sex

	FA index *Pearson r* *Sig. (1-tail)*	Hand index	Dermatoglyphic index	Face index	Total index	Rasch score
TOTAL	**Hand Index**	1.000				
		0.000				
	Dermatoglyphic Index	*0.172	1.000			
		0.023	0.000			
	Face Index	−0.051	−0.080	1.000		
		0.505	0.289	0.000		
	Total Index	*0.214	**0.556	**0.770	1.000	
		0.004	0.000	0.000	0.000	
	Rasch Score	0.129	0.010	−0.010	0.017	1.000
		0.087	0.895	0.893	0.819	0.000
MALE	**Hand Index**	1.000				
		0.000				
	Dermatoglyphic Index	*0.178	1.000			
		0.092	0.000			
	Face Index	0.017	−0.015	1.000		
		0.451	0.456	0.000		
	Total Index	*0.283	**0.653	**0.732	1.000	
		0.017	0.000	0.000	0.000	
	Rasch Score	0.194	*0.249	0.089	*0.255	1.000
		0.074	0.031	0.255	0.028	0.000
FEMALE	**Hand Index**	1.000				
		0.000				
	Dermatoglyphic Index	*0.172	1.000			
		0.031	0.000			
	Face Index	−0.087	−0.096	1.000		
		0.174	0.150	0.000		
	Total Index	*0.178	**0.523	**0.785	1.000	
		0.027	0.000	0.000	0.000	
	Rasch Score	0.126	−0.137	0.030	−0.034	1.000
		0.086	0.069	0.372	0.357	0.000

* Significance uncorrected ($P < 0.05$).
** Significant after Bonferroni correction ($P < 0.005$).

Females, and Total Sample. The focus here is whether or not there is a positive correlation between the Rasch and the indices and there does appear to be a trend for a positive correlation between Rasch score and Dermatoglyphic and Total Indices for males.

DISCUSSION

FAs are not much different between the sexes but the behavioral measures provide a striking contrast. FAs in Table 3 are lowest for hand and face and somewhat higher for ridge counts. Males and females differ little except for outer eye width and Total Face Index. Although not evident in head and face measures, Livshits and Smouse (1993) found small variance and covariance differentials in FA between the sexes. Trivers et al. (1999) found significant sex differences in the composite FAs for Jamaican children and boys more symmetric than girls, particularly in the elbow and fourth digit. Wilson and Manning (1996) also found higher FAs in girls relative to boys but the differences were not significant. Thus, there does not seem to be strong evidence for significant sex differences, in general, for FA.

The relatively high level of WURS symptoms in both college males and females summarized in Table 4 relative to Wender's (1995:247) "normal" sample is somewhat intriguing. Others have reported significant levels of AD/HD symptoms in college samples (e.g., Richards et al., 1999; Turnock et al., 1998; Weyandt et al., 1995). In this case it may represent self-selection by students more likely to participate in the study because of concerns about AD/HD symptoms. What may also be likely is that these are issues of salience for this sample. Most were freshmen and sophomores and the challenge of college relative to high school may make an individual more aware of concentration difficulties, inattention, or problems in self-control. A lower performance in college relative to high school may lead some students to question their native abilities.

Table 4 shows that male college students exhibit higher rates of WURS symptoms than do college females. Fossati et al. (2001) also found that male university students exhibited a significantly higher WURS total score than females (in contrast to no difference in psychiatrically identified subjects). This follows from the overall, more frequent rate of diagnosis for AD/HD in males relative to females (e.g., Gingerich et al., 1998). By contrast, the relatively close match between males and females for item level difficulty evident in Figure 3 suggests that the significance of symptoms for the experience of AD/HDness may be similar. This picture may support different polygenic multiple threshold models rather than intrinsic differences in how AD/HD is experienced (Rhee et al., 1999).

The univariate analysis presented in Table 5 also supports a sex difference with the significant interaction effect of sex. There are also statistically significant trends for FA as measured by a Dermatoglyphic Index or as measured by a Total Index to increase with increases in the Rasch logit values for AD/HDness *but only in males*, evident in Figure 4A,B. Again, the correlation matrix of Table 6 also supports this picture. This is consistent with the discovery by Reid and Norvilitis (2000) that anomalous brain lateralization, as reflected in hand, foot, ear, and eye, was associated with AD/HD behaviors measured by the short version of the WURS. This difference is apparent in males and not females and is also consistent with the older literature on minor physical anomalies (MPA), another proxy for developmental instability. MPAs vary along a continuum and are significantly higher in children with "deviant behavior" compared to normal controls and this relationship was strongest in males (Firestone and Peters, 1983).

Each of the indices likely represents somewhat different interacting genetic and environmental pathways. A trend for the Dermatoglyphic FA to increase with increases in AD/HDness may reflect issues that arise early in development. Dermal ridges differentiate during the 3rd and 4th months of fetal life (Holt, 1968). They start to form when the hand is about 3.5 mm in length and do not change after the hand is about 15.6 mm long. Secondary ridges, if they occur, form by the 5th and 6th fetal months. There are then no developmental changes in dermal ridges after the 7th fetal month. Thus, dermal ridges provide a window into growth disturbances during fetal life.

These relationships are weak but are suggestive, considering the small sample size (particularly of males) and may provide an example of how females may be buffered (more canalized) relative to males for some aspects of development relative to environmental insults that may affect behaviors associated with AD/HD (Stinson 1985, 2000).

CONCLUSIONS

The fluctuating asymmetries measured for hands, fingerprints, and face differed little between the sexes except for outer eye width and Total Face Index.

Males relative to females exhibited statistically significantly higher levels of AD/HD symptoms as measured by the WURS and both sexes of this college sample exhibited an intermediate level of symptoms, more than a "normal" sample and less than a clinical sample with AD/HD.

"AD/HDness" does appear to fit the Rasch measurement model well and represents a unidimensional construct. A Rasch measurement model analysis of individual responses to the WURS produced a true interval score (logit) for each person as a measure of individual "AD/HDness." Both sexes expressed similar levels of difficulty in endorsing AD/HD symptoms.

A univariate regression analysis including all the variables for both sexes indicate a significant interaction effect for sex. Regression analyses of individual indices for Hand, Dermatoglyphics, Face, and Total on Rasch logit values (AD/HDness) for males and females show that there are statistical trends for FA to increase (as measured either as a Dermatoglyphic Index or as a Total Index) as the behavioral measure (the Rasch logit values) for AD/HDness increases in males but these trends are not found in females.

ACKNOWLEDGMENTS

We thank the students for patience during measurement, J.T. Manning for answering many questions, and two anonymous reviewers for excellent suggestions. We also thank the Biology Department for use of the digital camera.

LITERATURE CITED

Anastopoulos AD, Shelton TL. 2001. Assessing attention-deficit/hyperactivity disorder. New York: Kluwer Academic/Plenum Publishers.

APA (American Psychiatric Association). 1994. Diagnostic and statistical manual of mental disorders, 4th edition. Washington, DC: American Psychiatric Association.

Auerbach JG, Benjamin J, Faroy M, Geller V, Ebstein R. 2001. DRD4 related to infant attention and information processing: a developmental link to ADHD? Psychiatr Genet 11:31–35.

Baird J, Stevenson JC, Williams DC. 2000. The evolution of ADHD: a disorder of communication? Q Rev Biol 75:17–35.

Barkley RA. 1997a. Behavioral inhibition, sustained attention, and executive functions: constructing a unifying theory of ADHD. Psychol Bull 121:65–94.

Barkley RA. 1997b. ADHD and the nature of self-control. New York: Guilford Press.

Barkley RA. 2001. The executive functions and self-regulation: an evolutionary neuropsychological perspective. Neuropsych Rev 11:1–29.

Blum K, Braverman ER, Holder JM, Lubar JF, Monastra VJ, Miller D, Lubar JO, Chen TJ, Comings DE. 2000. Reward deficiency syndrome: a biogenetic model for the diagnosis and treatment of impulsive, addictive, and compulsive behaviors. J Psychoactive Drugs 32:1–112.

Bond TG, Fox CM. 2001. Applying the Rasch model: fundamental measurement in the human sciences. Mahwah, NJ: Lawrence Erlbaum Associates.

Casey BJ, Castellanos FX, Giedd JN, Marsh WL, Hamburger SD, Schubert AB, Vauss YC, Vaituzis AC, Dickstein DP, Sarfatti SE, Rapoport JJ. 1997. Implication of right frontostriatal circuitry in response inhibition and attention-deficit/hyperactivity disorder. J Am Acad Child Psy 36:374–383.

Castellanos FX, Giedd JN, Eckburg P, Marsh WL, Vaituzis AC, Kaysen D, Hamburger SD, Rapoport JL. 1994. Quantitative morphology of the caudate nucleus in attention deficit hyperactivity disorder. Am J Psychiatry 151:1791–1796.

Castellanos FX, Giedd JN, Marsh WL, Hamburger SD, Vaituzis AC, Dickstein DP, Sarfatti SE, Vauss YC, Snell JW, Lange N, Kaysen D, Krain AL, Ritchie GF, Rajapakse JC, Rapoport JL. 1996. Quantitative brain magnetic resonance imaging in attention-deficit hyperactivity disorder. Arch Gen Psychiat 53:607–616.

Comings DE. 2001. Clinical and molecular genetics of ADHD and Tourette syndrome. Two related polygenic disorders. Ann NY Acad Sci 931:50–83.

Comings DE, Gade-Andavolu R, Gonzalez N, Wu S, Muhleman D, Blake H, Chiu F, Wang E, Farwell K, Darakjy S, Baker R, Dietz G, Saucier G, MacMurray JP. 2000. Multivariate analysis of associations of 42 genes in ADHD, ODD and conduct disorder. Clin Genet 58:31–40.

Cox DJ, Merkel L, Kovatchev B, Seward R. 2000. Effect of stimulant medication on driving performance of young adults with attention-deficit hyperactivity disorder: a preliminary double-blind placebo controlled trial. J Nerv Ment Dis 188:230–234.

DeRoos Y, Allen-Meares P. 1998. Application of Rasch analysis: exploring differences in depression between African-American and White children. J Soc Serv Res 23:93–107.

Dufour KW, Weatherhead PJ.1996. Estimation of organism-wide asymmetry in red-winged blackbirds and its relation to studies in mate selection. Proc R Soc Lond B Biol 263:769–775.

Durfee KE. 1974. Crooked ears and the bad boy syndrome: asymmetry as an indicator of minimal dysfunction. B Menninger Clin 38:305–316.

Edelbrock CS, Rende R, Plomin R, Thompson L. 1995. A twin study of competence and problem behavior in childhood and early adolescence. J Child Psychol Psyc 36:775–786.

Ernst M, Zametkin AJ, Matochik JA, Jons PH, Cohen RM. 1998. DOPA decarboxylase activity in attention deficit hyperactivity disorder in adults. A [fluorine-18] fluorodopa positron emission tomographic study. J Neurosci 18:5901–5907.

Faraone SV, Doyle AE, Mick E, Biederman J. 2001. Meta-analysis of the association between the 7-repeat allele of the dopamine D(4) receptor gene and attention deficit hyperactivity disorder. Am J Psychiat 158:1052–1057.

Filipek PA, Semrud-Clikeman M, Steingard RJ, Renshaw PF, Kennedy DN, Biederman J. 1997. Volumetric MRI analysis comparing subjects having attention-deficit hyperactivity disorder with controls. Neurology 48:589–601.

Firestone P, Peters S. 1983. Minor physical anomalies and behavior in children: a review. J Autism Dev Disord 13:411–425.

Firestone P, Peters S, Rivier M, Knights RM. 1978. Minor physical anomalies in hyperactive, retarded, and normal children and their families. J Child Psychol Psychiatry 19:155–160.

Fisher SE, Francks C, McCracken JT, McGough JJ, Marlow AJ, MacPhie IL, Newbury DF, Crawford LR, Palmer CG, Woodward JA, Del'Homme M, Cantwell DP, Nelson SF, Monaco AP, Smalley SL. 2002. A genomewide scan for loci involved in attention-deficit/hyperactivity disorder. Am J Hum Genet 70: 1183–1196.

Fossati A, Di Ceglie A, Acquarini E, Donati D, Donini M, Novelia L, Maffei C. 2001. The retrospective assessment of childhood attention deficit hyperactivity disorder in adults: reliability and validity of the Italian version of the Wender Utah Rating Scale. Compr Psychiatry 42:326–336.

Gangestad SW, Thornhill R. 1999. Individual differences in developmental precision and fluctuating asymmetry: a model and its implications. J Evol Biol 12:402–416.

Gaub M, Carlson CL. 1997. Gender differences in ADHD: a meta-analysis and critical review. J Am Acad Child Psychiatry 36:1036–1045.

Gillis JJ, Gilger JW, Pennington BF, DeFries JC. 1992. Attention deficit disorder in reading-disabled twins: evidence for a genetic etiology. J Abnorm Child Psychol 20:303–315.

Gingerich KJ, Turnock P, Litfin JK, Rosén A. 1998. Diversity and attention deficit hyperactivity disorder. J Clin Psychol 54:415–426.

Holt SB. 1968. The genetics of dermal ridges. Springfield, IL: Charles C. Thomas.

Hume DK, Montgomerie R. 2001. Facial attractiveness signals different aspects of "quality" in women and men. Evol Hum Behav 22:93–112.

Hynd GW, Hern KL, Novey ES, Eliopulos D, Marshall R, Gonzalez JJ, Voeller KK. 1993. Attention deficit-hyperactivity disorder and asymmetry of the caudate nucleus. J Child Neurol 8:339–347.

Krebs CJ. 1989. Ecological methodology. New York: Harper and Row.

Krouse JP, Kauffman JM. 1982. Minor physical anomalies in exceptional children: a review and critique of research. J Abnorm Child Psychol 10:247–264.

Lara-Muñoz C, Herrera-García S, Romero-Ogawa T, Torija L, García ML. 1998. Características psicométricas de la Escala de Evaluación Retrospectiva del

Trastorno por Déficit de Atención e Hiperactividad Wender-Utah en español. Actas Luso-Esp Neur 26:165–171.
Leamy L. 1999. Heritability of directional and fluctuating asymmetry for mandibular characters in random-bred mice. J Evol Biol 12:146–155.
Leung B, Forbes MR. 1997. Modeling fluctuating asymmetry in relation to stress and fitness. Oikos 78:397–405.
Leung B, Forbes MR, Houle D. 2000. Fluctuating asymmetry as a bioindicator of stress: comparing efficacy of analyses involving multiple traits. Am Nat 155:101–115.
Levy F, Hay DA, McStephen M, Wood C, Waldman I. 1997. Attention-deficit hyperactivity disorder: a category or a continuum? Genetic analysis of a large-scale twin study. J Am Acad Child Psychol 36:737–744.
Likert R. 1932. A technique for the measurement of attitudes. Arch Psychol 140:55.
Linacre J, Wright BD. 2000. WINSTEPS: multiple choice, rating scale and partial credit Rasch analysis (computer software). Chicago, IL: MESA Press.
Livshits G, Kobyliansky K. 1987. Dermatoglyphic traits as possible markers of developmental processes in humans. Am J Med Genet 26:111–122.
Livshits G, Smouse PE. 1993. Multivariate fluctuating asymmetry in Israeli adults. Hum Biol 65:547–578.
Markow TA. 1992. Genetics and developmental stability: an integrative conjecture on aetiology and neurobiology of schizophrenia. Psychol Med 22:295–305.
Massof RW. 2002. The measurement of vision disability. Optom Vision Sci 79L516–552.
Miller GM, De La Garza RD 2nd, Novak MA, Madras BK. 2001. Single nucleotide polymorphisms distinguish multiple dopamine transporter alleles in primates: implications for association with attention deficit hyperactivity disorder and other neuropsychiatric disorders. Mol Psychiatry 6:50–58.
Møller AP, Swaddle JP. 1997. Asymmetry, developmental stability, and evolution. New York: Oxford University Press.
Nolan EE, Gadow KD, Sprafkin J. 2001. Teacher reports of *DSM-IV* ADHD, ODD and CD symptoms in schoolchildren. J Am Acad Child Psychol 40:241–249.
Pechenkina EA, Benfer RA Jr, Vershoubskaya GG, Kozlov AI. 2000. Genetic and environmental influence on the asymmetry of dermatoglyphic traits. Am J Phys Anthropol 111:531–543.
Quinn PO, Rapoport JL. 1974. Minor physical anomalies and neurologic status in hyperactive boys. Pediatrics 53:742–747.
Quist JF, Barr CL, Schachar R, Roberts W, Malone M, Tannock R, Basile VS, Beitchman J, Kennedy JL. 2000. Evidence for the serotonin HTR2A receptor gene as a susceptibility factor in attention deficit hyperactivity disorder (ADHD). Mol Psychiatry 5:537–541.
Rapoport JL, Quinn PO. 1975. Minor physical anomalies (stigmata) and early developmental deviation: a major biologic subgroup of "hyperactive children." Int J Ment Health 4:29–44.
Reid HM, Norvilitis JM. 2000. Evidence for anomalous lateralization across domain in ADHD children as well as adults identified with the Wender Utah rating scale. J Psychiatr Res 34:311–316.
Rhee SH, Waldman ID, Hay DA, Levy F. 1999. Sex differences in genetic and environmental influences on DSM-III-R attention-deficit/hyperactivity disorder. J Abnorm Psychol 108:24–41.
Richards TL, Rosen LA, Ramirez CA. 1999. Psychological functioning differences among college students with confirmed ADHD, ADHD by self-report only, and without ADHD. J Coll Student Dev 40:299–304.
Rossini ED, O'Connor MA. 1995. Retrospective self-reported symptoms of attention-deficit hyperactivity disorder: reliability of the Wender Utah Rating Scale. Psychol Rep 77:751–754.
Scahill L, Schwab-Stone M. 2000. Epidemiology of ADHD in school-age children. Child Adolesc Psychol Clin 9:541–555.
Shackelford TK, Larsen RJ. 1997. Facial asymmetry as an indicator of psychological, emotional, and physiological stress. J Pers Soc Psychol 72:456–466.
Sonuga-Barke, EJS. 2002. Psychological heterogeneity in AD/HD—a dual pathway model of behaviour and cognition. Behav Brain Res 130:29–36.
Stein MA, Sandoval R, Szumowski E, Roizen N, Reinecke MA, Blondis TA, Klein Z. 1995. Psychometric characteristics of the Wender Utah Rating Scale (WURS): reliability and factor structure for men and women. Psychopharmacol Bull 31:425–433.
Stevenson JC, Williams DC. 2000. Parental investment, self-control, and sex differences in the expression of ADHD. Hum Nat 11:405–422.
Stinson S. 1985. Sex differences in environmental sensitivity during growth and development. Yearb Phys Anthropol 28:123–147.
Stinson S. 2000. Growth variation: biological and cultural factors. In: Stinson S, Bogin B, Huss-Ashmore R, O'Rourke D, editors. Human biology: an evolutionary and biocultural perspective. New York: Wiley-Liss. p 425–463.
Tannock R. 1998. Attention deficit hyperactivity disorder: advances in cognitive, neurobiological, and genetic research. J Child Psychol Psychiatry 39:65–99.
Thornhill R, Møller AP. 1997. Developmental stability, disease and medicine. Biol Rev 72:407–548.
Trivers R, Manning JT, Thornhill R, Singh D, McGuire M. 1999. Jamaican symmetry project: long-term study of fluctuating asymmetry in rural Jamaican children. Hum Biol 71:417–430.
Tukey J. 1977. Exploratory data analysis. Princeton, NJ: Addison-Wesley.
Turnock P, Rosén LA, Kaminski PL. 1998. Differences in academic coping strategies of college students who self-report high and low symptoms of attention deficit hyperactivity syndrome. J Coll Stud Dev 39:484–493.
Waldrop MF, Halverson CF Jr. 1971. Minor physical anomalies and hyperactive behavior in young children. In: Hellmuth J, editor. Exceptional infant: studies in abnormalities. London: Butterworths. p 343–379.
Ward MF, Wender PH, Reimherr FW. 1993. The Wender Utah Rating Scale: an aid in the retrospective diagnosis of childhood attention deficit hyperactivity disorder. Am J Psychiatry 150:885–890.
Waynforth D. 1998. Fluctuating asymmetry in human male life-history traits in rural Belize. Proc R Soc Lond B Biol 265:1497–1501.
Wender P. 1995. Attention-deficit hyperactivity disorder in adults. New York: Oxford University Press.
Wender P. 1997. Attention deficit hyperactivity disorder in adults: a wide view of a widespread condition. Psychiatry Ann 27:556–562.
Wender PH, Reimherr FW, Wood DR. 1981. Attention deficit disorder ('minimal brain dysfunction') in adults. Arch Gen Psychiatry 38:449–456.
Wender PH, Ward MF, Reimherr FW, Marchant BK. 2000. ADHD in adults. J Am Acad Child Psychol 39:543.
Weyandt LL, Linterman I, Rice JA. 1995. Reported prevalence of attentional difficulties in a general sample of college students. J Psychopathol Behav 17:293–304.

Wilson JM, Manning JT. 1996. Fluctuating asymmetry and age in children: evolutionary implications for the control of developmental stability. J Hum Evol 30:529–537.
Zahn-Waxler C, Schmitz S, Fulker D, Robinson J. 1996. Behavior problems in 5-year-old monozygotic and dizygotic twins: genetic and environmental influences, patterns of regulation, and internalization of control. Dev Psychopathol 8:103–122.
Zar JH. 1996. Biostatistical analysis. Upper Saddle River, NJ: Prentice-Hall.