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Masculinized Finger-Length Ratios of Boys, but Not Girls, Are Associated With Attention-Deficit/Hyperactivity Disorder

Michelle M. Martel,

Department of Psychology, Michigan State University, and Learning Support Center of Child Psychology, Texas Children's Hospital

Kyle L. Gobrogge,

Department of Psychology, Florida State University

S. Marc Breedlove, and

Department of Psychology and the Neuroscience Program, Michigan State University

Joel T. Nigg

Department of Psychology, Michigan State University

Abstract

Gonadal hormones may exert permanent organizational effects on sexually dimorphic finger-length ratios and sexually dimorphic behavior expressed in childhood attention deficit–hyperactivity disorder (ADHD). This study extended recent work examining associations between finger-length ratios (specifically, 2D:4D) and ADHD in a well-characterized, clinically diagnosed, community-recruited sample of boys and girls. A multistage, diagnostic procedure was utilized to identify 113 children with ADHD and 137 non-ADHD comparison children. Right-hand digit ratios showed significant mean differences by gender, as well as associations with ADHD diagnosis. Boys with ADHD had more masculinized digit ratios than control-group boys. More masculine right 2D:4D and 3D:4D ratios were correlated with parent- and teacher-rated inattentive and hyperactive–impulsive symptoms in boys but not in girls. Masculinized finger-length ratios were associated with hyperactive–impulsive and oppositional–defiant symptoms, but associations were largest with symptoms of inattention. It is concluded that prenatal, organizational effects of gonadal hormones may play a role in the development of ADHD and contribute to explaining sex differences in the prevalence rates of this childhood disorder.

Keywords

testosterone; finger-length ratio; 2D:4D; ADHD; sex differences

Males are more vulnerable than females to developing a range of learning and behavioral disorders during childhood. Attention deficit–hyperactivity disorder (ADHD) is one of the most common and impairing sexually dimorphic clinical psychopathologies, with boys about three times more likely to be identified with ADHD than girls in population samples (American Psychiatric Association, 2000; Pastor & Reuben, 2002; Wolraich, Hannah, Baumgaertel, & Feurer, 1998). In addition, there may be sex differences in the correlates of the disorder. Although boys with ADHD appear to be more inattentive and hyperactive–impulsive than girls

with the disorder, particularly in school (Gaub & Carlson, 1997; Gershon, 2002; Hartung et al., 2002), girls with ADHD may have worse cognitive impairment than boys with the disorder (Biederman et al., 1999; Gaub & Carlson, 1997; Newcorn et al., 2001). The precise neurobiological mechanisms underlying sex differences in the prevalence rates of developmental learning and behavioral disorders are complex and underresearched.

The male sex bias in ADHD and its precursor behaviors is apparent during early preschool years (Hart, Lahey, Loeber, Applegate, & Frick, 1995), a period marked by rapid neural organization (Breedlove & Hampson, 2002). Males are typically exposed to testosterone perinatally, whereas females develop in the relative absence of testosterone (Morris, Jordan, & Breedlove, 2004). This differential exposure to steroid hormones produces specific male-like and/or female-like brain-behavior patterns (Hines, 2002). Therefore, these prenatal events may be related to sex differences in prevalence rates of ADHD. However, research on hormonal effects in ADHD is sparse, despite consensus over a decade ago that such research was needed (Arnold, 1996).

Unfortunately, measuring prenatal levels of steroid hormones and patterns of childhood behavior are both invasive and costly, respectively. Therefore, one potential indirect measure of steroid hormone exposure is the relative length and pattern of phalangeal digits. On average, males have a shorter index finger (2D) relative to ring finger (4D), whereas females have a 2D:4D ratio that is nearly equal (Manning, 2002). These sex differences emerge by the 14th week of fetal growth (Malas, 2006; Manning et al., 2005; McIntyre, Cohn, & Ellison, 2006; Trivers, Manning, & Jacobson, 2006). Longitudinal research suggests that the pattern of 2D:4D ratios may change slightly over the course of development, but this growth is not associated with rank order changes in ratios (McIntyre et al., 2006; Trivers, Manning, & Jacobson, 2006). These findings are consistent with the notion that prenatal androgen exposure influences digit ratio development.

Research examining finger-length ratios in individuals with congenital adrenal hyperplasia (CAH), a condition marked by high levels of exposure to prenatal androgen, is also consistent with organizational theories of prenatal androgen influences on finger-length ratios. Girls with CAH exhibit lower 2D:4D digit ratios, compared with same-sex controls (Brown, Hines, Fane, & Breedlove, 2002; Okten, Kalyancu, & Yaris, 2002). In addition, Manning, Scutt, Wilson, and Lewis-Jones (1998) found a negative correlation between circulating testosterone and digit ratios in men. Other research suggests that lower finger-length ratios reflect higher prenatal testosterone exposure, whereas higher 2D:4D ratios are associated with higher prenatal estrogen exposure (Williams, Greenhalgh, & Manning, 2003). It should be noted that there are substantial ethnic differences in finger-length ratios, in addition to sex differences (Cohen-Bendahan, van de Beek, & Berenbaum, 2005).

Several studies have examined associations between finger-length patterns and childhood behavior relevant to ADHD. Williams et al. (2003) examined right 2D:4D ratio associations with childhood problem behaviors measured by the Social Difficulties Questionnaire and the Social Cognition Questionnaire in a community sample of approximately 200 preschool children. Lower 2D:4D ratios were related to increased levels of hyperactivity in girls but not boys. Fink, Manning, Williams, and Podmore-Nappin (2007) examined relationships between digit ratios and child problem behavior, measured by caregiver report on the Strength and Difficulties Questionnaire, in a sample of 58 Caucasian children (ages 5–7 years; 25 boys, 33 girls) from the United Kingdom and 56 Caucasian children (ages 6–11 years; 29 boys, 27 girls) from Austria. In the U.K. sample, hyperactivity–inattention and conduct problems were correlated with lower 2D:4D ratios in boys but not in girls. In the Austrian sample, lower 2D:4D ratios was related to more externalizing problems in girls and more social problems in boys (Fink, Manning, Williams, & Podmore-Nappin, 2007). All studies found that more masculine

ratios were correlated with increased symptoms; however, some studies found these relations only in girls, whereas other studies found these relations only in boys.

Another study examined finger-length ratios in relation to ADHD symptoms, measured by the Wender Utah Rating Scale and in accordance with the *Diagnostic and Statistical Manual of Mental Disorders* (4th ed.; *DSM-IV*; American Psychiatric Association, 2004), in a college sample of 238 women and men between the ages of 18 and 47 (Stevenson et al., 2007). In this sample (which was primarily female), more masculine left 2D:4D was related to higher numbers of both inattentive and hyperactive–impulsive ADHD symptoms but only in females (Stevenson et al., 2007). Together, the results of these initial studies provided partial support, using nondiagnosed samples, for the hypothesis that prenatal androgen exposure may increase childhood and adolescent problem behavior, including inattentive and hyperactive–impulsive ADHD symptoms, in girls and boys.

Only two previous studies examined finger-length ratios in clinically diagnosed samples. de Bruin, Verheij, Wiegman, and Ferdinand (2006) examined 144 boys with psychiatric disorders and 96 boys without psychiatric disorders. Consistent with other studies, boys with autism/Asperger disorder and ADHD/oppositional–defiant disorder had lower digit ratios than boys with anxiety disorders, and boys with autistic spectrum disorders had more masculine digit ratios than boys without psychiatric disorders. This study suggested that higher prenatal androgen exposure may increase risk for a range of childhood male-biased disorders.

McFadden, Westhafer, Pasanen, Carlson, and Tucker (2005) utilized a well-characterized, clinical sample, similar to that used in the present investigation, to study finger-length ratios and click-evoked otoacoustic emissions (CEOAEs). They noted that CEOAEs are weak sounds made by the cochlea that are stronger in females, are present early in life, and appear to be affected by exposure to androgens during early development. Children (ages 7–15) with the inattentive subtype of ADHD ($n = 61$) had smaller CEOAEs and smaller finger-length ratios—specifically right 2D: 5D, 3D:5D, and 4D:5D—than children with the combined subtype of ADHD or typically developing control children, suggesting higher prenatal androgen exposure in the inattentive group. Thus, two studies utilizing diagnosed child samples (de Bruin et al., 2006; McFadden et al., 2005) found that children with externalizing disorders had more masculinized finger-length ratio patterns, findings that are consistent with the idea that higher levels of prenatal testosterone exposure are related to increased incidence of externalizing disorders and increased inattentive symptoms in childhood.

We sought to replicate and extend recent work examining associations between finger-length ratios and ADHD by investigating distinct effects on inattentive and hyperactive–impulsive symptoms and ADHD subtypes in a large, well-characterized, clinically evaluated sample of children with ADHD. In addition, handedness and number of older brothers were examined in relation to finger-length ratios and ADHD symptoms in a more exploratory fashion.

Method

Participants

Participants were 113 children with ADHD and 137 non-ADHD comparison children. There were 149 boys and 101 girls in the total group, and 72 boys and 41 girls in the ADHD group. Twenty-six percent of the total sample was considered to be ethnic minorities. Sample descriptive statistics are shown in Table 1. The two groups had similar percentages of males. All participants, both non-ADHD controls and children with ADHD, were recruited from the local community through widespread public advertisements and announcements, mass mailings, and outreach. Invitations and announcements sought children, between the ages of 6 and 18 years, with possible or definite problems with inattention or hyperactivity and

impulsivity as well as healthy children with no apparent learning or behavioral problems. The sampling strategies were intended to obtain as representative and diverse a sample as possible while maintaining a similar recruitment source for all children. All potential participants underwent the same case finding procedure involving a multistage screening process.

The first stage was a screen that precluded any children who took hormones or slow-acting psychotropic medication (e.g., prednisone, birth control, antidepressant, antipsychotic, or anticonvulsant medications); any children who had major medical or neurological conditions, including genetic, gonadal, or hormonal abnormalities, as reported by the parent; and any children who did not speak English as their first language. At the second stage, remaining children's parents and teachers completed normative behavior rating scales: the ADHD Rating Scales (DuPaul, Power, Anastopolous, & Reid, 1998), the Parent and Teacher Behavior Assessment System for Children (Reynolds & Kamphaus, 1992), and the Conners (1997) Parent and Teacher Rating Scales—Revised Short Forms. These scales were chosen on the basis of their empirically validated cutoffs, thorough symptom coverage, and adequate reliability and validity. Furthermore, these scales are among the most widely accepted rating scales currently used and were judged to have the best sensitivity and specificity data for screening (Ostrander, Weinfurt, Yarnold, & August, 1998; Power et al., 1998). Test-retest reliabilities for these scales ranging from .62 to .95, internal reliabilities in this study ranging from .84 to .96, and validity coefficients ranging from .78 to .98 (Conners, 1997; DuPaul et al., 1998; Reynolds & Kamphaus, 1992) were judged to be sufficient because the questionnaires were only used for initial screening purposes.

For the third stage, participants completed a semistructured clinical interview (i.e., the Schedule for Affective Disorders and Schizophrenia for School-Age Children, Epidemiologic Version; KSADS-E; Orvaschel, Tabrizi, & Chambers, 1980). The KSADS-E is a widely used, well-established, standard method of interviewing for child disorders, and it provides a thorough coverage of childhood disease sequelae (Biederman et al., 1990; 1992). In addition to ADHD, the interview assessed all Axis I disorders (e.g., anxiety disorders, mood disorders, posttraumatic stress disorder, obsessive-compulsive disorder, tic disorders, oppositional defiant disorder, and conduct disorder). IQ and achievement were assessed with the Wechsler Intelligence Scale for Children (Wechsler, 1949), and the Wechsler Individual Achievement Test (2nd ed.; Wechsler, 2001). Basic Reading and Mathematical Reasoning subtests were used to enable evaluation of learning disabilities or cognitive impairments. The entire file for each study proband was then reviewed, and a "best estimate" diagnostic process was implemented, in which a board-certified child psychiatrist and licensed clinical psychologist independently arrived at a clinical decision regarding ADHD diagnosis, ADHD subtype, and presence of comorbid disorders. Their agreement rates were acceptable (all kappas > 0.80). Disagreements were always able to be resolved by conference, and no participants were rejected on the basis of clinician disagreement.

Exclusion criteria—The presence of autistic disorder, mental retardation (IQ less than 70), schizophrenia, bipolar disorder, fetal alcohol syndrome, known neurological disorder, cerebral palsy, Tourette's disorder, and genetic, gonadal, and/or hormonal abnormalities were considered exclusionary criteria.

ADHD symptom counts—Parent and teacher symptom counts were used as a continuous measure of inattentive and hyperactive-impulsive ADHD symptoms in some analyses. Parent-rated ADHD symptoms were obtained with the KSADS-E, and teacher-rated ADHD symptoms were obtained with the ADHD Rating Scale (a rating of 2 or 3 counting as present on the 0–3 scale). After the *DSM-IV* field trials (Lahey et al., 1994), we chose to create symptom count scores by combining across reporters; we counted a symptom as present if it was endorsed by either a parent or a teacher. These scores are emphasized, although effects within reporter are

also noted because of the marked discrepancies observed sometimes between parent and teacher ratings (Power et al., 1998; Verhulst, Koot, & Van der Ende, 1994).

Measures

Handedness—Handedness was assessed with the Edinburgh Handedness Questionnaire (Oldfield, 1971). Handedness was coded as right or nonright (i.e., left-handed or ambidextrous).

Finger-length measurements—We obtained finger-length images using a photocopier with 2400×600 dots per inch black resolution. Participants were asked to remove any jewelry and place their two hands, side by side, fingers together, onto a clear template marked with a ruler. A white plastic bag filled with rice was placed over their hands to provide a background contrast for optimal image resolution and to standardize pressure on the hands to obtain flat scans. Hands were then photocopied. Two independent raters measured (in centimeters, to the nearest millimeter) the index, middle, ring, and little finger on each hand from the hand scans. The ratings were reliable (all interrater $r_s > .91$; all $p_s < .01$), with an average correlation between raters' measurement of finger lengths of .97. Ratios were then computed between each of the pairs of fingers on both hands. For example, the length of the right index finger was divided by the length of the right ring finger to calculate right 2D:4D. Each finger-length ratio was calculated in this manner. In addition, the examiner asked the child's attending parent (most often the mother) a series of questions regarding the number of older and younger siblings of the child proband, birth order, and the presence of any miscarriages (and sex, if known) before the birth of the child proband.

Data Analysis Rationale

First we conducted a multivariate ANOVA (MANOVA) to examine sex differences in digit ratios, followed by univariate t tests. Ethnicity was covaried in all analyses involving finger-length ratios, because finger-length ratios also show ethnic differences. To further control for ethnic differences in finger-length ratios, we also conducted analyses in a subsample entirely comprised of Caucasians. Preliminary tests of Sex $\times$ Predictor (i.e., finger-length ratio) interactions predicting ADHD symptoms were all nonsignificant ($p > .05$), as tested by regressions and MANOVA. We therefore conducted primary analyses on the pooled data set of boys and girls because effects of testosterone are theorized to be similar in boys and in girls, albeit operating at far lower prenatal exposure levels in girls. This strategy also maximized statistical power. Sex was covaried as a precaution, although groups were well matched with regard to percentage of males. Because of its interest, we note specific effects for boys and girls where relevant.

Results

To address potential Type I error in view of the number of ratios examined, we conducted a multivariate analysis of covariance (MANCOVA) to confirm expected sex differences. When all finger-length ratios were analyzed together, covarying ethnicity, results showed significant sex differences, $F(12, 235) = 2.68, p < .01, \eta^2 = 0.12$. When the right and left hand ratios were examined separately with a MANOVA, again covarying ethnicity, significant sex differences were found for the right, $F(6, 242) = 3.33, p < .01, \eta^2 = 0.08$; and left hand ratios, $F(6, 241) = 3.56, p < .01, \eta^2 = 0.08$. We therefore examined all individual ratio comparisons without correction, using t tests, to evaluate which ratios differed between boys and girls.

As shown in Table 2, the right 2D:4D, 2D:5D, 2D:3D, 3D:4D, 3D:5D ratios and the left 2D:5D and 4D:5D ratios showed significant mean differences by sex (all $p_s < .05$). Within the Caucasian subsample, the same pattern of significant sex differences emerged, with the

exceptions that the right 4D:5D ratio also exhibited significant sex differences in the expected direction, whereas the sex difference in the left 4D:5D ratio was not statistically significant. Male digit ratios were smaller for all of the mean differences that were statistically significant; therefore, we regard smaller ratios as more masculine (consistent with expectations from the literature). Most of the finger-length ratios that exhibited significant sex differences in the present study have also been observed in previous work (de Bruin et al., 2006; Fink et al., 2007; McFadden & Shubel, 2002; McFadden et al., 2005; Stevenson et al., 2007; Williams et al., 2003). To further control Type I error, we selected a subset of finger-length ratios to analyze further on the basis of conceptual and empirical grounds; these were the right 2D:4D, 2D:5D, and 3D:4D ratios. The right 2D:4D ratio was emphasized because it has been well validated with regard to links with prenatal testosterone (Malas et al., 2006; Manning et al., 1998) and has been studied most in relation to ADHD (de Bruin et al., 2006; Fink et al., 2007; Stevenson et al., 2007; Williams et al., 2003).

Question 1: Are Finger-Length Ratios Masculinized in ADHD Versus Control Children?

We conducted a MANCOVA to examine whether children with ADHD had more masculine digit ratios (i.e., right 2D:4D, 2D:5D, and 3D:4D) than children without ADHD. Sex and ethnicity were covaried in this analysis, although groups differed only slightly by sex. Consistent with previous literature (de Bruin et al., 2006; Fink et al., 2007; McFadden et al., 2005; Stevenson et al., 2007; Williams et al., 2003), we found that children with ADHD had significantly more masculine finger-length ratios than control children when controlling for ethnicity, $F(3, 244) = 2.73, p < .05, \eta^2 = 0.03$. Boys with ADHD had more masculine digit ratios than control boys, $F(3, 144) = 2.78, p < .05, \eta^2 = 0.06$, whereas the effect in girls was trivial in size ($\eta^2 = 0.02$) and nonsignificant. This last finding was consistent with studies that examined these associations in clinically diagnosed samples (de Bruin et al., 2006; McFadden et al., 2005), but it contradicts other work utilizing rating scale data in nondiagnosed samples (Fink et al., 2007; Stevenson et al., 2007; Williams et al., 2003).

Question 2: Are There Differences in Right 2D:4D Finger-Length Ratios Between ADHD Subtypes?

We conducted a univariate ANCOVA, with sex and ethnicity covaried, looking at only the right 2D:4D in children with ADHD. Children with the combined subtype exhibited more masculine (i.e., lower) digit ratios than children with the inattentive subtype, but this difference was not significant, $F(1, 102) = 0.80, p > .05; \eta^2 = 0.01$. This finding contradicts McFadden et al. (2005)'s findings of hypermasculinization of the inattentive subtype.

Question 3: Are More Masculine Finger-Length Ratios Related to Particular ADHD Symptom Domains Within the Total Group?

We conducted bivariate correlations, shown in Table 3, to examine whether more masculine (i.e., lower) finger-length ratios were related to more inattentive or hyperactive-impulsive ADHD symptoms in the total group of children (i.e., boys and girls examined together). We examined correlations in the full group, rather than only the ADHD or the control group, to maximize the range of scores available and to assure oversampling for more extreme scores, recognizing that group differences were also influencing these correlations. As noted earlier, we emphasize the teacher + parent composite of ADHD. However, for completeness, reporter effects are noted later because discrepancies are often noted between parent and teacher reports of ADHD symptoms (Power et al., 1998; Verhulst, Koot, & Van der Ende, 1994). As shown in Table 3, more masculine ratios were related to both inattentive and hyperactive-impulsive ADHD symptoms, which is consistent with previous work (de Bruin et al., 2006; Fink et al., 2007; McFadden et al., 2005; Stevenson et al., 2007; Williams et al., 2003). When correlations were examined in the Caucasian subsample, the same pattern of significant findings emerged.

We conducted multivariate regression analyses to determine whether there was some specificity in relations between ADHD symptom domains and finger-length ratios. With inattentive symptoms as the criterion, hyperactive–impulsive symptoms and ethnicity (as a covariate) were entered at Step 1, and then digit ratio was entered at Step 2. The same procedure was used in reverse to statistically predict hyperactive–impulsive symptoms. To reduce Type I error, we examined only the right 2D:4D ratio on the basis of its emphasis in previous work. Right 2D:4D was associated with parent + teacher-rated inattention ($\beta = -0.10, p < .05$) but not hyperactivity–impulsivity ($\beta = -0.02, p > .05$) within the full sample (i.e., boys and girls analyzed together).

Question 4: Are More Masculine Finger-Length Ratios Related to Particular ADHD Symptom Domains Within Boys and Girls Examined Separately?

Bivariate correlations between digit ratios and ADHD symptoms were also conducted separately within each sex; they are shown in Table 4. Using a composite of teacher- and parent-rated symptom counts, a lower right 2D:4D ratio and right 3D:4D ratio were correlated with parent- and teacher-rated inattentive ($r = -.21$ and $-.20$, respectively; $p < .05$) and hyperactive–impulsive symptoms ($r = -.17$ for both; $p < .05$) in boys. In girls, these relations were not significant ($r = -.14$ and $-.09$, respectively, for inattention; and $-.10$ and $-.06$, respectively, for hyperactivity–impulsivity; all $ps > .05$). Relations between right 2D:5D and ADHD symptoms were not significant for either boys or girls ($p > .05$). The same pattern of correlations was found in the Caucasian subsample. The interaction between sex and right 2D:4D was not significant in predicting either inattentive or hyperactive–impulsive symptoms. There were no significant relations between right 2D:4D and ADHD symptom domains (when they were partialled from one another) within each sex.

Examination of Possible Confounds and Post Hoc Comparisons

Reporter and handedness effects—It should be noted that effects varied when parent and teacher symptom counts were examined separately, as shown in Tables 3 and 4. We observed more significant relations between digit ratios and ADHD symptoms using parent ratings (vs. teacher ratings) of symptoms. In addition, handedness analyses differed substantially by reporter. One hundred forty-two children had data on handedness. Nine of 76 children with ADHD and four of 66 non-ADHD comparison children were nonright-handed (i.e., left-handed or ambidextrous). There was no significant difference between the ADHD and non-ADHD groups in the percentage of nonright handedness, $\chi^2(1) = 1.42, p > .05$. Despite the small number of nonright-handers, a Right 2D:4D $\times$ Handedness interaction emerged in predicting teacher-rated hyperactive–impulsive symptoms ($\beta = -2.68, p < .05$), such that right 2D:4D predicted hyperactive–impulsive symptoms in nonright-handers ($\beta = -0.80, p < .01$) but failed to do so in right handers ($\beta = -0.04, p > .05$). Furthermore, within nonright-handers, there was a marginally significant Right 2D:4D $\times$ Sex interaction in predicting teacher-rated hyperactive–impulsive ADHD symptoms ($\beta = 7.05, p = .05$), with left-handed boys showing a stronger relation between right 2D:4D and symptoms than girls did (for boys, $\beta = -0.87, p < .01$; for girls, $\beta = -0.84, p > .05$). However, significant interactions were not observed predicting parent-reported symptoms or the composite parent- and teacher-rated symptoms.

Age, IQ, circulating testosterone/estradiol, and comorbid symptoms—We conducted bivariate correlations and hierarchical multiple regressions to examine the possible confounds of age, IQ, current testosterone/estradiol levels, and oppositional–defiant and conduct disorder symptoms. Externalizing (oppositional and conduct) symptoms were considered a potential confound because they may also be associated with testosterone and they overlap with ADHD; thus, they may account for any apparent ADHD effect. Bivariate correlations were not significant between right 2D:4D and age ($r = .07, p > .05$) or IQ ($r = .14, p > .05$). Bivariate correlations also were not significant between current blood testosterone or

estradiol levels (available for only a sub-sample of children, $n = 90$) and parent- and teacher-rated ADHD symptoms ($r_s = -.16$ to $-.07$, $p > .05$).

We did note significant correlations between smaller 2D:4D and oppositional–defiant symptoms ($r = -.19$, $p < .01$) but not conduct symptoms ($r = .11$, $p > .05$). In hierarchical multiple regression analyses controlling for symptoms of oppositional–defiant disorder, right 2D:4D continued to significantly predict inattentive symptoms ($\beta = -.21$, $p < .01$) but not hyperactive–impulsive symptoms ($\beta = -.12$, $p = .08$). In hierarchical multiple regression analyses controlling for symptoms of conduct disorder, right 2D:4D continued to significantly predict inattentive symptoms ($\beta = -.25$, $p < .01$) and hyperactive–impulsive symptoms ($\beta = -.18$, $p < .05$). When all symptom counts (i.e., inattentive, hyperactive–impulsive, oppositional, and conduct) were analyzed in a regression simultaneously, only inattentive symptoms were significantly related to right 2D:4D ($\beta = -.25$, $p < .05$).

Number of older brothers or sisters—Recent research suggests that the number of older brothers may influence prenatal androgen exposure through a maternal immune response to male-specific antigens (Blanchard, 2001). For this reason, bivariate correlations were conducted between sexually dimorphic finger-length ratios, ADHD symptoms, and number of older brothers and sisters. For boys, number of older brothers and sisters was not significantly related to more masculine or feminine finger-length ratios. However, number of older sisters was marginally related to more teacher-rated hyperactive–impulsive symptoms ($r = .16$, $p < .10$). For girls, number of older brothers was marginally related to a more masculine (i.e., smaller) right 2D:4D finger-length ratio ($r = -.19$, $p < .10$). However, no significant associations were found between the number of older brothers/sisters and ADHD symptoms in girls.

Discussion

In the present study, we sought to replicate and extend previous work examining associations between finger-length ratios and ADHD (de Bruin et al., 2006; Fink et al., 2007; McFadden et al., 2005; Stevenson et al., 2007; Williams et al., 2003). We report four main findings. First, the right 2D:4D, 2D:5D, 2D:3D, 3D:4D, 3D:5D and the left 2D:5D and 4D:5D showed significant mean differences by sex and by ADHD diagnosis (with sex controlled). We followed up on three ratios to constrain Type I error. Second, boys with ADHD showed more masculinized digit ratios than control boys. Third, more masculine (i.e., lower) right 2D:4D and 3D:4D ratios were correlated with a composite of parent- and teacher-rated inattentive and hyperactive–impulsive symptoms in boys but not in girls, although the statistical interaction with sex of the child did not reach significance. Fourth, masculinized finger-length patterns appeared to be more specific to the domain of inattention than hyperactivity–impulsivity in boys and girls when the shared variance between the two symptom domains was eliminated. Controlling for, or covarying, ethnicity did not alter the results.

These findings are consistent with recent studies in humans indicating that ADHD symptoms may be related to more masculinized finger-length ratios (de Bruin et al., 2006; Fink et al., 2007; McFadden et al., 2005; Stevenson et al., 2007; Williams et al., 2003). However, the present findings contradict several studies that found these relations in girls but not boys (Fink et al., 2007; Stevenson et al., 2007; Williams et al., 2003). It should be noted that studies finding relations in girls utilized nonclinical samples that relied on rating scale variation in the normal population and examined younger, preschool-age children (Fink et al., 2007; Williams et al., 2003) or older, college-age adults (Stevenson et al., 2007). In contrast, the present study and McFadden and colleagues (2005) found effects in boys. Both McFadden and colleagues (2005) and the present study utilized a well-defined, clinically diagnosed sample recruited for extreme ADHD-specific symptomatology. For this reason, differences between findings in the

present study and the McFadden et al. (2005) study, which also used a clinically recruited sample of school-age children, are most informative.

Two notable differences were seen between the results of the present study and that of McFadden et al. (2005). The present study did not replicate McFadden et al.'s (2005) finding that children with the inattentive subtype of ADHD had significantly more masculine digit ratios as compared with children with the combined subtype. It should be noted that differences between study definitions of the inattentive subtype likely explain this difference in findings. McFadden et al. (2005) defined the inattentive subtype more strictly than specified in the *DSM-IV* and in the present study, requiring the inattentive subtype to have four or fewer hyperactive-impulsive symptoms, rather than less than six hyperactive-impulsive symptoms. In the present study, more masculinized digit ratios were related most to inattentive symptoms, after controlling for hyperactive-impulsive symptoms; thus, it was not surprising that subtype differences did not emerge between the inattentive and combined subtypes, as both subtypes are defined by six or more inattentive symptoms.

Another notable difference between the two studies was the predominantly strong effects seen for right 2D:4D in the present study, as compared with the strong effects seen for right 2D:5D, 3D:5D, and 4D:5D in McFadden et al. (2005) study. It should be noted that measurements of finger-length ratios were conducted differently in the two studies (photocopy images and ruler measurements in the present study vs. photocopy/scanner images and canvas measurements in the McFadden et al. study). This measurement difference might explain differences in the specific effects of finger-length ratio between the two studies.

In the present study, there was some specificity of relations between masculinized digit ratios and inattention. However, it is also possible that prenatal testosterone exposure serves as more of a generalized risk factor for externalizing disorders and not just for ADHD. The association between more masculine digit ratios and hyperactivity-impulsivity may be at least partly accounted for by oppositional-defiant disorder symptoms. Thus, there may be a generalized association between prenatal testosterone exposure and disruptive behaviors, including inattention, hyperactivity, and oppositional defiance. Alternatively, prenatal testosterone exposure may increase the risk for childhood inattention and hyperactivity, in turn increasing risk for the subsequent development of oppositional defiance. It is important to note that age and circulating testosterone levels did not appear to account for these results, supporting the argument that these effects may be a direct result of prenatal testosterone exposure.

Exploratory analyses investigated the association between handedness, number of older siblings, and digit ratios. Although the sample was small, relations between digit ratios and hyperactive-impulsive ADHD symptoms appeared to be even stronger in children, especially boys, who were not right-handed. Older siblings did not show significant relations with digit ratios for boys or girls in this sample, although having older brothers was marginally related to more masculine digit ratios in girls.

Our results make a significant contribution to existing literature in this area of investigation. This was the largest study to date to employ a clinically well-characterized sample, and our finding of effects in boys but not girls replicates and extends previous work using such samples. The associations between digit ratios and ADHD symptoms in boys, but not in girls, suggest that the organizational effects of gonadal hormones such as testosterone may contribute to the sex-biased prevalence rate of ADHD in childhood. It is also possible that there is a threshold effect of androgen on ADHD, such that in girls with low absolute levels of prenatal testosterone, normal variation in prenatal androgen more rarely reaches threshold to produce ADHD symptoms.

We conclude that the organizational effects of gonadal hormones such as testosterone may influence the development of ADHD, possibly by acting generally on dopaminergic neural circuitry underpinning inattentive and hyperactive–impulsive symptoms of ADHD. Why would prenatal hormone exposure affect ADHD? Lesion studies using laboratory subjects suggest that such effects may be modulated by mesocorticolimbic dopamine circuitry (Chudasama & Robbins, 2006; Salazar et al., 2004; Taylor, Latimer, & Winn, 2003). Thus, gonadal hormones may influence the development of ADHD (and perhaps other learning and behavior problems) by permanently organizing the central dopamine system operating in cognitive control and reward processing (Sagvolden, Johansen, Aase, & Russell, 2005). Deficits in this neuronal architecture may in turn be manifested by behavioral deficits including inattention and hyperactivity–impulsivity (Sonuga-Barke, 2005).

High levels of testosterone early in gestation (measured indirectly through lower digit ratios) may permanently organize the development of the ascending midbrain dopamine system in a way that makes boys more vulnerable to the development of ADHD. For example, high levels of prenatal testosterone may lead to increased cell death within the right hemisphere of the brain, increased neural lateralization, slower development of the brain, and/or differential modulation of neurotransmission (Etgen, 2002; Goodman, 1991; Lyon & Gadisseux, 1991; Morris, Jordan, & Breedlove, 2004). In boys, the central dopamine system may be preferentially sensitive to these hormonal effects because it is slower to develop prenatally, thereby providing a longer period of time during which hormone exposure can influence prenatal dopaminergic gene expression. Sex differences in dopamine neural circuitry, evident in the striatum, the nucleus accumbens, and the prefrontal cortex, may be due to prenatal interactions between steroid hormones and specific dopaminergic genes (e.g., DRD4 and DAT1), operating synergistically in the brain to control attention, movement, and cognition (Becker & Rudick, 1999; see also Posner, 2004).

Although these findings are provocative, limitations should be noted. The interaction between child sex and digit ratio did not reach significance in predicting ADHD symptoms. Thus, the present study's finding of male-specific effects needs replication. Second, photocopies of finger length may have introduced some distortion into the absolute value of digit ratios in this study (Manning et al., 2005), although this distortion could not readily explain the group differences we found. Third, our findings for nonright-handers were limited to a very small sample and must be considered preliminary. Additional studies seem warranted given that several other groups have already reported similar associations between digit ratios and ADHD (de Bruin et al., 2006; Fink et al., 2007; McFadden et al., 2005; Stevenson et al., 2007; Williams et al., 2003). Furthermore, none of the studies to date, including the present report, indicate less masculine 2D:4D ratios being associated with greater ADHD symptoms, so it seems very unlikely that Type I error could account for the repeated finding of this association.

To summarize our findings, sexually dimorphic digit ratios were significantly related to ADHD symptoms in boys but not in girls. Prenatal testosterone exposure may serve as a general risk factor for the development of externalizing disorders in childhood with specific effects on inattention. These results suggest that prenatal organizational effects of gonadal hormones such as testosterone may contribute to sex differences in prevalence rates of ADHD.

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Table 1

Descriptive Statistics for ADHD, Control, and Total Groups of Children

Descriptive statistic	ADHD (<i>n</i> = 113)	Control (<i>n</i> = 137)	Total (<i>n</i> = 250)
<i>n</i> (%)			
Males	72 (63.7)	77 (56.2)	149 (59.6)
Ethnic minority	24 (21.3)	41 (30.0)	65 (26.0)
Alaska Native	1 (0.9)	0 (0.0)	1 (0.4)
American Indian	1 (0.9)	0 (0.0)	1 (0.4)
Asian	2 (1.8)	2 (1.5)	4 (1.6)
Black	14 (12.4)	29 (21.2)	43 (17.2)
Latino	4 (3.5)	6 (4.4)	10 (4.0)
Middle Eastern	1 (0.9)	0 (0.0)	1 (0.4)
Pacific Islander	0 (0.0)	3 (2.2)	3 (1.2)
White	89 (78.8)	96 (70.1)	185 (74.0)
Other/mixed	1 (0.9)	1 (0.7)	2 (0.8)
<i>M</i> (<i>SD</i>)			
Child age in years	14.25 (2.32)	15.01 (1.89)	14.67 (2.12)**
Inattentive symptoms (P)	7.37 (1.7)	1.82 (2.4)	4.33 (3.48)**
Hyperactive–impulsive symptoms (P)	4.05 (3.15)	1.01 (1.62)	2.38 (2.86)**
Inattentive symptoms (T)	5.29 (2.5)	1.9 (2.23)	3.5 (2.9)**
Hyperactive–impulsive symptoms (T)	2.83 (2.69)	1.09 (1.84)	1.91 (2.43)**

Note. Significant differences between ADHD and control groups, measured by *t* tests and chi-square tests, are indicated under Total column. ADHD = attention deficit-hyperactivity disorder; P = parent rated; T = teacher rated.

* $p < .05$.

** $p < .01$.

Table 2

Sexual Dimorphism of Finger-Length Ratios, Measured as Female–Male Mean Differences

Finger-length ratio	<i>t</i>	<i>df</i>	$M_{\text{girl}} - M_{\text{boy}}$	Cohen's <i>d</i>
R 2D:3D	−2.48	248	0.85–0.84	0.25*
R 2D:4D	−3.96	248	0.98–0.95	0.50**
R 2D:5D	−3.34	248	1.56–1.48	0.47**
R 3D:4D	−3.62	248	1.14–1.12	0.50**
R 3D:5D	−2.55	248	1.81–1.75	0.16*
R 4D:5D	−1.56	248	1.59–1.56	0.21
L 2D:3D	0.60	248	0.84–0.85	−0.25
L 2D:4D	−1.50	248	0.96–0.95	0.14
L 2D:5D	−3.17	248	1.54–1.47	0.50**
L 3D:4D	−1.33	248	1.23–1.12	1.57
L 3D:5D	−1.71	248	1.97–1.75	1.29
L 4D:5D	−2.34	248	1.60–1.56	0.33*

Note. R = right hand. L = left hand. For boys, $n = 149$. For girls, $n = 101$. MANOVA F test (ethnicity covaried) with 12 $df = 2.68$, $p < .01$. Note that some mean differences are large, but not significant, because of increased variability in these ratios' measurements.

*
 $p < .05$.

**
 $p < .01$.

Table 3

Correlations Between Relevant Right-Digit Ratios and ADHD Symptoms in All Children

ADHD symptom	R 2D:4D	R 2D:5D	R 3D:4D
Parent-rated inattention	-.18**	-.10	-.17**
Parent-rated hyperactivity	-.22**	-.17**	-.22**
Teacher-rated inattention	-.16*	-.12	-.16*
Teacher-rated hyperactivity	-.10	-.06	-.07
Parent- + teacher-rated inattention	-.21**	-.12	-.18**
Parent- + teacher-rated hyperactivity	-.16*	-.12	-.15*

Note. Correlations conducted in the Caucasian subsample alone do not differ from the primary effects observed in the total group analyzed together, with the exception that the correlation between teacher-rated inattention and right 3D:4D drops to nonsignificant.

*
 $p < .05$.

**
 $p < .01$.

Table 4
Correlations Between Relevant Right Digit Ratios and ADHD Symptoms in Boys and Girls

ADHD symptom	Boys			Girls		
	R 2D:4D	R 2D:5D	R 3D:4D	R 2D:4D	R 2D:5D	R 3D:4D
Parent-rated inattention	-.19*	-.07	-.19*	-.14	-.13	-.11
Parent-rated hyperactivity	-.26**	-.16	-.26**	-.10	-.15	-.08
Teacher-rated inattention	-.11	-.05	-.15	-.12	-.12	-.04
Teacher-rated hyperactivity	-.03	.05	-.03	-.16	-.19	-.08
Parent + teacher-rated inattention	-.21*	-.06	-.20*	-.14	-.13	-.09
Parent + teacher-rated hyperactivity	-.17*	-.08	-.17*	-.10	-.15	-.06

Note. Correlations conducted in the Caucasian subsample alone do not differ from the primary effects observed in the total group analyzed together. ADHD = attention deficit-hyperactivity disorder.

* $p < .05$.

** $p < .01$.