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Personality and Congenital Adrenal Hyperplasia: Possible effects of prenatal androgen exposure

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Abstract

Influences of early androgen exposure on personality were investigated. Participants were either exposed to abnormal levels of androgens prenatally due to congenital adrenal hyperplasia (CAH, 40 females, 29 males), or were unaffected relative controls (29 females, 30 males). Compared to female controls, females with CAH were less tender-minded ($p < .001$; 16 Personality Factor Inventory (16PF)), and reported greater physical aggression ($p = .03$; Reinisch Aggression Inventory) and less interest in infants ($p < .001$; Melson's Questionnaire), but did not differ in dominance (16PF). Males with CAH did not differ from male controls in interest in infants but were less dominant ($p = .008$), and more tender-minded ($p = .033$) and reported reduced physical aggression ($p = .025$). Thus, both males and females with CAH showed alteration in three of the four constructs assessed. Prenatal androgen exposure may shift some, but not all, personality characteristics in the male-typical direction in females. It may also be associated with a decrease in some aspects of male-typical personality development in males, although personality differences in males with CAH could relate to illness.

Keywords

androgen; dominance; aggression; prenatal; CAH; tender-minded; empathy; testosterone; gender; sex difference

Certain personality traits show sex differences (Maccoby and Jacklin, 1974; Feingold 1994; Costa, Terracciano, and McCrae, 2001). According to a meta-analysis based on data

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collected using a broad range of personality inventories (Feingold, 1994), the biggest sex differences are seen for dominance (a construct including measures of dominance, assertiveness, and ascendancy), with men scoring higher than women, and tender-mindedness (a construct including measures of tender-mindedness, nurturance, and empathy), with women scoring higher than men. In terms of the effect size index, d (the difference in means divided by the combined standard deviation; Cohen, 1988), dominance shows a moderate sex difference ($d = 0.50$), and tender-mindedness shows a large sex difference ($d = 0.97$). Regarding other personality traits, Feingold's (1994) meta-analysis suggested that anxiety ($d = .28$) and trust ($d = .25$) are somewhat more pronounced in females than in males, but that negligible or no sex differences ($d < .20$) are apparent in impulsiveness ($d = .06$), activity ($d = .09$), openness to ideas ($d = .03$), gregariousness ($d = .15$) and order ($d = .13$).

The nine traits investigated by Feingold (1994) are among 30 traits, each of which can be conceptualized as contributing to one of five broad factors (Neuroticism (N), Extraversion (E), Openness to Experience (O), Agreeableness (A) and Conscientiousness (C)) that comprise personality according to the Five-Factor Model (Costa and McCrae, 1992). A cross-cultural review of findings using a single personality inventory, the Revised NEO (Costa et al., 2001), investigated the five factors as well as their components, and produced results broadly in agreement with Feingold's. However, it reported smaller sex differences for dominance ($d = 0.19$ for U.S. adults, d ranges from 0.10 to 0.27 for all cultures) and tender-mindedness ($d = .31$ for U.S. adults, d ranges from 0.26 to 0.31 for all cultures), and a larger sex difference for anxiety ($d = .40$ for U.S. adults, d ranges from 0.32 to 0.43 for all cultures). In addition, there were small sex differences cross-culturally in several other personality facets (d s typically about 0.25), with women scoring higher than men on depression, self-consciousness, impulsiveness, vulnerability, warmth, gregariousness, positive emotions, openness to aesthetics, openness to feelings, straightforwardness, altruism, compliance, and modesty, and men scoring higher than women on excitement seeking, and openness to ideas. With respect to the five factors, women scored higher than men on N, E and A (d ranges from 0.28 to 0.59 in all cultures). All other factors and traits showed negligible sex differences ($d < 0.20$).

Differences in the sizes of sex differences seen in the two meta-analyses probably resulted because Feingold (1994) used a range of different measures of personality, whereas Costa et al. (2001) used a single inventory. Effect sizes for sex differences can vary substantially for different measures of the same construct. For example, smaller sex differences are found for dominance assessed by the California Personality Inventory (d ranges from 0.06 to 0.18) than by the Sixteen Personality Factor Questionnaire (d ranges from 0.49 to 1.09). Similarly, the sex difference in nurturance assessed by the Personality Research Form is smaller than the sex difference in tender-mindedness assessed by the Sixteen Personality Factor Questionnaire (d ranges from 0.56 to 1.22 and 0.97 to 1.37, respectively) (Feingold, 1994).

Causes of Sex Differences in Personality

Sex differences in personality have been suggested to result from both inborn factors and socialization (Costa et al., 2001). For instance, the differing adaptive problems faced by the two sexes during evolution might be expected to produce inborn sex differences in personality traits (Buss, 1995). In contrast, the social role model (Eagly, 1987) posits that gender differences in behavior and personality arise from sex differences in social roles.

One of the most powerful proximal determinants of sex differences in non-human mammals is the early hormone environment (De Vries and Simerly, 2002; Goy and McEwen, 1980; Hines, 2004). Experimental studies in species ranging from rodents to non-human primates support the view that prenatal or neonatal levels of gonadal hormones (especially the

androgen, testosterone) permanently influence behaviors that show sex differences. Gonadal hormones also appear to influence the development of at least some human behaviors (Collaer and Hines, 1995; Hines, 2004). This conclusion is based largely on studies of individuals who experienced anomalies in hormones during fetal life (e.g., as a result of genetic conditions or treatments to the mother). One such condition is classical congenital adrenal hyperplasia (CAH), an autosomal recessive disorder occurring in about 1 in 14,000 live births in North America and Western Europe (Pang, Wallace, Hofman, Thuline, Dorche, Lyon, Dobbins, Kling, Fujieda, and Suwa, 1988). Classical CAH involves a deficiency of the 21-hydroxylase (21-OH) enzyme (New, 1998). This deficiency leads to impaired cortisol synthesis. Increased compensatory uptake of substrate leads to the over production of androgens, including testosterone, because the pathway to cortisol secretion is blocked. There are two forms of classical CAH, a more severe, salt-losing form, and a less severe, simple virilizing form (Miller & Styne, 1999; New & Levine, 1984).

In females with CAH, the elevated prenatal androgens (Pang et al., 1980; Wudy, Dörr, Solleder, Djalali and Homoki, 1999) cause varying degrees of genital masculinization evident at birth, ranging from mild clitoral hypertrophy and labial fusion to a male-typical phallus and complete fusion of the labioscrotal folds. The genitalia of males with CAH appear normal at birth, and male fetuses with CAH appear to have normal levels of testosterone, at least at those points during gestation when testosterone has been measured (Pang et al., 1980; Wudy et al., 1999). Girls with CAH are typically detected soon after birth, owing to their genital virilization, and treated with corticosteroids to regulate the postnatal hormone environment. In the absence of neonatal screening, boys with CAH are typically diagnosed when they suffer salt-wasting crises, and are subsequently also treated with corticosteroids.

There is evidence that girls with CAH show behavioral, as well as physical, virilization. Numerous studies have documented increased male-typical toy, playmate, and activity preferences, in comparison to both unaffected female relatives and matched controls (Ehrhardt, Epstein and Money, 1968; Ehrhardt and Baker, 1974; Slijper, 1984; Dittmann et al., 1990; Berenbaum and Hines, 1992; Zucker et al., 1996; Nordenstrom, Servin, Bohlin, Larsson and Wedell, 2002, Pasterski et al., 2005). This behavioral masculinization occurs despite surgical feminization in infancy, hormone treatment to normalize cortisol and androgen levels postnatally and female sex of rearing. Boys with CAH have not been studied as extensively as girls, but generally show no alteration in toy or playmate preferences (Ehrhardt and Baker, 1974; Berenbaum and Hines, 1992; Hines and Kaufman, 1994). One study, however, found reduced rough-and-tumble play (a male-typical behavior) in boys with CAH (Hines and Kaufman, 1994).

Research on personality characteristics in individuals with CAH is less extensive than research on play behavior. The best studied aspect of personality is physical aggression, and although early studies produced inconsistent results (Ehrhardt & Baker, 1974; Money & Schwartz, 1976; Berenbaum and Resnick, 1997), a recent report, on a larger sample than used in prior studies, found increased tendencies to fighting and physical aggression in 3 to 11 year old girls with CAH (Pasterski et al., 2007). Also, a study using a standardized personality inventory, the Karolinska Scales of Personality, found that females with CAH showed increased (i.e., more male-typical) Detachment and Indirect Aggression scores, but did not differ from matched controls on six other traits that showed sex differences (Helleday, Edman, Ritzén and Siwers, 1993). Another focus has been interest in infants and motherhood. Females with CAH show reduced interest in female-typical toys, including dolls (Ehrhardt, Epstein and Money, 1968; Ehrhardt and Baker, 1974; Berenbaum and Hines, 1992; Pasterski et al., 2005), and interview data suggest that girls with CAH are less interested in infants and in motherhood than are other girls (Ehrhardt and Baker, 1974;

Dittmann et al., 1990). Using a questionnaire measure (Melson, 1987), Leveroni and Berenbaum (1998) found that females with CAH showed less interest in infants compared to unaffected female relatives. However, this difference was not significant at the conventional level ($p < .05$), when an item about interest in doll play was omitted from the scale. In addition, the girls with CAH also showed increased interest in pets, a characteristic that, unlike interest in infants, does not vary with sex.

Possible causes of inconsistencies in prior studies include the use of small samples (often fewer than 20, or even 10, participants per group), the use of measures that do not show substantial sex differences, and the use of inappropriate control groups, or no control group at all. Also, few studies have included males with CAH or assessed personality traits using standardized inventories, such as those that have been found to show sex differences in meta-analyses. In this study, we examined dominance and tender-mindedness, personality facets that have been found to show sex differences on a wide range of personality inventories, as well as propensities to physical aggression and interest in infants, in a relatively large sample of males and females with CAH. To further increase the power of the study, we used measures that previous investigators have found to reveal particularly large sex differences. In addition, unlike most prior studies, we assessed several personality facets in one sample, to determine if hormonal influences are uniform across a range of personality outcomes.

We hypothesized that, if sex differences in personality relate to the different levels of androgens experienced by male and female fetuses during development, females with CAH (who are exposed to increased androgens prenatally) would show increases in male-typical personality characteristics compared to unaffected female relatives. No specific predictions were made for males with CAH for three reasons: 1. it is not clear that testosterone levels are elevated prenatally in males with CAH (Pang et al., 1980; Wudy et al. 1999); 2. results for other behaviors in males with CAH have been inconsistent, with some reports suggesting a reduction in male-typical behavior but most suggesting no changes (Hines, 2004); and 3. studies of male animals treated with androgens during early life have produced similarly inconsistent results (Baum and Schretlen, 1975; Diamond, Llacuna and Wong, 1973; Dohler et al., 1984).

Method

Participants

The 128 participants in this study belonged to one of four groups: (1) Females with CAH ($n = 40$); (2) males with CAH ($n = 29$); (3) unaffected female relative controls ($n = 29$); and (4) unaffected male relative controls ($n = 30$). Individuals with CAH were recruited through consultant endocrinologists at Middlesex and Great Ormond Street Hospitals in London ($n = 35$; 22 female, 13 male) and through a national CAH Support Group in the UK ($n = 34$; 18 female, 16 male). Unaffected relatives were recruited through the families of participants with CAH. This unaffected relative control group was composed of unaffected siblings ($n = 57$) and first cousins ($n = 2$).

All the CAH families were British nationals and came from a broad range of educational and socio-economic backgrounds (described more fully in Hines et al., 2003). Participants were between 12 and 45 years of age. The ranges in age for the four groups were: (1) females with CAH—12 to 44 years ($M = 19.5$, $SD = 7.3$); (2) males with CAH—12 to 40 years ($M = 20.27$, $SD = 8.43$); (3) unaffected female relative controls—12 to 32 years ($M = 19.26$, $SD = 5.95$) and (4) unaffected male relative controls—12 to 45 years ($M = 18.00$, $SD = 6.81$). No group differed significantly from any other in age.

All 69 participants with CAH had 21-OH deficiency. Sixty-five had the more severe salt losing form of the disorder and four had the less severe simple virilizing form. Written consent was obtained from all participants. Written parental consent also was obtained for those participants under the age of 18 years. Participants received £50.00 (about \$80.00) and travel expenses for their participation.

Design and Procedure

A six-hour battery of cognitive, motor, physical and personality tests was administered. In this paper we report data on aspects of personality. Prior publications have reported data on targeting and mental rotations ability (Hines et al., 2003), language lateralization and hand preferences (Mathews et al., 2004); finger length ratios (Brown et al., 2002), sexuality (Hines, Brook and Conway, 2003), and characteristics related to autistic spectrum conditions (Knickmeyer et al., 2006).

Participants completed the items assessing dominance (Factor E) and tender-mindedness (Factor I) of the 16PF (Cattell and Eber, 1962) at home, and brought the completed questionnaires to the laboratory when they came in for testing. The Reinisch Aggression Inventory (RAI) (Reinisch, 1981) and Melson's Questionnaire of Interest in Infants and Interest in Pets (from Leveroni and Berenbaum, 1998) were completed at home by the participants after they had been tested in the laboratory. The Melson inventory was also completed by a parent of the participant (describing the interests of the child) after the laboratory visit. The RAI and Melson's Questionnaire were returned by post.

Factors E and I from the 16PF

Factor E (Dominance) is described as assessing whether a person is humble (mild, accommodating, easily led, conforming) versus assertive (independent-minded, aggressive, authoritative, competitive, stubborn) and Factor I (Tender-mindedness) as assessing whether a person is tough-minded (self-reliant, realistic, no-nonsense) versus tender-minded (intuitive, unrealistic, sensitive) (Cattell and Eber, 1962). The 1962 version of the 16PF was used because it showed large sex differences in prior studies. Internal consistency co-efficients (standardized alphas) for the subscales in the current study were 0.61 for the 13 items assessing dominance and 0.41 for the 10 items assessing tender-mindedness. Because of low internal consistency for the tender-mindedness subscale, item-total correlations were examined and three items were deleted on this basis, producing a seven item sub-scale with acceptable internal consistency (standardized alpha = 0.61).

The RAI—The RAI for adolescents and adults is a paper and pencil test assessing responses to six descriptions of hypothetical situations involving interpersonal conflict. For each situation, participants are presented with six pairs of forced-choice alternatives (all possible pair-wise combinations of four responses—physical aggression, verbal aggression, withdrawal and non-aggressive coping) and instructed to indicate how they would have responded as 12–13 year olds. Therefore, participants make six responses for each of the six situations from which scores for physical aggression, verbal aggression, withdrawal and non-aggressive coping are calculated. Internal consistency co-efficients (standardized alphas) for the four response categories were 0.86, 0.61, 0.71 and 0.82, in order. Previous work with the RAI has shown a large sex difference (d ranges from 0.7 to 1.1) for physical aggression, with boys and men more likely than girls and women to choose physically aggressive responses (Reinisch, 1981; Reinisch and Sanders, 1986). Completed RAIs were received from 32 females with CAH, 26 males with CAH, 26 female relative controls and 26 male relative controls.

Interest in Infants and Pets—Melson's Questionnaire

Melson's (1987) Questionnaire contains 16 items that assess interest in infants (11 items) and pets (five items). Participants (32 females with CAH, 23 males with CAH, 21 female relative controls and 23 male relative controls) and their parents (of 28 females with CAH, 22 males with CAH, 22 female relative controls and 23 male relative controls) indicated on a five-point scale ranging from 1 (never) to 5 (almost every day) how often the participant engaged in each of 16 activities when they were between two and seven years of age. The score for interest in infants reflected responses to the 11 items related to caring for, playing with, or being interested in infants, and the score for interest in pets reflected responses to the 5 items related to pets. Internal consistency co-efficients (standardized alpha) for the infant-related items were 0.83 and 0.90 for participants' and parents' completion, in order, and, for the pet-related items were 0.83 and 0.75 in the same order. We also calculated and analyzed alternative summary scores for interest in infants excluding items that depended on the target child having a younger sibling, because not all participants had a younger sibling, and excluding an item assessing interest in dolls, because a prior study found that differences between females with and without CAH were not present when the item related to dolls was removed (Leveroni and Berenbaum, 1998).

Two-way, between subjects ANOVAs were used to evaluate main effects of sex and group (CAH, Control) and their interaction on personality variables. In addition, planned comparisons examined *a priori* hypotheses regarding differences between (i) unaffected male and female relative controls to assess sex differences, (ii) females with and without CAH to determine if prenatal exposure to higher than normal levels of androgen influences personality, and (iii) males with and without CAH to clarify if CAH is associated with alterations in personality in males. Correlations also were computed to examine relationships among measures.

Results

16PF (See Tables 1 and 2)

The two-way ANOVA for Factor E (dominance) yielded no main effects of sex or group, but their interaction was significant (F values, degrees of freedom and p values for all two-way ANOVAs are provided in Table 2). Planned comparisons revealed that relative control males reported greater dominance than relative control females ($F_{(1, 57)} = 8.66, p = .005$), females with CAH did not differ from relative control females ($F_{(1, 67)} = 0.11, p = .740$), and males with CAH reported less dominance than relative control males ($F_{(1, 57)} = 7.69, p = .008$).

The two-way ANOVA for the ten item measure of Factor I (tender-mindedness) yielded a significant main effect of sex, no significant main effect of group, and a significant interaction. Planned comparisons revealed that relative control females reported more tender-mindedness than relative control males ($F_{(1, 57)} = 32.85, p < .001$) and than females with CAH ($F_{(1, 67)} = 22.21, p < .001$). The difference between males with and without CAH approached significance ($F_{(1, 57)} = 4.00, p = .050$). Analysis excluding the three items for which item total correlations were low (i.e., using the seven item subscale with improved internal consistency), produced the same pattern of results and statistical significance in the two-way ANOVA, and the same outcomes for group comparisons, with the exception that males with and without CAH now differed significantly; males with CAH showed significantly more tender-mindedness than unaffected males ($F_{(1, 57)} = 4.78, p = .033$).

RAI (see Tables 1 and 2)

The two-way ANOVA revealed a main effect of sex for physical aggression, no main effect of group, and a significant interaction. Planned comparisons showed that relative control males scored higher than relative control females ($F_{(1,50)} = 15.38, p < .001$), as did females with CAH ($F_{(1,56)} = 3.79, p = .057$, a difference that was in line with prediction and significant at the conventional level ($p < .05$) using a one-tailed statistic ($t = 1.95, df = 56, p = .0285$). Also, males with CAH scored significantly lower than control males ($F_{(1,50)} = 5.33, p = .025$).

Two-way ANOVA for withdrawal revealed a main effect of sex, no main effect of group and an interaction. Planned comparisons showed that relative control females scored higher than relative control males ($F_{(1,50)} = 9.48, p = .003$), and that the difference between females with and without CAH approached significance ($F_{(1,56)} = 4.01, p = .050$), but males with CAH did not differ from relative control males ($F_{(1,50)} = 1.84, p = .181$).

Two-way ANOVAs showed no main effects and no interactions for verbal aggression or non-aggressive coping. Planned comparisons also showed no differences in verbal aggression or non-aggressive coping between relative control males and females ($F_{(1,50)} = 0.33, p = .568$ and $F_{(1,50)} = 3.40, p = .071$, respectively), females with and without CAH ($F_{(1,56)} = 0.58, p = .450$ and $F_{(1,56)} = 0.76, p = .39$, respectively) or males with and without CAH ($F_{(1,50)} = 0.06, p = .804$ and $F_{(1,50)} = 2.728, p = .105$, respectively).

Melson's Questionnaire of Interest in Infants and Interest in Pets (See Tables 2 and 3)

This questionnaire was not completed by all participants or by all parents. However, scores derived from parents' and participants' responses correlated highly for interest in infants ($r = .70, n = 81, p < .001$), and for interest in pets ($r = .65, n = 89, p < .001$). Therefore, results are based on parents' or participants' responses, or when both were available, the average of parents' and participants' responses.

Two-way ANOVAs revealed significant main effects and an interaction of sex and group for interest in infants. Planned comparisons showed that relative control females scored higher than relative control males ($F_{(1,50)} = 34.47, p < .001$), and scored higher than females with CAH, ($F_{(1,59)} = 22.22, p < .001$), but that males with and without CAH did not differ in interest in infants ($F_{(1,50)} = 0.29, p = .591$). Data analyses using alternative composites created by deleting items that depended on having younger siblings, or that involved dolls, produced results that were statistically identical to those for the composite based on all the items (see Table 3).

There were no main effects and no interaction for interest in pets. Planned comparisons also revealed no differences between unaffected males and females ($F_{(1,50)} = 0.91, p = .345$), females with and without CAH ($F_{(1,59)} = 0.116, p = .734$), or males with and without CAH ($F_{(1,50)} = 0.52, p = .474$).

Correlations among measures—Because of group differences in outcome measures, correlations were calculated separately for each of the four groups of participants. Measures were largely independent of one another. Only three of the 24 correlations calculated were statistically significant. Among females with CAH, tender-mindedness correlated positively with interest in infants ($r = 0.38, df (34), p = .026$) and dominance correlated positively with physical aggression ($r = .41, p = .022$). Among males without CAH, tender-mindedness correlated negatively with physical aggression ($r = -0.39, df (25), p = .048$).

Discussion

Differences between unaffected males and females

All of the anticipated sex differences in personality were seen among unaffected males and females, replicating results obtained in prior studies. Males scored higher than females on dominance and on physical aggression and lower than females on tender-mindedness and interest in infants. Also as expected, there was no sex difference among unaffected controls for interest in pets, or for verbal aggression or non-aggressive coping. One unpredicted sex difference was observed, however. Unaffected males scored lower than unaffected females on the withdrawal scale of the RAI. This unpredicted difference may have occurred because the RAI is an ipsative measure, and higher scores on one scale, such as physical aggression, necessitate lower scores on others. In fact, although prior reports using the RAI have not typically investigated sex differences on subscales other than physical aggression, Reinisch and Sanders (1986) did so, and, like us, found lower withdrawal scores in males than in females.

The four predicted sex differences that we observed (in dominance, physical aggression, tender-mindedness and interest in infants) were similar in direction and magnitude to those observed by prior researchers using the same measures. For dominance and tender-mindedness on the 16 PF, Feingold (1994) reported sex differences ranging from .49 to 1.09 ($M = .83$, males greater than females) for dominance and .97 – 1.54 ($M = 1.33$, females greater than males) for tender-mindedness. We observed the same pattern of sex differences with mean differences of .77 for dominance and 1.49 for tender-mindedness (Table 1). For physical aggression (assessed using the RAI) and interest in infants (assessed using the Melson Inventory), prior researchers reported sex differences ranging from 0.7 to 1.9 for physical aggression ($M = 1.23$, males greater than females; Reinisch, 1981; Reinisch and Sanders, 1986) and 0.98 for interest in infants (females greater than males; Leveroni and Berenbaum, 1998), and we observed sex differences of 1.09 and 1.6 respectively (Tables 1 and 2). These large and reliable sex differences suggest that the measures we used were suitable for detecting hormonal influences on human psychological development, if such influences exist.

Differences between females with and without CAH

Three of the four personality measures differed in the predicted way for females exposed to higher than normal levels of androgen prenatally. Females with CAH were less tender-minded and less interested in infants, and gave more physically aggressive responses, than unaffected female relatives. In contrast, one characteristic hypothesized to relate to prenatal androgen exposure, dominance, did not differ for affected and unaffected females. Dominance also showed the smallest sex difference of the four measures ($d = 0.77$), raising the possibility that reduced experimental power could explain the absence of a relationship to the prenatal hormone environment. The difference in dominance scores for women with and without CAH was negligible ($d = 0.09$), however, arguing against this interpretation.

Outcomes for women with and without CAH for control measures that did not show sex differences also accorded with predictions. The two groups did not differ in interest in pets, or in verbal aggression or non-aggressive coping. Although women with CAH tended to score lower than women without CAH on the withdrawal scale of the RAI, this difference paralleled the sex difference on the same scale.

The current study not only provides additional empirical support for androgenic influences on the development of physical aggression and interest in infants, it also included broader constructs of traits than have been assessed in prior studies, measured using inventories that have been standardized on large samples of men and women in the general population. Our

results indicate that at least one such trait, tender-mindedness, relates to prenatal levels of androgen, suggesting that androgens during early development contribute to differential development of this personality facet in males and females. However, a second trait, dominance, did not differ in females with and without CAH, suggesting that the prenatal hormone environment does not contribute to the sex difference in this area. Thus, our results, and those of others, suggesting that prenatal hormones influence tender-mindedness, physical aggression and interest in infants, cannot be generalized automatically to other personality characteristics that show sex differences. Each of these needs to be investigated to see if hormones contribute to differential development in males and females.

The disorder, CAH, involves abnormalities in hormones in addition to testosterone and other androgens. Most notably, cortisol is deficient and adrenocorticotrophic hormone (ACTH) is elevated prior to treatment (Stewart, 2003). In addition, individuals with CAH are treated postnatally with glucocorticoids. However, there are no experimental data from other species suggesting that either cortisol or ACTH abnormality during early development influences sexual differentiation of behavior. It seems unlikely, therefore, that these other hormones, rather than androgens, are the agents responsible for the changes seen in sex-linked personality traits. Similarly, although postnatal corticosteroid treatment can produce non-physiological levels of hormones, there is no reason to predict effects on sex-related behavior or personality, reducing the likelihood that postnatal treatment is the responsible factor. The lack of group differences on control measures, such as verbal aggression and interest in pets, also argues against causation by factors not linked to processes of sexual differentiation.

Consequences of CAH also are not limited to hormone abnormality. As noted before, girls with CAH are born with virilized genitalia. Despite surgical feminization, female sex of rearing and treatment to normalize cortisol and androgens postnatally, it has been suggested that parents' awareness of the child's masculine appearance at birth could alter their treatment of the child and thus the child's gender related development (e.g., Fausto-Sterling, 2000; Quadagno, Briscoe and Quadagno, 1977). However, alterations in the play behavior of girls with CAH do not appear to result from parental encouragement of male-typical behavior or discouragement of female-typical behavior. Parents of girls with CAH do not differ from parents of unaffected girls in self-reported encouragement of their daughters "to act as a girl should" (Berenbaum and Hines, 1992). In addition, a study that observed parents interacting with their children as they played with sex-typical and sex-atypical toys found that parents gave more, rather than less, encouragement of female-typical behavior to daughters with CAH than to daughters without CAH (Pasterski et al., 2005).

Data linking normal variability in androgen prenatally to normal variability in sex-typical behavior postnatally, also argue for testosterone as the mechanism of behavioral change. Levels of maternal testosterone during pregnancy correlate with the amount of male-typical behavior in female offspring (Hines et al., 2002), and testosterone measured prenatally in amniotic fluid positively predicts male-typical behavior in both girls and in boys (Auyeung et al., in press) despite none of the offspring exhibiting genital abnormality.

Differences between males with and without CAH

Prior research on psychological outcomes in males with CAH has produced inconsistent results, as has research in which male rodents are treated with androgen during early development. Therefore, we did not make specific predictions about comparisons between males with and without CAH. Nevertheless, we found that males with CAH gave reduced male-typical responses in regard to three of the four characteristics measured. In comparison to male controls, they were less dominant, less physically aggressive, and more tender-minded. As with results for females with CAH, where dominance appeared to be unaffected,

the effect of CAH on personality in males was not evident for all four measures. Among males, interest in infants appeared unaffected. As was the case for the one unaffected characteristic in females (dominance), this did not seem to reflect insufficient experimental power, because the difference between males with and without CAH was negligible ($d = 0.14$). Also, as for females, effects of CAH in males were limited to personality facets that showed sex differences. Facets that did not differ for unaffected males and females (verbal aggression, non-aggressive coping and interest in pets) did not differ for males with and without CAH.

The pattern of differences observed in males suggests that CAH leads to a selective reduction in masculinization of some personality facets. Reduced masculinization has been reported previously for some other behaviors in males with CAH. As mentioned in the introduction, males with CAH have been found to show decreased rough-and-tumble play (Hines and Kaufman, 1994). They also show less male-typical (i.e., reduced) performance on measures of mental rotations abilities (Hampson et al., 1998; Hines et al., 2003). In contrast, other characteristics, including toy choices, playmate preferences and targeting abilities appear to be unaffected (Berenbaum and Hines, 1992; Ehrhardt and Baker, 1974; Hines and Kaufman, 1994; Hines et al., 2003). These inconsistent outcomes (sometimes reduced masculinization, sometimes no effect) resemble the inconsistent outcomes reported in males of other species following androgen administration during early development (Baum & Schretlen, 1975; Diamond et al, 1973; Dohler et al., 1984). The causes of the inconsistent outcomes are unclear, both in humans and other species.

There are at least three possible explanations for the effects of CAH on personality in males. The first possibility relates to neural feedback mechanisms that regulate androgen production in male mammals, and to the existence of separate sensitive periods for hormonal influences on different behaviors. The fetal testes produce androgens, including testosterone, and neural feedback mechanisms allow adjustment of this testicular androgen production in response to androgen elevation from other sources. For instance, a single injection of hormone during early development in male rats reduces subsequent testosterone production (Brown-Grant et al. 1975). Thus, in males with CAH, testosterone levels may be elevated initially, but then readjust to normal levels, or even lower than normal levels, because of feedback and reduced testicular production. Consistent with this possibility, levels of testosterone in midgestation amniotic fluid are elevated in female fetuses with CAH, who do not have testes and so cannot reduce testicular production, compared to healthy female fetuses, but are generally not elevated in male fetuses with CAH compared to healthy males, at least at midgestation when testosterone has been measured (Pang et al., 1980; Wudy, 1999). In addition, glucocorticoid treatment during early infancy can reduce testosterone levels in boys with CAH to levels below those of normal boys (Pang et al., 1979). In regard to critical periods, in rodents and non-human primates, different behaviors are susceptible to hormonal influences at different times during early development (Christensen and Gorski, 1978; Goy, Bercovitch and McBair, 1988). Thus, it is possible that demasculinization of certain characteristics in males with CAH reflects androgen deficits at some timepoints during development. Further research on the developmental profile of testosterone and other androgens in males with CAH would be helpful in evaluating this possibility.

A second possibility is that increased testosterone, beyond that seen in healthy male fetuses, reduces some aspects of male-typical neural and behavioral development. However, this explanation seems unlikely, because, as noted above, male fetuses with CAH do not show unusually high testosterone, at least at midgestation (Pang et al., 1980; Wudy, 1999).

A third possibility is that the observed personality differences between males with and without CAH reflect non-hormonal factors, particularly illness, associated with the disorder.

Most of the boys in our study had the more severe, salt losing, form of CAH. Boys with CAH, unlike girls, are not born with genital abnormalities and so are less likely to be diagnosed and treated from birth, thus increasing their risk of hospitalization for salt losing crises in infancy. Therefore, reduced male-typical characteristics in males with CAH could result from illness and its impact on personality. Specifically, illness during early life could lead to increased empathy and reduced dominance and physical aggression.

In support of illness effects on gender-related development, Green (1987) found that boys with extremely cross-gendered behavior were more likely than other boys to have been seriously ill in childhood. In addition, Hines and Kaufman (1994) found that boys with CAH had spent more time in the hospital during the first two years of life than had girls with CAH. This sex difference probably occurred because genital virilization in girls with CAH leads to rapid diagnosis and treatment, whereas boys with CAH appear normal at birth and are often not diagnosed until salt losing crises result in hospitalization. Hines and Kaufman (1994) also found that the number of days spent in the hospital related negatively to subsequent rough-and-tumble play in boys. These findings are consistent with the possibility that illness during infancy contributes to reduced male-typical behavior in boys with CAH. It would be of interest to explore whether similar relationships exist between illness in early life and subsequent empathy, dominance and physical aggression, both in boys with CAH and in children who suffer from other serious illnesses in childhood (e.g., insulin dependent diabetes). To our knowledge, the idea that early illness per se could have enduring influences on these particular personality characteristics is novel.

In summary, we found alterations in males and females with CAH in some personality facets that show sex differences. Among females, tendencies to physical aggression, tender-mindedness and interest in infants, were less female-typical in females with CAH than in unaffected females. These CAH-related alterations in personality may reflect the influence of androgen acting prenatally, as has been documented previously for the childhood play interests of girls with CAH. Dominance was the only sex-linked trait examined that was not masculinized in females with CAH, and this aspect of our results suggests that the sex difference in dominance is not due to the differing hormonal milieus experienced by males and females during prenatal development. Males with CAH were less male-typical than controls in dominance, tendencies to physical aggression and tender-mindedness, but showed no differences in interest in infants. The causes of decreased male-typical personality development in males with CAH are less clear than the causes of increased male-typical development in females. The reduced masculinization observed could result from deficits in testosterone at certain critical phases of development. Alternatively, the illness and hospitalization during early life that is common in boys with the salt-losing form of CAH could reduce masculinization of these aspects of personality. Regardless, our results suggest that CAH in both males and females is associated with alterations in sex-related aspects of personality development, most likely caused by hormonal action during critical periods of early life in females, and by either hormonal action, or illness-related experiences, in males.

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Dominance, tender-mindedness and aggression in female and male participants with CAH and unaffected female and male relative control participants

Table 1

	16PF ^a	Reinisch Aggression Inventory ^b				
		E	I	P	V	W
		M ± SD	M ± SD	M ± SD	M ± SD	M ± SD
(1) CAH female		13.05 ± 4.49	8.18 ± 2.80	6.56 ± 3.72	11.91 ± 1.96	8.72 ± 2.93
(2) Control female		12.72 ± 3.22	11.28 ± 2.55	4.63 ± 3.82	11.37 ± 3.34	10.35 ± 3.25
(3) CAH male		12.72 ± 3.65	8.83 ± 2.67	6.12 ± 4.79	11.65 ± 3.26	8.84 ± 3.56
(4) Control male		15.33 ± 3.58	7.47 ± 2.56	9.02 ± 4.24	11.87 ± 2.83	7.54 ± 3.33
<i>d</i> (2) & (4)		-0.77**	1.49***	-1.09***	0.13	0.85**
<i>d</i> (1) & (2)		0.09	-1.16***	0.51*	0.20	-0.53**
<i>d</i> (3) & (4)		-0.84**	0.52**	-0.72**	0.16	0.38
						0.46

Notes: E = Dominance, I = tender-mindedness, P = physical aggression, V = verbal aggression, W = withdrawal, N = non-aggressive coping, M = mean, SD = standard deviation, *d* = effect size (Cohen's *d*), *ns* for (1) – (4), in order are

^a = 40, 29, 29 and 30;

^b = 32, 26, 26 and 26.

* *p* ≤ .05, one tailed;

** *p* ≤ .05 and

*** *p* ≤ .001.

Table 2

F, degrees of freedom and *p* values for Two-Way ANOVAs

Factor E (dominance)		
Sex	$F_{(1,124)} = 2.79$	$p = .098$
Group (CAH, Control)	$F_{(1,124)} = 2.79$	$p = .098$
Sex X Group	$F_{(1,124)} = 4.61$	$p = .034$
Factor I (tender-mindedness)		
Sex	$F_{(1,124)} = 11.07$	$p = .001$
Group (CAH, Control)	$F_{(1,124)} = 3.36$	$p = .069$
Sex X Group	$F_{(1,124)} = 22.11$	$p < .001$
Physical Aggression		
Sex	$F_{(1,106)} = 6.20$	$p = .014$
Group (CAH, Control)	$F_{(1,106)} = 0.37$	$p = .544$
Sex X Group	$F_{(1,106)} = 9.29$	$p = .003$
Withdrawal		
Sex	$F_{(1,106)} = 4.66$	$p = .033$
Group (CAH, Control)	$F_{(1,106)} = 0.07$	$p = .791$
Sex X Group	$F_{(1,106)} = 5.50$	$p = .021$
Verbal aggression		
Sex	$F_{(1,106)} = 0.05$	$p = .825$
Group (CAH, Control)	$F_{(1,106)} = 0.09$	$p = .768$
Sex X Group	$F_{(1,106)} = 0.47$	$p = .496$
Non-aggressive coping		
Sex	$F_{(1,106)} = 0.82$	$p = .367$
Group (CAH, Control)	$F_{(1,106)} = 0.313$	$p = .58$
Sex X Group	$F_{(1,106)} = 3.18$	$p = .077$
Interest in infants		
Sex	$F_{(1,109)} = 28.48$	$p < .001$
Group (CAH, Control)	$F_{(1,109)} = 14.72$	$p = .001$
Sex X Group	$F_{(1,109)} = 9.80$	$p = .002$

Interest in pets			
Sex	$F_{(1,109)} = 3.44$	$p = .066$	
Group (CAH, Control)	$F_{(1,109)} = 0.61$	$p = .435$	
Sex X Group	$F_{(1,109)} = 0.12$	$p = .729$	

Interest in infants and pets in female and male participants with CAH and unaffected female and male relative control participants

Table 3

	Interest in Infants			Interest in Pets		
	C 1 ^a	C 2 ^b	C 3 ^c	C 4 ^d	P 1 ^e	
	M ± SD	M ± SD	M ± SD	M ± SD	M ± SD	
(1) CAH female	23.7 ± 6.9	22.1 ± 6.6	22.6 ± 6.5	20.0 ± 5.9	17.7 ± 3.4	
(2) Control female	31.9 ± 6.4	30.8 ± 7.4	30.0 ± 6.1	27.0 ± 6.3	18.0 ± 4.3	
(3) CAH male	21.1 ± 5.3	19.6 ± 4.9	20.6 ± 5.3	18.3 ± 4.8	16.0 ± 4.2	
(4) Control male	21.9 ± 5.8	20.3 ± 5.7	21.6 ± 5.8	19.0 ± 5.5	16.9 ± 4.4	
<i>d</i> (2) & (4)	1.6***	1.6***	1.4***	1.4***	0.3	
<i>d</i> (1) & (2)	-1.3***	-1.2***	-1.2***	-1.1***	-0.08	
<i>d</i> (3) & (4)	-0.1	-0.1	-0.2	-0.1	-0.2	

Notes: C 1 = all 11 infant-related items, C 2 = C 1 without "Takes care of younger siblings," C 3 = C 1 without "Plays with baby dolls," C 4 = C 1 without "Takes care of younger siblings" and "Plays with baby dolls," P 1 all five pet-related items. *ns* for (1) – (4) in order are 35, 25, 26 and 26; M = mean. SD = standard deviation. *d* = effect size (Cohen's *d*).

p < .001.