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BRIEF REPORT

Brief Report: Autistic Traits in Mothers and Children Associated with Child's Gender Nonconformity

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Abstract We examined relationships between autistic traits in children, mothers, and fathers and gender nonconformity (GNC) in children using data from the Nurses' Health Study II and the Growing Up Today Study 1. Autistic traits of mothers, fathers and children were measured using the Social Responsiveness Scale (SRS). GNC in children was measured using questions from the Recalled Childhood Gender Identity/Gender Role Questionnaire. In multivariable analyses increase in child's SRS score was associated with increased odds (OR 1.35; $p = 0.03$) of being in a higher GNC category. Increase in maternal SRS score was also associated with increased odds (OR 1.46; $p = 0.005$) of the child being in a higher GNC category. Paternal SRS scores were not related to child's GNC category.

Keywords Autism spectrum disorder · Gender · Gender identity · Gender nonconformity · Transgender

Abbreviations

ASD	Autism spectrum disorder
GNC	Gender nonconformity
NHSII	Nurses' Health Study II
GUTS1	Growing Up Today Study 1
SRS	Social Responsiveness Scale
IQR	Interquartile range

Introduction

Gender nonconformity (GNC) refers to having a gender expression that is not conforming to one's sex, such as a girl preferring to play with toys generally considered masculine (Zucker and Wood 2011). This concept is related to but distinct from *gender identity*, which refers to the

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internal sense of one's gender as man/boy or woman/girl, and *gender dysphoria*, which denotes incongruence in one's sex and gender identity, causing significant distress (American Psychiatric Association 2013; Shechner 2010). Both GNC and gender dysphoria describe individuals with characteristics and internal feelings diverging from typical gender norms.

There have been case reports suggesting co-occurrence of gender dysphoria and ASD (Gallucci et al. 2005; Kraemer et al. 2005; Landén and Rasmussen 1997; Mukaddes 2002; Perera et al. 2003; Robinow 2009; Tateno et al. 2008). A Dutch study reported a 7.8 % prevalence of ASD in a gender dysphoria clinic (de Vries et al. 2010), much higher than expected based on the prevalence of ASD in the general population, estimated to be approximately 1 % (Baird et al. 2006). Similarly, children seen at a US hospital-based pediatric neuropsychology program with ASD were found to be 7.6 times more likely to express gender variance compared to non-referred children as reported by their parents on the Child Behavior Checklist. Gender variance was assessed on the Child Behavior Checklist via a single item question, "wishes to be of opposite sex" with options "not true," "somewhat or sometimes true," and "very true or often true" (Strang et al. 2014).

Neither gender development nor the etiology of ASD are well understood. However, prenatal hormonal exposures, specifically androgen exposure, may influence both gender development (Berenbaum and Beltz 2011; Hines 2011) and development of ASD (Baron-Cohen 2002; Baron-Cohen et al. 2011; Knickmeyer et al. 2006; Voracek 2010). Genetic causes of autism have been described, with about 25 % of children with autism carrying an autism-related genetic variation (Miles 2011). Genetics have also been implicated as a contributing factor in gender dysphoria in a twin study demonstrating increased concordance of gender dysphoria among monozygotic twins compared to dizygotic twins. However, the study did not investigate specific genes or hormonal exposures in its evaluation (Heylens et al. 2012). ASD traits and diversity of gender identity could also be related in a more complex polygenetic or epigenetic fashion. The co-occurrence may be better framed as an example of *neurodiversity*, a term used by some in the ASD literature to frame autism symptoms and other neurologic symptoms as personality differences existing in a distribution as opposed to pathology (Jaarsma and Welin 2012; Kapp et al. 2013). In addition, postnatal environmental factors, such as the social relationship between the parent and infant (Fausto-Sterling 2012, pp. 408–9) and cognitive learning about parental expectations and societal norms (Martin et al. 2002) may influence gender development. As suggested by de Vries et al. (2010), the social rigidity present in children with ASD may lead to inflexibility with regard to gender and contribute to co-occurrence of ASD and GNC.

To date, there have been no national cohort studies examining the co-occurrence of autistic traits in children and GNC, nor have there been studies examining autistic traits in parents and the gender development of their offspring. The current study aims to fill these empirical gaps by analyzing data from two related US national cohorts, the Nurses' Health Study II (NHSII) and the Growing Up Today Study 1 (GUTS1). We hypothesized that young adults with more autistic traits would have higher degrees of recalled GNC in childhood. We also postulated that mothers and fathers with higher autistic traits would have offspring with higher GNC.

Methods

NHSII is a prospective cohort initially consisting of 116,430 female nurses, the majority of whom are white, originally from 14 populous US states when the study began in 1989. Since baseline, the nurses have been followed biennially with mailed questionnaires. Details of the NHSII cohort have been reported in Solomon et al. (1997). GUTS1 is a prospective cohort study of children who are the offspring of female nurses participating in NHSII. Participants of NHSII who had children ages 9–14 years in 1996 were contacted and asked for permission to enroll their offspring. The initial cohort in 1996 consisted of 16,882 participants. Follow-up questionnaires were sent annually or biennially (Field et al. 1999).

In 2005, NHSII participants were asked if they had a child diagnosed with autism, Asperger's syndrome, or another autism spectrum disorder. In 2007, follow-up mailings were sent to mothers of the 756 ASD cases and 3,000 controls. Following exclusions, 2,144 participants were included in this nested case–control study; details have been described (Lyall et al. 2012).

As part of the nested case–control study, participants completed the Social Responsiveness Scale (SRS; Constantino and Gruber 2005, 2012). The SRS is a 65-item questionnaire that assesses social functioning, reciprocal social interaction, and restrictive or stereotypical behaviors associated with ASD. It has been validated against the gold standard Autism Diagnostic Interview-Revised (ADI-R) with excellent agreement and has been shown to be stable over time and unrelated to age and IQ (Constantino et al. 2003). The mothers of index children (cases and controls) were sent SRS questionnaires regarding the index child.

The SRS was also used to assess the social functioning of the index children's parents; forms rating the fathers' social functioning were completed by the mothers, and forms rating the mothers' social functioning were completed by the children's fathers or a close relative. In total, SRS for 2,128 index children, 1,247 mothers and 1,629

fathers were collected (Lyall et al. 2014). SRS questionnaires were scored according to the scale's instructions. The scale is designed to have a mean score of 50 and standard deviation of 10 for the general population, with higher scores representing more autistic traits (Constantino and Gruber 2005, 2012).

In the 2005 and 2007 GUTS1 questionnaires, four questions were included from the Recalled Childhood Gender Identity/Gender Role Questionnaire asking about recalled behaviors during childhood, up to age 11 years (Zucker et al. 2006). GUTS1 participants ranged in age from 19 to 27 years in 2007 (mean age = 22.7 years). The GNC-related questions asked about recalled GNC from childhood: media characters imitated or admired, roles taken in pretend play, favorite games and toys, and feelings of femininity or masculinity. Response options ranged from "always women or girls"/"very feminine" to "always boys or men"/"very masculine" on a five-point Likert-type scale. A GNC score was calculated by averaging scores from the four questions (Cronbach's $\alpha = 0.78$). In order to identify participants with moderate nonconformity and more extreme nonconformity, GNC scores were grouped into three categories: scores below the median, scores above the median but below the top decile, and scores above the top decile. This method of score grouping has been used in other GUTS1 publications because the relation between GNC and health outcomes appears to be non-linear, with strongest associations found in the top 10 % of nonconforming children (Roberts et al. 2012a, b, 2013).

To test our hypotheses, we took advantage of the unique overlap in NSHII and GUTS1 data. Respondents eligible for inclusion in the current analysis were as follows: Of the 2,144 children with SRS data from the nested NHSII study, 94 of these individuals are also GUTS1 participants who had data collected regarding their recalled childhood gender GNC. Of the 1,247 mothers with SRS data, 198 of them had children who are GUTS1 participants with data collected regarding their recalled childhood GNC. Of the 1,629 fathers with SRS data, 269 of them had children in GUTS1 with recalled childhood GNC data.

We analyzed child SRS score by their GNC category using cumulative logit models recommended for the analysis of ordinal response data (Lee 1992). To evaluate our first hypothesis, that people with higher autistic traits have higher GNC, we examined data from the 94 children with both SRS scores from the nested NHSII study and GNC scores from GUTS1. Child SRS scores were compared to GNC score category (below median, above median but below top decile, and top decile) by calculating a median SRS score for each of these three GNC score categories. We used a cumulative logit model to determine how a one interquartile range (IQR) increase in SRS score statistically predicted the odds of being in a higher GNC score category. This model was adjusted for the sex and age of the child. In addition to an analysis of all 94 children, separate analyses were performed for males (n = 47) and females (n = 47), adjusted for age of the child.

Table 1 Sex, birth year, gender non-conformity, and social responsiveness scale scores of index children by case status

	Cases (n = 19)	Controls (n = 75)
Sex		
Males, n	12	35
Females, n	7	40
Year of birth, median	1985	1985
Childhood gender nonconformity		
Below median, n (%)	6 (31.58)	44 (58.56)
Above median, below top decile, n (%)	9 (47.37)	22 (29.33)
Top decile, n (%)	4 (21.05)	9 (12.00)
Social responsiveness score[a], mean (SD)	103.16 (33.25)	13.05 (12.38)

Cases are children identified by their mothers as having ASD as part of the nested study of autistic traits performed within NHSII. Controls are children not identified by their mothers as having ASD who participated in the same nested study

[a] Social Responsiveness Scores increase as autistic traits increase

In order to evaluate our second hypothesis, that parents with higher autistic traits have children with more GNC, we examined data from the 198 mothers and 269 fathers with SRS scores who also had a child with a GNC score. We analyzed mothers' and fathers' SRS scores and their children's GNC score category by calculating median SRS scores of mothers and fathers by GNC score category of their child. We used cumulative logit models to determine how a one IQR increase in the mother's SRS score or the father's SRS score predicted odds of their child being in a higher GNC score category. These models were adjusted for the sex and age of the child. In addition, analyses were performed stratified by child sex.

We also examined the relationship between SRS scores and GNC categories with the Kruskal–Wallis test. The Kruskal–Wallis test assigns ranks to SRS scores and compares ranks across GNC score categories. It does not assume a parametric distribution but sacrifices statistical power (Chan and Walmsley 1997). Using a nonparametric Kruskal–Wallis test, child SRS scores were compared to the child's GNC across the three GNC score categories. In addition, maternal SRS scores were compared to their child's GNC across the three GNC score categories using the Kruskal–Wallis test, and similarly, paternal SRS scores

Table 2 Child, Mother, and Father Social Responsiveness Scale (SRS) Scores by child's gender nonconformity

	N	Gender nonconformity of the child			Odds of higher level of gender nonconformity associated with one interquartile range (IQR) greater SRS score[a]	*p* value
		Below median	Above median, below top decile	Top decile		
		Median SRS score (interquartile range)			Odds ratio (95 % confidence interval)	
Child's SRS score	94	10 (20)	16 (77)	15 (97)	1.35 (1.04, 1.76)	0.03
Males	47	9 (17)	23 (72)	15 (97)	1.37 (0.94, 1.99)	0.10
Females	47	13 (24)	11.5 (19)	12.5 (119)	1.30 (0.87, 1.94)	0.21
Mother's SRS score	198	13 (17)	19 (20)	18 (23)	1.46 (1.12, 1.90)	0.005
Father's SRS score	269	17 (32)	15 (23)	19.5 (35)	1.06 (0.82, 1.38)	0.66

[a] Models for Child's SRS Score, Mother's SRS Score, and Father's SRS Score adjusted for year of birth and sex of index child. Models stratified by sex adjusted for year of birth of index child

were compared to their child's GNC across the three GNC score categories using the Kruskal–Wallis test.

Results

Characteristics of the index children are shown in Table 1. Data are stratified by whether the child participated in the nested SRS study within NHSII as an index case, identified by their mother as having ASD, or as a control participant.

In analyses of child SRS scores in relation to GNC, as the SRS score increases by one IQR, the odds ratio (OR) for being in a higher GNC score category (from below median, to above median but below top decile, to top decile) is 1.35 (95 % CI = 1.04, 1.76), indicating that higher SRS scores are associated with a higher GNC score category ($p = 0.03$; Table 2). For males, as the SRS score increases by one IQR, the OR for being in a higher GNC score category is 1.37 (95 % CI = 0.94, 1.99; $p = 0.10$) and for females the OR is 1.30 (95 % CI = 0.87, 1.94; $p = 0.21$; Table 2).

In analyses of maternal SRS scores by child GNC, when a mother's SRS score is higher by one IQR, the OR for their child being in a higher GNC score category is 1.46 (95 % CI = 1.12, 1.90), indicating that mothers with higher SRS have children with higher GNC score category ($p = 0.005$; Table 2).

When a father's SRS score is higher by one IQR, the OR of their child being in a higher GNC score category is 1.06 (95 % CI = 0.82, 1.38), indicating no significant relationship between a father's SRS score and his child's GNC score category ($p = 0.66$; Table 2).

Kruskal–Wallis analysis demonstrates a significant association between the maternal SRS score and their child's GNC category ($p = 0.03$). Kruskal–Wallis analyses does not demonstrate significant associations between the child's SRS score and the child's GNC category ($p = 0.18$) nor between the paternal SRS score and their child's GNC category ($p = 0.40$).

Discussion

This is the first evaluation of a link between autistic traits in either children or their parents and childhood GNC in a national cohort. Our results suggest that higher autistic traits in children or their mothers are associated with higher degrees of GNC in the child. These results strengthen evidence of a relationship between ASD and gender development. Causality cannot be established using this research design, however these findings could be consistent with common hormonal (Berenbaum and Beltz 2011; Hines 2011; Knickmeyer et al. 2006; Voracek 2010) or genetic causes, and may also support the assertion that the social relationship between parent and child is important for gender development (Fausto-Sterling 2012). Specifically, findings may support maternal social responsiveness as a factor in child gender expression. Alternatively, the co-occurrence may reflect another overarching characteristic, termed *neurodiversity* in some ASD literature, instead of an overlap of two distinct characteristics (Jaarsma and Welin 2012; Kapp et al. 2013).

It is interesting that we did not find an association between paternal SRS score and child GNC. This may give more credence to the prenatal hormonal environment as a cause of co-occurring ASD and GNC, or suggest a higher level of influence of the mother–infant/child dyad compared to the father–infant/child dyad in gender development. This latter hypothesis is consistent with related data regarding other dimensions of child development. For example, a study of 112 two-parent families found mothers to be more involved than fathers in socialization of their children (Schoppe-Sullivan et al. 2013). However, a study of familiarity of autism traits suggested higher SRS scores among fathers of children with ASD, but not mothers

(De la Marche et al. 2012). Future research is warranted to understand the relation of maternal versus paternal autistic traits to the development of children's gender nonconforming expression.

It is important to note that both the cumulative logit analysis and the Kruskal–Wallis test assessing association between maternal SRS score and their child's GNC score category were statistically significant. However, when analyzing for association between child SRS score and the child's GNC score category, only the cumulative logit analysis and not the Kruskal–Wallis test was significant. This may be related to the more limited power of the nonparametric Kruskal–Wallis test and the smaller number of analyzed pairs of child SRS scores and child GNC scores ($n = 94$) compared to the larger number of pairs of maternal SRS scores and their child's GNC scores ($n = 198$). Alternatively, this may represent a stronger relationship between maternal autism traits and child GNC compared to a child's autism traits and their own GNC. Future research is warranted to examine these associations.

This study has several limitations. We found a significant association between child SRS score and GNC score category. However, our sample size was too small to capture significant associations when stratified by sex, although point estimates are similar in males, females, and the combined sample. Also, while the parent–infant dyad model of gender development was the basis for our initial hypotheses, we cannot make claims of causality using this study design, as we are unable to adjust for possible confounding factors including genetics and the hormonal milieu of pregnancy. Additionally, autistic traits and GNC are measured indirectly. Autistic traits in the children were measured by SRS reports as assessed by the mothers, whereas formal autism evaluations in a clinical setting would have provided a more rigorous assessment of autistic traits. The measurement of GNC was performed using a subjective questionnaire assessing recalled GNC in childhood and not an objective in-person gender conformity assessment. Variability in the acceptance of GNC across participants was not collected and therefore could not be controlled for in modeling. Finally, the NHSII and GUTS1 are not racially or ethnically diverse, and this limits the generalizability of our findings. Research with diverse samples assessing autistic traits of parents and children and gender nonconforming expression represents an important future endeavor.

The overlap of the NHSII and GUTS1 cohorts provided a unique opportunity to explore autistic traits in children and their parents and their associations to GNC in children. This study should serve as a basis for further investigation into the importance of the parent–infant/child dyad on gender development and expression. Providers should be sensitive to the diversity of gender expression in children with autistic traits and in children of mothers with autistic traits. This sensitivity could help identify children who would benefit from services and support for both ASD and gender dysphoria.

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