

Brief Report: Female-To-Male Transsexual People and Autistic Traits

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Abstract The ‘extreme male brain’ theory suggests females with Autism Spectrum Conditions are hyper-masculinized in certain aspects of behavior. We predicted that females with Gender Identity Disorder (who are masculinized) would have elevated Autism Spectrum Quotient (AQ) scores. AQ scores from five groups were compared: (1) $n = 61$ transmen (female-to-male transsexual people); (2) $n = 198$ transwomen (male-to-female transsexual people); (3) $n = 76$ typical males; (4) $n = 98$ typical females; and

(5) $n = 125$ individuals with Asperger Syndrome (AS). Transmen had a higher mean AQ than typical females, typical males and transwomen, but lower than individuals with AS. Transmen have more autistic traits and may have had difficulty socializing with female peers and thus found it easier to identify with male peer groups.

Keywords Autism Spectrum Conditions · Gender Identity Disorder · Autism Spectrum Quotient (AQ) · Co-occurrence

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Introduction

Autism Spectrum Conditions (ASC) are characterized by difficulties in social interaction and communication, alongside restricted interests and repetitive behavior (APA 1994). ASC occur in approximately 1% of the population (Baird et al. 2006), with more males than females receiving a diagnosis. The Extreme Male Brain (EMB) theory of autism (Baron-Cohen and Hammer 1997) states that individuals with an ASC display an extreme of the typical male pattern of cognition and behavior (Baron-Cohen 2002). This pattern has been found on tests of intuitive physics (Baron-Cohen et al. 2001a), systemizing (Baron-Cohen et al. 2003; Wheelwright et al. 2006), and attention to detail (Jolliffe and Baron-Cohen 1997). In each of these domains, typical males score higher than females, and people with ASC (of both sexes) score even higher than typical males. This same pattern has also been found on the Autism Spectrum Quotient (AQ) which quantifies autistic traits in individuals (Baron-Cohen et al. 2001b; Wheelwright et al. 2006).

Given that in all the studies of ASC cited above, females with ASC are hyper-masculinized in specific aspects of behaviour and cognition, it may well be that they identify

more readily with the other sex. A recent study of play by girls with ASC found they show masculinization in choosing toys that do not require pretend play (Knickmeyer et al. 2008), and women with ASC report higher rates of tomboyism in childhood (Ingudomnukul et al. 2007). Females with ASC may therefore feel that they don't belong in a typical female peer group and in a minority of cases it may even lead to develop Gender Identity Disorder (GID). An elevated number of autistic traits would confer a rigidity on their perceived gender identity, similar to that which is observed in individuals with persistent GID from childhood to adulthood (Di Ceglie 1998).

Individuals with GID have a strong and persistent pre-occupation to live as the other gender than that of their biological sex (APA 1994). The co-occurrence of GID and ASD has been reported in several case histories (Kraemer et al. 2005; Landen and Rasmussen 1997; Mukaddes 2002; Tateno et al. 2008), observations from sexual disorder clinics (Robinow 2009) and recently, in a larger sample of adolescents and children (de Vries et al. 2010). There is evidence of similarities between girls with ASC and girls with GID. Girls with GID show more male-typical play and pastimes more associated with the other sex (APA 1994; Zucker 2005) and so do girls with ASC (Knickmeyer et al. 2008). Also, female-to-male (FM) transsexuals (referred to as 'transmen') follow a handedness pattern more similar to genetic males (less exclusively right-handed) (Green and Young 2001; Orlebeke et al. 1992) and the same has been found among women with ASC (Soper et al. 1986).

Given the above similarities and prior reports in the literature (de Vries et al. 2010; Kraemer et al. 2005; Landen and Rasmussen 1997; Mukaddes 2002; Tateno et al. 2008) we use the AQ to test the specific prediction from the EMB theory that transmen will have more autistic traits than typical women, and that a higher proportion will score in the ASC range for autistic traits. We also include male-to-female transsexual people ('transwomen') as a comparison group. The selection of atypical groups in the population thus allows us to test predictions from the EMB theory of autism.

Methods

Participants

Transmen and Transwomen

Transmen (female-to-male transsexuals) and transwomen (male-to-female transsexuals) participants were recruited by one of the authors (RG) at the Charing Cross Gender Identity Clinic, and via our website at www.cambridgepsychology.com. The participants completed online personal data

questions including age, occupation, and whether they had received a diagnosis of an intersex, transsexual or other DSM-IV major axis condition. A total of 77 transmen and 352 transwomen completed the AQ. This ratio of transmen:transwomen mirrors that found in other transsexual studies (Michel et al. 2001).

In the transmen group, 3 volunteers were excluded because they reported a diagnosis of bipolar disorder or schizophrenia. Data were only analyzed for participants who reported they were living in the gender role in line with their perceived gender identity. This led to data from 11 volunteers being excluded, and a further 2 because they did not answer that question. Data from 61 transmen were fully analyzed (mean age 34.0; range 19–52.7). Of these 61, 10 reported having lived as their perceived gender for less than 12 months, 27 for between one and 4 years, and 24 for more than 4 years. 48 of the 61 participants reported they were taking testosterone. 12 reported that they sexually preferred men, 28 preferred women, 3 endorsed liking neither sex, and 18 endorsed both sexes. One participant reported a diagnosis of AS and analyses were conducted both with and without this individual.

In the transwomen group, 4 volunteers were excluded because they reported a diagnosis of bipolar disorder. As with the transmen group, data were only analyzed for participants who reported they were currently living as their perceived gender: 148 volunteers were excluded because they did not meet this criterion and a further 2 did not answer this question. The data from 198 transwomen were fully analyzed (mean age 45.1; range 16–75). Of these 198, 43 reported having lived as their perceived gender for less than 12 months, 94 for between one and 4 years, and 61 for more than 4 years. 170 of the 198 reported they were taking oestrogen. 59 reported that they sexually preferred women, 69 preferred men, 25 endorsed liking neither sex, and 45 endorsed both sexes. Six reported a diagnosis of autism or AS and, as with the transmen group, analyses were conducted both with and without these individuals.

Male and Female Controls

The AQ scores from the male and female general population were taken from the data reported elsewhere (Baron-Cohen et al. 2001b) (76 males; 98 females; mean age 37.0; range 18.1–60.0).

Participants with Asperger Syndrome (AS)

The AQ scores for the participants with AS were taken from the data collected elsewhere (Wheelwright et al. 2006). These participants were recruited via the National Autistic Society (UK), specialist clinics and advertisements in newsletters/web pages. All participants had received a

diagnosis from a psychiatrist according to DSM-IV criteria (APA 1994). Of the 125 participants, 110 had a diagnosis of Asperger Syndrome (AS) and 15 a diagnosis of High Functioning Autism (HFA). The data from 69 males and 56 females were analyzed (mean age 37.6; range 17.6–71.1).

Autism Spectrum Quotient

The AQ is a 50-item, self-report questionnaire, which assesses social skills, communication skills, imagination abilities, attention switching, and attention to details (Baron-Cohen et al. 2001b) and which predicts clinical diagnosis of Asperger Syndrome (Woodbury-Smith et al. 2005). There are 10 statements in each of the 5 categories and participants choose whether they ‘strongly agree’, ‘slightly agree’, ‘slightly disagree’, or ‘strongly disagree’ with each statement. 1 point is given for the ‘autistic’ type response and 0 for the non-autistic type response. The higher the score on the AQ, the more autistic traits the individual possesses, for complete details of the questionnaire see Baron-Cohen et al. (2001a, b). In the version used in the present study, the AQ was presented online and took about 5 min to complete.

Results

Mean AQ scores from each group are displayed graphically in Fig. 1. An ANOVA with between subject factors of Group (Transsexuals vs. Controls vs. ASC group) and Genetic Sex and the covariate of age revealed significant main effects of Group ($F(2, 558) = 303.0, p < 0.0001$) and Genetic Sex ($F(1, 558) = 7.5, p < 0.01$) and a significant interaction ($F(2, 558) = 16.9, p < 0.0001$), but no effect of age ($p > 0.6$). Inspection of Fig. 1 suggests that the interaction arises because control males ($x = 17.8, SD = 6.8$) score

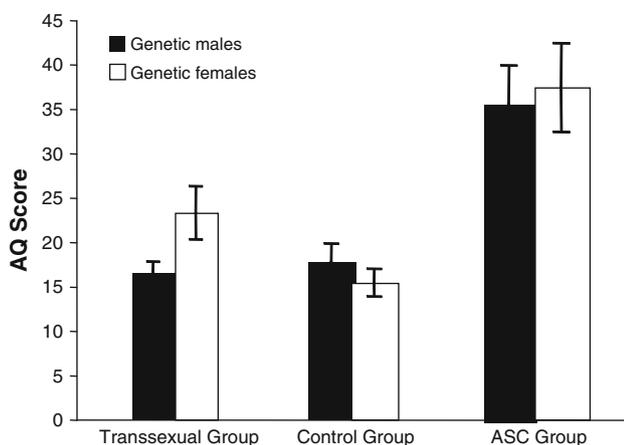


Fig. 1 Mean AQ scores in the three groups. Error bars denote standard error of the mean (SEM)

higher than control females ($x = 15.4, SD = 5.7$) ($t(172) = -2.5, p < 0.02$, cohen's $d = 3.8$), while in the transsexual group transmen ($x = 23.2, SD = 9.1$) score higher than transwomen ($x = 16.6, SD = 6.9$) ($t(257) = 5.3, p < 0.001$, cohen's $d = 0.83$). In addition, transmen score higher than control females ($t(157) = 6.1, p < 0.001$, cohen's $d = 1.0$) but there is no difference between transwomen and control males ($t(272) = 1.3, p > 0.2$). The ASC group scored ($x = 36.7, SD = 8.0$) significantly higher than all other participants (all p 's < 0.0001), and as in previous research, there was no sex difference in this group (ASC females: $x = 37.4, SD = 8.2$; ASC males: $x = 35.9, SD = 7.8$) ($t(123) = 1.0, p > 0.3$). When these analyses were repeated without the participants with ASC in the transsexual groups, the results remained the same.

The transsexual and control groups only were reanalyzed with an ANOVA, using the between subject factors of Group and Perceived Gender. This allowed control males and transmen, and control females and transwomen, to be compared directly. There were significant main effects of Group ($F(1, 433) = 20.5, p < 0.0001$), with transsexual people scoring higher than controls and Perceived Gender ($F(1, 433) = 38.2, p < 0.0001$), with males scoring higher than females. There was also a significant interaction ($F(1, 433) = 8.3, p < 0.001$). Post hoc t-tests revealed that the significant interaction arose because transmen scored higher than control males ($t(135) = 3.9, p < 0.001$, cohen's $d = 0.7$) but there was no difference between transwomen and control females ($t(294) = 1.6, p > 0.1$).

To investigate the effect of hormone treatment, those taking hormones were compared with those who did not report hormone treatment, in both transsexual groups separately. We found no significant difference between those on hormone treatment in either the transwomen ($p > 0.6$) or in the transmen group ($p = 0.07$), although there was a trend. To determine the effect of sexual orientation on AQ scores in the transsexual group, we compared those who reported to be homosexual (in relation to birth sex) versus not homosexual (this included those who endorsed liking both or neither sex) and found a significant interaction between the transwomen and transmen and sexual orientation ($F(1,259) = 4.8, p < 0.03$). Transwomen who reported not to be homosexual in relation to birth sex (i.e. endorsed liking women, both sexes or neither sex) had a greater AQ score ($x = 17.4, SD = 7.4$) than those who reported to be homosexual (i.e. preferred men) ($x = 15, SD = 5.6$) ($t(196) = 2.3, p < 0.03$, cohen's $d = 0.4$), whereas there was no difference among the transmen ($t(59) = 1.0, p > 0.3$).

Scores on the AQ can be used to categorise individuals as having the ‘broader autism phenotype’ (BAP: defined as AQ 23–28), ‘medium autism phenotype’ (MAP: defined as AQ 29–34) or ‘narrow autism phenotype’ (NAP: defined

Table 1 Percent of participants with each autism phenotype

	AQ score	Transsexuals		Controls	
		Transwomen	Transmen	Males	Females
Broader autism phenotype	23–28	13.6	21.3	15.8	10.2
Medium autism phenotype	29–34	3.5	14.8	5.3	2.0
Narrow autism phenotype	35+	1.5	14.8	1.3	0

as AQ 35+) (Wheelwright et al. 2010). These 3 AQ bands represent 1, 2, or 3 standard deviations above the population mean, respectively. The percent of participants from the transsexual and control groups with each of these phenotypes is shown in Table 1. The transmen had significantly more autistic traits than control men and their mean AQ score lies in the BAP range. Approximately 30% of the transmen group had an AQ in the MAP or NAP range. Transmen had a 11-fold increase in the rate of NAP relative to typical males.

Discussion

This study confirms clinical case studies and reports in adolescents and children that genetic females with Gender Identity Disorder (GID) have an increased number of autistic traits. Our results demonstrate that transmen (relative to control women) have more autistic traits, as measured by the Autism Spectrum Quotient (AQ). They also had significantly more autistic traits than control men, and their mean AQ score lies in the Broader Autism Phenotype (BAP) range. Approximately 30% of the transmen had an AQ in the Medium (MAP) or Narrow Autism Phenotype (NAP) range, which would warrant clinical investigation if this level of autistic traits was causing suffering or impacting on daily functioning (Woodbury-Smith, et al. 2005). Transmen had a 11-fold increase in the rate of NAP relative to typical males. We speculate that this increased number of autistic traits is likely to have made the transmen (in their childhood and adolescence) less able to assimilate in a female peer group, instead gravitating towards males. This may also have led to difficulties socializing in a female peer group, and a feeling of belonging more in a male group, thus increasing the probability of GID.

Assuming AQ is relatively stable throughout life, transmen can be assumed to have had an increased number of autistic traits in early development, possibly because of elevated foetal testosterone (FT) levels (Auyeung et al. 2009; 2010) and/or genetic factors (Hoekstra et al. 2007). Such an idea fits with research based on studies of amniotic testosterone levels that show a correlation between foetal testosterone (FT) and autistic traits (Auyeung et al. 2009; 2010). This is not to say that current testosterone levels are

average in adults with GID and ASC, since testosterone-linked medical conditions (such as polycystic ovary syndrome (PCOS) and delayed menarche) are reported to be elevated in women with ASC (Ingudomnukul, et al. 2007; Knickmeyer et al. 2006b) and there are some reports of increased PCOS in transmen (Baba et al. 2007) while others have not found this increase (Mueller et al. 2008). This may reflect a continuing elevation of androgen levels from foetal through to current circulation. The fact that a similar pattern of PCOS rates is also found in mothers of women with ASC suggests this may be occurring for genetic reasons (Ingudomnukul et al. 2007). A recent genetic study from our lab suggests that genes that control sex steroid hormones are associated with both ASC and autistic traits (Chakrabarti et al. 2009).

We found there was no difference between transwomen and control males on the AQ: transwomen have a mean AQ score that lies in the average range for both control males and females, and is not significantly different from either. Similarly, the proportion of transwomen with BAP and MAP also lies between control males and control females. Interestingly, within the 198 transwomen group, there were 6 individuals (i.e. 3%) with a diagnosis of AS. This rate is about 3 times as many as in the general population (Baird et al. 2006), although we have not calculated confidence intervals for this, it is consistent with previous studies (de Vries et al. 2010). Interestingly, among the transwomen sexual preference influenced AQ scores, consistent with findings that there are different typologies among transwomen based upon their sexual orientation (Lawrence 2010). Future research should explore this connection, as it appears that the association between GID and ASC in transwomen is complex.

It is important to consider certain limitations of the present study. First, it is based purely on self-report. It would be worthwhile repeating the study with an informant completing the AQ. Second, recruitment was conducted through multiple sources (the clinic and online) which may have resulted in a more heterogeneous participant population, which could be explored in future studies. Lastly, a proportion of the transsexual groups were taking hormone treatments and for obvious ethical reasons it was not possible to control for this factor but it is of interest that analysis comparing those on or off testosterone treatment did not lead to significantly different AQ scores. This raises

the idea that *current* sex steroid levels do not affect AQ, while *foetal* levels of sex steroids may affect AQ.

The relevance of these results for clinicians working with transmen is clear. These are chromosomally female individuals, who have felt masculinized since childhood, and rather than simply assuming the GID is the primary problem, the link between autistic traits and GID should be explored. Clinically, even if only for a minority of individuals considering sex reassignment surgery (SRS), the formulation of undiagnosed AS might be a helpful alternative to explore. Score on the AQ need not affect whether the individual continues on their chosen path for SRS but may help the person in examining the reasons behind their choices and consequently make better informed decisions about treatment and physical interventions.

In closing, this study provides evidence that transmen have an elevated number of autistic traits. This may be a reflection of elevated FT levels since both normative amniotic testosterone studies (Chapman et al. 2006; Knickmeyer et al. 2005) and studies of rare genetic conditions in which FT levels are abnormally high (such as in females with Congenital Adrenal Hyperplasia) (Knickmeyer et al. 2006a) indicate that higher FT is correlated with reduced empathy, reduced social interest, reduced social skills, and higher AQ scores. Quite how this is expressed in terms of neural masculinization (Baron-Cohen et al. 2005) is an important question for further study, which we are currently seeking to answer using MRI in children whose amniotic FT levels are known. The causation of GID and its development is complex and this paper is a contribution regarding a particular association between GID and ASC.

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References

- APA. (1994). *DSM-IV Diagnostic and statistical manual of mental disorders* (4th ed.). Washington DC: American Psychiatric Association.
- Auyeung, B., Baron-Cohen, S., Ashwin, E., Knickmeyer, R., Taylor, K., & Hackett, G. (2009). Fetal testosterone and autistic traits. *British Journal of Psychology*, *100*(1), 1–22.
- Auyeung, B., Taylor, K., Hackett, G., & Baron-Cohen, S. (2010). Foetal testosterone and autistic traits in 18 to 24-month-old children. *Mol Autism*, *1*(1), 11.
- Baba, T., Endo, T., Honnma, H., Kitajima, Y., Hayashi, T., Ikeda, H., et al. (2007). Association between polycystic ovary syndrome and female-to-male transsexuality. *Human Reproduction*, *22*(4), 1011–1016.
- Baird, G., Simonoff, E., Pickles, A., Chandler, S., Loucas, T., Meldrum, D., et al. (2006). Prevalence of disorders of the autism spectrum in a population cohort of children in South Thames: The special needs and autism project (SNAP). *Lancet*, *368*(9531), 210–215.
- Baron-Cohen, S. (2002). The extreme male brain theory of autism. *Trends in Cognitive Science*, *6*, 248–254.
- Baron-Cohen, S., & Hammer, J. (1997). Is autism an extreme form of the male brain? *Advances in Infancy Research*, *11*, 193–217.
- Baron-Cohen, S., Wheelwright, S., Scahill, V., Lawson, J., & Spong, A. (2001a). Are intuitive physics and intuitive psychology independent? *Journal of Developmental and Learning Disorders*, *5*, 47–78.
- Baron-Cohen, S., Wheelwright, S., Skinner, R., Martin, J., & Clubley, E. (2001b). The Autism Spectrum Quotient (AQ): Evidence from Asperger syndrome/high functioning autism, males and females, scientists and mathematicians. *Journal of Autism and Developmental Disorders*, *31*, 5–17.
- Baron-Cohen, S., Richler, J., Bisarya, D., Gurunathan, N., & Wheelwright, S. (2003). The Systemising Quotient (SQ): An investigation of adults with Asperger syndrome or high functioning autism and normal sex differences. *Philosophical Transactions of the Royal Society, Series B*, *358*, 361–374.
- Baron-Cohen, S., Knickmeyer, R. C., & Belmonte, M. K. (2005). Sex differences in the brain: Implications for explaining autism. *Science*, *310*(5749), 819–823.
- Chakrabarti, B., Dudbridge, F., Kent, L., Wheelwright, S., Hill-Cawthorne, G., Allison, C., et al. (2009). Genes related to sex steroids, neural growth, and social-emotional behavior are associated with autistic traits, empathy, and Asperger syndrome. *Autism Res*, *2*(3), 157–177.
- Chapman, E., Baron-Cohen, S., Auyeung, B., Knickmeyer, R., Taylor, K., & Hackett, G. (2006). Fetal testosterone and empathy: Evidence from the empathy quotient (EQ) and the “Reading the mind in the eyes” test. *Social Neuroscience*, *1*(2), 135–148.
- de Vries, A. L., Noens, I. L., Cohen-Kettenis, P. T., van Berckelaer-Onnes, I. A., & Doreleijers, T. A. (2010). Autism spectrum disorders in gender dysphoric children and adolescents. *Journal of Autism and Developmental Disorders*, *40*(8), 930–936.
- Di Ceglie, D. (1998). Reflections on the nature of the “atypical gender identity organization”. In D. Di Ceglie & D. Freedman (Eds.), *A stranger in my own body: Atypical gender identity development and mental health* (pp. 9–25). London: Karnac.
- Green, R., & Young, R. (2001). Hand preference, sexual preference and transsexualism. *Archives of Sexual Behavior*, *30*, 565–574.
- Hoekstra, R. A., Bartels, M., Hudziak, J. J., Van Beijsterveldt, T. C., & Boomsma, D. I. (2007). Genetic and environmental covariation between autistic traits and behavioral problems. *Twin Res Hum Genet*, *10*(6), 853–860.
- Ingudomnukul, E., Baron-Cohen, S., Wheelwright, S., & Knickmeyer, R. (2007). Elevated rates of testosterone-related disorders in women with autism spectrum conditions. *Hormones and Behavior*, *51*(5), 597–604.
- Jolliffe, T., & Baron-Cohen, S. (1997). Are people with autism or Asperger’s syndrome faster than normal on the embedded figures task? *Journal of Child Psychology and Psychiatry*, *38*, 527–534.
- Knickmeyer, R. C., Baron-Cohen, S., Raggatt, P., & Taylor, K. (2005). Foetal testosterone, social relationships, and restricted interests in children. *Journal of Child Psychology and Psychiatry*, *46*(2), 198–210.
- Knickmeyer, R. C., Baron-Cohen, S., Fane, B. A., Wheelwright, S., Mathews, G. A., Conway, G. S., et al. (2006a). Androgens and autistic traits: A study of individuals with congenital adrenal hyperplasia. *Hormones and Behavior*, *50*(1), 148–153.
- Knickmeyer, R. C., Wheelwright, S., Hoekstra, R., & Baron-Cohen, S. (2006b). Age of menarche in females with autism spectrum

- conditions. *Developmental Medicine and Child Neurology*, 48(12), 1007–1008.
- Knickmeyer, R. C., Wheelwright, S., & Baron-Cohen, S. B. (2008). Sex-typical play: Masculinization/defeminization in girls with an autism spectrum condition. *Journal of autism and developmental disorders*, 38(6), 1028–1035.
- Kraemer, B., Delsignore, A., Gundelfinger, R., Schnyder, U., & Hepp, U. (2005). Comorbidity of asperger syndrome and gender identity disorder. *European Child and Adolescent Psychiatry*, 14, 292–296.
- Landen, M., & Rasmussen, P. (1997). Gender identity disorder in a girl with autism—a case report. *European Child and Adolescent Psychiatry*, 6(3), 170–173.
- Lawrence, A. A. (2010). Sexual orientation versus age of onset as bases for typologies (subtypes) for gender identity disorder in adolescents and adults. *Archives of Sexual Behavior*, 39, 514–545.
- Michel, A., Mormont, C., & Legros, J. (2001). A psycho-endocrinological overview of transsexualism. *European Journal of Endocrinology*, 145, 365–376.
- Mueller, A., Gooren, L. J., Naton-Schötz, S., Cupisti, S., Beckmann, T. W., & Dittrich, R. (2008). Prevalence of polycystic ovary syndrome (PCOS) and hyperandrogenemia in female-to-male transsexuals. *The Journal of Clinical Endocrinology and Metabolism*, 93(4), 1408–1411.
- Mukaddes, N. M. (2002). Gender identity problems in autistic children. *Child: Care, Health and Development*, 28(6), 529–532.
- Orlebeke, J. F., Boomsma, D. I., Gooren, L. J., Verschoor, A. M., & Van Den Bree, M. J. M. (1992). Elevated sinistrality in transsexuals. *Neuropsychology*, 6(4), 351–355.
- Robinow, O. (2009). Paraphilia and transgenderism: A connection with asperger disorder? *Sexual and Relationship Therapy*, 24(2), 143–151.
- Soper, H., Satz, P., Orsini, D., Henry, R., Zvi, J., & Schulman, M. (1986). Handedness patterns in autism suggests subtypes. *Journal of Autism and Developmental Disorders*, 16(155–167).
- Tateno, M., Tateno, Y., & Saito, T. (2008). Comorbid childhood gender identity disorder in a boy with Asperger syndrome. *Psychiatry and Clinical Neurosciences*, 62(2), 238.
- Wheelwright, S., Baron-Cohen, S., Goldenfeld, N., Delaney, J., Fine, D., Smith, R., et al. (2006). Predicting Autism Spectrum Quotient (AQ) from the systemizing quotient-revised (SQ-R) and empathy quotient (EQ). *Brain Research*, 1079(1), 47–56.
- Wheelwright, S., Auyeung, B., Allison, C., & Baron-Cohen, S. (2010). Defining the broader, medium and narrow autism phenotype among parents using Autism Spectrum Quotient (AQ). *Molecular Autism*, 1(10), 1–9.
- Woodbury-Smith, M. R., Robinson, J., Wheelwright, S., & Baron-Cohen, S. (2005). Screening adults for Asperger syndrome using the AQ: A preliminary study of its diagnostic validity in clinical practice. *Journal of Autism and Developmental Disorders*, 35(3), 331–335.
- Zucker, K. (2005). J. Gender identity disorder in girls. In D. J. Bell, S. L. Foster, & E. J. Mash (Eds.), *Handbook of behavioral and emotional problems in girls* (pp. 285–319). New York: Kluwer Academic Publishers.