In: Personality and Individual Differences, 48(4), 357-366. doi:10.1016/j.paid.2009.09.018
The original publication is available at www.sciencedirect.com
Note that this contains additional references that were excluded from the original version.

BIOLOGICAL AND PSYCHOSOCIAL CORRELATES OF ADULT GENDER-VARIANT IDENTITIES: A REVIEW

Jaimie F. Veale, M.A.[1], Dave E. Clarke, Ph.D.

School of Psychology, Massey University, Albany Campus, New Zealand.

Terri C. Lomax, Ph.D.

Independent researcher, Wellington, New Zealand.

ABSTRACT

This article reviews research on biological and psychosocial factors relevant to the etiology of gender-variant identities. There is evidence for a genetic component of gender-variant identities through studies of twins and other within-family concordance and through studies of specific genes. Evidence that prenatal androgens play a role comes from studies that have examined finger length ratios (2D:4D), prevalence of polycystic ovary syndrome among female-to-male transsexuals, and individuals with intersex and related conditions who are more likely to have reassigned genders. There is also evidence that transsexuals have parts of their brain structure that is typical of the opposite birth-assigned gender. A greater likelihood of non-right-handedness suggests developmental instability may also contribute as a biological factor. There is a greater tendency for persons with gender-variant identities to report childhood abuse and a poor or absent relationship with parents. It is unclear if this is a cause or effect of a gender-variant identity. Parental encouragement of gender-variance is more common among individuals who later develop a gender-variant identity. We conclude that biological factors, especially prenatal androgen levels, play a role in the development of a gender-variant identity and it is likely that psychosocial variables play a role in interaction with these factors.

KEY WORDS: transsexualism; transvestism; development; etiology; gender identity; prenatal androgens; gender dysphoria; abuse.

[1] To whom correspondence should be addressed at School of Psychology, Massey University, Auckland Campus, Private Bag 102- 904, North Shore Mail Centre, Auckland 0745, New Zealand; e-mail: jfveale@yahoo.co.nz

INTRODUCTION

Although they do not necessarily imply causation, biological and psychosocial correlates can give insight into the etiology of transsexualism and other gender-variant identities (e.g. transvestites, cross-dressers, drag artists). A *gender-variant identity* occurs when a person identifies as a gender other than what they were assigned at birth (Docter, 1988). In this article, the term *gender-variance* is used to refer to the expression of this identity. Transsexuals are persons who have a sustained gender identity that is discrepant with their birth-assigned sex and have a desire to alter their bodily appearance to match this. Cross-dressers/transvestites are defined as persons who dress in clothing of the opposite sex for emotional relief and/or sexual arousal. These persons almost always report of history of heterosexual or bisexual partner preference, and although they may experience a gender identity that is at variance with their birth-assigned sex, this identity is not strong or persistent enough to occupy the individual's entire gender identity (Docter, 1988). All findings reported in this review were statistically significant.

It has been suggested that there are two types of female gender identity that can develop in birth-assigned males. One of these types has its origins in the femininity expressed by some homosexual males and the other has its origins in the cross-gender eroticism experienced also by heterosexual male transvestites (Blanchard, 2005; Freund, Steiner, & Chan, 1982). Proponents of this typology believe that these male-to-female (MF) transsexuals can be distinguished by one group having exclusive sexual attraction to males (androphilic) and the other group not (non-androphilic). Because it has been proposed that these types have distinct biological etiologies (Bailey & Triea, 2007; Freund & Blanchard, 1993; Freund et al., 1982), we report the sexual orientation of MF transsexuals in the research we are reviewing where this information is available. An analogous typology is generally not applied to female-to-male (FM) transsexuals due to the belief that this group seldom experiences cross-gender eroticism (Chivers & Bailey, 2000).

BIOLOGICAL CORRELATES

Genetics

Within-family concordance

A number of case reports have noted within-family concordance of gender-variant identities. Ball (1981) reported on three probably androphilic MF transsexual siblings in one

family who had an exclusively homosexual brother. Sabalis, Frances, Appenzeller, and Moseley (1974) also described three such siblings in one family. Hore, Nicolle, and Calnan (1973) and Stoller and Baker (1973) both reported occurrence of two androphilic MF transsexual siblings in one family. Hastings (1974, as cited in Freund, 1985) described two MF transsexuals who were half-siblings. Joyce and Ding (1985) reported a pair of sibling FM transsexuals. Edelstein (1960, as cited in Freund, 1985) described a transvestite patient with a transvestic older brother, Liakos (1967) described two sons sharing their transvestism with their father. There are three case reports of fathers and sons concordant for transvestism (Kasantikul & Roback, 1978; Krueger, 1978; Zucker & Blanchard, 1997).

These case reports give us little insight as to whether a family member of a person with a gender-variant identity is more likely to also develop a gender-variant identity than persons in the general population. Fortunately, six additional reports have examined within-family concordance among samples of persons with gender-variant identities. Results of these studies are summarized in Table 1.

Overall, these studies indicate the prevalence of transsexuality in the relatives of transsexuals appears to be higher than population prevalence estimates[1]. However, given transvestism has been estimated to occur among 2%-6% of the male population (see Lawrence, 2009 for a review), there is no evidence for elevated familial co-occurrence of transvestism.

Arguing a case for a genetic component for transsexuality, Green (2000b) noted that social learning could not have been a factor in his parent-child cases because in all of the cases the child did not know about their parent's gender-variant identity before they were aware of their own cross-gender feelings.

Genetic factors can be measured as the amount of greater concordance of transsexualism among monozygotic (MZ) twins than dizygotic (DZ) twins. Table 2 outlines twin pairs concordant and discordant for transsexualism that have been reported in the academic literature. From this table it can be seen that MZ twins are more likely to be concordant for transsexualism than DZ twins, $\chi^2(4) = 15.05$, $p < .01$, $n = 83$.

It is worth noting that this analysis drew 41% of cases from one conference paper that has not been peer-reviewed (Diamond & Hawk, 2004). When these cases are removed from

[1] The most liberal population estimates for transsexualism have been 1:2,900 MF and 1:8,300 FM in Singapore (Tsoi, 1988), and 1:4,470 MF and 1:26,818 among New Zealanders (Veale, 2008).

Table 1. Summary of studies of within-family concordance of gender-variance

Reference	Sample	Within-family concordance
Randell (1971)	340 transvestic clinical patients	Three "familial cases" (2.6%)
Buhrich (1978)	70 male members of cross-dressing clubs	Three (4.3%) had a first degree relative that cross-dressed – one father, one brother, and one sister
Croughan, Saghir, Cohen, and Robins (1981)	70 male members of cross-dressing clubs	One (1.4%) father, two (3.2%) brothers, and one (1.4%) sister cross-dressed
Green (2000b)	Clinic sample of 1500 transsexuals	Four MF transsexual sibling sets[1]; one MF transsexual with a gender-dysphoric birth-assigned female sibling; one FM transsexual siblings set; one MF transsexual with a gender dysphoric father[2]; one MF transsexual with a transvestite son[2]; one transvestite father with a gender dysphoric son; one transvestite father with a FM transsexual offspring.
Veale, Clarke, and Lomax (2008a)	Internet sample of 579 transsexuals, 574 other gender-variant, and 552 NGVs of both genders.	Three transsexuals reported transsexual relatives, giving prevalence ratios of 1:1,567 in MF transsexuals' relatives and 1:1,209 in FM transsexuals' relatives. The transsexual and other gender-variant groups reported a higher proportion of gender-variant relatives than the NGV group.
Gómez-Gil et al. (in press)	Clinic sample of 677 MF transsexuals and 318 FM transsexuals	Eleven MF transsexuals (1.6%) had a transsexual sibling (nine MF siblings and two FM siblings) and one FM transsexual (.003%) had a FM sibling.

Note: [1] sexual orientation not given for these pairs although one of these pairs had both been married; [2] both probands reported a history of gynephilic sexual attraction.

this analysis it is no longer statistically significant, $\chi^2(4) = 7.88$, $p = .10$, $n = 49$. It is also worth noting that there is no check on the equal environments assumption in this analysis. This is the assumption that MZ twins are not treated more similarly than DZ twins in ways that might affect their gender identity. Bailey, Dunne, and Martin (2000) were able to test this assumption among a large representative sample of Australian twins and found that twins that

Table 2. *Reports of twins at least one case of transsexualism in the academic literature*

Zygocity and birth assignment	Number concordant	References	Number discordant	References	Total
MZ Male	16 (10.5)	(Anchersen, 1956; Diamond & Hawk, 2004 [7]; Gooren, 1984, Gooren et al., 1989, and Latour, 1997, as cited in Diamond & Hawk, 2004; Green, 2000b [2]; Hyde & Kenna, 1977; Tsur, Borenstein, & Seidman, 1991; Zucker & Bradley, 1995)	15 (20.5)	(Diamond & Hawk, 2004 [8]; Chazen, 1995, Gooren et al., 1989 [3], and Stoller, 1976, as cited in Diamond & Hawk, 2004; Hepp, Milos, & Braun-Scharm, 2004; Zucker & Bradley, 1995)	31
DZ Male	1 (4.7)	(Gooren et al., 1989, as cited in Diamond & Hawk, 2004)	13 (9.3)	(Diamond & Hawk, 2004 [6]; Gooren, 1984 [2], Gooren et al., 1989 [3], and Maghazji, 1985, as cited in Daimond & Hawk, 2004; Vujovic, Popovic, Sbutega-Milosovec, Djordevic, & Gooren, 2009)	14
MZ Female	11 (9.1)	(Benjamin, 1971 [2]; Diamond & Hawk, 2004 [2]; de Vaal, 1975, Harima, 2003, and Hewitt & Warren, 1996, as cited in Diamond & Hawk, 2004; Green, 2000b; Broadbent, 1996, as cited in Green 2000b; Knoblauch, Busjahn, & Wegener, 2007; Sadeghi & Fakhrai, 2000)	16 (17.9)	(Diamond & Hawk, 2004 [5]; Gooren, 1984 [2], Hammond, 1995, and Stoller, 1976, as cited in Diamond & Hawk, 2004; Martin, 1981, as cited in Freund, 1985; Garden & Rothery, 1992; Green & Stoller, 1971; Segal, 2006 [2]; Hewitt, 1995 and Hutchinson, 2000, as cited in Segal, 2006)	27
DZ Female	0 (1.7)		5 (3.3)	(Diamond & Hawk, 2004 [4]; Vujovic et al., 2009)	5
DZ Male and Female	0 (2.0)		6 (4.0)	(Diamond & Hawk, 2004 [2]; de Vaal, 1975 [3] and Gooren, 1984, as cited in Diamond & Hawk, 2004)	6
Total	28		55		83

Note – figures in parentheses are the expected value if concordance for transsexualism was equally split between MZ and DZ twins (the null hypothesis); figures in brackets refer to the number of cases observed in this reference – references without a figure observed one case.

were more similar on adult gender identity were not more likely to report a more similar environment than those that were less similar. However, this study was not able to test any environmental factors that may be specifically relevant to gender identity development. Bailey et al. estimated that 31% (95% confidence intervals 0%-44%) of the variance of adult gender identity was accounted for genetic factors, 69% (53%-85%) was accounted for by non-shared environmental factors, and 0% (0%-30%) was accounted for by shared environmental factors among males. Among females the corresponding percentages were 24% (0%-42%), 67% (56%-79%), and 9% (0%-35%). However, because this study was not likely to include many participants with gender-variant identities, the reader should be cautious in extrapolating these findings to this population.

In all cases of twins reported to be concordant for transsexualism, sexual orientation was also reported to be concordant where this data was given. This should be expected to a certain degree, as a significant concordance of sexual orientation among non-transsexual twins has also been reported (Bailey et al., 2000; Kendler, Thornton, Gilman, & Kessler, 2000). No twin data on non-transsexual gender-variant persons has previously been reported.

Studies of specific genes

There is some evidence that three genes that have been associated with sexual differentiation of brain structures correlate with transsexualism. Henningson et al. (2005) found 29 MF transsexuals scored differently from 229 males with no gender-variant identity (NGV) on the estrogen receptor gene, although most transsexuals' scores were still within normal range. Henningsson et al. suggested that specific combinations of androgen receptor, aromatase, and estrogen receptor genes may be more relevant to the development of transsexualism and they found evidence for this with an interaction effect of these three genes in predicting transsexuality using a logistic regression analysis. This method was replicated by Hare and colleagues (2009) among 112 MF transsexuals and 258 NGV males. The results did not to replicate the estrogen receptor finding, but Hare et al. did find that MF transsexuals differed on the androgen receptor gene, suggesting a greater likelihood of reduced sensitivity to androgens among transsexuals.

Hengstschläger et al. (2003) found no detectable genetic abnormalities among 30 MF or 31 FM transsexuals at the chromosomal level, or at the molecular level on a gene associated with androgen receptor or the SRY gene (sex determining region). Another study found no genetic differences between transsexuals (MF or FM) and NGVs of the same birth-

assigned sex in the steroid 5α reductase gene which is involved with androgen metabolism (Bentz et al., 2007). The same research team also found that a variant of the CYP17 gene which influences the metabolism of sex hormones and leads to above average tissue concentrations of both estrogens and androgens was carried by more FM transsexuals (44%) than female NGVs (31%). MF transsexuals did not differ from male NGVs (Bentz et al., 2008). Data on sexual orientation of MF transsexuals was not collected in any of these genetic studies.

Care is needed in interpreting the findings of these genetic studies. Although the differences are statistically significant, there is still large proportion of the population who have these gene patterns yet are not transsexuals and a large proportion of transsexuals who do not have these gene patterns. There is also a lack of power in many of the studies that found non-significant results. The strongest conclusion these genetic studies allow us to draw is that genetic determinants of hormones play a small role in increasing the likelihood of transsexualism.

A recent study has found FM transsexuals have metric teeth features that are intermediary between NGV males and females (Antoszewski, Zadzinska, & Foczpanski, 2009). The authors suggested this finding provides evidence for a genetic cause of FM transsexuality.

Prenatal Hormones

Almost all of the evidence of sex-atypical hormone levels among persons with gender-variant identities has been of androgen levels. Androgen levels are much higher in men than women and these hormones play a role in masculinity development both pre- and post-natally (Nelson, 2005).

The most compelling evidence for the effects of prenatal androgens on adult gender identity in humans comes from studies of the gender identity of persons with intersex and related conditions. These persons are exposed to prenatal androgen levels that are at variance to either their genetic make-up (male or female sex chromosomes) or the gender they were assigned at birth. Recent reviews have shown that these individuals are much more likely to change from the gender they were assigned at birth than persons without these conditions. Veale, Clarke, and Lomax (2008b) summarized 451 cases reported in the academic literature of persons with prenatal androgen exposure at variance with their birth-assigned gender followed up in adulthood. Table 3 summarizes gender identity outcomes of these cases and it is clear that persons who are exposed to levels of prenatal androgens that are intermediary

between male- and female-typical levels are more likely to be reported as gender dysphoric and/or reassign their gender than persons from the general population, and persons exposed to male-typical prenatal androgen levels but assigned female at birth are even more likely to be reported as gender dysphoric and/or reassign gender. There have not been reports in the academic literature of persons exposed to female typical prenatal androgen levels assigned the male gender at birth.

Table 3. *Summary of gender dysphoria and gender change in adults with intersex and related conditions*

Condition	Prenatal androgen level	Gender assignment	Gender-variant identity outcomes
Congenital adrenal hyperplasia	Intermediary	147 female	3.4% male, 4.1% gender dysphoric
		14 male	14.3% female
Partial androgen insensitivity	Intermediary	39 female	7.7% male
		16 male	31.3% female
Micropenis	Intermediary	6 female	None
		69 male	2.9% gender dysphoric
Mixed gonadal dysgenesis	Intermediary	10 female	10.0% male, 20.0% gender dysphoric
		14 male	7.1% female
5α reductase deficiency	Male-typical	86 female	68.6% male
17β hydroxysteroid dehydrogenase	Male-typical	29 female	55.2% male
Cloacal exstrophy	Male-typical	13 female	30.8% male, 15.4% gender dysphoric
Penile agenesis	Male-typical	4 female	25.0% male
Penile ablation	Male-typical	4 Female	50.0% male, 25.0% gender dysphoric

See Veale et al. (2008b) for references

Veale et al. (2008b) analyzed these outcomes using a logistic regression and found that prenatal androgen exposure and sex of assignment at birth equally predicted adult gender identity among this population. They also found that sex chromosomes and time delayed before sex assignment were not significant predictors of adult gender identity, although these variables had a restricted range with the population tested.

Care is needed in extrapolating findings of change of sex in persons with intersex and related conditions with those who do not have these conditions. Meyer-Bahlburg et al. (1996)

argued that transsexuals experience more gender *dysphoria* whilst intersex persons experience more gender *confusion or uncertainty*, and that the gender-variant identity formation appears to develop later in intersex persons than those with early-onset transsexualism. Intersex persons often also experience ambiguous genitalia, early genital surgery, and sometimes develop cross-gender secondary sex characteristics – all of which non-intersex transsexuals generally do not experience. The effects of these on an emerging gender identity are unclear.

Only one study has directly tested the relationship between prenatal androgen levels and adult gender-variance. This study found prenatal androgen levels measured by maternal blood samples positively related to adult gender-role behaviour among females (Udry, Morris, & Kovenock, 1995).

Associations have also been made between a number of less direct measures of prenatal androgen exposure and gender-variance. The ratio between the length of the second and fourth fingers (2D:4D) is widely believed to be an indicator of prenatal sex hormone levels (e.g. Putz, Gaulin, Sporter, & McBurney, 2004). A number of studies have shown that females have greater 2D:4D ratios than males on average (Manning et al., 2000; Manning, Churchill, & Peters, 2007; Manning, Scutt, Wilson, & Lewis-Jones, 1998). Studies of 2D:4D of persons with gender-variant identities are outlined in Table 4. All of these studies used experimenter measures from photocopies except for Veale, Clarke, and Lomax (2008a) who used participant self-measure. Generally, these studies have either found transsexuals do not differ from NGV persons of the same birth-assigned gender or there were differences in the expected direction, providing some further evidence that prenatal androgens play a role in gender-variant identity development.

One study found 3 transsexuals (one MF and two FM) out of a group of 243 Dutch persons who had been exposed to anticonvulsants prenatally. This is a much higher prevalence than expected due to chance, and anticonvulsants have been demonstrated to alter prenatal hormone levels (Dessens et al., 1999).

Women with polycystic ovary syndrome (PCOS) experience elevated levels of androgen exposure postnatally and possibly prenatally (Xita & Tsatsoulis, 2006). Some studies have shown elevated rates of PCOS among FM transsexuals who have not yet begun cross-sex hormone treatment (reviewed in Table 5). Many of the earlier studies lacked a consistent definition of PCOS, so the newer Rotterdam criteria (Rotterdam ESHRE/ASRM-Sponsored PCOS Consensus Workshop Group, 2004) was applied to these. Overall, the prevalence of 29% of 294 FM transsexuals (Wald 95% confidence intervals 24%-35%) was

Table 4. *Summary of studies of 2D:4D among persons with gender-variant identities*

Reference	Sample	Findings[1]
Schneider, Pickel, and Stalla (2006)	63 MF and 43 FM transsexuals; 58 male and 65 female NGVs	MF transsexuals differed from male NGVs on right hand but not left hand; no differences between FM transsexuals and NGV females
Wallien, Zucker, Steensma, and Cohen-Kettenis (2008)[2]	96 MF and 51 FM transsexuals; 90 male and 112 female NGVs	No differences between MF transsexuals and NGV males; FM transsexuals differed from female NGVs on both right and left hands
Kraemer et al. (2009)	39 MF and 17 FM transsexuals; 176 male and 190 female NGVs	MF transsexuals differed from male NGVs on right hand but not left and no effect of sexual orientation on this; right handed FM transsexuals had hyper-feminized 2D:4D compared to NGV females
Veale et al. (2008a)	Convenience internet sample of 453 MF and 126 FM transsexuals; 574 other gender-variant persons (382 birth-assigned male and 192 birth-assigned female); 207 male and 345 female NGVs	No between group differences; 2D:4D predicted adult gender-variance (continuously measured) in regression models for both birth-assigned genders; 2D:4D was unrelated to sexual orientation in this sample

Note: [1] All findings in the expected direction (i.e. a lower 2D:4D signaling a more feminine gender identity and a higher 2D:4D signaling a more masculine gender identity) unless otherwise noted. [2] This study also did not find differences between children diagnosed with gender identity disorder and NGV children of the same birth-assigned gender.

greater than the most liberal population estimates of PCOS in NGV females, 13% (Mueller et al., 2008).

Table 5. *Summary of studies of PCOS among FM transsexuals*

Reference	Sample	Proportion meeting Rotterdam criteria for PCOS[1]
Futterweit, Weiss, and Fagerstrom (1986)	40 FM transsexuals	Eleven (28%)
Spinder, Spijkstra, Gooren, and Burger (1989)	16 FM transsexuals	None, but ultrasound was not measused
Balen, Schachter, Montgomery, Reid, & Jacobs (1993)	16 FM transsexuals	Seven (44%)
Bosinski et al. (1997)	16 FM transsexuals	Eight (50%)
Baba et al. (2007)	69 FM transsexuals	40 (58%)
Mueller et al. (2008)	61 FM transsexuals and 94 NGV females	Nine (15%), this was not more than NGVs (13%)
Vujovic, Popovic, Sbutega-Milosovec, Djordevic, and Gooren (2009)	76 FM transsexuals	Eleven (14%)
Total	294 FM transsexuals	86 (29%)

Note: [1] To meet this criteria, must have two of: clinical (hursutism) and/or bio-chemical signs of androgen excess, oligo-amenohhrea (nine or fewer menstrual cycles per year), or ultrasound evidence of polycystic ovaries.

Finally, scores on the "Systematizing Quotient" has been associated with prenatal androgen exposure (Auyeung et al., 2006). Systematizing refers to a persons' ability to understand and construct systems and it has used in explaining the development of autism which has been conceptualized by some as having an extreme male brain (Baron-Cohen, 2002). Veale et al. (2008a) found differences between participants with gender-variant identities (including transsexuals) and NGV participants in scores on the Systematizing Quotient and that this variable was a strong predictor of adult gender-variance in a regression model among both sexes. The Systematizing Quotient was not related to social desirability in this study.

Neuroanatomical Correlates

One research team has found a sex differentiation in the neuron volume of the central subdivision of the bed nucleus of the stria terminalis (BSTc) of the hypothalamus and in postmortem examinations of transsexuals' brains found that their BSTc neuron volume matched that of their opposite birth-assigned sex (Kruijver et al., 2000; Zhou, Hofman, Gooren, & Swaab, 1995). Their sample included six MF transsexuals and one FM transsexual who had received hormone treatment for a significant period of time. These MF transsexuals were all non-androphilic (Garcia-Falgueras & Swaab, 2008). Kruijver et al. also included a male who "had very strong cross-gender identity feelings" (p. 2039) but never received hormone treatment in their sample and found a BSTc neuron volume within the female range for this person. Both studies also included six "sex hormone disorder" cases that had atypical hormone levels but NGV, and came to the conclusion that the BSTc differences were not associated with hormone treatment in either the transsexuals or NGVs. Given their findings, Zhou et al. and Kruijver et al. proposed a neurobiological basis as an explanation of transsexualism.

More recent research has discovered that sex differences in BSTc volume do not develop until well into adulthood (Chung, De Vries, & Swaab, 2002) and yet the majority of transsexuals report that their transsexual feelings began before adulthood (Veale, Clarke, & Lomax, 2009). Lawrence (2007) also argued that there is insufficient evidence that these brain differences were not the effect of hormone treatment, given studies finding hormone treatment for MF transsexuals decreased overall brain volume, and in FM transsexuals both hypothalamic and overall brain volume increased with hormone treatment (Hulshoff Pol et al., 2006). A study has also found that pedophilic offenders also have a smaller BST size than non-pedophilic males (Schiltz et al., 2007). Finally, there is also evidence from experimental studies of animals and correlational studies of humans that childhood stress (including abuse) has an impact on brain structure, including the hypothalamus (Kaufman, Plotsky, Nemeroff, & Charney, 2000; Teicher, Tomoda, & Andersen, 2006). Clearly, further independent research using larger samples is needed to assess the BSTc as a proposed neurobiological basis as an explanation of transsexualism.

The interstitial nucleus of the anterior hypothalamus nuclei 3 (INAH-3) has previously been shown to be sexually dimorphic and it has been reported that NGV homosexual males also have a female-typical INAH-3 size (Byne et al., 2001; LeVay, 1991). Garcia-Falgueras and Swaab (2008) found 11 MF transsexuals had an INAH-3 that was a

smaller, female-typical size. The group differed from 14 NGV males but not from 11 NGV females. They also reported the INAH-3 size of one FM transsexual was within the male range. Given that most of the MF transsexuals in this research were non-androphilic this finding has interesting implications for the relationship between sexual orientation and gender identity. Garcia-Falgueras and Swaab found that INAH-3 size of five castrated males was intermediary between male and female NGVs, suggesting that androgen level changes in adulthood may have an effect on INAH-3 size.

Other research has found that 22 MF and 28 FM transsexuals have corpus collosum midsaggital plane shapes (measured using MRI scans) on average more typical of those of the gender they identify as when compared to NGVs (Yokota, Kawamura, & Kameya, 2005). A recent study has found that non-androphilic MF transsexuals who had not been taking cross-sex hormones showed a female-typical hypothalamus activation pattern when smelling odorous steroids known to activate the hypothalamus (Berglund, Lindstrom, Dhejne-Helmy, & Savic, 2009). Another study found that 6 androphilic and 18 gynephilic MF transsexuals who had not commenced hormone therapy had gray matter variation that was more similar to NGV males than NGV females, although transsexuals of both sexual orientations had a female-typical volume of grey matter in the left putamen (Luders et al., 2009).

Underlying differences in neuroanatomic structure can also be manifested in cognitive test performance. Studies of cognitive abilities of persons with gender-variant identities are outlined in Table 6. Almost all of this research has been conducted on transsexuals. Overall, these results are mixed, with some studies showing no differences between transsexuals and NGVs of the same birth-assigned sex and some showing transsexuals' cognitive test performance in line with their gender identity. It also appears that transsexuals' hormone treatment does not have much effect on the results (e.g. Haraldsen, Egeland, Haug, Finset, & Opjordsmoen, 2005). The most likely reason for these mixed findings is the small sample sizes of both transsexuals and NGVs in most of the studies. It is also possible that studies that included more transsexuals who were sexually attracted to the same birth-assigned sex (Collaer, Reimers, & Manning, 2007; Peters, Manning, & Reimers, 2007) were more likely to report between-group difference because this sexual orientation has been associated with sex-atypical results on these cognitive tests among NGVs.

Table 6. *Summary of studies of cognitive tests among persons with gender-variant identities*

Reference	Sample	Findings
La Torre, Gossmann, and Piper (1976)	8 MF transsexuals, 12 male and 14 female NGVs	MF transsexuals differed from male NGVs but not female NGVs on an embedded figures test
Cohen-Kettenis et al. (1998)	44 MF and 34 FM transsexuals pre hormone treatment, 51 male and 29 female NGVs	Transsexuals scored atypically of the same birth-assigned sex on a verbal memory test but not on a two-dimensional test of spatial ability
van Goozen, Slabbekoorn, Gooren, Sanders, & Cohen-Kettenis (2002)	22 MF and 19 FM transsexuals pre hormone treatment, 20 male and 23 female NGVs	No differences between groups on a two dimensional mental rotation task and verbal reasoning test; MF and FM transsexuals scored intermediary between NGV groups on judgement of line orientation, three-dimensional mental rotation, and targeted throwing
Haraldsen et al. (2003)	22 MF and 30 FM transsexuals pre hormone treatment, 14 male and 15 female NGVs	No effect of gender identity on two-dimensional rotation, visualization, perception, verbalization, logic, or artithmetic tests.
Menaged (2004)	20 MF transsexuals; 20 male and 20 female NGVs	Transsexuals and NGV females did not differ from each other but scored lower than NGV males on a three-dimensional mental rotation task.
Wisniewski, Prendeville, and Dobs (2005)	27 hormone treated MF transsexuals receiving and 16 NGV males	These groups did not differ on perceptual speed, map memory, and three-dimensional mental rotation tests
Veale et al. (2008a)	Large convenience internet sample of birth-assigned males and females; see Table 4	Three dimensional mental rotation performance was not related to gender identity or gender-variance continuously measured in either gender.

Handedness and Dermatoglyphics

Research into the handedness of persons with gender-variant identities is outlined in Table 7. Most of this research has been conducted on transsexuals. A consistently high proportion of non-right-handedness has been found among transsexuals in all of the controlled and non-controlled studies outlined[2]. Three studies have shown this occurs independently of sexual orientation in MF transsexuals (Green & Young, 2001; Veale et al., 2008a; Watson & Coren, 1992). Herman-Jeglinska, Dulko, and Grabowska (1997) noted that the elevated levels of non-right handedness was more pronounced in those transsexuals who did not have an immediate relative who was non-right handed compared to NGVs who also did not have an immediate relative who was non-right handed. They suggested this is a reflection of a non-genetic determinant of the relationship between transsexuality and non-right handedness. Herman-Jeglinska et al. also noted a greater prevalence of extreme-right handedness, which they operationally defined as scoring the maximum possible for right-handedness on a continuous scale. However, Veale et al. found no greater prevalence of extreme-right-handedness among participants with gender-variant identities and no interaction effect between handedness and number of older brothers that has been reported among NGV homosexual males (e.g. Blanchard & Lippa, 2008).

The causes of non-right handedness are not completely understood. There is evidence that the cause of at least some non-right handedness is genetic (e.g. Medland et al., 2009). However, two other explanations for non-right handedness are more likely to have relevance to gender-variant identity development: elevated prenatal androgens and developmental instability. There is evidence that non-right handedness is associated with increased prenatal androgen levels from a studies of females with CAH (Helleday, Siwers, Ritzen, & Hugdahl, 1994; Kelso, Nicholls, Warne, & Zacharin, 2000; Nass et al., 1987) and another study showing a link between handedness and genes associated with the androgen receptor (Medland et al., 2005). Whilst this explanation of elevated prenatal androgen levels causing non-right handedness is consistent with theory and research of prenatal hormones among birth-assigned females with gender-variant identities, lower levels of non-right handedness would be expected among birth-assigned males with gender-variant identities who theory and research has aligned with low prenatal hormone levels.

---

[2] One study has also found have elevated rates of left-handedness among boys diagnosed with childhood gender identity disorder (Zucker, Beaulieu, Bradley, Grimshaw, & Wilcox, 2001).

Table 7. *Summary of studies of handedness among persons with gender-variant identities*

Reference	Sample	Findings
Orlebeke, Boomsma, Gooren, and Verschoor (1992)	93 MF and 44 FM transsexuals	Both transsexual groups were almost twice as likely to be left-handed than estimates of the general population
Watson and Coren (1992)	45 MF transsexuals of varying sexual orientations and 225 age-matched NGV males	Transsexuals were more than three times more likely to be left-handed than age-matched males
Herman-Jeglinska et al. (1997)	12 MF and 70 FM transsexuals; 148 male and 331 female NGVs	FM transsexuals more likely to be non-right handed than NGV females; a non-significant trend towards non-right handedness among MF transsexuals
Cohen-Kettenis van Goozen, Doorn, and Gooren (1998)	46 MF and 47 FM transsexuals	3 MF (7%) and 13 (28%) transsexuals were non-right handeded
Slabbekoorn et al. (2000)	184 MF and 110 FM transsexuals; 158 male and 164 female NGVs	Both transsexual groups more likely to be non-right handed than NGVs (our analysis – *p*s <.01, Fisher's exact test)
Green and Young (2001)	443 MF and 93 FM transsexuals; 144 male and 140 female NGVs	Both transsexual groups more likely to be non-right handed than NGVs, for MF transsexuals
Veale et al. (2008a)	Large convenience internet sample of birth-assigned males and females; see Table 4	Gender-variant (including transsexual) participants were more likely to score non-right handed than NGVs among both sexes.

A second causal explanation of non-right handedness, developmental instability, can account for the elevated level of non-right handedness among persons with gender-variant identities of both birth-assigned sexes is. Developmental instability refers to a persons' susceptibility to developmental disturbances; such disturbances could be environmental (e.g. exposure to pathogens) or genetic (e.g. gene mutations) during early development. These result in reduced reproductive fitness and increased likelihood of developing

neurodevelopment disorders such as autism and schizophrenia. According to this theory, non-right handedness (as well as extreme-right handedness) is an indicator of developmental instability and the finding of elevated levels of non-right handedness among transsexuals suggests that such developmental disturbances have a role in the cause of transsexualism (see Lalumiere, Blanchard, & Zucker, 2000 for a review of the theory of developmental instability and its application to sexual orientation development).

Another morphological phenomenon relevant to prenatal androgens and developmental instability is dermatoglyphics. It has been proposed that total finger ridge count is positively associated with prenatal androgen levels and finger ridge fluctuating asymmetry is associated with developmental instability. Two studies have assessed dermatoglyphics among both FM and MF transsexuals. Slabbekoorn, van Goozen, Sanders, Gooren, and Cohen-Kettenis (2000) did not find any differences between transsexuals (FM or MF) and NGVs of the same birth-assigned sex on total finger ridge count or fluctuating asymmetry. Green and Young (2000) also found no differences between total ridge count between transsexuals and NGVs but found a difference between androphilic MF transsexuals and non-androphilic MF transsexuals and NGVs on fluctuating asymmetry.

Familial Correlates

Differences in birth order have also been found among transsexuals. Tsoi, Kok, and Long (1977) found androphilic MF transsexuals from Singapore have a later than expected birth order, although they did not employ a comparison group. Three studies have found that androphilic MF transsexuals have a greater number of brothers than sisters and later birth order than non-androphilic MF transsexuals and FM transsexuals (Blanchard & Sheridan, 1992; Blanchard, Zucker, Cohen-Kettenis, Gooren, & Bailey, 1996; Green, 2000a). Veale et al. (2008a) also found an elevated numbers of older brothers but not an elevated sibling sex ratio among birth-assigned males with gender-variant identities; however, they also found that this birth-order effect occurred regardless of sexual orientation typology.

While two studies have found that transvestites have shown the opposite birth order effect – a tendency to be more likely to be the oldest birth-assigned male or only child (Prince & Bentler, 1972; Schott, 1995), these studies did not include comparison groups and this finding has not been replicated in two other studies (V. L. Bullough, Bullough, & Smith, 1983; Veale et al., 2008a).

Green and Keverne (2000) found MF transsexuals, regardless of sexual orientation, had a greater number of maternal aunts than maternal uncles. However, this finding was not replicated among a large internet-based sample (Veale et al., 2008a).

PSYCHOSOCIAL CORRELATES

We included birth order and sibling sex ratio under the biological correlates heading because of convincing evidence that this correlate has a biological origin among NGV homosexual males (Bogaert, 2000, 2006).

Evidence from Persons with Intersex and Related Conditions

In the review by Veale et al. (2008b; see Prenatal Hormones section) of persons with intersex and related conditions, it was found that after controlling for biological factors (prenatal androgen levels) those with who were assigned a female gender at birth or early childhood were 61 times more likely to develop a female gender identity in adulthood than those who were assigned male at birth. The gender a child was reared as had an effect on gender identity that was at least as strong as the effect of prenatal androgens, indicating that psychosocial factors play a significant role in gender identity development among these persons.

Parental factors

A less warm, more emotionally distant, controlling or rejecting father has been associated with transsexualism in two studies with NGV comparison groups (Cohen-Kettenis & Arrindell, 1990; Parker & Barr, 1982). However, two studies have found that androphilic MF transsexuals reported being no less close to their father than homosexual males (Buhrich & McConaghy, 1978; Freund, Langevin, Zajac, Steiner, & Zajac, 1974). There is also evidence from studies of NGV participants that this poor relationship is associated with the child's feminine expression rather than later sexual orientation (Freund & Blanchard, 1983).

Two studies have found that MF transsexuals and birth-assigned male transvestites were more likely to report their parents wishing for a girl than NGV males (Buhrich & McConaghy, 1978; Hogan-Finlay, 1995). Buhrich and McConaghy also found that transvestites and MF transsexuals reported higher mothers' involvement and lower fathers' involvement in their upbringing. However, they reported no evidence of "abnormal relationships" with the participants' mothers. Two studies have found that transvestites viewed their fathers as more dependent and affiliative than NGV men (Newcomb, 1985;

Schott, 1995). Schott found that 68% of his sample of 85 transvestites reported a neutral or negative relationship with their father, compared to only 41% of a comparison group of 44 NGV male.

One study has shown that gender clinic patients were more likely than psychiatric patients to report parent death – especially fathers and during adolescence and early adulthood (Bernstein, Steiner, Glaister, & Muir, 1981). Among a population sample, Långstrom and Zucker (2005) found an increased likelihood of separation from parents during childhood among males reporting transvestic fetishism. However, studies of NGV populations have not found any evidence for an absent father having an effect on gender development (Stevens, Golombok, Beveridge, & ALSPAC Study Team, 2002; Stevenson & Black, 1988) and two further studies have found that MF transsexuals transvestites, and NGV males did not differ in their reported parent relations to each other or likelihood of living with both parents during their childhood (Hogan-Finlay, 1995; Veale et al., 2008a).

Four studies have looked at familial encouragement of childhood gender-variance. Schott (1995) reported that 22% of his sample had had their cross-dressing initiated, and openly encouraged by a female family member during their formative years. Prince and Bentler (1972) reported that 4% of their sample of 504 transvestites were "made to wear dresses as punishment" and 6% were "kept in curls till longer than other boys" (p. 912). Hogan-Finlay (1995) found that MF transsexuals were more likely to report being dressed as a girl in childhood than transvestites who in turn were more likely to report this than NGV males. However, Talamini (1982) reported that few of his male transvestite research participants remembered being treated like a girl during childhood.

Veale et al. (2008a) found that gender-variant birth-assigned females had a tendency to have older mothers. However, this finding was inconsistent with one previous study (Blanchard & Sheridan, 1992).

Abuse

Studies of the prevalence of emotional, physical, and sexual abuse among persons with gender-variant identities are outlined in Table 8. Many of these studies have small samples and/or lack adequate comparison groups. In the studies that include comparisons there is evidence that persons with gender-variant identities are more likely to have experienced abuse. Two studies have also found increased levels of gender-nonconforming behavior and identity in girls who had been sexually abused (Aiosa-Karpas, Karpas, Pelcovitz, & Kaplan, 1991; Cosentino, Meyer-Bahlburg, Alpert, & Gaines, 1993).

Table 8. *Summary of studies of abuse among persons with gender-variant identities*

Reference	Sample	Emotional abuse	Physical abuse	Sexual abuse
Pauly (1974)	80 FM transsexuals	Not assessed	24 (30%)	23 (29%)
Lothstein (1983)	53 FM transsexuals	Not assessed	26 (49%)	12 (23%)
Devor (1994)[1]	45 FM transsexuals	13 (29%)	17 (38%)	14 (31%)
Hogan-Finlay (1995)	27 MF transsexuals, 64 transvestites, 101 male NGVs	Gender-variant groups scored higher	No differences between groups	No differences between groups
B. Bullough and Bullough (1997)	41 MF transsexuals, 331 transvestites	Not assessed	Not assessed	37 (10%)
Kersting et al. (2003)	29 MF and 12 FM transsexuals, 56 male and 59 female psychiatric patients	Transsexuals scored higher	No differences between groups	Transsexuals scored lower
Gehring and Knudson (2005)	34 MF and 8 FM transsexuals	50-80%	20-30%	23 (55%) unwanted sexual event
Långstrom and Zucker (2005)	36 reporting sexual arousal from cross-dressing from a population sample of 2,450	Not assessed	Not assessed	Increased likelihood among those reporting transvestism
Grossman, D'Augelli, and Salter (2006)	31 young MF transsexuals who used a drop-in centre	27 (87%) reporting "verbal abuse"[2]	11 (35%)[2]	5 (16%)
Wharton (2007)	Online-recruited gender-variant persons; birth-assignment 132 female, 163 male	Not assessed	63 (48%) birth-assigned females and 53 (33%) birth-assigned males reported physical or sexual abuse	
Veale et al. (2008a)	Large convenience internet sample of birth-assigned males and females; see Table 4	Gender-variant groups scored higher	Gender-variant groups scored higher	Gender-variant groups scored higher

Note: [1] A total of 60% of this sample reported at least one of these forms of abuse. [2] Those who had been more gender atypical in childhood (i.e. reported being called a sissy) were more likely to report experiencing verbal and physical abuse.

The meaning of this higher prevalence of abuse among transsexuals is not clear. Reflecting on interviews with FM transsexuals, Devor stated "I have speculated…that, in some cases, transsexualism may be an adaptive extreme dissociative survival response to severe child abuse" (1994, p. 49). On the other hand Gehring and Knudson (2005) believed that children with gender-variance are more of a target to abusers. They stated that their findings "do not support any notion that childhood trauma is associated with … the formation of transsexualism … but rather has more to do with society's prejudices about being transsexual" (p. 29). Using path analysis, Veale et al. (2008a) found that recalled childhood gender-variance mediated all of the significant abuse relationship among birth-assigned females but mediated only part of this effect among birth-assigned males. This indicates the abuse is the effect of birth-assigned females being targeted for abuse for expressing their gender-variant identity and that indicating the abuse plays a role in causing the gender-variant identity in some birth-assigned males.

CONCLUSIONS

There is evidence that biological factors, especially prenatal androgen exposure, play a significant role in the etiology of gender-variant identities. While there is also evidence for other biological correlates, this does not necessarily imply more than one biological factor plays a role – it is likely that they are related and share a common precursor. For instance, it is entirely plausible that there is a causal pathway from genes causing atypical prenatal hormones levels causing neuroanatomical differences and an adult gender-variant identity.

We know from studies of individuals with intersex and related conditions that it is not uncommon for an individual to have a male-typical prenatal environment (including androgen levels), be assigned a female sex at birth, and develop a female gender identity. Therefore, psychosocial factors also have a role to play in the etiology of gender-variant identities. There is evidence that a poor or absent parental relationship, childhood abuse, and parental encouragement of gender-variance are more common amongst gender-variant populations. It is unclear whether these are a cause or effect of gender-variance. It is likely that any psychosocial variables that play a causative role in the development of gender-variant identities are complex and work in interaction with biological variables.

REFERENCES

Aiosa-Karpas, C. J., Karpas, R., Pelcovitz, D., & Kaplan, S. (1991). Gender identification and sex role attribution in sexually abused adolescent females. *Journal of the American Academy of Child and Adolescent Psychiatry, 30*, 266-271.

Anchersen, P. (1956). Problems of transvestism. *Acta Psychiatrica et Neurologica (KjoBenhavn), 106*, 249-256.

Antoszewski, B., Zadzinska, E., & Foczpanski, J. (2009). The metric features of teeth in female-to-male transsexuals. *Archives of Sexual Behavior, 38*, 351-358.

Auyeung, B., Baron-Cohen, S., Chapman, E., Knickmeyer, R., Taylor, K., & Hackett, G. (2006). Foetal testosterone and the child systemizing quotient. *European Journal of Endocrinology, 155*, S123-S130.

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*, 1011-1016.

Bailey, J. M., Dunne, M. P., & Martin, N. G. (2000). Genetic and environmental influences on sexual orientation and its correlates in an Australian twin sample. *Journal of Personality and Social Psychology, 78*, 524-536.

Bailey, J. M., & Triea, K. (2007). What many transgender activists don't want you to know and why you should know it anyway. *Perspectives in Biology and Medicine, 50*, 521-534.

Balen, A. H., Schachter, M. E., Montgomery, D., Reid, R. W., & Jacobs, H. S. (1993). Polycystic ovaries are a common finding in untreated female to male transsexuals. *Clinical Endocrinology, 38*, 325-329.

Ball, J. R. (1981). Thirty years experience with transsexualism. *Australian and New Zealand Journal of Psychiatry, 15*, 39-43.

Baron-Cohen, S. (2002). The extreme male brain theory of autism. *Trends in Cognitive Sciences, 6*, 248-254.

Benjamin, H. (1971). Should surgery be performed on transsexuals? *American Journal of Psychotherapy, 25*, 74-82.

Bentz, E. K., Hefler, L. A., Kaufmann, U., Huber, J. C., Kolbus, A., & Tempfer, C. B. (2008). A polymorphism of the CYP17 gene related to sex steroid metabolism is associated with female-to-male but not male-to-female transsexualism. *Fertility and Sterility, 90*, 56-59.

Bentz, E. K., Schneeberger, C., Hefler, L. A., van Trotsenburg, M., Kaufmann, U., Huber, J. C., et al. (2007). A common polymorphism of the SRD5A2 gene and transsexualism. *Reproductive Sciences, 14*, 705-709.

Berglund, H., Lindstrom, P., Dhejne-Helmy, C., & Savic, I. (2009). Male-to-female transsexuals show sex-atypical hypothalamus activation when smelling odorous steroids. *Cerebral Cortex, 18*, 1900-1908.

Bernstein, S. M., Steiner, B. W., Glaister, J. T., & Muir, C. F. (1981). Changes in patients with gender-identity problems after parental death. *American Journal of Psychiatry, 138*, 41-45.

Blanchard, R. (2005). Early history of the concept of autogynephilia. *Archives of Sexual Behavior, 34*, 439-446.

Blanchard, R., & Lippa, R. A. (2008). The sex ratio of older siblings in non-right-handed homosexual men. *Archives of Sexual Behavior, 37*, 970-976.

Blanchard, R., & Sheridan, P. M. (1992). Sibship size, sibling sex ratio, birth order, and parental age in homosexual and nonhomosexual gender dysphorics. *Journal of Nervous and Mental Disease, 180*, 40-47.

Blanchard, R., Zucker, K. J., Cohen-Kettenis, P. T., Gooren, L. J. G., & Bailey, J. M. (1996). Birth order and sibling sex ratio in two samples of Dutch gender-dysphoric homosexual males. *Archives of Sexual Behavior, 25*, 495-514.

Bogaert, A. F. (2000). Birth order and sexual orientation in a national probability sample. *Journal of Sex Research, 37*, 361-368.

Bogaert, A. F. (2006). Biological versus nonbiological older brothers and men's sexual orientation. *Proceedings of the National Academy of Sciences, 103*, 10771-10774.

Bosinski, H. A. G., Peter, M., Bonatz, G., Arndt, R., Heidenreich, M., Sippell, W. G., et al. (1997). A higher rate of hyperandrogenic disorders in female-to-male transsexuals. *Psychoneuroendocrinology, 22*, 361-380.

Buhrich, N. (1978). Motivation for cross-dressing in heterosexual transvestism. *Acta Psychiatrica Scandinavica, 57*, 145-152.

Buhrich, N., & McConaghy, N. (1978). Parental relationships during childhood in homosexuality, transvestism and transsexualism. *Australian and New Zealand Journal of Psychiatry, 12*, 103-107.

Bullough, B., & Bullough, V. L. (1997). Are transvestites necessarily heterosexual? *Archives of Sexual Behavior, 26*, 1-12.

Bullough, V. L., Bullough, B., & Smith, R. (1983). A comparative study of male transvestites, male to female transsexuals, and male homosexuals. *Journal of Sex Research, 19*, 238-257.

Byne, W., Tobet, S., Mattiace, L. A., Lasco, M. S., Kemether, E., Edgar, M. A., et al. (2001). The interstitial nuclei of the human anterior hypothalamus: An investigation of variation with sex, sexual orientation, and HIV status. *Hormones and Behavior, 40*, 86-92.

Chivers, M. L., & Bailey, J. M. (2000). Sexual orientation of female-to-male transsexuals: A comparison of homosexual and nonhomosexual types. *Archives of Sexual Behavior, 29*, 259-278.

Chung, W. C., De Vries, G. J., & Swaab, D. F. (2002). Sexual differentiation of the bed nucleus of the stria terminalis in humans may extend into adulthood. *Journal of Neuroscience, 22*, 1027-1033.

Cohen-Kettenis, P. T., & Arrindell, W. A. (1990). Perceived parental rearing style, parental divorce and transsexualism: A controlled study. *Psychological Medicine, 20*, 613-620.

Cohen-Kettenis, P. T., van Goozen, S. H. M., Doorn, C. D., & Gooren, L. J. (1998). Cognitive ability and cerebral lateralisation in transsexuals. *Psychoneuroendocrinology, 23*, 631-641.

Collaer, M. L., Reimers, S., & Manning, J. T. (2007). Visuospatial performance on an internet line judgment task and potential hormonal sarkers: Sex, sexual orientation, and 2D:4D. *Archives of Sexual Behavior, 36*, 177-192.

Cosentino, C. E., Meyer-Bahlburg, H. F. L., Alpert, J. L., & Gaines, R. (1993). Cross-gender behavior and gender conflict in sexually abused girls. *Journal of the American Academy of Child and Adolescent Psychiatry, 32*, 940-947.

Croughan, J. L., Saghir, M., Cohen, R., & Robins, E. (1981). A comparison of treated and untreated male cross-dressers. *Archives of Sexual Behavior, 10*, 515-528.

Dessens, A. B., Cohen-Kettenis, P. T., Mellenbergh, G. J., Poll, N. V. D., Koppe, J. G., & Boer, K. (1999). Prenatal exposure to anticonvulsants and psychosexual development. *Archives of Sexual Behavior, 28*, 31-44.

Devor, H. (1994). Transsexualism, dissociation, and child abuse: An initial discussion based on nonclinical data. *Journal of Psychology and Human Sexuality, 6*, 49–72.

Diamond, M., & Hawk, S. T. (2004). *Concordance for gender identity among monozygitic and dizygotic twin pairs* Research paper for American Psychological Association. Honolulu, HI: University of Hawaii.

Docter, R. F. (1988). *Transvestites and transsexuals: Toward a theory of cross-gender behaviour*. New York: Plenum Press.

Freund, K. (1985). Cross-gender identity in a broader context. In B. W. Steiner (Ed.), *Gender dysphoria: Development, research, management* (pp. 261-324). New York: Plenum Press.

Freund, K., & Blanchard, R. (1983). Is the distant relationship of fathers and homosexual sons related to the sons' erotic preference for male partners, or to the sons' atypical gender identity, or to both. *Journal of Homosexuality, 9*, 7–25.

Freund, K., & Blanchard, R. (1993). Erotic target location errors in male gender dysphorics, paedophiles, and fetishists. *British Journal of Psychiatry, 162*, 558-563.

Freund, K., Langevin, R., Zajac, Y., Steiner, B. W., & Zajac, A. (1974). Parent-child relations in transsexual and non-transsexual homosexual males. *British Journal of Psychiatry, 124*, 22-23.

Freund, K., Steiner, B. W., & Chan, S. (1982). Two types of cross-gender identity. *Archives of Sexual Behavior, 11*, 49-63.

Futterweit, W., Weiss, R. A., & Fagerstrom, R. M. (1986). Endocrine evaluation of forty female-to-male transsexuals: Increased frequency of polycystic ovarian disease in female transsexualism. *Archives of Sexual Behavior, 15*, 69-78.

Garcia-Falgueras, A., & Swaab, D. F. (2008). A sex difference in the hypothalamic uncinate nucleus: relationship to gender identity. *Brain, 131*, 3132-3146.

Garden, G. M., & Rothery, D. J. (1992). A female monozygotic twin pair discordant for transsexualism: Some theoretical implications. *British Journal of Psychiatry, 161*.

Gehring, D., & Knudson, G. (2005). Prevalence of childhood trauma in a clinical population of transsexual people. *International Journal of Transgenderism, 8*, 23-30.

Gómez-Gil, E., Esteva , I., Almaraz, M. C., Pasaro, E., Segovia, S., & Guillamon, A. (in press). Familiality of gender identity disorder in non-twin siblings. *Archives of Sexual Behavior*.

Green, R. (2000a). Birth order and ratio of brothers to sisters in transsexuals. *Psychological Medicine, 30*, 789-795.

Green, R. (2000b). Family coocurrence of "gender dysphoria": Ten siblings or parent-child pairs. *Archives of Sexual Behavior, 29*, 499-507.

Green, R., & Keverne, E. B. (2000). The disparate maternal aunt-uncle ratio in male transsexuals: An explanation invoking genomic imprinting. *Journal of Theoretical Biology, 202*, 55-63.

Green, R., & Stoller, R. J. (1971). Two monozygotic identical twin pairs discordant for gender identity. *Archives of Sexual Behavior, 1*, 321-327.

Green, R., & Young, R. (2000). Fingerprint asymmetry in male and female transsexuals. *Personality and Individual Differences, 29*, 933-942.

Green, R., & Young, R. (2001). Hand preference, sexual preference, and transsexualism. *Archives of Sexual Behavior, 30*, 565-574.

Grossman, A. H., D'Augelli, A. R., & Salter, N. P. (2006). Male-to-female transgender youth: Gender expression milestones, gender atypicality, victimization, and parents' responses. *Journal of GLBT Family Studies, 2*, 71-92.

Haraldsen, I. R., Egeland, T., Haug, E., Finset, A., & Opjordsmoen, S. (2005). Cross-sex hormone treatment does not change sex-sensitive cognitive performance in gender identity disorder patients. *Psychiatry Research, 137*, 161-174.

Haraldsen, I. R., Opjordsmoen, S., Egeland, T., & Finset, A. (2003). Sex-sensitive cognitive performance in untreated patients with early onset gender identity disorder. *Psychoneuroendocrinology, 28*, 906-915.

Hare, L., Bernard, P., Sánchez, F. J., Baird, P. N., Vilain, E., Kennedy, T., et al. (2009). Androgen receptor repeat length polymorphism associated with male-to-female transsexualism. *Biological Psychiatry, 65*, 93-96.

Helleday, J., Siwers, B., Ritzen, E. M., & Hugdahl, K. (1994). Normal lateralization for handedness and ear advantage in a verbal dichotic listening task in women with congenital adrenal hyperplasia (CAH). *Neuropsychologia, 32*, 875-880.

Hengstschläger, M., van Trotsenburg, M., Repa, C., Marton, E., Huber, J. C., & Bernaschek, G. (2003). Sex chromosome aberrations and transsexualism. *Fertility and Sterility, 79*, 639-640.

Henningsson, S., Westberg, L., Nilsson, S., Lundström, B., Ekselius, L., Bodlund, O., et al. (2005). Sex steroid-related genes and male-to-female transsexualism. *Psychoneuroendocrinology, 30*, 657-664.

Hepp, U., Milos, G., & Braun-Scharm, H. (2004). Gender identity disorder and anorexia nervosa in male monozygotic twins. *International Journal of Eating Disorders, 35*, 239-243.

Herman-Jeglinska, A., Dulko, S., & Grabowska, A. M. (1997). Transsexuality and adextrality: Do they share a common origin? In L. Ellis & L. Ebertz (Eds.), *Sexual orientation: Toward a biological understanding*. Westport, CT: Praeger.

Hogan-Finlay, M. (1995). *Development of the cross gender lifestyle and comparison of cross gendered men with heterosexual controls.* Unpublished Ph.D. thesis, Carleton University, Ottawa, Canada.

Hore, B. D., Nicolle, F. V., & Calnan, J. S. (1973). Male transsexualism: Two cases in a single family. *Archives of Sexual Behavior, 2*, 317-321.

Hulshoff Pol, E. H., Cohen-Kettenis, P. T., Van Haren, N. E. M., Peper, J. S., Brans, R. G. H., Cahn, W., et al. (2006). Changing your sex changes your brain: Influences of testosterone and estrogen on adult human brain structure. *European Journal of Endocrinology, 155(Suppl. 1)*, S107-S114.

Hyde, C. E., & Kenna, J. C. (1977). A male MZ twin pair, concordant for transsexualism, discordant for schizophrenia. *Acta Psychiatrica Scandinavica, 56*, 265-275.

Joyce, P. R., & Ding, L. (1985). Transsexual sisters. *Australian and New Zealand Journal of Psychiatry, 19*, 188-189.

Kasantikul, D., & Roback, H. (1978). Sex role identification [Letter to the editor]. *American Journal of Psychiatry, 135*, 1426.

Kaufman, J., Plotsky, P. M., Nemeroff, C. B., & Charney, D. S. (2000). Effects of early adverse experiences on brain structure and function: Clinical implications. *Biological Psychiatry, 48*, 778-790.

Kelso, W. M., Nicholls, M. E., Warne, G. L., & Zacharin, M. (2000). Cerebral lateralization and cognitive functioning in patients with congenital adrenal hyperplasia. *Neuropsychology, 14*, 370-378.

Kendler, K. S., Thornton, L. M., Gilman, S. E., & Kessler, R. C. (2000). Sexual orientation in a US national sample of twin and nontwin sibling pairs. *American Journal of Psychiatry, 157*, 1843-1846.

Kersting, A., Reutemann, M., Gast, U., Ohrmann, P., Suslow, T., Michael, N., et al. (2003). Dissociative disorders and traumatic childhood experiences in transsexuals. *Journal of Nervous and Mental Disease, 191*, 182–189.

Knoblauch, H., Busjahn, A., & Wegener, B. (2007). Monozygotic twins concordant for female-to-male transsexualism: A case report. *Archives of Sexual Behavior, 36*, 135-137.

Kraemer, B., Noll, T., Delsignore, A., Milos, G., Schnyder, U., & Hepp, U. (2009). Finger length ratio (2D:4D) in adults with gender identity disorder. *Archives of Sexual Behavior, 38*, 359-363.

Krueger, D. W. (1978). Symptom passing in a transvestite father and three sons. *American Journal of Psychiatry, 135*, 739-742.

Kruijver, F. P., Zhou, J. N., Pool, C. W., Hofman, M. A., Gooren, L. J., & Swaab, D. F. (2000). Male-to-female transsexuals have female neuron numbers in a limbic nucleus. *Journal of Clinical Endocrinology and Metabolism, 85*, 2034-2041.

La Torre, R. A., Gossmann, I., & Piper, W. E. (1976). Cognitive style, hemispheric specialization, and tested abilities of transsexuals and nontranssexuals. *Perceptual and Motor Skills, 43*, 719-722.

Lalumiere, M. L., Blanchard, R., & Zucker, K. J. (2000). Sexual orientation and handedness in men and women: A meta-analysis. *Psychological Bulletin, 126*, 575-592.

Långstrom, N., & Zucker, K. J. (2005). Transvestic fetishism in the general population: Prevalence and correlates. *Journal of Sex and Marital Therapy, 31*, 87-95.

Lawrence, A. A. (2007). A critique of the brain-sex theory of transsexualism. Retrieved 17 April 2007, from http://www.annelawrence.com/brain-sex_critique.html

Lawrence, A. A. (2009). Erotic target location errors: An underappreciated paraphilic dimension. *Journal of Sex Research, 46*, 194-215.

LeVay, S. (1991). A difference in hypothalamic structure between heterosexual and homosexual men. *Science, 253*, 1034.

Liakos, A. (1967). Familial transvestism. *British Journal of Psychiatry, 113*, 49-51.

Lothstein, L. M. (1983). *Female-to-male transsexualism: Historical, clinical, and theoretical issues*. Boston: Routledge & Kegan Paul.

Luders, E., Sánchez, F. J., Gaser, C., Toga, A. W., Narr, K. L., Hamilton, L. S., et al. (2009). Regional gray matter variation in male-to-female transsexualism. *Neuroimage, 46*, 904-907.

Manning, J. T., Barley, L., Walton, J., Lewis-Jones, D. I., Trivers, R. L., Singh, D., et al. (2000). The 2nd: 4th digit ratio, sexual dimorphism, population differences, and reproductive success: Evidence for sexually antagonistic genes. *Evolution and Human Behavior, 21*, 163–183.

Manning, J. T., Churchill, A. J. G., & Peters, M. (2007). The effects of sex, ethnicity, and sexual orientation on self-measured digit ratio (2D:4D). *Archives of Sexual Behavior, 36*, 223-233.

Manning, J. T., Scutt, D., Wilson, J., & Lewis-Jones, D. I. (1998). The ratio of 2nd to 4th digit length: A predictor of sperm numbers and concentrations of testosterone, luteinizing hormone and oestrogen. *Human Reproduction, 13*, 3000-3004.

Medland, S. E., Duffy, D. L., Wright, M. J., Geffen, G. M., Hay, D. A., Levy, F., et al. (2009). Genetic influences on handedness: Data from 25,732 Australian and Dutch twin families. *Neuropsychologia, 47*, 330-337.

Medland, S. E., Dully, D. L., Spurdle, A. B., Wright, M. J., Geffen, G. M., Montgomery, G. W., et al. (2005). Opposite effects of androgen receptor CAG repeat length on increased risk of left-handedness in males and females. *Behavior Genetics, 35*, 735-744.

Menaged, M. (2004). *The incongruence of sex and gender in the male transsexual's performance on the mental rotation test.* Unpublished Ph.D. thesis, Adelphi University, Garden City, NY, USA.

Meyer-Bahlburg, H. F. L., Gruen, R. S., New, M. I., Bell, J. J., Morishima, A., Shimshi, M., et al. (1996). Gender change from female to male in classical congenital adrenal hyperplasia. *Hormones and Behavior, 30*, 319-332.

Mueller, A., Gooren, L. J., Naton-Schötz, S., Cupisti, S., Beckmann, M. W., & Dittrich, R. (2008). Prevalence of polycystic ovary syndrome and hyperandrogenemia in female-to-male transsexuals. *Journal of Clinical Endocrinology and Metabolism, 93*, 1408-1411.

Nass, R., Baker, S., Speiser, P., Virdis, R., Balsamo, A., Cacciari, E., et al. (1987). Hormones and handedness: Left-hand bias in female congenital adrenal hyperplasia patients. *Neurology, 37*, 711-715.

Nelson, R. J. (2005). *An introduction to behavioral endocrinology* (3rd ed.). Sunderland, MA: Sinauer Associates

Newcomb, M. D. (1985). The role of perceived relative parent personality in the development of heterosexuals, homosexuals, and transvestites. *Archives of Sexual Behavior, 14*, 147-164.

Orlebeke, J. F., Boomsma, D. I., Gooren, L. J., & Verschoor, A. M. (1992). Elevated sinistrality in transsexuals. *Neuropsychology, 6*, 351-355.

Parker, G., & Barr, R. (1982). Parental representations of transsexuals. *Archives of Sexual Behavior, 11*, 221-230.

Pauly, I. B. (1974). Female transsexualism: Part I. *Archives of Sexual Behavior, 3*, 487-507.

Peters, M., Manning, J. T., & Reimers, S. (2007). The effects of sex, sexual orientation, and digit ratio (2D: 4D) on mental rotation performance. *Archives of Sexual Behavior, 36*, 251-260.

Prince, V., & Bentler, P. M. (1972). Survey of 504 cases of transvestism. *Psychological Reports, Vol. 31*, 903-917.

Putz, D. A., Gaulin, S. J. C., Sporter, R. J., & McBurney, D. H. (2004). Sex hormones and finger length: What does 2D:4D indicate? *Evolution and Human Behavior, 25*, 182-199.

Randell, J. B. (1971). Indications for sex reassignment surgery. *Archives of Sexual Behavior, 1*, 153-161.

Rotterdam ESHRE/ASRM-Sponsored PCOS Consensus Workshop Group. (2004). Revised 2003 consensus on diagnostic criteria and long-term health risks related to polycystic ovary syndrome. *Fertility and Sterility, 81*, 19-25.

Sabalis, R. F., Frances, A., Appenzeller, S. N., & Moseley, W. B. (1974). The three sisters: Transsexual male siblings. *American Journal of Psychiatry, 131*, 907-909.

Sadeghi, M., & Fakhrai, A. (2000). Transsexualism in female monozygotic twins: A case report. *Australian and New Zealand Journal of Psychiatry, 34*, 862-864.

Schiltz, K., Witzel, J., Northoff, G., Zierhut, K., Gubka, U., Fellmann, H., et al. (2007). Brain pathology in pedophilic offenders: Evidence of volume reduction in the right amygdala and related diencephalic structures. *Archives of General Psychiatry, 64*, 737-746.

Schneider, H. J., Pickel, J., & Stalla, G. K. (2006). Typical female 2nd–4th finger length (2D:4D) ratios in male-to-female transsexuals—possible implications for prenatal androgen exposure. *Psychoneuroendocrinology, 31*, 265-269.

Schott, R. L. (1995). The childhood and family dynamics of transvestites. *Archives of Sexual Behavior, 24*, 309-327.

Segal, N. L. (2006). Two monozygotic twin pairs discordant for female-to-male transsexualism. *Archives of Sexual Behavior, 35*, 347-358.

Slabbekoorn, D., van Goozen, S. H. M., Sanders, G., Gooren, L. J. G., & Cohen-Kettenis, P. T. (2000). The dermatoglyphic characteristics of transsexuals: Is there evidence for an organizing effect of sex hormones? *Psychoneuroendocrinology, 25*, 365-375.

Spinder, T., Spijkstra, J. J., Gooren, L. J. G., & Burger, C. W. (1989). Pulsatile luteinizing hormone release and ovarian steroid levels in female-to-male transsexuals compared to heterosexual women. *Psychoneuroendocrinology, 14*, 97-102.

Stevens, M., Golombok, S., Beveridge, M., & ALSPAC Study Team. (2002). Does father absence influence children's gender development? Findings from a general population study of preschool children. *Parenting: Science and Practice, 2*, 47-60.

Stevenson, M. R., & Black, K. N. (1988). Parental absence and sex role development: A meta-analysis. *Child Development, 49*, 227-232.

Stoller, R. J., & Baker, H. J. (1973). Two male transsexuals in one family. *Archives of Sexual Behavior, 2*, 323-328.

Talamini, J. T. (1982). *Boys will be girls: The hidden world of the heterosexual male transvestite*. Washington, DC: University Press.

Teicher, M. H., Tomoda, A., & Andersen, S. L. (2006). Neurobiological consequences of early stress and childhood maltreatment: Are results from human and animal studies comparable? *Annals of the New York Academy of Sciences, 1071*, 313-323.

Tsoi, W. F. (1988). The prevalence of transsexualism in Singapore. *Acta Psychiatrica Scandinavica, 78*, 501-504.

Tsoi, W. F., Kok, L. P., & Long, F. Y. (1977). Male transsexualism in singapore: A description of 56 cases. *British Journal of Psychiatry, 131*, 405-409.

Tsur, H., Borenstein, A., & Seidman, D. S. (1991). Transsexualism. *Lancet, 338*, 945-946.

Udry, J. R., Morris, N. M., & Kovenock, J. (1995). Androgen effects on women's gendered behaviour. *Journal of Biosocial Science, 27*, 359-368.

van Goozen, S. H. M., Slabbekoorn, D., Gooren, L. J., Sanders, G., & Cohen-Kettenis, P. T. (2002). Organizing and activating effects of sex hormones in homosexual transsexuals. *Behavioral Neuroscience, 116*, 982-988.

Veale, J. F. (2008). The prevalence of transsexualism among New Zealand passport holders. *Australian and New Zealand Journal of Psychiatry, 42*, 887-889.

Veale, J. F., Clarke, D. E., & Lomax, T. C. (2008a). Biological and psychosocial correlates of adult gender-variant identities: New findings. Manuscript submitted for publication.

Veale, J. F., Clarke, D. E., & Lomax, T. C. (2008b). The relative influence of assigned gender and prenatal androgen exposure on gender identity in intersex and related conditions. Manuscript submitted for publication.

Veale, J. F., Clarke, D. E., & Lomax, T. C. (2009). Test of the Identity-Defence Model of gender-variance development. Manuscript submitted for publication.

Vujovic, S., Popovic, S., Sbutega-Milosovec, G., Djordevic, M., & Gooren, L. (2009). Transsexualism in Serbia: A twenty-year follow-up study. *Journal of Sexual Medicine, 6*, 1018-1023.

Wallien, M. S. C., Zucker, K. J., Steensma, T. D., & Cohen-Kettenis, P. T. (2008). 2D:4D finger-length ratios in children and adults with gender identity disorder. *Hormones and Behavior, 54*, 450-454.

Watson, D., & Coren, S. (1992). Left-handedness in male-to-female transsexuals. *JAMA: Journal of the American Medical Association, 267*, 1342.

Wharton, V. W. (2007). *Gender variance and mental health: A national survey of transgender trauma history, posttraumatic stress, and disclosure in therapy.* Unpublished Master's dissertation, Smith College, Northampton, MA, United States of America.

Wisniewski, A. B., Prendeville, M. T., & Dobs, A. S. (2005). Handedness, functional cerebral hemispheric lateralization, and cognition in male-to-female transsexuals receiving cross-sex hormone treatment. *Archives of Sexual Behavior, 34*, 167-172.

Xita, N., & Tsatsoulis, A. (2006). Review: Fetal programming of polycystic ovary syndrome by androgen excess: Evidence from experimental, clinical, and genetic association studies. *Journal of Clinical Endocrinology and Metabolism, 91*, 1660-1666.

Yokota, Y., Kawamura, Y., & Kameya, Y. (2005). Callosal shapes at the midsagittal plane: MRI differences of normal males, normal females, and GID. *Proceedings of the 2005 IEEE engineering in medicine and biology 27th annual conference, Shanghai, 20*, 3055-3058.

Zhou, J. N., Hofman, M. A., Gooren, L. J. G., & Swaab, D. F. (1995). A sex difference in the human brain and its relation to transsexuality. *Nature, 378*, 68-70.

Zucker, K. J., Beaulieu, N., Bradley, S. J., Grimshaw, G. M., & Wilcox, A. (2001). Handedness in boys with gender identity disorder. *Journal of Child Psychology and Psychiatry, 42*, 767-776.

Zucker, K. J., & Blanchard, R. (1997). Transvestic fetishism: Psychopathology and theory. In D. R. Laws & W. T. O'Donohue (Eds.), *Sexual deviance: Theory, assessment, and treatment* (1st ed., pp. 253–279). New York: Guilford Press.

Zucker, K. J., & Bradley, S. J. (1995). *Gender identity disorder and psychosexual problems in children and adolescents*. New York: Guilford Press.