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A Biopsychosocial Model of Adolescent Gender Dysphoria

presented by Adam Omary

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A Biopsychosocial Model of Adolescent Gender Dysphoria

A dissertation presented

by

Adam Omary

to

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A Biopsychosocial Model of Adolescent Gender Dysphoria

Abstract

Gender dysphoria is a mental health condition characterized by clinically significant distress or impairment associated with marked gender incongruence, a feeling of discordance between one's gender identity and biological sex. Gender incongruence and dysphoria prevalence have risen substantially in recent years, particularly in adolescent females. My dissertation aims to understand the etiology of these phenomena through a historical overview of clinical gender dysphoria, case studies of gender reassignment in populations with disorders of sex development, theories of the neurobiology of sexual development, and four empirical studies examining gender incongruence across early adolescent development. My empirical chapters make use of the Adolescent Brain Cognitive Development (ABCD) Study sample, a demographically diverse longitudinal sample of 11,864 youths in the United States followed from ages 9-10 years in 2016-2018 to ages 14-15 years in 2021-2023.

Chapter 1 reports developmental trajectories of self- and parent-reported gender incongruence, gender expression, and gender identity across five timepoints. While gender expression was stable with age, gender incongruence and transgender or nonbinary (TNB) self-identification increased with age, particularly in females. At baseline, 0.5% of males and 1.1% of females identified as TNB. By Wave 5, 1.2% of males and 9.6% of females identified as TNB. Females were also more likely to report gender incongruence and elevated gender nonconformity (i.e., sex-atypical gender expression). TNB youth reported elevated gender nonconformity across

all ages, but showed increased gender incongruence only at later timepoints. Parents reported lower gender incongruence across all measures.

Chapter 2 makes use of ABCD's large sample of 1,970 same-sex twins to estimate the heritability of gender incongruence. Gender incongruence was more common in females, and concordance was higher in female twin pairs than male twin pairs. Binary probandwise concordance for TNB gender identity was 46% among monozygotic pairs compared to 13% among dizygotic pairs. ACE models revealed that genetic factors explained 47% of the variance of gender incongruence, with the remaining variance explained by non-shared environmental factors. Estimates had large standard errors, and thus high uncertainty, but suggest a large genetic contribution to gender incongruence in adolescents.

Chapter 3 examined concurrent and longitudinal cross-lagged associations between self-reported gender incongruence and a range of biopsychosocial variables known or hypothesized to be associated with gender dysphoria: internalizing symptoms, autism spectrum traits, sexual orientation, pubertal timing, body mass index, family conflict, peer victimization, cyberbullying, screen time, and number of TNB friends. All predictors of interest were significantly associated with gender incongruence except family conflict and number of TNB friends. Cross-lagged models tested competing hypotheses between the theory of rapid-onset gender dysphoria (ROGD) and minority stress theory on the directionality of these associations. Higher internalizing symptoms and screen time predicted later gender incongruence, but not vice versa, in females across all timepoints, while other associations were mixed. Longitudinal results largely favored predictions made by ROGD over minority stress theory.

Chapter 4 aimed to document predictors of longitudinal persistence and desistance of TNB gender identity within the subsample of 670 TNB adolescents, and to test whether a three-

cluster solution of latent gender incongruence classes resembled a hypothesized typology distinguishing between classical gender dysphoria, autogynephilia, and ROGD. Finite mixture regression identified three latent profiles of gender incongruence, but these appeared to describe mild, moderate, and pronounced subtypes of ROGD rather than the triune typology. Participants in the "mild" cluster were exclusively nonbinary (as opposed to transgender-identified), had a 100% longitudinal desistance rate, sex-typical gender expression, and low levels of internalizing symptoms and social stress. Conversely, participants in the "pronounced" cluster had the highest rates of transgender identification, nonheterosexual orientation, internalizing symptoms, and social stress, and the highest longitudinal persistence rates at 45%. Biopsychosocial correlates of gender incongruence largely did not predict longitudinal TNB persistence or desistance; the strongest predictors were gender identity and sexual orientation.

I conclude by proposing a new biopsychosocial model of adolescent gender dysphoria, synthesizing these findings on adolescent gender incongruence with existing clinical and neurobiological theories of gender dysphoria. This model accounts for recent rises in nonbinary identification among adolescents without gender dysphoria symptoms by introducing a subtype of gender incongruence not captured in existing typologies, characterized by identity exploration, low persistence rates, and sex-typical gender expression and psychosocial adjustment. Sociocultural and neuroendocrine pathways to gender incongruence are discussed.

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I am thankful that my studies have set me on a path connecting with brilliant researchers similarly fascinated by sex differences, including Carole Hooven, Alex Byrne, and Hollie Hammond. Through these friendships I am grateful to have been introduced to my committee members, Michael Bailey and Kenneth Zucker, whose support made this dissertation possible and whose feedback has been tremendously valuable. I am also indebted to the transgender people my work has allowed me to connect with and learn from through their lived experiences, especially my friend Ramblin. This is a sensitive topic and I appreciate those who have approached it with nuance and grace in the public forum, especially Lisa Littman, Helen Joyce, Abigail Shrier, Leor Sapir, and James Morandini, all of whom I also had the good fortune of meeting during my graduate studies.

As an interdisciplinary researcher with eclectic interests, I am fascinated by my dissertation topic because it threads so many existing topics I have dabbled in throughout my research career, including: hormones and brain development, evolution and sex differences, puberty and mental health, adolescent social development, and philosophical questions of mind, identity, and bioethics, all while addressing an ongoing public health crisis and unfortunately

politicized but important social issues. I hope that my dissertation moves scientific and public understanding of gender dysphoria forward with greater empathy and nuance.

While the path is clear in retrospect, my interests were not initially threaded together so nicely. I am grateful to my professors and peers for discussing these and my many other interests of nature and nurture. I am especially thankful to professors Birdie Shirtcliff, Jill Hooley, Rich McNally, and Omar Sultan Haque for their wisdom and encouragement. I am grateful to other minds who challenged and supported me in our department, including David Lin, Linas Nasvytis, Rachel Calcott, Daniella Rothstein, Erinn Acland, and my dear friends Pusillanimus and Pusillanima. I thank Patrick Mair, Katherine Grisanzio, and Laura Wesseldijk for statistical consulting, without which parts of my dissertation would not have been possible.

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The eternal unknown—nature, metaphorically speaking, creative and destructive, source and destination of all determinant things—is generally ascribed an affectively ambivalent feminine character (as the "mother" and eventual "devourer" of everyone and everything). The eternal known, in contrast—culture, defined territory, tyrannical and protective, predictable, disciplined and restrictive, cumulative consequence of heroic or exploratory behavior—is typically considered masculine (in contradistinction to "mother" nature). The eternal knower, finally—the process that mediates between the known and the unknown—is the knight who slays the dragon of chaos, the hero who replaces disorder and confusion with clarity and certainty, the sun-god who eternally slays the forces of darkness, and the "word" that engenders creation of the cosmos.

— Jordan Peterson, *Maps of Meaning*

Quod supplantandum, prius bene sciendum.
(What you hope to supplant, you must first know thoroughly.)

— Latin aphorism

In so far as we are intelligent beings, we cannot desire anything save that which is necessary, nor yield absolute acquiescence to anything, save to that which is true: wherefore, in so far as we have a right understanding of these things, the endeavor of the better part of ourselves is in harmony with the order of nature as a whole.

— Baruch Spinoza, *Ethics*

General Introduction

Are sex and gender the same thing? Historically and in most contexts, gender is synonymous with sex (Byrne, 2023). However, gender is an evolving and polysemous concept. Gender is sometimes used to describe continuous variation in the expression of masculinity and femininity, and other times used to describe gender identity as distinct from sex—one's "felt-sense" of gender (Turban, 2024). This latter definition of gender is central to the rare but increasingly prevalent experience of gender dysphoria.

Gender dysphoria, previously called gender identity disorder, is a mental health condition characterized by clinically significant distress or impairment associated with marked gender incongruence (American Psychiatric Association [APA], 2022). Gender incongruence has historically and colloquially been understood as gender-role incongruence: masculine females and feminine males who do not conform to stereotypical sex-typed roles (Green & Money, 1960). More recently, gender incongruence has been used to refer to transgender identity, an incongruence between one's felt-sense of gender identity and one's biological sex (Turban, 2024).

Transgender individuals often experience gender dysphoria, but may not meet diagnostic criteria without clinical distress or impairment (Ashley, 2019). Historically, gender dysphoria has been exceptionally rare, with prevalence estimated at below 14 per 100,000 males and below 3 per 100,000 females (APA, 2022; Arcelus et al., 2015). However, in the past fifteen years, rates of gender dysphoria have increased more than 30-fold in children and adolescents, particularly in adolescent females (Cass, 2024, p. 24; Zhang et al., 2020; K. Zucker, 2017). What are the drivers behind such a dramatic rise in incidence?

To understand changing rates of gender dysphoria, we must understand its etiology. Why do some people experience marked gender incongruence to such a degree that they feel trapped in the wrong body? Conversely, why do most people with atypical gender expression *not* experience gender dysphoria? With respect to both of these forms of gender diversity, the nature versus nurture question remains unsettled. To what degree are individual differences in gender expression and felt-identity the result of innate biological influences, and to what degree are they learned? Under what circumstances will gender dysphoria resolve, and under what circumstances, if any, is it advisable to change genders—is this even possible?

A Case Study of Acquired Gender Dysphoria

The strongest test-case for these questions would be to sexually reassign infants and rear them as the opposite gender. This would be highly unethical, yet the wrought history of medicine has produced precisely such an example in the case of David Reimer (Colapinto, 2000). Born in 1965, David and his identical twin Brian were both male. Both were soon circumcised, but complications in David's procedure damaged his penis beyond repair. His parents were then referred to Dr. John Money, a psychologist specializing in intersex conditions. The nature of David's injury made a vaginoplasty more feasible than a phalloplasty, and Money, a prominent social constructivist, believed that David fared better chances of growing into a psychologically and sexually healthy adult were he to be reared as a girl. If gender identity is moulded during early childhood, then alignment between one's body and socialization should matter more for well-being than the particular gender assigned.

David thus had his testes removed and a vagina constructed, and was reared as the "female" Brenda. Money had yearly psychological check-ins with Brenda, Brian, and their

parents, and initially reported success (Money, 1975). Brenda seemingly embraced her feminine identity and name, had a functional urogenital tract, dressed femininely and socialized with other girls, and was parented much differently than her brother. However, later reports revealed that David—who ultimately retransitioned to male—struggled with depression and suicidal ideation, often felt uncomfortable in his body, attempted to urinate while standing, stole his brother's toys and rejected dolls, desired to imitate his father more than his mother, had crushes on girls, and did not feel like a girl himself (Colapinto, 2000; M. Diamond & Sigmundson, 1997). Put simply, David experienced gender dysphoria.

The case of David Reimer indicates that it is indeed possible for an otherwise healthy individual to feel trapped in the wrong body. His genetically identical twin brother did not experience gender dysphoria, and David likely would not have without his forced gender reassignment. Presumably, then, both Reimer twins carried a genetically innate sense of maleness. Yet a much larger clinical population—without the same confounds of parents and physicians knowing that the birth sex of the child was different from their attempted socialization—indicates that genetics are not likely to be the sole determinant of gender identity, either.

Biological Mechanisms of Sexual and Gender Identity Development

Human fetuses are sexually indistinct until approximately six weeks into fetal development, when the sex chromosome begins to differentiate the gonads (Wilhelm et al., 2007). In the absence of a Y chromosome, whose SRY gene produces testis-determining factor proteins which activate genes involved in the development of testes, gonadal tissue by default develops into ovaries. From there on, the gonads produce sex-specific hormones—primarily

testosterone by the testes, and estradiol and progesterone by the ovaries, although all of these hormones are present in both sexes in varying quantities. The concentrations of these sex hormones, and not chromosomal differences, are the primary determinant of further sex differentiation, including of the brain and external genitalia.

Genetic males with Complete Androgen Insensitivity Syndrome (CAIS), a rare genetic condition which results in non-functional androgen receptors, possess a Y chromosome and functional testes, which produce male-typical levels of testosterone and other androgens (Oakes et al., 2008). However, their inert androgen receptors lead to female-typical brain and genital development (Hamann et al., 2014; Hines et al., 2003). Genetic males with CAIS are typically assigned female at birth and reared as girls. CAIS therefore serves as a natural experiment to examine gender identity development in individuals who are genetically male, hormonally and phenotypically female, and socialized as girls by parents and physicians with no knowledge of their condition. Children with CAIS overwhelmingly identify as girls, exhibit female-typical play preferences, and are typically not diagnosed until late adolescence (Hines et al., 2003).

Despite having no ovaries to produce female-typical levels of estrogens and progestins, testosterone produced by the testes—which remain functional, albeit undescended outside of the abdomen—is aromatized (reacting with the enzyme aromatase and converted) into estrogen. Aromatization is typical in both sexes with excess unbound testosterone and is the primary source of estrogen in males (Carreau et al., 2002). In males with CAIS, all testosterone is inert and subject to aromatization, producing female-typical estrogen levels and facilitating the development of female secondary sex characteristics, including breast growth (Tyutyusheva et al., 2021).

Therefore, adolescents with CAIS have no reason to suspect they are anything but females experiencing amenorrhea. It is typically only after seeking medical evaluation for absent menstruation that they discover they lack ovaries, and are in fact chromosomal males experiencing a rare condition affecting fewer than roughly 1 out of every 40,000 births (Oakes et al., 2008). Even once diagnosed, the vast majority of people with CAIS continue to live their lives as infertile heterosexual women (and would be unable to consider hormone replacement therapy even were they to desire to live as males, due to their complete insensitivity to androgens; Hines et al., 2003).

CAIS suggests that androgens are critical for psychological as well as physiological sex differentiation. However, it is still possible that feminine outcomes for males with CAIS are confounded by gendered socialization. Unlike David Reimer, newborns with CAIS are assigned and reared female from birth, possibly making a critical difference on parenting compared to someone with knowledge of their child's sexual reassignment. Evidence from additional disorders of sex development (a.k.a. intersex conditions) helps to further elaborate the role of sex hormones on gender identity development (Berglund et al., 2025).

Congenital Adrenal Hyperplasia (CAH) is a rare genetic condition resulting in deficiency of 21-hydroxylase, an enzyme necessary to synthesize cortisol (Speiser & White, 2003). This deficiency results in compensatory overactive adrenal glands, which as a byproduct produce excess androgens, including precursors to testosterone. Males with CAH are largely unaffected with regard to sexual development, but females undergo varying degrees of somatic masculinization as a result of high androgen levels within the male-typical range. Similarly to males with moderate Partial Androgen Insensitivity Syndrome (PAIS), females with severe CAH may develop ambiguous genitalia; and, similarly to males with severe PAIS, females with mild

CAH may develop slightly masculinized but otherwise fully functional vaginas (Lucas-Herald et al., 2016; Speiser & White, 2003). Though CAH is typically diagnosed at birth by blood tests for 17-hydroxyprogesterone, an androgen also overproduced as a result of adrenal hyperplasia, and treated with glucocorticoid supplementation, prenatal androgen exposure has lasting effects on the neurodevelopment of females with CAH (Khalifeh et al., 2022).

Despite their feminine phenotypes and socialization, girls with CAH likewise show masculinization on a number of behavioral and cognitive traits. Behaviorally, females with CAH show male-typical play preferences, higher scores on personality measures of dominance and aggression, and lower agreeableness compared to female controls (Berenbaum & Resnick, 1997; Hines & Kaufman, 1994; Mathews et al., 2009; Wong et al., 2013). These psychological and behavioral differences may be driven by altered brain structure and function in the amygdala, hippocampus, and prefrontal cortex, as a result of prenatal androgen exposure (Khalifeh et al., 2022; Omary et al., 2023).

Prenatal androgen exposure is likely to also influence sexual orientation, which is highly interrelated to both gender identity and gender dysphoria (Bailey et al., 2016; Köhler et al., 2013; K. Zucker, 2017). Genetic males with CAIS, phenotypically female due to androgen insensitivity and having been raised as girls, overwhelmingly identify as cisgender heterosexual women (Oakes et al., 2008). However, 15-60% of women with CAH identify as bisexual or lesbian, depending on subtype of CAH and degree of prenatal androgen exposure (for a review, see Daae et al., 2020), compared to around 5% of typically developing adult women (Flores & Conron, 2023). Females with severe CAH and genital masculinization may also exhibit higher rates of gender dysphoria and higher likelihood to identify as transgender men (Jones et al., 2022), though the majority of females with CAH are reared as girls and identify as women (de Jesus et

al., 2019; Dessens et al., 2005). However, evidence relating prenatal sex hormone exposure to subsequent gender dysphoria in other intersex conditions and in typically-developing populations has been mixed (Levin et al., 2023; Mazur, 2005; Köhler et al., 2013).

Experimental animal research supports the hypothesis that prenatal androgen exposure predicts sexual behavior after the onset of puberty (Adkins-Regan, 1988). In rodents, lordosis (i.e., arching of the back as an invitation for mounting) is typically exhibited by females, while mounting itself is typically initiated by males. However, female rodents exposed to male-typical levels of testosterone prenatally will not display lordosis upon sexual maturation and will instead attempt to mount other females (Phoenix et al., 1959). Conversely, males given androgen blockers will display lordosis behavior upon sexual maturation (Adkins-Regan, 1988; Hotchkiss et al., 2002). These results, combined with opposite patterns of sexual orientation in women with CAIS and CAH, indicate that sexual orientation may be explained by prenatal organizational effects on the brain activated during puberty (Schulz et al., 2009).

Genetic evidence also corroborates the theory that androgen levels during early critical windows of brain development shape later gender and sexual identity development. Gender expression, gender dysphoria, and sexual orientation have all been shown to be moderately heritable, with estimates averaging around 30% (Bailey et al., 2016; Coolidge & Stillman, 2020; Verweij et al., 2016). Genome- and exome-wide association studies have shown that thousands of genes are associated with gender and sexual diversity, each with small polygenic influence (Ganna et al., 2019; Sanders et al., 2021; Theisen et al., 2019). The few candidate gene studies which have replicated indicate that genes involved in sex steroid signaling and receptor function are associated with sexual orientation and gender dysphoria (Hamer et al., 1993; Foreman et al., 2019; Sanders et al., 2021).

A Critical Window?

Evidence from case studies such as David Reimer, clinical populations such as males with CAIS and females with CAH, experimental neuroendocrinology, and behavioral genetics research suggests that gender and sexual identity are innate and largely mediated by prenatal sex hormone exposure. Yet even identical twins average only 20-30% concordance of transgender identity, and heritability studies attribute the majority of gender and sexual identity variance to non-genetic factors (Coolidge & Stillman, 2020; L. Diamond, 2013). And astonishingly, even the infamous case of David Reimer has failed to replicate. As unreplicable as the tragic circumstances may seem, there has been at least one other documented example of male sexual reassignment following an ablated penis in early infancy, followed into adulthood (Bradley et al., 1998).

This case, referred to here as Jane, begins with seemingly identical circumstances to David's, but with an opposite outcome. Jane was born male, had her penis destroyed during an electrocautery circumcision at age 2 months, was sexually reassigned as female at age 7 months, and grew up to identify as a woman, without gender dysphoria (Bradley et al., 1998). The case details that Jane at 26 years of age had been on estrogen therapy starting at age 10, had received follow-up surgeries to improve her sexual functioning, identified as bisexual with a preference towards women, and recalled identifying as a tomboy during childhood, with masculine play preferences. Physicians reported her to be psychologically healthy and content living as a woman.

How could the same intervention on two male infants have such drastically different outcomes? The authors of Jane's case report wrote: "The most plausible explanation of our patient's differentiation of a female gender identity is that sex of rearing as a female, beginning

around age 7 months, overrode any putative influences of a normal prenatal masculine sexual biology" (Bradley et al., 1998). Why did that not apply to David Reimer as well?

One possibility, consistent with both the prenatal androgen theory of psychosexual development and the powerful effects of socialization indicated by the Jane case, is an early postnatal critical window of gender plasticity. David's accident happened at 7 months, rather than 2 months old, and he was surgically reassigned at 21 months, rather than 7 months old. Jane's parents not only had a nearly two year head start on socializing their child as a girl, but Jane had much lower postnatal androgen exposure by having her testes removed much earlier.

During the first 6 months of infancy, the hypothalamic-pituitary-gonadal (HPG) axis activates in a phenomenon known as minipuberty (Kuiri-Hänninen et al., 2014). In both sexes, sex hormones surge to their highest pre-pubertal levels during minipuberty, more than any other point in childhood. Minipuberty is thought to be critical for postnatal sex differentiation, particularly for penile and testicular growth in males and maturation of the ovarian follicles in females (Kuiri-Hänninen et al., 2014). Minipuberty may also play a critical role in infant neurodevelopment, during the first year of life when most synapse formation occurs (Hines, 2009). Experimental manipulation of androgen levels in early postnatal rodents has shown to influence structural development in the sexually dimorphic nucleus of the hypothalamus and apoptosis—programmed cell death as part of neural pruning—throughout the brain (Arnold & Gorski, 1984; de Vries & Simerly, 2002).

In males, pre-pubertal testosterone levels peak at near-pubertal levels by 3 months old, then steadily decline, with HPG activation fully declining by age 9 months (Kuiri-Hänninen et al., 2014). It is possible that, by having her testes removed at age 7 months, Jane did not receive the full amount of male-typical androgen exposure necessary to catalyze a male identity, while

David, during his first 21 months of life with functional testes, did. It is possible that psychological sex differentiation follows a dose-response relationship that is critical not just on the amount of perinatal sex hormones, but duration of cumulative exposure.

Perhaps, as in the case with genital development where a default female template becomes progressively masculinized, innately gendered psychological traits begin feminine and become progressively masculinized with early androgen exposure. If this is the case, one would expect feminine identity, feminine gender expression, and androphilia (attraction to males) not just in typically-developing females but males with CAIS; feminine identity but slightly masculinized personality and gender expression in children with low but above female-typical androgen levels, such as females with mild CAH; feminine identity but masculine gender expression and a higher likelihood of gynephilia (attraction to females) in children with high but below male-typical androgen levels, such as females with severe CAH or males who were castrated early; and masculine identity, gender expression, and gynephilia in children with male-typical androgen levels throughout the critical window, including males later reared as girls. At either extreme of this spectrum, one would expect that children reared opposite to their "brain sex" would experience gender dysphoria.

Animal evidence also favors the hormonal hypothesis, as indicated by the fact that prenatal androgen exposure in females or lack thereof in males predicts gender-atypical sexual, aggressive, and play behavior rodents (Adkins-Regan, 1988; Hotchkiss et al., 2002; Phoenix et al., 1959). However, it is debated whether animals experience a subjective sense of gender identity in the way that even young children express (Byrne, 2023; de Waal, 2022). Gender dysphoria is therefore inherently impossible to study in animals.

Further supportive of the theory that gender identity and sexual orientation are partly determined by genetics and/or prenatal hormone exposure, but problematic for the theory that a critical window for brain sex development extends postnatally, is evidence from cloacal exstrophy (K. Zucker, 1999). Cloacal exstrophy is a rare and devastating disorder affecting fewer than 1 in 200,000 births, in which the cloaca—the embryonic cavity uniting urinary, genital, and intestinal tracts—and its overlying membrane fail to develop properly, resulting in exstrophy of the bladder and bowel (Woo et al., 2010). Infants with cloacal exstrophy will be born with these organs outside of the abdomen and impaired urogenital development, requiring prompt surgical intervention, for which the survival rate is now near 100% (Woo et al., 2010).

Historically, for reasons similar to David Reimer's case, most males with cloacal exstrophy were believed to be better reassigned as girls, as the nature of their disorder left them with absent or severely malformed male genitalia (Reiner, 2004; Reiner & Gearheart, 2004; Woo et al., 2010). Unlike Reimer's and even Jane's case, because cloacal exstrophy requires immediate surgical intervention, males who were sex reassigned typically had their testes removed within the first month of life. One would therefore predict, if the pattern from Jane's case holds, that sexually reassigned males with cloacal exstrophy would adjust well to their feminine identities. Results have been mixed. One longitudinal study found that out of 14 males with cloacal exstrophy sexually reassigned as female shortly after birth, followed up at ages ranging from 8 to 21 years, only 5 (36%) were still living as female (Reiner & Gearheart, 2004). Six (43%) had transitioned to male and 3 (21%) were presenting as sexually ambiguous, two of whom experienced gender dysphoria. All subjects, including those still identifying as girls, exhibited masculine play preferences (Reiner & Gearheart, 2004).

Social Influences on Gender Identity Development

It remains unclear why some individuals with disorders of sex development develop gender dysphoria and others do not, and it remains unclear why some children with early sexual reassignment persist in that gender and others retransition to their natal sex. For that matter, it remains unclear why typically developing children differ in their gender expression and later sexual orientations. It is possible that these individual differences are attributed to more subtle hormonal and neurological differences, including not just hormone levels and brain structure but receptor sensitivity and brain connectivity. Likewise, it is possible that these individual differences are attributable to more subtle differences in the expression of the thousands of genes which have been shown to be associated with gender and sexual diversity (Ganna et al., 2019; Sanders et al., 2021; Theisen et al., 2019).

However, behavioral genetics studies of gender expression, gender dysphoria, and sexual orientation have averaged heritability estimates of around 30% (Bailey et al., 2016; Coolidge & Stillman, 2020; Verweij et al., 2016). Heritability studies attribute the majority of gender and sexual identity variance to non-genetic factors, and even identical twins average only 50% concordance of homosexual orientation and 20-30% concordance of transgender identity (Bailey & Pillard, 1991; Bailey et al., 2016; Coolidge & Stillman, 2020; L. Diamond, 2013).

What social and environmental factors influence gender identity development? Many books have been written on the subject (e.g., Eagly, 1987; Thorne, 1993), and gender expression is strongly shaped by cultural norms and expectations (Best & Williams, 2001; Wood & Eagly, 2002). These broad questions have been covered extensively elsewhere and are outside the scope of this dissertation. I am interested in a more narrow set of questions: given the vast diversity of gender expression and societal expectations, under what conditions does gender incongruence

manifest into gender dysphoria? Under what conditions is gender identity dissociable from gender expression and sexual orientation? Why do most individuals who are highly gender nonconforming and even same-sex attracted continue to identify with their natal sex, and why do others identify differently, sometimes to such a degree as to cause dysphoria?

Typology of Gender Dysphoria

Regardless of innate or environmental causes, there is an abundance of evidence that gender identity is cemented during early childhood, and that gender dysphoria is best characterized as a mismatch between one's body and subjective experience of gender. However, this essentialist view of gender identity does not explain why some children see their gender dysphoria resolve with age and without transition, or why other cohorts of people only begin to experience gender dysphoria during adolescence or adulthood.

Historically, gender dysphoria when manifest typically emerges in early childhood (Lawrence, 2010; K. Zucker, 2017). Historically, a majority of children with gender dysphoria were male, and a majority would grow up to identify as cisgender homosexual men, who would see their gender dysphoria resolve (Bailey & K. Zucker, 1995). The subset whose gender dysphoria persisted into adulthood would typically come to identify as transgender women, and were still typically androphilic or homosexual with respect to their natal sex. Likewise, a majority of transgender men (i.e., natal females) have historically been shown to be gynephilic, or homosexual with respect to their natal sex (Coleman et al., 1993).

Throughout the twentieth century, most research on transsexualism focused on male-to-female homosexuals, and homosexuality was assumed to be a central feature of the diagnosis of gender identity disorder (Blanchard et al., 1987). In the 1980s, research began to attend to a

growing number of transgender women who desired medical transition but who were gynephilic, or heterosexual with respect to their natal sex. Blanchard (1989) later introduced a typology for male gender dysphoria, distinguishing between homosexual cases which are typically child-onset and immutable, and heterosexual adolescent- or adult-onset cases termed autogynephilia. Autogynephilia is a sexual paraphilia or "erotic target location error" involving attraction to oneself as a woman, and has been hypothesized to be a causal factor behind gender dysphoria and gender transition in some transgender women (Blanchard, 1991; Freund & Blanchard, 1993; Lawrence, 2009, 2010). Likewise, it has been hypothesized that some transgender men may experience autoandrophilia, or paraphilic attraction to oneself as a man (Dickey & Stephens, 1995; Illy, 2025; Lawrence, 2010).

In recent decades, gender dysphoria has become more prevalent, particularly among children and adolescents and particularly in females (Cass, 2024; K. Zucker, 2017). A third proposed conceptualization of gender dysphoria, distinct from the essentialist conceptualization likely shaped by genetics, hormones, and early socialization (Turban, 2024), and the controversial autoerotic conceptualization (Lawrence, 2010), has been termed rapid-onset gender dysphoria (ROGD; Littman, 2018). ROGD is hypothesized to manifest primarily in adolescent females, through a combination of peer influence, changing sociocultural gender norms, mental health problems—particularly internalizing symptoms, body image issues, and autism—which are thought to interfere with gender expression and identity formation during adolescence. These problems are thought to be exacerbated by puberty, which itself has been linked to body image issues and a rise in internalizing symptoms, particularly in females (Barendse et al., 2022; Pfeifer & Allen, 2021). Social contagion of eating disorders and self-harm behavior in adolescents has been documented following similar proposed mechanisms (Allison et al., 2014; Prinstein et al.,

2010). However, limited empirical evidence exists documenting ROGD beyond demographic trends in diagnoses of gender dysphoria and accounts from parents on their children's absence of gender nonconformity in childhood (Littman, 2018).

The conceptualizations of gender dysphoria that link it intrinsically to sexual orientation in essentialist accounts, classify it as a paraphilia in autosexuality accounts, attribute it to social contagion in ROGD accounts, or otherwise frame it as a mental health disorder rather than a natural expression of human variation remain controversial and are still being debated (Byrne, 2023; Serano, 2010; Turban, 2024). Understudied in each of these debates, and necessary to resolve them, is what the specific causal mechanisms are behind each proposed manifestation of gender dysphoria, and particularly behind its recent increased incidence. Additionally understudied, even in contemporary research which has paid more attention to nonbinary gender identities, is how these individuals may differ in their experience of gender incongruence and other psychological outcomes as compared to transgender individuals (e.g., Martinez Agulleiro et al., 2024; Turban et al., 2018).

Research Questions

My dissertation proposes to address gaps in the literature concerning both the etiology of adolescent-onset gender dysphoria and gender incongruence more broadly through three studies utilizing the large, demographically diverse Adolescent Brain Cognitive Development (ABCD) Study dataset of over 11,000 youths aged 9 to 15 years in the United States, across five longitudinal waves collected between 2016 and 2023 (Barch et al., 2018; Garavan et al., 2018). Chapter 1 will characterize gender identity development across adolescence using the full suite of measurement options available in ABCD—including self- and parent-reported categorical

gender identity and continuous gender expression and incongruence—and evaluate their longitudinal trajectories, demographic differences, and parent-child concordance. Chapter 2 will estimate the heritability and twin concordance of gender incongruence using ABCD's large sample of same-sex twins. Chapter 3 will test associations between gender incongruence and a range of biopsychosocial variables, including pubertal timing, mental health, and social stressors using longitudinal data to show directional mechanisms behind the measured increase in adolescent-onset gender incongruence. Finally, Chapter 4 will examine how these biopsychosocial factors differ across transgender and nonbinary adolescents and contribute to longitudinal gender transition, persistence, and desistance likelihood, and will attempt to validate a data-driven typology of gender incongruence in adolescents. Together, these studies will inform a biopsychosocial model of how gender incongruence manifests across adolescence with potential to revise and improve competing theories and typologies of gender dysphoria.

I. Longitudinal Measurement of Adolescent Gender Identity Development

Despite decades of study, psychological research still faces challenges in conceptualizing and measuring gender (Byrne, 2023). This chapter examines adolescent gender identity development by evaluating research designs and methodologies, with a focus on psychometric properties and developmental trajectories of each measure of gender in the Adolescent Brain Cognitive Development (ABCD) Study, a large demographically diverse study of 11,864 adolescents across the United States. These include distinct categorical measures of gender identity and continuous measures of gender expression and gender incongruence, each reported separately by parent and child (Potter et al., 2022).

The ABCD Study dataset offers an opportunity to test how epidemiological trends in adolescent gender incongruence in the contemporary United States compare to recent trajectories in Europe (Cass, 2024) and historical trends in North America (K. Zucker, 2017). In addition to documenting trajectories in gender identity and gender incongruence, ABCD offers the opportunity to examine gender identity and gender expression as a trait-like disposition (e.g., Johnson et al., 2004) in relation to gender incongruence, age, sex, and other demographic characteristics. ABCD also offers an opportunity to examine parent-child concordance of gender identity, which is important given the potential for parental support in gender identification to moderate other mental health outcomes (Martinez Agulleiro et al., 2024; Olson et al., 2016). Family discordance in child gender identity—such as when a child identifies as transgender but their parents do not affirm that identity—has also been hypothesized as a feature of rapid-onset gender dysphoria, in which children may socially transition genders amongst peers without the knowledge or assent of parents (Littman, 2018).

Hypotheses

This study measures gender identity development in the ABCD Study by examining longitudinal developmental trajectories of self- and parent-reported gender identity, gender expression, and gender incongruence, across age, sex, and sociodemographics. This includes documenting the age of onset and sex ratio of transgender and nonbinary identification, which have changed in recent years and are still debated (Cass, 2024; K. Zucker, 2017; K. Zucker et al., 1997). I hypothesize that gender incongruence will increase with age and will examine whether this effect is larger in females, as has been documented in clinical samples (Cass, 2024; K. Zucker, 2017). Likewise, I hypothesize that transgender and nonbinary-identified youth will score higher on both gender incongruence and gender nonconformity (i.e., sex-atypical gender expression), compared with cisgender age- and sex-matched peers (Johnson et al., 2004; Potter et al., 2022). Additionally, I will examine the correspondence between self- and parent-reported gender measures, which is hypothesized to be low under ROGD but high in traditional accounts of gender incongruence (deMayo et al., 2025; Littman, 2018). All other analyses with regard to demographics and gender expression are exploratory.

Other researchers have examined subsets of these questions using cross-sectional or early longitudinal ABCD data (Calzo & Blashill, 2018; Ignatova et al., 2025; Martinez Agulleiro et al., 2024; Nagata et al., 2025; Potter et al., 2021). The present research builds on previous reports of gender incongruence as the first to include five timepoints of longitudinal data extending through ages 9 to 15 years, and as the first to comprehensively examine age-related changes, sex differences, and parent-child concordance in all gender-related measures in the ABCD Study.

Method

Participants

The Adolescent Brain Cognitive Development (ABCD) Study features a large longitudinal cohort of 11,864 youths aged 8-11 years at baseline in 2016-2018. Participants were recruited from 21 sites across the United States and are demographically diverse (Garavan et al., 2018). Baseline demographic characteristics are shown in Table 1. These participants will be followed annually for 10 years and complete psychological batteries and physiological measurements at each visit ("wave"). At present, five waves of data comprising baseline data plus four years of follow-up data are publicly available, following the participants through to ages 12-15 years at Wave 5. The total sample includes 49,134 observations from 11,864 unique participants across five timepoints (ages 8-15, 52% male). All data collection was approved by the institutional review boards of all participating sites (Barch et al., 2018; Garavan et al., 2018).

Table 1. Participant Demographics by Study Visit

Variable	**Categorical Variables: Frequency (%) Continuous Variables: Mean (SD)**				
Data Wave	**Wave 1** (N = 11,864)	**Wave 2** (N = 11,216)	**Wave 3** (N = 10,969)	**Wave 4** (N = 10,332)	**Wave 5** (N = 4,753)*
Age (Years)	9.91 (0.62)	10.92 (0.64)	12.03 (0.67)	12.91 (0.65)	14.08 (0.68)
Sex					
Female	5,676 (47.8%)	5,349 (47.7%)	5,217 (47.5%)	4,909 (47.5%)	2,265 (47.6%)
Male	6,188 (52.2%)	5,867 (52.3%)	5,752 (52.5%)	5,423 (52.5%)	2,488 (52.4%)
Race/Ethnicity					
White	6,172 (52.0%)	5,985 (53.3%)	5,860 (53.4%)	5,591 (54.1%)	2,681 (56.4%)
Hispanic	2,410 (20.3%)	2,221 (19.8%)	2,164 (19.7%)	2,075 (20.1%)	979 (20.6%)
Black	1,784 (15.0%)	1,597 (14.2%)	1,561 (14.2%)	1,361 (13.2%)	509 (10.7%)
Asian	253 (2.1%)	241 (2.1%)	231 (2.1%)	221 (2.1%)	110 (2.3%)
Multiracial	1,247 (10.5%)	1,175 (10.5%)	1,155 (10.5%)	1,086 (10.5%)	474 (10.0%)
Household Income					
< $50k	3,068 (28.3%)	2,824 (27.2%)	2,688 (26.9%)	2,435 (26.3%)	1,089 (25.4%)
$50-100k	3,222 (29.7%)	2,823 (27.2%)	2,487 (24.9%)	2,129 (23.0%)	896 (20.9%)
> $100k	4,558 (42.0%)	4,721 (45.5%)	4,831 (48.3%)	4,702 (50.7%)	2,298 (53.7%)
Parent Education					
< High School	419 (3.5%)	341 (3.1%)	482 (4.4%)	404 (4.0%)	183 (3.0%)
High School	1,269 (10.7%)	1,084 (9.7%)	1,272 (11.7%)	1,107 (11.0%)	466 (10.0%)
Some College	3,051 (25.8%)	2,865 (25.7%)	1,417 (13.0%)	1,300 (12.9%)	603 (12.9%)
Bachelor's	3,007 (25.4%)	2,868 (25.7%)	3,089 (28.4%)	2,932 (29.1%)	1,411 (30.2%)
Graduate	4,092 (34.6%)	3,999 (35.8%)	4,628 (42.5%)	4,337 (43.0%)	2,014 (43.1%)

*Wave 5 data are only partially complete as of ABCD Release 5.1

Measures

Sex and Gender Identity

ABCD collects participants' parent-reported sex recorded at birth (natal sex), self-reported gender identity, and parent-reported gender identity. Gender identity was initially assessed within the Kiddie Schedule for Affective Disorders and Schizophrenia (KSADS) parent and child interviews (Barch et al., 2018; Kaufman et al., 1997). The KSADS asks children "Are you transgender?" and parents "Is your child transgender?" with options "Yes/No/Maybe." Participants who responded "I don't understand the question" or "Refuse to answer" were excluded (see Dube et al., 2021 for missingness rates). Beginning at Wave 4 (ages 12+ years), children and parents are asked "What is [your/your child's] current gender identity?" with options "Boy/Girl/Another gender (e.g., nonbinary)" (Potter et al., 2022).

In all primary analyses, a binary "transgender/nonbinary" (TNB) gender variable was used, encompassing any individual who endorsed "Yes" or "Maybe" to the KSADS transgender self-report variable or endorsed a gender identity discordant with their natal sex on the ABCD Gender Survey, including nonbinary options. This is consistent with the "two-step" approach of assessing gender diversity with respect to natal sex (Dube et al., 2024; Tordoff et al., 2019). Unless otherwise mentioned, gender in subsequent modeling and discussion refers to the TNB binary variable.

Gender Expression and Incongruence

Self-reported gender incongruence was operationalized as the average of the following three items on a five-point scale:

1. How much do you feel like a boy? (1 = Not at all, 5 = Totally)
2. How much do you feel like a girl? (1 = Not at all, 5 = Totally)
3. How much have you had the wish to be a [girl/boy]? (1 = Never, 5 = Always)

(Males are asked the "girl" form of the question and females are asked the "boy" form).

Parent-reported gender incongruence (of their child) was operationalized as the average of the following three items from the Gender Identity Questionnaire (GIQ; Johnson et al., 2004):

1. He states the wish to be a girl or woman (5 = Every day, 1 = Never)
2. He states that he is a girl or woman (5 = Every day, 1 = Never)
3. He talks about not liking his sexual anatomy (private parts) (5 = Every day, 1 = Never)

(Male version of the GIQ is shown. Females are asked the same questions in "She/her/boy or man" format).

For both the self- and parent-reported measures of gender incongruence, only complete data were retained; responses missing one or two of the three items were excluded. Self-reported internal reliability (Cronbach's α) was strong in females (α = .84) and acceptable in males (α = .72). Parent-reported internal reliability was acceptable in females (α = .71) and questionable in males (α = .63). Scoring highly on these measures does not necessarily indicate a clinical diagnosis of gender dysphoria.

Additionally, parents and children each reported two items measuring gender expression on a seven-point scale:

1. How do you think other people would describe [you/your child]? (1 = Very masculine, 4 = Equally feminine and masculine, 7 = Very feminine)

2. [How would you describe yourself?/How do you think your child would describe themselves?] (1 = Very masculine, 4 = Equally feminine and masculine, 7 = Very feminine)

(Parents are asked the "your child" form of the questions and children are asked the "yourself" form).

Parent- and child-reported gender expression were operationalized as the average of the two corresponding parent- or child-reported items; responses missing one item were excluded. Self-reported gender expression had excellent reliability in both females ($\alpha = .93$) and males ($\alpha = .91$). Parent-reported gender expression also had excellent reliability in both females ($\alpha = .92$) and males ($\alpha = .90$).

Missing Data

Out of 11,868 unique participants, 4 were missing sex at birth and excluded. The total sample was thus 11,864 youths at baseline. After attrition, the returning sample size was 11,216 at Wave 2, 10,969 at Wave 3, and 10,332 at Wave 4. As of ABCD Release 5.1, data from Wave 5 is only available for 4,753 participants, though the total retained sample size is expected to be around 10,000 by the time of complete data release. The present study therefore includes 49,134 observations from 11,864 unique participants across five timepoints. Because some participants skipped visits, the number of participants with two, three, four, or five waves of data do not equal the sample sizes at each wave. A total of 11,522 participants had data from at least two waves, 11,066 had data from at least three waves, 10,121 had data from at least four waves, and 4,561 had data from all five waves. Exactly 342 had one wave of data, 456 had exactly two, 945 had exactly three, and 5,560 had exactly four, from any combination of waves.

The self-reported gender incongruence measure was introduced at Wave 2, and was at least partially completed at at least one wave by 11,510 out of 11,522 possible respondents (99.9%), for a total of 37,088 complete or partially complete responses out of 37,270 possible responses (99.5%). A total of 397 participants (3.4%) had an incomplete response (one or two items) during at least one wave. Overall, 36,659 responses were complete (1.6% total missingness) from a total of 11,502 individuals with complete responses for at least one wave (99.8%).

The parent-reported gender incongruence measure was collected only at Waves 2 and 3, and was at least partially completed by 11,440 out of 11,522 possible respondents (99.3%), for a total of 22,031 complete or partially complete responses out of 22,185 possible responses (99.3%). A total of 3,554 unique parents had an incomplete response (one or two items) during at least one wave (30.8%). Overall, 17,633 responses were complete (20.5% total missingness) from a total of 10,434 parents with complete responses for at least one wave (90.6%).

The self- and parent-reported gender expression measures were introduced at Wave 4. Child-reported gender expression measures were at least partially completed by 9,085 out of 10,397 possible respondents (87.4%), for a total of 9,465 out of 15,085 possible responses (62.7%). Parent-reported gender expression measures were at least partially completed by 7,100 out of 10,397 possible respondents (68.3%), for a total of 7,118 out of 15,085 possible responses (47.2%). All parents and children who completed the gender expression survey answered both items; no individuals responded to only one item. Because the gender expression measure was considered complete if either parent or child provided a complete response, the number of complete observations was 9,486 (27.7% total missingness) from 9,102 unique participants (87.5%). Of these, 7,097 (74.8%) of observations were from only one reporter.

Longitudinal Gender Transition

Longitudinal trajectories of categorical gender identity will be shown in a contingency table of gender identity by longitudinal visit for each categorical gender measure and reporter. Chi-square (χ^2) tests of independence were used to test whether gender identity compositions significantly differed across study timepoints, beyond change expected by chance due to attrition or measurement error. Because sample sizes will change across timepoints due to attrition and missing data, bar graphs will plot percentages of each gender across timepoints for each measure.

Parent-Child Concordance

Concordance between parent- and child-reported categorical gender identity variables will be calculated using Cohen's kappa ($\varkappa$) tests of agreement. Agreement will be calculated across parent- and child-reports of categorical gender identity for both the KSADS and ABCD Gender Survey gender identity questions.

Statistical Analyses

Longitudinal trajectories of continuous gender measures were modeled using linear mixed-effects models. Random effects are used to account for repeated measures within participants across longitudinal waves and to capture the nested structure of the data, with subject ID nested within household ID to account for shared variance between siblings recruited from the same household.

Developmental trajectories of continuous gender measures were modeled with a three-way age-by-sex-by-gender identity interaction term, in order to assess whether age-related changes in gender expression differ between males and females, and between cisgender and TNB

youth. Age was centered at 9 years for interpretability. Additionally, race/ethnicity, household income, and highest parent level of education were included as covariates to test whether gender expression differs across demographics. Four separate models were run for each outcome: self- and parent-reported gender expression and gender incongruence.

$$\textit{Gender Measure} \sim \textit{Age*Sex*TNB} + \textit{Race} + \textit{Income} + \textit{Education} + (\textit{1} \mid \textit{Family ID/Subject ID})$$

Post hoc tests in significant covariates are assessed to interpret specific demographic contrasts of gender expression and gender incongruence using the "emmeans" package (Lenth, 2025). False discovery rate correction (FDR) was performed to account for repeated tests between predictor variables with a significance threshold of $\alpha = .05$ (Benjamini & Hochberg, 1995). All *p*-values presented in multivariate tables are FDR-corrected.

Results

Self- and Parent-Reported Gender Identity

Across all timepoints and both gender identity self-report measures, 133 (2.2%) out of 6,187 males and 537 (9.5%) out of 5,677 females identified as TNB during at least one timepoint. The total population of TNB youth was 80.1% female. Self- and parent-reported gender identity by sex across each of the five timepoints are shown in Table 2. Self-reported gender identity distributions by sex and timepoint are visualized in Figure 1. Parent-reported gender identity distributions by child's sex and timepoint are visualized in Figure 2.

Self-reported TNB identification increased over time, particularly during Waves 4-5 (ages 11-15) and particularly in females. At Wave 1 (ages 8-11), 0.5% of males and 1.1% of females identified as transgender or gender-questioning. By Wave 5 (ages 12-15), 0.9% of males

and 6.6% of females identified as transgender or gender-questioning. Self-reported transgender identity compositions significantly increased across timepoints in females ($\chi^2(8) = 257.46$, $p < .001$), but not males ($\chi^2(8) = 15.27$, $p = .054$). Similarly, self-reported gender identity, though only administered at Waves 4 and 5, increased across timepoints. At Wave 4, 0.8% of males identified as girls or nonbinary, while 3.8% of females identified as boys or nonbinary. By Wave 5, 1.2% of males identified as girls or nonbinary, while 9.6% of females identified as boys or nonbinary, with 7.8% of females identifying as nonbinary. This increase was statistically significant in females ($\chi^2(2) = 29.78$, $p < .001$), but not males ($\chi^2(2) = 0.66$, $p = .721$).

Parent-reported TNB identification also increased over time, again particularly during Waves 4–5 (ages 11–15) and in females, though to a lesser extent. At Wave 1 (ages 8–11), 1.1% of males and 1.1% of females were reported by parents as transgender or gender-questioning. By Wave 5 (ages 12–15), 1.9% of males and 5.5% of females were reported as transgender or gender-questioning. Parent-reported transgender identification showed a significant increase across timepoints in both females ($\chi^2(8) = 231.79$, $p < .001$) and males ($\chi^2(8) = 24.52$, $p = .002$). Similarly, parent-reported gender identity increased in both sexes, but more so in females. At Wave 1, 0.1% of males were reported as identifying as girls or nonbinary, while 0.2% of females were reported as boys or nonbinary. By Wave 5, 1.1% of males were reported as girls or nonbinary, while 4.9% of females were reported as boys or nonbinary, with 2.9% identifying specifically as nonbinary. Parent-reported gender identification significantly differed across timepoints in both females ($\chi^2(8) = 339.71$, $p < .001$) and males ($\chi^2(8) = 48.28$, $p < .001$).

Figure 1. Self-reported gender and transgender identity across study timepoints, by sex. Results are shown in percentage format. Axis break at 10%—proportions above this threshold are not shown to scale. A) Self-reported transgender identity in males; B) self-reported transgender identity in females; C) self-reported gender identity in males; D) self-reported gender identity in

females. Waves differ in their total number of observations: Wave 1, N = 7,163; Wave 2, N = 9,130; Wave 3, N = 10,269; Wave 4, N = 9,957; Wave 5, N = 4,641.

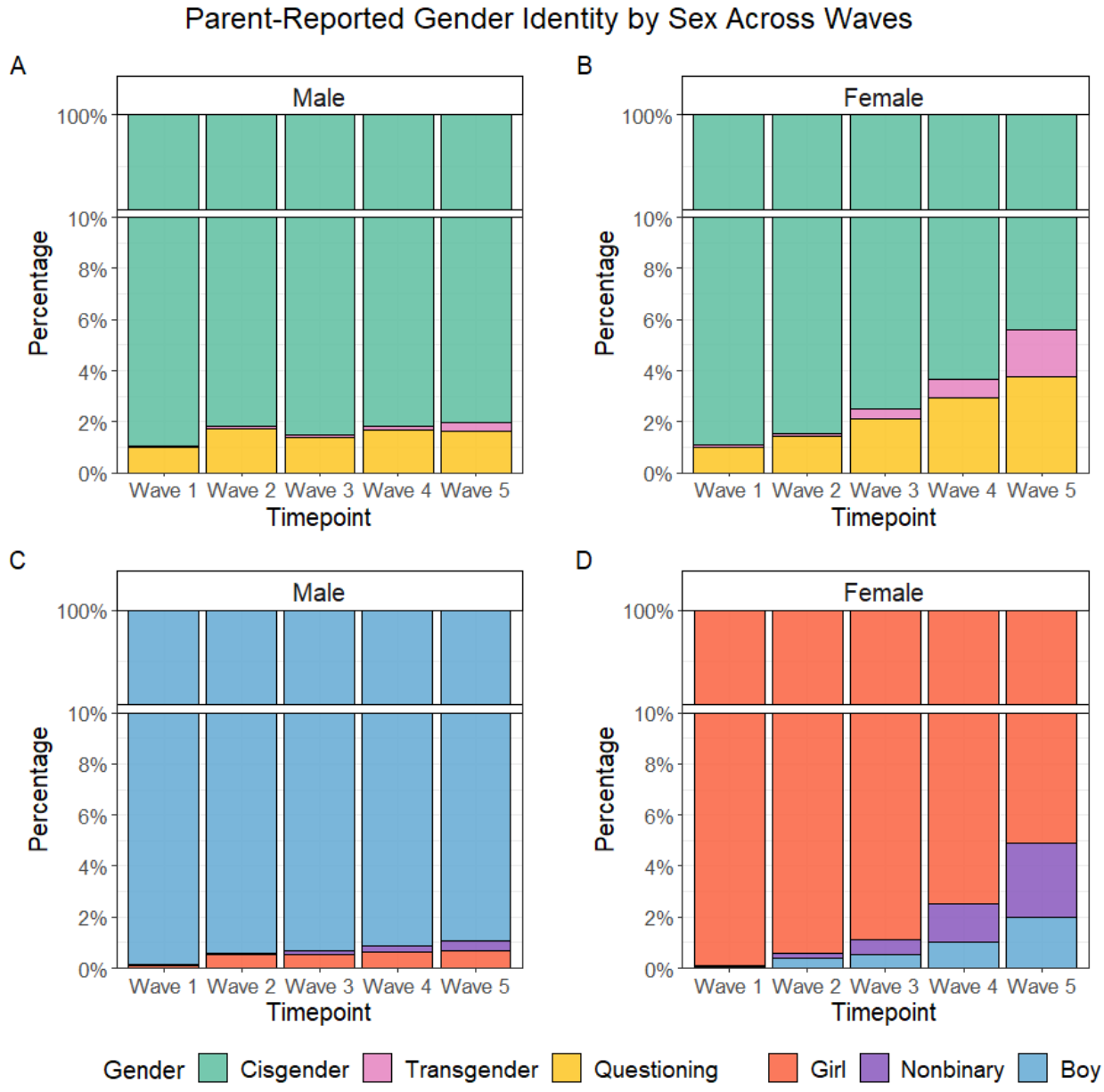


Figure 2. Parent-reported gender and transgender identity across study timepoints, by sex. Results are shown in percentage format. Axis break at 10%—proportions above this threshold are not shown to scale. A) Parent-reported transgender identity for males; B) parent-reported

transgender identity for females; C) parent-reported gender identity for males; D) parent-reported gender identity for females. Waves differ in their total number of observations: Wave 1, N = 11,802; Wave 2, N = 11,156; Wave 3, N = 10,850; Wave 4, N = 10,060; Wave 5, N = 4,649.

Table 2. Gender Identity by Study Visit by Sex and Reporter

Data Wave	Wave 1	Wave 2	Wave 3	Wave 4	Wave 5*
Male Self-Report					
Gender Identity Boy	Not Collected	Not Collected	Not Collected	5,294 (99.2%)	2,434 (98.8%)
Girl				19 (0.4%)	10 (0.4%)
Nonbinary				26 (0.5%)	19 (0.8%)
Transgender Yes	4 (0.1%)	4 (0.1%)	5 (0.1%)	14 (0.3%)	9 (0.4%)
Maybe	16 (0.4%)	22 (0.5%)	18 (0.3%)	23 (0.4%)	12 (0.5%)
No	3,728 (99.5%)	4,720 (99.5%)	5,334 (99.6%)	5,155 (99.3%)	2,408 (99.1%)
Female Self-Report					
Gender Identity Boy	Not Collected	Not Collected	Not Collected	56 (1.2%)	39 (1.8%)
Girl				4,569 (95.1%)	1,991 (90.4%)
Nonbinary				179 (3.7%)	172 (7.8%)
Transgender Yes	8 (0.2%)	12 (0.3%)	43 (0.9%)	91 (1.9%)	84 (3.8%)

Maybe	30 (0.9%)	60 (1.4%)	52 (1.1%)	88 (1.8%)	63 (2.8%)
No	3,377 (98.9%)	4,312 (98.4%)	4,817 (98.1%)	4,586 (96.2%)	2,065 (93.4%)
Parent-Report for Males					
Child's Gender Identity Boy	6,176 (99.9%)	5,818 (99.4%)	5,675 (99.3%)	5,259 (99.1%)	2,419 (98.9%)
Girl	6 (0.1%)	32 (0.5%)	32 (0.6%)	33 (0.6%)	17 (0.7%)
Nonbinary	2 (0.0%)	3 (0.1%)	7 (0.1%)	13 (0.2%)	9 (0.4%)
Transgender Child Yes	5 (0.1%)	6 (0.1%)	5 (0.1%)	8 (0.2%)	8 (0.3%)
Maybe	61 (1.0%)	101 (1.7%)	80 (1.4%)	88 (1.7%)	40 (1.6%)
No	6,079 (98.9%)	5,730 (98.2%)	5,603 (98.5%)	5,205 (98.2%)	2,395 (98.0%)
Parent-Report for Females					
Child's Gender Identity Boy	3 (0.1%)	20 (0.4%)	27 (0.5%)	48 (1.0%)	44 (2.0%)
Girl	5,662 (99.9%)	5,305 (99.4%)	5,105 (98.9%)	4,635 (97.5%)	2,096 (95.1%)
Nonbinary	4 (0.1%)	11 (0.2%)	31 (0.6%)	72 (1.5%)	64 (2.9%)
Transgender Child Yes	3 (0.1%)	6 (0.1%)	20 (0.4%)	34 (0.7%)	41 (1.8%)

Maybe	58 (1.0%)	76 (1.4%)	108 (2.1%)	140 (2.9%)	83 (3.7%)
No	5,596 (98.9%)	5,237 (98.5%)	5,034 (97.5%)	4,601 (96.4%)	2,093 (94.4%)

**Wave 5 data is only partially complete as of ABCD Release 5.1*

Parent-Child Concordance of Gender Identity

Table 3 shows concordance between self- and parent-reported gender identity by sex. On the KSADS, agreement between parent and child was modest ($\kappa = 0.26$, 95% CI [0.23-0.29]). Cohen's weighted kappa showed stronger agreement ($\kappa = 0.46$, 95% CI [0.17-0.74]), meaning that agreement was more likely for partial agreement, such as neither parent or child endorsing cisgender but one reporting transgender and the other gender-questioning. Of the 264 observations of children who identified as transgender on the KSADS (0.65% of total sample), 171 parent observations (64.8%) endorsed that their child was either transgender or gender questioning and 93 parent observations (35.2%) identified their child as cisgender.

On the ABCD Gender Survey, agreement was computed separately across sexes because the response options are inherently confounded by sex, which would otherwise inflate agreement. With this method, Cohen's kappa still showed higher agreement between parent- and child-reported gender identity than transgender identity, more so in females ($\kappa = 0.50$, 95%CI [0.45-0.54]) than males ($\kappa = 0.45$, 95%CI [0.34-0.55]). Weighted kappa did not significantly differ in either sex. Of the 357 observations of children who identified as nonbinary, 127 (35.6%) identified their child as nonbinary or transgender, while 230 (64.4%) identified their child as cisgender.

Table 3. Parent-Child Concordance of Gender Identity by Sex

Male Self-Reported Gender				
Parent-Reported Gender of Child		**Boy**	**Girl**	**Nonbinary**
	Boy	7,559 (98.6%)	10 (0.1%)	27 (0.4%)

	Girl	29 (0.4%)	16 (0.2%)	1 (0.0%)
	Nonbinary	7 (0.1%)	0 (0.0%)	14 (0.2%)
Female Self-Reported Gender				
Parent-Reported Gender of Child		Boy	Girl	Nonbinary
	Boy	47 (0.7%)	31 (0.5%)	10 (0.1%)
	Girl	29 (0.4%)	6,366 (93.4%)	203 (3.0%)
	Nonbinary	9 (0.1%)	19 (0.3%)	102 (1.5%)
Male Self-Reported Transgender				
Parent-Reported Transgender Child		**Transgender**	**Questioning**	**Cisgender**
	Transgender	20 (0.1%)	1 (0.0%)	5 (0.0%)
	Questioning	9 (0.0%)	14 (0.1%)	293 (1.4%)
	Cisgender	7 (0.0%)	76 (0.4%)	20,814 (98.0%)
Female Self-Reported Transgender				
Parent-Reported Transgender Child		**Transgender**	**Questioning**	**Cisgender**
	Transgender	75 (0.4%)	10 (0.1%)	12 (0.1%)
	Questioning	67 (0.3%)	51 (0.3%)	303 (1.6%)
	Cisgender	86 (0.4%)	226 (1.2%)	18,611 (95.7%)

Gender Expression and Incongruence Descriptives

The distributions of continuous gender measures in cisgender participants are shown in Figure 3. Means and SDs of continuous gender measures (self- and parent-reported gender expression and gender incongruence) by reporter, sex, and gender identity are shown in Table 4. Cisgender participants had bimodal gender expression scores across both reporters, with highly masculine average scores in males (self-report M = 1.89, SD = 0.99; parent-report M = 1.78, SD = 0.86) and highly feminine average scores in females (self-report M = 5.59, SD = 1.24; parent-report M = 5.81, SD = 1.07). Sex differences in gender expression were extremely large: self-report d = 3.79, parent-report d = 4.68; more androgynous scores were reported by children than parents (t(7096) = -3.736, p < .001). The vast majority of cisgender participants endorsed never experiencing gender incongruence, though self-report scores were higher than parent-report scores (t(17309) = 45.002, p < .001), particularly in females.

The distributions of continuous gender measures in TNB participants are shown in Figure 4. TNB participants had more normally distributed gender expression scores centered around androgyny (M = 4.01, SD = 1.29) with moderate sex differences (d = 0.65). Parents of TNB children reported more androgynous gender expression (M = 4.37, SD = 1.44) than parents of cisgender children (M = 3.64, SD = 2.27; t(654) = 10.105, p < .001), but scores remained more gender-typical than their children's self-reports with large sex differences (d = 1.62). While the modal score of gender incongruence in TNB participants was still “never,” most reported at least mild incongruence (M = 2.14, SD = 1.13). TNB youth reported significantly higher gender incongruence than cisgender youth (t(2247) = 41.385, p < .001). TNB females in particular reported elevated gender incongruence relative to TNB males (t(648) = 7.041, p < .001). Parent-

reported gender incongruence was higher in TNB children (M = 1.18, SD = 0.58) than in cisgender children (M = 1.02, SD = 0.13; $t(1021 = 8.642, p < .001)$, particularly for females.

Table 4. Gender Measures by Sex and Gender Identity

Mean (SD)	**Males**	**Females**	**Cisgender**	**TNB**
Self-Report				
Gender Expression	1.89 (0.99)	5.59 (1.24)	3.63 (2.20)	4.01 (1.29)
Gender Incongruence	1.09 (0.31)	1.34 (0.65)	1.15 (0.38)	2.14 (1.13)
Parent-Report				
Child's Gender Expression	1.78 (0.86)	5.81 (1.07)	3.64 (2.27)	4.37 (1.44)
Child's Gender Incongruence	1.02 (0.14)	1.02 (0.24)	1.02 (0.13)	1.18 (0.58)

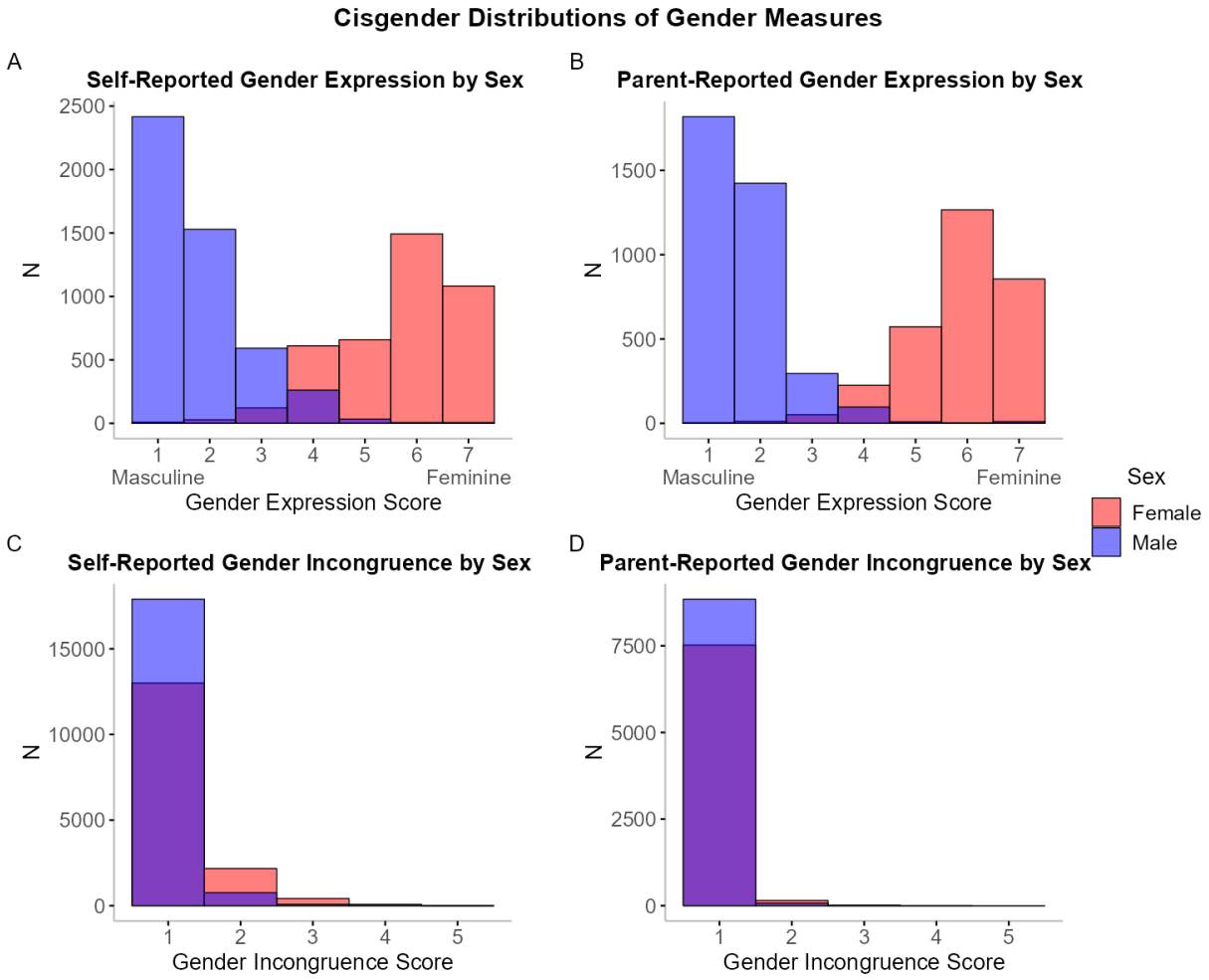


Figure 3. Distribution of gender expression and gender incongruence scores by reporter and sex among cisgender participants (N = 11,194). Self-reported gender expression (A) includes 8,847 observations from 8,847 unique individuals across Waves 4-5 (ages 11-15, 54.6% male). Parent-reported gender expression (B) includes 6,644 observations from 6,626 unique individuals across Waves 4-5 (ages 11-15, 55.0% male). Self-reported gender incongruence (C) includes 34,443 observations from 10,835 unique individuals across Waves 2-5 (ages 9-15, 54.3% male). Parent-reported gender incongruence (D) includes 16,618 observations from 9,832 unique individuals across Waves 2-3 (ages 9-14, 53.9% male).

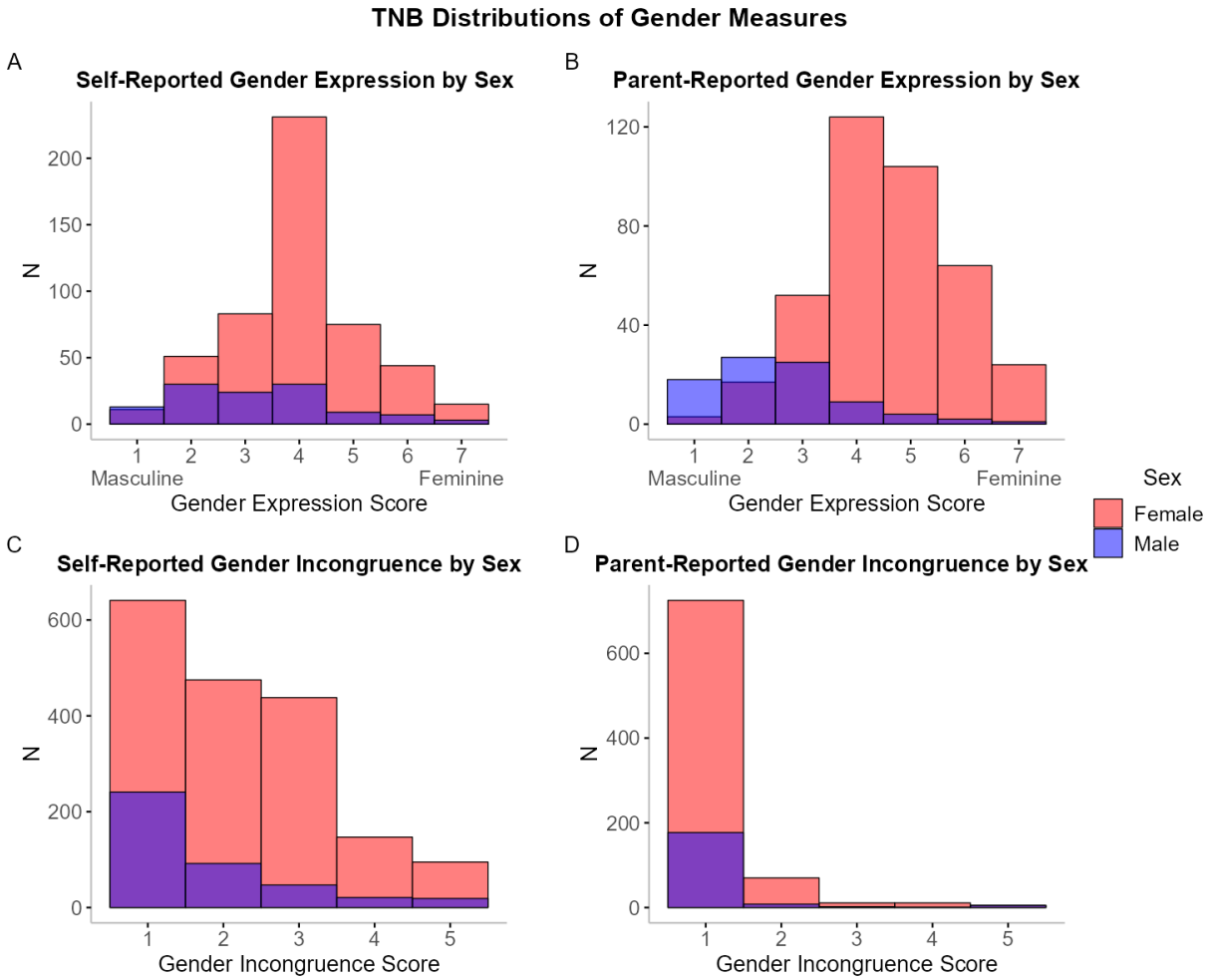


Figure 4. Distribution of gender expression and gender incongruence scores by reporter and sex among TNB participants (N = 670). Self-reported gender expression (A) includes 626 observations from 589 unique individuals across Waves 4-5 (ages 11-15, 81.0% female). Parent-reported gender expression (B) includes 474 observations from 474 unique individuals across Waves 4-5 (ages 11-15, 81.9% female). Self-reported gender incongruence (C) includes 2,216 observations from 667 unique individuals across Waves 2-5 (ages 9-13, 80.4% female). Parent-reported gender incongruence (D) includes 1,015 observations from 602 unique individuals across Waves 2-3 (ages 9-13, 80.7% female).

Gender Measure Correlations

Correlations between age and self- and parent-reported gender expression and gender incongruence are shown in Figure 5. Gender expression scores were rescaled in females so that for both sexes, lower scores indicate typical gender expression and higher scores indicate gender nonconformity.

In males, age was not significantly associated with any gender measure except parent-reported gender expression, where parent-reported gender nonconformity (i.e., sex-atypical gender expression) was slightly negatively correlated with age (r = -0.08, p = .025). In females, both self-reported (r = 0.06, p < .001) and parent-reported (r = 0.02, p < .001) gender incongruence were slightly positively associated with age, while neither self- or parent-reported gender expression were associated with age. In both sexes, gender measures were moderately correlated with each other across reporters, more so in females (range: r = 0.31-0.60, p's < .001) than males (range: 0.24-0.35, p's < .001). No correlations were available between parent-reported gender incongruence and gender expression, because these measures were administered at non-overlapping study waves.

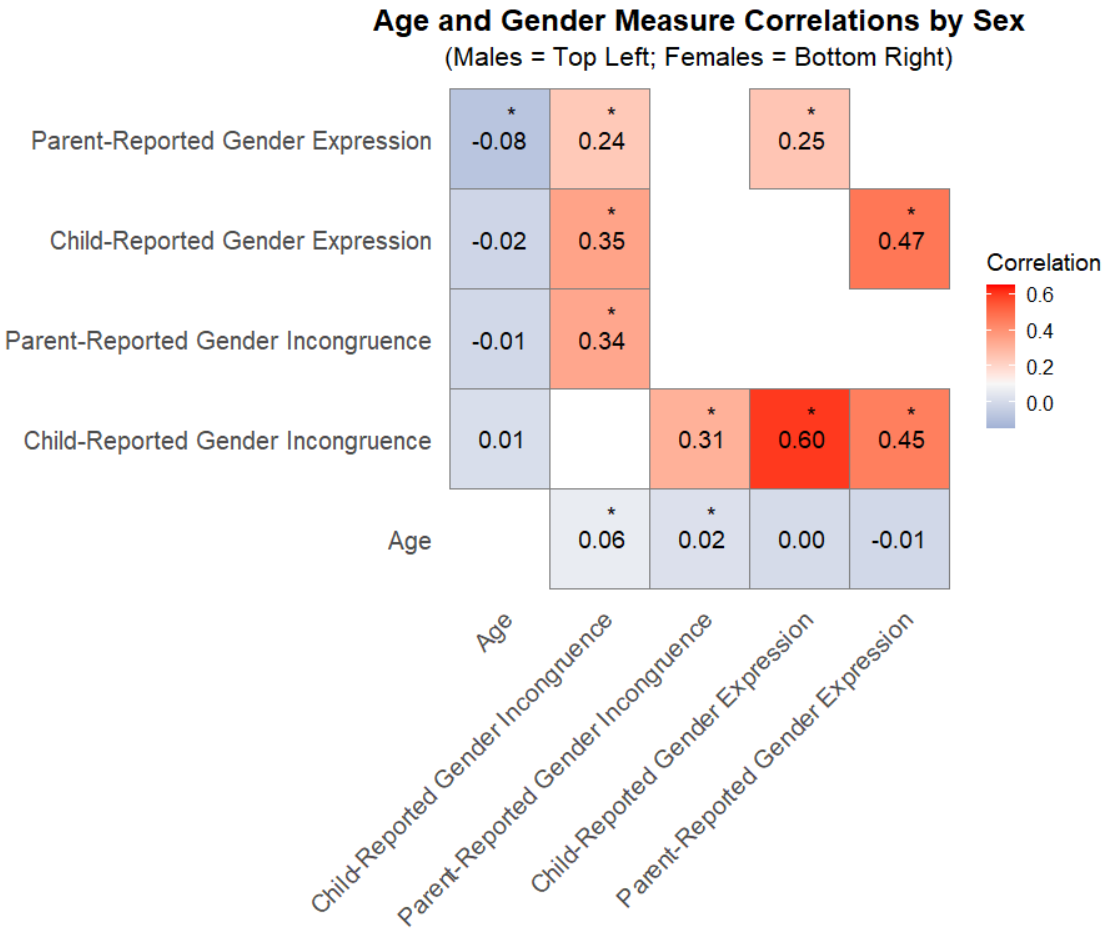


Figure 5. Age and Gender Measure Correlations by Sex. Correlations are of the full data averaged across longitudinal timepoints: child-reported gender incongruence has 36,659 observations across Waves 2-5; parent-reported gender incongruence has 17,633 observations across Waves 2-3; child-reported gender expression has 9,473 observations across Waves 4-5; parent-reported gender expression has 7,118 observations across Waves 4-5.

Longitudinal Trajectories of Gender Expression

The linear mixed-effects model estimating age-related changes in self-reported gender expression by sex and gender, after controlling for demographic covariates and making use of

the longitudinal data from Waves 4-5, is visualized in Figure 6 and summarized in Table 5. There were no significant changes in gender expression with age, regardless of sex or gender. On average, males had masculine gender expression (M = 2.61, 95% CI [2.50, 2.73]) and females had feminine gender expression (M = 4.96, 95% CI [4.89, 5.02]). No significant differences in gender expression existed between TNB and cisgender youth when averaged across sex. However, post hoc tests of the two-way sex-by-gender interaction effect revealed that TNB females were significantly more masculine than cisgender females (B = -1.620, z = -30.405, p < .001) and TNB males were significantly more feminine than cisgender males (B = 1.532, z = 14.483, p < .001). No demographic differences in self-reported gender expression were significant after FDR correction.

Table 5. Self-Reported Gender Expression Linear Mixed-Effects Model.

Term	Estimate (SE)	Test Statistic
Intercept	5.769 (0.116)	$t(7776) = 49.87, p < .001$***
TNB	-1.669 (0.283)	$t(8312) = -5.90, p < .001$***
Male	-3.800 (0.123)	$t(8008) = -30.78, p < .001$***
Age	0.002 (0.020)	$t(8132) = 0.11, p = .967$
Income < $50k	0.068 (0.036)	$t(6737) = 1.86, p = .188$
Income > $100k	0.039 (0.029)	$t(6483) = 1.35, p = .401$
High School	-0.040 (0.072)	$t(6643) = -0.56, p = .861$
Some College	-0.025 (0.072)	$t(6607) = -0.35, p = .933$
Bachelor's Degree	0.003 (0.070)	$t(6570) = 0.04, p = .967$
Graduate Degree	-0.011 (0.069)	$t(6604) = -0.16, p = .967$
Black	-0.098 (0.041)	$t(6421) = -2.41, p = .058$
Hispanic	-0.036 (0.033)	$t(6773) = -1.08, p = .561$
Asian	0.033 (0.083)	$t(7239) = 0.40, p = .933$

Multiracial	-0.055 (0.040)	$t(6229) = -1.39$, $p = .401$
TNB × Male	3.572 (0.633)	$t(8366) = 5.64$, $p < .001$***
TNB × Age	0.011 (0.062)	$t(8285) = 0.18$, $p = .967$
Male × Age	-0.027 (0.027)	$t(7889) = -0.99$, $p = .579$
TNB × Male × Age	-0.093 (0.136)	$t(8372) = -0.68$, $p = .809$

*$p < .05$, **$p < .01$, ***$p < .001$

Figure 6. Age-Related Changes in Self-Reported Gender Incongruence by Sex and Gender Identity. N = 8,386 observations from 8,071 unique participants across 2 timepoints (Waves 4-5).

TNB refers to participants identifying as transgender, nonbinary, or gender questioning at any timepoint. Cisgender participants are those whose self-reported gender aligned with their natal sex at every time point. Small points are raw data and large points are mean data within quartile year age bins, from 11.50 to 15.75 years.

The linear mixed-effects model estimating age-related changes in parent-reported gender expression by sex and gender, after controlling for demographic covariates and making use of the longitudinal data from Waves 4-5, is visualized in Figure 7 and summarized in Table 6. On average, parents of males reported highly masculine gender expression (M = 2.31, 95% CI [2.20, 2.42]) and parents of females reported highly feminine gender expression (M = 5.36, 95% CI [5.30, 5.43]). This sex difference was larger than that of self-reported gender expression. Averaging across sex, there was no significant difference in parent-reported gender expression of cisgender and TNB youths. However, post hoc tests of the two-way sex-by-gender interaction effect revealed that parents of TNB females reported significantly more masculine gender expression than parents of cisgender females (B = -1.20, z = -23.098, $p < .001$) and parents of TNB males reported significantly more feminine gender expression than parents of cisgender males (B = 1.03, z = 9.946, $p < .001$).

Post hoc tests of the significant age-by-sex and sex-by-gender interaction terms revealed that age was significantly associated with parent-reported gender expression scores only for cisgender males. Parents reported that these males' gender expression became more masculine with age (B = -0.079, 95% CI [-0.115, -0.042]). No associations between demographic variables and parent-reported gender expression were statistically significant after FDR correction.

Table 6. Parent-Reported Gender Expression Linear Mixed-Effects Model.

Term	Estimate (SE)	Test Statistic
Intercept	5.872 (0.121)	$t(6065) = 48.49, p < .001$***
TNB	-1.294 (0.296)	$t(6417) = -4.37, p < .001$***
Male	-3.714 (0.132)	$t(6174) = -28.17, p < .001$***
Age	0.018 (0.020)	$t(6249) = 0.90, p = .509$
Income < $50k	0.082 (0.036)	$t(5220) = 2.27, p = .071$
Income > $100k	0.035 (0.030)	$t(5006) = 1.19, p = .348$
High School	-0.099 (0.071)	$t(5217) = -1.38, p = .300$
Some College	-0.036 (0.071)	$t(5202) = -0.51, p = .647$
Bachelor's Degree	-0.040 (0.070)	$t(5174) = -0.57, p = .647$
Graduate Degree	-0.053 (0.068)	$t(5196) = -0.78, p = .557$
Black	0.077 (0.040)	$t(5104) = 1.93, p = .121$
Hispanic	-0.049 (0.033)	$t(5389) = -1.48, p = .277$
Asian	0.107 (0.082)	$t(5869) = 1.30, p = .319$
Multiracial	-0.084 (0.039)	$t(5037) = -2.14, p = .084$
TNB × Male	1.895 (0.646)	$t(6416) = 2.93, p = .012$*
TNB × Age	0.020 (0.063)	$t(6413) = 0.32, p = .749$
Male × Age	-0.097 (0.028)	$t(6073) = -3.51, p = .002$**
TNB × Male × Age	0.072 (0.135)	$t(6413) = 0.53, p = .647$

*$p < .05$, **$p < .01$, ***$p < .001$

Figure 7. Age-Related Changes in Parent-Reported Gender Incongruence by Sex and Gender Identity. N = 6,445 observations from 6,432 unique participants across 2 timepoints (Waves 4-5). TNB refers to participants identifying as transgender, nonbinary, or gender questioning at any timepoint. Cisgender participants are those whose self-reported gender aligned with their natal sex at every time point. Small points are raw data and large points are mean data within quartile year age bins, from 11.50 to 15.75 years.

Longitudinal Trajectories of Gender Incongruence

The linear mixed-effects model estimating age-related changes in self-reported gender incongruence by sex and gender, after controlling for demographic covariates and making use of the longitudinal data from Waves 2-5, is visualized in Figure 8 and summarized in Table 7. Because gender expression did not change with age, it was not included as a covariate. On average, TNB youth had significantly higher gender incongruence (M = 2.02, 95% CI [1.98, 2.05]) than cisgender participants (M = 1.17, 95% CI [1.16, 1.18]), and females (M = 1.74, 95% CI [1.72, 1.76]) had significantly higher gender incongruence than males (M = 1.44, 95% CI [1.41, 1.48]). Relative to middle-income children (those whose families earned $50,000-100,000 per year), those from low-income families (< $50,000 per year) had slightly higher average gender incongruence (B = 0.026, $t(26939) = 3.16$, $p = .003$) and children from high-income families (> $100,000 per year) had slightly lower average gender incongruence (B = -0.022, $t(23815) = -3.18$, $p = .003$) after controlling for age, sex, gender, and other demographic covariates. Relative to white participants, black (B = 0.028, $t(9412) = 2.46$, $p = .021$) and multiracial (B = 0.042, $t(8246) = 3.59$, $p < .001$) participants had slightly higher average gender incongruence after controlling for age, sex, gender, and other demographic covariates. No other demographic differences were significant for self-reported gender incongruence after FDR correction.

There was a significant three-way interaction between age, sex, and gender, as well as significant two-way age-by-sex, age-by-gender, and sex-by-gender interaction effects on self-reported gender incongruence (Table 7). Namely, the effects of age differed by sex, the effects of age and sex differed by gender (cisgender vs. TNB), and furthermore the effect of age differed by the intersection of sex and gender (e.g., cisgender females vs. TNB females). Post hoc tests

revealed that mean gender incongruence significantly increased with age in all groups except cisgender males (B = -0.001, 95% CI [-0.006, 0.003]). Cisgender females saw only a slight mean increase in gender incongruence with age (B = 0.009, 95% CI [-0.006, 0.003]). TNB males saw a more substantial mean increase in gender incongruence with age (B = 0.236, 95% CI [0.205, 0.267]). TNB females had the greatest mean increase in gender incongruence with age (B = 0.405, 95% CI [0.391, 0.420]).

Table 7. Self-Reported Gender Incongruence Linear Mixed-Effects Model

Term	Estimate (SE)	Test Statistic
Intercept	1.195 (0.020)	$t(23968) = 59.04$, $p < .001$***
TNB	-0.286 (0.030)	$t(32865) = -9.66$, $p < .001$***
Male	-0.137 (0.013)	$t(32948) = -10.46$, $p < .001$***
Age	0.009 (0.003)	$t(29326) = 3.49$, $p = .001$**
Income < $50k	0.026 (0.008)	$t(26939) = 3.16$, $p = .003$**
Income > $100k	-0.022 (0.007)	$t(23815) = -3.18$, $p = .003$**
High School	0.027 (0.017)	$t(25280) = 1.54$, $p = .158$
Some College	0.028 (0.017)	$t(21617) = 1.59$, $p = .156$
Bachelor's Degree	-0.002 (0.018)	$t(21003) = -0.10$, $p = .923$
Graduate Degree	0.012 (0.017)	$t(21642) = 0.72$, $p = .530$
Black	0.028 (0.011)	$t(9412) = 2.46$, $p = .021$*
Hispanic	0.010 (0.010)	$t(9210) = 1.06$, $p = .347$
Asian	-0.006 (0.024)	$t(9447) = -0.24$, $p = .858$
Multiracial	0.042 (0.012)	$t(8246) = 3.59$, $p < .001$***
TNB × Male	0.249 (0.068)	$t(32941) = 3.68$, $p < .001$***
TNB × Age	0.396 (0.008)	$t(28430) = 49.54$, $p < .001$***
Male × Age	-0.010 (0.004)	$t(28897) = -2.88$, $p = .007$**
TNB × Male × Age	-0.159 (0.018)	$t(29113) = -8.86$, $p < .001$***

*$p < .05$, **$p < .01$, ***$p < .001$

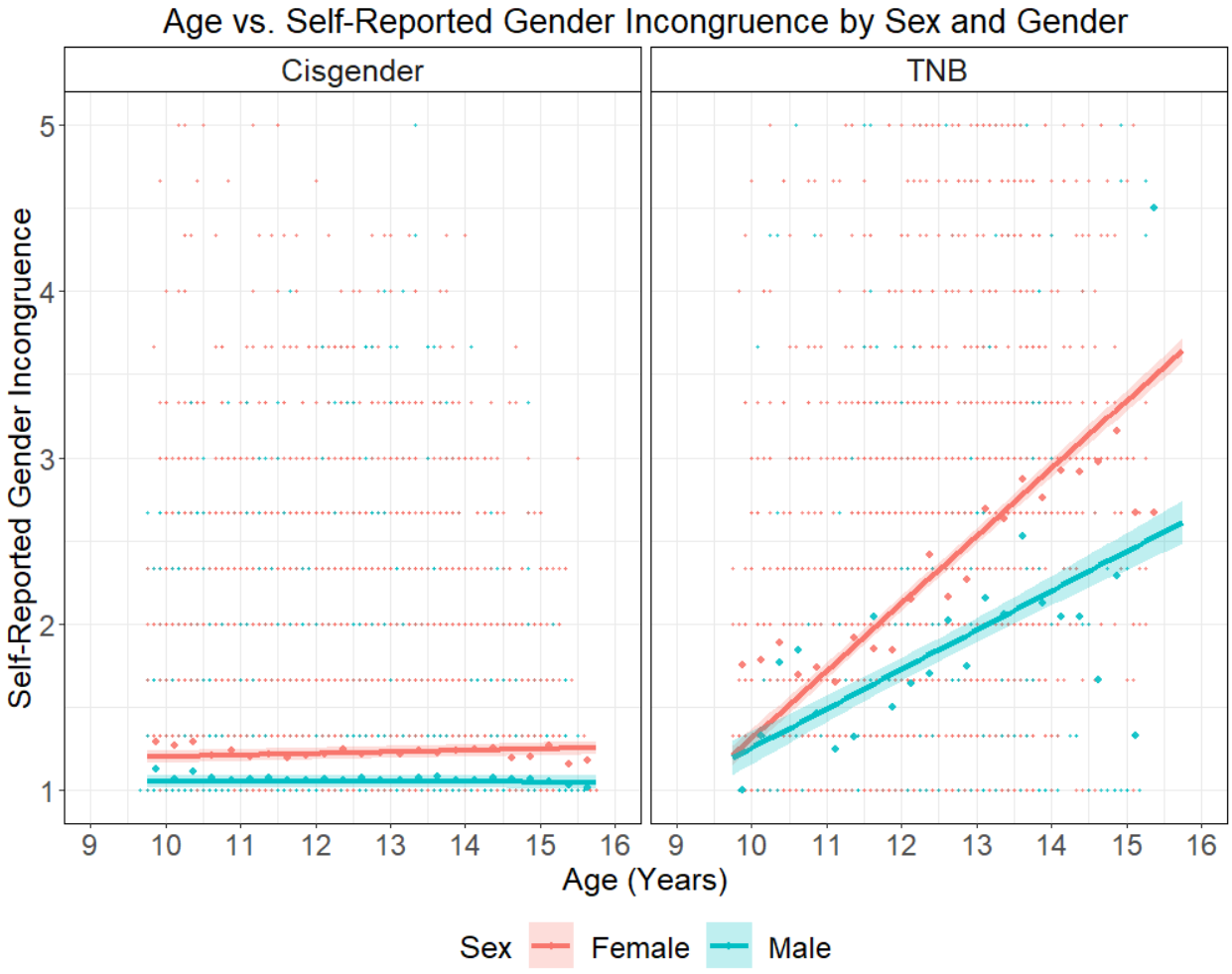


Figure 8. Age-Related Changes in Self-Reported Gender Incongruence by Sex and Gender Identity. N = 33,359 observations from 11,099 unique participants across 4 timepoints (Waves 2-5). TNB refers to participants identifying as transgender, nonbinary, or gender questioning at any timepoint. Cisgender participants are those whose self-reported gender aligned with their natal sex at every time point. Small points are raw data and large points are mean data within quartile year age bins, from 9.75 to 15.75 years.

The linear mixed-effects model estimating age-related changes in parent-reported gender incongruence by sex and gender, after controlling for demographic covariates and making use of the longitudinal data from Waves 2-3, is visualized in Figure 9 and summarized in Table 8. On average, parents of TNB youth reported significantly higher gender incongruence (M = 1.20, 95% CI [1.18, 1.22]) than parents of cisgender youth (M = 1.03, 95% CI [1.02, 1.03]). However, this difference was much smaller than the difference in self-reported gender incongruence between TNB and cisgender youth. There were no significant differences in parent-reported gender incongruence between parents of male or female children. Relative to parents of white participants, parents of multiracial children reported slightly higher average gender incongruence after controlling for age, sex, gender, and other demographic covariates (B = 0.015, $t(7843 = 2.42$, $p = .047$). No other demographic differences were significant for parent-reported gender incongruence after FDR correction.

There was a significant three-way interaction between age, sex, and gender, as well as significant two-way age-by-gender and sex-by-gender interaction effects on parent-reported gender incongruence (Table 8). Effects of age and sex differed by gender (cisgender vs. TNB), and furthermore, the effect of age differed by the intersection of sex and gender (e.g., cisgender females vs. TNB females). Post hoc tests revealed that parent-reported gender incongruence was significantly associated with age only for TNB females, for whom the parent-reported increase (B = 0.109, 95% CI [0.094, 0.125]) was about one-fourth that for self-report.

Table 8. Parent-Reported Gender Incongruence Linear Mixed-Effects Model

Term	Estimate (SE)	Test Statistic
Intercept	1.025 (0.012)	$t(14328) = 82.68$, $p < .001$***
TNB	-0.109 (0.021)	$t(16153) = -5.12$, $p < .001$***

Male	-0.011 (0.009)	$t(16096) = -1.23$, $p = .496$
Age	0.001 (0.003)	$t(15545) = 0.36$, $p = .763$
Income < $50k	0.010 (0.005)	$t(12936) = 1.91$, $p = .144$
Income > $100k	-0.002 (0.004)	$t(11843) = -0.54$, $p = .703$
High School	0.008 (0.010)	$t(13770) = 0.74$, $p = .650$
Some College	0.009 (0.010)	$t(12729) = 0.92$, $p = .645$
Bachelor's Degree	-0.004 (0.010)	$t(12684) = -0.42$, $p = .755$
Graduate Degree	0.002 (0.010)	$t(12945) = 0.16$, $p = .873$
Black	-0.004 (0.006)	$t(8472) = -0.67$, $p = .650$
Hispanic	-0.003 (0.005)	$t(8328) = -0.67$, $p = .650$
Asian	0.014 (0.012)	$t(8775) = 1.10$, $p = .540$
Multiracial	0.015 (0.006)	$t(7843) = 2.42$, $p = .047$*
TNB × Male	0.331 (0.049)	$t(15981) = 6.71$, $p < .001$***
TNB × Age	0.108 (0.008)	$t(15501) = 13.05$, $p < .001$***
Male × Age	-0.003 (0.004)	$t(15387) = -0.78$, $p = .650$
TNB × Male × Age	-0.124 (0.019)	$t(15443) = -6.63$, $p < .001$***

*$p < .05$, **$p < .01$, ***$p < .001$

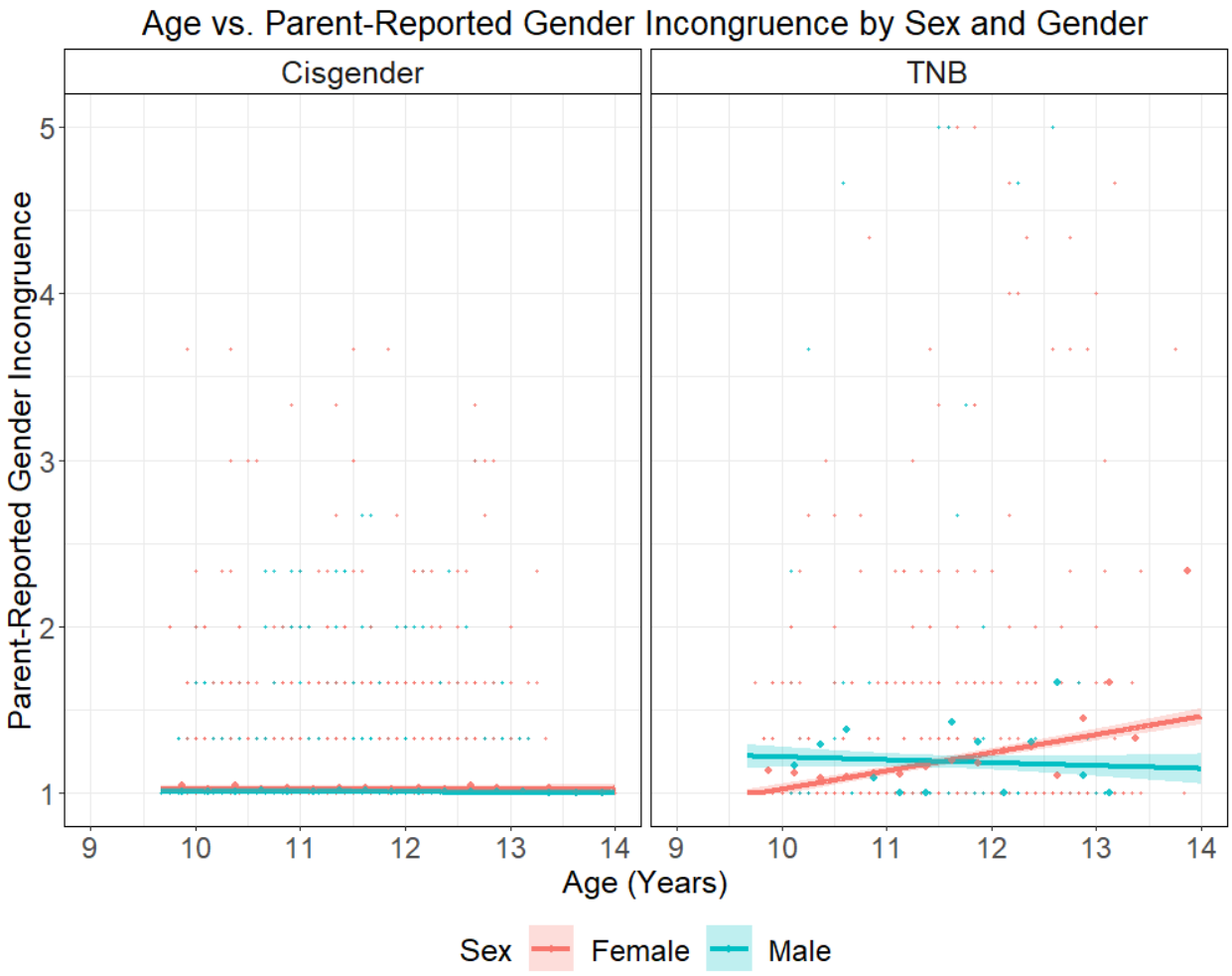


Figure 9. Age-Related Changes in Parent-Reported Gender Incongruence by Sex and Gender Identity. N = 16,234 observations from 9,866 unique participants across 2 timepoints (Waves 2-3). TNB refers to participants identifying as transgender, nonbinary, or gender questioning at any timepoint. Cisgender participants are those whose self-reported gender aligned with their natal sex at every time point.

Discussion

This study examined gender identity development using the large, longitudinal, demographically diverse Adolescent Brain Cognitive Development (ABCD) Study dataset. This

study expands on previous reports of ABCD gender measures (e.g., Calzo & Blashill, 2018; Dube et al., 2024) by including five full waves of longitudinal data, and estimating longitudinal trajectories of every gender measure rather than a subset of them: continuous gender expression and gender incongruence, categorical gender identity and transgender identity, each reported by both child and parent.

For both males and females, measures of gender incongruence increased over the study period. At baseline, 0.5% of males and 1.1% of females identified as transgender or nonbinary (TNB). By Wave 5, 1.2% of males and 9.6% of females identified as TNB. Across all waves, 133 (2.2%) out of 6,187 males and 537 (9.5%) out of 5,677 females self-identified as TNB during at least one timepoint. How do these data compare with other general population samples from the U.S.? A recent nationally representative survey of 20,103 adolescents in Grades 9 through 12 found that 4.0% of males and 7.6% of females self-identified as transgender or gender questioning (Suarez et al., 2024). In a recent smaller sample, 40 of 285 (14.0%) children recruited as cisgender identified as TNB at least once in the following five years, the majority of whom were nonbinary females (deMayo et al., 2025).

The total population of TNB youth was 80.1% female, the majority of whom "came out" at later timepoints. Females were also more likely to report gender incongruence and elevated gender nonconformity (i.e., sex-atypical gender expression). Parents also reported higher gender incongruence and gender nonconformity among their female children, although parent reports were more conservative. While 64% of parents of transgender-identifying children identified their child as transgender, nonbinary, or gender-questioning, only 36% of parents of nonbinary-identifying children identified their child as transgender, nonbinary, or gender-questioning. This difference might reflect greater visibility of gender variance in the former.

These results support the importance of studying cross-informant agreement in this area. Indeed, recent research suggesting low correspondence between parents and their adolescent children with respect to the latter's history of gender dysphoria has motivated the hypothesis of a new etiology contributing to the sharp rise in clinical referrals of adolescent females during the past 15 years (Littman, 2018; Sapir et al., 2024); however, this characterization has been challenged, arguing that recent demographic changes do not represent a new clinical phenomenon but are the byproduct of greater mental health awareness and lower levels of stigma within young generations (Ashley, 2020; Restar, 2020; Turban et al., 2023).

TNB youth in particular were, not surprisingly, at highest risk for gender incongruence and displayed strong gender nonconformity, as has been reported in smaller contemporary samples (deMayo et al., 2025). Among TNB youth, and even accounting for the overall higher prevalence in females, TNB females reported much stronger severity of gender incongruence, and this effect and sex difference increased with age. Most TNB youth did not experience gender incongruence at baseline. While youth who identified at TNB at any given timepoint had higher gender incongruence than cisgender youth at that timepoint, the majority of TNB youth reported little to no gender incongruence at ages 10-11 years. By contrast, the population of youths who identified as TNB at any timepoint did display higher gender nonconformity across all ages, according to both self-report and parent-report.

Although ABCD Study gender data did not include a clinical diagnosis of gender dysphoria, the finding that TNB identification was significantly more common among females than males is consistent with findings from clinic-referred adolescent samples since 2010, in both North America and Europe (e.g., Aitken et al., 2015; Fahrenkrug et al., 2025). Although older samples had a male-biased sex ratio, that has reversed (Zucker, 2017). Continued follow-up of

the participants in the ABCD Study will allow study of the stability of a TNB identity and whether TNB youth are at disproportionate risk for psychiatric outcomes (e.g., Ignatova et al., 2025).

It is unknown whether the high rates of TNB identification on ABCD's gender surveys, relative to historic rates in North America (e.g., Zucker, 2017), correspond to children who have socially transitioned, including using opposite-sex pronouns and potentially receiving new identity documentation or medical treatment (e.g., Olson et al., 2016, 2022). It is possible that TNB youth, perhaps especially nonbinary youth with high levels of parental discordance in particular, are experimenting with gender identity and expression in developmentally normative ways that do not bear on gender incongruence as clinically understood (deMayo et al., 2025). In either case, this represents a psychologically interesting and unprecedented social developmental change. Future research is warranted as to biopsychosocial mechanisms underlying increases in gender incongruence with age, higher rates of gender incongruence in females, parent-child discordance, and relevance to clinical gender dysphoria and mental health outcomes.

II. Heritability of Adolescent Gender Incongruence

There is growing recognition and cross-cultural evidence of an unprecedented rise in adolescent-onset gender dysphoria, particularly in females (Cass, 2024; K. Zucker, 2017). Even with this emerging empirical consensus of the changes in prevalence and demographics of adolescent gender dysphoria, its causes are unknown and debated (Ashley, 2020; Littman, 2018; Restar, 2020; Sapir et al., 2024; Turban et al., 2023). Clarifying the etiology of gender incongruence—discordance between one's biological sex and felt-gender—is therefore a necessary first step toward making sense of the recent epidemiology of gender dysphoria. A behavior genetics perspective offers particular value in estimating the proportion of variance that can be explained by genetic or environmental factors (Iacono et al., 2018).

Twin studies comparing monozygotic (MZ) and dizygotic (DZ) pairs are commonly used in order to assess the proportion of variance explained by genetic factors, or heritability, of psychological traits (e.g., Polderman et al, 2015; Smith et al., 2023; Vukasović & Bratko, 2015). Heritability is calculated by leveraging the fact that MZ twins share, on average, 100% of their genes and DZ twins share, on average, 50% of their genes, while on average not otherwise differing systematically in their environments. Therefore, greater correlations of a trait among MZ twins compared to DZ twins can be attributed to genetic differences.

Similarly, twin samples can be leveraged to distinguish between shared and non-shared environmental factors. Shared environmental factors refer both to environmental constants shared between household members, such as geography and socioeconomic status, as well as environmental factors which systematically covary between twins, but not necessarily different-aged siblings, such as school grade and classroom placement (Pinker, 2002; Smith et al., 2023). Shared environmental variance refers to residual similarity between MZ and DZ twins which

cannot be explained by shared genetic factors. Any residual differences, or the proportion of remaining variance, is attributed to non-shared environmental factors or measurement error. Importantly, not-shared environmental factors do not only include unique environmental experiences (e.g., unique conversations, diet, injury, and chance events) but also unique non-shared biological factors of the "internal environment" (e.g., unique patterns of neuronal wiring or cell death, random mutations, immune responses) which cumulatively differ across the lifespan (Pinker, 2002; Turkheimer, 2000).

Although several twin studies have examined traits related to gender incongruence, including sexual orientation and child gender nonconformity (see reviews in Bailey et al., 2016; Sanders et al., 2021), few have examined gender incongruence itself. Two small sample studies have identified higher odds ratios of gender dysphoria or transgender identity between twins and siblings compared with unrelated controls (Gómez-Gil et al., 2010; Karamanis et al., 2022), and one identified 39% concordance in gender dysphoria for MZ twins but no concordance for DZ twins (Heylens et al., 2012).

The only comparable study on the heritability of gender incongruence in youth, using a sample including 96 MZ pairs and 61 DZ pairs, estimated a 62% genetic contribution to parent-reported gender dysphoria variance among children and adolescents aged 4 to 17 years (Coolidge et al., 2002). By modern behavioral genetics research standards, this sample size is modest and underpowered, particularly for low-prevalence traits across a wide age range (Verhulst, 2017). Furthermore, parent-report measures can produce different heritability estimates compared with self-report measures (Kan et al., 2014). Finally, it is unclear whether this estimate, if accurate, is applicable to the recent manifestation of what has been dubbed "rapid-onset gender dysphoria"

(ROGD; Littman, 2018), because relevant data were collected prior to the aforementioned sharp rise in prevalence and include child-onset cases.

The Adolescent Brain Cognitive Development (ABCD) Study offers an opportunity to study adolescent development in a large, longitudinal, demographically diverse, contemporary sample (Barch et al., 2018). The ABCD Study dataset has been used to document substantial recent increases in gender incongruence during adolescence, consistent with predictions made by the ROGD hypothesis (see Chapter 1). Additionally, ABCD has strategically over-recruited 1,970 same-sex twins (985 pairs; 419 MZ pairs) in order to assess heritability of various outcomes (Iacono et al., 2018). Although the heritability of cognitive phenotypes have been comprehensively assessed in ABCD (Smith et al., 2023), the heritability of gender incongruence has not yet been examined.

This study seeks to assess the proportion of variance of adolescent gender incongruence that can be attributed to genetic, shared environmental, and unique environmental factors. I will estimate these for both binary gender incongruence (i.e., discordance between biological sex and self-reported gender identity) as well as a continuous measure of gender incongruence considered to be subclinical measures of gender dysphoria.

Method

Participants

The participants are the subset of the 1,970 same-sex twins out of the full ABCD sample of 11,864 youths. The full twin sample consists of 419 monozygotic pairs and 566 same-sex dizygotic pairs. Participants were recruited at ages 9-10 between 2016 and 2018 and are followed

annually thereafter, with data up through Wave 5 (ages 13-15) publicly available as of ABCD Release 5.1. Because gender incongruence measures of interest were assessed beginning only at Wave 2, the full data in this study consist of four timepoints, Waves 2-5 (ages 9-15). Demographics of the twin sample are shown in Table 1.

Table 1. Sample Demographics by Zygosity

Variable	**Full Sample** (N = 1,970)	**Monozygotic** (N = 838)	**Dizygotic** (N = 1,132)
Age (years)	11.88 (±1.49)	11.91 (±1.50)	11.86 (±1.49)
Sex			
Female	980 (49.7%)	410 (48.9%)	570 (50.4%)
Male	990 (50.3%)	428 (51.1%)	562 (49.6%)
Gender Identity			
Cisgender	1,900 (96.4%)	799 (95.3%)	1,101 (97.3%)
TNB	70 (3.6%)	39 (4.7%)	31 (2.7%)
Race/Ethnicity			
White	1,264 (64.2%)	534 (63.8%)	730 (64.5%)
Hispanic	223 (11.3%)	105 (12.5%)	118 (10.4%)
Black	280 (14.2%)	104 (12.4%)	176 (64.5%)
Asian	7 (0.4%)	3 (0.4%)	4 (0.4%)
Multiracial	194 (9.9%)	91 (10.9%)	103 (9.1%)
Household Income			
< $50k	323 (17.5%)	132 (17.1%)	191 (17.8%)
$50-100k	535 (29.0%)	230 (29.7%)	305 (28.5%)
> $100k	987 (53.5%)	412 (53.2%)	575 (53.7%)
Parent Education			
< High School	31 (1.6%)	12 (1.4%)	19 (1.7%)
High School	120 (6.1%)	46 (5.5%)	74 (6.5%)
Some College	499 (25.4%)	243 (29.0%)	256 (22.7%)
Bachelor's	616 (31.3%)	259 (30.9%)	257 (31.6%)
Graduate	702 (35.7%)	278 (33.2%)	424 (37.5%)

TNB = Transgender or Nonbinary

Gender Incongruence Measures

Gender incongruence is measured both continuously and categorically. Categorical measures of gender incongruence are operationalized as discordance between self-reported gender identity and parent-reported sex at birth, consistent with the "two-step" approach of assessing gender variance (Dube et al., 2024; Tordoff et al., 2019). As in Chapter 1, a binary "transgender/nonbinary" (TNB) gender variable was used, encompassing any individual who endorsed a gender identity discordant with their natal sex, including nonbinary and gender-questioning.

A continuous measure of gender incongruence was operationalized as the average of the following three items on a five-point scale:

1. How much do you feel like a boy? (1 = Not at all, 5 = Totally)
2. How much do you feel like a girl? (1 = Not at all, 5 = Totally)
3. How much have you had the wish to be a [girl/boy]* (1 = Never, 5 = Always)

*Males are asked the "girl" form of the question and females are asked the "boy" form.

This measure does not assess the dimensions of distress or impairment relevant to clinical gender dysphoria, but is conceptually related to the gender incongruence facet of gender dysphoria (American Psychiatric Association, 2022; Potter et al., 2022).

Twin Concordance for the Binary Measure

Categorical gender incongruence will be examined by zygosity. Specifically, I will calculate probandwise concordance rates to assess whether, when one twin reports a TNB gender identity, monozygotic twins are more likely to report gender incongruence as compared to dizygotic twins. Male and female concordance rates will be calculated separately. As saturated

models revealed that the TNB twin sample was underpowered for binary univariate twin modeling, twin concordance will be assessed using probandwise concordance estimates. These are calculated directly from pair counts using the formula *2C / (2C + D)*, where C is the number of concordant pairs and D the number of discordant pairs.

Heritability Analyses for the Continuous Measure

Heritability was operationalized by the univariate twin (ACE) model which partitions shared variance between monozygotic and dizygotic twins into estimates of (A) additive genetic effects, (C) common environmental effects, and (E) unique environmental effects and randomness (Martin & Eaves 1977). Analyses were conducted in R using the OpenMx package (v2.21.13), following the standard structural equation modeling approach for continuous outcomes in twin samples (Boker et al., 2011; Posthuma et al., 2003).

Saturated models were first fit to the raw twin data to estimate means and covariances separately for monozygotic (MZ) and dizygotic (DZ) twin pairs. This unconstrained model provided a baseline assessment of twin similarity, allowing us to verify that the phenotype exhibited sufficient variance and covariance structure for ACE modeling. Then, ACE models were run to partition variance of gender dysphoria into ACE influences, while controlling for age and sex. When multiple timepoints were available, the participant's highest score was used. This choice follows prior work suggesting that gender incongruence reflects a stable trait-like disposition (Bailey et al., 2016), making peak expression the most informative indicator. Including the full longitudinal series would have required modeling a more complex nested structure, for which the available data were insufficiently powered.

Missing Data

The twin sample had no missing data for self-reported gender identity. A total of 1,940 out of 1,970 participants (98.5%) had at least one usable self-reported gender incongruence score. Eighteen twin pairs (1.8%) had missing gender incongruence scores for both twins, while four pairs (0.4%) had gender incongruence scores for one twin but not the other. Therefore, ACE models were fit to the pairwise complete data of 1,926 observations from 963 unique twin pairs.

Results

In the full sample, probandwise concordance for TNB identity was 46% (95% CI: 30–63%) among monozygotic pairs, compared to 13% (95% CI: 4–30%) among dizygotic pairs (Table 2). Stratified analyses by sex showed similar patterns, though gender incongruence was much more common in females. Among female pairs, concordance was 50% (95% CI: 32–68%) in monozygotic twins and 17% (95% CI: 5–37%) in dizygotic twins. Among male pairs, probandwise concordance was 29% (95% CI: 4–71%) for monozygotic twins and 0% (95% CI: 0–41%) for dizygotic twins. Concordance estimates were consistently higher in monozygotic than dizygotic twins, indicating greater similarity in gender identity among genetically identical twins.

Table 2. Twin Pair Concordance in Gender Identity by Zygosity and Sex

Zygosity	Concordant TNB	Discordant	Concordant Cisgender	Probandwise
Full Sample Pairs				
Monozygotic	9	21	389	.46 [.30, .63]
Dizygotic	2	27	537	.13 [.04, .30]

Male Pairs				
Monozygotic	1	5	208	.29 [.04, .71]
Dizygotic	0	7	274	0 [0, .41]
Female Pairs				
Monozygotic	8	16	181	0.50 [.32, .68]
Dizygotic	2	20	263	0.17 [.05, .37]

For continuous measures of gender incongruence, ACE models estimated that additive genetic variance accounted for approximately 47% (95%CI: 33–100*%) of the variance of gender dysphoria (A = .339, 95%CI: [.241, 1.0*]) after controlling for age and sex. Shared environmental influences accounted for less than 3% (95%CI: 0–14%) of variance (C = .019, 95%CI: [.00*, .101]) while unique environment and error accounted for the remaining 50% (95%CI: 0–55%) of variance (E = .357, 95%CI: [.00*, .390]). (*Lower bounds of .00 and upper bounds of 1.0 indicate that the estimate failed to converge.)

Discussion

The present study provides a genetically informed analysis of gender incongruence in a large, contemporary sample of 985 twin pairs from the Adolescent Brain Cognitive Development (ABCD) Study. There were two main findings. First, categorical treatment of gender incongruence indicated higher probandwise concordance for transgender or nonbinary (TNB) identity among monozygotic (MZ) compared to dizygotic (DZ) twins. That is, if one twin expressed a TNB identity, the other twin was more likely to also identify as TNB if that twin was

MZ rather than DZ, indicating genetic influence on gender identity variance. Second, continuous measures of gender incongruence were also shown to have strong heritability.

In the full sample, probandwise concordance for TNB identity was 46% among monozygotic pairs, compared to only 13% among dizygotic pairs. TNB identity was more common in females among the twin sample, as in the full sample presented in Chapter 1, and had stronger twin concordance compared to males. Among female pairs, MZ concordance was 50% (i.e., of all females who were MZ twins and identified as TNB, half of them had an identical twin who also identified as TNB, while the other half did not) while DZ concordance was only 17%. Among male pairs, MZ concordance was 29% while DZ concordance was 0%. As TNB identity concordance is much more common in MZ than DZ twin pairs, these results indicate a large genetic influence on gender identity variance.

These results are corroborated by heritability analyses which partitioned the variance of continuous measures of gender incongruence into additive genetic effects (A), shared environmental effects (C), and unique environmental effects or error (E) using structural equation modeling (Boker et al., 2011; Martin & Eaves 1977; Posthuma et al., 2003). Consistent with probandwise concordance results for the categorical measure, ACE models estimated that genetic effects contributed 47% of the variance of gender incongruence in the ABCD twin sample, with shared environmental effects accounting for approximately 3% of the variance and unique environmental effects or error accounting for the remaining 50% of variance.

Combined, these findings indicate a large genetic component to gender incongruence, consistent with prior research (Bailey et al., 2016; Coolidge et al., 2002; Gómez-Gil et al., 2010; Karamanis et al., 2022). However, results should be contextualized against other psychological outcomes. Sexual orientation, for example, which is known to have many polygenic influences,

is only roughly 30% heritable, despite common intuitions of innateness (Bailey et al., 2016). Conversely, clinical outcomes such as depression and anxiety, known to be impacted by adverse childhood experiences, have been shown to be more than 50% heritable in adolescents (Polderman et al., 2015). As such, strong heritability does not necessarily imply immutability of the trait in question.

This pattern of roughly half genetic and half unique environmental influence is entirely typical of psychological traits, consistent with the Three Laws of Behavioral Genetics (Turkheimer, 2000). Namely: (1) all human behavioral traits are partially heritable; (2) the effect of shared environment is typically smaller than the effect of genes; (3) a large amount of variance is attributable to non-shared environmental effects. By definition, heritability refers only to the partitioning of variance within a particular sample at a particular time, rather than the proportion of an effect that is "genetically caused" (for example, number of fingers is genetically determined yet has close to zero heritability, because the overwhelming majority of variance is explained by injuries as opposed to genetic defects).

Heritability estimates, therefore, must be interpreted cautiously in light of several limitations. The present findings reflect genetic influences in this specific cohort of American adolescents during the unusual social context of the late 2010s and early 2020s, during which the prevalence of gender incongruence was rapidly changing (Cass, 2024; K. Zucker, 2017). Different historical or cultural contexts may yield different estimates. Moreover, it is unclear whether these genetic influences pertain directly to one's felt experience of gender, as classical and nativist theories of gender incongruence predict (Turban, 2024), or whether it may be confounded by other highly heritable mental health or personality traits, as ROGD would predict

(Littman, 2018). Indeed, genetics research has shown that factors influencing gender identity, gender expression, and sexual orientation are highly correlated (Bailey et al., 2016).

Though the overall twin sample was large with 1,970 individuals, only 70 (3.6%) of these identified as TNB. Of these, 48 (68.5%) were from discordant pairs (i.e., one twin identified as TNB and the other did not) while 22 (31.5%) were from concordant pairs (i.e., both twins identified as TNB). With the small proportion of TNB twins, there was insufficient power for formal binary ACE modeling, and probandwise concordance estimates had large confidence intervals indicating high uncertainty. While continuous measures of gender incongruence were completed by the full sample and had sufficient power for ACE modeling, outcomes were skewed and lacked sufficient power to test for sex differences. This is an important limitation given recent large sex differences in gender dysphoria prevalence (Cass, 2024), and the possibility that results presented here may only be applicable in females, who made up the majority of the TNB sample.

Despite these limitations, this study demonstrates that gender incongruence in adolescence is substantially influenced by genetic factors, with little evidence for shared environmental effects. These results may inform treatment outcomes for gender dysphoria, promoting a biological psychiatric approach, and may indicate that it is a stable trait-like phenomenon rather than a transient clinical state. Future research is necessary to explore sex differences in the heritability of gender incongruence, whether this differs by age, and whether these results are generalizable to clinical populations.

III. Biopsychosocial Correlates of Adolescent Gender Incongruence

Gender incongruence, defined as a mismatch between one's felt sense of gender identity and natal sex, has emerged as a central construct in contemporary debates about the etiology of gender dysphoria (Byrne, 2023). Gender incongruence may or may not be accompanied by psychological distress, social transition, or clinical gender dysphoria. Historically, such incongruence most often appeared in early childhood, was more common in males, and frequently resolved by adolescence, often coinciding with the emergence of same-sex attraction (Bailey & K. Zucker, 1995). In contrast, recent epidemiological shifts show sharp increases in adolescent-onset incongruence, particularly among females, raising questions about whether new developmental pathways are at play (Cass, 2024; K. Zucker, 2017). One proposed account of this shift has been dubbed rapid-onset gender dysphoria (ROGD), a hypothesized form of adolescent-onset incongruence thought to arise in the context of peer influence, internalizing psychopathology, and other psychosocial vulnerabilities (Littman, 2018).

Although ROGD remains controversial and has not been rigorously tested (see critiques in Ashley, 2020; Restar, 2020; Turban et al., 2023), it makes many testable predictions. Namely, the increased incidence in gender dysphoria being attributable to ROGD would predict that it is primarily adolescent-onset and primarily affecting females, unlike classical gender dysphoria manifesting in early childhood and primarily affecting males. While Chapter 1 showed that adolescent-onset gender incongruence disproportionately affects females, many causes could be at play not necessarily aligning with ROGD. Though associations between gender incongruence and internalizing symptoms (e.g., depression and anxiety) have been well-documented, even outside of the context of clinical gender dysphoria, directionality is debated (Gibson et al., 2021; Martinez Agulleiro et al., 2024; Turban & Ehrensaft, 2018). Minority stress theory suggests that

gender incongruence, or holding any form of stigmatized identity including being transgender or nonbinary, will precede internalizing symptoms and subsequently exacerbate them (Frost & Meyer, 2023). Conversely, ROGD would predict that individuals with high internalizing symptoms or other pre-existing problems, such as family conflict, peer victimization, and cyberbullying, will be more likely to subsequently develop gender incongruence as a coping mechanism (Littman, 2018). Longitudinal data are needed to rigorously test these competing frameworks; however, the two are not mutually exclusive, and the relationships between gender incongruence and internalizing symptoms or social stress may be bidirectional (K. Zucker et al., 2014).

The primary aim of this study is to establish biopsychosocial mechanisms associated with adolescent gender incongruence. As mentioned, minority stress theory and ROGD make opposite predictions with regard to directionality of the association between gender incongruence and internalizing symptoms, family conflict, peer victimization, and cyberbullying, which will be directly tested against each other. Relatedly, ROGD makes direct predictions concerning "social contagion," namely, that adolescents with high degrees of exposure to gender-nonconforming peers or online content will be at higher risk for developing gender incongruence (Ashley, 2020; Littman, 2018). Conversely, minority stress theory compatible with classical gender dysphoria would predict that gender incongruence would predict a higher number of transgender friends, or time spent online seeking community, as a form of support or refuge following onset of gender incongruence, not preceding it. I hypothesize that both screen time, as a proxy for online community seeking, and number of transgender or nonbinary friends will be positively associated with gender incongruence. Directionally, I will test predictions made by minority stress theory and ROGD against each other.

Physiologically, ROGD predicts that body dysmorphia not intrinsically related to gender, such as perceptions of attractiveness, may contribute to gender incongruence, particularly in adolescent females (Littman, 2018). If this is true, gender incongruence may relate to high body mass index (BMI) and early pubertal timing, which have both been associated with higher risk of internalizing symptoms and body image issues, particularly in females (Kaplowitz, 2008; Milano et al., 2020; Pfeifer & Allen, 2021; Ullsperger & Nikolas, 2017). Though it is theoretically possible that gender incongruence may alter BMI through over- or under-eating, as a result of stress of gender-nonconforming body ideals, and likewise alter pubertal timing through stress, I hypothesize that these relationships will only exist prospectively. That is, higher BMI and early pubertal timing will predict subsequent gender incongruence, but not vice-versa, with stronger relationships expected in females.

Additionally, autism spectrum traits have been shown to be overrepresented in people experiencing gender incongruence, particularly in females, as reviewed by Kallitsounaki & Williams (2023). Adults with clinical referrals of gender dysphoria are as much as 11 times more likely to be diagnosed with comorbid autism spectrum disorder as compared to people without gender dysphoria (Kallitsounaki & Williams, 2023). Additionally, children with higher levels of autistic traits tend to report greater gender nonconformity, potentially due to unique social and cognitive pathways influencing identity development (Kallitsounaki & Williams, 2023). As such, I hypothesize that these relationships will be replicated in the present study; that is, adolescents with higher levels of autistic traits will be more likely to experience gender incongruence, particularly in females.

Lastly, as gender nonconformity and sexual orientation have strong links, particularly as observed in classical gender dysphoria (Bailey & K. Zucker, 1995), I will also examine the

directional association between gender incongruence and sexual orientation. It is possible that children experiencing early-onset gender incongruence, who subsequently come out as homosexual or bisexual, will better assimilate their gender nonconformity and see their gender incongruence decrease, as has been historically reported (Bailey & K. Zucker, 1995). If this is the case, gender incongruence is expected to precede subsequent sexual minority status, but gay or bisexual orientation is expected to be followed by a decrease in gender incongruence likelihood indicating opposite directional associations.

Altogether, the proposed study will test hypothesized biopsychosocial mechanisms associated with the previously reported increase in gender incongruence with age. These results will test competing theories on the directionality of previously shown associations between gender dysphoria and other psychological outcomes, and test novel associations between physiological and environmental effects on gender incongruence across early adolescence.

Method

Participants

The participants are the subset of the 11,502 participants (52.3% male) who completed the self-reported gender incongruence measure at least once, out of the full ABCD Study sample described in Chapter 1. All data collection was approved by the institutional review boards of all participating sites (Barch et al., 2018; Garavan et al., 2018). Because self-reported gender incongruence was assessed beginning only at Wave 2, the full data in this study consist of four timepoints, with 36,659 total observations from 11,502 unique individuals collected across Waves 2-5 (ages 9-15). Wave 2 data were collected between 2017 and 2019 when participants were 9-12 years old; Wave 3 between 2018 and 2020 when participants were 10-14 years old;

Wave 4 between 2019 and 2021 when participants were 11-14 years old; and Wave 5 between 2020 and 2022 when participants were 12-15 years old.

Measures

Gender Incongruence

As in Chapter 1, self-reported gender incongruence is operationalized as the average of the following three items on a five-point scale:

1. How much do you feel like a boy? (1 = Not at all, 5 = Totally)
2. How much do you feel like a girl? (1 = Not at all, 5 = Totally)
3. How much have you had the wish to be a [girl/boy]* (1 = Never, 5 = Always)

*Males are asked the "girl" form of the question and females are asked the "boy" form.

This measure does not assess the dimensions of distress or impairment relevant to clinical gender dysphoria (American Psychiatric Association, 2022). As such, results may not generalize to clinical populations, but are conceptually useful for understanding developmental changes in the gender incongruence facet of gender dysphoria. Parent-reported gender incongruence is not included here, as results in Chapter 1 showed it had only modest correlations with self-report (r's $\leq .34$) and did not change as significantly with age.

Internalizing Symptoms

Internalizing symptoms were assessed by the parent-reported Child Behavior Checklist (CBCL; Achenbach & Rescorla, 2001). The CBCL is administered to parents at every timepoint in the ABCD Study (Barch et al., 2018). There are a total of 31 items corresponding to internalizing symptoms, with 14 items corresponding to the Anxious/Depressed subscale, 9 items

to the Withdrawn/Depressed subscale, and 8 items to the Somatic Symptoms subscale. As I did not have specific hypotheses across sub-dimensions, internalizing symptoms were computed as the average score across all items. Average scores from incomplete responses were retained so long as more than half of the items were complete (at least 16 items); responders missing more than half of the items were excluded.

Sexual Orientation

At every timepoint, children are asked "Are you gay or bisexual?" (Yes, No, Maybe, I do not understand this question, Decline to answer), as part of the KSADS (Barch et al., 2018; Kaufman et al., 1997; Kobak et al., 2013; Potter et al., 2022). Children who responded "Yes" or "Maybe" to this question were coded as "LGB" (lesbian, gay, or bisexual) and children who responded "No" were coded as "Straight." Children responding "I do not understand this question" or "Decline to answer" were treated as missing (see Missing Data section for rates). Children whose sexual orientation changes across timepoints will have each response counted, as longitudinal models will use wave-specific data.

Autism Spectrum Traits

Autism spectrum traits were assessed by the 11-item parent-reported brief adaptation of the Social Responsiveness Scale (SRS; Constantino et al., 2003; Reiersen et al., 2008). The SRS is not a clinical diagnosis and measures social difficulties which may be experienced by non-autistic children, but which are more common in autistic children. The average scores across all 11 items were used; incomplete responses were retained so long as more than half of the items are complete. The SRS was only administered at Wave 2 (N = 11,201, ages 9-12).

Pubertal Timing

Pubertal development was assessed at every timepoint using the Pubertal Development Scale (PDS; Petersen et al., 1988). Both parents and children completed the PDS at every timepoint. Only self-reported scores were used, as these have been shown to have higher reliability than parent-reported scores beginning at age 9 years (Dorn et al., 1990; Koopman-Verhoeff et al., 2020; Omary et al., 2025). The self-reported PDS asks participants of both sexes questions such as "Have you noticed any skin changes, especially pimples?" and sex-specific questions such as "Have you noticed that your breasts have begun to grow?" for females and "Have you begun to grow hair on your face?" for males. All raw scores are on a scale of 1 (Has not yet started growing/changing) to 4 (Seems complete).

Two transformations were made to the self-reported PDS scores. Firstly, PDS scores were transformed onto the Tanner Stage scale following the methodology of Shirtcliff et al. (2009), which is more comparable to physician ratings and uses a more developmentally appropriate weighting system so as not to over-score or under-score children, particularly females with early or late menstruation. Secondly, Shirtcliff-adjusted PDS scores were *z*-scored and residualized for age within each sex, following the methodology of Omary et al. (2025). This operationalizes pubertal scores as pubertal timing, and provides an estimate of standard deviations above or behind the mean pubertal stage of age- and sex-matched peers.

Body Mass Index

At each visit, participants' height and weight were measured on site (Barch et al., 2018). BMI was calculated using the standard formula of weight in kilograms divided by height in meters squared.

Family Conflict

At every timepoint, parents completed the Family Environment Scale, which includes a 9-item Family Conflict subscale (Moos & Moos, 1994; R. Zucker et al., 2018). The total family conflict score was computed as the sum score of each binary item (e.g., "In our family we argue a lot"; "My family members sometimes are violent"). If responses were missing one or two items, their average scores across all complete items were imputed. If participants were missing more than two items, their responses were treated as missing.

Peer Victimization

ABCD administers the 18-item Peer Experiences Questionnaire (PEQ; Prinstein et al., 2001) to participants beginning at Wave 3 and every year thereafter. The PEQ features 6 items each assessing Overt (e.g., "A kid threatened to hurt or beat me up"), Relational (e.g., "Some kids left me out of an activity or conversation I really wanted to be included in"), and Reputational (e.g., "Another kid said mean things about me so that people would think I was a loser") victimization on a 5-point scale (1 = Never, 5 = A few times a week). Peer victimization was operationalized as the average score across all 18 items. Subscores will not be utilized as I did not have specific hypotheses across victimization types. Incomplete data were included so long as participants completed at least half of the items on the scale.

Cyberbullying

Also beginning at Wave 3 and administered every year thereafter, youth were surveyed on cyberbullying (Hoffman et al., 2019). Participants are asked:

> "Have you ever been cyberbullied, where someone was trying on purpose to harm you or be mean to you online, in texts, or group texts, or on social media (like Instagram or Snapchat)?" (Yes, No, Refuse to answer)

And if yes:

> "Has this happened to you in the past 12 months?" (Yes, No)

Following the methodology of Shao et al. (2024) and Liu et al. (2025), participants were scored as having experienced cyberbullying if they respond "Yes" to both questions and "No" otherwise. Responses of "Refuse to answer" were treated as missing.

Screen Time

Youth and their parents completed various questionnaires regarding screen, technology, and social media usage at each wave as part of the ABCD Youth Screen Time Survey (Bagot et al., 2022). Beginning at Wave 3 (ages 10-14) and every wave thereafter, youth were asked to endorse the number of hours for the following for both a typical weekday and weekend:

> "On a typical [WEEKDAY/WEEKEND], how much TIME per day do you spend in TOTAL on a computer, phone, tablet, iPod, or other device or video game? Please do NOT include time spent on school related work, but do include watching TV, shows or videos, texting or chatting, playing games, or visiting social networking sites (Facebook, Twitter, Instagram)."

Total average screen time was calculated as the weighted average hours from weekdays and weekends: (Weekday*5 + Weekend*2)/7.

Transgender/Nonbinary Friends

Lastly, participants were administered the Youth Resilience Scale beginning at Wave 3 and every wave thereafter (Hoffman et al., 2019). This measure includes three open numeric response questions which ask:

1. "How many friends that are boys do you have?"
2. "How many friends that are girls do you have?"
3. "How many friends that are another gender do you have (for example, nonbinary)?"

For purposes of this study, only responses to the last question were assessed. Numeric responses to this question are referred to as "TNB friends" and were used as a proxy for peer groups familiar with and supportive of gender incongruence.

Multivariate Analyses

As in Chapter 1, linear mixed-effects models were used to estimate gender incongruence. Random effects were used to account for repeated measures within participants across longitudinal waves and to capture the nested structure of the data, with subject ID nested within household ID to account for shared variance between siblings recruited from the same household. Multivariate models will build onto the three-way age-by-sex-by-gender developmental trajectory model from Chapter 1, with the same demographic covariates:

$$Gender\ Incongruence \sim Age*Sex*TNB + Race + Income + Education + (1 \mid Family\ ID/Subject\ ID)$$

The multivariate model will add to the base model all of the aforementioned biopsychosocial predictors: internalizing symptoms, sexual orientation, autism spectrum traits, pubertal timing, body mass index, screen time, cyberbullying, peer victimization, family conflict,

and number of TNB friends, as well as study timepoint ("wave") to control for possible period effects. All predictor variables were initially modeled with an exploratory sex interaction effect to test potential sex-specific effects:

*Gender Incongruence ~ Age*Sex*TNB + Race + Income + Education + Sex*(Internalizing + Sexual Orientation + Autism + Pubertal Timing + BMI + Family Conflict + Victimization + Cyberbullying + Screen Time + TNB Friends) + Study Wave + (1 | Family ID/Subject ID)*

If sex interaction terms were nonsignificant, the exploratory model was re-fit into a final model without these interactions.

Prior to interpretation of the final multivariate model, false discovery rate correction (FDR) was applied to account for repeated tests between predictor variables with a significance threshold of $\alpha = .05$ (Benjamini & Hochberg, 1995). Variables with significant effects after applying FDR correction were carried over into cross-lagged analyses, while variables not surviving FDR will be discarded.

Cross-Lagged Analyses

Longitudinal cross-lagged panel models were conducted using the "lavaan" package version 0.6.14 in R (Rosseel, 2012) to examine prospective bidirectional relationships between gender incongruence and significant biopsychosocial predictors identified in the multivariate analyses. Cross-lagged models allow for the simultaneous estimation of autoregressive paths (stability of constructs over time) and cross-lagged paths (prospective influence between constructs across timepoints), while controlling for within-time covariances (S. Mackinnon et al., 2022).

Only variables with significant associations in the final multivariate model after false discovery rate correction were included. For each significant predictor, a univariate cross-lagged model was fit to test the bidirectional association between the predictor and self-reported gender incongruence across all available timepoints (Waves 2-3, 3-4, and/or 4-5, depending on the variable). If a sex interaction term was identified in the final multivariate model, cross-lagged models for that variable were stratified by sex. All cross-lagged models included age, sex, and gender as covariates (unless stratified by sex, in which case sex was not included as covariate).

Missing Data

Out of 37,270 total possible observations from 11,522 unique individuals who had any data at Waves 2-5, there were 611 total missing observations (1.6%) for self-reported gender incongruence. A total of 11,502 individuals (99.8%) had at least one complete self-reported gender incongruence score.

The following variables were collected at every timepoint and had a total of 36,659 possible observations from 11,502 unique participants: internalizing symptoms, sexual orientation, pubertal timing, body mass index, and family conflict. A total of 11,487 participants (99.9%) had at least one complete internalizing symptoms score with 383 total missing observations (1.0%). A total of 11,426 participants (99.3%) had at least one complete sexual orientation response with 1,895 total missing observations (5.2%). Of these, 83 (0.2%) were true missing, 303 (0.8%) were "Decline to answer," and 1,509 (4.1%) were "I do not understand this question." A total of 11,496 participants (99.9%) had at least one complete puberty score with 183 (0.5%) total missing observations. A total of 11,281 participants (98.1%) had at least one

BMI score with 11,461 (31.3%) total missing observations. Lastly, a total of 11,484 participants (99.8%) had at least one family conflict score with 381 (1.0%) total missing observations.

The following variables were collected at only Waves 3-5 and had a total of 25,680 possible observations from 11,134 unique participants: peer victimization, cyberbullying, screen time, and number of TNB friends. A total of 11,129 participants (99.9%) had at least one peer victimization score with 28 (0.1%) total missing observations. A total of 11,125 participants (99.9%) had at least one cyberbullying score with 92 (0.4%) total missing observations. Of these, 29 (0.1%) were true missing and 63 (0.2%) were "Refuse to answer." A total of 11,127 participants (99.9%) had at least one screen time report with 34 (0.1%) total missing observations. Lastly, a total of 8,869 participants (79.7%) had at least one number of TNB friends report with 16,011 (62.4%) total missing observations.

Finally, autism spectrum traits were only measured at Wave 2, which had a total of 11,502 possible observations. A total of 10,966 participants (95.3%) had complete scores with 536 (4.7%) total missing observations.

Multiple Imputation

Missing data of biopsychosocial predictor variables were imputed using multiple imputation with chain equations using the "MICE" package in R (van Buuren & Groothuis-Oudshoorn, 2011). Gender incongruence as an outcome variable was not imputed if missing. Additionally, demographic variables were not imputed if missing, including gender identity and sexual orientation. Even the relatively high missingness rates for sexual orientation, BMI, and number of TNB friends had enough complete observations which demonstrated strong enough covariance with other complete subject-level data for MICE assumptions to be met. MICE

generated 20 multiply imputed datasets which stably converged within 10 iterations each. All multivariate and cross-lagged analyses were conducted on these 20 imputed datasets, and all estimates presented are pooled results across all 20 sets of estimates. Each imputed dataset had 36,659 observations from 11,502 unique participants.

Results

Correlations

Correlations between all continuous variables, averaged across all timepoints, are shown in Figure 1. Every variable of interest had a significant association with self-reported gender incongruence except for BMI in males and family conflict in males. The strongest associations with gender incongruence were peer victimization and autism symptoms in males, and peer victimization, screen time, number of TNB friends, and internalizing symptoms in females.

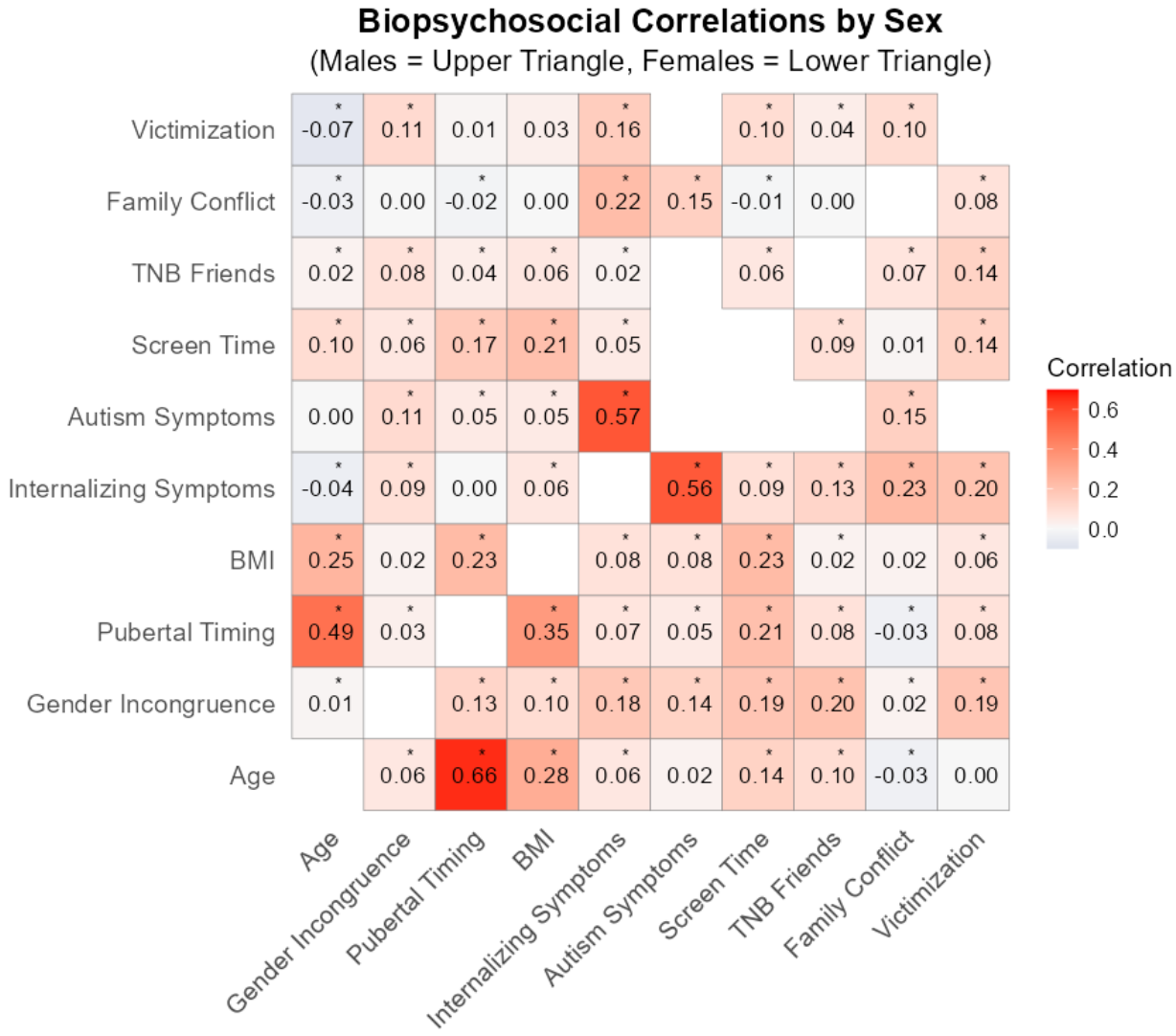


Figure 1. Correlations Between All Continuous Variables. Correlations among the male sample (N = 6,016) are shown in the upper left of the diagonal. Correlations among the female sample (N = 5,486) are shown in the lower right of the diagonal.

Multivariate Analyses

An initial exploratory model, with interaction terms by sex for each predictor of interest on gender incongruence, found that the effects of internalizing symptoms, sexual orientation,

pubertal timing, family conflict, cyberbullying experience, and number of TNB friends did not significantly differ by sex. Therefore, the model was re-fit without these interaction terms.

Table 1 summarizes the final multivariate linear mixed-effects model on gender incongruence. All variables of interest, except family conflict and number of TNB friends, were significantly associated with gender incongruence after false discovery rate correction. The strongest predictors of gender incongruence were TNB self-identity (β = 1.124, z = 19.99, p < .001), LGB sexual orientation (β = 0.631, z = 37.37, p < .001), and female sex (β = 0.254, z = 18.93, p < .001). Furthermore, a significant sex-by-gender interaction showed that TNB females were additionally likely to report higher gender incongruence, beyond the additive effects (β = 0.378, z = 6.05, p < .001). Additionally, there was a significant three-way age-by-sex-by-gender interaction effect, as well as significant age-by-sex and age-by-gender interaction effects on gender incongruence. Post hoc tests revealed that, after controlling for study wave, demographics, and other biopsychosocial predictors, mean gender incongruence decreased with age for cisgender males (β = -0.05, 95%CI [-0.07, -0.03]) and females (β = -0.11, 95%CI [-0.13, -0.08]), and increased with age for TNB males (β = 0.48, 95%CI [0.40, 0.55]) and females (β = 0.74, 95%CI [0.69, 0.78]).

Table 1. Multivariate Linear Mixed-Effects Model on Gender Incongruence.

Term	**β (SE)**	**Test Statistic**
Intercept	-0.325 (0.036)	z = -8.96, p < .001***
Wave 3	-0.006 (0.013)	z = -0.47, p = .681
Wave 4	0.042 (0.020)	z = 2.15, p = .049*
Wave 5	0.107 (0.030)	z = 3.56, p < .001***
Age	-0.050 (0.012)	z = -4.24, p < .001***
Female	0.254 (0.013)	z = 18.93, p < .001***

TNB	1.124 (0.056)	$z = 19.99$, $p < .001$***
Black	0.055 (0.021)	$z = 2.63$, $p = .017$*
Hispanic	0.027 (0.017)	$z = 1.55$, $p = .158$
Asian	0.026 (0.043)	$z = 0.60$, $p = .616$
Multiracial	0.050 (0.021)	$z = 2.42$, $p = .028$*
Income < $50,000	0.027 (0.015)	$z = 1.75$, $p = .112$
Income > $100,000	-0.029 (0.013)	$z = -2.24$, $p = .040$*
Parent High School	0.049 (0.032)	$z = 1.52$, $p = .160$
Parent Some College	0.031 (0.033)	$z = 0.96$, $p = .392$
Parent Bachelor's Degree	-0.013 (0.033)	$z = -0.40$, $p = .705$
Parent Graduate Degree	0.012 (0.032)	$z = 0.38$, $p = .705$
Internalizing Symptoms	0.025 (0.007)	$z = 3.35$, $p = .002$**
LGB Sexual Orientation	0.631 (0.017)	$z = 37.37$, $p < .001$***
Autistic Traits	0.019 (0.008)	$z = 2.42$, $p = .028$*
Pubertal Timing	0.018 (0.006)	$z = 2.94$, $p = .007$**
Body Mass Index	-0.013 (0.007)	$z = -1.72$, $p = .117$
Family Conflict	-0.005 (0.005)	$z = -0.97$, $p = .392$
Victimization	0.022 (0.006)	$z = 3.35$, $p = .002$**
Cyberbullied	0.055 (0.022)	$z = 2.47$, $p = .028$*
Screen Time	0.004 (0.008)	$z = 0.47$, $p = .681$
TNB Friends	0.020 (0.011)	$z = 1.86$, $p = .108$
Age*Female	-0.057 (0.009)	$z = -6.14$, $p < .001$***
Age*TNB	0.526 (0.039)	$z = 13.52$, $p < .001$***
Female*TNB	0.378 (0.063)	$z = 6.05$, $p < .001$***
Female*Autistic Traits	0.057 (0.013)	$z = 4.34$, $p < .001$***
Female*Body Mass Index	0.036 (0.013)	$z = 2.80$, $p = .012$*
Female*Victimization	0.041 (0.011)	$z = 3.88$, $p < .001$***
Female*Screen Time	0.050 (0.010)	$z = 4.89$, $p < .001$***
Age*Female*TNB	0.317 (0.044)	$z = 7.29$, $p < .001$***

Internalizing symptoms (β = 0.025, z = 3.35, p = .002), cyberbullying experience (β = 0.055, z = 2.47, p = .028), and earlier pubertal timing (β = 0.018, z = 2.94, p = .007) were all associated with increased gender incongruence, and these associations did not differ by sex. Autism spectrum traits, body mass index, peer victimization, and screen time were all significantly associated with gender incongruence, with significant interaction effects by sex. Post hoc tests revealed that autism spectrum traits were positively associated with gender incongruence, with a stronger effect in females (β = 0.02, 95% CI [0.00, 0.03]) than in males (β = 0.08, 95% CI [0.05, 0.10]). Body mass index was not significantly associated with gender incongruence in males (β = –0.01, 95% CI [–0.03, 0.00]) but was significantly positively associated in females (β = 0.02, 95% CI [0.00, 0.05]). Peer victimization was positively associated with gender incongruence in both males (β = 0.02, 95% CI [0.01, 0.03]) and females (β = 0.06, 95% CI [0.04, 0.08]), with a stronger effect in females. Screen time was not significantly associated with gender incongruence in males (β = 0.00, 95% CI [–0.01, 0.02]) but was positively associated in females (β = 0.05, 95% CI [0.04, 0.07]).

Controlling for all other effects and compared to Wave 2, where the gender incongruence measure was first collected, Wave 4 (β = 0.042, z = 2.15, p = .049) and Wave 5 (β = 0.107, z = 3.56, p < .001) collections had significantly higher mean gender incongruence scores, pointing to a possible period effect. Waves 2 and 3 did not significantly differ. Additionally, compared to white participants, black (β = 0.055, z = 2.63, p = .017) and multiracial (β = 0.042, z = 2.15, p = .049) participants had significantly higher mean gender incongruence. Lastly, relative to middle-income households ($50-100k), participants from high-income households (>$100k) had significantly lower mean gender incongruence scores after controlling for all other predictors (β = -0.029, z = -2.24, p = .040).

Cross-Lagged Analyses

Multivariate results showed that all predictors of interest except family conflict and number of TNB friends were significantly associated with gender incongruence, after false discovery rate correction. Therefore, univariate cross-lagged models were run for internalizing symptoms, sexual orientation, pubertal timing, body mass index, peer victimization, cyberbullying experience, and screen time, in order to examine the longitudinal directionality of these effects. Autism spectrum traits were not included in cross-lagged modeling because this measure was collected at only one timepoint. Because significant interactions by sex were identified for the effects of peer victimization, this model was stratified by sex. Additionally, because the effects of body mass index and screen time on gender incongruence were only significant in females, these analyses were only run on the female subsample. All cross-lagged *p*-values are FDR-adjusted to account for repeated measurements across lags and labeled as *q*; all other autoregressive, concurrent, and covariate effects are unadjusted.

Internalizing Symptoms

Cross-lagged relationships between gender incongruence and internalizing symptoms across Waves 2-5 (ages 9-15) are visualized in Figure 2 and summarized in Table 2. Autoregressive paths indicated moderate stability for gender incongruence (β's = .342-.492, *p*'s < .001) with decreasing stability over time, and strong stability for internalizing symptoms (β's = .676-.706, *p*'s < .001) with increasing stability over time, after controlling for age, sex, and gender identity. Concurrent relationships between internalizing symptoms and gender incongruence were small but significant at every timepoint (β's = .061-.107, *q*'s < .001), and largest at Wave 5. Cross-lagged effects revealed directional specificity: internalizing symptoms

predicted weak increased likelihood of gender incongruence at every subsequent timepoint (β's = .032-.045, *q*'s ≤ .001), but gender incongruence did not significantly predict subsequent internalizing symptoms.

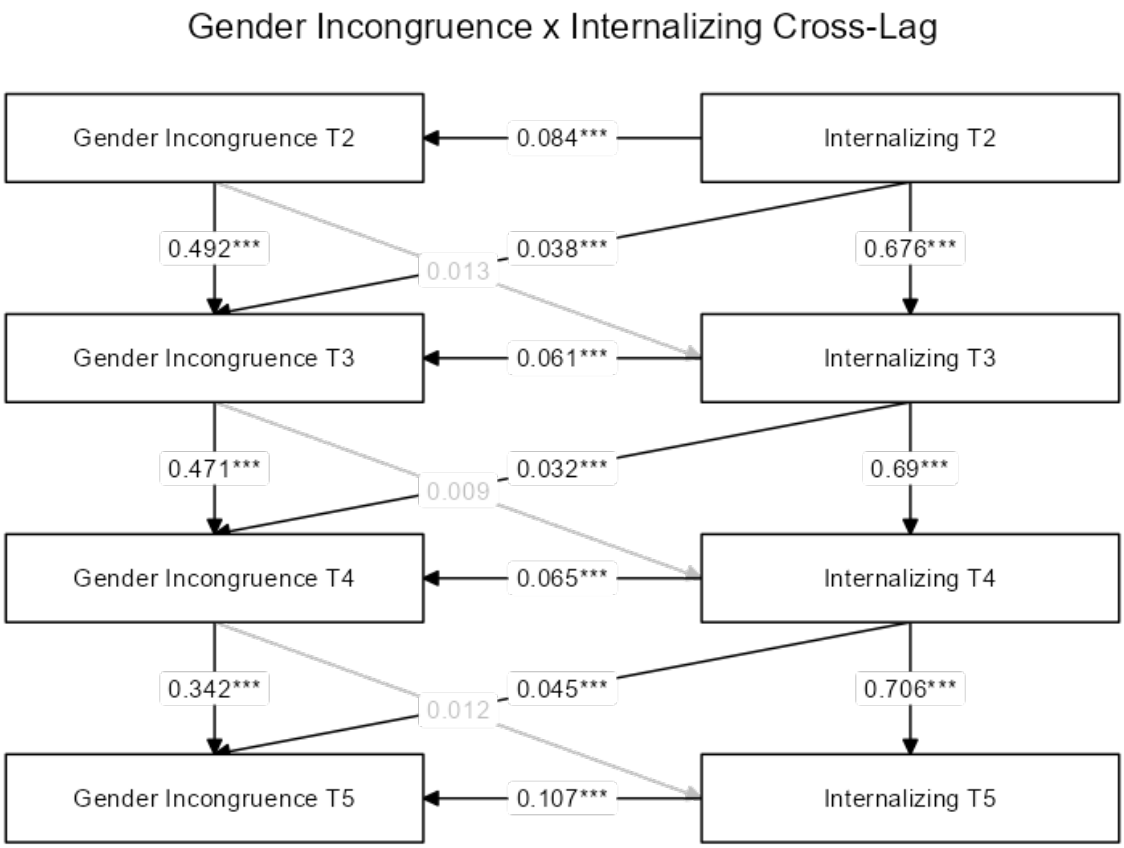


Figure 2. Cross-Lagged Effects of Internalizing Symptoms on Gender Incongruence.

Table 2. Cross-Lagged Effects of Internalizing Symptoms on Gender Incongruence.

Autoregressives	**Std. β**	**Unstd. B**	**SE**	***z***	***p***
Gender Incongruence T3 ~ Gender Incongruence T2	0.492	0.494	0.02	25.141	< .001***
Internalizing T3 ~ Internalizing T2	0.676	0.686	0.012	56.112	< .001***
Gender Incongruence T4 ~ Gender Incongruence T3	0.471	0.549	0.021	25.889	< .001***
Internalizing T4 ~ Internalizing T3	0.690	0.727	0.013	57.594	< .001***

Gender Incongruence T5 ~ Gender Incongruence T4	0.342	0.415	0.034	12.212	< .001***
Internalizing T5 ~ Internalizing T4	0.706	0.73	0.017	42.478	< .001***
Concurrent Effects	**Std. β**	**Unstd. B**	**SE**	***z***	***p***
Gender Incongruence T2 ~~ Internalizing T2	0.084	0.214	0.029	7.259	< .001***
Gender Incongruence T3 ~~ Internalizing T3	0.061	0.095	0.019	4.942	< .001***
Gender Incongruence T4 ~~ Internalizing T4	0.065	0.108	0.024	4.472	< .001***
Gender Incongruence T5 ~~ Internalizing T5	0.107	0.194	0.04	4.844	< .001***
Cross-Lagged Effects	**Std. β**	**Unstd. B**	**SE**	***z***	***q***
Gender Incongruence T3 ~ Internalizing T2	0.038	0.003	0.001	4.087	< .001***
Internalizing T3 ~ Gender Incongruence T2	0.013	0.163	0.111	1.465	.214
Gender Incongruence T4 ~ Internalizing T3	0.032	0.003	0.001	3.499	.001***
Internalizing T4 ~ Gender Incongruence T3	0.009	0.123	0.135	0.906	.438
Gender Incongruence T5 ~ Internalizing T4	0.045	0.005	0.001	3.535	.001***
Internalizing T5 ~ Gender Incongruence T4	0.012	0.136	0.192	0.71	.478
Covariates	**Std. β**	**Unstd. B**	**SE**	***z***	***p***
Gender Incongruence T3 ~ Age T3	0.033	0.023	0.006	4.107	< .001***
Gender Incongruence T3 ~ Sex	-0.098	-0.09	0.007	-12.508	< .001***
Gender Incongruence T3 ~ TNB	0.246	0.481	0.036	13.237	< .001***
Internalizing T3 ~ Age T3	0.023	0.19	0.06	3.171	.002**
Internalizing T3 ~ Sex	-0.023	-0.259	0.082	-3.153	.002**
Internalizing T3 ~ TNB	0.053	1.28	0.209	6.135	< .001***

Gender Incongruence T4 ~ Age T4	0.004	0.003	0.006	0.562	.574
Gender Incongruence T4 ~ Sex	-0.107	-0.114	0.008	-14.262	< .001***
Gender Incongruence T4 ~ TNB	0.332	0.757	0.043	17.702	< .001***
Internalizing T4 ~ Age T4	0.002	0.023	0.066	0.343	.732
Internalizing T4 ~ Sex	-0.039	-0.469	0.088	-5.327	< .001***
Internalizing T4 ~ TNB	0.081	2.066	0.262	7.883	< .001***
Gender Incongruence T5 ~ Age T5	-0.014	-0.013	0.009	-1.521	.128
Gender Incongruence T5 ~ Sex	-0.101	-0.131	0.013	-9.913	< .001***
Gender Incongruence T5 ~ TNB	0.48	1.328	0.068	19.404	< .001***
Internalizing T5 ~ Age T5	-0.004	-0.035	0.09	-0.385	.700
Internalizing T5 ~ Sex	-0.065	-0.8	0.126	-6.362	< .001***
Internalizing T5 ~ TNB	0.068	1.787	0.41	4.353	< .001***

Sexual Orientation

Cross-lagged relationships between gender incongruence and sexual orientation across Waves 2–5 (ages 9–15) are visualized in Figure 3 and summarized in Table 3. Autoregressive paths indicated moderate stability for gender incongruence (β's = .308–.476, *p*'s < .001) with decreasing stability over time, and moderate stability for sexual orientation (β's = .332–.452, *p*'s < .001) with increasing stability over time, after controlling for age, sex, and gender identity. Concurrent relationships between gender incongruence and LGB orientation were moderate and significant at every timepoint (β's = .210–.319, *p*'s < .001). Cross-lagged effects revealed bidirectional associations: LGB orientation predicted small increases in subsequent gender incongruence across all intervals (β's = .077–.127, *q*'s < .001), and gender incongruence predicted small increases in subsequent LGB orientation from T2 to T3 and T3 to T4 (β's = .076–.094, *q*'s < .001), but not from T4 to T5.

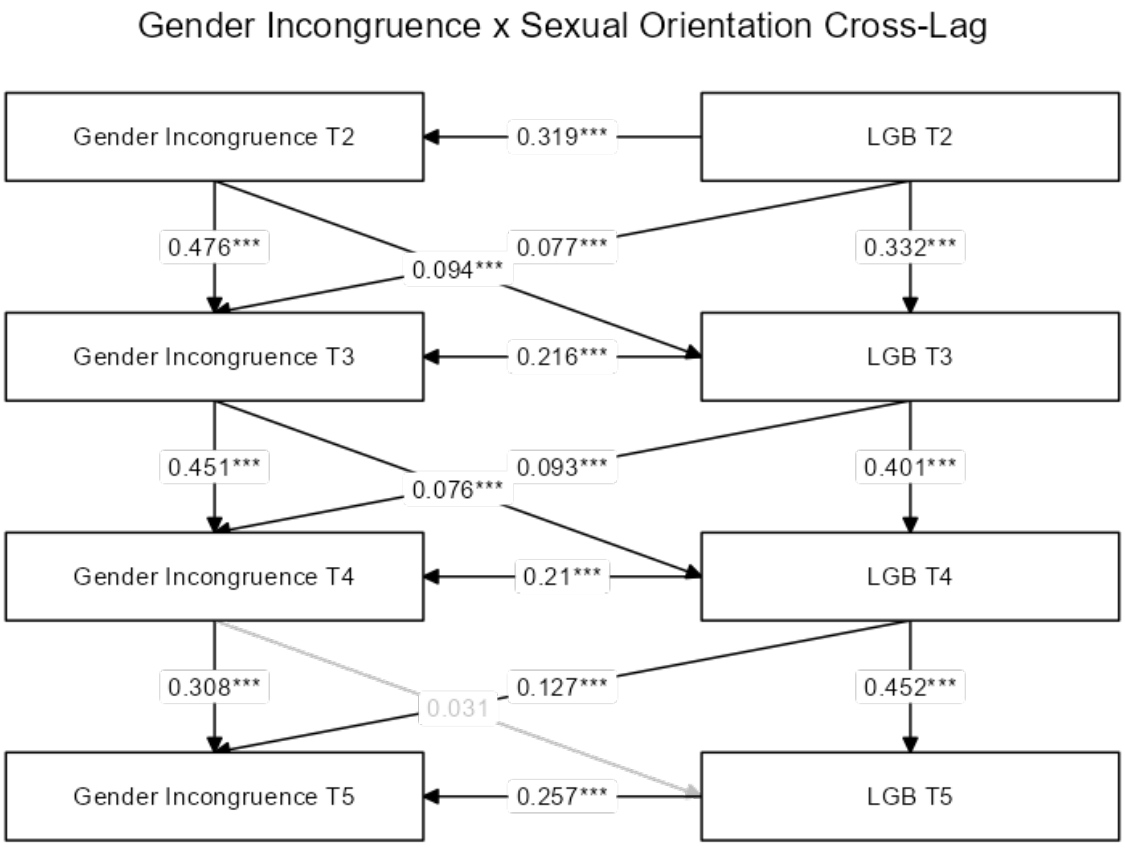


Figure 3. Cross-Lagged Effects of Sexual Orientation on Gender Incongruence.

Table 3. Cross-Lagged Effects of Sexual Orientation on Gender Incongruence.

Autoregressives	**Std. β**	**Unstd. B**	**SE**	***z***	***p***
Gender Incongruence T3 ~ Gender Incongruence T2	0.476	0.477	0.02	24.006	< .001***
LGB T3 ~ LGB T2	0.332	0.461	0.027	17.233	< .001***
Gender Incongruence T4 ~ Gender Incongruence T3	0.451	0.524	0.022	23.879	< .001***
LGB T4 ~ LGB T3	0.401	0.513	0.02	26.212	< .001***
Gender Incongruence T5 ~ Gender	0.308	0.375	0.035	10.672	< .001***

Incongruence T4					
LGB T5 ~ LGB T4	0.452	0.535	0.023	22.892	< .001***
Concurrent Effects	**Std. β**	**Unstd. B**	**SE**	***z***	***p***
Gender Incongruence T2 ~~ LGB T2	0.319	0.028	0.002	12.38	< .001***
Gender Incongruence T3 ~~ LGB T3	0.216	0.019	0.002	11.364	< .001***
Gender Incongruence T4 ~~ LGB T4	0.210	0.023	0.002	12.653	< .001***
Gender Incongruence T5 ~~ LGB T5	0.257	0.035	0.003	11.308	< .001***
Cross-Lagged Effects	**Std. β**	**Unstd. B**	**SE**	***z***	***q***
Gender Incongruence T3 ~ LGB T2	0.077	0.181	0.042	4.276	< .001***
LGB T3 ~ Gender Incongruence T2	0.094	0.056	0.01	5.822	< .001***
Gender Incongruence T4 ~ LGB T3	0.093	0.182	0.029	6.352	< .001***
LGB T4 ~ Gender Incongruence T3	0.076	0.057	0.011	5.081	< .001***
Gender Incongruence T5 ~ LGB T4	0.127	0.238	0.039	6.026	< .001***
LGB T5 ~ Gender Incongruence T4	0.031	0.024	0.016	1.49	.136
Covariates	**Std. β**	**Unstd. B**	**SE**	***z***	***p***
Gender Incongruence T3 ~ Age T3	0.031	0.021	0.005	3.9	< .001***
Gender Incongruence T3 ~ Sex	-0.095	-0.087	0.007	-12.063	< .001***
Gender Incongruence T3 ~ TNB	0.23	0.45	0.036	12.659	< .001***
LGB T3 ~ Age T3	0.049	0.02	0.004	5.558	< .001***
LGB T3 ~ Sex	-0.122	-0.066	0.005	-13.547	< .001***
LGB T3 ~ TNB	0.27	0.311	0.021	14.84	< .001***
Gender Incongruence T4 ~ Age T4	0.002	0.002	0.006	0.267	.790
Gender Incongruence T4 ~ Sex	-0.098	-0.104	0.008	-13.147	< .001***
Gender Incongruence T4 ~ TNB	0.31	0.702	0.044	16.114	< .001***
LGB T4 ~ Age T4	0.015	0.008	0.004	1.83	.067
LGB T4 ~ Sex	-0.151	-0.104	0.006	-16.845	< .001***
LGB T4 ~ TNB	0.183	0.269	0.022	12.106	< .001***
Gender Incongruence T5 ~ Age T5	-0.018	-0.017	0.009	-1.97	.049*
Gender Incongruence T5 ~ Sex	-0.089	-0.115	0.013	-8.883	< .001***
Gender Incongruence T5 ~ TNB	0.457	1.26	0.07	18.116	< .001***

LGB T5 ~ Age T5	0.015	0.009	0.007	1.332	.183
LGB T5 ~ Sex	-0.2	-0.163	0.011	-15.25	< .001***
LGB T5 ~ TNB	0.149	0.259	0.031	8.365	< .001***

Pubertal Timing

Cross-lagged relationships between gender incongruence and pubertal timing across Waves 2–5 (ages 9–15) are visualized in Figure 4 and summarized in Table 4. Autoregressive paths indicated moderate stability for gender incongruence (β's = .344–.492, *p*'s < .001) and stronger stability for pubertal timing (β's = .483–.547, *p*'s < .001), after controlling for age, sex, and gender identity. Concurrent relationships between gender incongruence and pubertal timing were small at T2 (β = .123, *p* < .001), significant but weak at T3 (β = .041, *p* < .001), and nonsignificant at T4 and T5. Cross-lagged effects revealed that earlier pubertal timing predicted weak increases in subsequent gender incongruence across all intervals (β's = .032–.042, *p*'s ≤ .004), whereas gender incongruence did not predict later pubertal timing at T3 or T4, and showed only a negligible negative effect at T5 (β = –.028, *p* = .030).

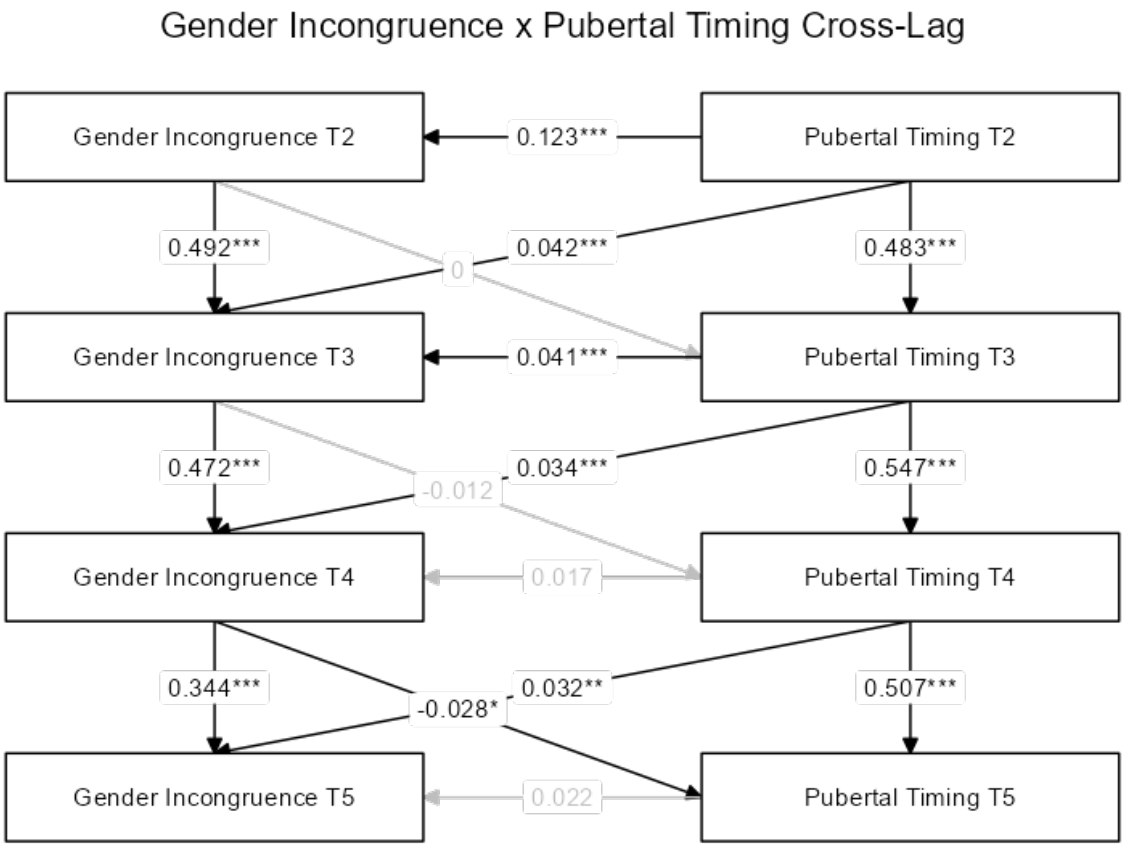


Figure 4. Cross-Lagged Effects of Pubertal Timing on Gender Incongruence.

Table 4. Cross-Lagged Effects of Pubertal Timing on Gender Incongruence.

Autoregressives	**Std. β**	**Unstd. B**	**SE**	***z***	***p***
Gender Incongruence T3 ~ Gender Incongruence T2	0.492	0.494	0.020	25.080	< .001***
Pubertal Timing T3 ~ Pubertal Timing T2	0.483	0.530	0.009	58.159	< .001***
Gender Incongruence T4 ~ Gender Incongruence T3	0.472	0.550	0.021	25.781	< .001***
Pubertal Timing T4 ~ Pubertal Timing T3	0.547	0.574	0.008	67.875	< .001***
Gender Incongruence T5 ~ Gender Incongruence T4	0.344	0.418	0.034	12.262	< .001***
Pubertal Timing T5 ~ Pubertal Timing	0.507	0.429	0.011	38.047	< .001***

T4					
Concurrent Effects	**Std. β**	**Unstd. B**	**SE**	***z***	***p***
Gender Incongruence T2 ~~ Pubertal Timing T2	0.123	0.053	0.005	11.090	< .001***
Gender Incongruence T3 ~~ Pubertal Timing T3	0.041	0.013	0.003	4.060	< .001***
Gender Incongruence T4 ~~ Pubertal Timing T4	0.017	0.005	0.003	1.680	.093
Gender Incongruence T5 ~~ Pubertal Timing T5	0.022	0.006	0.004	1.637	.102
Cross-Lagged Effects	**Std. β**	**Unstd. B**	**SE**	***z***	***q***
Gender Incongruence T3 ~ Pubertal Timing T2	0.042	0.020	0.005	4.515	< .001***
Pubertal Timing T3 ~ Gender Incongruence T2	0.000	0.000	0.019	0.017	.986
Gender Incongruence T4 ~ Pubertal Timing T3	0.034	0.017	0.004	4.012	< .001***
Pubertal Timing T4 ~ Gender Incongruence T3	-0.012	-0.030	0.020	-1.520	.154
Gender Incongruence T5 ~ Pubertal Timing T4	0.032	0.019	0.006	3.085	.004**
Pubertal Timing T5 ~ Gender Incongruence T4	-0.028	-0.048	0.021	-2.322	.030*
Covariates	**Std. β**	**Unstd. B**	**SE**	***z***	***p***
Gender Incongruence T3 ~ Age T3	0.026	0.018	0.006	3.124	.002**
Gender Incongruence T3 ~ Sex	-0.088	-0.081	0.007	-11.127	< .001***
Gender Incongruence T3 ~ TNB	0.246	0.482	0.036	13.280	< .001***
Pubertal Timing T3 ~ Age T3	0.255	0.397	0.012	33.228	< .001***
Pubertal Timing T3 ~ Sex	-0.306	-0.638	0.017	-37.564	< .001***
Pubertal Timing T3 ~ TNB	0.023	0.105	0.033	3.165	.002**
Gender Incongruence T4 ~ Age T4	-0.006	-0.005	0.006	-0.791	.429
Gender Incongruence T4 ~ Sex	-0.094	-0.100	0.008	-11.865	< .001***

Gender Incongruence T4 ~ TNB	0.334	0.759	0.043	17.807	< .001***
Pubertal Timing T4 ~ Age T4	0.170	0.286	0.013	21.902	< .001***
Pubertal Timing T4 ~ Sex	-0.215	-0.471	0.018	-26.256	< .001***
Pubertal Timing T4 ~ TNB	0.025	0.115	0.033	3.465	.001**
Gender Incongruence T5 ~ Age T5	-0.025	-0.023	0.010	-2.418	.016*
Gender Incongruence T5 ~ Sex	-0.090	-0.116	0.014	-8.346	< .001***
Gender Incongruence T5 ~ TNB	0.484	1.338	0.069	19.430	< .001***
Pubertal Timing T5 ~ Age T5	0.128	0.172	0.016	10.833	< .001***
Pubertal Timing T5 ~ Sex	-0.264	-0.488	0.022	-22.009	< .001***
Pubertal Timing T5 ~ TNB	0.007	0.027	0.038	0.718	.473

Body Mass Index in Females

Cross-lagged relationships between gender incongruence and body mass index (BMI) in females across Waves 2–5 (ages 9–15) are visualized in Figure 5 and summarized in Table 5. Autoregressive paths indicated moderate-to-high stability for gender incongruence (β's = .342–.503, *p*'s < .001) with decreasing stability over time, and strong stability for BMI from T2 to T3 (β = .792, *p* < .001), followed by moderate stability from T3 to T5 (β's = .284–.316, *p*'s < .001), after controlling for age and gender identity. Concurrent relationships between gender incongruence and BMI were small and significant at T2 and T4 (β's = .057–.069, *p*'s ≤ .005), but nonsignificant at T3 and T5. Cross-lagged effects were inconsistent: higher BMI predicted small increases in subsequent gender incongruence from T2 to T3 and T3 to T4 (β's = .039–.042, *q*'s = .009), but not from T4 to T5, while gender incongruence predicted only a small increase in subsequent BMI from T2 to T3 (β = .027, *q* = .036) and was nonsignificant thereafter.

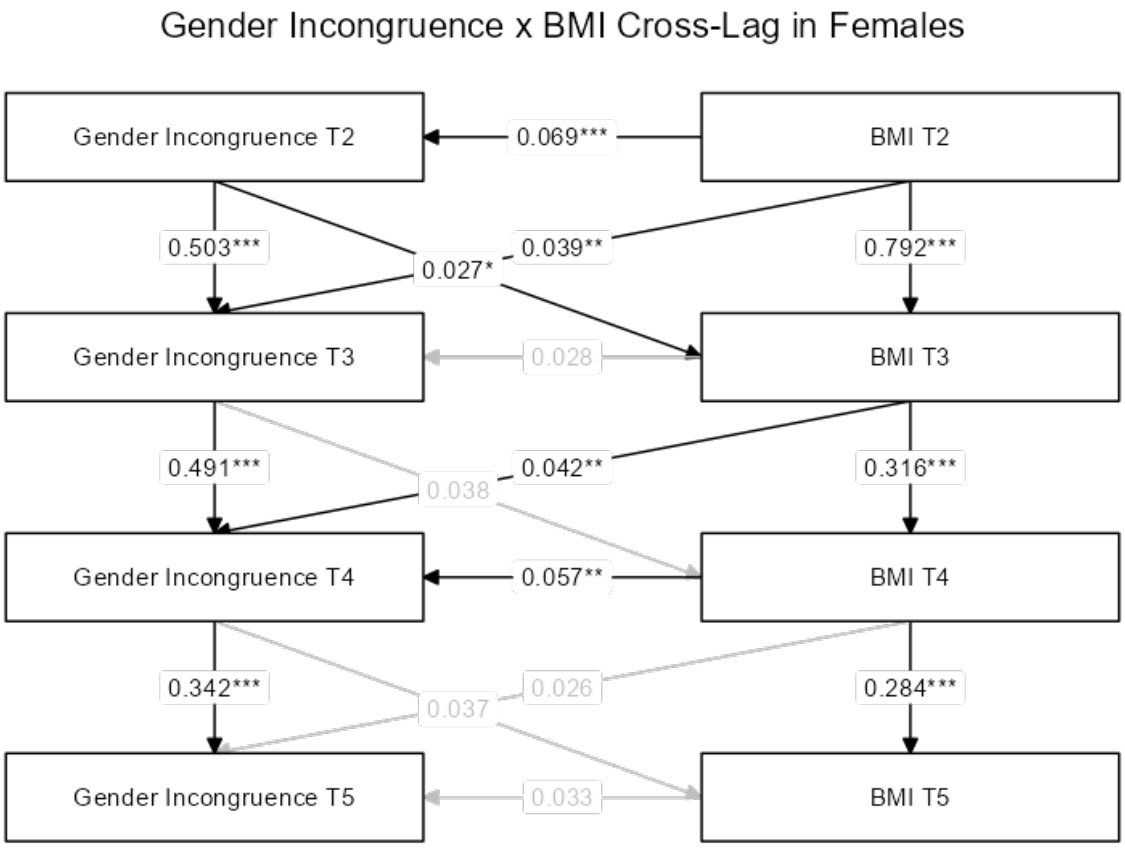


Figure 5. Cross-Lagged Effects of Body Mass Index on Gender Incongruence in Females.

Table 5. Cross-Lagged Effects of Body Mass Index on Gender Incongruence in Females.

Autoregressives	**Std. β**	**Unstd. B**	**SE**	***z***	***p***
Gender Incongruence T3 ~ Gender Incongruence T2	0.503	0.515	0.025	20.456	< .001***
BMI T3 ~ BMI T2	0.792	0.864	0.023	37.433	< .001***
Gender Incongruence T4 ~ Gender Incongruence T3	0.491	0.566	0.027	20.976	< .001***
BMI T4 ~ BMI T3	0.316	0.345	0.026	13.247	< .001***
Gender Incongruence T5 ~ Gender Incongruence T4	0.342	0.411	0.043	9.613	< .001***
BMI T5 ~ BMI T4	0.284	0.303	0.034	8.811	< .001***
Concurrent Effects	**Std. β**	**Unstd. B**	**SE**	***z***	***p***

Gender Incongruence T2 ~~ BMI T2	0.069	0.191	0.047	4.096	< .001***
Gender Incongruence T3 ~~ BMI T3	0.028	0.044	0.028	1.565	.120
Gender Incongruence T4 ~~ BMI T4	0.057	0.155	0.054	2.892	.005**
Gender Incongruence T5 ~~ BMI T5	0.033	0.102	0.075	1.354	.177
Cross-Lagged Effects	**Std. β**	**Unstd. B**	**SE**	***z***	***q***
Gender Incongruence T3 ~ BMI T2	0.039	0.005	0.002	2.968	.009**
BMI T3 ~ Gender Incongruence T2	0.027	0.249	0.105	2.379	.036*
Gender Incongruence T4 ~ BMI T3	0.042	0.005	0.002	3.010	.009**
BMI T4 ~ Gender Incongruence T3	0.038	0.373	0.204	1.827	.105
Gender Incongruence T5 ~ BMI T4	0.026	0.004	0.002	1.470	.174
BMI T5 ~ Gender Incongruence T4	0.037	0.344	0.267	1.286	.199
Covariates	**Std. β**	**Unstd. B**	**SE**	***z***	***p***
Gender Incongruence T3 ~ Age T3	0.048	0.042	0.011	3.635	< .001***
Gender Incongruence T3 ~ TNB	0.258	0.507	0.048	10.617	< .001***
BMI T3 ~ Age T3	0.045	0.351	0.080	4.379	< .001***
BMI T3 ~ TNB	0.010	0.177	0.181	0.980	.329
Gender Incongruence T4 ~ Age T4	0.002	0.002	0.012	0.153	.878
Gender Incongruence T4 ~ TNB	0.353	0.799	0.054	14.680	< .001***
BMI T4 ~ Age T4	0.055	0.488	0.149	3.282	.001**
BMI T4 ~ TNB	0.013	0.262	0.354	0.740	.462
Gender Incongruence T5 ~ Age T5	-0.018	-0.022	0.018	-1.212	.226
Gender Incongruence T5 ~ TNB	0.525	1.429	0.083	17.120	< .001***
BMI T5 ~ Age T5	0.044	0.402	0.202	1.989	.047*
BMI T5 ~ TNB	-0.002	-0.042	0.471	-0.088	.930

Peer Victimization by Sex

In females, cross-lagged relationships between gender incongruence and peer victimization across Waves 3–5 (ages 10–15) are visualized in Figure 6 and summarized in Table

6. Autoregressive paths indicated moderate-to-high stability for gender incongruence (β's = .332–.524, *p*'s < .001) and peer victimization (β's = .462–.528, *p*'s < .001), both with decreased stability over time, after controlling for age and gender identity. Concurrent relationships between gender incongruence and victimization in females were moderate at Wave 3 (β = .227, *p* < .001) and weak at Waves 4 and 5 (β's = .085–.091, *p*'s < .001). Increased peer victimization at T4 was associated with slightly increased gender incongruence at T5 (β = .052, *q* = .012); no other cross-lagged effects were significant between victimization and gender incongruence in females.

In males, cross-lagged relationships between gender incongruence and peer victimization across Waves 3–5 are visualized in Figure 7 and summarized in Table 7. Autoregressive paths indicated moderate stability for gender incongruence (β's = .342–.440, *p*'s < .001) and moderate-to-high stability for victimization (β's = .472–.524, *p*'s < .001), both with decreasing stability over time, after controlling for age and gender identity. Concurrent relationships between gender incongruence and victimization in males were small but significant at every wave (β's = .061–.114, *p*'s ≤ .015). Cross-lagged effects revealed no significant associations between peer victimization and gender incongruence in either direction at any timepoint in males.

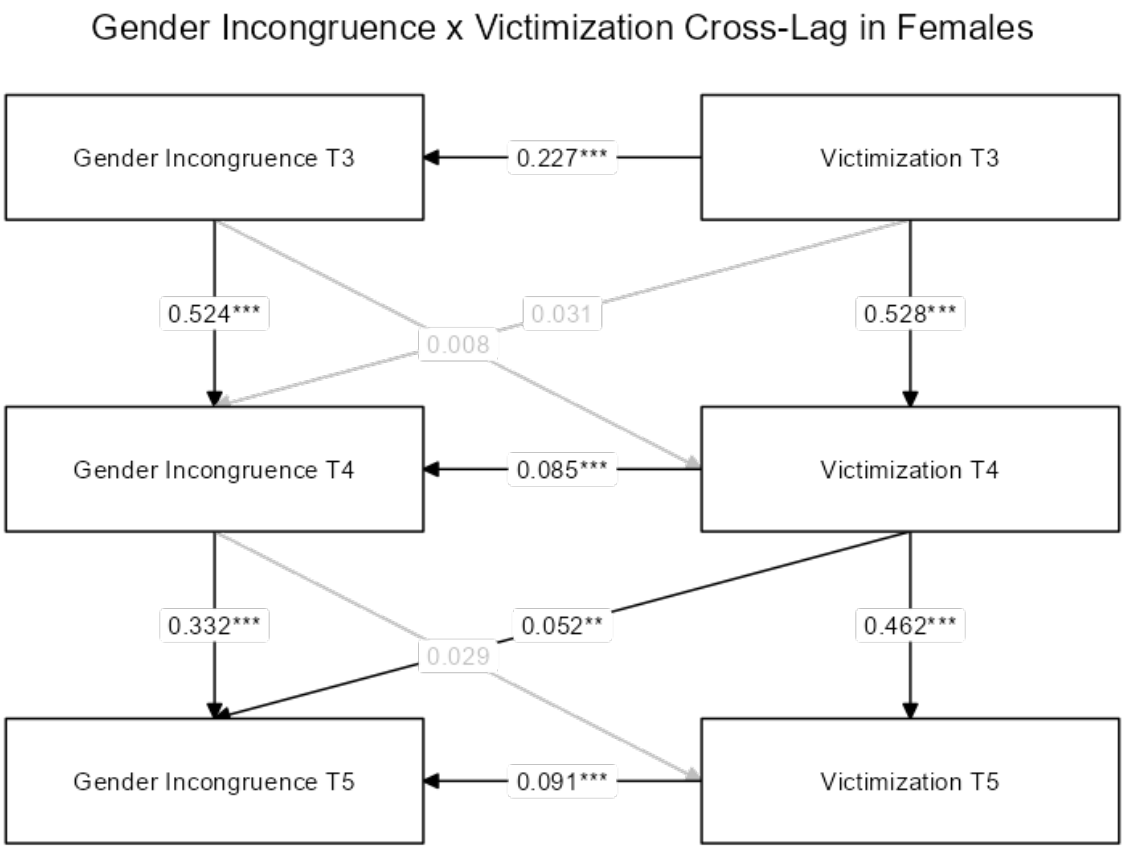


Figure 6. Cross-Lagged Effects of Peer Victimization on Gender Incongruence in Females.

Table 6. Cross-Lagged Effects of Peer Victimization on Gender Incongruence in Females.

Autoregressives	**Std. β**	**Unstd. B**	**SE**	***z***	***p***
Gender Incongruence T4 ~ Gender Incongruence T3	0.524	0.564	0.024	23.098	< .001***
Victimization T4 ~ Victimization T3	0.528	0.489	0.019	25.307	< .001***
Gender Incongruence T5 ~ Gender Incongruence T4	0.332	0.402	0.039	10.355	< .001***
Victimization T5 ~ Victimization T4	0.462	0.445	0.026	16.893	< .001***
Concurrent Effects	**Std. β**	**Unstd. B**	**SE**	***z***	***p***
Gender Incongruence T3 ~~ Victimization T3	0.227	0.063	0.006	10.896	< .001***
Gender Incongruence T4 ~~	0.085	0.015	0.003	4.409	< .001***

Victimization T4					
Gender Incongruence T5 ~~ Victimization T5	0.091	0.018	0.005	3.870	< .001***
Cross-Lagged Effects	**Std. β**	**Unstd. B**	**SE**	***z***	***q***
Gender Incongruence T4 ~ Victimization T3	0.031	0.043	0.023	1.843	.131
Victimization T4 ~ Gender Incongruence T3	0.008	0.006	0.014	0.417	.676
Gender Incongruence T5 ~ Victimization T4	0.052	0.095	0.032	2.970	.012*
Victimization T5 ~ Gender Incongruence T4	0.029	0.019	0.021	0.883	.503
Covariates	**Std. β**	**Unstd. B**	**SE**	***z***	***p***
Gender Incongruence T4 ~ Age T4	0.005	0.005	0.011	0.501	.617
Gender Incongruence T4 ~ TNB	0.368	0.803	0.049	16.408	< .001***
Victimization T4 ~ Age T4	0.041	0.027	0.008	3.344	.001**
Victimization T4 ~ TNB	0.065	0.093	0.024	3.962	< .001***
Gender Incongruence T5 ~ Age T5	-0.022	-0.025	0.016	-1.574	.116
Gender Incongruence T5 ~ TNB	0.538	1.420	0.074	19.101	< .001***
Victimization T5 ~ Age T5	-0.009	-0.005	0.011	-0.500	.617
Victimization T5 ~ TNB	0.012	0.017	0.035	0.490	.624

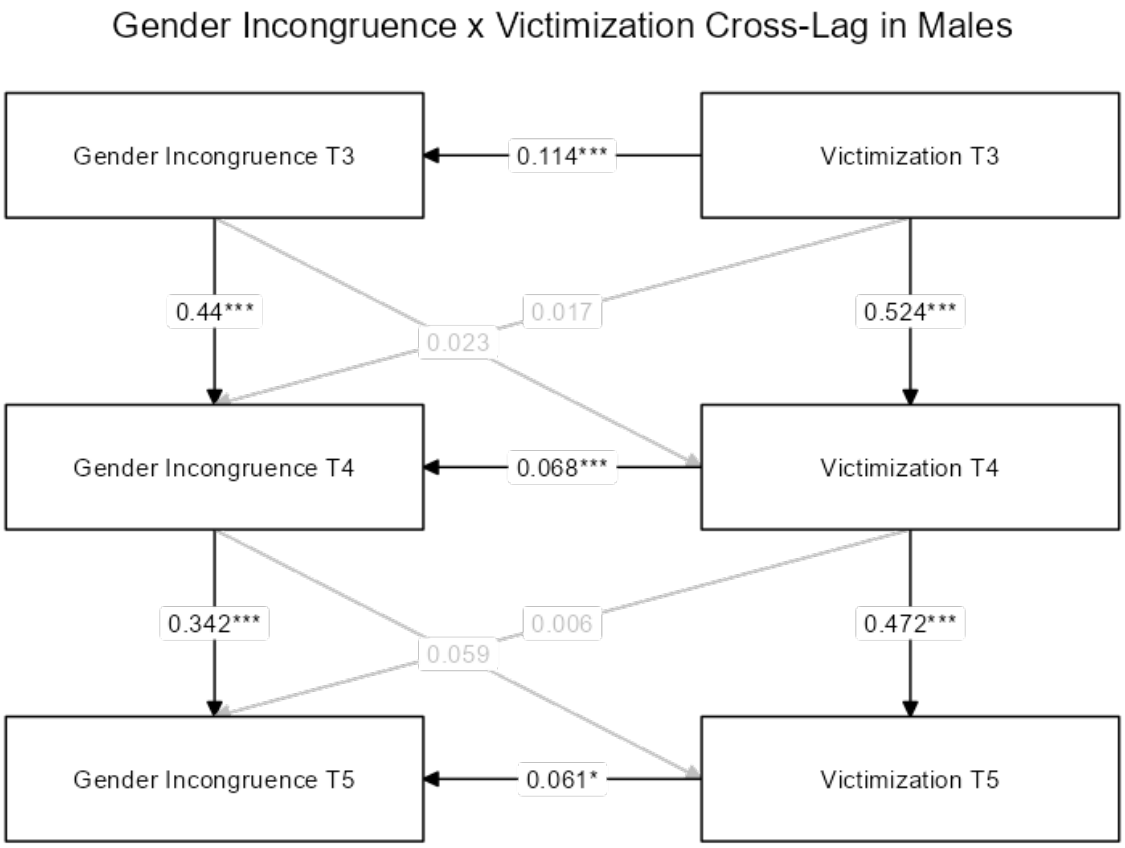


Figure 7. Cross-Lagged Effects of Peer Victimization on Gender Incongruence in Males.

Table 7. Cross-Lagged Effects of Peer Victimization on Gender Incongruence in Males.

Autoregressives	**Std. β**	**Unstd. B**	**SE**	***z***	***p***
Gender Incongruence T4 ~ Gender Incongruence T3	0.440	0.488	0.047	10.469	< .001***
Victimization T4 ~ Victimization T3	0.524	0.485	0.018	26.707	< .001***
Gender Incongruence T5 ~ Gender Incongruence T4	0.342	0.411	0.079	5.226	< .001***
Victimization T5 ~ Victimization T4	0.472	0.429	0.034	12.740	< .001***
Concurrent Effects	**Std. β**	**Unstd. B**	**SE**	***z***	***p***
Gender Incongruence T3 ~~ Victimization T3	0.114	0.015	0.003	5.695	< .001***
Gender Incongruence T4 ~~	0.068	0.007	0.002	4.228	< .001***

Victimization T4					
Gender Incongruence T5 ~~ Victimization T5	0.061	0.006	0.003	2.435	.015*
Cross-Lagged Effects	**Std. β**	**Unstd. B**	**SE**	***z***	***q***
Gender Incongruence T4 ~ Victimization T3	0.017	0.011	0.009	1.195	.309
Victimization T4 ~ Gender Incongruence T3	0.023	0.035	0.023	1.497	.269
Gender Incongruence T5 ~ Victimization T4	0.006	0.005	0.016	0.320	.749
Victimization T5 ~ Gender Incongruence T4	0.059	0.074	0.033	2.225	.104
Covariates	**Std. β**	**Unstd. B**	**SE**	***z***	***p***
Gender Incongruence T4 ~ Age T4	0.007	0.003	0.005	0.632	.527
Gender Incongruence T4 ~ TNB	0.294	0.633	0.087	7.232	< .001***
Victimization T4 ~ Age T4	0.006	0.004	0.008	0.486	.627
Victimization T4 ~ TNB	0.027	0.079	0.051	1.546	.122
Gender Incongruence T5 ~ Age T5	-0.012	-0.007	0.009	-0.737	.461
Gender Incongruence T5 ~ TNB	0.380	0.983	0.161	6.110	< .001***
Victimization T5 ~ Age T5	-0.015	-0.008	0.010	-0.806	.420
Victimization T5 ~ TNB	0.043	0.115	0.081	1.427	.154

Cyberbullying

Cross-lagged relationships between gender incongruence and cyberbullying across Waves 3–5 (ages 10–15) are visualized in Figure 8 and summarized in Table 8. Autoregressive paths indicated moderate-to-high stability for gender incongruence (β's = .339–.512, *p*'s < .001) with decreasing stability over time, and weak-to-moderate stability for cyberbullying (β's = .221–.224, *p*'s < .001), after controlling for age, sex, and gender identity. Concurrent

relationships between gender incongruence and cyberbullying were small but significant at every timepoint (β's = .050–.087, *p*'s ≤ .001). Cross-lagged effects revealed that gender incongruence predicted small increases in subsequent cyberbullying from T3 to T4 (β = .049, *q* = .002); no other longitudinal associations between gender incongruence and cyberbullying were significant.

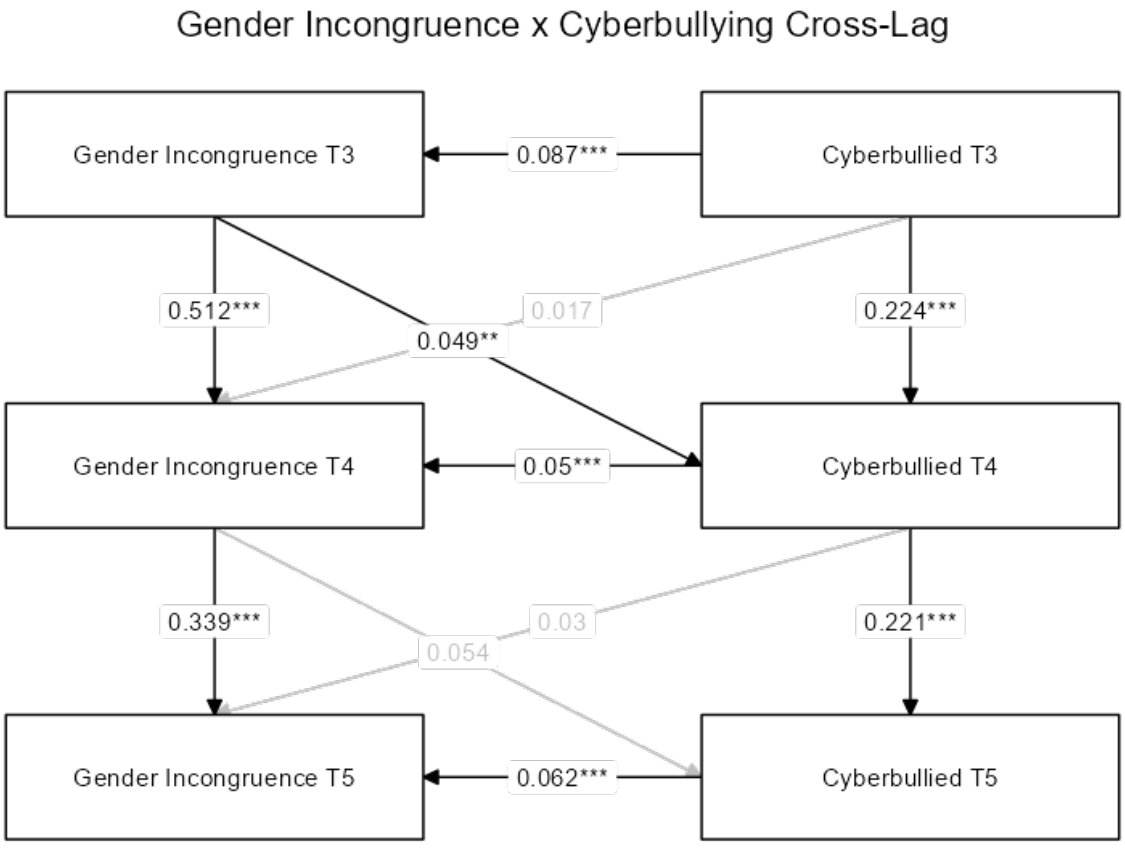


Figure 8. Cross-Lagged Effects of Cyberbullying on Gender Incongruence.

Table 8. Cross-Lagged Effects of Cyberbullying on Gender Incongruence.

Autoregressives	**Std. β**	**Unstd. B**	**SE**	***z***	***p***
Gender Incongruence T4 ~ Gender Incongruence T3	0.512	0.551	0.021	25.962	< .001***
Cyberbullied T4 ~ Cyberbullied T3	0.224	0.235	0.019	12.397	< .001***
Gender Incongruence T5 ~ Gender Incongruence T4	0.339	0.416	0.034	12.152	< .001***

Cyberbullied T5 ~ Cyberbullied T4	0.221	0.226	0.027	8.311	< .001***
Concurrent Effects	**Std. β**	**Unstd. B**	**SE**	***z***	***p***
Gender Incongruence T3 ~~ Cyberbullied T3	0.087	0.010	0.002	6.355	< .001***
Gender Incongruence T4 ~~ Cyberbullied T4	0.050	0.005	0.001	3.731	< .001***
Gender Incongruence T5 ~~ Cyberbullied T5	0.062	0.007	0.002	3.328	.001***
Cross-Lagged Effects	**Std. β**	**Unstd. B**	**SE**	***z***	***q***
Gender Incongruence T4 ~ Cyberbullied T3	0.017	0.037	0.021	1.746	.081
Cyberbullied T4 ~ Gender Incongruence T3	0.049	0.026	0.007	3.502	.002**
Gender Incongruence T5 ~ Cyberbullied T4	0.030	0.075	0.037	2.014	.059
Cyberbullied T5 ~ Gender Incongruence T4	0.054	0.027	0.013	2.125	.059
Covariates	**Std. β**	**Unstd. B**	**SE**	***z***	***p***
Gender Incongruence T4 ~ Age T4	0.006	0.005	0.006	0.781	.435
Gender Incongruence T4 ~ Sex	-0.113	-0.116	0.008	-14.490	< .001***
Gender Incongruence T4 ~ TNB	0.352	0.773	0.043	18.089	< .001***
Cyberbullied T4 ~ Age T4	0.000	-0.000	0.004	-0.008	.993
Cyberbullied T4 ~ Sex	-0.011	-0.005	0.005	-1.082	.279
Cyberbullied T4 ~ TNB	0.036	0.039	0.015	2.653	.008**
Gender Incongruence T5 ~ Age T5	-0.015	-0.014	0.009	-1.539	.124
Gender Incongruence T5 ~ Sex	-0.106	-0.133	0.013	-9.999	< .001***
Gender Incongruence T5 ~ TNB	0.498	1.341	0.069	19.560	< .001***
Cyberbullied T5 ~ Age T5	0.021	0.008	0.005	1.512	.130
Cyberbullied T5 ~ Sex	-0.014	-0.007	0.007	-0.944	.345
Cyberbullied T5 ~ TNB	0.061	0.068	0.022	3.090	.002**

Screen Time in Females

Lastly, cross-lagged relationships between gender incongruence and screen time in females across Waves 3–5 (ages 10–15) are visualized in Figure 9 and summarized in Table 9. Autoregressive paths indicated moderate-to-high stability for gender incongruence (β's = .335–.514, *p*'s < .001) with decreasing stability over time, and moderate stability for screen time (β's = .446–.469, *p*'s < .001), after controlling for age and gender identity. Concurrent relationships between gender incongruence and screen time were moderate at T3 (β = .209, *p* < .001) and small at T4 and T5 (β's = .051–.059, *p*'s ≤ .023). Cross-lagged effects revealed that higher screen time predicted small increases in subsequent gender incongruence from T3 to T4 (β = .083, *q* < .001) and T4 to T5 (β = .052, *q* = .026), but earlier gender incongruence did not predict subsequent screen time, indicating directional specificity of this relationship in females.

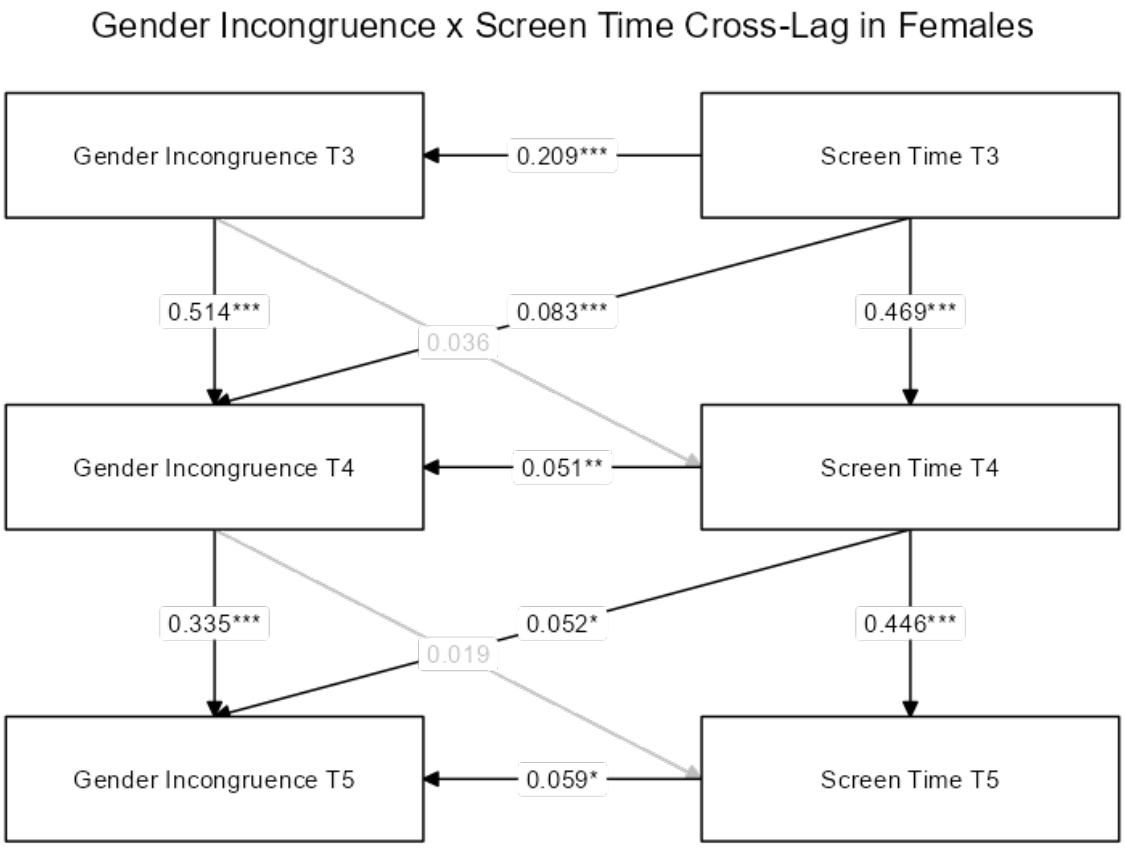


Figure 9. Cross-Lagged Effects of Screen Time on Gender Incongruence in Females.

Table 9. Cross-Lagged Effects of Screen Time on Gender Incongruence in Females.

Autoregressives	**Std. β**	**Unstd. B**	**SE**	***z***	***p***
Gender Incongruence T4 ~ Gender Incongruence T3	0.514	0.553	0.024	22.612	< .001***
Screen Time T4 ~ Screen Time T3	0.469	0.489	0.023	21.628	< .001***
Gender Incongruence T5 ~ Gender Incongruence T4	0.335	0.406	0.038	10.588	< .001***
Screen Time T5 ~ Screen Time T4	0.446	0.439	0.028	15.783	< .001***
Concurrent Effects	**Std. β**	**Unstd. B**	**SE**	***z***	***p***
Gender Incongruence T3 ~~ Screen Time T3	0.209	0.397	0.036	11.048	< .001***
Gender Incongruence T4 ~~ Screen	0.051	0.071	0.026	2.788	.005**

Time T4					
Gender Incongruence T5 ~~ Screen Time T5	0.059	0.089	0.039	2.268	.023*
Cross-Lagged Effects	**Std. β**	**Unstd. B**	**SE**	***z***	***q***
Gender Incongruence T4 ~ Screen Time T3	0.083	0.017	0.003	5.691	< .001***
Screen Time T4 ~ Gender Incongruence T3	0.036	0.195	0.105	1.853	.085
Gender Incongruence T5 ~ Screen Time T4	0.052	0.012	0.005	2.481	.026*
Screen Time T5 ~ Gender Incongruence T4	0.019	0.094	0.124	0.757	.449
Covariates	**Std. β**	**Unstd. B**	**SE**	***z***	***p***
Gender Incongruence T4 ~ Age T4	0.001	0.001	0.011	0.108	.914
Gender Incongruence T4 ~ TNB	0.367	0.800	0.048	16.507	< .001***
Screen Time T4 ~ Age T4	0.027	0.136	0.063	2.146	.032*
Screen Time T4 ~ TNB	0.054	0.596	0.163	3.647	< .001***
Gender Incongruence T5 ~ Age T5	-0.019	-0.022	0.016	-1.388	.165
Gender Incongruence T5 ~ TNB	0.538	1.423	0.074	19.178	< .001***
Screen Time T5 ~ Age T5	-0.001	-0.006	0.090	-0.069	.945
Screen Time T5 ~ TNB	0.053	0.574	0.233	2.459	.014*

Discussion

This chapter examined biopsychosocial correlates of gender incongruence in a large, demographically diverse, longitudinal sample of contemporary adolescents. After controlling for age, sex, self-reported gender identity, and demographic covariates, almost all variables of interest—internalizing symptoms, sexual orientation, autism spectrum traits, pubertal timing,

body mass index, peer victimization, cyberbullying experience, and screen time—showed significant relationships with gender incongruence.

The largest effects on gender incongruence were observed for transgender or nonbinary (TNB) gender self-identity, lesbian, gay, or bisexual (LGB) sexual orientation, and female sex, each strongly associated with greater gender incongruence. Paralleling Chapter 1, a three-way age-by-sex-by-gender interaction effect revealed that older nonbinary female adolescents experienced the strongest gender incongruence. Notably, this relationship held even when controlling for other mental health, physiological, and social-environmental confounds.

Though univariate correlations showed significant association between gender incongruence and all predictor variables of interest, family conflict and number of TNB friends did not significantly relate to gender incongruence after controlling for other predictors of interest. This may indicate that, to the extent these associations are meaningful, they are explained by mental health and other social relations.

Other associations replicate well-established findings, namely that gender incongruence is associated with increased mental health risk, including higher internalizing symptoms (Gibson et al., 2021; Martinez Agulleiro et al., 2024; Turban & Ehrensaft, 2018). Additionally, gender incongruence being associated with increased screen time, cyberbullying, and peer victimization replicates findings that gender and sexual minority youth are at increased risk for peer victimization and cyberbullying experiences (Huang et al., 2024), and may devote increased time to screens to find online social belonging (Nagata et al., 2023). However, the directionality of all of these associations remains debated. Are gender-nonconforming youth at increased mental health risk and targeted by bullies because of their gender nonconformity? Or are vulnerable victimized youth more likely to change their identity to find belongingness in new social groups?

The largely untested theory of "rapid-onset gender dysphoria" (ROGD; Littman, 2018) posits that gender dysphoria may develop in adolescents who did not previously exhibit strong gender incongruence, but who were experiencing or at risk for other internalizing disorders, body dysmorphia, and social exclusion. To this end, ROGD predicts that gender incongruence will be predicted by, rather than preceding, internalizing psychopathology, peer victimization, and body-image related variables including pubertal timing and body mass index (BMI). By contrast, minority stress theory, a dominant framework for understanding mental health in gender and sexual minority youth, posits that gender incongruence predicts increased bullying and mental health risk as a result of stigma towards minoritized identities (Frost & Meyer, 2023).

Predictions made by ROGD and minority stress theory regarding the directionality of associations between gender incongruence and psychosocial stressors were directly tested against each other using longitudinal cross-lagged panel models. Results found that the association between gender incongruence and internalizing symptoms was present at all concurrent waves, and that internalizing symptoms predicted increased gender incongruence at all subsequent timepoints, after controlling for age, sex, and gender identity, but that increased gender incongruence did not predict subsequent internalizing symptoms. Therefore, longitudinal associations better fit predictions made by ROGD, though it should be noted that these relationships only refer to subclinical levels of internalizing symptoms and gender incongruence, and may not extend to clinical diagnoses of gender dysphoria or related internalizing disorders. Moreover, stigma and other relevant features of minority stress may still explain concurrent associations between gender incongruence and internalizing symptoms at each wave (Xu et al., 2024). Also potentially supportive of ROGD was that earlier pubertal timing and increased BMI in females were both associated with increased later likelihood of gender incongruence, and both

pubertal timing and BMI have been well-established to be related to body image issues in females (Kaplowitz, 2008; Milano et al., 2020; Pfeifer & Allen, 2021; Ullsperger & Nikolas, 2017).

Peer victimization was consistently associated with gender incongruence at every wave in both sexes, yet the only significant lagged effect was from victimization at Wave 4 predicting gender incongruence at Wave 5 in females (ages 11–15). No effects were observed of gender incongruence predicting later victimization, in contrast to predictions made by minority stress theory, nor were any lagged effects significant in males. This pattern is broadly consistent with the predictions of ROGD—that victimization precedes and contributes to emerging gender incongruence particularly in adolescent females. However, cyberbullying showed the reverse pattern. While concurrent associations were significant in both sexes at every timepoint, the only significant lagged effect was that gender incongruence at Wave 3 predicted greater cyberbullying at Wave 4 (ages 9–14). This aligns with minority stress theory, in that gender incongruence itself was associated with later increased risk for online harassment. No evidence was found for cyberbullying predicting later gender incongruence. Screen time was also associated with gender incongruence, but only in females. Cross-lagged results revealed a consistent pattern of earlier screen time predicting increased later likelihood of gender incongruence, but earlier gender incongruence did not predict later screen time. This is potentially consistent with ROGD insofar as increased screen time may capture exposure to online peer groups and identity exploration opportunities that precede the development of gender incongruence. Similarly small effect sizes have been found linking screen time and transgender identity in youth, though these have been challenged as lacking practical significance (Semenyna & Ferguson, 2025).

The largest longitudinal associations were those between gender incongruence and sexual orientation. As has historically been the case, youth experiencing higher gender incongruence at earlier timepoints were at increased likelihood of later coming out as lesbian, gay, or bisexual (Bailey & K. Zucker, 1995). However, this directional association weakened over time, and was not significant at the oldest Waves 4 to 5. In contrast, LGB sexual orientation also prospectively predicted increased gender incongruence between every timepoint, and this association grew with time. This falsifies my prediction that LGB sexual orientation would predict decreased gender incongruence, and may be explained by the fact that remittance of childhood gender dysphoria upon sexual maturation is only relevant at the clinical level, and not when considering a subclinical measure of gender incongruence which may also describe gender nonconformity as a personality trait (Johnson et al., 2004). Sexual orientation within the TNB subsample, in relation to longitudinal gender persistence and desistance, will be revisited in Chapter 4.

Autism spectrum traits were also strongly associated with gender incongruence, particularly in females, as has been previously reported (reviewed by Kallitsounaki & Williams, 2023). While these analyses did not include longitudinal cross-lags as autistic traits were only measured at one timepoint, it is implausible that gender incongruence would increase subsequent likelihood for autism. Therefore, it is reasonable to assume that autism is also a prospective predictor of gender incongruence in adolescent females, even after controlling for age and other possible psychosocial confounds.

Taken together, this study provides one of the most comprehensive longitudinal examinations to date of biopsychosocial correlates of adolescent gender incongruence. However, several limitations should be acknowledged when interpreting these findings. This study relied on self-reported measures of gender incongruence rather than clinical diagnoses of gender

dysphoria, which limits generalizability to clinical populations and theories tested here, including ROGD, which has been criticized (Ashley, 2020; Restar, 2020; Turban et al., 2023). Relatedly, while the longitudinal design permits stronger inferences about directionality than cross-sectional studies, effect sizes for the significant cross-lagged associations were small, and many hypothesized prospective pathways were null even where concurrent associations were present at every timepoint. This suggests that although psychosocial and physiological predictors may contribute to the onset or maintenance of gender incongruence, they likely operate in combination with unmeasured individual factors. Additionally, while gender identity was included as a control variable in all analyses, it is unclear whether effects may be driven entirely by transgender or nonbinary youth in the sample, or whether the two groups differ significantly in psychological profiles. Chapter 4 will address this question directly, with the addition of cluster analyses to detect whether there are diverse sub-populations of adolescents experiencing gender incongruence via distinct mechanisms, a process of equifinality (Cicchetti & Rogosch, 1996).

IV. Gender Persistence, Desistance, and Psychological Profiles of Transgender and Nonbinary Adolescents

According to the typology discussed in the General Introduction, gender dysphoria has at least three distinct manifestations. The first, classical gender dysphoria, begins in early childhood, is intuitively understood as being "trapped in the wrong body," is associated with homosexuality, and may be driven by genetic and hormonal factors influencing early sexual differentiation of the brain, perhaps exacerbated by social reinforcement of gender nonconformity beginning in early childhood (Bailey & K. Zucker, 1995; Bailey et al., 2016). The second, a byproduct of autoerotic paraphilia, emerges after puberty, particularly in heterosexual males, and has unknown cause (Blanchard, 1989; Lawrence, 2010). The third, rapid-onset gender dysphoria (ROGD), is hypothesized to occur during adolescence and affect predominantly females with risk factors including internalizing symptoms, autism, body image issues, and social exclusion (Littman, 2018).

Reported rates of gender persistence and desistance (i.e., whether and for how long people with gender incongruence remain transgender- or nonbinary-identified) among gender-variant persons vary substantially (Byrne, 2024; Temple Newhook et al., 2018; K. Zucker, 2018). Historically, for children diagnosed with gender dysphoria followed into adulthood, persistence rates have been low (< 12%) and desistance rates have been high (> 88%), with the majority of desisters coming to identify as cisgender homosexual adults (Bailey & K. Zucker, 1995; Byrne, 2024; Drummond et al., 2008; Singh et al., 2021). However, the majority of these studies occurred during a time where gender transition prior to puberty was virtually unheard of. Other studies using more recently collected data collected from youth who had socially transitioned genders prior to puberty have shown high rates (82-97%) of longitudinal persistence

in transgender or nonbinary identities into adolescence (deMayo et al., 2025; Olson et al., 2022). Moreover, the largest study of its kind, the 2015 U.S. Transgender Survey (James et al., 2016) showed a low detransition rate of 8% in a sample of over 27,000 transgender adults, indicating 92% persistence. (However, because this study collected data primarily through advertising online to transgender-affirming communities, individuals who had detransitioned may have been underrepresented.)

These seemingly inconsistent findings may all be reconciled purely within a framework of classical gender dysphoria. It may be the case that gender dysphoria persistence is high across childhood up until sexual maturation, accounting for the high persistence rates within the contemporary TransYouth samples (deMayo et al., 2025; Olson et al., 2022), and desistance rates may rise if a majority of previously gender-dysphoric youth reconcile their identities with homosexuality, as predicted by Bailey and K. Zucker (1995). It may also be the case that, of the minority of individuals who do persist with gender incongruence past puberty, persistence rates are then extremely stable across adulthood, accounting for findings from the U.S. Transgender Survey (James et al., 2016).

However, the U.S. Transgender Survey also reported that 16% of transgender adults were exclusively same-gender attracted (i.e., heterosexual with respect to their biological sex), and another 53% identified as bisexual, pansexual, or queer. Only 15% identified as straight (i.e., homosexual with respect to their biological sex), a much lower proportion than the homosexual theory of gender dysphoria would predict. Moreover, there was a large sex difference in same-gender attractedness: 27% of males (transgender women) were exclusively attracted to women, while only 12% of females (transgender men) were exclusively attracted to men. These results are better understood in the context of autoerotic theories of gender incongruence, which posits

that a large proportion of heterosexual males with gender dysphoria are autogynephilic (i.e., attracted to oneself as a woman; Blanchard, 1991; Freund & Blanchard, 1993). While this theory has been critiqued (e.g., Serano, 2010), there are reports of self-professed autogynephiles living as transgender women (Lawrence, 2009, 2010). Likewise, a complimentary theory of autoandrophilia (attraction to oneself as a man) has been proposed and self-adopted by some transgender men, though it is thought to be far less common (Dickey & Stephens, 1995; Illy, 2025; Lawrence, 2010).

A combination of classical and autoerotic theories of gender dysphoria may explain high persistence among transgender adults and prepubescent children, but cannot explain the large recent surge in gender dysphoria referrals among adolescents, particularly adolescent females (Cass, 2024; K. Zucker, 2017). In fact, this trend is exactly opposite what the classical binary typology of gender dysphoria would predict. The homosexual subtype of gender dysphoria is characterized by decreases in gender dysphoria across adolescence, rather than increases, except for the minority of cases who go on to become transgender adults (Bailey & K. Zucker, 1995). And, while autoerotic subtypes would predict increases in gender dysphoria around sexual maturation, they should be primarily concentrated in males rather than females, as autogynephilia is more common than autoandrophilia (Lawrence, 2010).

The theory of "rapid onset gender dysphoria" (ROGD; Littman, 2018) proposes to explain recent large increases in prevalence of adolescent-onset gender incongruence. Specifically, ROGD builds on a literature of other forms of body dysmorphia and internalizing psychopathology which adolescent females are particularly vulnerable to, such as eating disorders (Rohde et al., 2015). Eating disorders are known to be prone to "social contagion," or peer influence and media risk-factors, especially in adolescent girls (Allison et al., 2014;

Eisenberg & Neumark-Sztainer, 2010; Paxton et al., 1999). Moreover, other factors impacting body image in adolescents, such as early pubertal timing and high body mass index, have been shown to influence risk for internalizing psychopathology, particularly in females (Kaplowitz, 2008; Milano et al., 2020; Pfeifer & Allen, 2021; Ullsperger & Nikolas, 2017). Littman (2018) and others building on the theory of ROGD (e.g., Clayton, 2023) hypothesize that these pre-existing gendered risk-factors for body dysmorphia in a vulnerable population of youth susceptible to social contagion culminate in a "perfect storm" of gender dysphoria in girls not otherwise exhibiting strong gender incongruence prior to puberty.

Findings from extant literature on gender incongruence and original results from Chapters 1-3 are compatible with a typology of multiple different manifestations of gender incongruence. However, this typology has been criticized as overly simplistic, not diagnostically valid or medically useful, and unable to explain their lived experiences of transgender people (Turban, 2024; Turban et al., 2023), though some transgender scholars embrace such typologies (e.g., Lawrence, 2010).

Nevertheless, while this three-class typology promises to explain different manifestations of gender incongruence, with each theory supporting empirical evidence that the others cannot address without contradiction, the typology itself has never been empirically validated. How could this be done? Recent work in developmental science has validated tools for identifying distinct clusters of individuals with unique psychological profiles in a data-driven fashion using finite mixture modeling (Grisanzio et al., 2025). Finite mixture modeling identifies joint distributions between variables in the same model to identify latent clusters of individuals with similar data profiles, in this case gender incongruence subtypes.

This chapter therefore has four aims. First, I will examine longitudinal gender persistence and desistance rates within the transgender or nonbinary (TNB) subsample from the Adolescent Brain Cognitive Development (ABCD) Study, and examine sex and demographic differences in these trajectories. Second, I will examine how biopsychosocial correlates of gender incongruence discussed in Chapter 3 predict longitudinal persistence and desistance rates separately for transgender and nonbinary adolescents. Third, I will test a three-cluster finite mixture modeling solution to relationships between gender incongruence and these biopsychosocial and demographic characteristics within the TNB subsample, and evaluate whether the triune typology of gender incongruence discussed earlier can be documented empirically. If the typology can be discovered from the data, I expect to identify three latent classes: a classic subtype, with a higher likelihood of child-onset gender incongruence, and higher likelihood of homosexuality; an autogynephilic subtype, with a male-skewed sex ratio, higher likelihood of gender incongruence manifesting after puberty, and a higher likelihood of heterosexuality; and an ROGD subtype, with a female-skewed sex ratio, higher likelihood of gender incongruence after puberty than before puberty, and higher likelihood of internalizing psychopathology. Lastly, I will examine whether the finite mixture-identified clusters predict gender persistence or desistance likelihood, noting that even if the triune typology is not recoverable, latent classes with different psychological profiles may still differ in their gender persistence likelihood.

Method

Participants

The participants are the subsample of 670 TNB youths (ages 8-15, 80.1% female, 34.6% transgender) described in Chapter 1. All five waves of data were used, for a total of 2,944

observations. As participants were classified as TNB if they identified as transgender or nonbinary at any timepoint, this may include earlier waves of participants identifying as cisgender prior to transition or participants identifying as cisgender following desistance.

Gender Transition, Persistence, and Desistance

Gender transition is operationalized as any participant identifying as cisgender at an earlier timepoint coming to identify as transgender or nonbinary at a later timepoint. Desistance is operationalized as any participant identifying as transgender or nonbinary at an earlier timepoint and coming to identify as cisgender at a later timepoint. Persistence is operationalized as any participant identifying as transgender or nonbinary across two or more consecutive timepoints. For the purposes of this study, gender transition from transgender to nonbinary, or nonbinary to transgender, will both be counted as persistence.

Each timepoint from Waves 2-5 will have an individual score of Transitioned, Persisted, or Desisted. Wave 1 will be missing for all participants since there is no previous reference gender. Every other timepoint will be scored in reference to the previous timepoint. Participants persisting as cisgender for two or more timepoints in a row will be treated as the reference category. For example, an individual identifying as cisgender at Waves 1 and 2, nonbinary at Wave 3, transgender at Wave 4, and cisgender at Wave 5, will have missing values for Waves 1 and score Cisgender (reference) at Wave 2, Transitioned at Wave 3, Persisted at Wave 4, and Desisted at Wave 5.

Predictor Variables

The demographic variables are the same as those described in Chapter 1: age, sex, gender identity, race/ethnicity, household income, parent education. Age was centered at age 9 for interpretability. For the purposes of this study, the TNB variable refers to a binary "Transgender" or "Nonbinary" variable (as opposed to binary "TNB" or "Cisgender" in previous chapters) to identify possible group differences within the TNB sample.

The self-reported gender incongruence (i.e., the subclinical measure of gender dysphoria) and gender nonconformity (i.e., the personality measure of gender expression reverse-coded by sex) measures are the same as those described in Chapter 1. The biopsychosocial predictor variables are the same as those described in Chapter 3: internalizing symptoms, autism spectrum traits, sexual orientation, pubertal timing, body mass index, screen time, cyberbullying, peer victimization, family conflict, and number of TNB friends.

Multinomial Logistic Regression

Bayesian mixed-effects multinomial logistic regression was used to predict the likelihood of each level (Transitioned, Persisted, or Desisted) of the categorical gender tracking variable with respect to the reference Cisgender category. Models were specified in a lagged format, such that predictors from Waves 1-4 were aligned with outcomes from the immediately subsequent Waves 2-5. In other words, for all pairs of two consecutive timepoints, how do biopsychosocial factors at the earlier timepoint predict gender transition, persistence, or desistance at the later timepoint? Random effects were used to account for repeated measures within participants across longitudinal waves and to capture the nested structure of the data, with subject ID nested within

household ID to account for shared variance between siblings recruited from the same household.

Sex and gender interaction effects were not included because preliminary models showed no significant evidence of such interactions and their inclusion substantially increased model complexity and uncertainty. The final model was structured as follows:

Gender Change ~ Sex + TNB + Age + Internalizing + Sexual Orientation + Autism + Pubertal Timing + BMI + Family Conflict + Victimization + Cyberbullying + Screen Time + TNB Friends + Race + Income + Education + (1 | Family ID/Subject ID)

Cluster Analyses

Lastly, finite mixture modeling was performed using the *"flexmix"* package (Leisch, 2004). Finite mixture regression identifies joint distributions between variables in the same model to identify latent clusters of individuals with similar data profiles (Grisanzio et al., 2025). The outcome variable was the continuous measure of self-reported gender incongruence, with $k = 3$ to identify three latent clusters of gender incongruence scores conditional on covariates of interest, including gender nonconformity. The model was specified as follows:

Gender Incongruence ~ Age + Sex + TNB + Gender Nonconformity + Sexual Orientation + Internalizing + Pubertal Timing + BMI + Screen Time + Cyberbullying + Victimization + Family Conflict + TNB Friends + Autism | (Subject ID)

Subject ID was included as a random effect to account for repeated measurements across longitudinal timepoints. Racial and socioeconomic demographic covariates were not included as these were not considered relevant to the hypothesized latent subtypes of gender incongruence.

Missing Data and Multiple Imputation

As in Chapter 3, missing data of biopsychosocial predictor variables were imputed using multiple imputation with chain equations using the "MICE" package in R (van Buuren & Groothuis-Oudshoorn, 2011). Gender incongruence was not imputed if missing. Additionally, demographic variables were not imputed if missing, including gender identity and sexual orientation. Missingness rates across all timepoints where the relevant variable was measured were: gender incongruence 2.5%, internalizing symptoms 1.3%, sexual orientation 9.8%, autism spectrum traits 0.3%, pubertal timing 1.2%, body mass index 24.3%, family conflict 1.3%, peer victimization 0.4%, cyberbullying 0.9%, screen time 0.3%, number of TNB friends 50.7%. Even the relatively high missingness rates for sexual orientation, BMI, and number of TNB friends had enough complete observations which demonstrated strong enough covariance with other complete subject-level data for MICE assumptions to be met. MICE generated 20 multiply imputed datasets which stably converged within 10 iterations each. All analyses were conducted on these 20 imputed datasets, and all estimates presented are pooled results across all 20 sets of estimates. Each imputed dataset had 2,944 observations from 670 unique participants.

Results

Longitudinal Gender Stability

Figure 1 depicts longitudinal change scores of gender identity between Waves 1-2, 2-3, 3-4, and 4-5. Specific counts, by wave and sex, are shown in Table 1. Though all participants in the TNB subsample (N = 670) identified as TNB during at least one timepoint, results show that

the majority of participants identified as cisgender across Waves 1-2 and Waves 2-3, and a majority of transitions occurred during Waves 3-4 or Waves 4-5.

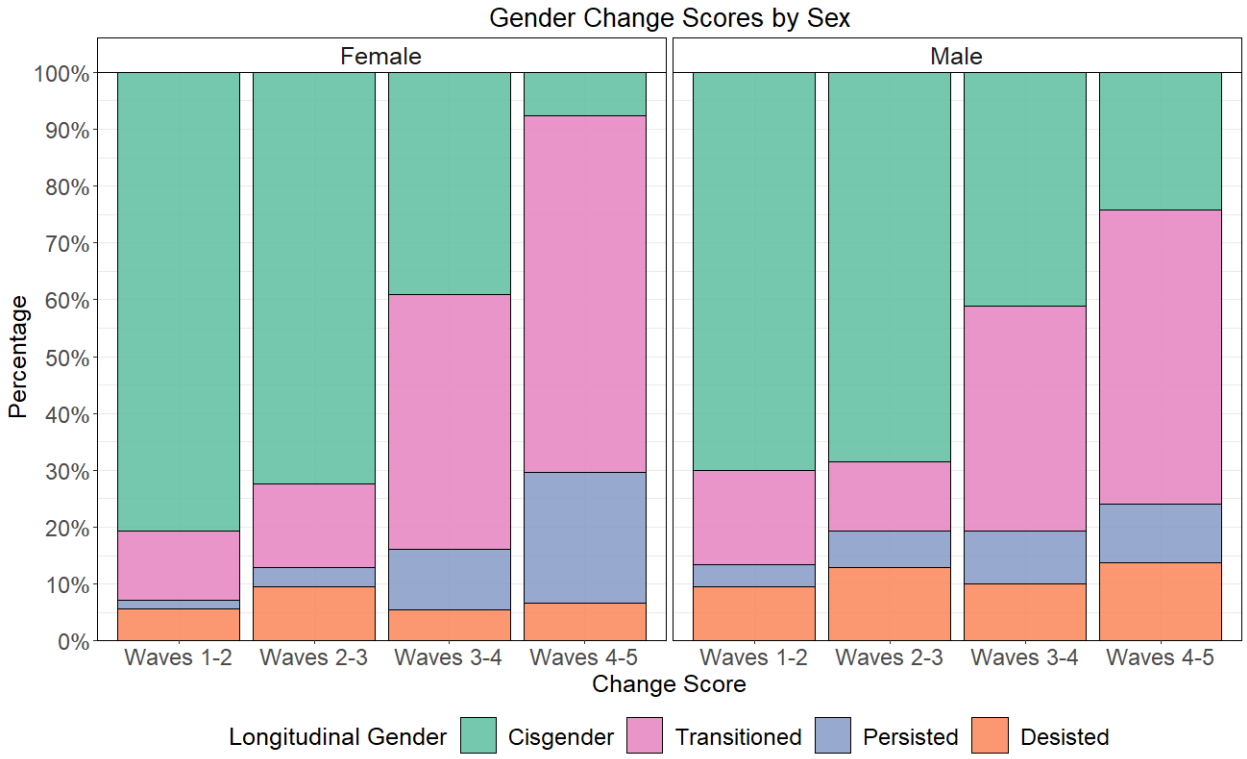


Figure 1. Longitudinal Gender Stability Among TNB Youth by Sex and Timepoints. All participants (N = 670) identified as TNB during at least one timepoint.

Table 1. Longitudinal Gender Stability Among TNB Youth by Sex and Timepoint. All participants (N = 670) identified as TNB during at least one timepoint.

Data Wave	**Waves 1-2**	**Waves 2-3**	**Waves 3-4**	**Waves 4-5**
Males (N = 133)				
Cisgender	89 (70.1%)	85 (68.5%)	53 (41.1%)	14 (24.1%)
Transitioned	21 (16.5%)	15 (12.1%)	51 (39.5%)	30 (51.7%)

Persisted	5 (3.9%)	8 (6.5%)	12 (9.3%)	6 (10.3%)
Desisted	12 (9.4%)	16 (12.9%)	13 (10.1%)	8 (13.8%)
Females (N = 537)				
Cisgender	425 (80.6%)	381 (72.4%)	199 (39.1%)	21 (7.7%)
Transitioned	64 (12.1%)	77 (14.6%)	228 (44.8%)	172 (62.8%)
Persisted	8 (1.5%)	18 (3.4%)	54 (10.6%)	63 (23.0%)
Desisted	30 (5.7%)	50 (9.5%)	28 (5.5%)	18 (6.6%)

Gender persistence and desistance rates, by wave and sex, are shown in Table 2. These counts are identical to those presented in Table 1, but their proportions and interpretations differ. Table 1 documents all gender change scores across all participants within the full sample of 670 participants who ever identified as TNB, whereas Table 2 documents persistence and desistance rates for the 272 participants who had at least one longitudinal observation following a TNB observation. The smaller sample is a result of the fact that 398 participants (59.4%) identified as TNB only at their final observation and had no persistence or desistance follow-up. These results are visualized in Figure 2.

Figure 2. Gender Persistence and Desistance Among TNB Youth by Sex and Timepoints. All participants began as TNB (N = 272, 61 male, 211 female) and had at least one wave of longitudinal follow-up.

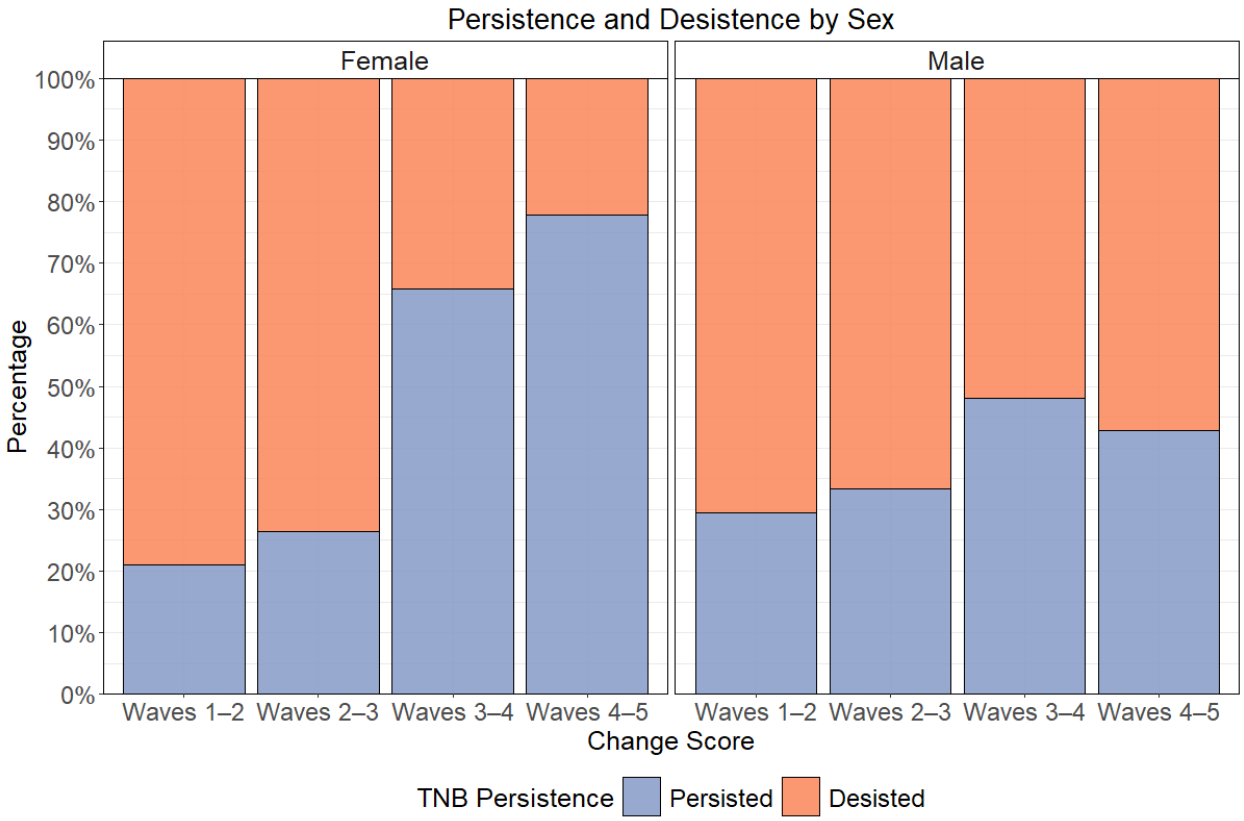


Table 2. Gender Persistence and Desistance by Sex and Timepoint

Data Wave	Waves 1-2	Waves 2-3	Waves 3-4	Waves 4-5	Full Study*
Full Sample (N = 272)					
Persisted	13 (23.6%)	26 (28.3%)	66 (61.7%)	69 (72.6%)	101 (37.1%)
Desisted	42 (76.4%)	66 (71.7%)	41 (38.3%)	26 (27.4%)	171 (62.9%)
Males (N = 61)					
Persisted	5 (29.4%)	8 (33.3%)	12 (48.0%)	6 (42.9%)	13 (21.3%)
Desisted	12 (70.6%)	16 (66.7%)	13 (52.0%)	8 (57.1%)	48 (78.7%)
Females (N = 211)					
Persisted	8 (21.1%)	18 (26.5%)	54 (65.9%)	63 (77.8%)	88 (41.7%)
Desisted	30 (78.9%)	50 (73.5%)	28 (34.1%)	18 (22.2%)	123 (58.3%)

**Full study counts are the number of unique participants with at least one wave of longitudinal follow-up following a TNB identity who desisted at least once. Column sums exceed these counts because participants with multiple longitudinal waves are included in multiple columns.*

Table 2 documents desisters as those who ever reported a cisgender identity following a TNB identity, which may underestimate unstable persistence (e.g., TNB-cisgender-TNB) and overestimate desistance. While 171 of participants desisted at least once, 46 of these (4 male, 42 female) later reverted to a TNB identity. Therefore, another way of interpreting the data is that 37% of the sample were stable persisters, 17% were unstable persisters or temporary desisters, and 46% were stable desisters. In this framing, 21% of males were stable persisters, 7% of males were unstable persisters or temporary desisters, and 72% of males were stable desisters, while 42% of females were stable persisters, 20% of females were unstable persisters or temporary desisters, and 38% of females were stable desisters.

Correlations

Figure 3 shows correlations between self-reported gender incongruence and all continuous predictor variables among TNB youth, by sex. All variables were correlated with gender incongruence in at least one sex, except for family conflict and peer victimization. The strongest associations with gender incongruence were age, pubertal timing, and internalizing symptoms in both sexes, with stronger associations in females.

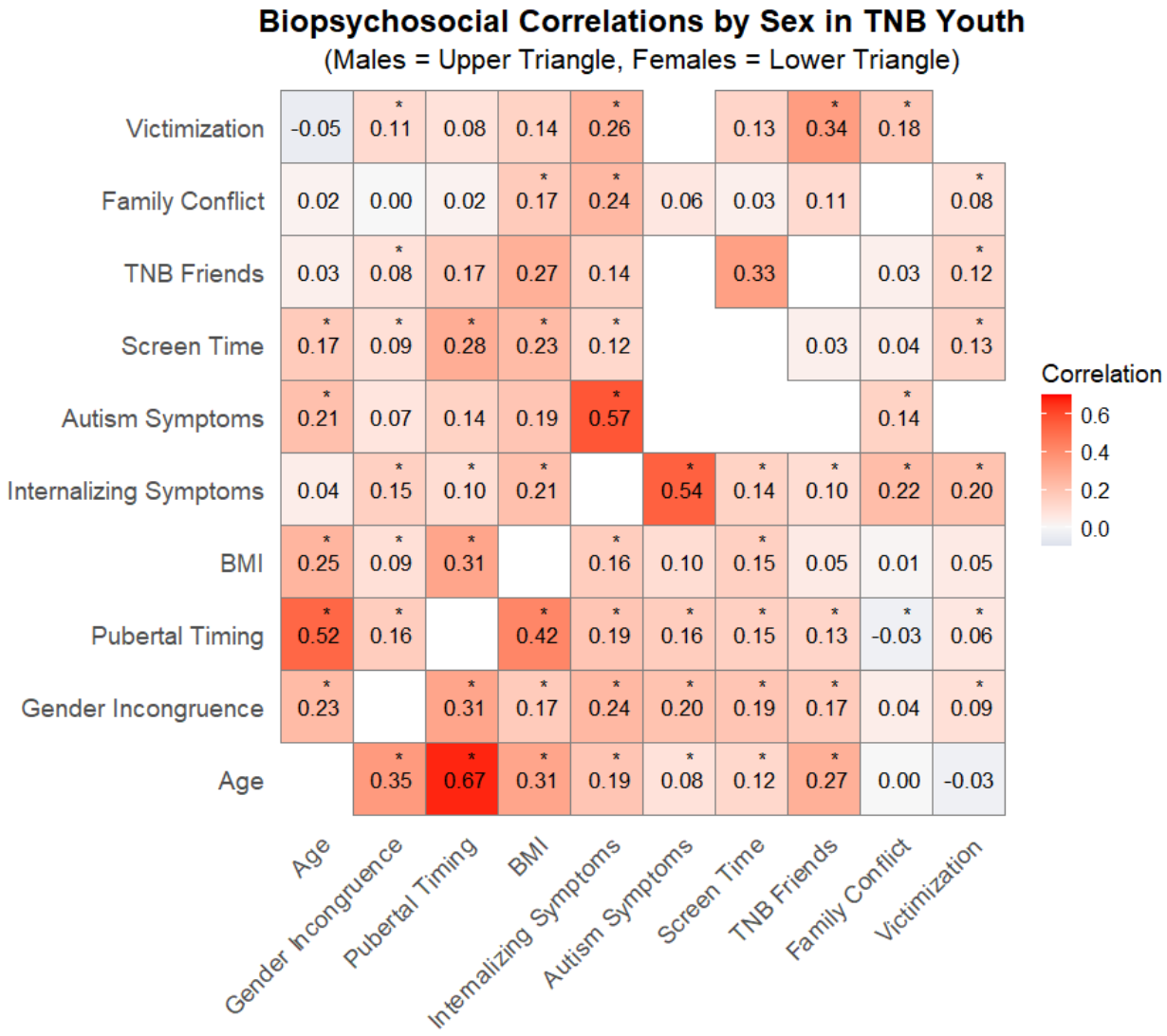


Figure 3. Correlations Between All Continuous Variables in the TNB Sample

Multinomial Logistic Regression

Multinomial logistic regression model results on gender persistence likelihood are summarized in Table 3. All continuous predictors were mean-centered for interpretability, except age which was centered at 9 years.

Cisgender persistence (i.e., two consecutive timepoints of a participant identifying as cisgender) was modeled as the reference category, or baseline against which all likelihoods were calculated. Because all participants identified as TNB during at least one timepoint, cisgender persistence refers to a baseline state before which participants "came out," or in rarer cases cisgender persistence after desisting in TNB identity.

Relative to cisgender persistence, the intercept identified a .09 times likelihood of gender transition (cisgender-TNB) at any given pair of timepoints. However, this increased by 2.56 times likelihood for each year of age beyond age 9. LGB sexual orientation was associated with 2.79 times greater likelihood of social gender transition, while cyberbullying experiences within the past year were associated with 0.48 times lower likelihood of transition. No other predictors were significantly associated with gender incongruence. As a matter of comparison, 9 year-old participants were .09 times less likely to transition than to persist as cisgender at age 10, while 11.5 year-old participants had roughly equal likelihood of cisgender persistence or gender transition at age 12.5, and nonheterosexual 14 year-old participants were 27.6 times more likely to transition than to persist as cisgender at age 15.

TNB persistence had a strong negative intercept corresponding to a low .002 times likelihood of nonbinary persistence relative to cisgender persistence. However, participants who came to identify as transgender as opposed to nonbinary had 1,380 times higher likelihood of persistence, corresponding to a 2.76 times higher likelihood of transgender persistence than cisgender persistence at any given pair of timepoints. These likelihoods of TNB persistence increased by 2.34 times per year increase in age beyond age 9, as well as a 1.90 times increase per Tanner Stage increase in earlier pubertal timing. Nonheterosexual participants had a 16.82 times higher likelihood of TNB persistence, while Black participants had 0.10 times lower

likelihood of TNB persistence as compared to cisgender persistence. No other predictors had significant associations with TNB persistence. As a matter of comparison, 9-year old nonbinary participants had a .002 times lower likelihood of TNB persistence at age 10, while 13-year old nonheterosexual nonbinary participants had roughly equal likelihood of nonbinary and cisgender persistence at age 14, and 13-year old transgender participants had 82.8 times higher likelihood of TNB persistence at age 14.

Desistance in TNB identity had 0.16 times lower odds than cisgender persistence, though baseline likelihoods did not differ significantly. Transgender as opposed to nonbinary identity was associated with 156.7 times higher likelihood of desistance, and LGB sexual orientation was associated with 14.43 times higher likelihood of desistance as compared to heterosexual identity. No other predictors, including age, were significantly associated with desistance likelihood. As a matter of comparison, nonheterosexual nonbinary participants were 2.31 times more likely to desist in their nonbinary identity than to persist as cisgender, while heterosexual transgender participants were 25.1 times more likely to desist in their transgender identity than to persist as cisgender between any two pairs of timepoints.

Table 3. Multinomial Logistic Regression on Gender Transition, Persistence, and Desistance

Transitioned			
Predictor	**B (SE)**	**95% CI**	**Odds Ratio**
Intercept	–2.36 (0.90)	[–4.13, –0.59]*	0.09 [0.02, 0.55]*
Age	0.94 (0.13)	[0.68, 1.20]*	2.56 [1.97, 3.32]*
Female	0.16 (0.30)	[–0.40, 0.84]	1.17 [0.67, 2.32]
Transgender	–2.32 (1.33)	[–5.15, 0.08]	0.10 [0.01, 1.08]
Black	–0.18 (0.40)	[–1.01, 0.57]	0.84 [0.36, 1.77]
Hispanic	0.15 (0.24)	[–0.34, 0.61]	1.16 [0.71, 1.84]
Asian	0.62 (0.61)	[–0.58, 1.83]	1.85 [0.56, 6.23]

Multiracial	–0.01 (0.30)	[–0.68, 0.54]	0.99 [0.51, 1.72]
Income < $50k	0.02 (0.27)	[–0.50, 0.57]	1.02 [0.61, 1.77]
Income > $100k	–0.11 (0.22)	[–0.52, 0.34]	0.90 [0.59, 1.41]
Parent High School	0.46 (0.51)	[–0.53, 1.47]	1.58 [0.59, 4.36]
Parent Some College	0.01 (0.46)	[–0.89, 0.93]	1.01 [0.41, 2.54]
Parent Bachelor's	–0.15 (0.48)	[–1.07, 0.79]	0.86 [0.34, 2.20]
Parent Graduate	0.18 (0.47)	[–0.72, 1.11]	1.20 [0.49, 3.03]
Internalizing	0.03 (0.02)	[–0.01, 0.07]	1.03 [0.99, 1.07]
LGB	1.03 (0.23)	[0.59, 1.48]*	2.79 [1.80, 4.39]*
Autism	–0.02 (0.04)	[–0.09, 0.06]	0.98 [0.91, 1.06]
Pubertal Timing	0.05 (0.10)	[–0.15, 0.24]	1.05 [0.86, 1.27]
BMI	0.02 (0.02)	[–0.03, 0.06]	1.02 [0.97, 1.06]
Family Conflict	–0.04 (0.04)	[–0.12, 0.05]	0.96 [0.89, 1.05]
Victimization	–0.13 (0.26)	[–0.63, 0.41]	0.88 [0.53, 1.51]
Cyberbullied	–0.74 (0.39)	[–1.55, –0.04]*	0.48 [0.21, 0.96]*
Screen Time	0.05 (0.03)	[–0.02, 0.12]	1.05 [0.98, 1.13]
TNB Friends	–0.06 (0.23)	[–0.42, 0.47]	0.94 [0.66, 1.60]
Persisted			
Predictor	**B (SE)**	**95% CI**	**Odds Ratio**
Intercept	–6.15 (1.84)	[–9.92, –2.71]*	0.002 [0.00, 0.07]*
Age	0.85 (0.26)	[0.36, 1.38]*	2.34 [1.43, 3.98]*
Female	–0.73 (0.61)	[–1.89, 0.50]	0.48 [0.15, 1.65]
Transgender	7.23 (0.95)	[5.41, 9.15]*	1380 [224, 9400]*
Black	–2.32 (1.10)	[–4.62, –0.29]*	0.10 [0.01, 0.75]*
Hispanic	0.80 (0.53)	[–0.24, 1.87]	2.23 [0.79, 6.48]
Asian	0.21 (1.35)	[–2.56, 2.73]	1.23 [0.08, 15.36]
Multiracial	0.01 (0.60)	[–1.22, 1.15]	1.01 [0.29, 3.16]
Income < $50k	0.64 (0.55)	[–0.42, 1.73]	1.90 [0.65, 5.64]
Income > $100k	0.25 (0.48)	[–0.69, 1.20]	1.28 [0.50, 3.32]
Parent High School	0.25 (0.96)	[–1.65, 2.15]	1.28 [0.19, 8.60]

Parent Some College	0.39 (0.88)	[–1.31, 2.14]	1.48 [0.27, 8.50]
Parent Bachelor's	–0.38 (0.91)	[–2.14, 1.42]	0.68 [0.12, 4.14]
Parent Graduate	0.75 (0.88)	[–0.94, 2.50]	2.12 [0.39, 12.18]
Internalizing	0.08 (0.04)	[–0.00, 0.15]	1.08 [1.00, 1.16]
LGB	2.82 (0.45)	[1.97, 3.76]*	16.82 [7.19, 42.77]*
Autism	0.02 (0.08)	[–0.13, 0.18]	1.02 [0.88, 1.20]
Pubertal Timing	0.64 (0.24)	[0.18, 1.13]*	1.90 [1.20, 3.10]*
BMI	–0.09 (0.05)	[–0.19, 0.00]	0.91 [0.83, 1.00]
Family Conflict	–0.14 (0.10)	[–0.35, 0.06]	0.87 [0.71, 1.06]
Victimization	–0.10 (0.46)	[–0.93, 0.85]	0.91 [0.39, 2.34]
Cyberbullied	–1.04 (0.80)	[–2.57, 0.56]	0.35 [0.08, 1.75]
Screen Time	0.07 (0.06)	[–0.05, 0.20]	1.07 [0.95, 1.22]
TNB Friends	–0.09 (0.21)	[–0.65, 0.24]	0.91 [0.52, 1.27]
Desisted			
Predictor	**B (SE)**	**95% CI**	**Odds Ratio**
Intercept	–1.85 (1.85)	[–5.72, 1.85]	0.16 [0.00, 6.35]
Age	–0.14 (0.22)	[–0.59, 0.27]	0.87 [0.55, 1.31]
Female	–0.57 (0.50)	[–1.51, 0.49]	0.57 [0.22, 1.64]
Transgender	5.05 (1.27)	[3.15, 8.52]*	156.7 [23.3, 5030]*
Black	–0.37 (0.73)	[–1.93, 0.98]	0.69 [0.15, 2.66]
Hispanic	0.05 (0.43)	[–0.81, 0.88]	1.05 [0.44, 2.42]
Asian	–1.53 (1.46)	[–4.63, 1.06]	0.22 [0.01, 2.90]
Multiracial	0.07 (0.48)	[–0.96, 0.97]	1.07 [0.38, 2.64]
Income < $50k	0.58 (0.55)	[–0.38, 1.87]	1.79 [0.68, 6.49]
Income > $100k	0.25 (0.39)	[–0.50, 1.04]	1.28 [0.61, 2.83]
Parent High School	0.10 (0.84)	[–1.56, 1.74]	1.11 [0.21, 5.71]
Parent Some College	–0.24 (0.75)	[–1.68, 1.28]	0.79 [0.19, 3.60]
Parent Bachelor's	–0.48 (0.81)	[–2.02, 1.16]	0.62 [0.13, 3.19]
Parent Graduate	–0.19 (0.75)	[–1.64, 1.32]	0.83 [0.20, 3.74]
Internalizing	0.00 (0.03)	[–0.06, 0.06]	1.00 [0.94, 1.06]

LGB	2.67 (0.42)	[1.92, 3.55]*	14.43 [6.82, 34.9]*
Autism	0.09 (0.06)	[–0.04, 0.23]	1.09 [0.96, 1.26]
Pubertal Timing	–0.15 (0.18)	[–0.52, 0.20]	0.86 [0.59, 1.22]
BMI	–0.02 (0.05)	[–0.12, 0.10]	0.98 [0.89, 1.11]
Family Conflict	–0.04 (0.08)	[–0.20, 0.11]	0.96 [0.82, 1.12]
Victimization	0.26 (0.58)	[–0.68, 1.74]	1.30 [0.51, 5.71]
Cyberbullied	–0.92 (1.08)	[–2.92, 2.59]	0.40 [0.05, 13.4]
Screen Time	0.05 (0.06)	[–0.07, 0.15]	1.05 [0.93, 1.16]
TNB Friends	–0.26 (0.75)	[–3.24, 0.51]	0.77 [0.04, 1.66]

Cluster Analyses

Flexmix analysis identified a three-cluster solution that captured distinct profiles of predictors associated with gender incongruence. Class-specific standardized regression coefficients on gender incongruence (cluster loadings) for all predictors of interest are summarized in Table 4. Cluster 1 was the smallest class with 128 individuals (20% of sample) and had weak to moderate loadings across all predictors of interest. Cluster 2 was the next largest class with 236 individuals (38%) and had distinct weak to moderate loadings across all predictors. Cluster 3 was the largest with 264 individuals (42%) and was almost entirely characterized by gender incongruence intercept values and gender identity, with miniscule loadings for all other predictors.

Table 4. Flexmix Cluster Loadings

Predictor	**Cluster 1**	**Cluster 2**	**Cluster 3**
Intercept	–0.293	0.647	–0.843
Age	< .001	0.026	< .001

Female	–0.066	–0.036	< .001
LGB	–0.228	–0.357	< .001
Gender Nonconformity	0.009	0.037	< .001
Internalizing	0.017	–0.001	< .001
Pubertal Timing	0.029	0.033	< .001
BMI	0.007	0.044	< .001
Screen Time	–0.013	0.021	< .001
Cyberbullied	–0.030	0.172	< .001
Victimization	0.019	0.033	< .001
Family Conflict	–0.025	0.025	< .001
TNB Friends	0.008	0.015	< .001
Autism	0.017	0.020	< .001
Transgender	0.514	1.631	4.085
Residual σ	0.354	0.860	< .001

Pooled and cluster-specific means and mean differences for all continuous measures are visualized in Figure 4 and summarized in Table 5. Cluster differences in categorical gender identity, sex, sexual orientation, and cyberbullying experiences are summarized in Table 6.

Class profiles revealed that Cluster 1 had the lowest mean gender incongruence, was exclusively characterized by nonbinary-identified participants, had the highest proportion of males across clusters, and the lowest proportion of nonheterosexuals. Cluster 1 participants additionally had lower internalizing symptoms, lower gender nonconformity, lower screen time and peer victimization, lower body mass index and puberty scores, and fewer TNB friends.

Cluster 2 participants had moderate gender incongruence and moderate scores in between

Clusters 1 and 3 for all predictors of interest except autistic traits, where their scores were lowest. Cluster 3, by contrast, had the highest mean levels of gender incongruence and all predictors of interest, and had the highest proportions of females, transgender identification, and LGB sexual orientation across clusters. Clusters did not significantly differ from each other in age, family conflict, or cyberbullying experiences.

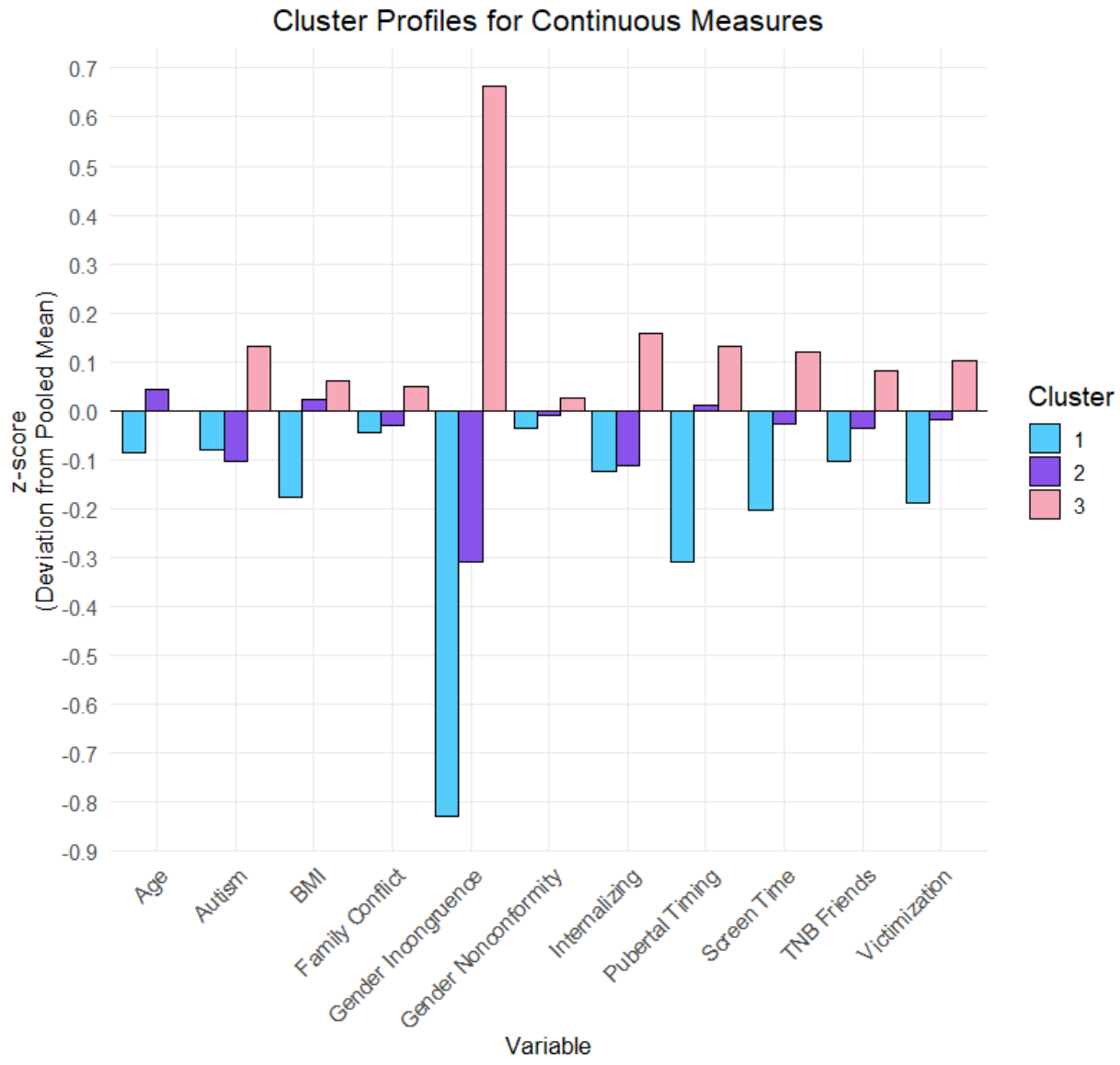


Figure 4. Cluster Profiles for All Continuous Measures

Table 5. Cluster Profiles for All Continuous Measures

Variable	Pooled Mean (SD)	Cluster 1	Cluster 2	Cluster 3	Cluster Differences
Gender Incongruence	1.83 (0.98)	1.01 (z = -0.83)	1.52 (z = -0.31)	2.48 (z = 0.66)	$F(2, 625) = 524.31$ $p < .001$***
Age	11.74 (0.98)	11.65 (z = -0.08)	11.78 (z = 0.04)	11.74 (z = 0.00)	$F(2, 625) = 1.27$ $p = 0.311$
Gender Nonconformity	3.52 (1.56)	3.47 (z = -0.03)	3.50 (z = -0.01)	3.56 (z = 0.03)	$F(2, 625) = 3.66$ $p = 0.036$*
Internalizing	7.28 (7.17)	6.41 (z = -0.12)	6.49 (z = -0.11)	8.42 (z = 0.16)	$F(2, 625) = 6.01$ $p = 0.004$**
PDSS	3.08 (1.13)	2.73 (z = -0.31)	3.09 (z = 0.01)	3.22 (z = 0.13)	$F(2, 625) = 11.49$ $p < .001$***
BMI	20.94 (4.81)	20.08 (z = -0.18)	21.05 (z = 0.02)	21.23 (z = 0.06)	$F(2, 625) = 3.43$ $p = 0.040$*
Screen Time	4.53 (3.29)	3.87 (z = -0.20)	4.44 (z = -0.03)	4.92 (z = 0.12)	$F(2, 625) = 11.20$ $p < .001$***
Victimization	1.56 (0.58)	1.45 (z = -0.19)	1.55 (z = -0.02)	1.62 (z = 0.10)	$F(2, 625) = 8.68$ $p < .001$***
Family Conflict	2.30 (1.94)	2.22 (z = -0.04)	2.24 (z = -0.03)	2.40 (z = 0.05)	$F(2, 625) = 0.67$ $p = 0.510$
TNB Friends	2.65 (4.23)	2.21 (z = -0.10)	2.49 (z = -0.04)	2.99 (z = 0.08)	$F(2, 625) = 32.73$ $p < .001$***
Autism	16.08 (5.26)	15.67 (z = -0.08)	15.53 (z = -0.10)	16.78 (z = 0.13)	$F(2, 625) = 10.37$ $p < .001$***

Table 6 . Cluster Differences Across Categorical Measures.

Variable		Cluster 1	Cluster 2	Cluster 3	Cluster Differences
Sex	Male	39 (30.5%)	43 (18.2%)	38 (14.4%)	$\chi^2(2) = 24.99$ $p < .001$***
	Female	89 (69.5%)	193 (81.8%)	226 (85.6%)	
Sexual	LGB	17 (13.3%)	65 (27.5%)	127 (48.1%)	$\chi^2(2) = 138.2$

Orientation	Straight	111 (86.7%)	171 (72.5%)	137 (51.9%)	$p < .001$***
Gender Identity	Transgender	0 (0%)	5 (2.1%)	16 (6.1%)	$\chi^2(2) = 36.6$ $p < .001$***
	Nonbinary	128 (100%)	231 (97.9%)	248 (93.9%)	
Cyberbullied	Yes	16 (12.5%)	39 (16.5%)	61 (23.1%)	$\chi^2(2) = 4.59$ $p = 0.101$
	No	112 (87.5%)	197 (83.5%)	203 (76.9%)	

Lastly, Table 7 summarizes gender transition, persistence, and desistance rates across clusters. Clusters significantly differed from each other, with Cluster 1 being composed of primarily longitudinal cisgender persistence and no TNB persistence, with low likelihood of transitioning and desisting. Cluster 2 was also primarily composed of cisgender persistence but had higher rates of longitudinal transition and TNB persistence. Cluster 3 had the lowest rates of cisgender persistence and the highest rates of gender transition, desistance, and TNB persistence.

Table 7. Gender Transition, Persistence, and Desistance Likelihood by Cluster

Cluster	Cisgender	Transitioned	Persisted	Desisted	Cluster Differences
1	234 (90.3%)	11 (4.2%)	0 (0.0%)	14 (5.4%)	$\chi^2(6) = 164.7$ $p < .001$***
2	443 (85.5%)	38 (7.3%)	10 (1.9%)	27 (5.2%)	
3	325 (57.7%)	144 (25.6%)	42 (7.5%)	52 (9.2%)	

Discussion

This study examined longitudinal gender transition, persistence, and desistance rates among 670 ever-identifying transgender and nonbinary (TNB) adolescents within the Adolescent Brain Cognitive Development (ABCD) Study. Results showed that even among ever-identifying TNB adolescents, gender identity was highly variable over time, and that the vast majority of TNB adolescents identified as cisgender until ages 12-15 years. The overall desistance rate of

TNB identity was high, averaging 63% across the full study (79% in males, 58% in females). Males were more likely to desist than persist at every timepoint, but persistence rates rose with time. Females were more likely to desist at ages 9-12 years but more likely to persist at ages 12-15 years, with the highest persistence rates of 78% observed between the final two timepoints in 13-15 year old females.

Biopsychosocial correlates of gender incongruence, as shown in Chapter 3 and replicated here in the TNB subsample, largely did not predict gender change scores across longitudinal timepoints. The largest factors increasing longitudinal gender transition (cisgender-TNB) likelihood was older age, with each year in age increasing transition likelihood by 156%, reflecting the older average age of gender incongruence onset. Lesbian, gay, and bisexual (LGB) sexual orientation was also associated with 179% increased likelihood of gender transition between any given successive pairs of timepoints. This reflects the consistent finding that sexual minorities are disproportionately likely to experience gender dysphoria, or alternatively that gender incongruence is a strong predictor of future sexual orientation (Bailey & K. Zucker, 1995; deMayo et al., 2025). Experiencing cyberbullying in the past year, by contrast, decreased likelihood of gender transition at the subsequent timepoint by 52%. Given that the entire sample is comprised of adolescents who came to identify as TNB at least once, decreased likelihood of transition is likely explained by remaining "in the closet" at earlier timepoints, as gender and sexual minority youth are known to experience high levels of stigma and bullying which may keep them from expressing their identities openly until older ages (Xu et al., 2024; Xu & Rahman, 2025). However, from the measures collected it is not clear whether cyberbullied youth were being bullied about their gender or other factors.

Rates of TNB persistence were much lower than has been reported in contemporary samples of youth with gender dysphoria (e.g., deMayo et al., 2025; Olson et al., 2022), but this is expected given that the majority of this TNB sample identified as nonbinary and may not have undergone a formal "gender transition" as is typically understood for transgender individuals. Subclinical gender incongruence, and nonbinary gender identities in particular, are thought to be markers of gender fluidity which may represent normative adolescent identity exploration (L. Diamond, 2020). This is exemplified by the fact that transgender as opposed to nonbinary youth had a 1,380 times higher likelihood of longitudinal persistence in their TNB identity. Additionally, maturation was associated with increased likelihood of persistence, with each year increase in age associated with a 134% increased likelihood of persisting as TNB and each Tanner Stage increase in pubertal stage, after controlling for age, associated with 90% increased likelihood of TNB persistence. This likely indicates that participants who identified as nonbinary at earlier ages or prior to pubertal maturation were more likely to desist in their nonbinary identities, whereas youth who came out as transgender earlier were most likely to persist and more likely to experience strong gender incongruence.

The strongest effect on TNB persistence, other than transgender as opposed to nonbinary identity, was LGB sexual orientation. LGB participants were 1,582% more likely to persist in TNB identity at the subsequent timepoint, which stands in contrast to the historical finding that homosexual orientation is strongly associated with remission of gender dysphoria once a person's felt gender incongruence is assimilated into their sexual identity (Bailey & K. Zucker, 1995; Byrne, 2024). However, LGB sexual orientation was also associated with 1,343% increased likelihood in longitudinal gender desistance, supporting the theory. Because this study lacked granularity on sexual orientation, it is possible that the positive effect is driven primarily

by high likelihood of bisexuality among TNB youth, as sexual and gender fluidity often covary (L. Diamond, 2020), while homosexuality may be associated with decreases in gender incongruence.

The only other effect predicting TNB desistance likelihood was transgender identity, which was associated with 156 times increased likelihood of desistance as opposed to nonbinary participants. While transgender identity is generally thought to reflect a stronger form of gender incongruence that is more stable than nonbinary identity (Turban, 2024), it is possible that these effects are confounded by amount of time spent living as TNB. While age did not significantly predict desistance likelihood, given that most nonbinary participants came out at the final study timepoint (ages 13-15 years), it is possible that this led to an underestimation of nonbinary desistance and a relative overestimation of transgender desistance. Future ABCD data will be able to address these questions directly with longitudinal follow-up on the 398 out of 670 (59%) of participants who came out as TNB during their most recent timepoint.

Strong effects of gender and sexual orientation on gender persistence likelihood suggests that discussion of gender incongruence as a unitary developmental phenomenon is misleading. As discussed in the Introduction, attempts at a typology of the diverse manifestations of gender incongruence has identified at least three subtypes relevant to gender dysphoria. The first, classic gender dysphoria, is argued to be driven by genetic and hormonal factors influencing early sexual differentiation of the brain, manifests during early childhood and often remits after puberty as a homosexual identity develops, if gender transition is not possible prior to puberty (Bailey & K. Zucker, 1995; Bailey et al., 2016). However, the minority of individuals who persist in their gender incongruence past puberty often transition and live stable lives as transgender adults, with high rates of persistence (Bailey, 2003; James et al., 2016). The second

hypothesized forms of gender incongruence, autogynephilia or autoandrophilia, are related to "erotic target location error," or attraction towards oneself in a particular gender role, which seek to explain relatively high rates of heterosexual orientation (with respect to natal sex) in transgender adults (Blanchard, 1991; Freund & Blanchard, 1993; Lawrence, 2009, 2010). The third, "rapid-onset gender dysphoria" (ROGD; Littman, 2018) is hypothesized to explain recent increases in adolescent-onset prevalence (U.S. Department of Health and Human Services [HHS], 2025), particularly in females at risk for internalizing psychopathology.

The second part of this study empirically tested triune typology of gender incongruence for the first time, using finite mixture regression to identify three latent subpopulations of TNB youth (Grisanzio et al., 2025; Leisch, 2004). A three-cluster solution was identified with unique psychological profiles, but these did not resemble the hypothesized subtypes of gender incongruence. This is likely in part because classical gender incongruence is exceptionally rare. While Chapter 1 showed that 12 out of 11,864 participants (0.1%) in the full ABCD sample identified as transgender at ages 9-10 years at baseline—a proportion in line with historical prevalence and possibly indicative of classical gender incongruence (K. Zucker, 2017)—this sample was likely not large enough to form a detectable cluster even if these dozen individuals had unique profiles. Similarly, autogynephilia is both rare and does not typically develop until adulthood. While in principle, autogynephilia or autoandrophilia could have been observed as a cluster of late-onset, heterosexual cases, with otherwise typical mental health and social functioning, the age range and sample size may have been underpowered to detect such a cluster if it was existable at all.

Instead, these clusters reflected gradations of severity of gender incongruence and psychosocial engagement across the full sample. Cluster 1 presented an exclusively nonbinary-

identified group who were less pubertally developed, reporting low gender incongruence and low gender nonconformity, paired with low internalizing symptoms and generally lower social stress. Cluster 2 presented an intermediate group, with relatively higher levels of gender incongruence, intermediate psychopathology and social stress, and somewhat higher rates of LGB sexual orientation. Cluster 3 had the highest levels of gender incongruence and nonconformity, the highest proportion of transgender identity and LGB sexual orientation, the highest internalizing symptoms and social stress, and were most pubertally developed. Across clusters, no significant differences were observed for age, family conflict, or cyberbullying, suggesting that the latent structure was primarily defined by gender and sexual identity-related variation.

While the latent clusters identified did not map onto the theorized typology, cluster membership did significantly predict persistence and desistance likelihood. Cluster 1 members observed no longitudinal persistence cases, adding to evidence that nonbinary identification is more likely to be a transient state than a stable identity (Byrne, 2023). Cluster 2 members had intermediate rates of both longitudinal gender persistence and desistance, while Cluster 3 members had the highest rates of both longitudinal persistence and desistance. This may reflect the fact that Cluster 3 had the highest rates of transgender identification, which is thought to be more stable, but also the highest levels of psychosocial stress, which may exacerbate identity instability and lead to a more ROGD-like presentation (Littman, 2018).

Together, these results suggest that gender incongruence throughout adolescence has distinct manifestations, both within individuals and from existing theories derived from the literature on clinical gender dysphoria. Gender incongruence does not necessarily result in clinical dysphoria, but as shown in Chapter 1 has increased dramatically in recent years, paralleling findings on clinical gender dysphoria referral rates (Cass, 2024; HHS, 2025; K.

Zucker, 2017). As the ABCD Study offers a demographically diverse snapshot of youth in the contemporary United States, changes in gender incongruence may reflect changing cultural attitudes towards gender expression and identity as a whole, and some have hypothesized that these changes are more visible in females because gender norms are more rigid in males (e.g., Turban, 2024). In either case, this evidence is largely consistent with the theory of ROGD (Littman, 2018). Rather than rapid-onset gender dysphoria, however, it suggests rapid-onset gender fluidity, which may be a part of normative adolescent identity exploration with unknown psychosocial consequences. As the cluster analyses showed, many nonbinary youth may express gender nonconformity and remain well-adjusted, while others with high risk for internalizing psychopathology and peer victimization may be at risk for ROGD.

As the ABCD Study cannot speak to clinical gender dysphoria directly, it is possible that the triune typology of gender dysphoria may still hold in clinical samples. It is also possible that, insofar as the ABCD gender incongruent sample is relative to gender dysphoria, it is most representative of ROGD, and that there is insufficient power to detect classical gender dysphoria or autogynephilia, which are hypothesized to be much rarer (Byrne, 2023).

However, irrespective of their link to clinical gender dysphoria, these findings are valuable for understanding adolescent gender identity development and updating our theories as to the etiology of gender incongruence and its longitudinal stability. Gender incongruence in adolescence cannot be reduced to a single developmental pathway or subtype. Continued longitudinal follow-up on the ABCD sample and others will be essential to understand how gender incongruence and identity continue to change into adulthood, and how mental health and social functioning changes with it.

General Discussion

Why do some people feel trapped in a body of the wrong gender? Why do others exhibit strong gender nonconformity, but never question their identity? How does gender expression and identity development change from childhood to adulthood? What are the causes of gender incongruence, and are there unique subgroups of individuals with different risk profiles?

My dissertation attempts to address these questions through a historical overview of theories of gender dysphoria informed by the neurobiology of sexual development, as discussed in the General Introduction, and four empirical studies examining gender incongruence across early adolescent development. These studies made use of the Adolescent Brain Cognitive Development (ABCD) Study sample, a demographically diverse longitudinal sample of 11,864 youths in the United States followed from ages 9-10 years in 2016-2018 to ages 14-15 years in 2021-2023 (Barch et al., 2018; Garavan et al., 2018). Combining existing evidence with results from these four chapters, summarized below, I conclude by proposing a new revised biopsychosocial model of adolescent gender dysphoria.

Longitudinal Measurement of Adolescent Gender Identity Development

Chapter 1 comprehensively assessed longitudinal trajectories of adolescent gender identity development in the ABCD Study across measures and reporters. Measures included categorical self- and parent-reported gender identity, as well as continuous self- and parent-reported gender incongruence and gender expression. For both males and females, gender incongruence increased over the study period. At baseline, 0.5% of males and 1.1% of females identified as transgender or nonbinary (TNB). By Wave 5, 1.2% of males and 9.6% of females identified as TNB. Across all waves, 133 (2.2%) out of 6,187 males and 537 (9.5%) out of 5,677

females self-identified as TNB during at least one timepoint. The total population of TNB youth was 80.1% female, the majority of whom "came out" at later timepoints. Females were also more likely to report gender incongruence and elevated gender nonconformity (i.e., sex-atypical gender expression).

On average, parents reported lower gender incongruence relative to youth self-report. While 64% of parents of transgender-identifying children identified their child as transgender, nonbinary, or gender-questioning, only 36% of parents of nonbinary-identifying children identified their child as transgender, nonbinary, or gender-questioning. Parents also reported higher gender incongruence and gender nonconformity among their female children, although parent reports on average remained lower than youth self-report. Among TNB youth, and even after accounting for the overall higher prevalence in females, TNB females reported much stronger severity of gender incongruence, and this effect and sex difference increased with age. While youth who identified at TNB at any given timepoint had higher gender incongruence than cisgender youth at that timepoint, the majority of TNB youth reported little to no gender incongruence at baseline at ages 10-11 years. By contrast, the population of youths who identified as TNB at any timepoint displayed high gender nonconformity by both self-report and parent-report at every timepoint.

These results indicate that recent rises in gender dysphoria prevalence—more than a 30-fold increase in the last two decades (Cass, 2024)—are not limited to clinical referrals. One might hypothesize that subclinical increases in gender incongruence are a result of recent cultural changes in gender expression, increased social acceptance of gender nonconformity, or both. However, gender expression did not change with age or across longitudinal timepoints in the ABCD sample. Gender incongruence measures were only modestly correlated with gender

nonconformity, and a strongly skewed distribution (with most participants indicating "Never" experiencing gender incongruence) indicates it functions more similarly to a subclinical measure of gender dysphoria (e.g., Johnson et al., 2004). Therefore, it is possible that the observed increases in gender incongruence with age, particularly in female adolescents, support the theory of "rapid-onset gender dysphoria" (ROGD; Littman, 2018). The following chapters assessing the heritability of gender incongruence and its biopsychosocial correlates, including internalizing symptoms, further tested this hypothesis.

Heritability of Adolescent Gender Incongruence

Evidence from clinical populations with disorders of sex development (i.e., intersex conditions) and case studies of gender reassignment during infancy, reviewed in the General Introduction, suggests gender identity is partially innate. When gender incongruence manifests early in childhood, it is likely heritable and influenced by genes relevant to sex hormone production and receptor sensitivity (Foreman et al., 2019). Indeed, twin studies of gender dysphoria in transgender adults show strong concordance of gender identity (L. Diamond, 2013; Heylens et al., 2012) with heritability estimates averaging 30%, similar to gender expression and sexual orientation, both argued to be innate and mediated by sex hormone-dependent mechanisms (Bailey et al., 2016; Coolidge & Stillman, 2020; Verweij et al., 2016).

However, the phenomenon of adolescent-onset gender incongruence described in Chapter 1 does not fit this innate profile. With the exception of 5-alpha-reductase-2 deficiency (5-ARD), the onset of gender incongruence after puberty is unlikely to be innate. 5-ARD is a unique exception in which genetic males are born lacking 5-alpha-reductase-2, an enzyme necessary to synthesize testosterone into dihydrotestosterone (DHT), a hormone necessary for constructing

male genitalia during fetal development (Imperato-McGinley & Zhu, 2002). Similarly to people with complete androgen insensitivity syndrome (CAIS; Hines et al., 2003; Oakes et al., 2008), whose androgen receptors are inert to all androgens including testosterone and DHT, genetically male fetuses with 5-ARD will develop a vagina along the "default" female pathway in absence of DHT stimulating urogenital tissue along the male pathway. Children with 5-ARD and CAIS are therefore typically assigned female at birth and reared as girls, despite their XY karyotype. However, unlike children with CAIS, who functionally develop into infertile female adults, even going through estrogen-mediated puberty as their unused testosterone is aromatized (Tyutyusheva et al., 2021), children with 5-ARD retain male-typical testosterone levels at puberty, only lacking DHT (Imperato-McGinley & Zhu, 2002). As a result, they will develop masculine secondary sex characteristics, including facial hair, deep voices, and muscularity. With intersex phenotypes, adolescents with 5-ARD often develop gender dysphoria and some choose to live their lives as men, while others opt for androgen blocker treatment and estrogen therapy to continue living as women (Imperato-McGinley et al., 1979; Nascimento et al., 2018; K. Zucker, 2002).

disorders of sex development such as 5-ARD and CAIS, as well as case studies of gender reassignment discussed in the General Introduction, provide robust evidence that gender incongruence can be driven by innate biological causes. However, these circumstances are exceptionally rare, with the most common intersex condition known to impact gender identity development, congenital adrenal hyperplasia, occurring in fewer than 1 out of every 15,000 births (Pang et al., 1993). In typically-developing populations, can adolescent-onset gender incongruence also be explained by genetic factors, or is it primarily a product of social learning and environmental stressors, as hypothesized by the theory of ROGD?

Chapter 2 sought to test this question by making use of the ABCD Study's large sample of 1,970 same-sex twins (985 pairs; 419 monozygotic pairs; 50.3% male). As in the full sample, gender incongruence was more common in females. Notably, probandwise concordance for TNB gender identity was 46% among monozygotic pairs compared to 13% among dizygotic pairs. Heritability analyses on the continuous measure of gender incongruence indicated that genetic factors explained 47% of the variance, with the remaining variance explained by non-shared environmental factors. These results suggest that genetic factors explain a large proportion of gender variance in adolescents.

Importantly, these results do not imply that adolescent-onset gender incongruence is innate but late-onset, like pubertal development. While results estimate higher heritability than sexual orientation, a trait commonly argued to be innate and manifesting only during adolescence (Bailey et al., 2016), they must be contextualized against other well-established findings in the field of behavioral genetics. The pattern of roughly half genetic and half unique environmental influence is entirely typical of psychological traits, consistent with the Three Laws of Behavioral Genetics (Pinker, 2002; Turkheimer, 2000). Additionally, clinical outcomes such as depression and anxiety, known to be strongly impacted by environmental stressors, have been shown to be more than 50% heritable in adolescents (Polderman et al., 2015). Internalizing symptoms in particular may serve as strong confounds if the theory of ROGD is correct. The next chapter therefore aimed to test biopsychosocial correlates of gender incongruence, in order to examine both potential confounds inflating heritability estimates and potential mediators of the remaining environmental variance.

Biopsychosocial Correlates of Adolescent Gender Incongruence

Chapter 3 examined concurrent and longitudinal cross-lagged associations between self-reported gender incongruence and a range of biopsychosocial variables known or hypothesized to be associated with gender dysphoria: internalizing symptoms, autism spectrum traits, sexual orientation, pubertal timing, body mass index, family conflict, peer victimization, cyberbullying, screen time, and number of TNB friends. All predictors of interest had univariate associations with gender incongruence, and all associations remained significant in multivariate analyses except family conflict and number of TNB friends. Therefore, insofar as family and social support are relevant to gender incongruence, these measures may be confounded by related variables such as internalizing symptoms, sexual orientation, and screen time. This may explain why family support has failed to show attenuation of mental health disparities between TNB and cisgender youth, also in the ABCD sample (Martinez Agulleiro et al., 2024).

Most findings have been reported before in cross-sectional studies, but researchers have debated the directionality of effects. My study directly tested predictions made by ROGD against predictions made by minority stress theory. ROGD posits that risk-factors for gender dysphoria—including higher internalizing symptoms, earlier pubertal timing, higher body mass index, higher levels of peer victimization and cyberbullying, and higher levels of screen time—are likely to longitudinally precede gender incongruence, particularly in adolescent females (Clayton, 2023; Littman, 2018). These relationships are predicted on the basis of mental health problems, social stressors, or atypical weight or maturation being known risk-factors for other forms of body dysmorphia and eating disorders, which adolescent females are most likely to develop (Allison et al., 2014; Barendse et al., 2022; Prinstein et al., 2010; Spiliadis, 2025).

Conversely, minority stress theory proposes that these associations are better understood as downstream consequences of gender incongruence (Frost & Meyer, 2023). Higher levels of peer victimization and cyberbullying may follow from adopting a stigmatized identity, which would also influence later mental health, and elevated body mass index and screen time may be the result of coping mechanisms for minority stress rather than risk factors. Even accelerated pubertal timing may hypothetically follow from increased minority stress, as research on early life adversity has shown that stress hormones accelerate pubertal maturation and biological aging, in accordance with evolutionary life history theory (Colich et al., 2020).

As a whole, cross-lagged associations largely favored predictions made by ROGD. Internalizing symptoms predicted later gender incongruence across every pair of longitudinal timepoints, but gender incongruence did not predict later internalizing symptoms. These results suggest that, among youth not otherwise exhibiting gender incongruence by early adolescence, mental health risk may increase likelihood of identity confusion or experimentation, and attempts to find belonging in a new community. This interpretation is corroborated by longitudinal associations between screen time and subsequent gender incongruence, but not vice versa, indicating that youth who struggle to find social belonging or who are exposed to novel forms of gender expression online may be drawn to such identity experimentation. Relatedly, in females, earlier pubertal timing and higher body mass index—both known risk factors for body image issues and internalizing psychopathology (Kaplowitz, 2008; Milano et al., 2020; Pfeifer & Allen, 2021; Ullsperger & Nikolas, 2017)—predicted longitudinal increases in gender incongruence. Taken together, these results indicate that the observed increase in adolescent-onset gender incongruence in ABCD bears similarities to ROGD (Littman, 2018), with the caveat that these are small effects applicable only to subclinical gender incongruence increases and not clinical

gender dysphoria. The final chapter aimed to explore whether these variables also predict longitudinal gender identity stability within the TNB subsample, and whether subtypes of gender incongruence which do not fit the subclinical ROGD profile are observable.

Gender Persistence, Desistance, and Psychological Profiles of Transgender and Nonbinary Adolescents

While evidence from the ABCD Study indicates that the observed increases in adolescent-onset gender incongruence best resemble subclinical ROGD, historically gender incongruence has presented very differently. Its classical form manifests during early childhood, and is likely partially mediated by sex-atypical prenatal androgen exposure, as evidenced by studies of people with disorders of sex development and case studies of gender reassignment. Such innate gender incongruence is often linked to homosexuality, and any clinically significant distress associated with gender incongruence often remits upon the healthy integration of one's sexual identity into adulthood (Bailey & K. Zucker, 1995). In the literature on clinical gender dysphoria, such remittance is called "desistence," while stability of gender dysphoria or transgender identity is called "persistence" (Byrne, 2024; K. Zucker, 2003, 2018). On average, child-onset gender dysphoria has low persistence and high desistance, but among transgender adults, persistence is high and desistance is low (Bailey & K. Zucker, 1995; deMayo et al., 2025; James et al., 2016).

A third form of documented gender incongruence is the autoerotic type, overwhelmingly observed as autogynephilia in males (Blanchard, 1991; Freund & Blanchard, 1993; Lawrence, 2009, 2010). In contrast to classical gender incongruence, autogynephilia manifests after puberty or later in adulthood, and is typically found in heterosexuals. In contrast to ROGD,

autogynephilia is most prevalent in males rather than females, and is not necessarily accompanied by internalizing symptoms, body dysmorphia, social exclusion, or signs of social contagion. Together, these characterizations—rapid-onset gender dysphoria, classical gender dysphoria, and autogynephilia—form what is known as the triune typology of gender dysphoria (Byrne, 2023).

The triune typology explains epidemiological inconsistencies in gender dysphoria incidence which cannot be explained by any single type (HHS, 2025), but has never been empirically validated. Chapter 4 aimed to test whether a data-driven three-cluster solution of latent gender incongruence classes in the TNB subsample of the ABCD Study resembles this triune typology. Additionally, this chapter aimed to document predictors of longitudinal gender persistence and desistance of TNB identity across adolescence.

Finite mixture regression identified three latent profiles of gender incongruence, but these did not resemble classical gender incongruence or autogynephilia. This likely reflects the rarity of both phenomena, particularly the latter being unlikely to be observable in a sample of youth. Instead, the profiles appeared to describe mild, moderate, and pronounced subtypes of ROGD. The "mild" case, in particular, likely represents its own unique phenomenon: participants in this cluster were exclusively nonbinary (as opposed to transgender-identified), had a 100% longitudinal desistance rate, sex-typical gender expression, and low levels of internalizing symptoms and social stress. In other words, youth in this cluster had no resemblance to ROGD other than adolescent-onset nonbinary self-identification, which in all cases was observed only during a single timepoint out of five. A total of 128 (20%) of the 670 TNB youths fit this "transient nonbinary" profile.

The pronounced cluster was the largest with 264 (42%) individuals who had the highest likelihood of transgender identification, nonheterosexual orientation, and longitudinal persistence, though persistence rates were still only 45%, far lower than samples of transgender youth who had socially transitioned genders prior to puberty (e.g., deMayo et al., 2025; Olson et al., 2022). Additionally, this cluster had the highest rates of peer victimization, cyberbullying, and internalizing symptoms, as well as earlier pubertal timing and higher mean body mass index. Though it is unknown whether any in this sample were ever diagnosed with gender dysphoria, this cluster bears the strongest resemblance to ROGD. The remaining 38% of TNB youth fell into a "moderate" cluster of mostly nonbinary females, with higher persistence rates and scores for ROGD-relevant risk-factors than the mild cluster, but lower than the pronounced cluster.

Across the full TNB sample (i.e., without regard to clusters), biopsychosocial correlates of gender incongruence identified in Chapter 3 were largely irrelevant to longitudinal persistence and desistance rates of TNB identity. The strongest predictors were transgender (as opposed to nonbinary) identity and lesbian, gay, or bisexual (LGB) sexual orientation. However, transgender identity and LGB orientation predicted desistance as strongly as persistence. It is possible that this is explained by transgender youth "coming out" earlier than nonbinary youth, and that persistence and desistance rates are confounded by timelines of gender identity exploration. In other words, if every participant were to persist as TNB for a fixed number of years then desist (i.e., return to cisgender identity), at the same rates for transgender- and nonbinary-identified participants but at different timelines, persistence rates would be artificially higher for transgender participants during earlier timepoints, and artificially lower during later timepoints. Follow-up studies of the ABCD cohort into adulthood are necessary to rule out this possibility.

Likewise, sexual orientation results may be confounded by homosexual or bisexual identity. In the ABCD Study, participants are asked "Are you gay or bisexual?" (Yes, No, Maybe), as part of the KSADS (Barch et al., 2018; Kaufman et al., 1997; Kobak et al., 2013; Potter et al., 2022). It is therefore unclear whether participants are gay or bisexual with mutual exclusivity. It is possible, consistent with theories of classical gender incongruence, that homosexual orientation is a strong predictor of persistence, while bisexual orientation is a strong predictor of desistance, leading to opposite observable effects when combined. Alternatively, it is possible that all participants who persist in TNB identity are homosexual with respect to their natal sex, but that the majority of homosexual participants desist in their TNB identities after assimilating their gender incongruence into their sexual identities, leading to positive predictions for both outcomes (Bailey & K. Zucker, 1995).

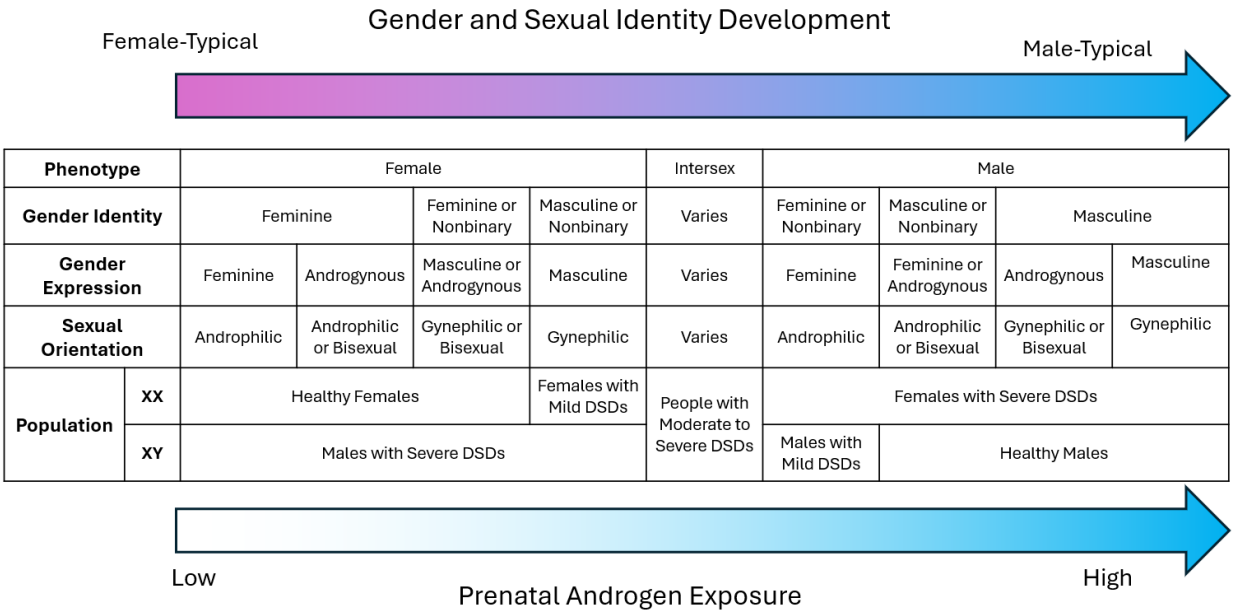
A Biopsychosocial Model of Adolescent Gender Dysphoria

Together, these four chapters advance our understanding of how gender incongruence manifests during adolescence and build towards a new biopsychosocial model of adolescent gender dysphoria. In particular, the present results identify a subclinical form of gender incongruence which promises to explain the rise in adolescent-onset gender dysphoria that has been dubbed ROGD, while also incorporating criticisms that the theory does not explain the lived experiences of many transgender and nonbinary youth (Littman, 2018; Turban, 2024).

Neuroendocrinology of Gender and Sexual Identity Development

The first premise of the proposed model is that individuals are born with a "brain sex" or starting "set point" of gender and sexual identity, largely determined by prenatal androgen

exposure. This is supported by evidence from clinical disorders of sex development and case studies of infant gender reassignment discussed throughout this dissertation. A schematic describing the neurodevelopmental set point of gender is shown in Figure 1.



Phenotype		Female				Intersex	Male			
Gender Identity		Feminine		Feminine or Nonbinary	Masculine or Nonbinary	Varies	Feminine or Nonbinary	Masculine or Nonbinary	Masculine	
Gender Expression		Feminine	Androgynous	Masculine or Androgynous	Masculine	Varies	Feminine	Feminine or Androgynous	Androgynous	Masculine
Sexual Orientation		Androphilic	Androphilic or Bisexual	Gynephilic or Bisexual	Gynephilic	Varies	Androphilic	Androphilic or Bisexual	Gynephilic or Bisexual	Gynephilic
Population	**XX**	Healthy Females			Females with Mild DSDs	People with Moderate to Severe DSDs	Females with Severe DSDs			
	XY	Males with Severe DSDs					Males with Mild DSDs	Healthy Males		

Figure 1. Neuroendocrine Model of Gender and Sexual Identity Development. CAIS = Complete Androgen Insensitivity Syndrome; DSD = Disorder of Sex Development.

Under this model, gender expression, gender identity, sexual orientation, and sexual phenotype (i.e., genitalia) all fall along a continuum mediated by prenatal androgen exposure. As discussed, independently of chromosomal sex, the brain and body follow a "female default" developmental trajectory, which is masculinized by androgens. The strongest evidence of this comes from males with complete androgen insensitivity syndrome (CAIS), who develop female genitalia and gender identities despite their XY karyotype (Hines et al., 2003; Oakes et al., 2008). Importantly, this model is dependent on functional androgen presence. Genetic males with CAIS have male-typical androgen levels, but non-functional androgen receptors, leading to an effective dose of zero. Conversely, individuals with highly sensitive androgen receptors may

be more progressively masculinized than individuals with higher absolute hormone levels but lower androgen sensitivity.

Individual differences in hormone levels and sensitivity are hypothesized to explain individual differences in gender expression and sexual orientation, though the most dramatic effects occur within disorders of sex development, in which genetic females are exposed to heightened androgen levels (e.g., CAH) or genetic males have reduced androgen function (e.g., partial AIS, 5-ARD) (Berglund et al., 2025). This model also parsimoniously explains the strong association between child gender nonconformity and later homosexuality, as well as predictions made by the homosexual theory of gender dysphoria (Bailey et al., 2016). As for why each of these traits are dissociable (i.e., why there are people who are gender nonconforming but not homosexual, and homosexual but not gender dysphoric), this is explained by its continuous dose-response relationship. This model hypothesizes a higher threshold of deviance from sex-typical hormone levels and sex-typical neurodevelopment for homosexuality to emerge than for gender nonconformity, and a still higher threshold for gender identity incongruence than for homosexuality. An innate "set point" of gender and sexual identity also explains why gender reassignment cases such as David Reimer's have dramatically failed and led to the development of strong gender dysphoria (Colapinto, 2000).

Plasticity of Gender and Sexual Identity

The only configurations of gender and sexual identity that cannot be explained by this model are homosexuality paired with sex-typical gender expression (e.g., Friedman & Stern, 1980), gender incongruence paired with heterosexuality (e.g., Blanchard et al., 1987), and why some cases of infant gender reassignment have been successful (e.g., Bradley et al., 1998). There

are two possible solutions to these open questions. The first is dependence on developmental timing, or the possibility that each trait in question (gender expression, gender identity, sexual orientation) have unique critical windows of plasticity during prenatal brain development. If this is true, in most cases, androgen levels typically remain above or below threshold for the sex-typical or sex-atypical development of each trait in question across all relevant critical windows. However, if there are dissociable critical windows, it is possible that hormonal fluctuations during one window of prenatal brain development may influence one trait and not the others. For example, if sexual orientation is determined during the third trimester but gender identity and expression are determined in the second trimester, and if an individual experienced hormonal imbalances only during the third trimester, it is possible that such a child would grow up to be gay without experiencing gender incongruence or displaying gender nonconformity (Bailey & K. Zucker, 1995).

A simpler, but not mutually exclusive, explanation is that the organizational effects of prenatal androgen exposure on neurodevelopment determines a gender and sexual identity "set point," but that it is not deterministic and gender and sexual plasticity remains throughout development. As in other cases of neuroplasticity, plasticity may be highest earlier in life, explaining why some cases of infant gender reassignment are successful (e.g., Bradley et al., 1998). Furthermore, plasticity may be greater for the traits known to be more variable. In other words, even if gender expression, sexual orientation, and gender identity all have set points determined in utero, gender expression may be most malleable, sexual orientation less malleable, and gender identity least malleable, though in all cases each may be able to change across the lifespan.

Gender plasticity throughout the lifespan is almost certainly the case given recent epidemiological changes in the prevalence of LGB orientations and TNB gender identities (Jones, 2025; HHS, 2025). It remains debated whether rising prevalence of gender and sexual minority identities represents a true increase, or a revealing of true neurobiological base rates as social stigma abates. In either case, these trends reveal plasticity. If the innate "set point" of gender and sexual diversity is higher than previously estimated, this offers robust evidence that generations were able to change, or at least hide, their gender and sexual identities under different cultural contexts, which arguably is its own form of plasticity. Conversely, if the set point itself is plastic, it is possible that changing social contexts may lead to genuinely higher gender and sexual diversity.

The Relationship Between Gender Incongruence and Gender Dysphoria

Bridging together the concept of an innate set point of gender identity and the reality of gender plasticity across the lifespan, it is possible to reconcile non-pathological increases in gender incongruence with the alarming rise in gender dysphoria incidence (Cass, 2024). Critics of the ROGD hypothesis often note that many transgender adolescents do not meet diagnostic criteria for gender dysphoria, and that even if gender incongruence is becoming more common as a result of increased cultural acceptance and media representations of gender variance, this is not indicative of socially contagious psychopathology (Ashley, 2020; Restar, 2020; Turban, 2024; Turban et al., 2023).

On the other hand, the increased incidence in clinical gender dysphoria has been dubbed a public health crisis, and increasing attention has been paid to cases of gender detransition, or people with gender dysphoria who have undergone "gender-affirming" hormonal interventions

or surgical sex reassignment, only to experience regret and revert to their natal gender (HHS, 2025; MacKinnon et al., 2025). In some cases, detransitioned individuals experience lasting harm as a result of procedures that have been characterized as medical malpractice (Clayton, 2022). How do we distinguish between adolescent-onset gender incongruence as a normative form of identity exploration and as a risk-factor for ROGD?

These may be two sides of the same coin. Figure 2 depicts a hypothesized relationship between population-level gender expression, gender incongruence (i.e., TNB self-identification), and gender dysphoria.

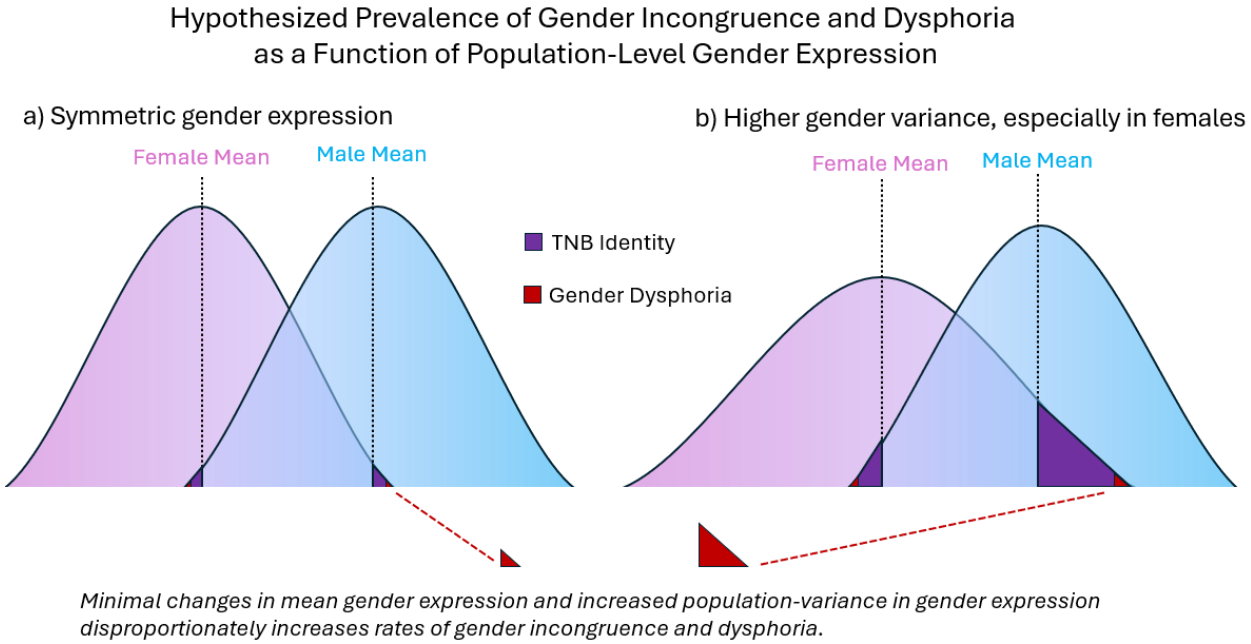


Figure 2. Hypothesized Prevalence of Gender Incongruence and Dysphoria as a Function of Population-Level Gender Expression.

Figure 2a depicts a hypothetical population of males and females with bimodally-distributed gender expression (i.e., male scores cluster around a masculine mean and female scores cluster around a feminine mean). In this idealized example, symmetric between sexes, the small proportion of males more feminine than the average female, and females more masculine than

the average male, experience gender incongruence and adopt a transgender or nonbinary (TNB) identity. A fixed small proportion of TNB individuals are said to experience gender dysphoria.

Figure 2b imagines the same population undergoing cultural change in gender expression. Becoming a more gender-diverse and gender-homogynous society, the female mean becomes slightly more masculine and the male mean becomes slightly more feminine. Additionally, the variance of male gender expression is slightly increased, and the variance of female gender expression is moderately increased. As a result, the proportion of individuals with gender incongruence—in this instance said to be more congruent with the opposite gender than a typical member of the opposite sex—substantially increases. As a fixed percentage of gender incongruent individuals, gender dysphoria prevalence likewise substantially increases. As a result, small shifts at the population level to gender norms and the variance of gender expression disproportionately influence the extremes of gender incongruence and dysphoria.

Under this model, such cultural changes in gender norms are driving both normative increases in gender incongruence and gender identity experimentation, as well as a public health crisis of rapid-onset gender dysphoria in people most predisposed to risk for internalizing psychopathology, namely adolescent females. The two should not be conflated but are intrinsically related.

Summary and Conclusion

The biopsychosocial model of adolescent gender dysphoria presented here largely preserves the triune typology of gender dysphoria (Byrne, 2023). There is robust evidence that gender identity is innate and influenced by prenatal androgen exposure. Some individuals, particularly those with disorders of sex development (i.e., intersex conditions), may experience

gender dysphoria innately as a result of atypical sex differentiation during early brain development. The mechanisms underlying this are intrinsically related to those which also dictate gender expression and sexual orientation, which explain child-onset gender incongruence and persistence rates. In most cases, children exhibiting gender incongruence and dysphoria will grow up to be gay and assimilate their gender nonconformity into their sexual identities (Bailey & K. Zucker, 1995). However, in a minority of cases, the remainder of people who persist in their gender incongruence will live their lives as transgender adults, and are best thought of as innately transgender, likely due to genetic factors mediated by early hormonal effects on brain development (Turban, 2024).

People who develop gender dysphoria during adolescence or adulthood do not fit this innate profile, and are best characterized as experiencing something of different origin. Historically, individuals experiencing adolescent- or adult-onset gender dysphoria were almost exclusively male (Bailey & Triea, 2007). A majority of those were heterosexual, distinguishing them further from the known early-onset neurobiological pathway to gender incongruence. Blanchard (1989) coined the term autogynephilia to describe a sexual paraphilia which can cause gender dysphoria, a controversial diagnosis but one which had met acceptance from some self-professed autogynephilic transgender women (e.g., Lawrence, 2010). For decades, this binary typology (homosexual vs. autoerotic) explained a majority of gender dysphoria cases, which was diagnosed at less than one-tenth the rate as it is today (K. Zucker, 2017).

Within the last two decades, aside from higher overall rates of gender dysphoria, a demographic shift began to emerge. A majority of gender dysphoria cases are now female, with particularly increased incidence in adolescent females (Cass, 2024; HHS, 2025; K. Zucker, 2017). This phenomenon was dubbed “rapid-onset gender dysphoria” (ROGD; Littman, 2018), a

controversial hypothesis proposing that gender dysphoria spreads by social contagion, similar to eating disorders and self-harm, in girls vulnerable to internalizing psychopathology and social exclusion. While this characterization certainly accurately portrays the circumstances of some adolescents with gender dysphoria (e.g., Shrier, 2020), it has been critiqued as overly pathologizing (e.g., Turban et al., 2023). After all, most youth experiencing gender incongruence (i.e., adopting transgender or nonbinary identities) are not clinically diagnosed with gender dysphoria, and many who are bear little resemblance to ROGD (deMayo et al., 2025). This is partly because transgender adolescents who have received the most intensive study had child-onset gender incongruence and may better fit the classical innate subtype, but many people with adolescent-onset gender incongruence bear no resemblance to ROGD, including in the large population sample from the Adolescent Brain Cognitive Development (ABCD) Study examined in this dissertation.

Results from the ABCD Study indicate that there is indeed an unprecedented surge in adolescent-onset gender incongruence, with up to 10% of contemporary adolescent females identifying as transgender or nonbinary (Chapter 1). Moreover, adolescent-onset gender incongruence is up to 50% heritable, though this is not necessarily indicative of innateness and could be confounded by covarying mental health traits which are equally or more heritable (Chapter 2). Adolescent-onset gender incongruence is consistent with hypotheses made by ROGD, including being longitudinally predicted by internalizing symptoms, social stress, and body composition, but effects were small and could not be said to bear clinical relevance (Chapter 3). Lastly, gender persistence rates were much lower than reported in clinical gender dysphoria samples or those who came out as transgender or nonbinary (TNB) during childhood,

and unique clusters of individuals were observed who bore both high relevance and no relevance to ROGD (Chapter 4).

The primary contribution of this dissertation is measuring and conceptualizing a new population of adolescents with gender incongruence which, I believe, has confounded many unresolved debates within contemporary research on adolescent gender dysphoria. The field routinely conflates gender incongruence and gender dysphoria, because, historically, TNB identification by definition characterized gender identity disorder, now known as gender dysphoria (Byne et al., 2018). While TNB individuals vary in gender-related distress, clinicians routinely misdiagnose gender dysphoria in order for patients presenting with gender incongruence to receive insurance coverage for "gender-affirming" care (Jasuja et al., 2020; Streed et al., 2023). As a result, clarity is often lost as to what claims generalize to which populations of people with clinically-distressing gender dysphoria and subclinical gender incongruence (Byrne, 2023). ROGD may be a real phenomenon without accurately characterizing the majority of adolescent female patients in gender clinics or experimenting with TNB social identities.

Within the ABCD cohort, largely representative of children born between 2006 and 2009 in the United States as a whole, an unprecedented proportion of youth identified as gender diverse, likely owing to sociocultural changes in understanding of and acceptance of gender nonconformity. Whether gender nonconformity is uniquely increasing in females, or whether social norms restricting gender expression remain stronger in males, remains unclear. However, a majority of these youth identify as nonbinary, without significant levels of gender nonconformity or psychosocial impairment, and a majority do not persist in this identity longitudinally. These changes are likely best thought of as normative identity exploration and

experimentation, though a minority of TNB youth do bear strong resemblance to ROGD and may be at increased risk for clinically-significant gender dysphoria and potentially harmful medical interventions.

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