A Psychological Investigation of the Link Between Autism Spectrum Disorder and Gender Dysphoria/Incongruence

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Declaration

I declare that the work presented in this thesis is my own carried out under the normal terms of supervision. To conduct this research, I was supported by a Vice Chancellor's Research Scholarship.

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Abstract

The clinical recognition of the high co-occurrence of autism spectrum disorder (ASD) and gender dysphoria/incongruence has received increased attention in recent years from researchers, as well as the lay press. Yet, many aspects of this phenomenon remain obscure. The overarching aim of this thesis was to develop an enhanced understanding of this intersection by answering three main questions. (1) Is there a link between ASD and gender dysphoria/incongruence? (2) What is the role of mentalising in the high co-occurrence of ASD and gender dysphoria/incongruence? (3) How does ASD affect gender-related cognition? To begin address these questions, we conducted a systematic literature review, two meta-analyses, two studies in the general population (original study; N = 101, followed by a replication study; N = 126), and a case-control study in neurotypical and autistic cisgender and transgender adults (N = 347). The findings in this thesis indicate the existence of a link between ASD and gender dysphoria/incongruence that is real and not a methodological artefact. Results of our studies also suggest that mentalising is *not* the shared underlying mechanism that underpins this link. Yet, our findings indicate that a mentalising difficulty might trigger subclinical gender dysphoric feelings in autistic cisgender people. Lastly, this thesis provides evidence that ASD hinders the explicit (and implicit at least among birth-assigned females) identification of autistic cisgender people with their experienced/reported gender. Autistic transgender people's explicit and implicit identification with their experienced/reported gender seems to be unaffected by ASD. The theoretical and clinical implications of these findings, as well as directions for future research are discussed.

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Chapter 1

Introduction to the Concept of Gender and Gender-Related Issues in Autism Spectrum Disorder

Gender Self-Concept

In the 1950s, the psychologist and sexologist John Money and his colleagues were among the first who introduced the concept of gender in psychology and differentiated it from sex (Money et al., 1955a, 1955b; Money et al., 1957). *Sex* connotates the state of being male or female. It is assigned at birth based on the appearance of external genitalia and is determined by genetic factors (American Psychological Association, 2012; Diamond et al., 2011; Johnson et al., 2009).[1] In contrast, *gender* emerges in a social context and refers to the cultural meanings (e.g., personality traits, roles, and behaviours) given to being male or female (American Psychological Association, 2012; Hines, 2004). Within the existing literature, there is a debate about the relation between sex and gender, but this discussion goes beyond the scope of this thesis (for a review, see Glasser & Smith, 2008).

Gender is a core part of our own sense of self and lies at the heart of our self-concept (Waltner, 1986). It is also one of the very first identities we adopt (Lewis & Brooks-Gunn, 1979) and plays an important role in the way we perceive the world (Frable, 1997; Markus et al., 1982). A person's sense of self as being a male, a female, or an alternative gender (e.g., nonbinary) is known as gender self-concept or gender identity (American Psychological Association, 2015). Gender self-concept is comprised of "personal and social attributes, social relationships, interests and abilities, symbolic and stylistic behaviours, and biological/physical/material

[1] Note that a discordance between chromosomal, gonadal, or phenotypic sex can occur. These rare congenital conditions are known as disorders of sex development (e.g., Witchel, 2018).

attributes" (Frable, 1997, p. 144). A number of definitions relating to sex and gender is provided in Table 1. Historically, the study of gender self-concept has been based on two major research traditions (Wood & Eagly, 2009, 2015) as outlined below.

Table 1

Definitions of Key Terms Relating to Sex and Gender

Term	Definition
Sex	The state of being male or female based on biological characteristics (chromosomes, genitals and hormones)
Gender	The attitudes, feelings, and behaviours that a given culture ascribes to a person's biological sex
Gender self-concept/identity	A person's sense of self as being a male, a female, or an alternative gender
Cisgender	A person whose experienced/reported gender corresponds to their birth-assigned sex
Transgender	Umbrella term used to describe a broad category of individuals who identify with a gender other than that associated with their birth-assigned sex
Nonbinary	Individuals whose experienced/reported gender is not exclusively male or female. It may include features of both genders or neither
Gender incongruence	Broad term used to describe the condition is which one's own experienced/reported gender is not in line with their birth-assigned sex
Gender dysphoria	General term that refers to the psychological distress that may accompany an individual's incongruence between their birth-assigned sex and their experienced/reported gender. In DSM-5, the term gender dysphoria is used as a diagnostic category
Gender nonconformity/variance	Umbrella term used to describe any situation in which the expression of one's own gender does not conform the societal norms
Femininity	Term that encompasses a set of traits, behaviours, and roles that are stereotypically attributed to the female sex
Masculinity	Term that encompasses a set of traits, behaviours, and roles that are stereotypically attributed to the male sex
Gender-typing	The process through which a child adopts behaviours, roles, and characteristics that are attributed to the gender associated with their birth-assigned sex
Transition	The process of affirming one's gender presentation and/or sex characteristics to be in line with their experienced/reported gender

Note. DSM-5 = Diagnostic and Statistical Manual of Mental Disorders.

Study of Gender Self-Concept

The first approach to the study of gender self-concept invokes personality traits and interests that are stereotypically attributed to males and females within society (Wood & Eagly, 2009, 2015). In its earliest form, this approach measured gender self-concept as a single *bipolar continuum* that ranged from masculinity to femininity (for a review, see Lippa, 2001). Yet, Constantinople (1973) criticised this this approach, noting that it was not in line with the evidence that masculinity and femininity constitute two separate constructs, for example. Following her critique, a new framework for studying gender self-concept emerged. The new approach, which reflects to a large extent the changes the modern women's movement brought in the early 1970s, conceptualises and measures masculinity and femininity as two *individual dimensions* (Wood & Eagly, 2009, 2015).

The direct, self-report measures of gender self-concept that follow this approach (e.g., the Bem's Sex Role Inventory; Bem, 1974 and the Personal Attributes Questionnaire; Spence et al., 1975) assess the extent to which individuals ascribe to themselves personality traits, roles, and interests that are stereotypically attributed either to males or females. Research has revealed significant sex differences in these types of self-report measures with cisgender (i.e., a person whose experienced/reported gender corresponds to their birth-assigned sex) birth-assigned males endorsing more masculine traits, roles, and interests as self-descriptive, and with cisgender birth-assigned females endorsing more feminine traits, roles, and interests to themselves, on average (Bem, 1974; Oswald, 2004; Spence & Helmreich, 1978; van Well et al., 2007; but see Donnelly & Twenge, 2017).

The second and more recent approach of examining gender self-concept draws on the social identity and self-categorisation theory (Tajfel & Turner,

1986/2004; Turner et al., 1987). In this approach gender self-concept constitutes a collective identity that individuals adopt when they explicitly identify with one gender group more than they identify with the other gender groups (Wood & Eagly, 2009, 2015). *Gender-group identification* is defined as "the descriptive or prescriptive categorisation of oneself as female or male, along with the importance of this categorisation for one's self-definition" (Wood & Eagly, 2015, p. 464). Following the principles of self-categorisation theory, this research tradition assumes that when people categorise themselves to one gender group (e.g., female), they ascribe to themselves stereotypes that are attributed to this group, feel more estranged with the members of the other gender groups, and are more favourably disposed towards the gender group that they belong to (Wood & Eagly, 2009, 2015).

The direct, self-report measures of gender self-concept that follow the principles of this research tradition require individuals to report the degree to which they identify with their gender (e.g., Greenwald et al., 2002), how typical they feel in relation to other people in their gender group (e.g., Bejerot & Eriksson, 2014; Egan & Perry, 2001), or to express the importance of their gender group for them (e.g., Copper et al., 2018). Research has shown that group membership is an important source of identity (Foels & Tomcho, 2005; Schmader, 2002; Tropp & Wright, 2001) and that, by middle childhood, children perceive themselves as typical members of their gender (Egan & Perry, 2001). Direct, self-report measures are widely used in gender research, but it is important to mention that people's responses in these measures may be biased due to social desirability, self-presentation, and limited introspective abilities (e.g., Devos et al., 2012; Nosek et al., 2005, 2007; Van de Mortel, 2008). An alternative approach to use of direct, self-report measures is to employ indirect measures.

Indirect measures assess *implicit cognition* by revealing automatic and spontaneous mental associations without requiring introspection, because implicit cognition cannot be reached through this ability (Nosek et al., 2007). Implicit cognition encompasses a series of constructs that are "introspectively unidentified (or inaccurately identified) trace[s] of past experience" (Greenwald & Banaji, 1995, p. 5) and as such, modulate the explicit and observable manifestation of behaviour in relation those constructs. To put it more simply, implicit cognition refers to cognitive processes that are beyond the conscious control of an individual and affect people's performance on tasks that measure self-concepts, attributes, stereotypes, etc. (Greenwald & Banaji, 1995).

The most widely used measure of implicit cognition is the Implicit Association Test (IAT; Greenwald et al., 1998). Since its initial publication, a number of different variations of the IAT have been developed and employed in a diverse range of branches of psychology, including developmental psychology (e.g., Baron & Banaji, 2006; Thomas et al., 2007), clinical psychology (Babchishin et al., 2013; Palfai & Ostafin, 2003), social and cognitive psychology (Fazio & Olson, 2003; Takarangi et al., 2013), political psychology (Albertson, 2011; Arcuri et al., 2008), and neuroscience (Ibáñez et al., 2010; Venkatraman et al., 2015). IATs are used in research to assess the strength of the automatic associations between concepts (Nosek et al., 2007).

Specifically, gender IATs measure the automatic associations of self-concept (using words related to self) with either gender-differentiated personality traits (e.g., gentle; Greenwald & Farnham, 2000) or gender groups (e.g., sister; Greenwald et al., 2002). In this task, individuals are asked to sort a number of stimulus exemplars from four concepts (i.e., self, other, female, male), using two response options, each of

which is assigned to two of the four concepts. The task is expected to be easier when two strongly associated concepts (e.g., me and female) share the same response option than when two unrelated or only weakly related concepts share the same response option (Nosek et al., 2007). Indeed, research has shown that cisgender birth-assigned females respond faster and more accurately when the same response option is shared between self-related words and female-targeted words than between self-related words and male-targeted words. Likewise, cisgender birth-assigned males perform better on the task when self-related words share the same response option with male-targeted words than with female-targeted words (Aidman & Carroll, 2003; Greenwald et al., 2002; Greenwald & Farnham, 2000; van Well et al., 2007; van Well et al., 2008). IATs show acceptable internal consistency (i.e., Cronbach's α around 0.80; Banse et al., 2001; Bosson et al., 2000; Cunningham et al., 2001) and are more reliable than other implicit measures (Bosson et al., 2000; Teige et al., 2004).

Overall, it can be argued that explicit and implicit measures are useful tools to provide an index of a construct (i.e., gender) that is more or less formulated, consolidated, and attached to the concept of self. But what do we know about the development of gender self-concept? What are the developmental trajectories of it, and what factors and mechanisms underlie its development?

Normative/Typical Development of Gender Self-Concept

The awareness of gender comes gradually after children have developed the capacity to recognise their selves, which is usually achieved after the age of 18 months (e.g., Bard et al., 2006; Nielsen et al., 2003; Nielsen et al., 2006). Cognitive-developmental theorists highlight that gender knowledge is acquired through several stages that are inherently related to child cognitive development (Kohlberg, 1966,

1969; Slaby & Frey, 1975). At around age 3, most children are able to label their own and other's gender (Ruble et al., 2006); this ability might be achieved as early as in 18 months to 2 years of age (Campbell et al., 2002; Campbell et al., 2004; Fagot et al., 1986; Levy, 1999). Gender-labelling is followed by the understanding that gender remains stable over time (e.g., a girl will grow up to be a woman and a boy will grow up to be a man). This stage is reached between the ages of 3 and 5 and by age 7, most children have acquired a good understanding that gender remains consistent, regardless of changes in appearance and interests (Ruble et al., 2006; Tobin et al., 2010). When these developmental milestones have been reached, it is believed that *gender constancy* has been achieved. Gender constancy refers to children's belief that gender is a permanent and irreversible part of self (Kohlberg, 1966).

The emergence of a rudimentary understanding of gender coincides with the first signs of gender-typed preferences and behaviour in children. Research has shown that gender-typed toy preferences may appear in children younger than 2 years (Alexander et al., 2009; Campbell et al., 2000; Jadva et al., 2010), become stronger in the course of development (Davis & Hines, 2020; Jadva et al., 2010; Todd et al., 2018), and can predict their occupational interests in early adolescence (Kung, 2021). From an early age, children also prefer to play with children of their own sex (Fabes, 1994; Maccoby, 1990; Maccoby & Jacklin, 1987) and to engage in activities and behaviours that are stereotypically attributed to males or females (e.g., boys are more likely than girls to engage in rough-and-tumble play behaviours; Harbin, 2016). Interestingly, when children have consolidated a secure sense of gender self-concept, rigidity in gender-typed behaviours and gender stereotypes decreases (Ruble et al., 2007; Trautner et al., 2005). It is clear from this brief overview that cognitive-developmental theorists view the consolidation of gender

self-concept as the product of reaching a number of stages that are inextricably linked to the cognitive maturation of the individual. We should not forget, however, that gender self-concept occurs within a social context.

Social-cognitive theorists highlight the importance of the environmental influences (i.e., parents, peers, and media) in the development of gender self-concept (Bandura & Bussey, 2004; Bussey & Bandura, 1999). From the moment of birth, children are treated differently based on their birth-assigned sex. This is what usually determines the name they are given and the way they are dressed (Bussey, 2011). It is clear that even before children become aware of their own gender, they start acquiring knowledge about gender (Bussey, 2011). According to social-cognitive theorists, *modelling* is considered the most crucial mode of conveying gender role information in children (Bussey & Bandura, 1984, 1999). Through modelling children learn what behaviours, attitudes, and values are expected from them based on their birth-assigned sex. The development of gender self-concept is also achieved through *enactive experience*. Children observe and evaluate the social reactions towards gender-typed behaviours and regulate their behaviour accordingly. Lastly, through *direct tutoring* children learn that society stereotypically associates certain activities with a specific gender (Bussey & Bandura, 1984, 1999).

In addition to the role that cognitive maturation and social influences play in the development of gender self-concept, it is important to consider the role of biological factors. It is well established that prenatal exposure to gonadal hormones (especially testosterone) influences children's gender-typed toy preferences, activities, and preferences for playmates (Hines, 2011). Pasterski et al. (2005) found that girls with congenital adrenal hyperplasia showed an increased preference for male-typical toys and a decreased preference for female-typical toys than did

unaffected girls. Prenatal exposure to testosterone has also been found to influence sexual orientation and gender self-concept (Hines, 2011). Specifically, birth-assigned females with congenital adrenal hyperplasia are more likely to identify as homosexual, they recall less gender-typed behaviour from childhood, and report reduced satisfaction with their birth-assigned sex than unaffected birth-assigned females (e.g., Daae et al., 2020; Hines et al., 2004; Zucker, Bradley, Oliver, et al., 1996). Although we still know very little about the degree to which genetic factors affect the development of gender self-concept (Ristori et al., 2020), Loehlin et al. (2005) found that 40% of the total variability of gender diagnosticity (i.e., Bayesian probability that an individual is masculine or feminine based on some gender-related indicators; Lippa & Connelly, 1990) in male and female adults was explained by genetic factors. Yet, no strong candidate genes that could be involved in the development of gender self-concept have been found (Ristori et al., 2020).

In clinical practice, a "biopsychosocial model" rather than a pure "medical model" is used to explain the development of gender identity (de Vries et al., 2014). Based on this model, gender identity develops through a complex interplay between biological, psychological, and social factors (de Vries et al., 2014). Thus, clinical work supports gender identity exploration recognising people's right to formulate their own gender identity and to make informed decisions about their life (Stocks, 2019).

Variant/Atypical Development of Gender Self-Concept

Instead of following the normative pathways of gender self-concept development, a small number of children show signs of atypical development and express/display feelings of gender dysphoria. In early toddlerhood, these children typically show cross-gender interests and might express the wish to be the other

binary gender (de Vries et al., 2014). When these feelings are persistent, have an adverse impact on a child's everyday life, and last more than 6 months, the child might meet the diagnostic criteria of the Diagnostic and Statistical Manual of Mental Disorders (DSM-5; American Psychiatric Association, 2013) for *gender dysphoria*. Gender dysphoria (GD), formally known as transsexualism (for adolescents and adults) and gender identity disorder of childhood (for children) in DSM-III (American Psychiatric Association, 1980) and gender identity disorder in DSM-IV-TR (American Psychiatric Association, 2000), is a psychiatric condition characterised by an incongruence between one's own birth-assigned sex and one's experienced/reported gender that is accompanied by negative emotions about the characteristics of one's birth-assigned sex (American Psychiatric Association, 2013).[2] Most, but not all, gender incongruent people feel negative emotions about this incongruence (Olson-Kennedy et al., 2016). In the current thesis, we use the term *gender incongruence* as an umbrella term to describe the condition is which one's own experienced/reported gender is not in line with their birth-assigned sex, regardless of whether this incongruence is accompanied by significant levels of distress (Butler, 2020). Some gender dysphoric/incongruent people decide to *transition* socially and medically to another gender, while others transition only socially. Of course, not every gender dysphoric/incongruent person transitions to another gender. Social transition is defined as a person's:

> "change from living socially as the gender that matches the sex assigned at birth, to another gender, which may involve a change in name, pronouns,

[2] The definitions of gender dysphoria and autism spectrum disorder provided in this thesis are the definitions used in fifth edition of the Diagnostic and Statistical Manual of Mental Disorders (American Psychiatric Association, 2013).

> presentation, and a request that others recognize the child in their asserted gender rather than the gender that would match the sex assigned to them at birth. (Ehrensaft et al., 2018, p. 252)

Medical transition refers to gender-affirming medical care which may include the use of puberty blockers, hormone therapy, and/or gender-affirming surgeries to align one's body with their experienced/reported gender (Radix, 2016).

Clinical Manifestation of Gender Dysphoria

Children who meet the criteria for a diagnosis of GD (a) express a strong desire to be the opposite gender of that associated to their birth-assigned sex or they insist that they are the opposite gender, (b) show a strong preference for cross-dressing, and toys and activities that are stereotypically used or engaged in by the opposite gender, (c) adopt cross-gender roles in pretend or fantasy play, and (d) they might show a strong dislike of their sexual anatomy (American Psychiatric Association, 2013). Adults who meet the criteria for a diagnosis of GD might also desire to get rid of the primary and/or secondary sex characteristics of their birth-assigned sex, and/or to adopt the primary and/or secondary sex characteristics of the other sex, and show a strong desire to be and be treated as the opposite gender (or any other gender that is different from that associated to their birth-assigned sex) (American Psychiatric Association, 2013). GD as a diagnostic category follows two developmental pathways.

Early-onset GD is diagnosed when symptoms of this condition occur early in childhood, usually between the ages of 2 and 4 (American Psychiatric Association, 2013). This corresponds to the developmental period in which children show clear signs of gender-typed preferences and behaviours (Ruble & Martin, 1998). Research has shown that early-onset GD alleviates after childhood in most children, with the

estimates of children who cease to be gender dysphoric ranging from 61%–98% (Ristori & Steensma, 2016; Steensma et al., 2013; but see Ehrensaft et al., 2018; Temple Newhook et al., 2018)[3]. Early-onset GD seems to be a relatively strong predictor of a homosexual orientation later in life mostly for boys, and less so for girls (e.g., Steensma et al., 2013; Wallien et al., 2008). Late-onset GD is diagnosed when symptoms occur at puberty or later in life (American Psychiatric Association 2013; Zucker et al., 2016) and has been seen among birth-assigned males in the main, historically (Zucker et al., 2016). In recent years, however, a "new" subcategory of people with late-onset GD (also known as rapid-onset GD) has emerged and attracted the attention of clinicians and researchers. This subcategory includes mostly birth-assigned females whose GD emerges at puberty without any evidence of gender dysphoric feelings or behaviour indicative of gender variance in childhood (Littman, 2018; Zucker, 2019).

Prevalence and Sex Ratio of Gender Dysphoria/Incongruence

According to DSM-5, the prevalence of GD ranges from 0.005%–0.014% in adult birth-assigned males and from 0.002%–0.003% in adult birth-assigned females (American Psychiatric Association, 2013). As Zucker (2017) noted, no epidemiological studies have examined the prevalence of GD in children or adolescents. Based on the prevalence estimates reported in DSM-5, GD can be considered as a relatively rare condition. However, Zucker (2017) found that recent studies indicate that the prevalence of children, adolescents, and adults who *self-identify* as transgender, but who do not necessarily use clinical services, is higher ranging from 0.5%–1.3%. *Transgender* is umbrella term used to describe a broad range of individuals who identify with a gender other than that that matches their

[3] For a response to the critical commentary of Temple Newhook et al. (2018), see Zucker (2018).

birth-assigned sex (Butler, 2020).[4] Recent studies have also denoted an increase in the number of gender diverse adolescents (4%-10%; Ghassabian et al., 2022; Kidd et al., 2021).

The prevalence estimates of GD indicate that this condition is more common among birth-assigned males than birth-assigned females (American Psychiatric Association, 2013). Nonetheless, an alteration of sex ratio among adolescents with GD has been observed in the latest years. Specifically, research has showed that the prevalence estimates of transgender and clinically referred gender dysphoric adolescents are higher among birth-assigned females than among birth-assigned males (Aitken et al., 2015; Kaltiala-Heino et al., 2015; Zucker, 2017; Zucker & Aitken, 2019). Whether this reflects a real change in sex ratio is still debatable (see Zucker, 2019).

Aetiological Factors of Gender Variant/Atypical Development

With respect to the aetiology of gender-variant development, a number of factors seem to be implicated, yet its exact causes remain unknown. It is important to note here that the study of possible pathways that could lead to gender variance should only aim to increase our understanding of it in order to develop targeted and personalised interventions for people who suffer from a significant distress related to a mismatch between their birth-assigned sex and their experienced gender. Therefore, such endeavours might be best supported by community involvement (e.g., community-based participatory approaches that include sexual and gender minority individuals to help direct and contextualise such research). Research on the cognitive-developmental underpinnings of gender variance is very limited. Zucker et

[4] In this thesis, the terms “transgender” and “gender incongruent” are used interchangeably (definitions are provided in Table 1).

al. (1985) found that children clinically referred because of gender identity difficulties did not show the expected gender-typed preferences in toys as their gender conforming siblings did, instead showed a strong preference for toys consistent to their experienced/reported gender. In another study, Zucker et al. (1999) found a developmental lag in the acquisition of gender constancy among gender-referred children (i.e., children referred to gender clinics/services for gender-related issues). These children followed the cognitive stages of achieving gender constancy, but the rate with which they reached the milestones of gender labelling, gender stability, and gender consistency was slower than the rate of nongender-referred children. It should be noted that these studies were conducted in a time period that has been criticised by many for the practices employed to support gender-referred children (e.g., Langer & Martin, 2004).

More recent studies have shown that the development of gender self-concept in socially transitioned transgender children follows similar pathways to cisgender children (Fast & Olson, 2018; Olson et al., 2015; for a review, see Olson & Gülgöz, 2018). Transgender children believe that their experienced/reported gender is stable across time and remains consistent, regardless of changes in appearance and interests. They also show peer and object preferences for their experienced/reported gender and perceive implicitly and explicitly themselves in accordance with their experienced/reported gender (Fast & Olson, 2018; Olson et al., 2015).

At a biological level, to date, no study has provided strong evidence for a gene that could account for GD (Henningsson et al., 2005; Ujike et al., 2009), yet twin and family studies have suggested that genetic factors play a role in the development of gender dysphoria/incongruence (Gómez-Gil et al., 2010; Heylens et al., 2012). Specifically, the largest study to date in transgender twins showed that

28% of monozygotic co-twins of a proband who identified as transgender also identified as transgender. This compared to only 2.8% of dizygotic twins of a proband who identified as transgender (Diamond, 2013). This indicates a clear biological predisposition for gender incongruence. Prenatal exposure to abnormal levels of sex hormones might also play a role in the development of gender dysphoria/incongruence (Garcia-Falgueras & Swaab, 2010). Research has shown that 5.2% of birth-assigned females with congenital adrenal hyperplasia who were raised female experienced gender dysphoria and 2% of them initiated a transition (for a review, see Dessens et al., 2005). That is more than 666 times higher than the prevalence of birth-assigned females with gender dysphoria in the general population (American Psychiatric Association, 2013).

Psychosocial factors have also been suggested to play a role in gender-variant development. Older theories have implicated parents' prenatal sex preference, parental reinforcement of cross-gender interests and cross-dressing in children, and a symbiotic mother-son relation accompanied by a distant father-son relation as aetiological mechanisms for the development of GD (for a review, see Zucker, 2004). However, research has either provided evidence against these hypotheses, or hypotheses have not been examined extensively (Green et al., 1985; Zucker et al., 1994). More recent theories suggest that a number of biological and environmental parent-child risk factors are involved in the development of GD (Bradley & Zucker, 1990; Zucker & Bradley, 1995). The risk of developing GD is increased for example when general child and/or parent factors, such as anxious nature of the child, coincide with specific family risk factors, such as systemic conflicts within the family at a crucial developmental period (Bradley & Zucker, 1990; Zucker & Bradley, 1995). Indeed, research has provided some support about the general factors

that might predispose a child to develop GD, such as elevated levels of anxiety (e.g., Cohen-Kettenis et al., 2003; Wallien et al., 2007; Zucker et al., 2003; Zucker, Bradley, & Sullivan, 1996), yet, the model itself is still understudied (Ristori & Steensma, 2016). As de Vries et al. (2012) concluded:

> With the current state of knowledge, it remains most plausible that a complex interaction between a biological predisposition in combination with intra- and interpersonal factors (Crouter, Whiteman, McHale, & Osgood, 2007; Maccoby, 1998; Zucker & Bradley, 1995) contribute to a development of gender dysphoria, which may come in different forms and intensities. (p. 305)

Co-Occurring Conditions With Gender Dysphoria/Incongruence

Research has shown that mental health conditions occur among gender incongruent people at a significantly higher rate than among people from the general population (e.g., Dhejne et al., 2011; Nobili, Glazebrook, & Arcelus, 2018; Zucker et al., 2016). Specifically, depression and anxiety disorder are among the most frequent co-occurring conditions in transgender adults and adults with GD (e.g., Hepp et al., 2005; Heylens et al., 2014; Mazaheri Meybodi et al., 2014; for a review, see Dhejne et al., 2016). Emotional/behavioural problems are among the most frequent conditions in children with GD (Cohen-Kettenis et al., 2003; Wallien et al., 2007; Zucker, 2007; Zucker & Bradley, 1995). Research has also shown that transgender and gender dysphoric children and adults are at increased risk of self-harm and suicidality (e.g., Aitken et al., 2016; de Graaf et al., 2020; García-Vega et al., 2018; Grant et al., 2011; Peterson et al., 2017). Specifically, Aitken et al. (2016) found that children referred for GD were 8.6 times more likely to self-harm or attempt suicide than nonreferred children, and Grant et al. (2011) showed that transgender and

gender nonconforming adults were 25.63 times more likely to report a suicide attempt than people from the general population. Interestingly, in recent years, another co-occurrence has come to the attention of clinicians. As Wood et al. (2013) reported:

> Another factor that has impressed us in accounting for the increase in adolescent referrals pertains to youth with gender identity disorder who also have an autism spectrum disorder. As noted by others (de Vries, Noens, Cohen-Kettenis, van Berckelaer-Onnes, & Doreleijers, 2010), many clinicians are now reporting a co-occurrence of these two conditions. More than 10 years or so ago, it was rare in our clinic to see an adolescent with gender identity disorder who also appeared to have an autism spectrum disorder. (p. 5).

Indeed, clinicians seem now to witness this phenomenon almost on a daily basis (Strang, Janssen, et al., 2018). Yet, a number or aspects of this phenomenon remain obscure and underresearched.

Autism Spectrum Disorder

In 1943, the psychiatrist Leo Kanner reported the cases of 11 children who presented a fundamental *difficulty to relate themselves* to others and the outside world. The 6-years-old Frederick was one of these children. Kanner (1943) described Frederick in the following way:

> The child has always been self-sufficient…He mostly ignored other people…He acted as if people weren't there at all, even with his grandparents…He was never interested in hide-and-seek, but he'd roll a ball back and forth, watch his father shave, hold the razor box and put the razor back in, put the lid on the soap box. (pp. 222-223)

On year later, the paediatrician Hans Asperger (1944/1991) reported the cases of 4 boys with profound social problems. The 7-year-old Ernst was one of these children. Asperger (1944/1991) described Ernst in the following way:

> He spoke like "an adult"… He was "very precise": certain things always had to be in the same place, and certain events always had to happen in the same manner, or he would make a big scene… the eye gaze was highly characteristic, far away and unfocused. The eye did not seem to grasp anything and was vaguely aimed into the distance… He was never able to get on with other children. (p. 59)

By a remarkable coincidence, both Kanner (1943) and Asperger (1944/1991) used the term *autistic* (derived from the Greek word *autos* meaning self) to describe these children in their seminal reports. This condition is now recognised and known as *autism spectrum disorder*. Autism spectrum disorder (ASD) is a neurodevelopmental disorder diagnosed based on a dyad of severe difficulties with social communication, together with a restricted, repetitive pattern of interests and behaviours (American Psychiatric Association, 2013). ASD subsumes autistic disorder or autism, Asperger's disorder, and pervasive developmental disorder - not otherwise specified, that were previously included in DSM-IV-TR (American Psychiatric Association, 2013).

Clinical Manifestation, Prevalence, and Sex Ratio of ASD

The core features of ASD include (a) difficulties in social-emotional reciprocity, (b) absence or idiosyncratic verbal and nonverbal communicative behaviour, (c) deficits in developing, maintaining, and understanding social relationships, (d) insistence on sameness, inflexible adherence to routines, or ritualised patterns of behaviour, (e) highly restricted, fixated interests that are

abnormal in intensity or focus, (f) hyper- or hyporeactivity to sensory input or unusual interests in sensory aspects of the environment, and (g) stereotyped or repetitive motor movements, use of objects, or speech (American Psychiatric Association, 2013).

The behavioural features of ASD are present from early childhood (usually before the age of 3; Mandell et al., 2005) and affect significantly the daily functioning of the autistic individual (American Psychiatric Association, 2013).[5] It is well established in the literature that number of the aforementioned ASD traits is normally distributed in the general population (Constantino & Todd, 2000; 2003; Ronald et al., 2006), and that "unaffected" relatives of people with ASD report significantly more ASD traits than the general population (Frazier et al., 2014; Pickles et al., 2000; Piven et al., 1994; 1997). On this basis, it has been argued that ASD traits represent a single continuum that is known as "broad autism phenotype" (Bolton et al., 1994; Goldberg et al., 2005; Le Couteur et al., 1996; Murphy et al., 2000; Pickles et al., 2000; Piven et al., 1997; Szatmari et al., 2000). While people with low levels of ASD traits fall at the low end of the spectrum, people with a formal diagnosis of ASD fall at the high end of it. On this view, there is no distinct

[5] Throughout this thesis, we tend to use identity-first (i.e., autistic person) language, since this is preferred by a proportion of intellectually high-functioning autistic adults and autistic self-advocates (see Brown, n.d.; Kenny et al., 2016). Nonetheless, there is not a universal agreement about this in the community (Kenny et al., 2016). Therefore, person-first language (i.e., person with ASD) has been used in a few isolated places. The use of person-first language has been made with the premise that everyone is a person-first, regardless of their condition or identity (American Psychological Association, 2020) and with no intention of objectifying a person by their condition or identity.

cut-off for ASD, and diagnosis is based on an arbitrarily defined threshold of the number of traits or severity of impairment observed in an individual; the difference between those classified above this threshold (and thus gaining a diagnosis) and those classified below threshold is quantitative (and one of degree) rather than qualitative (and one of difference), according to this view.

The prevalence of ASD diagnosis in the general population is estimated to be approximately 1% (e.g., Baird et al., 2006; Baron-Cohen et al., 2009; Kim et al., 2011; Lai et al., 2014), yet more recent evidence indicates a prevalence of approximately 1.5% in developed countries (Lyall et al., 2017). Research has also indicated a male bias in ASD prevalence. A recent meta-analysis showed that the male-female ratio in ASD is 3:1 (Loomes et al., 2017).

Co-Occurring Conditions With ASD

As for co-occurring mental health conditions, there is strong evidence that these are significantly more prevalent in autistic people than in the general population (e.g., Lai et al., 2019; Lever & Geurts, 2016). Specifically, a number of studies have shown that about 70%–80% of autistic people have at least one co-occurring mental health condition, with attention-deficit/hyperactivity disorder, anxiety disorders, conduct disorders, depressive disorders, and obsessive-compulsive disorder being among the most common ones (e.g., Joshi et al., 2013; Lugnegård et al., 2011; Mattila et al., 2010; Simonoff et al., 2008; Vohra et al., 2017). Research has also shown that autistic people are at higher risk of suicide than the general population (Cassidy et al., 2014; Hirvikoski et al., 2016).

Aetiological Factors of ASD

Biological Factors.

ASD is highly heritable, which indicates an apparent biological basis of the disorder (e.g., Bailey et al., 1995; Colvert et al., 2015; Folstein & Rutter, 1977; Taniai et al., 2008). Specifically, a meta-analysis of twin studies showed that the heritability of ASD ranges from 74%–93% (Tick et al., 2016). Despite widespread research, however, the aetiology of ASD remains unclear. Regions on a number of chromosomes (e.g., 2q, 7q, and 16p) have been reliably linked to ASD (International Molecular Genetic Study of Autism Consortium, 2001; Shao et al., 2003), but no necessary and sufficient genes to explain the disorder have been found yet (for a review, see Rylaarsdam & Guemez-Gamboa, 2019). Furthermore, well-replicated findings from neurobiology have associated ASD with early brain overgrowth. Research has shown that compared to neurotypical children, autistic children in infancy and early childhood have a larger brain volume (Carper & Courchesne, 2005; Courchesne et al., 2001; Hazlett et al., 2011) and abnormalities in connectivity (Wolff et al., 2012). Further research is warranted, however, to understand how these variations is brain development and functioning may provide an explanation for ASD.

Environmental Factors.

A number of environmental factors have also been suggested to influence the risk of ASD. Specifically, research has indicated a number of prenatal, perinatal, and postnatal factors that are associated with an increased risk of developing ASD (for a meta-analysis, see Wang et al., 2017). Among the most common ones are advanced maternal and paternal age (e.g., Croen et al., 2007), prenatal exposure to antiepileptics (e.g., Bromley et al., 2013) and antidepressants (for a systematic review and meta-analysis, see Kobayashi et al., 2016), maternal hospitalization with infection during pregnancy (e.g., Lee et al., 2015), parental autoimmune diseases

(e.g., Keil et al., 2010), and short or long interpregnancy interval (e.g., Zerbo et al., 2015). It remains unclear, however, whether these factors increase people's liability specifically to ASD. Ultimately, it can be argued that the causes of ASD lie in a complex interplay between gene and environment (e.g., Emberti Gialloreti et al., 2019).

Cognitive Factors.

A number of cognitive accounts have attempted to explain the social-communication difficulties and/or the restricted and repetitive patterns of behaviour and interests that characterise ASD. Among these accounts are included the weak central coherence/enhanced perceptual processing theory (e.g., Happé & Frith, 2006) and the theory of executive dysfunction (e.g., Hill, 2004). One of the most prominent cognitive accounts, however, that has been proposed as a partial explanation of ASD symptomatology posits a primary deficit in mentalising (e.g., Brunsdon & Happé, 2014; Frith, 1994; Jones et al., 2018; Tager-Flusberg, 1999).

Mentalising (often called "theory of mind") is the ability to impute mental states to oneself and others (Carruthers, 2009; Frith & Happé, 1999; Premack & Woodruff, 1978). Mentalising is considered of fundamental importance for understanding and predicting other people's behaviour (e.g., Frith & Frith, 2005), and for fostering motives-based moral reasoning and prosocial behaviour (e.g., Baird & Astington, 2004; Bzdok et al., 2012; Imuta et al., 2016). The "false belief" task is one of the most classic and widely used paradigms employed to test mentalising, especially in preschool age children (Hogrefe et al., 1986; Wimmer & Perner, 1983). The classic "false belief" paradigm (e.g., "Sally-Anne" task; Wimmer & Perner, 1983) assesses children's ability to understand that a person can be mistaken in their belief about reality. It is well established in the literature that neurotypical children

pass reliably classic mentalising tasks at around age 4 (e.g., Gopnik & Astington, 1988; Perner et al., 1987; Wimmer & Perner, 1983; for a meta-analysis, see Wellman et al., 2001). In adults "advanced" mentalising tasks are usually employed. In these tasks, people are asked to interpret complex social situations and impute complex mental states to others, based on subtle/limited information. Among the most widely used mentalising tasks in adults are the Strange Stories Test (Happé, 1994), the Animations task (Abell et al., 2000), the Movie for the Assessment of Social Cognition (Dziobek et al., 2006), and the Reading the Mind in the Eyes test (Baron-Cohen, Wheelwright, Hill, et al., 2001).

Baron-Cohen et al. (1985) were the first who examined mentalising ability in autistic people. They found that autistic children showed a profound difficulty in passing the Sally-Anne task, compared to neurotypical children and children with Down's syndrome. Since then, many researchers have successfully replicated these findings within the autistic adult and childhood population, using both explicit and implicit measures of mentalising (e.g., Baron-Cohen, Wheelwright et al., 2001; Senju et al., 2009; Yirmiya et al., 1998). Poor performance on mentalising tasks has been considered as an "endophenotype" or "cognitive marker" for ASD (Gliga et al., 2014). This is based on evidence that mentalising difficulties not only are found in autistic people, but also run in families with a history of ASD (Eyuboglu et al., 2017; Gliga et al., 2014), are heritable (Hughes & Cutting, 1999), apparent even in optimal outcome children with a history of ASD (Kelley et al., 2006), and present in people with high levels of ASD traits, but not a diagnosis of ASD (Best et al., 2008).

Mentalising seems to play some role in the development of gender self-concept. Despite the limited research on this topic, evidence indicate that mentalising ability in children is positively related to their understanding of gender constancy

(Miller, 2007; Trautner et al., 2003; Zmyj & Bischof-Köhler, 2015) and negatively with use of gender stereotypes (Rizzo & Killen, 2018). This is important, because as previously mentioned, gender dysphoria/incongruence often co-occurs with ASD (e.g., Strang, Janssen, et al., 2018; Wood et al., 2013). A mentalising deficit has been proposed as a potential explanation for this apparent behavioural overlap (Glidden et al. 2016; Jacobs et al. 2014; Van Der Miesen et al. 2016; van der Miesen, Hurley, et al., 2018), but to date this hypothesis has not been examined systematically. Limited research has also been conducted on the development and expression of gender self-concept in autistic people.

Gender Self-Concept in ASD

Qualitative Evidence.

Listening to the accounts and experiences of autistic people, we learn that this population struggles not only to conceive the concept of gender, but also to express a gender self-concept that meets the societal expectations of masculinity and femininity. An analysis of autistic people's responses to online surveys, blogs, and autobiographical accounts indicated that, "not only does gender not constitute the definitive core of autistic experience, but for many, gender is barely present at all" (Davidson & Tamas, 2016, p. 61; see also Jack, 2012). Qualitative studies have shown that autistic people have a difficulty in understanding the concept of gender (Coleman-Smith et al., 2020) and that sometimes it is essential for them to explore this concept in order to identify if it exists in their own reality (Strang, Powers, et al., 2018). This might be related to a difficulty in consolidating a strong *self-concept* in general. Specifically, in Kourti et al.'s (2019) qualitative study, a number of autistic birth-assigned females expressed the idea that their fluid gender self-concept was part of a fluid sense of identity, and none of them felt able to conform to the societal expectations of expressing a female gender self-concept. Likewise, other evidence

from autobiographical accounts and qualitative studies has highlighted the atypicality autistic birth-assigned females have internalising attributes and roles that are stereotypically ascribed to them based on their birth-assigned sex (e.g., Bargiela et al., 2016; Kanfiszer et al., 2017; Miller, 2003). In Bargiela et al.'s (2016) study, an autistic women characteristically said "I knew that I wasn't being me" when she tried to adopt the role of a "wife" or "girlfriend" (p. 3288). In sum, autobiographical accounts and qualitative research seem to raise the hypothesis of a variant/atypical development and/or expression of gender self-concept in autistic people. Yet, only a very limited number of studies has conducted a quantitative examination of this hypothesis.

Quantitative Evidence.

To date, Abelson's study (1981) is the only quantitative study ever conducted on the development of gender self-concept in ASD. Abelson (1981) investigated the extent to which autistic children can develop a sense of gender identity, using the Michigan Gender Identity Test (MGIT) and the Gesell Question ("Are you a little boy or a little girl?"). Abelson found that only 48% of autistic children with mental age above 3 were able to label their own gender. He also found a significant positive correlation between participants' mental age and their performance on the MGIT, suggesting that the formation of gender self-concept in autistic children relied upon their cognitive development. It is important to mention, however, that the absence of a control group of neurotypical children limit the conclusions we can draw from this study. The examination of gender-typed play in autistic children could also increase our understanding of the development of gender self-concept in this population. To our knowledge, only one study has examined this topic. Knickmeyer et al. (2008) found that autistic birth-assigned male children showed a weaker preference for

male-typical play than neurotypical birth-assigned male children. Likewise, autistic birth-assigned female children showed a weaker preference for female-typical play than neurotypical birth-assigned female children. We also have limited knowledge of whether autistic adults attribute to themselves personality traits and gender role behaviours that stereotypically characterise males and females to the same extent as neurotypical adults do.

The sparse research on this topic has shown that autistic adults conform to masculine gender roles and traits (as measured with validated scales) to a lesser degree than neurotypical adults, yet no difference has been found in feminine gender roles and traits (Bejerot & Eriksson, 2014; Stauder et al., 2011). Interestingly, Bejerot and Eriksson (2014) also found that autistic people perceived themselves as being typical of their birth-assigned sex to the same degree as neurotypical adults. Furthermore, using a single item measure, Cooper et al. (2018) found that autistic birth-assigned females reported lower femininity and higher masculinity than neurotypical birth-assigned females and autistic birth-assigned males reported lower masculinity than neurotypical birth-assigned males. Cooper et al. (2018) also found that autistic people reported lower identification with their gender group and less gender self-esteem than neurotypical individuals.

Taken together, evidence from qualitative and quantitative studies indicate that the development and/or expression of gender self-concept in autistic people might not follow the normative pathways. Nonetheless, research on gender-related cognition in ASD is very limited, and we still know very little about the way autistic people formulate, consolidate, and express a gender self-concept. Given the high co-occurrence of ASD and gender dysphoria/incongruence, this is considered a critical gap in the literature.

The Current Thesis

Clinical experience yielded the first evidence of the co-occurrence of ASD and gender dysphoria/incongruence. Specifically, it was Williams et al. (1996) who reported the first cases in the literature of autistic people with co-occurring symptoms of GD. The authors presented the case of two boys (5 and 3 years old) who were referred to a child evaluation centre and received a diagnosis of ASD. Both boys engaged with cross-dressing and adopting cross-gender roles in imitative play. One year later, Landén and Rasmussen (1997) presented the case of an autistic girl who had a co-occurring gender identity disorder (now known as GD). Williams et al. (1996) suggested that the co-occurrence of ASD and GD could be more common than believed, albeit similar cases had not been reported in the literature before. They argued that the stigma associated with cross-gender interests and preoccupations in boys may deter parents from reporting these cases to clinicians.

It is worth noting that in the first decades of the 21st century, when transgender activism blossoms (Morris, 2009), there has been a remarkable increase in the number of case-reports of autistic people with a co-occurring diagnosis of GD or gender identity difficulties (Baker & Shweikh, 2016; Cain & Velasco, 2020; Gallucci et al., 2005; Jacobs et al., 2014; Kraemer et al., 2005; Lemaire et al., 2014; Mukaddes, 2002; Parkinson, 2014; Perera et al., 2003; Selinger, 2018; Tateno et al., 2008; Tateno et al., 2015; Violeta & Langer, 2017; Zupanič et al., 2021). Since then**,** the high co-occurrence of ASD and gender dysphoria/incongruence has received growing attention from researchers as well as the lay press.

Although the contribution of the lay press in the transmission of research findings to the community is considered very important, headlines such as “Do Transgender Children Just Have Autism?” (Prestigiacomo, 2017) have been

counterproductive to the study and understanding of a phenomenon with serious clinical implications. Research has provided strong evidence that the co-occurrence of ASD and gender dysphoria/incongruence is usually accompanied by an increase in internalised emotional problems (i.e., anxiety and depression) and suicidality (George & Stokes, 2018a; Mahfouda et al., 2019; Strang et al., 2021). This is of major importance, given that the prevalence of co-occurring mental health conditions and suicidality in both ASD and gender dysphoria/incongruence is already significantly higher than in the general population (e.g., Dhejne et al., 2016; Lai et al., 2019; Segers et al., 2014). Thus, the experienced psychological burden in people might be exponentially more severe in intensity when these conditions co-occur. This highlights the need for specialised and tailored support and care for this group.

Although some important steps have been made towards this direction (see Strang et al., 2020; Strang, Jarin, et al., 2018; Strang, Meagher, et al., 2018), it is important to understand the nature and foundations of the apparent overlap between gender dysphoria/incongruence and ASD so that appropriate interventions and support to autistic gender dysphoric/incongruent people can be provided (e.g., VanderLaan, Leef et al., 2015). In the current thesis, we aimed to elucidate some core aspects of this phenomenon, conducting a systematic literature review, two meta-analyses, and three empirical studies (i.e., a study in the general population, a replication study, and a case-control study). The results of these studies are presented in chapters according to different overarching research questions.[6]

[6] Chapters 3 and 5 (Experiment 1) contain the results of the study conducted in the general population. Chapters 4 and 5 (Experiment 2) contain the results of the replication study, and Chapter 6 contains the results of the case-control study.

Research Aims and Questions

Despite increasing evidence of a co-occurrence between ASD and gender dysphoria/incongruence that would not be expected by chance (e.g., Hisle-Gorman et al., 2019; Strauss et al., 2021; Warrier et al., 2020), a number of researchers have questioned the existence of a link between these conditions (Fortunato et al., 2021; Turban, 2018; Turban & van Schalkwyk, 2018). Thus, the first overarching aim of this thesis was to answer whether there is a link between ASD and gender dysphoria/incongruence. To answer this questions, we conducted a systematic review of the literature pertaining the co-occurrence of ASD and gender dysphoria/incongruence, a meta-analysis of studies on the prevalence of ASD *diagnoses* in gender dysphoric/incongruent people, and a meta-analysis of studies of ASD *traits* in gender dysphoric/incongruent people (Chapter 2). We also examined in the general population the relation between ASD traits, on the one hand, and current gender dysphoric feelings and recalled gender-typed behaviour, on the other hand (Chapters 3 & 4). Lastly, we investigated current gender dysphoric feelings and recalled gender-typed behaviour in autistic people, and whether transgender people with clinically significant levels of GD show the behavioural (i.e., ASD traits) and cognitive (i.e., mentalising) features of ASD (Chapter 6).

Furthermore, a number of clinicians and researchers in this field have expressed the need for further research on potential *mechanisms* that could explain the high co-occurrence of ASD and gender dysphoria/incongruence (e.g., Glidden et al., 2016; Van Der Miesen et al., 2016). Atypical/diminished mentalising ability has been proposed as a potential explanation for it (Glidden et al. 2016; Jacobs et al. 2014; Van Der Miesen et al. 2016; van der Miesen, Hurley, et al., 2018), but this hypothesis has not been examined systematically. Thus, the second overarching aim

of this thesis was to examine whether mentalising plays a role in the co-occurrence of ASD and gender dysphoria/incongruence. To answer this question, we examined the role of mentalising in the link between ASD traits and gender dysphoric feelings in the general population (Chapters 3 & 4) and investigated whether transgender people show a mentalising deficit that would indicate true co-morbidity between ASD and gender dysphoria/incongruence (Chapter 6).

Lastly, to acquire a deep understanding of this phenomenon, we considered it essential to know what gender means for autistic people and how gender self-concept is expressed in this population. As already mentioned, this is also a very underresearched area. Our knowledge on how ASD affects the mental processes of acquiring knowledge and understanding gender as well as formulating and consolidating a gender self-concept is very limited. Thus, the last overarching aim of this thesis was to investigate how ASD affects gender-related *cognition*, rather than only behaviour. To answer this question, we examined the relation between ASD traits and the explicit and implicit identification with gender stereotypical attributes and gender groups in the general population (Chapters 5 & 6) and investigated explicit and implicit gender-related cognition in autistic cisgender and transgender people (Chapter 6).

Chapter 2

Autism Spectrum Disorder and Gender Dysphoria/Incongruence. A Systematic Literature Review and Meta-Analysis

The number of publications on the suggested overlap between ASD and gender dysphoria/incongruence has more than doubled in the last two years, reflecting the increased attention this topic has received from clinicians, researchers, as well as the lay press (e.g., Seaman, 2016; Strang, Janssen, et al., 2018). Yet, it remains debated whether evidence supports this hypothesis (Fortunato et al., 2021; Turban & van Schalkwyk, 2018; Turban, 2018). Several reviews of the literature on the co-occurrence of ASD and gender dysphoria/incongruence have been conducted, but most of these were based on a very small number of studies published prior to 2016 (Glidden et al., 2016; Van Der Miesen et al., 2016; van Schalkwyk et al., 2015; Wood & Halder, 2014).

More recently, Øien et al. (2018) identified and catalogued studies on the co-occurrence of ASD and GD, but did not attempt to answer any research questions. In contrast, Thrower et al. (2020) conducted a systematic review of the literature to investigate whether there is an overrepresentation of ASD diagnoses/caseness (and attention deficit hyperactivity disorder) in people with GD. They found that the prevalence of ASD diagnoses in this population was increased (range: 4.8%–26%), but this was not examined statistically. This leaves a critical gap in the literature, which we propose to fill in the current chapter.

To examine whether the evidence indicate a link a between ASD and gender dysphoria/incongruence, we adopted two approaches in this chapter. First, in Part 1 we appraised the existing literature concerning the co-occurrence of ASD and gender dysphoria/incongruence, including not only studies conducted among autistic and

gender dysphoric/incongruent individuals, but also studies that examined the suggested overlap in the general population. As already mentioned in Chapter 1, the features of ASD represent a single continuum that is known as "broad autism phenotype". People with a diagnosis of ASD fall at the high end of this continuum and people with low levels of ASD traits fall at the low end of it (e.g., Bolton et al., 1994; Goldberg et al., 2005; Le Couteur et al., 1996; Murphy et al., 2000; Ronald et al., 2006). On this basis, it has been argued that important information about ASD can be obtained by investigating individual differences in ASD traits and their relation to other phenomena among people from the general population (e.g., Lind et al., 2020). Second, in Part 2 we conducted the first meta-analyses of studies of ASD diagnoses and ASD traits. Our aim was to report the first pooled prevalence estimate of ASD diagnoses in gender dysphoric/incongruent people and examine the hypothesis that gender dysphoric/incongruent people have elevated levels of ASD traits.

Part 1: Autism Spectrum Disorder and Gender Dysphoria/Incongruence: A Systematic Literature Review

Method

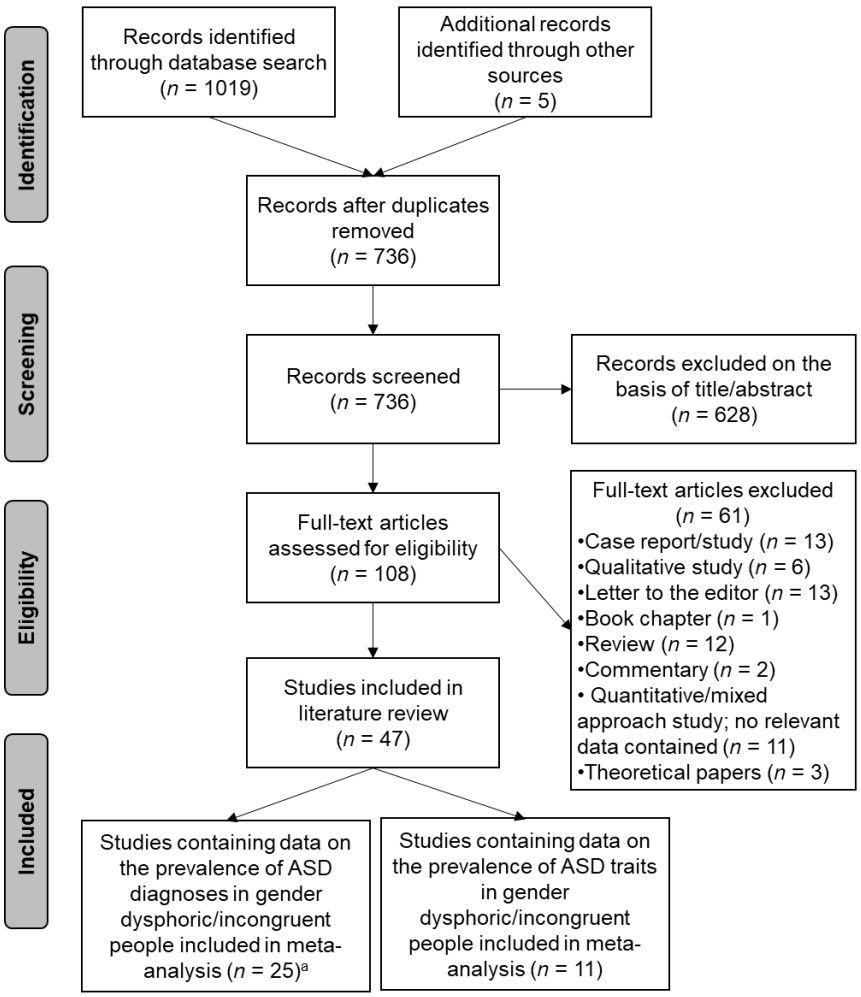
To perform the systematic literature review and meta-analyses (see Part 2), we followed the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) checklist guidelines where possible (Moher et al., 2015). A systematic literature search was conducted on Web of Science, Pub Med, and PsycINFO by the author of this thesis. Studies of interest were those examining the overlap between ASD/ASD traits and gender dysphoria/incongruence. To identify articles published prior to October 2020, we used combinations of the following search terms: "autism, autism spectrum disorder, autistic traits, autistic, ASD,

Asperger syndrome, gender dysphoria, transgender, gender dysphoric, gender identity disorder, transsexualism, transgenderism, sex reassignment, gender incongruence, non-binary/nonbinary, gender variance, gender non-conformity/gender nonconformity, and gender diversity", using the Boolean AND operator.

Articles that were considered eligible were those examined either the relation between ASD traits and gender dysphoria/incongruence in the general population, the prevalence of gender dysphoria/incongruence in autistic cohorts, or the prevalence of ASD diagnoses/caseness/ASD traits in gender dysphoric/incongruent cohorts. Articles that assessed ASD prevalence in gender dysphoric/incongruent cohorts should report rates of *formal* diagnoses of ASD (including Asperger's syndrome, autism, and pervasive developmental disorder), and articles that assessed ASD traits in gender dysphoric/incongruent cohorts should include a *control group* (either primary or secondary). Lastly, to be considered eligible, articles should report quantitative results, be published in peer-reviewed journals, and be written in English. As Figure 1 illustrates, the study selection was conducted in two stages. In the first stage, we screened articles by title and abstract for relevance. At this stage, we excluded proceedings papers and grey literature articles including dissertations. In the second stage, we evaluated the suitability for inclusion based on the full-text articles. Studies that reported nonquantitative results, such as reviews were excluded at this stage (for detailed information on excluded studies, see Table A1). Forty-seven articles met all the eligibility criteria and were included in the current systematic literature review.

Figure 1

PRISMA Flow Chart Illustrating Study Selection Process



Note. PRISMA = Preferred Reporting Items for Systematic Reviews and Meta-Analyses.

[a] Warrier et al.' s (2020) study included five datasets, all of which were independent from each other. As such, we decided to include them in the current meta-analysis as separate studies, hence $n = 25$.

Results

We were able to identify 1,019 studies through the database search and five studies through other sources. After duplicates were removed, we screened 736

studies and excluded irrelevant ones. From the remaining 108 full-text articles, we excluded 61 studies because they did not fulfil the criteria described in the method section. Hence, 47 studies that contained information on the overlap between ASD and gender dysphoria/incongruence were included in the current systematic literature review. Of these studies, five were conducted with children, 13 with children and adolescents, two with adolescents, two with children, adolescents, and adults, nine with adolescents and adults, and 16 with adults (for a summary of the identified articles, see Table A2).

Studies in the General Population

Shumer et al. (2015) were the first to report that elevated ASD traits in children *or* their mothers predicted high gender nonconformity in children. Nabbijohn et al. (2019) found a positive and significant association between ASD traits and gender variance in a sample of neurotypical children. The greater the number of ASD traits reported by parents in these children, the more parent-reported gender variance in these children. Interestingly, in a sample of neurotypical adults with no clinically significant levels of ASD traits, George and Stokes (2018b) reported a positive and significant association between the number of self-reported ASD traits and the number of concurrent gender dysphoric feelings. Kallitsounaki and Williams (2020a) successfully replicated this finding in cisgender people from the general population and extended this finding further by reporting a significant association between ASD traits and *recalled* gender-typed behaviour (i.e., behaviour that is considered stereotypically characteristic of a specific gender and recalled from childhood). That is, the higher the number of ASD traits self-reported by an individual, the more they reported experiencing current gender dysphoric feelings and the less they recalled childhood gender-typed behaviour. Results were

successfully replicated in an independent sample by Kallitsounaki et al. (2021), highlighting the existence of a robust and reliable link in the general population between ASD traits, on the one hand, and current gender dysphoric feelings and recalled childhood gender-typed behaviour, on the other hand.

Studies in the Autistic Population

Janssen et al. (2016), May et al. (2017), and Strang et al. (2014) used the item 110 from the Child Behaviour Checklist to investigate cross-gender wishes (i.e., wishes to be the other binary gender). They all found that parents of autistic children endorsed this item more frequently, compared to parents of nonreferred children. Adopting a similar methodology, van der Miesen, Hurley, et al. (2018) examined cross-gender wishes in autistic adolescents and adults, using the item 110 from the Youth Self-Report and the Adult Self-Report. This item also measures endorsement of the wish to be the gender opposite to birth-assigned sex. The study reported that autistic adolescents were 2.12 times more likely to endorse this item for themselves than were nonreferred adolescents. Likewise, autistic adults were 2.46 times more likely to endorse the item than nonreferred adults. Although this single item approach has been frequently used to investigate gender variance in autistic individuals, it is not entirely free of criticism. It can be argued that the item 110 taps ideation rather than behaviour. Therefore, it cannot be considered equivalent to a gender evaluation in which behaviours, wishes, and roles are all assessed. Also, Turban and van Schalkwyk (2018) offered an alternative explanation for the increased prevalence of the wish to be the gender opposite to birth-assigned sex observed in autistic people. They suggested that the well-established cognitive inflexibility in ASD might trigger *ephemeral* desires among autistic people to be the gender opposite to their birth-assigned sex.

Hisle-Gorman et al. (2019) has published the only study on the prevalence of a *formal diagnosis of GD* in children with a primary diagnosis of ASD. Collecting information from medical records, they conducted a matched case-cohort study and found that autistic children were over 4 times more likely to have a co-occurring diagnosis of GD than were neurotypical children. Furthermore, Nabbijohn et al. (2019) found that parents of autistic children reported significantly more gender variance in their children, compared to parents of neurotypical children. George and Stokes (2018b) utilized a standardised self-report measure of gender dysphoric feelings to compare autistic adults with neurotypical adults (i.e., Gender-Identity/Gender-Dysphoria Questionnaire for Adolescents and Adults; Deogracias et al., 2007). They found that the autistic group reported significantly more GD than the neurotypical group. However, it is important to note that the percentage of cisgender participants was 89.59% in the neurotypical group and 70.23% in the autistic group. Given the difference between the two groups, it could be argued that the inclusion of noncisgender people in the analysis could have artificially inflated the score of the autism group, creating a significant difference in gender dysphoric feelings between autistic and neurotypical people. Replication of these findings await, before strong conclusions can be drawn.

Research has also shown that autistic people report a more diverse range of gender identities than neurotypical individuals, on average (Bejerot, & Eriksson, 2014; George & Stokes, 2018b). In keeping with these findings, Cooper et al. (2018) found that autistic individuals were significantly more likely to be gender incongruent and to have or be planning a gender transition than neurotypical people. Indeed, Walsh et al. (2018) reported that 15% of autistic individuals who participated in their study reported trans and nonbinary identities. Surprisingly, also Dewinter et

al. (2017) found that 15.4% of the autistic participants who participated in their study reported trans, nonbinary, and other/unknown gender identities. However, the latter study did not include a population-based control group, so meaningful conclusions are difficult to draw from the results. Lastly, Pecora et al. (2020) found that autistic females were less likely to identify with their birth-assigned sex than neurotypical females.

Studies in the Gender Dysphoric/Incongruent Population

Prevalence of ASD Diagnoses

To investigate the prevalence of ASD *diagnoses* in gender dysphoric/incongruent cohorts, researchers have relied on (a) diagnostic instruments for ASD, (b) information obtained from patient files, and (c) self-reported ASD diagnosis. de Vries et al. (2010) published the first quantitative study on the prevalence of ASD diagnoses in gender dysphoric individuals. To date, this is the only study that has employed a clinical diagnostic tool to identify clinically diagnosable ASD in a sample of gender dysphoric/incongruent people. Specifically, de Vries et al. (2010) utilized the Dutch version of the Diagnostic Interview for Social and Communication Disorders-10th revision (DISCO-10) in 26 children and adolescents with suspected ASD who had been referred to a gender identity clinic for GD. The investigators reported that the incidence of ASD was 7.8% in the total sample of gender-referred individuals (N = 204).

Interestingly, among adolescents diagnosed with gender identity disorder, 6.5% received a co-occurring diagnosis of ASD, whereas only 1.9% of children with gender identity disorder were diagnosed with ASD. Turban and van Schalkwyk (2018) argued that since ASD is a neurodevelopmental disorder that is usually detected early in development, the high rates of clinically diagnosable ASD found in

adolescents and not children with gender identity disorder indicate that the diagnostic tool de Vries at al. (2010) used did not tap upon "true" ASD characteristics. Instead, psychosocial issues, such as anxiety and depression, that are particularly common in adolescents with GD might have artificially inflated adolescents' scores on DISCO-10. However, it is important to stress here that in de Vries at al.'s (2010) study only 12.7% of the sample received a diagnostic assessment for ASD. As such, it remains unclear whether all autistic children and adolescents were detected.

Compared to diagnostic instruments for ASD, the analysis of prerecorded, patient-centred data has been more frequently used for the investigation of the prevalence of ASD diagnoses in gender dysphoric/incongruent individuals. In chart reviews, the incidence of a diagnosis of ASD ranged from 3% to 21.3% in gender incongruent and gender-referred children and adolescents (Becerra-Culqui et al., 2018; Chen et al., 2016; Chiniara et al., 2018; Holt et al., 2016; Khatchadourian et al., 2014; Leef et al., 2019; Nahata et al., 2017; Peterson et al., 2017; Shumer et al., 2016; Skagerberg et al., 2015; Spack et al., 2012) and from 4.8% to 7.8% in gender incongruent and gender-referred adults (Cheung et al., 2018; Fielding & Bass, 2018; Heylens et al., 2018). When researchers relied solely upon self-reports, the percentage of gender-referred children and adolescents who reported possession of a diagnosis of ASD was 9.62% (Mahfouda et al., 2019), and the percentage of gender incongruent adults who reported possession of a diagnosis of ASD ranged from 2.7% to 82% (Jones et al., 2012; Kristensen & Broome, 2015; Murphy et al., 2020; Stagg & Vincent, 2019; Warrier et al., 2020).

Prevalence of ASD Caseness

To examine the prevalence of ASD *caseness* in gender dysphoric/incongruent cohorts, researchers have relied on cut-off scores from ASD screening

questionnaires. Studies have shown that the positive rates for ASD range from 14.5% to 68% in gender dysphoric/incongruent children and adolescents (Akgül et al., 2018; Leef et al., 2019; Mahfouda et al., 2019; Shumer et al., 2016; Skagerberg et al., 2015; VanderLaan, Leef, et al., 2015; van der Miesen, de Vries, et al., 2018) and from 1.2% to 40.3% in gender dysphoric/incongruent adults (Heylens et al., 2018; Jones et al., 2012; Kristensen & Broome, 2015; Lehmann et al., 2020; Murphy et al., 2020; Nobili et al., 2020; Nobili, Glazebrook, Bouman, et al., 2018; Pasterski et al., 2014; Stagg & Vincent, 2019; Vermaat et al., 2018). However, the incidence of ASD caseness ranges widely depending on the cut-off scores used. For example, while Pasterski et al. (2014) found that 5.5% of transgender adults diagnosed with GD or gender identity disorder scored ≥ 32 on the Autism-Spectrum Quotient (AQ-50; Baron-Cohen et al., 2001), suggesting clinically significant levels of ASD traits, Kristensen and Broome (2015) reported that 39% of gender-variant adults should be referred for an ASD diagnostic assessment as they scored > 6 on the AQ-10 (Allison et al., 2012).

Prevalence of ASD Traits

To examine the prevalence of ASD *traits* in gender dysphoric/incongruent cohorts, researchers have used parent/self-report measures that index ASD characteristics. VanderLaan, Postema, et al. (2015) and Zucker et al. (2017) assessed circumscribed preoccupations and intense interests (one diagnostic feature of ASD) in children referred to a gender identity clinic, using the items 9 and 66 from the Child Behaviour Checklist or Teacher's Report Form. Both studies found elevated obsession in gender-referred children, compared to nonreferred children and clinic-referred children. A significant increase in compulsion was also reported in gender-referred children, compared to nonreferred children.

van der Miesen, de Vries, et al. (2018) used the Children's Social Behaviour Questionnaire to examine the prevalence of ASD traits in children diagnosed with GD. The study found significantly increased ASD traits in gender dysphoric children, compared to neurotypical children. Another study compared children who satisfied the diagnostic criteria for GD to neurotypical controls, using the Social Responsiveness Scale (Akgül et al., 2018). The study found that the gender dysphoric group had significantly more ASD traits than the control group. Notwithstanding, when Leef et al. (2019) used the same measure to compare children diagnosed with gender identity disorder, GD or gender identity disorder not otherwise specified with clinic-referred children no difference was found in parent-reported ASD traits. However, when they employed the Social Communication Questionnaire to tap ASD traits, they did find elevated ASD traits in children with gender identity disorder, GD or gender identity disorder not otherwise specified. While the results of the aforementioned studies seem to indicate an increased prevalence on ASD traits in gender dysphoric children, the study of this topic in gender dysphoric/incongruent adults has produced mixed results.

In seven out of the eight identified studies that contained data on the prevalence of ASD traits in gender dysphoric/incongruent adults, researchers have used the AQ to measure ASD traits. While Jones et al.'s (2012) study found that transgender men reported significantly more ASD traits than neurotypical birth-assigned males and females, no difference was found between transgender women and either neurotypical birth-assigned males or females. Murphy et al. (2020) and Nobili, Glazebrook, Bouman, et al. (2018) replicated these findings. Likewise, Kung (2020) found that transgender men and nonbinary birth-assigned females reported significantly more ASD traits than control birth-assigned females from the general

population. No difference was observed between either transgender women or nonbinary birth-assigned males and control birth-assigned males from the general population, however. Vermaat et al. (2018) found that birth-assigned females referred for GD reported significantly more ASD traits than control birth-assigned females. A significant difference in ASD traits was also found between birth-assigned females referred for GD and one of the three control samples of birth-assigned males (i.e., Dutch AQ scores) they used, with birth-assigned females scoring higher than birth-assigned males. Birth-assigned males referred for GD scored significantly lower on the AQ than control birth-assigned males and no difference was found between birth-assigned males referred for GD and control birth-assigned females. It is also important to mention that in Pasterski et al.'s (2014) study, transgender birth-assigned females diagnosed with GD/GID scored higher on the AQ-50 than neurotypical birth-assigned females, but the difference was small (*d* = 0.31) and nonsignificant. In contrast, Stagg and Vincent (2019) reported a significant difference in the number of self-reported ASD traits between groups, with transgender and nonbinary individuals reporting more ASD traits than cisgender adults. Results were replicated by Warrier et al. (2020) in the largest study on this topic conducted to date. Using a different self-report measure (i.e., the Social Responsiveness Scale), Heylens et al. (2018) also found increased ASD traits in adults diagnosed with GD, compared to a normative sample.

Part 2: Meta-Analyses of Studies of ASD Diagnoses and ASD Traits in Gender Dysphoric/Incongruent Individuals

Method

Sample of Studies

Our full study selection strategy is described in the method section of Part 1. The inclusion criteria for the meta-analysis of studies of ASD diagnoses remained the same with the ones applied for the literature review (see Part 1). All the studies that reported quantitative results on the prevalence of ASD diagnoses in gender dysphoric/incongruent people were meta-analysed. Warrier et al. (2020) reported evidence from five datasets, all of which were independent from each other. As such, we included them in the current meta-analysis as separate studies.

For the meta-analysis of studies of ASD traits, in addition to the inclusion criteria reported in Part 1, studied should include a *nonclinical* control group (either primary or secondary). Of the studies that reported evidence about the prevalence of ASD traits, we excluded two studies (i.e., VanderLaan, Postema, et al., 2015; Zucker et al., 2017) because they did not employ an ASD screening questionnaire. We also excluded Warrier et al.'s (2020) IMAGE and LifeLines datasets because the data reported did not allow an approximation of a standardized bias-corrected effect size to be calculated. Lastly, we excluded Leef et al.'s (2019) study because their control group was selected from a clinical population. Jones et al. (2012) and Pasterski et al. (2014) took the data of their control group from the same source (i.e., Baron-Cohen, Wheelwright, Skinner, et al., et al., 2001). To satisfy the assumption of independence of effects in the current meta-analysis, we decided to include Jones et al.'s (2012) study based on temporal criteria. Results of the meta-analysis did not change substantively when Pasterski et al.'s (2014) study was included instead (for the results, see Appendix A). Also, results did not change substantively when in Kung's (2020) study we used the control group from Ruzich, Allison, Chakrabarti et al. (2015), instead of the control group from Baron-Cohen et al. (2014) (for the results, see Appendix A).

Meta-Analytic Procedure

To estimate the prevalence of ASD diagnoses in gender dysphoric/incongruent people, we conducted a meta-analysis of proportions. That is a widely used method that aims to provide an accurate estimate of the frequency of a disorder (Barendregt et al., 2013; Lai et al., 2019). In the current meta-analysis, the event rate of gender dysphoric/incongruent people with a diagnosis of ASD reported in each study was transformed into a logit event rate effect size, and the corresponding standard error was calculated. The transformed logit event rates were meta-analysed, using the inverse of the variance of each of the transformed rate as their study weight. The calculated pooled prevalence of ASD diagnoses in the gender dysphoric/incongruent population and its confidence intervals were then retransformed into event rates (Lipsey & Wilson, 2001).

To conduct the meta-analysis of studies containing data on the prevalence of ASD traits in gender dysphoric/incongruent individuals, we used Cohen's *d* as index of standardised mean difference in ASD traits between gender dysphoric/incongruent and nonclinical/population-based control participants. Cohen's *d* was calculated based on means and standard deviations provided by the authors, using Lipsey and Wilson's (2001) web-based effect size calculator. When means and standard deviations were reported only separately for birth-assigned males at birth-assigned females within each group, combined means and standard deviations were calculated using the standard mean and standard deviation formula (Altman et al., 2000). The same formula was also used to combine the groups of transgender and nonbinary people included in Kung's (2020) study into a single group.

To control for an overestimation of Cohen's *d* effect size in studies with small sample size, we calculated Hedges'*g* (Hedges, 1981; Hedges & Olkin, 1985). That is

the unbiased version of Cohen's *d* and is interpreted in a similar way with Cohen's *d* (i.e., $g \geq .20$ = small effect, $g \geq .50$ = moderate effect, $g \geq .80$ = large effect; Cohen, 1988). Inverse variance weights were applied to effect sizes to control for sample size differences between studies (Borenstein et al., 2009; Lipsey &Wilson, 2001). Ninety-five percent confidence intervals were computed as an index of variation of the estimate and the significance of the mean effect size.

The studies we selected to include in the current meta-analyses cannot be considered functionally equivalent, as they utilize a range of measures, samples, and settings. Therefore, we used a random-effects model to compute a weighted pooled estimate of the prevalence of ASD diagnoses in gender dysphoric/incongruent individuals and a weighted mean effect size of the difference in ASD traits between gender dysphoric/incongruent and nonclinical/population-based control participants (e.g., Borenstein et al., 2009; Lipsey & Wilson 2001; Pigott & Polanin, 2020). In the meta-analysis of studies of ASD traits, a positive effect size indicates that relative to neurotypical/population-based control people, gender dysphoric/incongruent individuals have more ASD traits.

To quantify heterogeneity in effect sizes (or else variation in the true effect sizes) a series of measures were employed. We used the *Q* statistic to test the hypothesis that the true effect size is the same across studies. A significant *p*-value provides evidence that studies included in the meta-analysis do not share a common effect size. To examine what proportion of the observed variance reflects variation in the true effect sizes rather than sampling error, we calculated I^2 values, and to identify how much the true effects vary across studies, we estimated prediction intervals (Borenstein et al., 2009; Borenstein et al., 2017; Higgins et al., 2003).

To examine potential sources of heterogeneity, we conducted a series of random-effects categorical analyses and meta-regression analyses. In the categorical analyses we conducted, we compared prevalence estimates of ASD diagnoses and mean differences in ASD traits between clinical-based and population-based studies. In clinical-based studies participants were recruited primarily from specialised clinics providing care to gender dysphoric/incongruent individuals. Whereas in population-based studies, participants were recruited either randomly from the general population or from health care consortiums. Prevalence estimates of ASD diagnoses were also compared among studies that included people referred to clinics/services for gender-related issues (mainly gender dysphoria), studies that included people with a diagnosis of GD, gender identity disorder, or gender identity disorder not otherwise specified, and studies that included gender incongruent people. Furthermore, mean differences in ASD traits were compared between studies that used primary sources of data for the control group and studies that used secondary sources. Lastly, we compared mean differences in ASD traits between studies that included primarily children (mean age < 18) and studies that included primarily adults (mean age > 18). In the meta-regression analysis we conducted, we examined the effect of age (mean) and percentage of birth-assigned males in the prevalence estimates of ASD diagnoses in gender dysphoric/incongruent people.

If data were missing for a potential moderator, that study was excluded from the analysis. When mean age was not reported, the midpoint between the minimum and maximum of the age range was calculated (Lai et al., 2019; Loomes et al., 2017). If the mean age was reported separately for birth-assigned males and birth-assigned females, a combined mean was calculated using the standard mean and standard deviation formula (Altman et al., 2000). In the absence of a good rationale and a

solid theoretical background to assume that there will be greater variation in one group compared to the other group, we used a pooled estimate of variance component to conduct our categorical analyses (e.g., Borenstein et al., 2009).

To detect the effect of publication bias, we conducted a *cumulative meta-analysis*. Studies were added one by one based on their *N* (largest to smallest), and a cumulative meta-analysis was performed with the addition of each study. An increase in the mean effect size with the addition of smaller studies provide evidence for bias (e.g., Borenstein et al., 2009). Lastly, a *one-study-removed analysis* was conducted to assess the influence of any one particular study on the results of the meta-analyses. All the analyses described above were performed using Comprehensive Meta-Analysis Software Version 3 (CMA; Borenstein et al., 2013), and prediction intervals were calculated using the spreadsheet created by Borenstein (2019).

Results

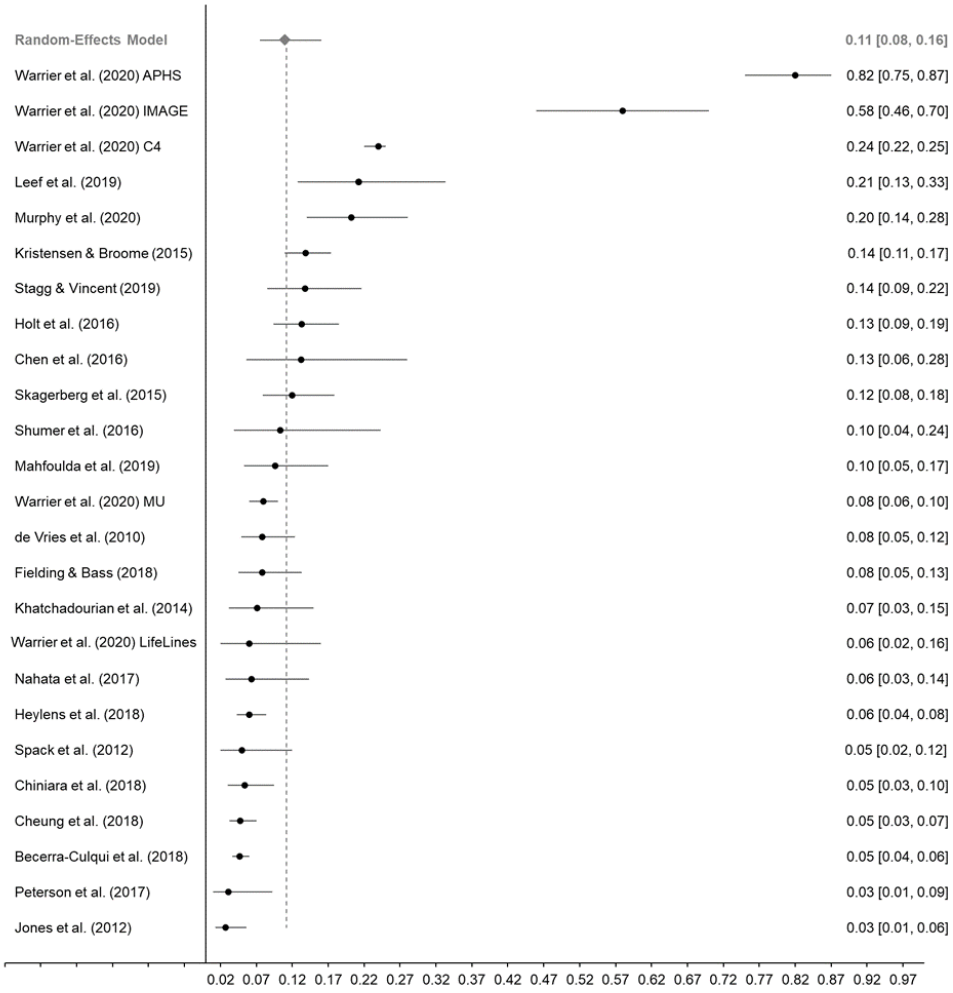
Prevalence of ASD *Diagnoses* in Gender Dysphoric/Incongruent People

A total of 8,662 gender dysphoric/incongruent participants from 25 studies were included in the meta-analysis of studies of ASD diagnoses, using a random-effects model (for a summary of the included studies, see Table A3). Of the 25 studies, 12 were conducted primarily with adults, three were conducted primarily with adolescents, nine were conducted primarily with children and adolescents, and one was conducted with children. Results from the analysis revealed that the pooled prevalence estimate of ASD diagnoses in gender dysphoric/incongruent individuals was 0.11. The 95% confidence interval for the pooled prevalence estimate was 0.08 to 0.16 (z = -9.43, p < .001). A forest plot for this meta-analysis is depicted in Figure 2.

The *Q*-value was 735.61, $df = 24$, $p < .001$, indicating that the effect sizes included in the analysis were significantly different from each other. The I^2 statistic was 96.74, suggesting that 96.74% of the variance in the observed effects reflects variance in true effects rather than sampling error. The variance of true effects (Tau^2) was 1.11, the standard deviation of true effects (Tau) was 1.05, and the prediction interval was 0.01 to 0.54. Results indicate very substantial levels of heterogeneity. To investigate the sources of this heterogeneity, we conducted two random-effects categorical analyses and two random-effects univariable meta-regression analyses.

Figure 2

Forest Plot for Event Rate and 95% Confidence Interval for the Studies of ASD Diagnosis in Gender Dysphoric/Incongruent Individuals Included in the Meta-Analysis



Note. The grey diamond marks the mean weighted effect and its 95% confidence interval.

Moderation Analysis

As Table 2 shows, both participant type and study design were significant categorical moderators. Specifically, the prevalence estimate of ASD diagnoses was higher among gender incongruent participants than among people with a diagnosis of

GD/GID/GID-NOS (gender identity disorder not otherwise specified) and people referred to specialised clinics/services for gender-related issues. Furthermore, the prevalence estimate from clinical-based studies was lower than that from population-based studies. However, the univariable meta-regression analyses we conducted showed that neither the mean age of participants (Q_{model} = 0.38, df = 1, p = .539; $Q_{residual}$ = 692.73, df = 22, I^2 = 96.60, p < .001; R^2 = .00) nor the percentage of birth-assigned males in the sample (Q_{model} = 0.37, df = 1, p = .542; $Q_{residual}$ = 84.45, df = 15, I^2 = 81.81, p < .001; R^2 = .00) predicted significantly the prevalence estimate of ASD diagnoses.

Table 2

Summary of Random-Effects Categorical Analysis Results for the Prevalence of ASD Diagnoses in Gender Dysphoric/Incongruent Individuals

Moderators	*Q*	*df*	*p*	Tau^2	*k*	Prevalence	95% CI	*p*
Participants	7.80	2	.020	0.79				
Referred					9	0.09	[0.05, 0.15]	<.001
Diagnosed					7	0.07	[0.03, 0.13]	<.001
Incongruent					9	0.20	[0.12, 0.31]	<.001
Study design	5.99	1	.014	0.95				
Clinical					15	0.08	[0.05, 0.13]	<.001
Population					9	0.20	[0.12, 0.33]	<.001

Note. Study codes are presented in Table A3. Referred = participants referred to gender identity clinics/services for gender related issues (mainly gender dysphoria); Diagnosed = participants diagnosed with gender dysphoria, gender identity disorder or gender identity disorder not otherwise specified; Incongruent = gender incongruent participants; *Q* = heterogeneity test; *df* = degrees of freedom; Tau^2 = pooled variance component; *k* = number of studies included in each group; prevalence = event rate of ASD diagnoses; 95% CI = 95% confidence interval.

Publication Bias

To examine the impact of publication bias, we conducted a cumulative meta-analysis. We found that the point estimate increased when studies with smaller sample sizes were included (for a forest plot, see Figure A1). Although this provides evidence that studies with small sample size introduced bias, it should be noted that even if the meta-analysis was limited to the first 10 largest studies ($N > 200$), the pooled prevalence estimate of ASD diagnoses in gender incongruent/dysphoric people would be 0.08 (95% CI 0.04 to 0.13).

Sensitivity Analysis

Conducting a one-study-removed analysis, we found that none of the studies had a strong influence on the results of the meta-analysis. The pooled estimates of the prevalence of ASD diagnoses in gender dysphoric/incongruent individuals ranged from 0.10 to 0.12 (for a forest plot, see Figure A2).

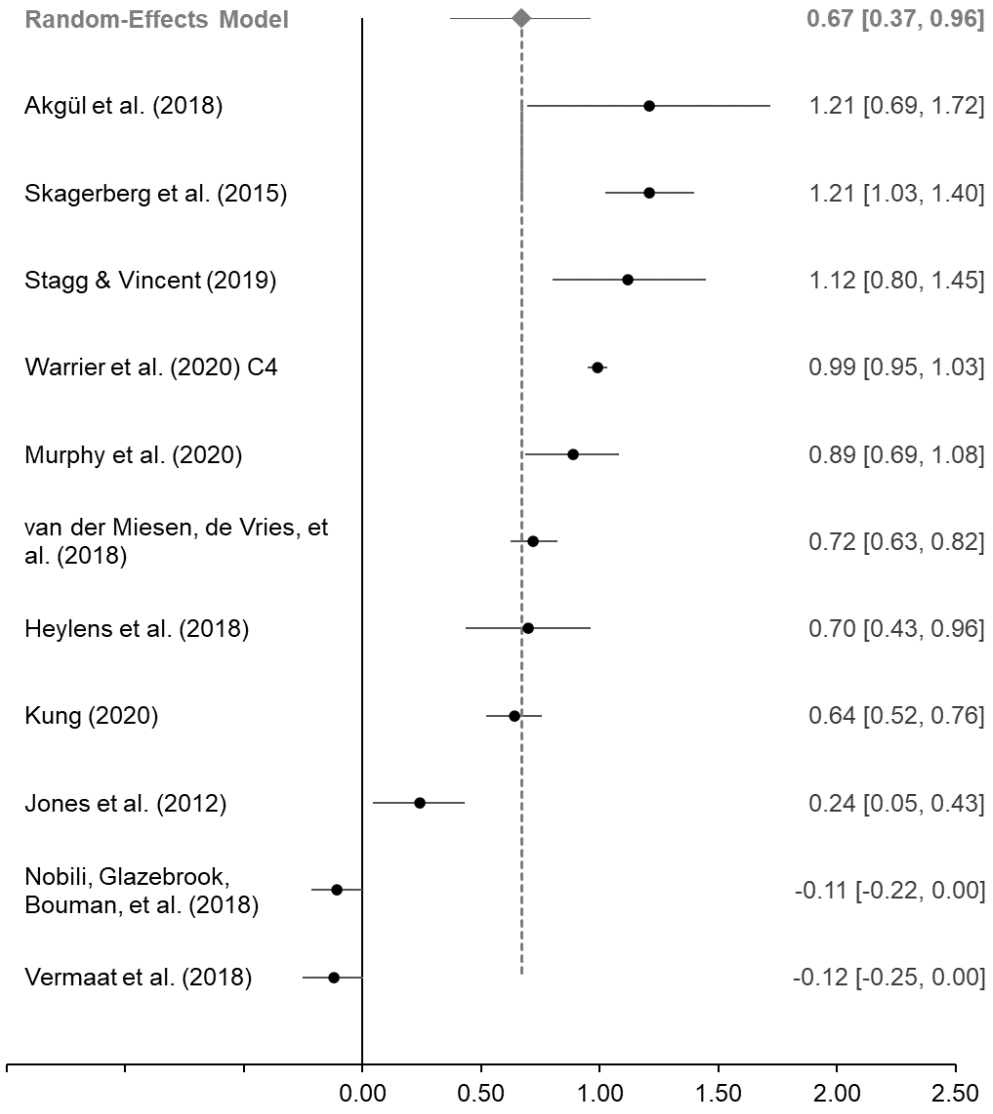
Difference in ASD *Traits* Between Gender Dysphoric/Incongruent and Control Individuals

A total of 4,664 gender dysphoric/incongruent participants and 494,791 neurotypical/population-based control participants from 11 studies were included in the meta-analysis of studies of ASD traits, using a random-effects model (for a summary of the included studies, see Table A4). Of the 11 studies, three were conducted primarily with children and adolescents, and eight were conducted primarily with adults. Results from the analysis revealed that the weighted standardised mean difference in the number of reported ASD traits between gender dysphoric/incongruent and neurotypical/population-based control participants was moderate, $g = 0.67$ ($SE = 0.15$). On average, gender dysphoric/incongruent people reported more ASD traits than neurotypical/population-based control participants.

The 95% confidence interval for the standardised mean difference was 0.37 to 0.96 (z = 4.38, p < .001). A forest plot for this meta-analysis is depicted in Figure 3.

Figure 3

Forest Plot for Effect Sizes (g) and 95% Confidence Interval for the Studies of ASD Traits in Gender Dysphoric/Incongruent Individuals Included in the Meta-Analysis



Note. The grey diamond marks the mean weighted effect and its 95% confidence interval.

The *Q*-value was 608.03 ($df = 10$, $p < .001$), indicating that the true effect size was not identical in all studies. The I^2 statistic was 98.36, which tells us that the 98.36% of the variance in observed effects reflects variance in true error rather than sampling error. The variance of true effects (Tau^2) was 0.24, the standard deviation of true effects (Tau) was 0.49, and the prediction interval was -0.50 to 1.83. Results indicate very substantial levels of heterogeneity. To investigate the sources of this heterogeneity, three random-effects categorical analyses were conducted.

Moderation Analysis

As Table 3 shows, none of the categorical variables we examined were significant moderators. Specifically, there was not a significant difference in the mean effect size between studies that used secondary and primary sources of data for their control groups. Likewise, no significant difference in the mean effect size was found either between clinical-based studies and population-based studies, or between studies conducted among children and adolescents and studies conducted among adult cohorts.

Publication Bias

To examine the impact of publication bias, we conducted a cumulative meta-analysis. We found that the point estimate did not increase when studies with smaller sample sizes ($N < 200$) were included (for a forest plot, see Figure A3). This indicates that there is no reason to assume that the inclusion of studies with smaller sample size has introduced bias.

Sensitivity Analysis

Conducting a one-study-removed analysis, we found that none of the studies had a strong influence on the results of the meta-analysis. The overall weighted effect sizes of the difference in the number of reported ASD traits between gender

dysphoric/incongruent participants and neurotypical/population-based control participants ranged from 0.61 to 0.75 (for a forest plot, see Figure A4).

Table 3

Summary of Random-Effects Categorical Analysis Results for the Prevalence of ASD traits in Gender Dysphoric/Incongruent Individuals

Moderators	*Q*	*df*	*p*	Tau^2	*k*	*g*	*SE*	95% CI	*p*
Age group	1.71	1	.191	0.30					
Child & adolescent					3	1.04	0.33	[0.39, 1.68]	.002
Adult					8	0.54	0.20	[0.15, 0.92]	.006
Control group	0.49	1	.483	0.30					
Secondary					6	0.56	0.23	[0.12, 1.01]	.013
Primary					5	0.80	0.25	[0.31, 1.30]	.002
Study design	1.32	1	.250	0.19					
Clinical					6	0.57	0.19	[0.21, 0.93]	.002
Population					4	0.90	0.22	[0.47, 1.34]	<.001

Note. Study codes are presented in Table S4. Control group = source of data; *Q* = Heterogeneity test; *df* = Degrees of freedom; *p* = Probability value; Tau^2 = Pooled variance component; *k* = number of studies included in each group; *g* = Hedges'*g* Standardised bias-corrected effect size; *SE* = Standard error; 95% CI = 95% Confidence intervals.

General Discussion

The review of the evidence presented in Part 1 as well as the evidence from the meta-analyses presented in Part 2 are suggestive of a link between ASD and gender dysphoria/incongruence. The literature review, specifically, provided consistent evidence about a positive relation between ASD traits and gender dysphoria/variance in the general population (e.g., George & Stokes, 2018b; Kallitsounaki & Williams, 2020a) and a high prevalence of gender dysphoria/incongruence in autistic individuals (e.g., George & Stokes, 2018b; Hisle-

Gorman et al., 2019; Pecora et al., 2020). However, we should note that despite the consistency of the findings, research in these populations is sparse. The bulk of research has focused on the prevalence of ASD/ASD traits in gender dysphoric/incongruent people.

From the studies we reviewed, it was estimated that the positive rates for ASD caseness in gender dysphoric/incongruent people range from 1.2% to 68% (e.g., Akgül et al., 2018; Vermaat et al., 2018). Nonetheless, the variation in ASD screening questionnaires used (AQ, SRS, etc.) and cut-off points used (e.g., AQ score > 26 in some studies, over 32 in other studies etc.) across different studies, does not allow us to make accurate interpretations of these findings. In contrast, the evidence about the rates of autistic gender dysphoric/incongruent people was less obscure and showed an increased prevalence of ASD diagnoses in this population (e.g., Akgül et al., 2018; Kristensen & Broome, 2015; Skagerberg et al., 2015; Warrier et al., 2020). Likewise, the results of the meta-analysis we conducted indicated that ASD frequently occurs in gender dysphoric/incongruent individuals. Specifically, the prevalence of ASD diagnoses in this population was 11 times higher than the ASD prevalence estimate of approximately 1% in the general population (e.g., Lai et al., 2014). We should note, however, that the prediction intervals of the prevalence estimate were very wide, indicating that not all gender dysphoric/incongruent people are affected by ASD to the same degree. Based on the characteristics of the literature pertaining to the prevalence of ASD diagnoses/ASD traits in gender dysphoric/incongruent individuals, as discussed hereunder, wide prediction intervals were expected.

Furthermore, the findings we reviewed indicated that gender dysphoric/incongruent *children* have higher ASD traits than control children (e.g.,

Akgül et al., 2018; Skagerberg et al., 2015). Yet, conflicting evidence emerged about the difference in ASD traits between gender dysphoric/incongruent *adults* and neurotypical/population-based control adults (e.g., Nobili, Glazebrook, Bouman, et al., 2018; Stagg & Vincent, 2019; Warrier et al., 2020). Results of the second meta-analysis we conducted yielded a moderate (g = .67) and significant difference in the number of reported ASD traits between gender dysphoric/incongruent and neurotypical/population-based control individuals, indicating a high prevalence of ASD traits among gender dysphoric/incongruent people. As expected, the prediction intervals were very wide, denoting that not all gender dysphoric/incongruent people report high and clinically significant levels of ASD traits.

An important point to make is that we investigated the impact of a number of potential methodological moderators on the prevalence estimate of ASD diagnoses in gender dysphoric/incongruent people and on the mean difference in the number of ASD traits between gender dysphoric/incongruent and neurotypical/population-based control participants. Results showed that the *study design* and *participant type* were the only significant moderators. The prevalence of ASD diagnoses was lower in clinical-based studies than in population-based studies. Also, the prevalence of ASD diagnoses was lower in people with a diagnosis of GD/GID/GID-NOS and people referred to gender clinics/services for gender-related issues than in gender incongruent people. We should mention here that in clinical-based studies and studies that included people with a diagnosis or symptoms of GD, information about the diagnosis of ASD was collected through medical records and in most of the studies the diagnosis was verified, whereas in studies that included gender incongruent people from the general population information was collected mainly through self-reports. This suggests that the prevalence of ASD diagnoses in gender

incongruent people might be overestimated when ASD diagnosis is self-reported and that clinical-based studies might provide more precise estimates. Alternative explanations, however, are also possible. For example, ASD might be underdiagnosed in clinical setting due to a “different phenotype”. Future studies might usefully investigate whether the presentation of ASD follows similar patterns and trajectories in gender incongruent and cisgender people and whether “camouflaging” is particularly pronounced in the former group (Cook et al., 2021).

To further understand and place the findings of the meta-analyses in context, a number of limitations are discussed. In both meta-analyses, the effect sizes varied substantially across studies. Although we attempted to identify factors that contribute to this variation, only two of the moderation analyses we conducted yielded significant results. We should note, however, that some of the categorical moderation analyses in this study were underpowered (Borenstein et al., 2009; Fu et al., 2011). Therefore, the absence of statistical significance does not provide strong evidence that the factors we examined do not have a contributing role in the observed variation. Furthermore, it is important to stress that the 95% prediction intervals were wide in both meta-analyses. This indicates that some of the future studies on this topic will find effect sizes that denote an overlap between ASD and gender dysphoria/incongruence, yet other studies will not find similar results. Of course, high heterogeneity does not reduce the quality or the importance of a meta-analysis. Rather, it determines the conclusions that can be drawn from the findings and provides a better understanding of the phenomenon under investigation (Berlin, 1995; Lau et al., 1998).

The high heterogeneity observed in the current meta-analyses was not surprising, as it is common in meta-analyses that focus on ASD or gender

dysphoria/incongruence (e.g., Arcelus et al., 2015; Lai et al., 2019; Loomes et al., 2017). It likely reflects some fundamental limitations of the literature pertaining to the prevalence of ASD diagnoses/ASD traits in gender dysphoric/incongruent individuals. First, the targeted population cannot be considered homogeneous, as it includes transgender people, nonbinary individuals, people formally diagnosed with GD, and people who do not conform to the societal expectations of their birth-assigned sex and present gender dysphoric feelings. Future studies might usefully apply stricter eligibility criteria to elucidate which of the aforementioned categories are mostly influenced by ASD. Of equal importance is another limitation identified through the current study. That is the paucity of studies that have employed standardised diagnostic measures of ASD to identify autistic gender dysphoric/incongruent individuals. The vast majority of information about the prevalence of ASD diagnoses in this population has been collected from patient files or it has been obtained by self-reports. As such, essential information about the diagnosis itself is missing. In future studies, it might be useful to collect a copy of the diagnostic report or when this is not possible to collect information about the type of clinician who diagnosed ASD and the type and time of the diagnosis. Along with a formal diagnosis of ASD, standardised diagnostic tools that tap *current* as well as *early* symptoms of ASD (e.g., Autism Diagnostic Observation Schedule and Autism Diagnostic Interview-Revised; Lord et al., 2000; Rutter et al., 2003) could also be used in an attempt to elucidate the high heterogeneity we observe in the prevalence of ASD diagnoses in this population.

We should also note that although results of the second meta-analysis indicate an increased prevalence of ASD traits in gender dysphoric/incongruent people, further research is required to examine whether *neurotypical* gender

dysphoric/incongruent people have increased ASD traits. To date, only four studies have examined this hypothesis. This was achieved either by excluding gender dysphoric/incongruent people with a diagnosis of ASD and reanalysing the data or by including only neurotypical gender dysphoric/incongruent people in their samples (i.e., Akgül et al., 2018; Jones et al., 2012; Murphy et al., 2020; Warrier et al., 2020). Three out of the four studies have shown an increased prevalence of ASD traits in neurotypical gender dysphoric/incongruent people (i.e., Akgül et al., 2018; Murphy et al., 2020; Warrier et al., 2020).

Taken together, results from the literature review and the meta-analyses indicate that the chances there is not a link between ASD and gender dysphoria/incongruence are negligible, yet absolute conclusions about the size of the link cannot be drawn. It is well established that both autistic and gender dysphoric/incongruent individuals are at increased risk of mental health conditions and suicidal behaviour (Grant et al., 2011; Hirvikoski et al., 2016; Hofvander et al., 2009; Holt et al., 2016). When ASD and gender dysphoria/incongruence co-occur in a person, it is possible that the risk to mental health is multiplied. Thus, it is important to understand that link. It is still unclear, for example, whether the overlap between ASD and gender dysphoria/incongruence reflects true comorbidity. As Williams (2017, p. 274) noted, “just because behaviourally-defined disorders A and B co-occur in a person does not mean that the underlying causes of those disorders are the same as the causes of A or B in isolation”.

One explanation for the co-occurrence of ASD with gender dysphoria/incongruence is that some of the core features of ASD predispose an individual to develop gender dysphoric feelings or disidentify with their birth-assigned gender, creating a temporal relation between these conditions (e.g., Leef et

al., 2019). For example, autistic people's difficulty representing mental states (namely mentalising; e.g., Yirmiya et al., 1998) has been proposed as one of the mechanisms that could explain the link between ASD and gender dysphoria/incongruence (Glidden et al., 2016; Jacobs et al., 2014; Van Der Miesen et al., 2016). Recent studies have provided some tentative, preliminary evidence in support of this hypothesis (Kallitsounaki & Williams, 2020a; Kallitsounaki et al., 2021). Of course, reverse causal relations among ASD, gender dysphoria/incongruence, and mentalising are certainly possible (R. J. Walsh, personal communication, 8 September, 2020) and, therefore, these links need further investigation. At a biological level, a potential explanation of the co-occurrence of ASD and gender dysphoria/incongruence among birth-assigned females that needs to be systematically studied is the prenatal exposure to high testosterone levels (e.g., Van Der Miesen et al., 2016).

Furthermore, future research might usefully investigate the development of gender identity in ASD (van Schalkwyk et al., 2015). Research on this topic is surprisingly limited, and many questions remain unanswered. For example, it is unclear whether the development of gender identity in autistic and neurotypical children follows the same cognitive and developmental pathways. This is important, because it could provide a unique insight into the mechanisms that are involved in the formation and consolidation of gender identity in cisgender and gender dysphoric/incongruent individuals. We also hope that by establishing a link between ASD and gender dysphoria/incongruence, the current study will give new impetus to future research on the size of the link and on potential aetiological mechanisms that could explain this phenomenon. Although a number of explanations have been suggested for the co-occurrence of ASD and gender dysphoria/incongruence (for a

review, see Van Der Miesen et al., 2016), there is a paucity of research on aetiological mechanisms. Establishing that the link between ASD and gender incongruence/dysphoria is "real" at the behavioural and cognitive level, will allow clinicians to start tracking the outcomes of treatment/support at a more fine-grained level (i.e., by group) rather than as a whole. Ultimately, this will promote the development of tailored services and interventions for autistic gender dysphoric/incongruent people.

It is also important to note that the high co-occurrence between ASD and gender dysphoria/incongruence is underrecognised among health care professionals (Murphy & Livesey, 2017). Evidence about a link between ASD and gender dysphoria/incongruence might stimulate the development of appropriate trainings to raise their awareness (Strauss, et al., 2021) so that gender dysphoric/incongruent people are screened for ASD and autistic people for gender related issues (Janssen et al., 2016; Mahfouda et al., 2019; Strang, Meagher, et al., 2018). Gender related issues in autistic people could be detected by using self/parent-report measures, such as the Gender Identity/Gender Dysphoria Questionnaire for Adolescents and Adults (Deogracias et al., 2007) and the Gender Identity Questionnaire for Children (Johnson et al., 2004) and by including a few gender-related questions on a clinical intake form or a clinical interview (Strang, Meagher, et al., 2018). We should note, however, that these self/parent-report measures have not been validated in the autistic population, so they should be used with caution. Autistic people with suspected GD should be referred to gender specialists for further assessment and support (Strang, Meagher, et al., 2018). It is essential to provide timely support and care to these people, as the burden they suffer seems to be doubly distressing (George & Stokes, 2018a; Hall et al., 2020).

In conclusion, this is the most up-to-date systematic review of the literature pertaining the overlap between ASD and gender dysphoria/incongruence and the first meta-analysis of the prevalence of ASD diagnoses and ASD traits in gender dysphoric/incongruent people. The findings of the current literature review and meta-analyses suggest that (a) there is a positive relation between ASD traits and gender dysphoric/incongruent feelings among people from the general population, (b) an increased prevalence of gender dysphoria/incongruence in the autistic population, and (c) an increased prevalence of ASD diagnoses and ASD traits in the gender dysphoric/incongruent population. Overall, these findings suggest the existence of a link between ASD and gender dysphoria/incongruence that warrants the investigation of mechanisms that could underpin that link and the intensification of clinical attention to autistic gender dysphoric/incongruent individuals.

Chapter 3

Mentalising Moderates the Link between ASD Traits and Current Gender Dysphoric Feelings in Primarily Non-Autistic, Cisgender Individuals

The previous chapter provided an overview of the literature pertaining the suggested overlap between ASD and gender dysphoria/incongruence and the results of two meta-analyses. In the systematic literature review, we included among other studies those that examined the relation between ASD traits and gender dysphoria/incongruence in the general population. Two of these studies were conducted as part of this project (i.e., Kallitsounaki & Williams, 2020a; Kallitsounaki et al., 2021). This chapter presents the study conducted by Kallitsounaki and Williams (2020a).[7] In this study, we adopted an individual differences approach to examine: (1) the relation between ASD traits, on the one hand, and gender dysphoric feelings and recalled childhood gender-typed behaviour, on the other hand, (2) the relation between mentalising and gender dysphoric feelings, and (3) the role of mentalising in the relation between ASD traits and gender dysphoric feelings.

As already mentioned in the previous chapters, despite clear phenotypic differences between ASD and gender dysphoria/incongruence, recent research findings suggest the existence of a link between them (e.g., Strang, Janssen, et al., 2018; van der Miesen, Cohen-Kettenis, et al., 2018). Within the autistic population, retrospective chart reviews have shown that affected people are significantly more likely than neurotypical individuals to express the wish to be the other binary gender (Janssen et al., 2016; May et al., 2017; Strang et al., 2014; van der Miesen, Hurley, et al., 2018). Furthermore, George and Stokes (2018b) used the Gender Identity/Gender

[7] Note that the study conducted by Kallitsounaki et al. (2021) is presented in Chapter 4.

Dysphoria Questionnaire for Adolescents and Adults (Deogracias et al., 2007) and found that autistic adults reported significantly increased gender dysphoric feelings relative to neurotypical individuals. Within the gender dysphoria population, research has shown that the rate of a diagnosis of ASD is significantly higher than the population estimate of ASD (e.g., de Vries et al., 2010; Heylens et al., 2018), which is approximately 1% (e.g., Baird et al., 2006). Compared to neurotypical individuals, an increased number of ASD traits has also been found in gender dysphoric/incongruent people (e.g., Akgül et al., 2018; Nobili, Glazebrook, Bouman, et al., 2018; van der Miesen, de Vries, et al., 2018; but see Turban 2018 and Turban & van Schalkwyk, 2018).

The above studies involved diagnosed or clinically referred cases of ASD and gender dysphoria/incongruence, respectively, using a variety of research designs. Yet, both ASD and gender dysphoria/incongruence are considered dimensional conditions (e.g., Constantino & Todd, 2003; Ehrensaft, 2018; Ronald et al., 2006). As such, adopting an individual differences approach among individuals from the general population can tell us something important about the nature of ASD and gender dysphoria/incongruence themselves (e.g., Nicholson et al., 2018; Williams, Bergström, & Grainger, 2018; Williams, Nicholson, & Grainger, 2018; Williams, Nicholson, Grainger, Lind, et al., 2018).

To date, only one study has examined the overlap between ASD and gender dysphoria/incongruence in adults from the general population. George and Stokes (2018b) found a positive and significant association between current gender dysphoric feelings and number of self-reported ASD traits as measured with the Autism-Spectrum Quotient (AQ; Baron-Cohen, Wheelwright, Skinner, et al., et al., 2001) among neurotypical individuals with an AQ score below 32. One aim of the

current study was to attempt to replicate George and Stokes' (2018b) finding of a significant association between number of ASD traits and current gender dysphoric feelings among adults from the general population. Given concerns about the reliability of results in psychological research, independent replication serves a crucial function in the discipline (e.g., Amir & Sharon, 1990; Makel et al., 2012; Pashler & Wagenmakers, 2012). The current study also moved beyond George and Stokes' by including a second behavioural measure that assessed *retrospective* childhood gender-typed behaviour, rather than only *current* gender dysphoric feelings. Such a historical approach would increase our confidence that gender dysphoric traits measured in the current study reflect persistent gender identity difficulties that started early in life. Perhaps most important, the current study moves beyond previous studies in this domain by including an objective cognitive measure of mentalising.

What relatively little research has been conducted on this topic appears to provide evidence of a link between ASD and gender dysphoria/incongruence, but there is an ongoing debate on whether that link is anything more than superficial (Strang, Janssen, et al., 2018; Turban, 2018; Turban & van Schalkwyk, 2018; van der Miesen, Cohen-Kettenis, et al., 2018). Indeed, behavioural overlap between two conditions represents only a weak source of evidence that the two conditions are truly comorbid, because any given behaviour (including self-reported behavioural traits) can have a number of different underlying causes (see Williams, 2017; Williams et al., 2008; Williams & Lind, 2013).

Arguably, it is only if the overlapping behavioural features have the same underlying cause that we should consider the two conditions comorbid and as Williams (2017, p. 274) noted, "just because behaviourally-defined disorders A and

B co-occur in a person does not mean that the underlying causes of those disorders are the same as the causes of A or B in isolation". For example, it may be that when the behavioural features of GD are apparent in a person with a primary diagnosis of ASD, those features have a different underlying cause (cognitive/neurobiological/genetic/environmental) to the underlying cause of behavioural features of GD in a person with a diagnosis of GD. It may be, for example, that the ASD itself predisposes affected individuals to develop gender dysphoric features (or vice versa). In that case, GD in people with ASD would represent a "phenomimic" of GD (see Bishop 2006, 2010). In other words, GD might be a different entity that just appears at the behavioural level to be the same in both ASD and GD, because it is defined by behaviour only. Behaviour is a sub-optimal means of diagnosing disorders, and so we may be dealing with two different entities that just appear to be similar.

At the psychological level, one candidate mechanism proposed as an explanation for the apparent overlap between ASD and gender dysphoria/incongruence is diminished *mentalising* (Glidden et al., 2016; Jacobs et al., 2014; Van Der Miesen et al., 2016; van der Miesen, Hurley, et al., 2018). Mentalising is the ability to represent mental states in order to explain and predict behaviour (Premack & Woodruff, 1978). It is widely considered to be diminished in autistic people and to contribute to the social-communication impairments that are diagnostic of the disorder (see Brunsdon & Happé, 2014). Moreover, mentalising difficulties may qualify as a cognitive marker of ASD (Gliga et al., 2014). Some have proposed that mentalising may be diminished in people with GD (Jacobs et al., 2014), but this hypothesis had not been tested until very recently. Stagg and Vincent

(2019) found that neurotypical individuals had significantly better mentalising ability than nonbinary people, but equivalent to transgender individuals.

Theoretically, diminished mentalising could contribute to gender dysphoria/ incongruence in a number of ways. First, it could influence gender constancy, which is the representation of gender as a construct that can remain stable even when temporary changes in physical appearance take place (e.g., a boy does not become a girl as soon as he grows long hair or puts on a dress). Gender constancy is central to theories of gender self-concept development (e.g., Kohlberg 1966) and bears close resemblance to the ability to distinguish between appearance and reality, which is a core aspect to mentalising ability. Indeed, among neurotypical children, mentalising ability is associated positively with performance on tests of gender constancy (Trautner et al., 2003; Zmyj & Bischof-Köhler, 2015), and negatively with use of gender stereotypes (Rizzo & Killen, 2018). Second, any internalisation of gender-related traits requires an accurate awareness of those attributes in others. It seems theoretically plausible to suggest that the development of gender self-concept would be affected by a difficulty representing gender-related internal states/traits in others. Third, it is well known that diminished mentalising leads to reduced experience of self-conscious emotions (e.g., embarrassment/shame) (Hobson et al., 2006). Self-conscious emotions contribute to a desire for social conformity (we conform to society's expectations in part to avoid the feelings of embarrassment/shame that results from others judgments when we do not conform). As such, these emotions may lead to a discontinuation of gender incongruent behaviours in children who have a tendency toward an incongruent identity.

In the current study, we used an individual differences approach to investigate whether mentalising plays a role in the suggested link between ASD and

gender dysphoria/incongruence. Participants completed online a self-report measure of ASD traits (the AQ; Baron-Cohen, Wheelwright, Skinner, et al., 2001) and the Reding the Mind in the Eyes test (RMIE; Baron-Cohen, Wheelwright, Hill, et al., 2001), which is a widely used cognitive-experimental task of mentalising. In addition, they completed a self-report measure of retrospective childhood gender-typed behaviour (the Recalled Childhood Gender Identity/Gender Role Questionnaire; RCGI; Zucker et al., 2006) and a self-report measure of gender dysphoric feelings (the GIDYQ-AA; Deogracias et al., 2007).

First, we aimed to examine the extent to which the number of self-reported ASD traits is associated with *recalled* childhood gender-typed behaviour and/or *current* gender dysphoric feelings. To our knowledge, no study has examined the link between ASD and retrospective childhood gender-typed behaviour. We predicted that AQ score would be negatively associated with both RCGI and GIDYQ-AA score (lower scores on RCGI and GIDYQ-AA are indicative of less recalled childhood gender-typed behaviour and more current gender dysphoric feelings, respectively). Second, given the theoretical link between mentalising and the gender self-concept, we predicted that RMIE score would be positively associated with both RCGI and GIDYQ-AA score. Third, we tested the hypothesis that mentalising modulates the predicted association between ASD traits and gender dysphoric feelings by conducting a moderation analysis. Moderation analysis examines whether the direction or strength of the relation between two variables is influenced by a third variable. In the current study, we predicted that performance on the mentalising task would moderate the relation between ASD traits and current gender dysphoric feelings.

Method

Participants and Procedure

One hundred and one adults (50 birth-assigned female) participated in the current study through the Amazon's online crowdsourcing platform MTurk. The average age of the sample was 36.93 (*SD* = 10.11; range = 22 to 70) years. No participant reported incongruence between their birth-assigned sex and their experienced/reported gender. Ninety-four percent of participants reported being native English speakers. Thirteen participants had a formal diagnosis of ASD, according to self-report. All participants took part after they had given written, informed consent and received compensation for their time. This study was approved by the University of Kent's Psychology Research Ethics Committee (ID: 201815434977895393).

Measures

Autism-Spectrum Quotient

The AQ; (Baron-Cohen, Wheelwright, Skinner et al., 2001) is a reliable and valid self-report questionnaire that measures ASD traits in clinical and nonclinical populations (e.g., Ruzich, Allison, Smith, et al., 2015; Williams, Nicholson, & Grainger, 2018). Participants are presented with 50 self-referential statements (e.g., "I find social situations easy"), and they are asked to indicate their agreement with each statement, using a 4-point Likert scale that ranges from "definitely agree" to "definitely disagree". The AQ sum score ranges from 0 to 50, and a value of 26 or above indicates a clinically significant number of ASD traits (Woodbury-Smith et al., 2005). The sensitivity of the cut-off ranges from 88%–95% in people with a diagnosis of ASD, and the specificity ranges from 52%–80% in people from the general population (Booth et al., 2013; Woodbury-Smith et al., 2005). The test-retest

reliability scores of the AQ range from .70 to .95 (e.g., Baron-Cohen, Wheelwright, Skinner et al., 2001; Broadbent et al., 2013), and it has shown convergent validity with the Social Responsiveness Scale in clinical (r = .64; Armstrong & Iarocci, 2013) and nonclinical samples (r = .55; Ingersoll et al., 2011).

Reading the Mind in the Eyes

The RMIE (Baron-Cohen, Wheelwright, Hill, et al., 2001) is a widely used measure of mentalising. Participants are presented with a series of 36 photographs each showing the eye region of people who are experiencing a particular emotion/thought. Participants are asked to choose which one of four presented words best describes the emotional/mental state of the depicted person. Scores range from 0 to 36, with higher scores indicating better task performance. The task has been employed in over 250 studies and shows acceptable test-retest reliability of over .60 in most studies (e.g., Dehning et al., 2012; Fernández-Abascal et al., 2013; Voracek & Dressler, 2006), including over long periods of time (e.g., Fernández-Abascal et al., 2013 report an intraclass coefficient of .63 for 1 year test-retest reliability). Task performance clearly distinguishes groups of participants with and without ASD, even when participant groups are closely matched for verbal ability (e.g., Nicholson et al., 2019; Williams, Nicholson, & Grainger, 2018) and is associated negatively with the number of ASD traits shown by individuals in large population studies (e.g., Baron-Cohen, Wheelwright, Skinner, et al., 2001). Performance on the task is also correlated with performance on other measures of mentalising, even after the influence of IQ is controlled statistically (e.g., Jones et al., 2018) and is associated with activation of a well-established mentalising network of brain regions including the temporo-parietal junction and medial prefrontal cortex (for a meta-analysis, see Schurz et al., 2014).

The Recalled Childhood Gender Identity/Gender Role Questionnaire

The RCGI (Zucker et al., 2006) is a 23-item self-report measure that provides a retrospective assessment of childhood gender-typed behaviour and closeness to parents in the first 12 years of life (e.g., "As a child, my favourite playmates were: a. always boys, b. usually boys, c. boys and girls equally, d. usually girls, e. always girls, f. I did not play with other children"). For the purposes of the current study, participants were asked to respond only to the 18 items comprised factor one in the original Zucker et al.'s (2006) study, using a 5-point scale. These items particularly assess gender role behaviour and gender identity. The questionnaire has parallel male and female versions and as such, participants completed the version according to their birth-assigned sex. A mean score was calculated for each participant, with means scores ranging from 1 to 5 and with lower scores denoting less childhood gender-typed behaviour. With respect to its psychometric properties, RCGI has shown convergent validity with the GIDYQ-AA, r = .70 (Singh et al., 2010).

Gender Identity/Gender Dysphoria Questionnaire for Adolescents and Adults

The GIDYQ-AA (Deogracias et al., 2007) is a 27-item self-report measure of gender identity and gender dysphoria. The GIDYQ-AA presents participants with a series of questions about their feelings, wishes, thoughts, and behaviours regarding their birth-assigned sex and their experienced/reported gender within a 1 year period (e.g., "In the past 12 months, have you felt uncertain about your gender, that is, feeling somewhere in between a woman and a man?"). The questionnaire has two analogous versions, one for birth-assigned males and one for birth-assigned females. In the current study, participants completed the version that was consistent with their birth-assigned sex and responded to each of the items, using a 5-point scale. A mean score was calculated for each participant, with scores ranging from 1 to 5 and with

lower scores indicating increased gender dysphoric feelings. A mean score of 3 is the cut-off point that indicates clinically significant levels of gender dysphoria (Deogracias et al., 2007). The original validation study showed that this self-report measure is reliable (Cronbach's alpha = .97) and the sensitivity of its cut-off point is 90.4% among people with a diagnosis of GD and its specificity 99.7% among a university student sample (Deogracias et al., 2007).

Statistical Analysis

According to a well-documented dimensional approach (namely "broad autism phenotype"), ASD is a spectrum disorder that ranges from people in the general population with low levels of ASD traits to people who hold a clinical diagnosis of ASD (e.g., Bolton et al., 1994; Goldberg et al., 2005; Le Couteur et al., 1996; Murphy et al., 2000; Pickles et al., 2000; Piven et al., 1997; Szatmari et al., 2000). Therefore, in the current study all participants were included in the analyses described below, independently on whether they reported possession of a formal diagnosis or not (for the results of the analyses when autistic participants were excluded from the sample, see Appendix B). An alpha level of .05 was used to determine statistical significance.

To examine the relations between ASD traits, recalled childhood gender-typed behaviour, current gender dysphoric feelings, and mentalising, a series of zero-order correlations was conducted involving scores on the AQ, RCGI, GIDYQ-AA, and RMIE. Following the suggestion of an anonymous reviewer of the published version of this chapter, a series of post-hoc correlation analyses was conducted among AQ subscales, GIDYQ-AA, and RCGI (for the results, please see Appendix B). Coefficients *r* are reported as measures of effect size (≥ .10 = small effect, ≥ .30 = moderate effect, ≥ .50 = large effect; Cohen, 1992). Furthermore, a moderation

analysis was employed to test the hypothesis that RMIE moderates the predicted relation between ASD traits and current gender dysphoric feelings. To perform this analysis we used PROCESS v3.3 operated in SPSS, employing bootstrapping with 5,000 resamples (Hayes, 2018). To mitigate the potential risk of multicollinearity, products of interactions were mean centred prior to the analysis (Dawson, 2014).

In moderation analysis, it is crucial to investigate whether there is a significant interaction between a focal variable (F) and a moderator variable (M), when predicting an outcome (Y) variable (Hayes & Matthes, 2009). In our moderation analysis, we examined whether there was a significant interaction between AQ score (F) and performance on the RMIE (M), when predicting current gender dysphoric feelings (Y). If so, this result would imply that the predicted association is present to a lesser degree (or not at all) in participants with a high RMIE score than people with a low RMIE score (or vice versa).

According to standard practice, in order to understand the conditions, or else the values of the moderator under which the relation between a focal variable and an outcome variable holds its statistical significance or its effect size, the significant interaction should be further probed (Hayes & Matthes, 2009). The "gold standard" method to probe the source of a significant interaction effect in a moderation analysis, *without* losing power by dichotomising continuous predictors, is to perform simple slopes analysis (Field, 2013; Hayes & Matthes, 2009). To do so, we employed the widely used "pick-a-point" approach (e.g., Bauer & Curran, 2005; Dawson & Richter, 2006), which involves probing interactions at one standard deviation above (+1SD) and below (-1SD) the mean for the moderator (Aiken et al., 1991).

In a series of exploratory analyses, we also compared the data from the 13 participants who reported possession of a formal diagnosis of ASD to the data from

the remaining 88 non-autistic participants. To examine between-group (autistic/non-autistic) differences in the number of self-reported ASD traits, recalled gender-typed behaviour, current gender dysphoric feelings, and in performance on the RMIE, independent samples *t*-tests were conducted. In cases when the homogeneity of variance assumption was violated results from the Welch's *t*-test were reported (Field, 2013). Cohen's *d* values were reported as measures of effect size (≥ 0.20 = small effect, ≥ 0.50 = moderate effect, ≥ 0.80 = large effect; Cohen, 1969). Finally, a Fisher's exact test was also employed to explore the extent to which there was a significant association between the diagnostic group (autistic/non-autistic) and GIDYQ-AA score (above clinical threshold/below clinical threshold). A phi coefficient was reported as measure of effect size. Its values fall between 0 and 1 and is interpreted as coefficient *r*.

Results

Table 4 presents the results from the correlation analyses conducted to examine the relations between ASD traits, recalled childhood gender-typed behaviour, current gender dysphoric feelings, and mentalising. As predicted, AQ score was negatively and significantly correlated with both RCGI and GIDYQ-AA mean score (i.e., the more ASD traits, the less recalled gender-typed behaviour and the more current gender dysphoric feelings). Furthermore, RMIE score was positively and significantly correlated with RCGI score (i.e., the poorer the RMIE performance, the less recalled gender-typed behaviour), but this was the only association that lost its significance when participants who reported a formal diagnosis of ASD were excluded from the analysis (see Appendix B). There was also a large and significant positive correlation between performance on the RMIE task

and GIDYQ-AA score (i.e., the poorer the RMIE performance, the more the current gender dysphoric feelings).

Table 4

Bivariate Correlations Between AQ, GIDYQ-AA, RCGI, and RMIE

Variable	1	2	3	4
1. AQ	-			
2. GIDYQ-AA	-.32**	-		
3. RCGI	-.33**	.53***	-	
4. RMIE	-.18*	.70***	.33**	-

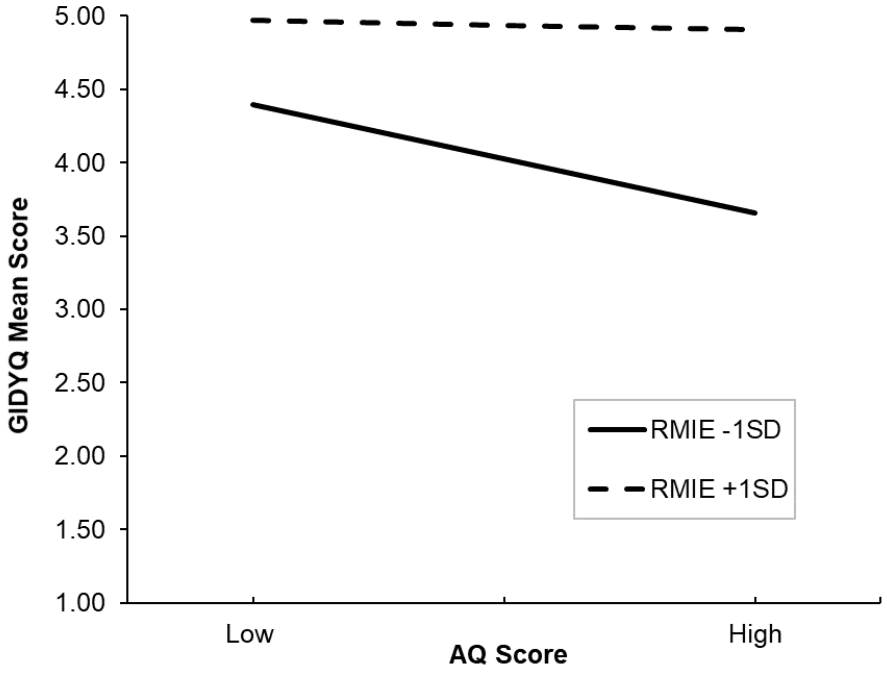
Note. N = 101; AQ = Autism-Spectrum Quotient; GIDYQ-AA = Gender Identity/Gender Dysphoria Questionnaire for Adolescents and Adults (low scores = more gender dysphoric feelings); RCGI = The Recalled Childhood Gender Identity/Gender Role Questionnaire (low scores = less gender-typed behaviour); RMIE = Reading the Mind in the Eyes test. *p < .05 (one-tailed). **p < .01. ***p < .001.

Next, we examined whether the association between AQ total score and GIDYQ-AA mean score was moderated by performance on the RMIE test. The overall model of AQ score, RMIE score, and their interaction was significant, $F(3, 97) = 42.51$, $p < .001$, $R^2 = .57$, and the interaction added $R^2 = .04$ above the main effects. AQ score negatively predicted GIDYQ-AA, $b = -0.03$, $t(97) = -3.72$, $p < .001$, and performance on RMIE positively predicted GIDYQ-AA, $b = 0.06$, $t(97) = 8.78$, $p < .001$. As expected, the interaction between AQ and RMIE also predicted GIDYQ-AA, $b = 0.003$, $t(97) = 2.94$, $p = .004$, indicating that mentalising moderated significantly the relation between ASD traits and current gender dysphoric feelings. Figure 4 depicts the effect of interaction on GIDYQ-AA. Simple slopes analysis showed that when performance on RMIE was low (-1SD), AQ score predicted

negatively and significantly current gender dysphoric traits, b = -0.05, $t(97)$ = -4.02, p < .001, whereas, when it was high (+1SD), AQ score did not predict current gender dysphoric traits significantly, b = -0.004, $t(97)$ = -0.49, p = .625.

Figure 4

Regression Plot Showing the Interaction Between ASD Traits (AQ) and High/Low Performance on RMIE on Current Gender Dysphoric Feelings (GIDYQ-AA)



Exploratory Analyses

Table 5 presents mean (*SD*) scores on the AQ, RCGI, GIDYQ-AA, and RMIE among cisgender individuals who reported a formal diagnosis of ASD and those who did not. In line with the findings from multiple previous studies, relative to comparison participants, autistic cisgender adults showed significantly higher AQ scores (in keeping with their diagnosis) and significantly lower RMIE scores (suggesting a mentalising difficulty). More important for the current investigation,

autistic cisgender participants reported significantly less childhood gender-typed behaviour and significantly more current gender dysphoric feelings than comparison participants.

Table 5

Means (SDs) and Inferential Statistics for Group Differences

Variable	Diagnostic Group		Group Differences		
	Autistic (n = 13)	Non-autistic (n = 88)	t	p	d
AQ	24.92 (5.56)	18.88 (7.24)	2.88	.005	0.94
GIDYQ-AA	3.27 (0.81)	4.62 (0.55)	-7.72	<.001	1.95
RCGI [a]	3.05 (0.37)	3.77 (0.60)	-5.97	<.001	1.45
RMIE	14.15 (7.07)	25.50 (6.52)	-5.79	<.001	1.67

Note. AQ = Autism-spectrum Quotient; GIDYQ-AA = Gender Identity/Gender Dysphoria Questionnaire for Adolescents and Adults; RCGI = The Recalled Childhood Gender Identity/Gender Role Questionnaire; RMIE = Reading the Mind in the Eyes test.

[a] Welch's t test.

Across the total sample (N = 101), there were nine participants who scored less than or equal to 3 on the GIDYQ-AA, denoting clinically significant levels of gender dysphoria. Seven (77.78%) of these nine participants reported being in receipt of a formal diagnosis of ASD (see Table 6). A Fisher's exact test indicated that there was a significant association between the diagnostic group (autistic/non-autistic) and GIDYQ-AA score (above clinical threshold/below clinical threshold), $p < .001$, phi = .61. The odds of denoting clinically significant levels of gender dysphoria were 50.17

times (95% CI: 8.49 to 296.31) greater for autistic cisgender participants, compared to non-autistic participants.

Table 6

Contingency Table of Participants who Scored Either Below or Above the Threshold on GIDYQ-AA by Group

Group	Gender dysphoric feelings	
	Above threshold	Below threshold
Autistic	6 (6.52%)	7 (77.78%)
Non-autistic	86 (93.48%)	2 (22.22%)
Total	92 (100%)	9 (100%)

Note. $N = 101$. Above threshold = more than 3; Below threshold = less than or equal to 3.

Discussion

In keeping with our predictions, this study found that AQ score was significantly associated with both the RCGI and the GIDYQ-AA score, indicating that the more an individual's ASD traits, the less their recalled gender-typed behaviour in childhood *and* the more their current gender dysphoric feelings tended to be. To our knowledge, this is the first study to replicate George and Stokes (2018b) findings of a significant relation between ASD traits and current gender dysphoric feelings in adults from the general population. It is also the first study to demonstrate a link between ASD traits and *recalled* gender-typed behaviour. This highlights a developmental continuity of the association, which is present in both children (Nabbijohn et al., 2018) and adults (current study and George & Stokes, 2018b). We also found that RMIE task performance was significantly associated

with both the RCGI and the GIDYQ-AA score (poorer mentalising = less recalled childhood gender-typed behaviour and more current gender dysphoric feelings). Furthermore, RMIE task performance moderated significantly the relation between ASD traits and current gender dysphoric feelings. Specifically, only when mentalising ability was relatively low was there a significant association.

The results from exploratory group contrasts complemented results from the main association/moderation analyses; cisgender participants who identified as having a diagnosis of ASD showed significantly diminished performance on the RMIE task and (clinically and statistically) significantly elevated levels of gender dysphoric feelings. Of course, the results from these exploratory analyses should be treated with caution, given the small number of ASD cases involved and the fact that participants were recruited via opportunity sampling, which may have biased selection (i.e., perhaps those individuals with ASD who also had gender identity difficulties were attracted to take part in the study, given its focus).

From a theoretical perspective, the results of the current study come to add to the discussion on whether the link between ASD and gender dysphoria/incongruence exists purely at a behavioural level (Turban, 2018; Turban & van Schalkwyk, 2018). Our findings indicate that mentalising *moderates* the relation between ASD traits and gender dysphoric feelings. As such, it could be argued that in ASD, a weakness in the process of mentalising may contribute to increased fluidity of gender identity. A child in whom the process of mentalising is diminished/atypical is likely to experience elevated feelings of gender fluidity, as they fail to internalise a typical proportion of the attributes that stereotypically define their biological sex. It is also possible that this child, in whom the experience of guilt and embarrassment is

reduced, will be less affected by societal pressures and prejudices and therefore less likely to discontinue cross-gender behaviour as they develop cross-gender interests.

Nonetheless, it is unclear whether this approach accounts for the conviction and conduct of those who assume gender roles out of keeping with their biological sex (i.e., gender incongruent individuals/individuals diagnosed with GD). Could it be the case that increased ASD traits in gender dysphoric/incongruent people reflect genuine ASD features *only* when mentalising difficulties are present? This question remains open until directly investigated. Given that mentalising can be considered as a cognitive marker of ASD (e.g., Gliga et al., 2014), it could be argued that by measuring mentalising we tap a core component of ASD itself. This could provide an alternative explanation of why mentalising moderated the relationship between ASD traits and gender dysphoric feelings in the current study.

It is important to highlight here that the investigation of mechanisms that could explain gender dysphoria/incongruence and/or its co-occurrence with ASD is a very sensitive topic and, as such, it should be treated with caution. Ideally, participatory research methods should be employed in studies that could have a direct impact on people's lives (e.g., Strang et al., 2019). Due to limited resources this was not possible in the current project.

One thing to note is that that participant recruitment was conducted using an online platform. Although online studies yield results that are equivalent in terms of their reliability with other widely employed methods of data collection (e.g., Buhrmester et al., 2011), firm conclusions might await independent replication of the current findings in studies that employ multiple methodological approaches and sampling procedures. Moreover, future research might usefully examine the extent to which ASD and gender dysphoria/incongruence share underlying causes at more

than one levels of explanation (biological, genetic, environmental), in order to provide insight on the extent to which there is true comorbidity between them (Williams & Lind, 2013).

In conclusion, the current study shows for the first time (a) an association between ASD traits and recalled childhood gender-typed behaviour, (b) an association between gender dysphoric feelings and mentalising, and (c) that the link between ASD and current gender dysphoric feelings is moderated by mentalising ability in the general population.

Chapter 4

Links Between ASD Traits, Feelings of Gender Dysphoria, and Mentalising Ability: Replication and Extension of Previous Findings From the General Population

The findings presented in the previous chapter provide further support to the hypothesis of a link between ASD and gender dysphoria/incongruence and indicate that mentalising may play a role in this link. Given the tentative and preliminary nature of these findings, however, we attempted to replicate and extend them further. This chapter presents the results of this attempt (for the published version of this chapter, see Kallitsounaki et al., 2021).

In recent years evidence has accumulated for a high co-occurrence of ASD and gender dysphoria/incongruence. Research has shown that autistic adults present significantly more gender dysphoric feelings than people from the general population (George & Stokes, 2018b), are more likely to express the wish to be the gender opposite to their birth-assigned sex (van der Miesen, Hurley, et al., 2018), and to be gender incongruent (Cooper et al., 2018; George & Stokes, 2018b; Walsh et al., 2018). It is still unclear, however, what underlying neurocognitive mechanisms could explain this phenomenon and few suggestions have been made (for a review, see Van der Miesen et al., 2016).

One potential candidate mechanism/ability that could contribute to gender dysphoria/incongruence in ASD is the ability to represent mental states, otherwise known as mentalising (Glidden et al., 2016; Jacobs et al., 2014; Van Der Miesen et al,. 2016; van der Miesen, Hurley, et al., 2018). While, only initial steps have been made to explore this hypothesis, the findings presented in the previous chapter provided the first evidence in favour of a potential link. In Chapter 3, we investigated

the relation between ASD traits (using the Autism-Spectrum Quotient; Baron-Cohen, Wheelwright, Skinner, et al., 2001), current gender dysphoric feelings (using the Gender Identity/Gender Dysphoria Questionnaire for Adolescents and Adults; Deogracias et al., 2007), and recalled gender-typed behaviour (using the Recalled Childhood Gender Identity/Gender Role Questionnaire; Zucker et al., 2006) among cisgender individuals from the general population. Importantly, we also examined the role of mentalising (using the Reading the Mind in the Eyes test; Baron-Cohen, Wheelwright, Hill, et al., 2001) in the relations between these traits.

We found that the number of self-reported ASD traits was significantly associated with the number of gender dysphoric feelings reported (more ASD traits = more current gender dysphoric feelings). We also extended this finding further by reporting a significant association between ASD traits and recalled gender-typed behaviour (more ASD traits = less recalled gender-typed behaviour in childhood) for the first time. Furthermore, to our knowledge this was the first study that observed a large and significant association between mentalising and gender dysphoric feelings (poorer mentalising = more current gender dysphoric feelings). Of equal importance was the finding that mentalising moderated significantly the relation between ASD traits and gender dysphoric feelings. Further analysis showed that this relation was significant when mentalising ability was low, but it was nonsignificant when mentalising ability was high. Based on these results and on research evidence that suggests ASD is associated with a mentalising deficit (e.g., Brunsdon & Happé, 2014; Jones et al., 2018; Kaland et al., 2008; Yirmiya et al., 1998), we concluded that in autistic people “a weakness in the process of mentalising may contribute to increased fluidity of gender identity” (Kallitsounaki & Williams, 2020a, p.6).

Despite the potential importance of the findings described above, we were rightly cautious about drawing strong conclusions, given the preliminary nature of the findings. Arguably, these findings await replication before conclusions can be drawn from–and theory built on the basis of–them with confidence. Replication is of eminent importance, considering the crisis of confidence that has affected the discipline of psychology in recent years (Pashler & Wagenmakers, 2012). The necessity for conducting replication is now well recognised (Asendorpf et al., 2013; Makel et al., 2012; Pashler & Wagenmakers, 2012), and replicability is being characterised as the cornerstone of science (Simons, 2014). Furthermore, as Cesario (2014) suggested "researchers themselves need to provide repeated replications of their own work upon initial publication" (p. 41).

On this basis, the first aim of the current study was to attempt a direct replication of our recently published findings (Kallitsounaki & Williams, 2020a), presented in Chapter 3, in a new sample of adults from the general population to increase confidence in the veracity of the original findings. One thing to note is that in the original research, we took an individual differences approach to investigate these links. Therefore, that same approach was employed in the current replication study. It is well documented that there are personality characteristics in the neurotypical population that are qualitatively similar to the defining features of ASD, reflecting continuous liability to the disorder throughout the population. Research findings have indicated that ASD traits are normally distributed in the general population (Constantino & Todd, 2003; Ronald et al., 2006) and that "unaffected" relatives of autistic people report more ASD traits than people from the general population (Frazier et al., 2014; Pickles et al., 2000; Piven et al., 1994; 1997). As such, the investigation of the relations between ASD traits and other variables can be

informative about the nature of ASD itself (e.g., Lind et al., 2020; Nicholson et al., 2018).

The second aim of this study was to extend further the original findings about the moderator role of mentalising in the link between ASD traits and gender dysphoric feelings by conducting a mediation analysis. As noted previously, in Chapter 3 we found that the relation between ASD traits and gender dysphoric feelings was particularly pronounced when mentalising ability was low and completely absent when the level of this ability was high. However, the (unexpected and unusual) nonsignificant association between ASD traits and mentalising ability found in the original study did not allow a mediation model to be tested (e.g., Baron & Kenny, 1986).

In support of the original findings, we predicted that the number of self-reported ASD traits would be significantly associated with the number of self-reported current gender dysphoric feelings *and* with the recalled gender-typed behaviour in childhood (more ASD traits = more current gender dysphoric feelings and less recalled gender-typed behaviour). We also predicted that mentalising ability would be significantly associated with the number of current gender dysphoric feelings reported (poorer mentalising = more current gender dysphoric feelings). Furthermore, on the basis of previous research evidence (e.g., Baron-Cohen, Wheelwright, Skinner, et al., 2001; Williams, Bergström et al., 2018), we expected that the number of self-reported ASD traits would be significantly associated with mentalising ability (more ASD traits = poorer mentalising). Lastly, we predicted that mentalising ability would *mediate* the significant relation between ASD traits and current gender dysphoric feelings.

Method

Participants

One hundred and twenty-six people (97 birth-assigned female) took part in the current study. The average age of participants was 20.99 (*SD* = 4.10, range 18 to 45) years. One birth-assigned female participant identified as male, and one birth-assigned male participant did not identify either as female or male. English was the first language of 76.2% the sample, and two participants reported having a formal diagnosis of ASD. Student participants were rewarded with course credit in partial fulfilment of their degree, and people from the general population did not receive any kind of compensation for their participation. All participants gave informed consent, and the study was approved by City, University of London's Psychology Research Ethics Committee. A comparison between the replication and the original sample (see Chapter 3) is presented in Table C1.

Materials and Procedure

All the materials used in this study were identical to the ones we employed in the study presented in Chapter 3. Participants completed the Autism-Spectrum Quotient (AQ; Baron-Cohen, Wheelwright, Skinner, et al., 2001), the Reading the Mind in the Eyes test (RMIE; Baron-Cohen, Wheelwright, Hill, et al., 2001), the Gender Identity/Gender Dysphoria Questionnaire for Adolescents and Adults (GIDYQ-AA; Deogracias et al., 2007), and the Recalled Childhood Gender Identity/Gender Role Questionnaire (RCGI; Zucker et al. 2006).[8] Participants either took part in a laboratory administration of the measures or completed the study online through SONA (i.e., a web-based experiment management system for recruiting and rewarding participants who take part in research studies).

[8] For a detailed description of the measures used in this study, see Chapter 3.

Statistical Analysis

Following the approach we took in the original study, participants who had a history of ASD (*n* = 2) were not excluded from the analyses described below (for the results of the analyses when autistic participants were excluded from the sample, see Appendix C). To investigate the success of our replication attempt, we employed a series of statistical methods. First, we analysed whether those results reported as significant in the original study (see Chapter 3) were replicated in the current study. An alpha level of .05 was used as the cut-off for statistical significance. In addition, Bayesian analyses were conducted to examine whether the effects found in the original study were present or absent in the data from the current study. That is an increasingly used method that estimates the relative strength of the alternative hypothesis over the null, or vice versa (e.g., Dienes, 2014). According to Jeffreys' (1961) criteria, Bayes factors with values larger than 1 indicate increasing evidence for the alternative hypothesis ($BF_{10} > 3$ = substantial evidence, $BF_{10} > 10$ = strong evidence, $BF_{10} > 30$ = very strong evidence, $BF_{10} > 100$ = decisive evidence), whereas, scores < 1 indicate evidence for the null hypothesis ($BF_{10} < 0.33$ = substantial evidence, $BF_{10} < 0.10$ = strong evidence, $BF_{10} < 0.03$ = very strong evidence, $BF_{10} < 0.01$ = decisive evidence). Bayesian analyses were performed using JASP 0.8.1.2 (JASP Team, 2016). To directly compare the effect sizes of the current replication study with the ones of the original one, a series of Fisher's *Z* tests was conducted. For the zero order correlations that were carried-out, coefficients *r* are reported as measures of effect size (≥ .10 = small effect, ≥ .30 = moderate effect, ≥ .50 = large effect; Cohen, 1992).

To understand the mechanism through which ASD traits were related to current gender dysphoric feelings, a mediation analysis was conducted. Mediation

analysis indicates *how* or *by what means* a predictor variable (X) relates to an outcome variable (Y). In other words, mediation describes a situation where a relation between two variables is *explained* by a third variable that is called *mediator* (M) (Field, 2013; Preacher & Hayes, 2008). The relation between the predictor variable and the outcome variable, partialling out the effect of the mediator is called the *direct effect*, whereas the effect of the predictor on the outcome through the mediator is called the *indirect effect.* The sum of the direct and the indirect effects is the *total effect* (Field, 2013; Preacher & Hayes, 2008). In our mediation analysis, we examined whether mentalising (M) mediates the relation between ASD traits (X) and gender dysphoric feelings (Y). To test the significance of the indirect effect we employed bootstrapping procedure. That is a resampling technique "from which the sampling distribution of a statistic is estimated by taking repeated samples from the data set" (Field 2013, p. 871). In mediation analyses, this technique is used to compute confidence intervals for the indirect effects. A variable is said to significantly mediate the relation between X and Y, when the indirect effect is significantly different from zero (Preacher & Hayes, 2008). In the current study, a mediation analysis was conducted, using PROCESS v3.4.1 operated in SPSS, and unstandardised indirect effects were computed for each of 5,000 bootstrapped samples.

Results

Means (*SD*) for performance on the measures used in the current study are presented in Table 7.

Table 7

Mean (SD) Score on Self-Report Measures and Mentalising Performance

Variable	*n*	Mean (*SD*)
AQ	126	18.22 (5.89)
RMIE	126	23.06 (5.87)
GIDYQ-AA [a]	125	4.75 (0.44)
RCGI [a]	125	3.91 (0.65)

Note. AQ = Autism-Spectrum Quotient; RMIE = Reading the Mind in the Eyes test; GIDYQ-AA = Gender Identity/Gender Dysphoria Questionnaire for Adolescents and Adults; RCGI = The Recalled Childhood Gender Identity/Gender Role Questionnaire.

[a] Due to error, one birth-assigned male participant completed the female version of the GIDYQ-AA and RCGI. Hence, his data has not been included in the analyses.

Association Analyses

A series of Pearson *r* correlations was conducted to investigate the relations between ASD traits (measured with the AQ), mentalising (measured with the RMIE), current gender dysphoric feeling (measured with the GIDYQ-AA), and recalled childhood gender-typed behaviour (measured with the RCGI). Table 8 presents the results of the analyses, along with a comparison between the current findings and the results reported in the original study.

Table 8

Bivariate Correlations Differences Between the Current and the Original (Kallitsounaki & Williams, 2020a) Study

Association	Current study	Original study	Fisher's *Z* test
AQ × RMIE	r = -.33 [b] ***	r = -.18	Z = 1.18, p = .237
AQ × GIDYQ-AA	r = -.31 [a] ***	r = -.32 **	Z = -0.07, p = .948
AQ × RCGI	r = -.34 [b] ***	r = -.33 **	Z = 0.10, p = .921
RMIE × GIDYQ-AA	r = .55 [b] ***	r = .70 ***	Z = -1.91, p = .057

Note. AQ = Autism-Spectrum Quotient; RMIE = Reading the Mind in the Eyes test; GIDYQ-AA = Gender Identity/Gender Dysphoria Questionnaire for Adolescents and Adults; RCGI = The Recalled Childhood Gender Identity/Gender Role Questionnaire.

[a] BF_{10} = 30 - 99 (very strong evidence for alternative hypothesis). [b] $BF_{10} \geq 100$ (decisive evidence for alternative hypothesis).

** $p < .01$. *** $p < .001$.

In keeping with predictions, AQ score was moderately, negatively, and significantly associated with both GIDYQ-AA and RCGI. That is, the more ASD traits a person self-reported, the more current gender dysphoric feelings *and* the less recalled gender-typed behaviour they reported. Bayes factors indicated very strong and decisive evidence in support of the alternative hypotheses, respectively. Fisher's *Z* tests revealed that neither the AQ × GIDYQ-AA nor the AQ × RCGI correlations differed significantly from the equivalent correlations reported in the original study. AQ score was also found to be moderately, negatively, and significantly associated with performance on the RMIE task, suggesting that the greater a person's self-reported ASD traits, the lower their mentalising ability. Bayes factors indicated decisive evidence in support of the alternative hypothesis. Furthermore, as predicted the RMIE × GIDYQ-AA correlation was positive, large, and significant just as it

was in the original study, even though the size of the two associations was marginally significantly different (somewhat stronger in the original study). [9] Results suggest that the higher the score on the RMIE, the fewer the current gender dysphoric feelings, and Bayesian analysis indicated decisive evidence in support of the alternative hypothesis.

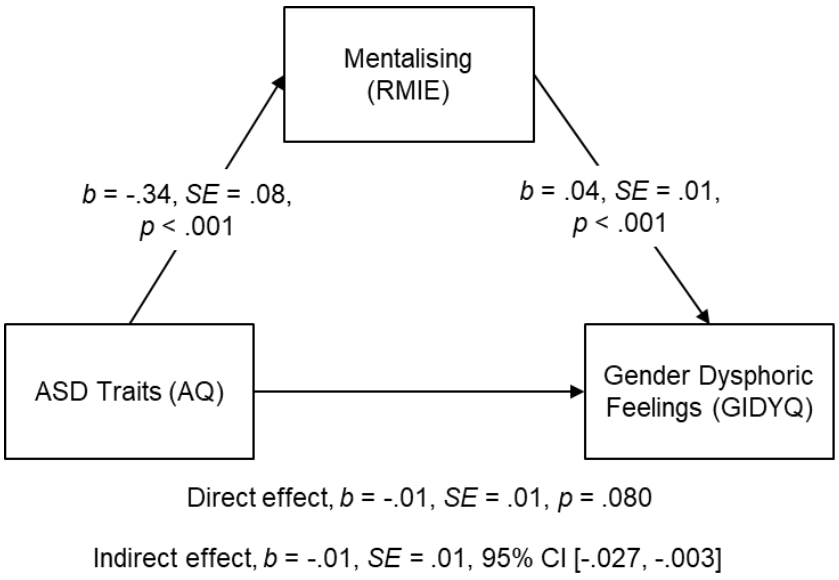
Mediation Analysis

A mediation analysis was carried out to examine whether mentalising mediated the effect of ASD traits on current gender dysphoric feelings. As Figure 5 illustrates, the indirect effect was significantly different from zero, indicating that mentalising mediated the significant relation between ASD traits and current gender dysphoric feelings. While the total effect between ASD traits and gender dysphoric feelings was negative and significant, b = -.02, SE = .01, p < .001, the direct effect between these variables was nonsignificant. Importantly, results indicate that mentalising ability accounted for most of the relation between ASD traits and current gender dysphoric feelings.

[9] It should be noted that when participants who reported possession of an ASD diagnosis were removed from the sample of the original study the RMIE × GIDYQ-AA correlation was almost identical (i.e., r = .59) to the one reported in the current study that contained only two participants who had a history of ASD.

Figure 5

Model of ASD Traits as Predictor of Gender Dysphoric Feelings, Mediated by Mentalising Ability



Note. AQ = Autism-Spectrum Quotient; RMIE = Reading the Mind in the Eyes test; GIDYQ-AA = Gender Identity/Gender Dysphoria Questionnaire for Adolescents and Adults.

Discussion

The first aim of the current study was to replicate the study we presented in Chapter 3. Our replication attempt yielded support for the hypothesis that ASD traits are significantly associated with both current gender dysphoric feelings and recalled gender-typed behaviour. In particular, we found that the more ASD traits a person self-reported, the more gender dysphoric feelings they had in the last 12 months and the less gender-typed behaviour they recalled from childhood. The significant association between ASD traits and gender dysphoric feelings observed in both the original *and* the current study was also consistent with George and Stokes' (2018b) findings. As such, the link between ASD traits and gender dysphoria in the general

population appears robust and reliable. Furthermore, our successful replication of the relation between ASD traits and recalled childhood gender-typed behaviour fits well with previous research evidence that autistic children show a weaker preference for gender-typical play than typically developing children (Knickmeyer et al., 2008). In keeping with the original research evidence, we also observed a significant association between mentalising ability and current gender dysphoric feelings. This supports the hypothesis that poorer mentalising ability relates to more gender dysphoric feelings. Given that the link between mentalising and gender dysphoric feelings was reported *for first time* in the study presented in Chapter 3, the replication of this link in the current research is striking and increases confidence in the reliability of the original findings.

The second aim of the current study was to extend our original findings about the role of mentalising in the link between ASD traits and gender dysphoric feelings, examining whether mentalising mediates this link. In contrast to previous research evidence (e.g., Baron-Cohen, Wheelwright, Hill, et al., 2001; Williams, Bergström, et al., 2018), in Chapter 3 we observed a nonsignificant association between ASD traits and mentalising. However, in the current study, the number of self-reported ASD traits was moderately, negatively, and significantly associated with mentalising ability and therefore, we were able to test our mediation hypothesis. Results from a mediation analysis revealed that the relation between ASD traits and gender dysphoric feelings in the general population was almost entirely explained by mentalising. As mentioned in Chapter 3, it could be argued that by measuring mentalizing, we in fact measured a core component of ASD itself. This can provide an alternative explanation of why mentalising mediated the relation between ASD traits and current gender dysphoric feelings.

Taken together, the replicated and novel findings of the current study suggest it is appropriate to draw a series of conclusions. First, results imply that people with low mentalising ability are more liable than people with high mentalising ability to experience gender dysphoric feelings and that people with high ASD traits report increased gender dysphoric feelings mainly because their mentalising ability is low. ASD is known to be characterised by diminished mentalising (e.g., Yirmiya et al., 1998), with some suggesting that this represents a cognitive marker of ASD (e.g., Brunsdon & Happé, 2014), and research has shown that autistic people experience significantly more gender dysphoric feelings than neurotypical people. They also show a more diverse range of gender identities (e.g., George & Stokes, 2018b) and are more likely to begin or plan to begin the process of gender transitioning (Cooper et al., 2018). As such, it is plausible to suggest that the results of the current study support the hypothesis that mentalising contributes to the overrepresentation of gender dysphoria/incongruence in ASD (Glidden et al., 2016; Jacobs et al., 2014; Van Der Miesen et al., 2016; van der Miesen, Hurley, et al., 2018). It is important to note that given its sensitive nature, the study of mechanisms that could explain the co-occurrence of ASD and gender dysphoria/incongruence should include participatory research practices. Due to limited resources, however, this was not possible in the current study.

From a theoretical perspective, a mentalising deficit could contribute to gender dysphoria/incongruence in a number of ways. Gender constancy - the understanding that one's own gender does not change, regardless of changes in gender-typed appearance, activities, and traits - is considered one of the major cognitive stages that a child needs to reach to be able to formulate a gender self-concept (e.g., Kohlberg, 1966). Indeed, a developmental lag in the acquisition of

gender constancy has been found in children with gender identity difficulties (Zucker et al., 1999). Crucially, the level of understanding of gender constancy is related to the ability to distinguish between appearance and reality (Trautner et al., 2003; Zmyj & Bischof-Köhler, 2015), which is one of the key components of mentalising. As such, it could be argued that an autistic child who has not reached an adequate level of understanding of gender constancy, due to difficulties in mentalising, could be susceptible to increased cross-gender behaviour in childhood and therefore greater likelihood of developing feelings of gender dysphoria in adolescence and adulthood (e.g., Drummond et al., 2008; Green, 1987; Wallien & Cohen-Kettenis, 2008).

Furthermore, people tend to conform to social conditioning and social norms partially to avoid feelings of guilt and embarrassment that result from others' judgments when they do not conform (e.g., Scheff, 1988; Suhay, 2015). The experience of these feelings, known as self-conscious emotions, is thought to depend on people's mentalising ability (Hobson, 2006). A person who experiences difficulties in attributing mental states to others will show a reduced propensity to experience self-conscious emotions and, therefore, might be less likely to comply with societal norms. Indeed, numerous studies have shown that autistic people experience self-conscious emotions less frequently than neurotypical people (e.g., Capps et al., 1992; Davidson et al., 2018; Losh & Capps, 2006). Arguably, if autistic people tend to feel fewer self-conscious emotions, they may be less affected by what other people think about their cross-gender behaviour and therefore less likely to feel pressure to conform of gender norms.

In sum, the current study successfully reproduced (a) the link between ASD traits and current gender dysphoric feelings/recalled gender-typed behaviour and (b) the relation between mentalising and current gender dysphoric feelings, we reported

in Chapter 3. Most importantly, we found, for the first time, that the relation between ASD traits and gender dysphoric feelings was to a significant degree explained by mentalising ability. Although direct replication by other laboratories awaits, the main implication of these findings is that mentalising ability could be one of the underlying neurocognitive mechanisms that explain the increased prevalence of gender dysphoria/incongruence in people with a diagnosis of ASD. This represents a highly novel contribution to the literature and provides motivation for future theory-building research on the role of mentalising in the formation of typical and atypical gender self-concepts.

Chapter 5

A Relation between ASD Traits and Gender Self-Concept: Evidence from Explicit and Implicit Measures

In the previous chapters, we presented a number of findings aiming to answer whether there is a link between ASD and gender dysphoria/incongruence, as well as whether mentalising underpins any such link. In this chapter, the focus shifts to the effect of ASD on gender-related cognition, and we present the findings of two studies (original study, followed by a replication study). In both studies, we adopted an individual differences approach to investigate the relation between explicit and implicit gender self-concept in people from the general population.

Gender describes a constellation of traits, behaviours, and roles attributed to males and females (Wood & Eagly, 2009). When individuals impute these cultural meanings of being male or female to themselves, then the process of formation and consolidation of a gender self-concept (or gender identity) begins (Wood & Eagly, 2009, 2010, 2015). As already described in detail in Chapter 1, the study of gender self-concept in adults has traditionally relied on self-reports of the extent to which individuals believe their personality traits and interests conform to societal standards of masculinity and femininity. Research has shown that people attribute to themselves both feminine and masculine traits, with birth-assigned males endorsing more masculine traits as self-descriptive, and with birth-assigned females endorsing more feminine traits to themselves, on average (Bem, 1974; Spence & Helmreich, 1978).

Despite their extensive use in research, these self-report measures rely on accurate self-awareness of one's own traits (which is unlikely to always be the case) and are considered susceptible to the effects of self-presentation (Devos et al., 2012;

Nosek et al., 2005, 2007). That is, people may (consciously or nonconciously) attempt to camouflage or manipulate presentation of aspects of their selves and identities for various reasons. An alternative approach to use of verbal self-report measures of gender self-concept is to employ implicit measures that rely on some or other behavioural indicator of identity. Such, implicit measures are considered less prone than self-report measures to the adverse effects of self-presentation and have, therefore, been widely employed in research as an alternative to explicit measures of self and identity (e.g., Stieger et al., 2014; van Well et al., 2008). As Nosek et al., (2007) argued:

> Implicit cognition could reveal associative information that people were either unwilling or unable to report. In other words, implicit cognition could reveal traces of past experience that people might explicitly reject because it conflicts with values or beliefs, or might avoid revealing because the expression could have negative social consequences. Even more likely, implicit cognition can reveal information that is not available to introspective access even if people were motivated to retrieve and express it. (p. 266)

One of the most prominent of these measures is the Implicit Association Test (IAT; Greenwald et al., 1998). The IAT measures the strength of the automatic associations between concepts and attributes based on the assumption that strong links between concepts and attributes trigger faster behavioural reactions than concepts and attributes that are only weakly related with each other (Greenwald et al., 1998). The IAT requires respondents to identify and sort items into one of four categories, using two response keys, with each of the keys being assigned to two of the four categories. For example, in the gender IAT respondents sort words (e.g., I, They, Forceful, and Warm) that belong to one of the four following categories:

Self/Other/Masculine/Feminine. Findings from the classic Greenwald and Farnham (2000) study showed that birth-assigned females tend to respond faster when the category of "self" shares the same response key as traditionally feminine attributes than when "self" shares response key with traditionally masculine attributes. In contrast, birth-assigned males tend to show the opposite pattern (faster responses when the category of "self" is paired with masculine attributes than with feminine attributes).

The study of gender self-concept is particularly important when it comes to understanding of disorders that have been linked with gender identity difficulties, such as autism spectrum disorder (ASD). Williams et al. (1996) were the first who described the case of two boys diagnosed with ASD with co-occurring gender identity difficulties, expressed by cross-gender stereotyped interests and behaviours. Since then, a series of other case-studies followed, indicating the existence of a link between ASD and gender dysphoria/incongruence (e.g., Jacobs et al., 2014; Kraemer et al., 2005; Landén & Rasmussen, 1997; Mukaddes, 2002).

Indeed, de Vries et al. (2010) found that the prevalence of ASD among individuals with gender dysphoria was almost 8 times greater than the population estimate of ASD. There has been also evidence about increased gender dysphoric feelings among individuals with ASD (George & Stokes, 2018b) and an increased likelihood to express the wish to be the other binary gender (Janssen et al., 2016; May et al., 2017; Strang et al., 2014; van der Miesen, Hurley, et al., 2018) and to report incongruent gender identities (e.g., Bejerot & Eriksson, 2014; George & Stokes, 2018b).

Despite a suggestive link between ASD and dysphoria/incongruence, only two studies have examined whether individuals with ASD internalise attributes and

roles that stereotypically define the gender associated with their birth-assigned sex to the same degree as neurotypical individuals do (Bejerot & Eriksson, 2014; Stauder et al., 2011). Both studies used self-report measures of gender self-concept, and found that autistic adults showed weaker conformity to masculine roles, traits, and interests than neurotypical adults. Interestingly, both studies found between-group differences in self-identification with feminine traits (e.g., $d = 0.63$ in Stauder et al., 2011), yet they did not reach the traditional threshold for statistical significance. Statistical power to detect even moderate group differences was relatively low in each study, which raises the possibility of a Type I error being made in each study.

Experiment 1: Research Aims and Hypotheses

In Experiment 1, we attempted to address the issue of gender self-concept in ASD by taking an individual differences approach among a general population sample. ASD is now considered by most to be a dimensional disorder with ASD traits being normally distributed in the general population (Constantino & Todd, 2000, 2003; Ronald et al., 2006). As such, people from the general population will vary in the number of ASD traits they have from very few at one end of the spectrum to very high at the other end of the spectrum. Only at an arbitrary point toward the upper end of the distribution (related to clinical urgency) are individuals with a sufficiently high number of ASD traits given an official clinical diagnosis of ASD (Bolton et al., 1994; Goldberg et al., 2005; Le Couteur et al., 1996; Murphy et al., 2000; Pickles et al., 2000; Piven et al., 1997; Szatmari et al., 2008). Therefore, we can learn important things about the nature and basis of gender in ASD by studying how gender self-concept varies according to the number of ASD traits manifested by people from the general population.

Crucially, we included in the current study not only an explicit self-report measure of gender self-concept, but also a gender IAT as an implicit behavioural measure of the strength of participants' identification with masculine and feminine traits, respectively. Inclusion of an implicit measure is crucial because one possible interpretation of previous findings of a reduced identification with gender-differentiated traits among people with ASD is that self-report measures are less valid than they are among neurotypical individuals. Given that aspects of self-awareness are considered by many researchers to be diminished in ASD (e.g., Carruthers, 2009; Gopnik, 1993; Williams, 2010), it may be that ASD is associated with gender identification difficulties not because of a diminished implicit self-experience of gender feelings, but merely because those feelings are not accurately represented and reported. If that was the case, then ASD traits would be associated (negatively) with explicit self-report strength of gender self-concept but not with implicit, behavioural measures of gender self-concept.

In the current study, we measured ASD traits, using the Autism-Spectrum Quotient (AQ; Baron-Cohen, Wheelwright, Skinner et al., 2001) and the strength of the explicit gender self-concept, using the Personal Attributes Questionnaire (PAQ; Spence & Helmreich, 1978). Based on the existing preliminary findings about a weaker self-attribution of masculine traits among autistic individuals (Bejerot & Eriksson, 2014; Stauder et al., 2011) and the presence of gender dysphoria/incongruence also in autistic birth-assigned females (e.g., Lemaire et al., 2014), we predicted that the number of ASD traits would be negatively and significantly associated with the strength of the explicit gender self-concept. In other words, we expected that as the number of ASD traits increase, the strength of explicit

gender self-concept (i.e., the extent to which a person reports themselves to identify themselves masculine and feminine traits) will decrease.

Perhaps most importantly, however, we explored the relation between ASD traits and implicit gender self-concept, for the first time, employing the gender IAT described by Greenwald and Farnham (2000). Following the theory that ASD is associated with an underlying difficulty "identifying" with others (e.g., Hobson, 2010; Hobson & Lee, 1999; Tomasello, 1999) we predicted that the strength of implicit gender self-concept would be also associated significantly with number of ASD traits, such that as ASD traits increase, so the strength of implicit gender self-concept will decrease.

Experiment 1: Method

Participants

One hundred and one adults (50 birth-assigned female) took part in the current online experiment. Their average age was 36.93 (*SD* = 10.11; range = 22 to 70) years. Ninety-four percent of participants reported English as their first language, and all were cisgender. Thirteen of the 101 participants had a formal diagnosis of ASD, according to self-report. Participants were recruited through the Amazon's online crowdsourcing platform MTurk and received compensation for their time. Informed consent was obtained from all individual participants included in the study. Ethical approval for this study was obtained from the University of Kent's Psychology Research Ethics Committee (ID: 201815434977895393)

Materials and Procedure

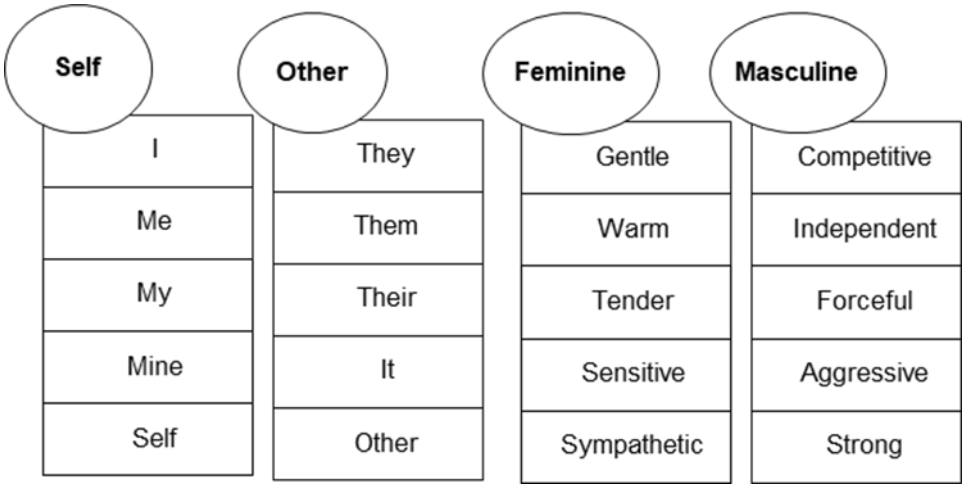
Implicit Association Test

To measure implicit gender self-concept, we employed the Implicit Association Test (IAT) described by Greenwald and Farnham (2000). The task

involved sorting words (see Figure 6) that belonged to one of four categories (self/other/feminine/masculine), using one of two possible keys. Words appeared on the middle of a computer screen in a random order and category labels were always presented in the upper right and left corners of the screen. All stimuli were presented to participants before the beginning of the task, and they were instructed to study the items that belonged to each category for 25s.

Figure 6

Contrast Concepts and Items Presented in the Implicit Association Test



In block 1 the “self” category label was presented in the upper left corner of the screen and the “other” category label in the upper right corner. Participants had to sort words that belonged either to the “self” or the “other” category by pressing the “z” key of a keyboard for words related to “self” and the “m” key for words related to “other”. In block 2 categories referred to “feminine” and “masculine” attributes. The “feminine” category label was presented in the upper left corner and the “masculine” category in the upper right corner. Participants were instructed to press the “z” key for words belonged to “feminine” category and the “m” key for items belonged to “masculine” category. In block 3 the four categories were presented

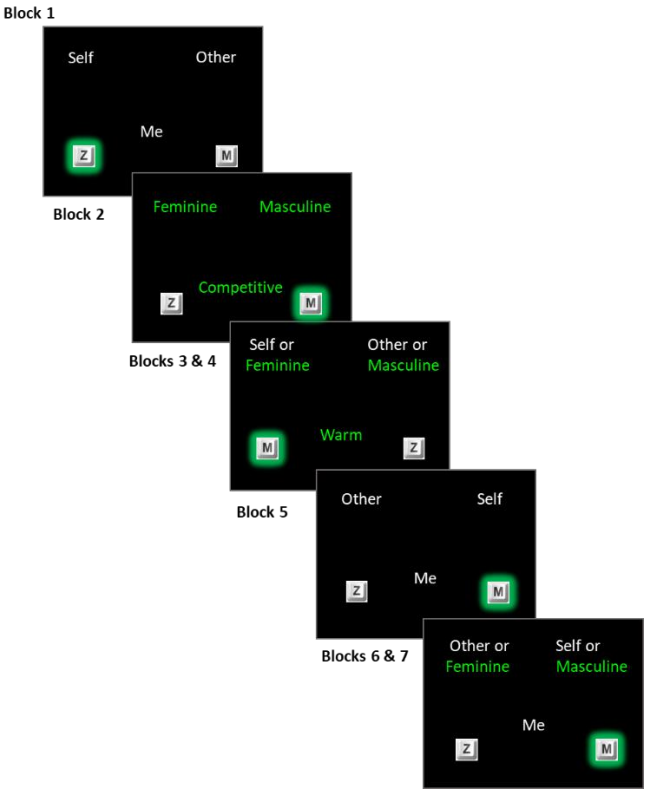
combined (“self/feminine” labels: upper left corner; “other/masculine” labels: upper right corner), and participants practiced the sorting task by pressing the key that was assigned to each category in the preceding two blocks (i.e., “z” key for items belonged to “self /feminine” categories and “m” key for items belonged to “other/masculine” categories). In block 4 they completed the first experimental condition of the sorting task. In block 5, the “self” category label was presented in the upper right corner and the “other” category label in the upper left corner, subsequently the assignment of the key for each category was reversed, compared to the first block. Participants had to press “m” for items belonged to the “self” category and “z” for items belonged to the “other” category. In block 6, they practiced the combined task, using the switched key assignments. That is, participants were instructed to press the “z” key to categorise items that belonged either to the “other” or to the “feminine” category and the “m” key for words that belonged either to the “self” or “masculine” category. In the last block, they completed the second experimental condition of the combined sorting task. The order of the blocks was counterbalanced across participants. Figure 7 illustrates the procedure and the number of trials used in each of the seven blocks of the task.

The dependent measure for the IAT was the strength of the automatic associations calculated with the scoring algorithm recommended by Greenwald, Nosek and Banaji (2003). That is, a standardised mean difference score (namely *D*) in response latencies between the two practical blocks (i.e., block 3 and block 6) and the two experimental blocks (i.e., bock 4 and block 7). The resulting *D* scores formulate a bipolar scale that ranges from masculinity (i.e., negative scores) to femininity (i.e., positive scores). The faster the responses in the “*self-masculine* vs *other-feminine”* combined sorting condition than in the “*self-feminine* vs *other-*

masculine" condition, the stronger the implicit masculine gender self-concept. Whereas, the faster the responses in the *"self-feminine vs other-masculine"* combined sorting condition than in the "*self-masculine* vs *other-feminine"* condition, the stronger the implicit feminine gender self-concept. However, in the current study, the key dependent variable on the IAT related to the strength of the implicit gender self-concept, regardless of whether the participant identified as male or female. Therefore, all *D* scores were transformed to positive values with higher scores denoting stronger implicit gender self-concept. The IAT was programmed using Inquisit Millisecond software package 4 (https://www.millisecond.com.), and it was administered using Inquisit Web Player 4.0.10. After the completion of the IAT, participants were automatically redirected to a Qualtrics survey to complete the rest of the tasks and questionnaires.

Figure 7

Illustration of the Procedure and Stimuli Used in the Implicit Association Test



Note. Responses indicate a strong association of self-concept with feminine traits.

Personal Attributes Questionnaire (PAQ)

The PAQ (Spence & Helmreich, 1978) is one of the most widely used self-report measures of explicit gender self-concept (Greenwald & Farnham, 2000; Van Well et al., 2007; Ward et al., 2006). Participants are presented with 24 trait dimensions, with their endpoints being labelled with contradictory attributes (e.g., "Not at all aggressive - Very aggressive"), and they are asked to indicate the extent to which each item applies to them, using a 5-point scale. The questionnaire consists of an 8-item bipolar scale of gender self-concept that ranges from extreme femininity to extreme masculinity (F-M) and of two 8-item unipolar scales that measure masculinity (M) and femininity (F) separately. As such, three mean scores were calculated for each participant. High scores on the bipolar scale (M-F) indicate a

masculine self-concept and low scores a feminine self-concept. The higher the score on the femininity (F) scale, the stronger the feminine self-concept and the higher the score on the masculinity (M) scale, the stronger the masculine self-concept. In terms of its psychometric properties, Spence and Helmreich (1978) reported that Cronbach's α was .85, .82, and .78 for the masculinity, femininity, and masculinity-femininity scale, respectively. Moreover, scale scores clearly distinguish birth-assigned males from birth-assigned females (e.g., Helmreich et al., 1981), and the measure shows convergent validity (coefficients range from .52 to .84) with other self-report measures of gender self-concept (e.g., Bem Sex Role Inventory; Lubinski et al., 1983; Spence, 1991).

Autism-Spectrum Quotient (AQ)

The AQ (Baron-Cohen, Wheelwright, Skinner, et al., 2001) is a reliable and valid and valid self-report measure of ASD traits that has been widely used in research among clinical and nonclinical populations (e.g., Robertson, & Simmons, 2013; Williams, Nicholson, & Grainger, 2018). Participants are asked to indicate their agreement with each of the 50 self-referential statements that the AQ comprises (e.g., "I find social situations easy"), using a 4-point Likert scale that ranges from "definitely agree" to "definitely disagree". The total score ranges from 0 to 50, and values ≥ 26 indicate clinically significant levels of ASD traits (Woodbury-Smith et al., 2005). [10]

Statistical Analysis

The focus of the current study was on the *strength* of gender self-concept, regardless of which direction the self-concept was in (i.e., masculine or feminine).

[10] For a detailed description of the psychometric properties of the Autism-Spectrum Quotient, see Chapter 3.

Therefore, only the dimensional scales of PAQ and the transformed *D* scores of the IAT were included in the analyses described below. The bipolar scale of PAQ was used only in an initial analysis to explore its association with the untransformed *D* scores from the IAT, for comparison with the equivalent result reported by Greenwald and Farnham (2000). To examine the relation between ASD traits and the strength of the explicit/implicit gender self-concept, a series of zero-order correlations was conducted, with coefficients *r* being reported as measures of effect size (≥ .10 = small effect, ≥ .30 = moderate effect, ≥ .50 = large effect; Cohen, 1992). Furthermore, a series of independent samples *t*-tests was employed to examine differences in the explicit and implicit measures of gender self-concept between a low-AQ group and a high-AQ group. To categorise participants as having high or low number of self-reported ASD traits, we employed the widely used median split approach (e.g., Bayliss & Kritikos, 2011; O'Keefe & Lindell, 2013). That is, individuals who scored below the median score on the AQ (i.e., *Mdn* = 20.00) were assigned in the low AQ-group, whereas people who scored above the median were categorised in the high-AQ group (we also used the AQ threshold of 26 to split participants; results of the analyses are reported in Appendix D). Cohen's *d* values (≥ 0.20 = small effect, ≥ 0.50 = moderate effect, ≥ 0.80 = large effect; Cohen, 1969) are reported as measures of effect size. A Chi-Square test was also conducted to explore whether there was a significant association between the group of participants (low-AQ/high-AQ) and gender. Cramer's V was reported as measure of effect size and was interpreted as a coefficient *r* (McHugh, 2013). To examine sex differences, a series of correlation analyses was conducted for birth-assigned males and birth-assigned females separately, and sex was used as a between-participants variable in a series of ANOVAs of the case-control data. Nonetheless, we should note here that

the investigation of sex differences was beyond the scope of the current study. Therefore, all the analyses were conducted post-hoc, and we did not make specific predictions about them. An alpha level of .05 was used as the conventional criterion for statistical significance, unless a priori directional predictions were made. In those cases, values for one-tailed tests are reported.

In the current study, 13 participants reported possession of a formal diagnosis of ASD. Nonetheless, we did not exclude them from any analysis conducted in this study on the basis that ASD describes a spectrum disorder that ranges from people in the general population with low levels of ASD traits to people who hold a clinical diagnosis of ASD (e.g., Murphy et al., 2000; Pickles et al., 2000; Szatmari et al., 2008). However, in order to ensure that the significant effects we found were not inflated by their inclusion, we reconducted the main analyses, including only non-autistic participants. Results did not change substantively (see Appendix D).

Experiment 1: Results

Preliminary Analyses

The average AQ score across participants was 19.65 (*SD* = 7.31). The average score on PAQ masculinity and PAQ femininity scale was 2.43 (*SD* = 0.73) and 2.85 (*SD* = 0.72), respectively. Participants were accurate when sorting items on the IAT. The mean proportion of correct item categorisation (*M* = 85.94; *SD* = 13.90) in the critical blocks was above chance, $t(100) = 62.12$, $p < .001$, $d = 6.18$. Likewise, participants showed the expected effect on the IAT, producing a mean *D* score (*M* = 0.39; *SD* = 0.32) that was significantly above zero, $t(100) = 12.13$, $p < .001$, $d = 1.21$.

A correlation analysis was conducted to examine the association between the explicit and the implicit measure of gender self-concept. The bipolar scale of PAQ was negatively and significantly associated with the untransformed *D* scores of the

IAT, r = -.26, p = .009, showing that the more a person explicitly endorses masculine traits as self-descriptive, the more they implicitly/automatically associate their self-concept with masculine traits on the IAT. A Fisher's Z test showed that the magnitude of the association was of equivalent size with that reported in the IAT original study by Greenwald and Farnham (2000), Z = -0.36, p = .720.

Furthermore, a series of independent samples t-tests was conducted to examine differences in the explicit and the implicit measure of gender self-concept between birth-assigned males and birth-assigned females. As shown in Table 9, birth-assigned females scored significantly higher on the PAQ femininity scale than birth-assigned males, whereas, a nonsignificant difference in the PAQ masculinity scale score was found between birth-assigned males and birth-assigned females. Results indicate that compared to birth-assigned males, birth-assigned females ascribed to their self-concept more feminine traits, as self-descriptive, whereas birth-assigned males and birth-assigned females ascribed to their self-concept masculine traits, as self-descriptive, to the same degree. Results also yielded a significant between-group difference in performance on the gender IAT, with birth-assigned females scoring significantly higher on the task than birth-assigned males. Results suggest that both birth-assigned females and birth-assigned males implicitly associated their self-concept more strongly with feminine than masculine traits, but birth-assigned females implicitly identified with feminine traits more strongly than birth-assigned males.

Table 9

Means (SDs) and Results of t-Tests for Sex Differences in the Explicit and Implicit Measures of Gender Self-Concept

Variable	Birth-Assigned Sex		Group Differences		
	Male (n = 51)	Female (n = 50)	t	p	d
PAQ masculinity	2.49 (0.69)	2.38 (0.78)	0.79	.433	0.16
PAQ femininity	2.71 (0.66)	3.00 (0.76)	-2.06	.043	-0.41
IAT (D score) [a]	0.10 (0.31)	0.38 (0.50)	-3.37	.001	-0.67

Note. PAQ = Personal Attributes Questionnaire; IAT = Implicit Association Test.

[a] Descriptive statistics are reported for untransformed D scores. Positive scores indicate an implicit female self-concept, whereas negative scores indicate an implicit male self-concept.

Association Analyses

A series of correlation analyses was conducted to explore the relation between ASD traits, on the one hand, and the explicit and implicit gender self-concept, on the other hand. In line with our predictions, AQ was negatively and significantly associated with both PAQ femininity and PAQ masculinity scale scores, r = -.43, p < .001 (one-tailed) and r = -.35, p < .001, respectively. These results indicate that the higher the number of ASD traits manifested, the lower the explicitly reported strength of gender self-concept (for both feminine and masculine traits). Next, we examined the relation between AQ score and implicit gender self-concept (indexed by D score). As predicted, AQ was negatively and significantly associated with D score, r = -.25, p = .006 (one-tailed), indicating a negative association

between the number of self-reported ASD traits and the strength of the implicit gender self-concept (high AQ score = low *D* score).

Case-Control Analyses

Table 10 presents mean (*SD*) scores on the PAQ femininity scale, PAQ masculinity scale, and the IAT among participants who scored above and below the median of the sample on the AQ. A series of independent samples *t*-tests was conducted to examine differences between the low-AQ group and the high-AQ group in the explicit and implicit gender self-concept. Results of the analyses are presented in Table 10. Groups were equated for age and birth-assigned sex. The average age was 38.61 (*SD* = 9.02) in the low-AQ group and 35.22 (*SD* = 10.93) in the high-AQ group, a difference that was nonsignificant, $t(94.85) = 1.70$, $p = .093$, $d = 0.38$. The low-AQ group included 23 birth-assigned males and 28 birth-assigned females, whereas the high-AQ group included 28 birth-assigned males and 22 birth-assigned females. The difference between the high and low AQ groups in ratio of birth-assigned males to birth-assigned females was nonsignificant, $\chi^2(1) = 1.20$, $p = .273$, Cramer's V = .11. As predicted, relative to participants in the low-AQ group, those in the high-AQ group had significantly lower scores on both PAQ scales, as well as significantly lower *D* scores on the IAT. When the AQ threshold of 26 (i.e., the clinical cut-off score) was used to split participants in a high-AQ group and in low-AQ group, results remained essentially the same. The results from these supplementary analyses are reported in Appendix D.

Table 10

Means (SDs) and Inferential Statistics for Group Differences

Variables	Group		Group Differences		
	Low AQ (n = 51)	High AQ (n = 50)	t	p [a]	d
IAT (D score)	0.46 (0.38)	0.31 (0.23)	2.57	.006	0.51
PAQ femininity	3.09 (0.55)	2.61 (0.80)	3.52	<.001	0.70
PAQ masculinity	2.69 (0.64)	2.18 (0.74)	3.71	<.001	0.74

Note. IAT = Implicit Association Test; PAQ = Personal Attributes Questionnaire.

[a] Values for one-tailed tests are reported.

Exploratory Analyses Among Each Birth-Assigned Sex Separately

In a set of exploratory analyses, we checked whether there were any of the effects reported above differed significantly between birth-assigned males and birth-assigned females.[11] Fisher's Z tests were used to establish whether the size of correlations between variables differed significantly between sexes. Birth-assigned sex was also used as a between-participants variable in a series of ANOVAs of the case-control data. Only two of these analyses were significant (all other ps > .246).

First, there was a significant difference between birth-assigned males and females in the size of the association between D score on the gender IAT and total score on the AQ, Z = 2.49, p = .013. The association between AQ and D score was significant within birth-assigned females, indicating that the higher the number of ASD traits, the lower the strength of the implicit gender self-concept, r = -.41, p = .003. However, this association was nonsignificant among birth-assigned males, r = .08, p = .584.

[11] Please note that non-autistic birth-assigned males and non-autistic birth assigned females reported similar levels of ASD traits, $t(86) = 0.42$, p = .679, d = .09.

Second, in a 2 (birth-assigned sex: male/female) × 2 (group: high AQ/low AQ) ANOVA on the gender IAT data, there was a significant Group × Birth-Assigned Sex interaction, $F(1,97) = 11.02$, $p = .001$, $\eta_p^2 = .10$. The IAT *D* score among birth-assigned females in the low-AQ group ($M = 0.65$, $SD = 0.40$) was significantly higher than among birth-assigned females in the high-AQ group ($M = 0.33$, $SD = 0.26$), $t(46.73) = 3.44$, $p = .001$, $d = 0.96$. This indicates that birth-assigned females with a high number of ASD traits had a weaker gender self-concept than birth-assigned females who had a low number of ASD traits. In contrast, the IAT *D* score among birth-assigned males in the low-AQ group ($M = 0.24$, $SD = 0.18$) was not significantly different from the IAT *D* score among birth-assigned males in the high-AQ group ($M = 0.29$, $SD = 0.20$), $t(49) = -0.90$, $p = .379$, $d = 0.25$.

Experiment 1: Discussion

The first notable set of findings of the current study was about the link between ASD traits and the explicit gender self-concept. As predicted, AQ score was negatively and significantly associated with both femininity and masculinity scale scores of PAQ. Results indicate that as the number of self-reported ASD traits increases, the strength of the explicit gender self-concept decreases. This is the first study, to our knowledge, to show a link between explicit gender self-concept and number of ASD traits among members of the general population. We also addressed this issue by dividing our sample into a high-AQ group and a low-AQ group. In keeping with our predictions, the results complemented the findings from the correlation analyses. That is, relative to the group of participants with low ASD traits, the group with high ASD traits scored significantly lower on both scales of PAQ. These results indicate that people with high ASD traits explicitly/consciously

identify themselves with less strong masculine and feminine attributes relative to individuals with low ASD traits; they have weaker gender self-concepts, on average.

Arguably more notable is the second set of findings from the current study, regarding the relation between number of ASD traits and implicit gender self-concept. To our knowledge, the current study is the first to investigate the potential link between ASD and gender self-concept at a level deeper than that tapped by self-report. In keeping with our predictions, we found that the strength of the implicit gender self-concept (indexed by *D* score) was negatively and significantly associated with number of self-reported ASD traits, indicating that as the number of reported ASD traits increases, the strength of the implicit gender self-concept decreases. The size of the association was relatively modest (small-to-medium), but it was highly statistically significant. Complementary to this, relative to the group of participants with low ASD traits, the group with high ASD traits scored significantly lower (*D* score) on the IAT, indicating that among people with high ASD traits the automatic identification of *self* with either masculine or feminine attributes was weaker relative to individuals with low ASD traits. Again, group differences were moderate-to-large in magnitude and statistically significant. These results suggest that high ASD traits in the general population might signify an implicit/unconscious gender self-concept that is neither strongly masculine nor strongly feminine, among individuals who otherwise report themselves to be cisgender.

Importantly, the significant association we found between the implicit and the explicit measure of gender self-concept was equivalent in size to the one reported by Greenwald and Farnham (2000) (r = -.26 in our study and r = .24 in their study). Moreover, these associations are of an order of magnitude suggested by Hofmann et al. (2005) to indicate consistency between implicit and explicit measures. This is

important, because it provides evidence that the two measures are tapping overlapping sets of representations about one's gender rather than tapping entirely different constructs. Of course, it should be noted that some divergence between implicit and explicit measures is always expected, given that "direct ratings are farther downstream in the processes of judgement and thus subject to more deliberation than the more spontaneous, automatic associations tapped by indirect measures" (Wood & Eagly, 2009, p. 112). As a result, direct measures can be prone to the influence of social desirability.

A note of caution is also needed when interpreting results from the gender IAT. In a series of post hoc analyses, we observed that the explicit effects (using the PAQ) observed in the whole sample of 101 participants held in *both* birth-assigned males (n = 51) and birth-assigned females (n = 50). That is, in the association analyses the strength of explicit gender self-concept was negatively associated with number of ASD traits in both birth-assigned males and females. Likewise, in the case-control analyses those with high ASD traits had a weaker gender self-concept than those with low ASD traits, regardless of whether they were birth-assigned male or female. However, results appeared to be not so straightforward when considering the effects of ASD traits on *implicit* gender self-concept. Here, in the association analyses number of ASD traits was negatively associated with strength of implicit gender self-concept in birth-assigned females only. Likewise, in the case-control analyses only birth-assigned females with high ASD traits had a weaker gender self-concept than birth-assigned females with low ASD traits (the same analyses in males revealed no between-group differences). What should be made of these apparent sex differences in the relation between ASD and implicit gender self-concept?

On the one hand, the existence of gender differences might be treated with some scepticism, given that the analyses were entirely post hoc and between-sex differences were not predicted. On the other hand, sex differences were reported by Nobili, Glazebrook, Bouman, et al. (2018) in their study of the association between ASD traits and transgender status. Nobili, Glazebrook, Bouman, et al. (2018) found that birth-assigned females in a transgender group reported significantly more ASD traits relative to birth-assigned females in a cisgender group. No differences between gender groups in number of ASD traits were seen among birth-assigned males in their study, however. As such, our results could be considered in line with these findings. Nevertheless, gender self-concept is different from transgender identity and therefore future studies need to examine sex differences in gender self-concept further before strong conclusions can be drawn.

Taken together, the results of this study imply that people with high ASD traits have a weaker inclination to explicitly identify with gender-differentiated attributes and that birth-assigned females at least have a weaker propensity to explicitly identify with, and incorporate into their self-concept gender-differentiated attributes. As levels of ASD increase, so too may be susceptible to gender identity difficulties. If this interpretation is correct, then this could explain gender dysphoria/incongruence in ASD (Glidden et al., 2016; Øien et al., 2018; Van Der Miesen et al., 2016).

Given that several researchers have argued (and provided evidence to show) that ASD is characterised by a diminished general tendency to represent the perspectives and attitudes of others (e.g., Baron-Cohen & Wheelwright, 2004; Hobson et al., 2006; Hobson & Lee, 1999; Tomasello, 1999), it could be that gender-

related traits are not internalised and incorporated into the self-concept of children with ASD in the same manner, or to the same depth, as among neurotypical children. In addition, difficulties representing others' perspectives also contribute to reduced experiences of self-conscious emotion in children with ASD (e.g., Hobson et al., 2006). Whereas a neurotypical child might feel self-conscious or embarrassed at others' reactions to their gender incongruent behaviour and thus seek to conform to gender-typical norms, a gender-incongruent child with ASD would likely be much less moved to change by the attitudes of others. As a result, with diminished pressure for conformity, gender-related attributes and roles might not easily become incorporated into the self-concept of children with ASD (e.g., Bargiela et al., 2016). This is important given, the dearth of evidence about a mechanism that could explain the link between ASD and gender dysphoria/incongruence (Glidden et al., 2016), but also because it denotes the continuous nature of gender and the influence of society upon the formation of the so-called binary gender identity (e.g., Ehrensaft, 2018; Turban & van Schalkwyk, 2018).

Moreover, this idea fits with some first person accounts by autistic people and about how they experience gender. As summarised by Davidson and Tamas (2016, p. 61), ""not only does gender not constitute the definitive core of autistic experience, but for many, gender is barely present at all". Our study suggests that this claim does not reflect difficulties with self-awareness (in birth-assigned females, at least). Rather, among people with high ASD traits there seems to be a match between their internal/nonconscious experience of gender self-concept and their explicit expression of this facet of self. The extent to which there is also the same level of consistency between the implicit and the explicit experience of gender self-concept among people with a diagnosis of ASD is still debatable, until it is directly explored.

Experiment 2: Research Aims and Hypotheses

To increase confidence in the veracity of the original findings and provide a safe ground to study gender-related cognition in the autistic population, we conducted a direct replication of our findings in a new sample. As noted in the previous chapter, it is very important researchers to conduct replication research on their own published work (Cesario, 2014). Based on the original findings, we predicted that the number of self-reported ASD traits would be negatively and significantly associated with the size of implicit and explicit gender self-concept (more ASD traits = less strong implicit/explicit gender self-concept).[12]

Experiment 2: Method

Participants

One hundred and twenty-six adults (97 birth-assigned female) took part in this study. The mean age of participants was 20.99 years (*SD* = 4.10; age range = 18 to 45 years). Two participants had a history of ASD, according to self-report, and 76.2% of participants reported English as their first language. One birth-assigned female participant identified as male, and one birth-assigned male participant did not identify either as female or male. Student participants were rewarded with course credit in partial fulfilment of their degree, and people from the general population did not receive any kind of compensation for taking part in the study. All participants gave informed consent, and the study was approved by City, University of London's Psychology Research Ethics Committee.

Just as in the original study, participants who scored below the median score on the AQ (i.e. *Mdn* = 18.00) were assigned in the low AQ-group, whereas people

[12] Please note that Experiment 2 was conducted after the publication of Experiment 1 (i.e., Kallitsounaki & Williams, 2020b).

who scored above the median were categorised in the high-AQ group. The low-AQ group included 16 birth-assigned males and 48 birth-assigned females, whereas the high-AQ group included 13 birth-assigned males and 49 birth-assigned females. The difference between the two groups in sex ratio was nonsignificant, $\chi 2(1) = 0.29$, p = .591, Cramer's V = .05. Also, participants in the low-AQ group (n = 64, M_{age} = 21.30, SD = 4.69) and those in the high-AQ group (n = 62; M_{age} = 20.68; SD = 3.40) did not differ significantly in age, $t(124) = 0.85$, $p = .399$, $d = 0.15$.

Materials and Procedures

All the materials employed and the procedures followed in the replication study were identical to the ones reported in the original study. As in the original study, participants completed Greenwald and Farnham's (2000) Implicit Association Test (IAT), the Personal Attributes Questionnaire (PAQ; Spence & Helmreich, 1978), and the Autism-Spectrum Quotient (AQ; Baron-Cohen, Wheelwright, Skinner, et al., 2001). To categorise participants as having high or low number of self-reported ASD traits, we employed the median split (we also used the AQ threshold of 26 to split participants; results of the analyses are reported in Appendix D). The main analyses of the study were reconducted including only non-autistic participants. Results did not change substantively (see Appendix D). Participants either took part in a laboratory administration of the measures or completed the study online through SONA.

Statistical Analyses

To investigate the success of our replication attempt, we followed the statistical methods described in Chapter 4. That is, we analysed whether those results reported as significant in the original study (see Experiment 1) were replicated in the current study, using an alpha level of .05 as the cut-off for statistical significance. We

also conducted a series of Fisher's *Z* tests and Bayesian analyses. According to Jeffreys's (1961) criteria, Bayes factors > 1 indicate increasing evidence for the alternative hypothesis over the null hypothesis (1 - 3 = anecdotal evidence; 3 - 10 = substantial evidence; 10 - 30 = strong evidence; 30 - 100 = very strong; values > 100 = decisive evidence). Whereas, scores < 1 indicate evidence for the null hypothesis over the alternative hypothesis (1 - 0.33 = anecdotal evidence; 0.33 - 0.10 = substantial evidence; 0.10 - 0.03 = strong evidence; 0.03 - 0.01 = very strong evidence; scores < 0.01 decisive evidence). When a priori directional predictions were made, BF_{10} values for one-tailed tests are reported. Bayesian analyses were performed using the statistical software package JASP 0.8.1.2 (JASP Team, 2016).

Experiment 2: Results

Preliminary Analyses

Means (*SD*) for participants' performance on the measures employed in this study are presented in Table 11. Participants' performance on the gender IAT task was above chance, $t(122) = 10.49$, $p < .001$, $d = 0.95$, $BF_{10} > 100$, as was the mean proportion of correct item categorisation in the critical blocks of the task, $t(122) = 114.42$, $p < .001$, $d = 10.32$, $BF_{10} > 100$.

Furthermore, a correlation analysis showed that the bipolar scale of PAQ was negatively and significantly associated with the untransformed *D* scores of the gender IAT, $r = -.28$, $p = .002$, $BF_{10} = 13.81$. Results indicate that the more a person explicitly endorsed masculine attributes as self-descriptive, the more they implicitly associated their self-concept with masculine attributes on the IAT. Likewise, the more a person explicitly endorsed feminine attributes, the more they implicitly associated their self-concept with feminine attributes. A Fisher's *Z* test showed that the magnitude of the association between the bipolar scale of PAQ and the

untransformed *D* scores of the gender IAT did not differ significantly from the size of the equivalent association reported in the original study, $Z = 0.17$, $p = .868$.

Table 11

Means (SDs) of Explicit and Implicit Gender Self-Concept and ASD Traits

Variable	Mean (*SD*)
Explicit gender-self concept	
PAQ femininity	2.87 (0.63)
PAQ masculinity	2.28 (0.63)
PAQ bipolar scale	1.80 (0.55)
Implicit gender self-concept [a]	
IAT (*D* score)	0.41 (0.44)
IAT accuracy rate	90.02 (8.73)
ASD traits	
AQ total	18.22 (5.89)

Note. PAQ = Personal Attributes Questionnaire; IAT = Implicit Association Test; AQ = Autism-Spectrum Quotient.

[a] Due to an error in the data collection, three birth-assigned females did not complete the IAT, hence $n = 123$.

Lastly, a series of independent samples *t*-tests was conducted to examine differences in the explicit and the implicit measure of gender self-concept between birth-assigned males and birth-assigned females. As shown in Table 12, birth-assigned males scored significantly higher on PAQ masculinity scale than birth-assigned females, yet significantly lower on PAQ femininity scale. Results indicate that birth-assigned males ascribed more masculine attributes to their self-concept, compared to birth-assigned females, whereas birth-assigned females ascribed more feminine attributes to their self-concept, compared to birth-assigned males. The

analysis also yielded a significant difference in the IAT *D* scores (untransformed) between birth-assigned females and birth-assigned males. Results suggest that birth-assigned females implicitly associated their self-concept more strongly with feminine than masculine attributes, whereas birth-assigned males did not show a strong implicit association of their self-concept either with masculine or feminine attributes.

Table 12

Means (SDs) and Results of t-Tests for Sex Differences in the Explicit and Implicit Measures of Gender Self-Concept

Variable	Birth-assigned sex		Group differences			
	Male (n = 29)	Female (n = 97)	t	p	d	BF_{10}
PAQ masculinity	2.71 (0.68)	2.15 (0.55)	4.46	<.001	0.94	>100
PAQ femininity	2.49 (0.72)	2.98 (0.56)	-3.89	<.001	-0.82	>100
IAT (*D* score) [a]	0.03 (0.43)	0.53 (0.37)	-6.17	<.001	-1.31	9.77

Note. PAQ = Personal Attributes Questionnaire; IAT = Implicit Association Test.

[a] Descriptive statistics are reported for untransformed *D* scores.

Association Analyses

A series of correlation analyses was conducted to investigate the relations between AQ score, PAQ femininity score, PAQ masculinity score, and IAT *D* score. The results of the analyses are reported and compared to that of the original study in Table 13. In keeping with our predictions, AQ was negatively and significantly associated with both PAQ femininity and PAQ masculinity scale scores, suggesting that the more ASD traits, the less strong the endorsement to feminine and masculine attributes as self-descriptive. The Bayes factors indicated that there is very strong and decisive evidence to support the alternative hypothesis, respectively. A Fisher's Z test revealed that neither the AQ × PAQ femininity correlation not the AQ × PAQ

masculinity association differ significantly from the equivalent associations reported in the original study. We also examined whether any of these effects differed significantly between birth-assigned males and birth-assigned females. Results showed that AQ was negatively and significantly associated with PAQ femininity and PAQ masculinity among birth-assigned males (AQ × PAQ femininity: r = -.54, p = .003, BF_{10} = 16.51; AQ × PAQ masculinity: r = - .49, p = .007, BF_{10} = 7.82) *and* birth-assigned females (AQ × PAQ femininity: r = -.27, p = .008; BF10 = 4.23; AQ × PAQ masculinity: r = -.46, p < .001, BF_{10} > 100).

Contrary to our hypothesis, AQ was not significantly associated with IAT *D* scores. Bayes factor provided anecdotal evidence for the null hypothesis. Nonetheless, the size of the AQ × IAT *D* did not differ significantly from that reported in the original study. The association between AQ and IAT (*D* score) was nonsignificant either among birth-assigned males (r = .02, p = .932, BF_{10} = 0.23) or birth-assigned females (r = -.16, p = .114, BF_{10} = 0.44).

Table 13

Bivariate Correlations Differences Between the Current and the Original Study

Association	Current study	Original study	Fisher's *Z* test
AQ × PAQ femininity	-.30 [b] ***	-.43***	Z = -1.12, p = .261
AQ × PAQ masculinity	-.44 [c] ***	-.35***	Z = 0.75, p = .451
AQ × IAT (*D* score)	-.13 [a] †	-.25**	Z = -0.93, p = .352

Note. PAQ = Personal Attributes Questionnaire; IAT = Implicit Association Test; AQ = Autism-Spectrum Quotient.

[a] BF_{10} = 0.34 - 0.99, one-tailed. [b] $BF_{10} \geq 30$, one-tailed. [c] $BF_{10} \geq 100$, one-tailed.

†$p \leq .10$, one-tailed. **$p < .01$, one-tailed. ***$p < .001$, one-tailed.

Case-Control Analysis

A series of independent samples *t*-tests was conducted to examine differences between the low-AQ group and the high-AQ group in the explicit and implicit measures of gender self-concept. Results of the analyses are presented in Table 14. As expected, relative to participants in the low-AQ group, those in the high-AQ group scored significantly lower in both PAQ scales. Contrary to predictions, there was not a significant difference in *D* score between participants in the low-AQ group and those in the high-AQ group. To examine whether any of these effects differed significantly between birth-assigned males and birth-assigned females, we conducted a series of 2 (birth-assigned sex: male/female) × 2 (group: low AQ/high AQ) ANOVAs. None of the analyses yielded a significant 2-way interaction (all *p*s > .062).

Table 14

Means (SDs) and Inferential Statistics for Group Differences

Variable	Group		Group Differences			
	Low AQ (n = 64)	High AQ (n = 62)	t	p [b]	d	BF_{10} [b]
IAT (*D* score)	0.53 (0.35)	0.47 (0.32) [a]	0.89	.189	0.16	0.44
PAQ femininity	2.98 (0.60)	2.75 (0.65)	2.00	.024	0.36	2.22
PAQ masculinity	2.52 (0.51)	2.04 (0.64)	4.66	<.001	0.83	> 100

Note. IAT = Implicit Association Test; PAQ = Personal Attributes Questionnaire.

[a] n = 59. [b] Values for one-tailed tests are reported.

Experiment 2: Discussion

In keeping with the original findings, we found that AQ score was associated negatively and significantly with both PAQ femininity and PAQ masculinity scale scores (more ASD traits = less strong explicit identification with masculine and

feminine attributes) . The size of the associations was equivalent to that reported in the original study, and Bayesian analysis suggested that the data supported the alternative hypothesis. Contrary to expectations, however, we failed to replicate the significant association between ASD traits and the strength of the implicit gender self-concept found in the original study. In this study, the AQ × IAT *D* score was negative as expected, but nonsignificant. Noteworthy, the size of the association was not significantly different from that reported in the original study, and Bayesian analysis provided only anecdotal evidence for the null hypothesis.

General Discussion

This chapter focuses on the effect of ASD on gender-related cognition. The continuously distributed nature of ASD traits allows us to gather important information about ASD itself by investigating among people from the general population the relation between these traits, on the one hand, and the strength of the explicit and implicit gender self-concept, on the other hand. That is the approach we followed in the studies (i.e., original and replication) included in this chapter. In both studies, we found that the number of self-reported ASD traits was negatively and significantly associated with the number of masculine and feminine attributes reported by people as self-descriptive. Results indicate that people with elevated ASD traits have a difficulty in identifying explicitly with attributes that are stereotypically ascribed to males and females.

These results are in keeping with previous findings that indicated people with a diagnosis of ASD tend to report a weaker masculine self-concept than do neurotypical individuals. This is important and increases our confidence in the reliability of previous findings linking ASD and weak masculine self-concept (Bejerot & Eriksson, 2014; Stauder et al., 2011). Given that only two previous

studies have explored this link, the replication of effects in the general population is notable and should make a valuable contribution to the field. More than this, however, the current study provides the first evidence that ASD is also linked to a diminished feminine self-concept. The studies by Bejerot and Eriksson (2014) and Stauder et al. (2011) found that ASD and control samples self-reported equally strong identification with feminine attributes, which led the authors of those studies to conclude that ASD was linked specifically with a reduced identification with masculine attributes. While such a conclusion was reasonable on the basis of their findings, alternative explanations are possible. For example, given that Stauder et al.'s (2011) study had statistical power of only .26 to detect a predicted moderately-sized difference between birth-assigned females with ASD (n = 9) and birth-assigned females without ASD (n = 9) in Gender Feminine scale, it is possible that a Type I error was made. Also, the Bem Sex Role Inventory, used by Bejerot and Eriksson (2014), includes items that are significantly more desirable for one sex than the other (Hoffman, 2001) and so could have confounded results in that study (e.g., if neurotypical birth-assigned males failed to report feminine attributes because of social undesirability).

Importantly, both studies included a self-report measure of gender self-concept that did not bias participants against self-endorsement of feminine attributes, as it includes psychological attributes that are equally desirable for both sexes (Hoffman, 2001). Moreover, Bayesian analyses were conducted in the replication study to provide a more complete picture of the results than available in previous studies of the link between gender self-concept and ASD. Given this, findings suggest that people with high ASD traits do report a less strong feminine self-concept, compared to people with low ASD traits.

Furthermore, in the original study, we found a negative and significant association between ASD traits and the strength of the implicit gender self-concept. An exploratory analysis, however, revealed that this relation was significant only among birth-assigned females. Given the preliminary nature of these findings, we attempted to replicate them in a new sample. Contrary to the findings of the original study, the relation between number of self-reported ASD traits and the strength of the implicit gender self-concept was nonsignificant. Results did not change substantively (i.e., from nonsignificant to significant) when this relation was examined among birth-assigned males and females separately. Of course, we cannot argue that this effect does not exist in the population, based on a single failure to replicate the original finding (Simons, 2014; Stroebe & Strack, 2014). Failure to replicate the original results is not sufficient to deem them false-positive (Simonsohn, 2016). Based on the results from the Bayesian analyses and the Fisher's Z tests we conducted, it can be argued that our replication was *inconclusive*. Therefore, we cannot conclude with confidence that people with high ASD have a weaker propensity to incorporate into their self-concept gender-differentiated attributes.

It is important to note that the findings presented in this chapter should be interpreted in light of the limitations of the gender research tradition we adopted. Although, direct and indirect measures of gender-stereotypical dimensions of personality have been widely used in research (e.g., Greenwald & Farnham, 2000; September et al., 2001; van Well et al., 2007; Yarnell et al., 2019), we should not overlook the fact that in recent years differences in gender-stereotypical attributes seem to be less prominent between birth-assigned males and birth-assigned females (Donnelly & Twenge, 2017; Hentschel et al., 2019). This was evidence in both studies we conducted. In the original study, birth-assigned males and birth-assigned

females ascribed to their self-concept masculine attributes, as self-descriptive to the same degree. Furthermore, none of the two studies indicated a strong implicit male self-concept among birth-assigned males. To overcome this potential limitation, researchers might usefully adopt the *gender self-categorisation* research approach in future studies (for a detailed description of this approach, see Chapter 1).

In conclusion, the findings presented in this chapter provide *strong evidence* that people with high ASD traits have a weaker propensity to explicitly identify with gender-differentiated attributes. Yet, it remains unclear whether this is accompanied by a difficulty incorporating gender-differentiated attributes into their self-concept. Some preliminary evidence suggests that this might be the case only among birth-assigned females. ASD might lead to a weaker implicit identification with gender-differentiated traits in a number of ways. Given that there may be qualitative differences in the underlying mechanisms that underpin ASD traits in people from general population and those in people with a diagnosis of ASD (e.g., Peterson et al., 2005), further research among people with a diagnosis of ASD is essential to shed light on this topic.

Chapter 6

Gender-Related Cognition, Gender Dysphoric Feelings, ASD Traits, and Mentalising Ability: Differences Between Autistic and Neurotypical Cisgender and Transgender Adults

In the previous chapters, we attempted to answer whether (a) there is a link between ASD and gender dysphoria/incongruence, (b) mentalising plays a role in the high co-occurrence of ASD and gender dysphoria/incongruence, and (c) ASD affects gender-related cognition. To do so, we presented the findings of a literature review, two meta-analyses, and two studies in the general population (i.e., original and replication).

As already noted, research has shown that the continuity of ASD traits in the general population allows us to gain important information about people with a diagnosis of ASD by investigating the relation between the number of ASD traits and other variables of interest in neurotypical people (e.g., Lind et al., 2020; Williams, Bergström, et al., 2018). We should not overlook, however, that there may be qualitative differences in the underlying mechanisms that underpin ASD traits in people from general population and those in people with a diagnosis of ASD (e.g., Peterson et al., 2005). Thus, before we drew strong conclusions from the findings presented in the previous chapters, we decided to test a number of hypotheses associated to our research questions within the autistic (cisgender and transgender) population rather than the general population. Specifically, this chapter presents the findings of a case-control study. The aim of this study was to inform our understanding of the high co-occurrence of ASD and gender dysphoria/incongruence, examining gender-related cognition, gender dysphoric feelings, recalled childhood gender-typed behaviour, ASD traits, and mentalising

ability in autistic and neurotypical cisgender and transgender individuals. We also explored alexithymia in these groups.

Research Question A: Does ASD Affect Gender-Related Cognition?

Little is known about the mental processes involved in the formation and consolidation of gender self-concept in ASD. The sparse research conducted on this topic has shown that autistic adults show a reduced propensity to identify explicitly with personality traits and gender roles that stereotypically characterise males (Bejerot & Eriksson 2014; Stauder et al., 2011). It has also been found that autistic people feel less positively and identify less strongly with the gender group that matches their experienced/reported gender than do neurotypical individuals (Cooper et al., 2018). Yet, a note of caution should be added here, as these findings are from self-report measures only. Self-report measures rely on people's accurate representation of their own feelings, emotions, traits, and thoughts. Given that aspects of self-awareness are considered to be atypical in ASD (e.g., Carruthers, 2009; Gopnik, 1993; Williams, 2010), it is unclear whether these findings reflect an atypical implicit self-experience of gender (and hence, accurate self-reporting) or difficulties in the explicit representation of this concept. Self-report measures are also considered problematic when evaluating sensitive topics (e.g., Rasinski et al., 2005), such as the study of gender self-concept/identity. To overcome these issues, implicit measures can also be employed (Wood & Eagly, 2009).

To our knowledge, no study has used an implicit measure to investigate gender-related cognition in autistic people. As described in Chapter 5, we adopted an individual differences approach and examined how gender self-concept varies according to the number of ASD traits manifested by people from the general population, using not only a self-report measure but also an *implicit* association test

(IAT; Greenwald & Farnham, 2000). In the original study (see Chapter 5, Experiment 1), we found that people from the general population with high ASD traits had a weaker inclination to ascribe explicitly to themselves, feminine *and* masculine gender stereotypical attributes. These findings were successfully reproduced in the replication study we conducted (see Chapter 5, Experiment 2), indicating a robust and reliable link between ASD traits and explicit identification with gender-differentiated attributes among people from the general population. In the original study, we also found that people from the general population with high ASD traits had a weaker inclination to ascribe *implicitly* to themselves feminine and masculine gender-differentiated attributes. An exploratory analysis, however, showed a selective influence of ASD traits on the implicit identification with gender-differentiated attributes among birth-assigned females only. Nonetheless, the replication study we conducted failed to reproduce these results.

Given the potential limitations of the research tradition we employed to study gender self-concept, the first aim the current study was to replicate conceptually, as well as extend our previous findings further, by examining *gender-group identification*, instead of identification with gender stereotypical *personality attributes*. As described in detail in Chapter 1, gender-group identification implicates people's sense of belonging to one gender group as opposed to another and is considered as one of the two traditions of research on gender self-concept (Wood & Eagly, 2015). In the current study, we examined *gender-group identification,* using an explicit self-report measure and an IAT (Greenwald et al., 2002). Based on our previous findings, we made the following hypothesis:

> *Hypothesis A1*. The number of self-reported ASD traits of neurotypical cisgender people would be negatively and significantly associated with the

strength of gender self-concept (more ASD traits = weaker explicit and implicit gender-group identification). This relation would hold its significance for both birth-assigned males (more ASD traits = weaker explicit and implicit identification with male gender groups) and birth-assigned females (more ASD traits = weaker explicit and implicit identification with female gender groups).

Next, we attempted to extend our previous findings further by examining gender-group identification among people with a self-reported diagnosis of ASD. Autistic people's sense of belonging to one gender group over another was examined in cisgender and transgender people separately, for the first time. This is important to understand how exactly ASD affects gender-related cognition. It is important to mention that Olson et al. (2015) were the first and only researchers to use an IAT to examine gender-related cognition in the transgender population. They found that transgender children implicitly perceived themselves in keeping with their experienced/reported gender. To our knowledge, no study has employed an IAT in transgender *adults*. On the basis of previous findings (e.g., Cooper et al., 2018; Kallitsounaki & Williams, 2020b; Olson et al., 2015), we made the following hypotheses:

Hypothesis A2. Within each group (autistic cisgender/autistic transgender/neurotypical cisgender/neurotypical transgender), scores on the explicit and the implicit measures of gender self-concept would align with experienced/reported gender rather than birth-assigned sex (i.e., individuals who identify as females would show an explicit and implicit female self-concept, whereas people who identify as males would show an explicit and implicit male self-concept).

Hypothesis A3. Regardless of birth-assigned sex, we expected autistic participants who identify either as cisgender or transgender to show a significantly weaker explicit and implicit gender self-concept than neurotypical people.

Research Question B: Do Autistic People Have Increased Gender Dysphoric Feelings and Recall Limited Gender-Typed Behaviour From Childhood?

The second aim of this study was to examine whether autistic people have increased current gender dysphoric feelings and whether they recall limited gender-typed behaviour from their childhood years. Research has shown that autistic people report a more diverse range of gender identities than neurotypical individuals (Bejerot & Eriksson, 2014; George & Stokes, 2018b), and that they are more likely to be planning or have transitioned (Cooper et al., 2018). To date, only George and Stokes (2018b) have employed a validated measure to examine gender dysphoric feelings in autistic people. Using the Gender Identity/Gender Dysphoria Questionnaire for Adolescents and Adults (GIDYQ-AA; Deogracias et al., 2007), George and Stokes found that autistic people reported significantly more current gender dysphoric feelings than neurotypical individuals. We should note, however, that in George and Stokes' online study the percentage of participants who did not identify as cisgender was 3 times higher in the autistic group (30%) than in the neurotypical group (10%). The inclusion of these people in the analysis could have artificially inflated the score of the autism group, creating a significant difference in the number of current gender dysphoric feelings between autistic and neurotypical people. As such, the extent to which autistic *cisgender* adults have increased gender dysphoric feelings remains unclear. To answer this question in the current study, we examined current gender dysphoric feelings among autistic cisgender and autistic

transgender people separately. We also examined, for the first time, whether these groups recall limited gender-typed behaviour from childhood. On the basis of previous findings that indicate an increase likelihood of autistic adults to be gender dysphoric/incongruent (e.g., Bejerot & Eriksson, 2014; Pecora et al., 2020; Pohl et al., 2014; Walsh et al., 2018) and an increased prevalence of a diagnosis of gender dysphoria in autistic children (Hisle-Gorman et al., 2019), we made the following hypotheses:

Hypothesis B1. We expected autistic people who identify either as cisgender or transgender to report significantly more gender dysphoric feelings than neurotypical cisgender people, autistic cisgender people to report significantly less gender dysphoric feelings than neurotypical transgender individuals, and autistic transgender people to report significantly more gender dysphoric feelings than neurotypical transgender individuals (autistic transgender > neurotypical transgender > autistic cisgender > neurotypical cisgender).

Hypothesis B2. We expected autistic people who identify either as cisgender or transgender to recall significantly less gender-typed behaviour from childhood than neurotypical cisgender people, autistic cisgender people to recall significantly more gender-typed behaviour from childhood than neurotypical transgender individuals, and autistic transgender people to recall significantly less gender-typed behaviour from childhood than neurotypical transgender individuals (autistic transgender < neurotypical transgender < autistic cisgender < neurotypical cisgender).

Research Question C: Do Transgender People Have Increased ASD Traits, Diminished Mentalising Ability, and High Levels of Alexithymia?

The third aim of the current study was to examine whether transgender people (autistic and neurotypical) have increased ASD traits, difficulties mentalising, and high levels of alexithymia. With respect to the prevalence of ASD traits in gender dysphoric/incongruent people, previous studies have produced mixed and inconclusive results. Specifically, Vermaat et al. (2018) did not find a significant difference in the number of self-reported ASD traits, measured with the Autism-Spectrum Quotient (AQ-50; Baron-Cohen, Wheelwright, Skinner, et al., 2001), between people referred to a gender identity clinic for GD and a sample of neurotypical individuals from the general population (d = 0.06). Yet, a number of studies in clinical and nonclinical transgender individuals have suggested there may be a selective difference in ASD traits between transgender and neurotypical control birth-assigned females only. Specifically, Jones et al. (2012) found that transgender birth-assigned females, recruited either from a gender identity clinic or via a website for participating in research projects, scored significantly higher on the AQ-50 than neurotypical birth-assigned females (d = 1.00), whereas the difference between transgender and control birth-assigned males was nonsignificant. Likewise, in an online study conducted by Kung (2020), transgender birth-assigned females reported significantly more ASD traits than control birth-assigned females from the general population (d = 0.77), whereas the difference in AQ-50 scores between transgender and control birth-assigned males from the general population was nonsignificant. Also, in Murphy et al.'s (2020) online study, transgender birth-assigned females scored significantly higher on the AQ-50 than cisgender birth-assigned males (d = 1.28), whereas a nonsignificant difference was observed between transgender and cisgender birth-assigned males. Similar results were found by Nobili, Glazebrook, Bouman, et al. (2018) in a sample of transgender participants recruited from a gender

identity clinic, but the size of the difference (d = 0.20) between transgender and cisgender birth-assigned females was smaller than that reported in previous studies. Surprisingly, transgender birth-assigned males scored significantly lower on the AQ-28 (Hoekstra et al., 2011) than cisgender birth-assigned males in this study. Lastly, in Pasterski et al.'s (2014) study, transgender birth-assigned females diagnosed with GD/GID scored higher on the AQ-50 than neurotypical birth-assigned females, but the difference was small (d = 0.31) and nonsignificant. It is important to note, however, that the study was underpowered to detect an effect of this size. In contrast to previous findings, Stagg and Vincent (2019) conducted an online study and found that transgender individuals scored significantly higher on the AQ-50 than cisgender people, regardless of participant birth-assigned sex. The difference in ASD traits between transgender and cisgender birth-assigned females was large (d = 1.74), and the difference between transgender and cisgender birth-assigned males was moderate (d = 0.66). Likewise, using a different measure (i.e., Social Responsiveness Scale for Adults), Heylens et al. (2018) found that birth-assigned males diagnosed with GD reported significantly more ASD traits than a norm group of birth-assigned males (d = 0.63) and birth-assigned females diagnosed with gender dysphoria reported significantly more ASD traits than a norm group of birth-assigned females (d = 0.78). Lastly, Warrier et al., (2020) examined ASD traits in transgender and gender-diverse individuals, using three independent samples. In all three, transgender and gender-diverse individuals reported significantly more ASD than cisgender people, yet sex differences were not examined.

We should note here that researchers, with the exception of Jones et al. (2012), Murphy et al. (2020), and Warrier et al. (2020), either did not control for (or did not collect/report information about) the presence of autistic people in gender

dysphoric/incongruent samples in their studies. This is important because research has shown a high prevalence of ASD diagnoses in this population (Strauss et al., 2021; Warrier et al., 2020), and as expected based on their diagnosis, autistic transgender people show significantly more ASD traits than neurotypical transgender people (Warrier et al., 2020). Arguably, the inclusion of autistic people in these studies could have inflated the AQ/SRS score of gender dysphoric/incongruent groups. Therefore, the extent to which *neurotypical* transgender people have increased ASD traits is not well studied. Furthermore, it still unclear whether the behavioural features of ASD in neurotypical transgender people are accompanied by the cognitive difficulties that are frequently encountered in ASD and which arguably underpin the behavioural features of the condition.

As already described in Chapter 1, ASD is characterized by well-established difficulties with mentalising ability (e.g., Baron-Cohen, Wheelwright, Hill, et al., 2001; Senju et al., 2009). Mentalising has been proposed as one of the mechanisms that could explain the high co-occurrence of ASD and gender dysphoria/incongruence (Glidden et al. 2016; Jacobs et al. 2014; Van Der Miesen et al. 2016; van der Miesen, Hurley, et al., 2018). To our knowledge, however, only two studies have examined mentalising in gender dysphoric/incongruent individuals. Stagg and Vincent (2019) did not find a significant difference in the Reading the Mind in the Eyes (RMIE; Baron-Cohen, Wheelwright, Hill, et al., 2001) task performance between cisgender and transgender people. In contrast, Kung (2020) found that transgender individuals performed significantly worse on the RMIE than control participants. We should note, however, that strong conclusions cannot be drawn from Stagg and Vincent's (2019) study because their sample was

underpowered to detect a small (d = 0.39), but potentially meaningful, difference between transgender and cisgender people.

Another important point we should mention here is that in neither Kung's (2020) nor Stagg and Vincent's (2019) study did researchers control for a possible overrepresentation of ASD diagnoses in the transgender group.[13] Recent evidence suggests that the prevalence of self-reported ASD diagnoses in transgender people might reach 22.5% (Strauss et al., 2021), when the equivalent prevalence in the general population is around 1% (e.g., Lai et al., 2014). Arguably, the inclusion of autistic people in transgender samples could artificially deflate this group's score on the RMIE. So, based on previous studies, we cannot conclude with confidence whether neurotypical transgender people show atypical mentalising ability. This leaves a critical gap in the literature that we aimed to fill by examining ASD traits and mentalising in *neurotypical* and *autistic* transgender individuals separately. On the basis of previous findings (Walsh et al., 2018; Warrier et al., 2020), we made the following hypotheses:

> *Hypothesis C1*. We expected neurotypical transgender participants to report significantly more ASD traits than neurotypical cisgender people, but significantly fewer than autistic cisgender individuals and autistic transgender people to report significantly more ASD traits than autistic cisgender people (autistic transgender > autistic cisgender > neurotypical transgender > neurotypical cisgender).

[13] Note that 14% of transgender and nonbinary individuals who participated in Stagg & Vincent's (2019) study reported being in receipt of a formal diagnosis of ASD. Also, 9% of Kung's (2020) transgender sample scored so highly (i.e., ≥ 35) on the AQ-50 that they likely had ASD.

Hypothesis C2. We expected neurotypical transgender individuals to perform significantly less well on the mentalising task than neurotypical cisgender people, but significantly better than autistic cisgender people and autistic transgender people to perform significantly less well on the task than autistic cisgender participants (autistic transgender < autistic cisgender < neurotypical transgender < neurotypical cisgender).

ASD also often co-occurs with alexithymia. Alexithymia describes a difficulty in identifying and describing emotions, as well as in distinguishing feelings from body sensations (Sifneos, 1973). Clinically significant levels of alexithymia are present in approximately 50% of autistic people (e.g., Hill et al., 2004), yet, little is known on whether alexithymia affects transgender people, as well. To our knowledge, only one study has examined this issue. Kessler et al. (2006) found that transgender individuals scored significantly higher than cisgender controls in the German version of the Toronto Alexithymia Scale (TAS; Kupfer et al., 2000), but the alexithymia traits they reported did not reach the level of clinical significance. Of course, in the absence of an autistic control group, no conclusions can be made on whether transgender and autistic people are affected by alexithymia to the same degree. To address this issue, in this study we explored alexithymia among autistic transgender, neurotypical transgender, autistic cisgender, and neurotypical cisgender people.

Research Question D: What are the Relations Between ASD Traits, Gender Dysphoric Feelings, and Mentalising?

The last aim of this study was to replicate and extend the findings presented in Chapters 3 and 4 by examining the links between ASD traits, gender dysphoric feelings, and mentalising ability in neurotypical cisgender, neurotypical transgender,

autistic cisgender, and autistic transgender people separately. The findings we presented in Chapter 3 indicated a link between ASD traits and current gender dysphoric feelings in the general population (more ASD = more gender dysphoric feelings). Results were in keeping with George and Stokes' (2018b) findings and successfully were reproduced in our replication study (see Chapter 4). Taken together, the link between ASD traits and gender dysphoric feelings seems robust and reliable in the general population. The findings presented in Chapter 3 also indicated a strong relation between mentalising and current gender dysphoric feelings (better mentalising = fewer gender dysphoric feelings), for the first time. These findings were successfully reproduced in the replication study included in Chapter 4. It remains unknown, however, whether these relations hold beyond the general population. This is a serious gap in the literature because the study of the links between ASD traits, gender dysphoric feelings, and mentalising ability in neurotypical cisgender, neurotypical transgender, autistic cisgender, and autistic transgender people separately, can provide important insight about the nature of the link between ASD and gender dysphoria/incongruence. Based on our findings from the general population, we made the following hypotheses:

Hypothesis D1. Within each group separately, AQ score would be negatively and significantly correlated with GIDYQ-AA score (more ASD traits = more current gender dysphoric feelings).

Hypothesis D2. Within each group, RMIE score would be positively and significantly correlated with GIDYQ-AA score (better mentalising = fewer current gender dysphoric feelings).

Method

Participants

One hundred and six neurotypical cisgender adults (51 birth-assigned female), 107 autistic cisgender adults (57 birth-assigned female), 78 neurotypical transgender adults (41 birth-assigned female), and 56 autistic transgender adults (27 birth-assigned female) took part in the current study. Participants who identified as gender nonconforming or unknown were excluded from the study (n = 4), whereas participants who identified as trans(masculine/male/female) nonbinary or masculine nonbinary (n = 4) were included in the transgender group. The number of birth-assigned females and males did not differ significantly between groups, χ^2 (3, N = 347) = 0.81, p = .846, φ = .05, but differences in age were found, F(3,343) = 26.99, p < .001, η_p^2 = .19 (please note that when participant groups were matched for age results of the analyses did not change substantively; see Appendix E). Ninety-nine percent of participants reported being native English speakers. According to self-report, all autistic participants had a formal diagnosis of ASD, and self-identifying transgender participants had clinically significant levels of gender dysphoria. Participants were recruited via the online crowdsourcing platform Prolific Academic, social media platforms, and a database of autistic individuals interested in taking part in psychological research. All participants completed the study online after they had given written, informed consent and received compensation for their time. This study was approved by the University of Kent's Psychology Research Ethics Committee (ID: 201915670711375862).

The current study was preregistered on Open Science Framework (preregistration can be viewed here: https://osf.io/bke5j). We should note, however, that none of the hypotheses about the autistic transgender group have been included in the preregistration. Yet, they were all made before any statistical analyses were conducted and if preregistered they would be exactly the same as the ones presented

in the current article (all deviations from the preregistration, as well as preregistered hypotheses and secondary analyses that have not been included here are reported in Appendix E).

Materials and Procedure

Implicit Measure of Gender Self-Concept

We assessed participants' implicit gender self-concept, using the Implicit Association Test (IAT) described by Greenwald et al. (2002). Participants were instructed to sort words that belonged to one of four categories, using one of two possible response keys. Categories and stimuli used in the task were (a) Self: I, me, my, mine, self; (b) Other: they, them, their, it, other; (c) Female: woman, girl, daughter, madam, lady, female; and (d) Male: man, boy, son, sir, gentleman, male.

In the first block (20 trials), the "self" category label was presented in the upper left corner of the screen and the "other" category label in the upper right corner. Participants were asked to sort words that belonged either to "self" or "other" category by pressing the "a" key of a keyboard for words related to "self" and the "l" key for words related to "other". In the second block (20 trials), categories referred to "female" and "male" categorisation. The "female" category label was presented in the upper left corner and the "male" category in the upper right corner. Participants were instructed to press the "a" key for words belong to "female" category and the "l" key for items belong to "male" category. In the third block (20 practice trials), the four categories were presented combined ("self/female" labels: upper left corner; "other/male" labels: upper right corner) and participants practiced the sorting task by pressing the key that was assigned to each category in the preceding two blocks (i.e., "a" key for items belonged to "self /female" categories and "l" key for items belonged to "other/male" categories). In the fourth block (40 experimental trials),

participants completed the first experimental condition of the combined sorting task. In the fifth block (40 trials), the “female” category label was presented in the upper right corner and the “male” category label in the upper left corner, subsequently the assignment of the key for each category was reversed, compared to the first block. Participants were instructed to press “l” for items belonged to the “female” category and “a” for items belonged to the “male” category. In the sixth block (20 practice trials), participants practiced the combined task, using the switched key assignments. They were instructed to press the “a” key to categorise items that belonged either to the “self” or to the “male” category and the “l” key for words that belonged either to the “other” or “female” category. In the last block (40 experimental trials), they completed the second experimental condition of the combined sorting task.

The dependent measure for the IAT was the strength of the automatic associations calculated with the scoring algorithm recommended by Greenwald et al. (2003). That is, a standardised mean difference score (namely *D*) in response latencies between the two practical blocks (i.e., block 3 and block 6) and the two experimental blocks (i.e., bock 4 and block 7). When participants respond faster in the “self-female and other-male” condition than in the “self-male and other-female” condition, they receive a positive *D* score indicating a female self-concept. When they respond faster in the “self-male and other-female” than in the “self-female and other-male” condition they receive a negative *D* score indicating a male self-concept. When error rate in the critical blocks (i.e., practical and experimental) exceeded 20%, participants (i.e., neurotypical cisgender: $n = 7$; neurotypical transgender: $n = 3$; autistic cisgender: $n = 14$; autistic transgender: $n = 8$) were not included in the analyses (Greenwald et al., 1998; van Well et al., 2008). Results of the analyses including all participants, regardless of their performance in the task, are reported in

Appendix E. The IAT was programmed using Inquisit Millisecond software package 4 (https://www.millisecond.com.) and administered using Inquisit Web Player 4.0.10. After the completion of the IAT, participants were automatically redirected to a Qualtrics survey to complete the rest of the tasks and questionnaires.

Explicit Measure of Gender Self-Concept

We assessed participants' explicit gender self-concept, using the self-report measure designed by Greenwald et al. (2002). Participants were asked to rate each of the six male and six female nouns used in the IAT, using a 7-point Likert scale ranging from "not at all characteristic to you" to "extremely characteristic of you". The measure was scored by subtracting participants' average score on the male nouns from that on the female nouns. Positive scores denote a female self-concept and negative scores denote a male self-concept.

Other Tasks and Self-Report Measures

Participants were asked to indicate their birth-assigned sex, such as on an original birth certificate (i.e., male or female) and their gender identity (i.e., male, female, trans male, trans female, gender nonconforming, or other). Participants whose birth-assigned sex was congruent to their gender identity were categorised as cisgender, and those whose birth-assigned sex was incongruent to their gender identity or identified as transgender were categorised as transgender.

Participants completed the Reading the Mind in the Eyes test (RMIE; Baron-Cohen, Wheelwright, Hill, et al., 2001), which is a widely used measure of adult mentalising, the Autism-Spectrum Quotient (AQ; Baron-Cohen, Wheelwright, Skinner, et al., 2001) that measures ASD traits (scores $\geq$ 26 denote clinically significant levels of ASD traits), the Gender Identity/Gender Dysphoria Questionnaire for Adolescents and Adults (GIDYQ-AA; Deogracias et al., 2007),

which is a reliable self-report measure that taps gender identity and gender dysphoria (scores ≤ 3 denote clinically significant levels of gender dysphoria), and the18 items of the Recalled Childhood Gender Identity/Gender Role Questionnaire (RCGI; Zucker et al., 2006) that assess gender role behaviour and gender identity.[14]

Lastly, participants completed the Toronto Alexithymia Scale (TAS-20; Bagby et al., 1994). That is widely used and reliable 20-item self-report measure that taps people's awareness and understanding of, and ability to describe, their own bodily sensations and feelings. The TAS presents participants individual statements (e.g., "I am often confused about what emotion I am feeling"), and they are asked to decide the extent to which they agree with each statement, responding on a 5-point Likert scale, ranging from "strongly disagree" to "strongly agree". Scores range from 20 to 100, with higher scores denoting more alexithymia scores. A score of > 60 indicate clinically significant alexithymia. TAS-20 shows good internal consistency (Cronbach's α around .80) in clinical and nonclinical samples (Bagby et al., 1994; Parker et al., 2003).

Results

Association Analyses between the Explicit and the Implicit Measures of Gender Self-Concept

In keeping with the preregistration, a correlation analysis was conducted to examine the association between the explicit and the implicit measures of gender self-concept. As predicted, we found that, within each group, scores from the explicit measure were positively and significantly associated with scores from the IAT

[14] For a detailed account of the Reading the Mind in the Eyes test, the Autism-Spectrum Quotient, the Gender Identity/Gender Dysphoria Questionnaire for Adolescents and Adults, and the Recalled Childhood Gender Identity/Gender Role Questionnaire, see Chapter 3.

(neurotypical cisgender: $r = .79$, $p < .001$, one-tailed; autistic cisgender: $r = .72$, $p < .001$, one-tailed; neurotypical transgender: $r = .65$, $p < .001$, one-tailed; autistic transgender: $r = .63$, $p < .001$, one-tailed). Results suggest that participants' explicit gender self-concept was in keeping with their implicit gender self-concept. That is, participants who identified explicitly with female groups identified implicitly with female groups as well, and participants who identified explicitly with male groups identified implicitly with male groups as well.

Gender-Related Cognition

Associations Between AQ, and Performance on the Explicit and the Implicit Measures of Gender Self-Concept

In keeping with the preregistration, a series of correlation analyses was conducted to investigate the relations between AQ, and performance on the explicit and implicit measures of gender self-concept among neurotypical cisgender individuals (note: scores from the explicit and implicit measures of gender self-concept were transformed to positive values so that higher scores denote a stronger gender self-concept, regardless of whether it is male or female). As predicted, AQ score was negatively and significantly related to the strength of the explicit gender self-concept (Hypothesis A1), $r = -.32$, $p < .001$ (one-tailed). Given that TAS score was positively and significantly correlated with AQ score, $r = .42$, $p < .001$ and negatively and significantly associated with the strength of the explicit gender self-concept, $r = -.27$, $p = .006$, we performed a series of partial correlations to examine whether the shared variance between AQ and the strength of the explicit gender self-concept can be attributed to alexithymia. The relation between AQ score and the strength of the explicit gender self-concept, *controlling for TAS*, remained significant, $r = -.24$, $p = .015$. In contrast, the relation between TAS and the strength

of the explicit gender self-concept, *controlling for AQ score,* was nonsignificant, r = -.15, p = .118. Results suggest that the more ASD traits reported by neurotypical cisgender individuals, the lower the strength of their explicit gender self-concept independent of the effects of alexithymia.

Following the preregistration, the also examined the relations between AQ, and performance on the explicit and implicit measures of gender self-concept among birth-assigned males and birth-assigned females separately (note: scores from the explicit and implicit measures of gender self-concept were untransformed so that positive scores denote a female self-concept and negative scores a male self-concept). As predicted (Hypothesis A1), the relation between AQ and the strength of the explicit gender self-concept held its significance for birth-assigned males, r = .41, p = .001, one-tailed (high ASD traits = weaker male self-concept) and birth-assigned females, r = -.25, p = .036, one-tailed (high ASD traits = weaker female self-concept).

Contrary to predictions, however, AQ did not correlate significantly with the strength of the implicit gender self-concept among neurotypical cisgender people (Hypothesis A1), r = -.06, p = .276 (one-tailed). Results remained nonsignificant when the analysis was performed for each birth-assigned sex separately (birth-assigned males: r = -.17, p = .127, one-tailed; birth-assigned females: r = -.08, p = .286, one-tailed).

Performance on the Explicit Measure of Gender Self-Concept

A 2 (birth-assigned sex: male/female) × 2 (diagnostic category: neurotypical/autistic) × 2 (gender identity: cisgender/transgender) ANOVA was conducted on participant scores from the explicit measure of gender self-concept. Nonsignificant main effects were detected for birth-assigned sex, $F(1,339) = 2.18$, p

= .140, η_p^2 = .01, gender identity, $F(1,339) = 0.05$, $p = .829$, η_p^2 = .00, and diagnostic category, $F(1,339) = 0.142$, $p = .707$, η_p^2 = .00. Nonetheless, the analysis revealed a significant Birth-Assigned Sex × Gender Identity interaction, $F(1,339) = 2843.37$, $p < .001$, η_p^2 = .89, a significant Birth-Assigned Sex × Diagnostic Category interaction, $F(1,339) = 34.46$, $p < .001$, η_p^2 = .09, a significant Gender Identity × Diagnostic Category interaction, $F(1,339) = 6.49$, $p = .011$, η_p^2 = .02, and a significant three-way interaction, $F(1,339) = 21.33$, $p < .001$, η_p^2 = .06.

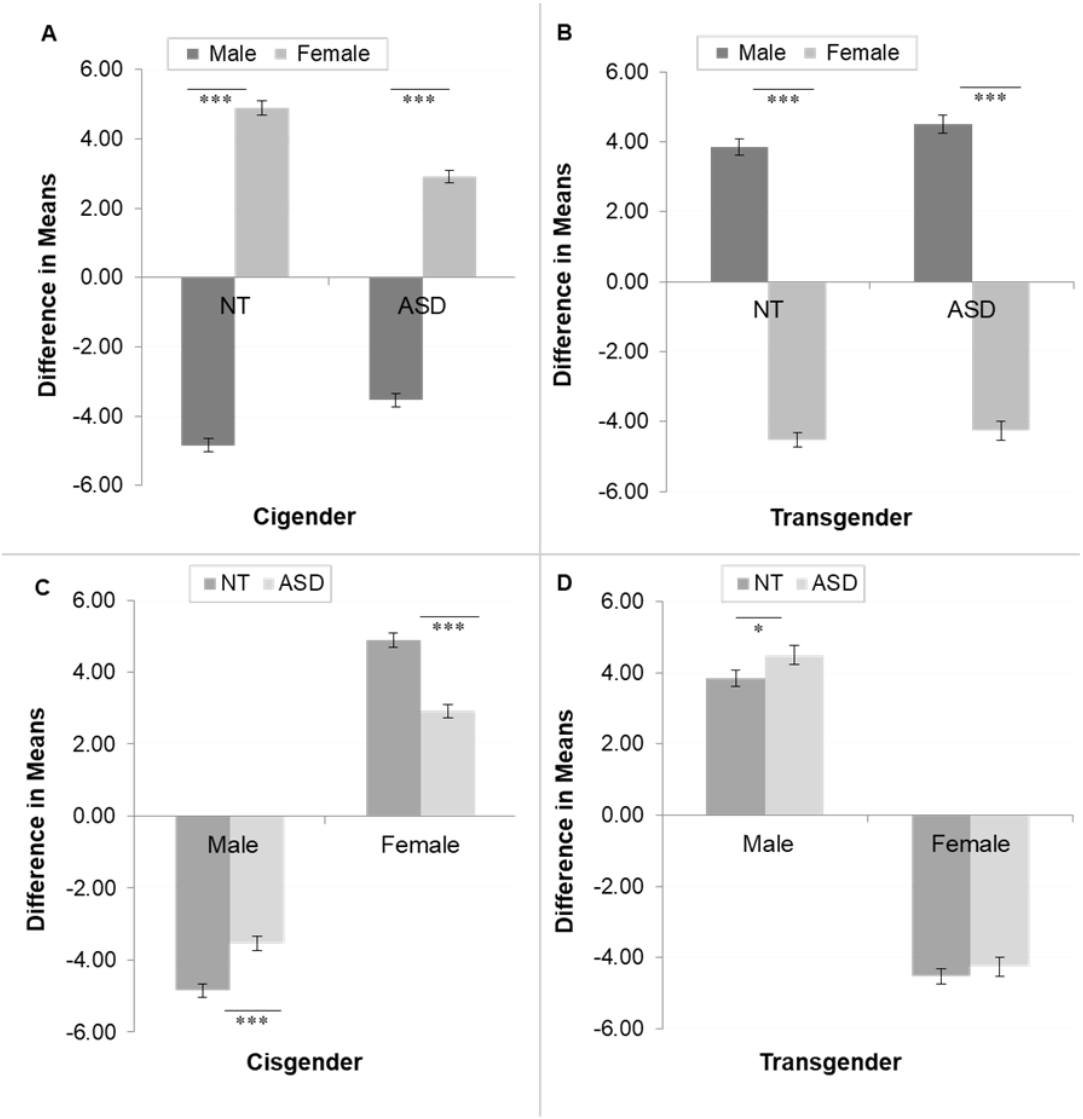
Breaking down the three-way interaction, a simple effects analysis of birth-assigned sex within gender identity and diagnostic category indicated that, in accordance with the preregistered Hypothesis A2, the explicit measure of gender self-concept was sensitive to gender identity differences. Results of the analysis are illustrated in Figure 8A and B. Specifically, we found that among neurotypical cisgender and autistic cisgender individuals, birth-assigned females scored significantly *higher* than birth-assigned males (note: mean scores of birth-assigned females were significantly above zero, and mean scores of birth-assigned males were significantly below zero, all *p*s < .001 one-tailed). This indicates that birth-assigned females who identify as females showed an *explicit female self-concept* and birth-assigned males who identify as males showed an *explicit male self-concept*. The opposite pattern was observed among neurotypical transgender and autistic transgender individuals, with birth-assigned females scoring significantly *lower* than birth-assigned males (note: mean scores of birth-assigned females were significantly below zero, and mean scores of birth-assigned males were significantly above zero, all *p*s < .001 one-tailed). This indicates that birth-assigned females who identify as males showed an *explicit male self-concept* in line with their experienced/reported (rather than birth-assigned) gender, whereas birth-assigned males who identify as

females showed an *explicit female self-concept* in line with their experienced/reported (rather than birth-assigned) gender. These results were entirely expected.

Next, we conducted a simple effects analysis of diagnostic category within birth-assigned sex and gender identity. The results of the analysis are illustrated in Figure 8C and D. In line with the preregistered Hypothesis A3, autistic cisgender birth-assigned males scored significantly higher on the explicit task than neurotypical cisgender birth-assigned males, whereas autistic cisgender birth-assigned females scored significantly lower on the task than neurotypical cisgender birth-assigned females. This indicates a weaker explicit gender self-concept among autistic cisgender participants than among neurotypical cisgender participants. Contrary to predictions, we found that autistic transgender birth-assigned males scored significantly higher on the explicit task than neurotypical transgender birth-assigned males. This shows that autistic transgender birth-assigned males showed a stronger explicit *female* self-concept than neurotypical transgender birth-assigned males. Also unexpectedly, no difference in the strength of the explicit gender self-concept was observed between autistic transgender birth-assigned females and neurotypical transgender birth-assigned females. Thus, autistic and neurotypical transgender birth-assigned females displayed an explicit *male* gender self-concept that was in keeping with their experienced/reported gender rather than birth-assigned gender, to the same degree.

Figure 8

Performance on the Explicit Measure of Gender Self-Concept as a Function of Birth-Assigned Sex and Diagnostic Category Within Cisgender and Transgender Participants



Note. NT = neurotypical; ASD = autism spectrum disorder. Birth-assigned males: NT cisgender $n = 55$; ASD cisgender $n = 50$; NT transgender $n = 37$; ASD transgender $n = 29$. Birth-assigned females: NT cisgender $n = 51$; ASD cisgender $n = 57$; NT transgender $n = 41$; ASD transgender $n = 27$. For ps $< .001$, $\eta_p^2 \geq .06$; for $p < .05$, $\eta_p^2 = .01$; for $p > .05$, $\eta_p^2 = .00$.

*$p < .05$ (one-tailed). *** $p < .001$ (one-tailed).

Performance on the Implicit Measure of Gender Self-Concept

A 2 (birth-assigned sex: male/female) × 2 (diagnostic category: neurotypical/autistic) × 2 (gender identity: cisgender/transgender) ANOVA was conducted on participant scores from the IAT. Significant main effects were detected for birth-assigned sex, $F(1,304) = 6.41$, $p = .012$, $\eta_p^2 = .01$, gender identity, $F(1,304) = 7.64$, $p = .006$, $\eta_p^2 = .03$, and diagnostic category, $F(1,304) = 13.83$, $p < .001$, $\eta_p^2 = .04$. The analysis also yielded a significant Birth-Assigned Sex × Gender Identity interaction, $F(1,304) = 302.40$, $p < .001$, $\eta_p^2 = .50$ and a significant Gender Identity × Diagnostic Category interaction, $F(1,304) = 4.98$, $p = .026$, $\eta_p^2 = .02$. Neither the Birth-Assigned Sex × Diagnostic Category interaction, $F(1,304) = 0.15$, $p = .701$, $\eta_p^2 = .00$ nor the three-way interaction, $F(1,304) = 2.80$, $p = .095$, $\eta_p^2 = .01$ were significant.

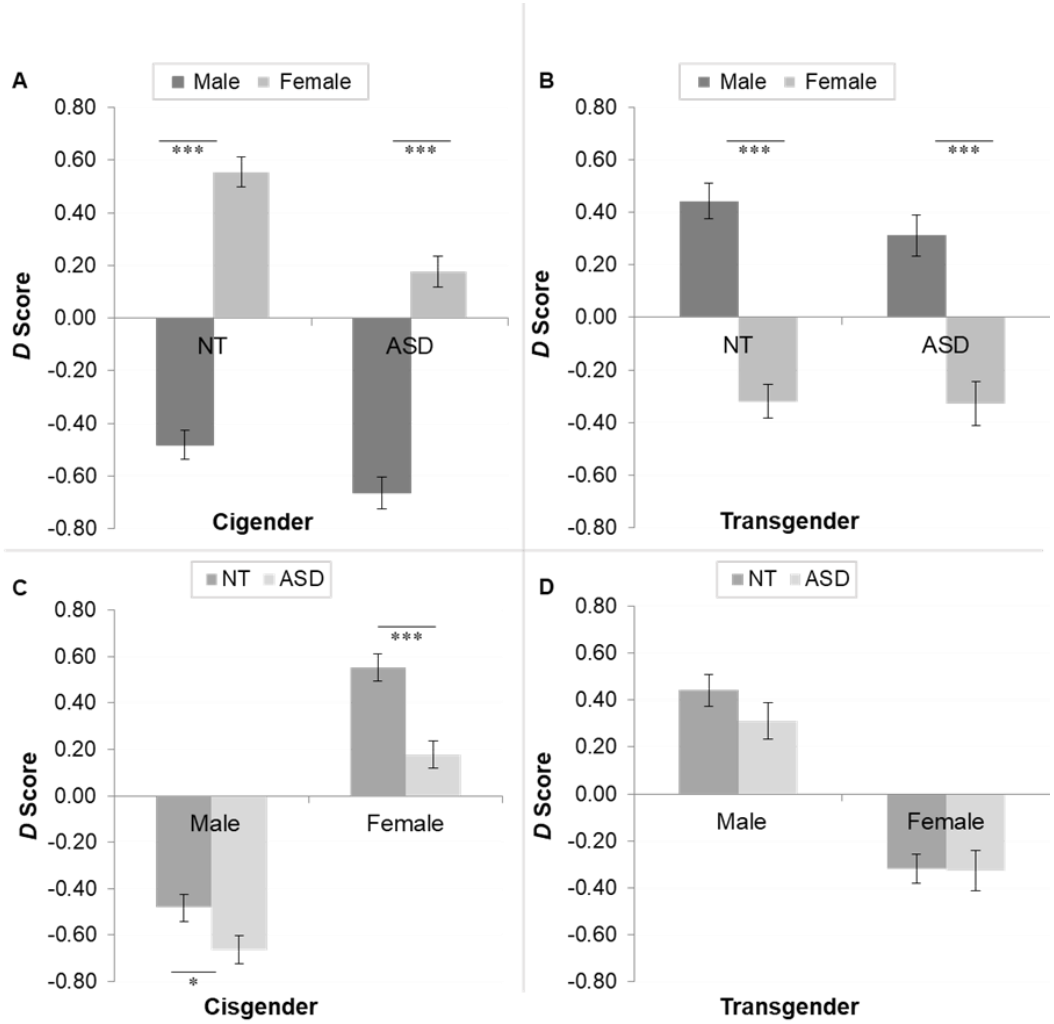
Breaking down the three-way interaction, a simple effects analysis of birth-assigned sex within gender identity and diagnostic category indicated that, in accordance with the preregistered Hypothesis A2, the IAT was sensitive to gender identity differences. Results of the analysis are illustrated in Figure 9A and B. Specifically, we found that among neurotypical cisgender and autistic cisgender individuals, birth-assigned females scored significantly *higher* than birth-assigned males (note: mean scores of birth-assigned females were significantly above zero (neurotypical $p < .001$ one-tailed; autistic $p = .003$ one-tailed), and mean scores of birth-assigned males were significantly below zero (all *p*s < .001 one-tailed)). This indicates that birth-assigned females who identify as females showed an *implicit female self-concept*, whereas birth-assigned males who identify as males showed an *implicit male self-concept*. The opposite pattern was observed among neurotypical transgender and autistic transgender individuals, with birth-assigned females scoring

significantly *lower* than birth-assigned males (note: mean scores of birth-assigned females were significantly below zero, and mean scores of birth-assigned males were significantly above zero, all *p*s < .001 one-tailed). This indicates that birth-assigned females who identify as males showed an *implicit male self-concept* in line with their experienced (rather than birth-assigned) gender, and birth-assigned males who identify as females showed an *implicit female self-concept* in line with their experienced (rather than birth-assigned) gender. These results were entirely expected.

Next, we conducted a simple effects analysis of diagnostic category within birth-assigned sex and gender identity. The results of the analysis are illustrated in Figure 9C and D. In line with the preregistered Hypothesis A3, we found that autistic cisgender birth-assigned females achieved a significantly lower *D* score on the IAT than neurotypical cisgender birth-assigned females, indicating a weaker implicit female self-concept. Contrary to predictions, we found that autistic cisgender birth-assigned males achieved a significantly lower *D* score on the IAT than neurotypical cisgender birth-assigned males, indicating a *stronger* implicit male self-concept. Also unexpectedly, a nonsignificant difference in the strength of implicit gender self-concept was found between autistic transgender and neurotypical transgender individuals. Both autistic and neurotypical transgender adults displayed an implicit gender self-concept that was in keeping with their experienced/reported gender rather than birth-assigned gender, to the same degree.

Figure 9

Performance on the Implicit Measure of Gender Self-Concept as a Function of Birth-Assigned Sex and Diagnostic Category Within Cisgender and Transgender Participants



Note. NT = neurotypical; ASD = autism spectrum disorder. Birth-assigned males: NT cisgender n = 50; ASD cisgender n = 43; NT transgender n = 34; ASD transgender n = 26. Birth-assigned females: NT cisgender n = 49; ASD cisgender n = 48; NT transgender n = 40; ASD transgender n = 22. For ps < .001, $\eta_p^2 \geq .07$; for $p < .05$, η_p^2 = .02; for ps > .05, $\eta_p^2 \leq .01$.

*$p < .05$ (one-tailed). *** $p < .001$ (one-tailed).

Performance on RMIE and Self-Report Measures

Table 15 shows descriptive statistics for participant scores on GIDYQ-AA, RCGI, AQ, RMIE, and TAS-20. A series of ANOVAs were conducted on these scores (see Table 16).

Table 15

Participant Characteristics and Mean (Standard Deviation) Performance on RMIE and Self-Report Measures

Groups	Age	RMIE	GIDYQ	RCGI[a,c]	AQ	TAS
	M (*SD*)	*M* (*SD*)	*M* (*SD*)[a,b]	*M* (*SD*)	*M* (*SD*)	*M* (*SD*)
NT cis	37.10	26.09	4.80	3.94	19.98	45.82
	(12.89)	(4.65)	(0.20)	(0.52)	(7.42)	(11.80)
NT trans	26.51	26.59	2.16	2.63	24.55	53.45
	(9.02)	(3.77)	(0.32)	(0.63)	(9.24)	(12.61)
ASD cis	31.55	19.61	4.18	3.52	31.45	61.42
	(7.86)	(7.80)	(0.71)	(0.63)	(7.21)	(9.09)
ASD trans	24.73	24.66	2.23	2.58	35.11	63.61
	(6.85)	(4.70)	(0.45)	(0.64)	(6.70)	(12.56)

Note. NT = neurotypical; ASD = autism spectrum disorder; Cis = cisgender; Trans = transgender; RMIE = Reading the Mind in the Eyes test; GIDYQ-AA = Gender Identity/Gender Dysphoria Questionnaire for Adolescents and Adults; RCGI =Recalled Childhood Gender Identity/Gender Role Questionnaire; AQ = Autism-Spectrum Quotient; TAS-20 = Toronto Alexithymia Scale.

[a] One neurotypical cisgender birth-assigned male completed the female version of the GIDYQ-AA and RCGI, and one autistic cisgender birth-assigned female completed the male version of the GIDYQ-AA and RCGI. Hence, their data has not been included in the analysis. [b] Low scores = more gender dysphoric feelings. [c] Low scores = less recalled childhood gender-typed behaviour.

Table 16

2 (Birth-Assigned Sex: Male/Female) × 2 (Diagnostic Category: Neurotypical/Autistic) × 2 (Gender Identity: Cisgender/Transgender) ANOVA Results

Measure	Effect	F	p	η_p^2	Direction of the main effects
GIDYQ-AA	Sex	12.55	<.001	.04	Birth-assigned females < Birth-assigned males
	Gender identity	1947.98	<.001	.85	Transgender < Cisgender
	Diagnostic category	28.73	<.001	.08	Autistic < Neurotypical
	Gender Identity × Diagnostic Category	41.11	<.001	.11	
	Sex × Gender Identity	3.35	.068	.01	
	Sex × Diagnostic Category	0.78	.378	.00	
	Sex × Gender Identity × Diagnostic Category	0.10	.756	.00	
RCGI	Sex	92.67	<.001	.22	Birth-assigned females < Birth-assigned males
	Gender identity	361.98	<.001	.52	Transgender < Cisgender
	Diagnostic category	16.63	<.001	.05	Autistic < Neurotypical
	Gender Identity × Diagnostic Category	7.37	.007	.02	
	Sex × Gender Identity	1.10	.295	.00	
	Sex × Diagnostic Category	0.83	.364	.00	
	Sex × Gender Identity × Diagnostic Category	0.99	.322	.00	
AQ	Sex	3.32	.069	.01	Birth-assigned females = Birth-assigned males
	Gender identity	22.79	<.001	.06	Transgender > Cisgender
	Diagnostic category	167.92	<.001	.33	Autistic > Neurotypical
	Gender Identity × Diagnostic Category	0.21	.645	.00	
	Sex × Gender Identity	2.67	.103	.01	
	Sex × Diagnostic Category	0.26	.608	.00	
	Sex × Gender Identity × Diagnostic Category	0.34	.563	.00	
RMIE	Sex	7.50	.006	.02	Birth-assigned females < Birth-assigned males

Measure	Effect	F	p	η_p^2	Direction of the main effects
	Gender identity	19.02	<.001	.05	Transgender > Cisgender
	Diagnostic category	45.40	<.001	.12	Autistic < Neurotypical
	Gender Identity × Diagnostic Category	12.63	<.001	.04	
	Sex × Gender Identity	2.33	.128	.01	
	Sex × Diagnostic Category	7.34	.007	.02	
	Sex × Gender Identity × Diagnostic Category	1.59	.209	.01	
TAS-20 [b]	Sex	0.20	.658	.00	Birth-assigned females = Birth-assigned males
	Gender identity	15.12	<.001	.04	Transgender > Cisgender
	Diagnostic category	107.23	<.001	.24	Autistic > Neurotypical
	Gender Identity × Diagnostic Category	4.84	.028	.01	
	Sex × Gender Identity	6.26	.013	.02	
	Sex × Diagnostic Category	0.18	.671	.00	
	Sex × Gender Identity × Diagnostic Category	0.63	.429	.00	

Note. Sex = Sex-assigned at birth.

Current Gender Dysphoric Feelings

A 2 (birth-assigned sex: male/female) × 2 (diagnostic category: neurotypical/autistic) × 2 (gender identity: cisgender/transgender) ANOVA was conducted on GIDYQ-AA scores. Significant main effects were detected for birth-assigned sex, such that birth-assigned females (marginal M = 3.25, SE = 0.04) reported significantly more current gender dysphoric feelings than birth-assigned males (marginal M = 3.43, SE = 0.04), gender identity, such that transgender individuals (marginal M = 2.20, SE = 0.04) reported significantly more current gender dysphoric feelings than cisgender people (marginal M = 4.49, SE = 0.03), and diagnostic category, such that autistic people (marginal M = 3.21, SE = 0.04) reported significantly more current gender dysphoric feelings than neurotypical individuals (marginal M = 3.48, SE = 0.04). The analysis also revealed a significant Gender Identity × Diagnostic Category interaction. The Birth-Assigned Sex × Gender Identity interaction was nonsignificant, as were the Birth-Assigned Sex × Diagnostic Category interaction, and the three-way interaction.

To test Hypothesis B1, the significant Gender Identity × Diagnostic Category interaction was treated as 4-level variable, and a series of planned t-tests was conducted. Results of the analyses are presented in Table 17. As predicted, autistic cisgender people reported significantly more current gender dysphoric feelings than neurotypical cisgender people, but significantly less than both neurotypical transgender and autistic transgender participants. Contrary to predictions, there was no significant difference in GIDYQ-AA score between neurotypical transgender and autistic transgender individuals (hence, autistic transgender = neurotypical transgender > autistic cisgender > neurotypical cisgender).

Table 17

Planned and Post-Hoc Comparisons Between Groups

Measure	*t*-tests	Cohen's *d*	95% CI
GIDYQ-AA			
	NT cis > NT trans ***	10.18	[9.09, 11.27]
	NT cis > ASD cis ***	1.18	[0.88, 1.47]
	NT cis > ASD trans ***	8.37	[7.39, 9.34]
	NT trans < ASD cis ***	-3.47	[-3.93, -3.01]
	NT trans = ASD trans	-0.18	[-0.52, 0.17]
	ASD cis > ASD trans ***	3.08	[2.61, 3.54]
RCGI			
	NT cis > NT trans ***	2.30	[1.92, 2.67]
	NT cis > ASD cis ***	0.73	[0.45, 1.01]
	NT cis > ASD trans ***	2.42	[2.00, 2.83]
	NT trans < ASD cis ***	-1.41	[-1.74, -1.08]
	NT trans = ASD trans	0.08	[-0.26, 0.43]
	ASD cis > ASD trans ***	1.49	[1.13, 1.85]
AQ			
	NT cis < NT trans ***	-0.56	[-0.85, -0.26]
	NT cis < ASD cis ***	-1.57	[-1.87, -1.26]
	NT cis < ASD trans ***	-2.11	[-2.50, -1.71]
	NT trans < ASD cis ***	-0.85	[-1.15, -0.54]
	NT trans < ASD trans ***	-1.28	[-1.65, -0.90]
	ASD cis < ASD trans **	-0.52	[-0.85, -0.19]
RMIE			
	NT cis = NT trans	-0.12	[-0.41, 0.18]
	NT cis > ASD cis ***	1.01	[0.72, 1.29]
	NT cis > ASD trans * [a]	0.31	[-0.02, 0.63]
	NT trans > ASD cis ***	1.09	[0.77, 1.40]
	NT trans > ASD trans **	0.46	[0.11, 0.81]
	ASD cis < ASD trans ***	-0.73	[-1.06, -0.40]
TAS-20 [b]			
	NT cis < NT trans ***	-0.63	[-0.93, -0.33]
	NT cis < ASD cis ***	-1.48	[-1.78, -1.18]
	NT cis < ASD trans ***	-1.47	[-1.83, -1.11]
	NT trans < ASD cis ***	-0.74	[-1.05, -0.44]
	NT trans < ASD trans ***	-0.81	[-1.16, -0.45]
	ASD cis = ASD trans	-0.21	[-0.53, 0.11]

Note. ASD = autism spectrum disorder; NT = neurotypical; Cis = cisgender; Trans = transgender; 95% CI = 95% Confidence Intervals.

[a] When groups were matched for age, $p < .01$ (one-tailed) and $d = 0.54$. [b] The analysis of TAS-20 was exploratory, therefore symbols for statistical significance reflect results from two-tailed tests. Tukey HSD correction was applied.

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

Recalled Gender-Typed Behaviour from Childhood

A 2 (birth-assigned sex: male/female) × 2 (diagnostic category: neurotypical/autistic) × 2 (gender identity: cisgender/transgender) ANOVA was conducted on RCGI scores. Significant main effects were detected for birth-assigned sex, such that birth-assigned females (marginal $M = 2.89$, $SE = 0.04$) recalled significantly less gender-typed behaviour from childhood than birth-assigned males (marginal $M = 3.46$, $SE = 0.04$), gender identity, such that transgender participants (marginal $M = 2.61$, $SE = 0.05$) recalled significantly less gender-typed behaviour from childhood than cisgender participants (marginal $M = 3.73$, $SE = 0.04$), and diagnostic category, such that autistic participants (marginal $M = 3.05$, $SE = 0.04$) recalled significantly less gender-typed behaviour than neurotypical participants (marginal $M = 3.29$, $SE = 0.04$). The analysis also revealed a significant Gender Identity × Diagnostic Category interaction. The Birth-Assigned Sex × Gender Identity interaction was nonsignificant, as were the Birth-Assigned Sex × Diagnostic Category interaction and the three-way interaction.

To test Hypotheses B2, the significant Gender Identity × Diagnostic Category interaction was treated as 4-level variable, and a series of planned *t*-tests was conducted. Results of the analyses are presented in Table 17. As predicted, autistic cisgender people recalled significantly less gender-typed behaviour from childhood than neurotypical cisgender people, but significantly more than both neurotypical transgender and autistic transgender participants. Contrary to predictions, there was no significant difference in RCGI score between neurotypical transgender and autistic transgender individuals (hence, autistic transgender = neurotypical transgender > autistic cisgender > neurotypical cisgender).

ASD traits

A 2 (birth-assigned sex: male/female) × 2 (diagnostic category: NT/ASD) × 2 (gender identity: cisgender/transgender) ANOVA was conducted on AQ scores. Significant main effects were detected for gender identity, such that transgender participants (marginal M = 29.82, SE = 0.67) reported significantly more ASD traits than cisgender participants (marginal M = 25.74, SE = 0.53) and diagnostic category, such that autistic participants (marginal M = 33.32, SE = 0.63) reported significantly more ASD traits than neurotypical participants (marginal M = 22.24, SE = 0.57). The main effect of birth-assigned sex was nonsignificant, as were the Birth-Assigned Sex × Gender Identity interaction, the Birth-Assigned Sex × Diagnostic Category interaction, the Gender Identity × Diagnostic Category interaction, and the three-way interaction.

To examine Hypothesis C1, a series of planned *t*-tests was conducted. Results of the analyses are presented in Table 17. As predicted, neurotypical transgender participants reported significantly more ASD traits than neurotypical cisgender people, but significantly fewer than autistic cisgender individuals. We also found, that autistic transgender individuals reported significantly more ASD traits than autistic cisgender people (hence, autistic transgender > autistic cisgender > neurotypical transgender > neurotypical cisgender).

Mentalising Ability

A 2 (birth-assigned sex: male/female) × 2 (diagnostic category: neurotypical/autistic) × 2 (gender identity: cisgender/transgender) ANOVA was conducted on RMIE scores. Significant main effects were detected for birth-assigned sex, such that birth-assigned males (marginal M = 25.11, SE = 0.44) performed significantly better on the task than birth-assigned females (marginal M = 23.43, SE = 0.43), gender identity, such that transgender participants (marginal M = 25.61, SE

= 0.48) performed significantly better on the task than cisgender participants (marginal M = 22.93, SE = 0.38), and diagnostic category, such that neurotypical participants (marginal M = 26.34, SE = 0.41) performed significantly better on the task than autistic participants (marginal M = 22.20, SE = 0.46).The analysis also revealed a significant Birth-Assigned Sex × Diagnostic Category interaction and a significant Gender Identity × Diagnostic Category interaction. Neither the Birth-Assigned Sex × Gender Identity interaction nor the three-way interaction were significant.

To test Hypothesis C2, the significant Gender Identity × Diagnostic Category interaction was treated as 4-level variable, and a series of planned *t*-tests was conducted. Results of the analyses are presented in Table 17. In contrast to predictions, no differences emerged in RMIE task performance between neurotypical transgender and neurotypical cisgender people, but as expected both groups performed significantly better than autistic cisgender people. In keeping with predictions, autistic transgender participants scored significantly lower on the task than both neurotypical cisgender and neurotypical transgender people, but contrary to our predictions, they scored significantly higher than autistic cisgender people (hence, autistic cisgender < autistic transgender < neurotypical transgender = neurotypical cisgender).

Alexithymia

A 2 (birth-assigned sex: male/female) × 2 (diagnostic category: neurotypical/autistic) × 2 (gender identity: cisgender/transgender) ANOVA was conducted on TAS-20 scores. Significant main effects were detected for gender identity, such that transgender participants (marginal M = 58.55, SE = 0.99) reported significantly higher levels of alexithymia than cisgender participants (marginal M =

53.66, *SE* = 0.78) and diagnostic category, such that autistic participants (marginal *M* = 62.62, *SE* = 0.93) reported significantly higher levels of alexithymia than neurotypical participants (marginal *M* = 49.59, *SE* = 0.84). The analysis also revealed a significant Birth-Assigned Sex × Gender Identity interaction and a significant Gender Identity × Diagnostic Category interaction. The main effect of Birth-Assigned Sex was nonsignificant, as were the Birth-Assigned Sex × Diagnostic Category interaction, and the three-way interaction.

To examine between-group differences, the significant Gender Identity × Diagnostic Category interaction was treated as 4-level variable, and a series of post-hoc *t*-tests was conducted. As shown in Table 17, neurotypical cisgender people reported significantly lower levels of alexithymia than neurotypical transgender, autistic cisgender, and autistic transgender people. In turn, neurotypical transgender reported significantly lower levels of alexithymia than autistic cisgender or transgender people, with no differences between the latter two groups (hence, neurotypical cisgender < neurotypical transgender < autistic transgender = autistic cisgender).

Association Analyses Between ASD Traits, Gender Dysphoric Feelings, and Mentalising

In keeping with the preregistration, we conducted a series of correlation analyses examining the relations between AQ, GIDYQ-AA, and RMIE within each group. As predicted, among neurotypical cisgender individuals, AQ score was negatively and significantly correlated with GIDYQ-AA score, $r = -.36$, $p < .001$ (one-tailed). Results indicate that the more ASD traits a person self-reported, the more their current gender dysphoric feelings. However, TAS score was positively and significantly correlated with AQ score, $r = .42$, $p < .001$ and negatively and

significantly correlated with GIDYQ-AA score, $r = .26$, $p = .008$. To investigate the extent to which the variance shared between ASD traits and current gender dysphoric feelings was due to alexithymia, a series of partial correlations was conducted. While, the relation between AQ score and GIDYQ-AA score, *controlling for TAS score*, remained significant, $r = -.29$, $p = .003$, the relation between TAS score and GIDYQ-AA score, *controlling for AQ score*, lost its significance, $r = -.13$, $p = .207$. Results highlight that the variance shared between AQ and GIDYQ-AA taps ASD characteristics rather than alexithymia.

As predicted, AQ score was also negatively and significantly correlated with GIDYQ-AA score, $r = -.43$, $p = .001$ (one-tailed) among autistic transgender people. Results indicate that the more ASD traits an autistic transgender person self-reported, the more their current gender dysphoric feelings. In contrast to predictions, however, AQ score was positively and significantly correlated with GIDYQ-AA score among autistic cisgender people, $r = .40$, $p < .001$ (one-tailed), indicating that the fewer ASD traits an autistic cisgender person self-reported the higher the level of current gender dysphoric feelings they reported. Also in contrast to predictions, the relation between AQ score and GIDYQ-AA score among neurotypical transgender individuals was nonsignificant, $r = -.12$, $p = .149$ (one-tailed).

Next, we examined the relation between RMIE score and GIDYQ-AA score. As predicted, performance on RMIE task was found to be positively and significantly associated with GIDYQ-AA score among autistic cisgender individuals, $r = .51$, $p < .001$ (one-tailed), suggesting that the better their mentalising ability, the fewer their current gender dysphoric feelings. Contrary to predictions, the relation between RMIE score and GIDYQ-AA score was nonsignificant among neurotypical cisgender

people, $r = .03$, $p = .377$ (one-tailed), neurotypical transgender people, $r = .11$, $p = .175$ (one-tailed) and autistic transgender people, $r = -.01$, $p = .468$ (one-tailed).

Discussion

Gender-Related Cognition in ASD

The first overarching aim of the current study was to examine whether ASD affects gender-related cognition. To do so, we first investigated the relation between ASD traits, on the one hand, and the strength of explicit and implicit gender self-concept, on the other hand, among neurotypical cisgender people. This approach followed was taken from our previous studies (see Chapter 5). In keeping with predictions, AQ score was negatively and significantly associated with the strength of the explicit gender self-concept. Results indicate that the more ASD traits a neurotypical cisgender person self-reported, the less they explicitly identified with gender groups. This finding adds to the evidence provided in Chapter 5 that cisgender people with high ASD traits display a weaker inclination to identify explicitly with personality traits that stereotypically characterise either females or males. In the current study, we also found that the more ASD traits neurotypical cisgender birth-assigned males self-reported, the weaker their explicit identification with male gender groups, and the more ASD traits neurotypical cisgender birth-assigned females self-reported, the weaker their explicit identification with female gender groups. These results were entirely expected.

Contrary to predictions, the number of self-reported ASD traits was not significantly associated with the strength of the implicit gender self-concept. Results did not change substantively when this association was examined among neurotypical cisgender birth-assigned males and among neurotypical cisgender birth-assigned females separately. These findings are inconsistent with those of

Experiment 1, presented in Chapter 5 (i.e., Kallitsounaki & Williams, 2020b). Contrary to the results of the current study, in our previous study we found the higher the ASD traits among cisgender people the weaker the automatic identification of *self* with either masculine or feminine personality attributes. One possible explanation for why the current study failed to replicate the results of the original study is that in this study we used an IAT that taps implicit self-identification with gender *groups*, whereas in the original study we used an IAT that taps implicit self-identification with gender-stereotypical personality *traits*. Although, both approaches have been traditionally used in gender identity research, they are not equivalent, as both have derived from different theoretical and research backgrounds (Wood & Eagly, 2015). It could be the case that neurotypical individuals with high ASD traits identify implicitly with gender stereotypical traits to a lesser degree than neurotypical individuals with low ASD traits, but nonetheless, show no difference in implicit identification with gender groups. We should not overlook, however, that we failed to reproduce the original findings in the replication study presented in Chapter 5. Although this does not exclude the possibility that the effect exists in the population, we cannot make firm conclusions about the robustness of this effect; in short, the relation between ASD traits and the strength of the implicit gender self-concept may well not be a reliable one.

Next, we aimed to extend our previous findings further by investigating, for the first time, gender-related cognition in autistic cisgender and transgender people, using an explicit and implicit measure of gender self-concept. As expected, both measures tapped the experienced/reported gender of participants. Specifically, among cisgender individuals, either neurotypical or autistic, birth-assigned females showed an explicit and implicit *female* self-concept, and birth-assigned males

showed an explicit and implicit *male* self-concept. Whereas, among transgender individuals, either neurotypical or autistic, birth-assigned females who identify as males showed an explicit and implicit *male* self-concept, and birth-assigned males who identify as females showed an explicit and implicit *female* self-concept.

To our knowledge, this is the first study to investigate implicit gender self-concept in transgender adults and found that neurotypical and autistic transgender adults perceive themselves explicitly *and* implicitly in terms of their experienced/reported gender. Olson et al. (2015) rightly, in our view, highlighted that "the IAT should not be seen as a lie detector test" (p. 468). Based on the results from the explicit and implicit task, however, it could be argued that the current study might provide counter evidence to the hypothesis that symptoms of gender dysphoria in ASD (e.g., cross-dressing) reflect more an obsession that arises from autistic people's inherent predisposition toward unusual interests and preoccupations than "genuine" gender identity difficulties (Parkinson, 2014; Tateno et al., 2008; Williams, 1996). Furthermore, results indicate that autistic people (cisgender and transgender) are able to formulate a gender self-concept, regardless of whether it matches their birth-assigned sex. It is unclear, however, whether autistic and neurotypical people identify with their experienced/reported gender to the same degree. To answer to this question, we first compared the strength of explicit and implicit gender self-concept between autistic and neurotypical cisgender people.

As predicted, autistic cisgender individuals (birth-assigned males and birth-assigned females) showed a weaker explicit identification with the gender groups associated with their birth-assigned sex than did neurotypical cisgender people. Based on our findings from the general population (current study and studies included in Chapter 5), this was expected. Results are also in line with previous

findings of lower explicit gender identification in autistic people (Cooper et al., 2018). Furthermore, on the basis of previous findings from the general population (Chapter 5; Experiment 1), we expected autistic cisgender birth-assigned females to show a significantly lower score in the implicit measure of gender self-concept than neurotypical cisgender birth-assigned females. Indeed, our hypothesis was confirmed. This important finding is the first of its kind, to our knowledge, and suggests that autistic cisgender birth-assigned females have a *weaker* inclination to incorporate into their self-concept a collective gender concept that matches their birth-assigned sex. We also predicted that autistic cisgender birth-assigned males would show a significantly higher score on the implicit measure of gender self-concept than neurotypical cisgender birth-assigned males. Unexpectedly, however, autistic cisgender birth-assigned males displayed a significantly lower score on the implicit measure of gender self-concept than neurotypical cisgender birth-assigned males, indicating a stronger inclination to incorporate into their self-concept a collective gender concept that matches their birth-assigned sex. Thus, there is an important effect of sex here.

Autistic cisgender birth-assigned females show weaker implicit *and* explicit identification with female gender groups than neurotypical cisgender birth-assigned females. In contrast, autistic cisgender birth-assigned males show weaker explicit, yet stronger implicit identification with male gender groups than neurotypical cisgender birth-assigned males. The observed mismatch between their explicit and implicit experience of male self-concept among autistic cisgender birth-assigned *males* might reflect a difficulty with self-awareness. The idea that self-awareness difficulties are involved in ASD has been expressed by many (e.g., Frith & Happé,

1999; Hobson, 1990; Williams, 2010), but to our knowledge, sex differences in self-awareness have not been examined in the autistic population.

Next, we compared the strength of explicit and implicit gender self-concept between autistic transgender and neurotypical transgender individuals. Contrary to predictions, autistic transgender birth-assigned females and neurotypical transgender birth-assigned females identified explicitly with the gender groups associated with their experienced/reported gender to the same degree, and autistic transgender birth-assigned males identified explicitly with the gender groups of their experienced/reported gender more strongly than neurotypical transgender birth-assigned males. Also unexpectedly, the performance of autistic transgender individuals (birth-assigned males and birth-assigned females) on the implicit measure of gender self-concept did not differ significantly from the performance of neurotypical transgender individuals. Both groups identified implicitly with their experienced/reported gender, rather than their birth-assigned gender, to the same degree.

In sum, we found that autistic cisgender people were able to identify *explicitly* with the gender groups associated with their birth-assigned sex, yet they identified less strongly than did neurotypical cisgender people. They also identified *implicitly* with the gender groups associated with their birth-assigned sex, but the strength of the identification was weaker only among birth-assigned females. In contrast, autistic transgender and neurotypical transgender people identified *explicitly* and *implicitly* with the gender groups of their experienced/reported gender to the same degree, at least. On this basis, it could be argued that these results provide preliminary evidence of a selective influence of ASD in the consolidation of a collective gender self-concept that matches people's birth-assigned sex. To put it

simply, ASD seems to hinder the explicit (and implicit among birth-assigned females) identification of people only with the gender groups associated with their birth-assigned sex. The explicit and implicit identification of autistic transgender people with the gender groups of their experienced/reported gender seems to be unaffected by ASD. To get a better understanding of the findings from the autistic adult population (cisgender and transgender), we believe it is essential to know how gender self-concept develops in autistic children. Given that research on this topic is almost nonexistent, future research will be needed to elucidate whether the development of gender self-concept follows the same cognitive and developmental trajectories in autistic and neurotypical children (van Schalkwyk et al., 2015).

Gender Dysphoric Feelings and Recalled Gender-Typed Behaviour in ASD

The second overarching aim of this study was to investigate gender dysphoric feelings and recalled childhood gender-typed behaviour in autistic people. Specifically, we first examined whether *autistic transgender* people report increased current gender dysphoric feelings and recall diminished gender-typed behaviour from childhood. To our knowledge, this is the first study that attempted to answer this question in the adult population. We found that, in accordance to the mismatch autistic transgender people expressed between their birth-assigned sex and their experienced/reported gender, they reported *clinically* significant levels of gender dysphoria and they recalled limited gender-typed behaviour from childhood. This indicates that autistic transgender people and neurotypical transgender individuals feel an extreme distress of their body, anatomy, and function to the same degree and that gender dysphoric/incongruent feelings have an early onset in both groups. Results are in keeping with Strang et al.'s (2021) findings. Although neurotypical transgender adolescents reported significantly more gender dysphoria than autistic

transgender adolescents in their study, both groups reported clinically significant levels of gender dysphoria. Taken together, these findings provide further support to the clinical recommendation that adequate support and care should be provided in transgender people, regardless of whether they have a co-occurring diagnosis of ASD (e.g., Strang, Meagher, et al., 2018).

Next, we examined whether *autistic cisgender* people report increased current gender dysphoric feelings and recall diminished gender-typed behaviour from childhood. In keeping with George and Stokes' (2018b) findings, we found that autistic cisgender individuals reported significantly more current gender dysphoric feelings than neurotypical cisgender people, but significantly less than autistic and neurotypical transgender people. We also extended this finding further by showing, for the first time, that autistic cisgender people *recalled* less gender-typed behaviour in their childhood memories than neurotypical cisgender individuals, but more than autistic and neurotypical transgender people. This is in line with recent findings that autistic people report a more diverse range of gender identities and are more likely to be gender incongruent and to have or be planning a gender transition than neurotypical people, (Bejerot & Eriksson, 2014; Cooper et al., 2018; George & Stokes, 2018b). This also supports the hypothesis that there is link between ASD and gender incongruence/dysphoria (e.g., Strang, Janssen, et al., 2018). Nonetheless, we should stress that the mechanisms that could underpin that link are still unclear and further research is required. Epidemiological studies are also required to get an estimate of the size of this link. To date the only study of the prevalence of GD diagnosis in the autistic population has been conducted among children (Hisle-Gorman et al., 2019).

ASD Traits, Mentalising Ability, and Alexithymia in Transgender Individuals

The third overarching aim of this study was to examine whether transgender individuals have increased ASD traits, diminished mentalising ability, and high levels of alexithymia. As predicted, *neurotypical transgender* individuals reported significantly more ASD traits than neurotypical cisgender individuals, but significantly fewer than autistic people (either cisgender or transgender). This is important because to date, only a very small number of studies (i.e., Jones et al., 2012; Murphy et al., 2020; Warrier et al., 2020) have examined whether increased ASD are observed in samples of *neurotypical* gender dysphoric/incongruent adults. In this study, we also found that *autistic transgender* people reported significantly more ASD traits than autistic cisgender individuals. This is in keeping with previous research findings (Walsh et al., 2018; Warrier et al., 2020), but further research is required to understand this finding. If high scores on the AQ tap solely ASD characteristics in both autistic cisgender and autistic transgender groups, then these results indicate an increased severity of ASD in autistic transgender people. If this is true, autistic transgender people should also score higher on standardised diagnostic tools for ASD (e.g., Autism Diagnostic Observation Schedule; Lord et al., 2000) than autistic cisgender people. To our knowledge, this has not been examined in the literature.

We should highlight here that it remains debatable whether ASD traits observed among gender dysphoric/incongruent people tap "true" ASD characteristics (Fortunato et al., 2021; Turban, 2028; Turban & van Schalkwyk, 2018). It has been suggested that the psychological impact of stigma, marginalization, and rejection that transgender people frequently experience increase their liability to develop features that "mimic" ASD characteristics (Fortunato et al., 2021; Turban, 2028; Turban & van Schalkwyk, 2018). To investigate this hypothesis we examined whether the

behavioural features of ASD observed among transgender individuals (neurotypical and autistic) were accompanied with (traditionally-observed) difficulties at the cognitive level. If ASD traits among neurotypical and autistic transgender people reflected true ASD characteristics, then these groups should show the mentalising difficulty that characterises ASD.

Interestingly, *neurotypical* transgender participants did not show the predicted mentalising deficit. Instead, their performance on the mentalising task was equivalent to the performance of neurotypical cisgender individuals. This is out of keeping with Kung (2020) who found that transgender males had significantly poorer mentalising ability than cisgender birth-assigned females and transgender females poorer mentalising ability than cisgender birth-assigned males. Nonetheless, as discussed above, Kung (2020) did not control for the presence of autistic participants in the transgender sample. Furthermore, we found, for the first time, that autistic transgender people scored significantly lower on the RMIE than neurotypical cisgender and neurotypical transgender people, indicating a mentalising difficulty. Noteworthy, neurotypical transgender people performed significantly better on the task than autistic cisgender people.

Lastly, conducting an exploratory analysis, we found that neurotypical transgender individuals reported significantly more alexithymia than neurotypical cisgender people, but significantly less than autistic people (cisgender and transgender). Although alexithymia is not well examined in transgender individuals, results are in keeping with Kessler et al.'s (2006) findings. Interestingly, no differences in the levels of alexithymia were observed between autistic transgender and autistic cisgender people. Both groups reported *clinically significant alexithymia*. Based on their diagnosis, this was to be expected.

From a theoretical perspective, these results add to the discussion as to whether ASD traits in transgender people represent true ASD characteristics (Fortunato et al., 2021; Turban, 2028; Turban & van Schalkwyk, 2018). We found that neurotypical transgender people did show increased ASD traits, yet they did not show the predicted mentalising deficit. Whereas, autistic transgender people showed not only increased ASD traits, but also a mentalising difficulty. Based on these results, it could be argued that ASD traits in neurotypical transgender people do not reflect "true" ASD characteristics, whereas they do tap upon ASD among autistic transgender individuals. Furthermore, in previous studies (see Chapters 3 & 4) we found a significant relation between mentalising ability and gender dysphoric feelings among people from the general population. Yet, the findings presented in this chapter indicate that this relation does not hold among neurotypical transgender people and thus contradict the hypothesis that mentalising is the shared underlying mechanism that explains the high co-occurrence of ASD and gender dysphoria/incongruence (Glidden et al. 2016; Jacobs et al. 2014; Van Der Miesen et al. 2016). Based on our previous findings, however, we cannot exclude the possibility that mentalising might play a role in the development of gender dysphoric feelings in *autistic cisgender* people (see Chapters 3 & 4). Of course, further research and replication of the current findings is required before major conclusions can be made. To increase confidence in the veracity of the current findings, future research might usefully examine mentalising ability in transgender (autistic and neurotypical) children and well as in their parents and siblings, employing not only explicit, but also implicit measures of mentalising (e.g., Senju et al., 2009).

Links Between ASD Traits, Current Gender Dysphoric Feelings, and Mentalising

The last overarching aim of this study was to examine the relations between ASD traits, current gender dysphoric feelings, and mentalising not only among neurotypical cisgender people, but also among neurotypical transgender, autistic cisgender, and autistic transgender individuals, for the first time. A correlation analysis conducted among neurotypical cisgender individuals revealed that in line with predictions, AQ score was significantly associated with GIDYQ-AA score, indicating that the more ASD traits a neurotypical cisgender individual self-reported, the more their current gender dysphoric feelings tended to be. Results were in line with previous evidence, reflecting a very robust and reliable link between ASD traits and gender dysphoric feelings in the general population (George & Stokes, 2018b; Kallitsounaki & Williams, 2020a; Kallitsounaki et al., 2021). Importantly, this link emerged also among autistic transgender individuals. We found that the more ASD traits an autistic transgender individual self-reported, the more their current gender dysphoric feelings. Contrary to our predictions, AQ score was not significantly associated with GIDYQ-AA score among neurotypical transgender individuals. Also unexpectedly, the relation between AQ and GIDYQ-AA among autistic cisgender individuals was significant, yet negative instead of positive. That is to say, the more ASD traits an autistic cisgender individual self-reported, the fewer their current feelings of gender dysphoria tended to be. Results indicate that ASD traits in neurotypical transgender people are unrelated to their levels of gender dysphoric feelings, yet it is very difficult to interpret the relation we found between ASD traits and gender dysphoric feelings among autistic cisgender people.

Next, we examined the relation between RMIE and GIDYQ-AA. Given that mentalising has been found to be significantly associated with gender dysphoric feelings (better mentalising = fewer gender dysphoric feelings) among cisgender

individuals (see Chapters 3 & 4), we expected to replicate this finding in the current study. Contrary to our predictions, RMIE score did not correlate significantly with GIDYQ-AA score either among neurotypical cisgender, neurotypical transgender, or autistic transgender individuals. Interestingly, the expected link was found to be significant among autistic cisgender individuals, indicating that the better the mentalising ability of autistic people, the fewer their current gender dysphoric feelings. Results might suggest that mentalising plays some role in the development and expression of gender dysphoric feelings in autistic cisgender people, but given the analysis we conducted, causal claims cannot be made based on these findings.

Limitations and Directions for Future Research

Although the findings of this study enhance our understanding of the high-occurrence between ASD and gender dysphoria/incongruence by elucidating some core aspects of this phenomenon, we should note a potential limitation in this study. In order autistic participants to take part in the current study had to report possession of a formal diagnosis of ASD. We cannot exclude the possibility, however, that self-diagnosed individuals also took part in the study. Future online studies should account for the issue of sample identifiability (i.e., internally valid results, but not necessarily representative; Rubenstein & Furnie, 2020) by asking participants to provide a copy of the diagnostic letter (see Warrier et al., 2020). Despite this potential limitation, it is important to mention that autistic participants in this study showed clinical levels of ASD and alexithymia, as well as poor mentalising ability. The presentation of ASD diagnosis is the autistic group of the current study was in keeping with that found in other studies where not only evidence of a formal diagnosis of ASD has been provided by autistic participants, but also an independent validation of the diagnosis has been made (e.g., Nicholson et al., 2018).

In sum, the main findings of this study indicate that (a) ASD appears to affect the consolidation of a strong explicit (and implicit in birth-assigned females) gender self-concept among autistic cisgender people only, (b) autistic cisgender people have increased current gender dysphoric feelings and recall limited gender-typed behaviour from childhood, and (c) neurotypical transgender people show only the behavioural features of ASD, whereas autistic transgender people show features of ASD at the behavioural and cognitive level. The current study enhances our understanding of a phenomenon that despite its important implications for both clinical practice and theory development remains to a large extent unexplained and insufficiently researched.

Chapter 7

General Discussion

Williams et al. (1996) reported the case of two autistic boys who presented an intense preoccupation with stereotypically feminine activities and roles. The publication of this case-study signified the emergence of a new research topic. That is the co-occurrence of ASD and gender dysphoria/incongruence. As the clinician and researcher Aron Janssen highlighted "like many research ideas, this is one that was born from clinical experience" (Seaman, 2016, p. 2). Clinicians specialised on gender-related issues and/or ASD witness the high co-occurrence of these conditions and its effect on clinical practice and policy-making almost on a daily basis (Strang, Janssen, et al., 2018). Yet, our understanding of this phenomenon remains scant. In the current thesis, we identified and examined three core aspects of the co-occurrence of ASD and gender dysphoria/incongruence, aiming to acquire a better understanding of it.

Summary of Findings and Discussion

Is There a Link Between ASD and Gender Dysphoria/Incongruence?

Chapter 2 provided the most recent systematic review of the literature pertaining the suggested link between ASD and gender dysphoria/incongruence. It also provided the *first* meta-analyses of studies of ASD diagnoses and ASD traits in gender dysphoric/incongruent people. Although the research investigating the link between ASD and gender dysphoria/incongruence in the general population is limited, the systematic review found consistent evidence of a positive relation between ASD traits and gender dysphoria/variance in general population samples. Specifically, high ASD traits in children or their mothers predict gender nonconformity in children (Shumer et al., 2015), and the more the ASD traits in

neurotypical children, as reported by their parents, the more parent-reported cross-gender behaviour in them (Nabbijohn et al., 2019). Noteworthy, these findings converge with those of studies conducted in the *adult* general population (George & Stokes, 2018b; Kallitsounaki & Williams, 2020a; Kallitsounaki et al., 2020).

Studies focusing on autistic samples are also limited, yet our systematic review indicated an increased likelihood of autistic people to be gender dysphoric/incongruent. Hisle-Gorman et al. (2019) found that the prevalence of a diagnosis of gender dysphoria in autistic children was 7%, as compared to 1% in neurotypical children. Research has also shown that autistic children, as well as adults, are more likely to desire to be the other binary gender (Janssen et al., 2016; May et al., 2017; Strang et al., 2014; van der Miesen, Hurley, et al., 2018) and that autistic adults are more likely to be gender incongruent than neurotypical people (Bejerot, & Eriksson, 2014; Cooper et al., 2018; George & Stokes, 2018b). Specifically, 15% of autistic people in Walsh et al.'s (2018) study reported being gender incongruent. In contrast to the limited number of studies examining the link between ASD and gender dysphoria/incongruence in the autistic population, the study of this topic in gender dysphoric/incongruent people is more extensive.

The literature review found that the positive rates for suspected ASD range from 1.2%–68% in gender dysphoric/incongruent samples (e.g., Akgül et al., 2018; Vermaat et al., 2018). Yet, the heterogeneity in ASD screening questionnaires, as well as the cut-off points used across different studies to identify gender dysphoric/incongruent people who might warrant a clinical assessment for ASD, make these findings difficult to interpret. The systematic review also found that studies focusing on gender dysphoric/incongruent samples suggest a prevalence of ASD diagnoses higher than expected by chance (e.g., Leef et al., 2019; Murphy et

al., 2020; Shumer et al., 2016; Warrier et al., 2018). Specifically, the meta-analysis indicated that the prevalence of ASD diagnoses in gender dysphoric/incongruent people was 11%. That is 11 times higher than the prevalence of ASD in the general population (approximately 1%; e.g., Baird et al., 2006). The systematic review also indicated evidence of increased ASD traits in gender dysphoric/incongruent children (Akgül et al., 2018; Skagerberg et al., 2015; van der Miesen, de Vries, et al., 2018). Yet, conflicting evidence emerged about the prevalence of ASD traits in gender dysphoric/incongruent adults (Heylens et al., 2018; Jones et al., 2012; Vermaat et al., 2018; Warrier et al., 2020). The meta-analysis of studies of ASD traits we conducted elucidated this issue, indicating a moderate (i.e., Hedges'g = 0.67) and a significant difference in ASD traits between gender dysphoric/incongruent people and nonclinical/population-based control individuals.

One of the purposes of Chapter 3 was to examine the relation between ASD traits, and current gender dysphoric feelings and recalled gender-typed behaviour in the adult general population. We found that the more ASD traits a person self-reported, the more gender dysphoric feelings they had in the last 12 months and the less gender-typed behaviour they recalled from childhood. Results were successfully reproduced in a replication study we conducted, the results of which are presented in Chapter 4[15]. It is important to note that no study had examined the relation between ASD traits and *recalled* gender-typed behaviour before, whereas the link between ASD traits and *current* gender dysphoric feelings had been examined only by George and Stokes (2018b). An independent replication of their preliminary findings was

[15] Note that the case-control study presented in Chapter 6 also found a significant relation between ASD traits and gender dysphoric feelings among neurotypical cisgender individuals (i.e., r = -.36). Hence, there is convergence evidence from three independent samples.

important to increase confidence in the reliability of the original findings. George and Stokes (2018b) found a positive relation (r = .34) between ASD traits and current gender dysphoric feelings among people who did not have clinically significant levels of ASD traits. Importantly, in both studies we conducted the relation between ASD traits and current gender dysphoric feelings was almost identical (i.e., r = .32 in Kallitsounaki & Williams, 2020a and r = .31 in Kallitsounaki et al., 2021) to the relation reported by George and Stokes (2018b). Taken together, results indicate that the link between ASD traits, on the one hand, and gender dysphoric feeling and recalled gender-typed behaviour, on the other hand is robust in the general population. The two hypotheses that stem from this link were examined and discussed in Chapter 6.

One of the purposes of the case-control study included in Chapter 6 was to examine whether autistic people have increased current gender dysphoric feelings and limited recalled gender-typed behaviour from childhood. George and Stokes (2018b) had examined this hypothesis before and found that autistic people reported significantly more current gender dysphoric feelings than neurotypical participants. However, a high proportion of the autism group in George and Stokes did not identify as cisgender in the first place and so conclusions as to whether autistic *cisgender* people have increased gender dysphoric feelings cannot be drawn from this study. To address this issue, we examined recalled gender-typed behaviour and current gender dysphoric feelings in autistic cisgender and autistic transgender people separately. We found that autistic *cisgender* people recalled significantly less gender-typed behaviour from their childhood memories and reported significantly more, yet not clinically significant, current gender dysphoric feelings than neurotypical cisgender people. Compared to autistic transgender people, autistic

cisgender individuals recalled significantly more gender-typed behaviour from childhood and reported significantly fewer current gender dysphoric feelings. Autistic transgender and neurotypical transgender people recalled similar levels of gender-typed behaviour from childhood and clinically significant levels of current gender dysphoria.

Another purpose of the case-control study included in Chapter 6 was to investigate whether transgender people have elevated levels of ASD traits and poor mentalising ability. In line with previous evidence (e.g., Warrier et al., 2020), we found that *neurotypical* transgender people reported significantly more ASD traits than neurotypical cisgender people, yet significantly fewer traits than both autistic cisgender and autistic transgender people. It is important to note that although neurotypical transgender people showed the behavioural features of ASD in our study, they did not show the social-cognitive deficits that arguably underlie ASD. Specifically, the performance of this groups on the mentalising task was equivalent to the performance of neurotypical cisgender individuals. This counter-indicates the evidence provided by Kung (2020), who observed significantly poorer mentalising ability in his group of gender incongruent adults than in his group of neurotypical adults. However, Kung's gender incongruent group might contain a high proportion of people already diagnosed with ASD and, thus, diminished mentalising in that group may have been the result of elevated rates of ASD rather than incongruent gender identity. Therefore, the study reported in Chapter 6 was the first to show that *neurotypical* transgender people's mentalising ability is intact. Furthermore, as expected based on their diagnosis, autistic transgender people reported significantly more ASD traits and performed significantly poorer in the mentalising task than neurotypical people (cisgender and transgender). We should also note, however, that

although autistic transgender people performed significantly better on the mentalising task than autistic cisgender people, they reported significantly fewer ASD traits than this group.

Taken together, the findings of the systematic literature review, the meta-analyses, the studies conducted in the general population, and the case-control study indicate that the chances there is not a link between ASD and gender dysphoria/incongruence are negligible. This is in keeping with the experiences and views expressed by many clinicians and researchers (e.g., Strang, Janssen, et al., 2018; van der Miesen, Cohen-Kettenis, et al., 2018). However, there is one more question that needs to be answered. Is the link between ASD and gender dysphoria/incongruence merely a methodological artefact?

The opponents of the hypothesis of a true link between ASD and gender dysphoria/incongruence have argued that the over-representation of ASD in the gender dysphoric/incongruent population is not well-supported by research evidence (Fortunato et al., 2021; Turban, 2018; Turban & van Schalkwyk, 2018). Specifically, they have posited that elevated scores of gender dysphoric/incongruent people on ASD screening tools are nonspecific to a diagnosis of ASD. Accordingly, gender dysphoric/incongruent people score high on these measures due to high rates of co-occurring internalising psychopathology and reflect social impairment that stems from reversible or modifiable processes such as minority stress, and peer and family rejection (Fortunato et al., 2021; Skagerberg et al., 2015; Turban, 2018; Turban & van Schalkwyk, 2018). To support this claim Fortunato et al. (2021) presented the case of a gender dysphoric birth-assigned female with a co-occurring diagnosis of ASD who did not meet the diagnostic criteria for ASD after four years of psychotherapy and a successful social transition from female to male. According to

Turban and van Schalkwyk, gender dysphoric/incongruent people might show the features of ASD at the behavioural level, but not the "social cognitive deficits that underlie ASD" (p. 9).

The current thesis partially supports this hypothesis by showing, for the first time, that *neurotypical* transgender people have increased ASD traits, yet not poor mentalising ability. In other words, neurotypical transgender people seem to show only the features of ASD at the behavioural level, as Turban and van Schalkwyk suggested (2018). Two recently published studies examined stability of ASD traits in transgender adolescents and adults after receiving medical treatment (i.e., puberty suppression treatment and cross sex hormone treatment). None of the two studies revealed a significant decrease in the number of reported ASD traits. Based on these findings, researchers argued that this provides counter evidence to the hypothesis that the overrepresentation of ASD traits in gender dysphoric/incongruent people is due adverse social experiences (Nobili et al., 2020; Russell et al., 2021). We have to acknowledge that this is a methodologically rigorous way to examine this hypothesis, yet two important limitations in these studies do not allow us to argue that their findings contradict our results.

First, in both studies the time span between the two measurements was relatively narrow (i.e., after one year of receiving medical treatment) for meaningful differences to be found. In previous studies that have assessed the stability of ASD-related features in different populations, though (i.e., congenitally blind children and Romanian orphans raised in institutions) the follow up assessment was conducted at least five years after the initial assessment (Hobson & Lee, 2010; Rutter et al., 2007). On this basis, it is questionable whether within only a year of receiving treatment a gender dysphoric/incongruent person would show a significant improvement in

social functioning, even though their anxiety levels may have dropped (Nobili et al., 2020). Second, it is unclear whether the positive effects of treatment (physical and psychological) are accompanied with fewer adverse social experiences. In future studies, this is an important factor that needs to be measured and controlled. If a gender dysphoric/incongruent people keep suffering at the social and interpersonal level (e.g., stigma, marginalisation, and rejection) despite receiving the appropriate treatment, reduction in ASD traits cannot be expected.

It is important to note that Fortunato et al. (2021) omitted to mention that the suggestion of a link between ASD and gender dysphoria/incongruence has not been based solely on the apparent overrepresentation of *ASD traits* in gender dysphoric/incongruent people. As highlighted in this thesis, this hypothesis is also supported by the increased prevalence of *ASD diagnoses* in this population. Of course, it could be argued that this is due to false positives, as in the case presented by Fortunato et al. (2021). Yet, the evidence presented in this thesis contradict this hypothesis. In the case-control study we conducted (see Chapter 6), we found that neither the ASD traits nor the alexithymia reported by neurotypical transgender people reached the threshold of clinical significance to warrant further assessment for ASD. Whereas, autistic transgender people reported clinically significant levels of ASD and alexithymia and showed poor mentalising ability. Thus, the presentation of the diagnosis of ASD in this group was *qualitatively* similar to how ASD was presented among autistic cisgender people in this thesis and in the autism literature in general (e.g., Nicholson et al., 2018). So, there is no evidence to indicate a misdiagnosis of ASD in autistic transgender people. It could be argued, however, that social impairment in gender dysphoric/incongruent people lead to fewer meaningful opportunities to learn about mental states. This could explain the relatively poor

performance on the mentalising task of autistic transgender people (Rutter et al., 2007). If this was true, however, then neurotypical transgender people should also show a mentalising deficit. The findings of our case-control study showed that this was not the case.

Overall, this thesis indicates that the link between ASD and gender dysphoria/incongruence exists and is real. We showed that there is a robust relation between ASD traits and gender dysphoric feelings in the general population, autistic cisgender people show increased gender dysphoric feelings, and there is a high prevalence of ASD diagnoses in gender dysphoric/incongruent people. Nonetheless, evidence of elevated levels of ASD traits in neurotypical transgender people should be interpreted with caution, as they might not tap core features of ASD. Given that the underpinnings of the link between ASD and gender dysphoria/incongruence remain unclear, the second overarching aim of this thesis was to examine the role of mentalising in this link.

What is the Role of Mentalising in the Link Between ASD and Gender Dysphoria/Incongruence?

This thesis provides the first systematic examination of the hypothesis that mentalising (i.e., the ability to impute mental states to oneself and others; Premack & Woodruff, 1978) is one the contributing factors of the high co-occurrence of ASD and gender dysphoria/incongruence (Glidden et al., 2016; Jacobs et al., 2014; Van Der Miesen et al., 2016). In Chapter 3, we demonstrated, for the first time, a strong and negative relation between mentalising and current gender dysphoric feelings among cisgender people from the general population. Results were successfully reproduced in the replication study included in Chapter 4, indicating a relatively reliable link between mentalising and gender dysphoric feelings in the general

population (poorer mentalising ability = more gender dysphoric feelings). [16] In Chapter 3, we also demonstrated that mentalising *moderates* the relation between ASD traits and gender dysphoric feelings so that high ASD traits were related with increased gender dysphoric feelings when mentalising ability was poor, but unrelated to ASD traits when mentalising ability was exceptional. In the latter case, people from the general population did not report any gender dysphoric feelings. Interestingly, in Chapter 4 we demonstrated that mentalising *mediates* the relation between ASD traits and gender dysphoric feelings.

At first reading, the findings presented in Chapters 3 and 4 might seem contradictory, yet research has shown that a variable can serve both as a moderator and a mediator (e.g., Beauchaine et al., 2005; Grech et al., 2016). A moderation provides insight on the group that it is mostly affected from a relation, whereas a mediation provides information on the mechanisms under the influence of which a link is created (Judd et al., 2001). On this basis, the findings presented in Chapter 3 indicate that exceptional mentalising ability plays a protective role in the relation between ASD traits and gender dysphoria, whereas poor mentalising ability places an individual with high ASD traits at greater risk of developing some gender dysphoric feelings, and the findings presented in Chapter 4 indicate that high ASD traits predict poor mentalising ability that leads to increased feelings of gender dysphoria. Taken together, these findings provide some preliminary evidence that mentalising is one of the mechanisms that could explain the high co-occurrence of ASD and gender dysphoria/incongruence. To further understand, however, the role of mentalising in

[16] Note that the case-control study presented in Chapter 6 did not find a significant relation between gender dysphoric feelings and mentalising among neurotypical cisgender people.

this link, it was important to investigate these effects in autistic and neurotypical cisgender and transgender people.

As already mentioned, the findings presented in Chapter 6 indicate that mentalising ability is intact among neurotypical transgender people. This finding is the first of its kind and very important because it indicates that mentalising is *not* the shared underlying mechanism that leads to the high co-occurrence of ASD and gender dysphoria/incongruence, it appears. This does not exclude the possibility, however, that gender dysphoria/incongruence in ASD results directly from the poor mentalising ability that is characteristic in ASD and by many the cause of social communication difficulties that are diagnostic of the disorder (e.g., Brunsdon & Happé, 2014; Frith, 1994; Jones et al., 2018; Tager-Flusberg, 1999). As demonstrated in Chapter 6, mentalising ability was significantly related to gender dysphoric feelings among autistic cisgender people (poorer mentalising = more gender dysphoric feelings). This group also showed poor performance on the mentalising task and increased gender dysphoric feelings, albeit a congruence between people's birth-assigned sex and experienced/reported gender. In contrast, mentalising ability was unrelated to gender dysphoric feelings in autistic transgender people. They also performed significantly better on the mentalising task, but reported significantly more ASD traits than autistic cisgender people. Taken together, results indicate that poor mentalising ability might trigger some gender dysphoric feelings in autistic people, yet it is not the mechanism that explains why some autistic people develop clinically significant gender dysphoria and/or disidentify with the gender associated with their birth-assigned sex.

How Does ASD Affect Gender-Related Cognition?

To further understand the phenomenon of the high co-occurrence of ASD and gender dysphoria/incongruence, it is important to know whether autistic people are able to develop a strong gender self-concept. Research on this topic is surprisingly limited and the sparse knowledge we have about it has stemmed entirely from self-reports. This thesis provides the first examination of implicit gender-related cognition in ASD. Specifically, in Chapter 5 we examined the relation between ASD traits and the strength of implicit and explicit gender self-concept in people from the general population. To tap gender self-concept we adopted the research tradition of identification with *gender stereotypical attributes* (Wood & Eagly, 2009; 2015).

In Experiment 1, we demonstrated a significant relation between ASD traits and the strength of the explicit *and* implicit gender self-concept, suggesting that cisgender people from the general population with high ASD traits have a weaker inclination to identify explicitly *and* implicitly with masculine and feminine stereotypical attributes. Yet, an exploratory analysis showed that the association hold its significance only among birth-assigned females, indicating a selective influence of ASD traits on the implicit experience of gender stereotypical attributes on birth-assigned females. In Experiment 2, we successfully replicated the relation between number of ASD traits and the strength of explicit gender self-concept (more ASD traits = weaker explicit identification with masculine and feminine attributes) in an independent sample of people from the general population. In contrast to the original study, however, the number of self-reported ASD traits was unrelated to the strength of people's implicit identification with gender stereotypical attributes. Taken together, Chapter 5 presents strong evidence of a robust and reliable link between ASD traits and the strength of the explicit gender self-concept in people from the general population, yet the findings about the relation between ASD traits and the

strength of the implicit gender self-concept were less straightforward. This relation was further examined in the study presented in Chapter 6, using gender self-categorisation to tap gender self-concept. That is a more recent approach to the study of gender self-concept (Wood & Eagly, 2015) and arguably less volatile than the approach we adopted in the studies presented in Chapter 5.

Once again, ASD traits among neurotypical cisgender people were found unrelated to the strength of implicit gender self-concept among neurotypical cisgender people. In keeping with the results presented in Chapter 5, however, the more ASD traits neurotypical cisgender people self-reported, the weaker their explicit gender self-concept. Specifically, the more ASD traits neurotypical cisgender birth-assigned females self-reported, the weaker their explicit identification with female groups and the more ASD traits neurotypical cisgender birth-assigned males self-reported, the weaker their explicit identification with male groups. The findings presented in Chapters 5 and 6 were the first of their kind. Based on evidence that the study of the relation between ASD traits and other phenomena of interest in the gender population can provide important information about the ASD itself (e.g., Lind et al., 2020; Williams, Bergström, et al., 2018), we would expect autistic people to show weak explicit and implicit (at least birth-assigned females) gender self-concepts.

The surprisingly limited research of gender related-cognition in this population has shown that autistic people have a weaker propensity to identify explicitly with masculine stereotypical traits and roles, but no difficulty in identifying with feminine traits and roles (Bejerot & Eriksson, 2014; Stauder et al., 2011). Methodological limitations (e.g., low statistical power), however, do not allow firm conclusions to be drawn from these studies. To our knowledge, the study

presented in Chapter 6 is the first study that has examined the implicit experience of gender in autistic people, to date.

In Chapter 6, we demonstrated that autistic cisgender people show a weaker *explicit* identification with the gender groups that are associated with their birth-assigned sex than neurotypical cisgender individuals. Results were entirely expected based on our findings from the general population. Autistic cisgender people also showed an atypical implicit identification; while, with autistic birth-assigned males identified *more* strongly with male gender than neurotypical birth-assigned males, autistic birth-assigned females identified *less* strongly with female gender than neurotypical birth-assigned females. It is important to note that the latter finding is in keeping with our previous evidence of a selective influence of ASD traits in the strength of the implicit gender self-concept among females only. In contrast, autistic transgender and neurotypical transgender people seem to identify explicitly and implicitly with gender groups that are associated with their experienced/reported gender almost to the same degree.

Taken together, the findings about the effect of ASD on gender-related cognition presented in this thesis indicate that ASD seems to hinder the development of a strong/typical explicit and implicit gender self-concept among autistic birth-assigned females and a strong/typical explicit gender self-concept among birth-assigned males. Gender identification difficulties in autistic cisgender birth-assigned females seem to stem from a diminished implicit experience of gender feelings, whereas gender identification difficulties in autistic cisgender birth-assigned males seem to stem from a diminished/atypical representation of them. Importantly, in autistic people who feel clinically significant gender dysphoria, ASD *does not* inhibit

the development of a strong gender self-concept that matches their experienced/reported gender.

Clinical and Theoretical Implications

Given the common overrepresentation of ASD and gender dysphoria/incongruence in clinical settings, it would be expected medical health professionals to be well aware of the existence of this phenomenon. However, research has shown that this is not the case. Murphy and Livesey (2017) found that 54% of secondary healthcare professionals, who were frequently seeing people for ASD or gender related issues, were unaware of the high co-occurrence of ASD and gender dysphoria/incongruence. Limited knowledge about this phenomenon among health care professionals might provide a partial explanation for the difficulty autistic people with gender dysphoria/incongruence face when it comes to receiving medical care for gender-related issues (Coleman-Smith et al., 2020) and being understood by health providers when they express these issues (Strang, Powers et al., 2018).

In this thesis we established the existence of a link between ASD and gender dysphoria/incongruence, and although the pathways that lead to gender-related issues might be different between neurotypical gender dysphoric/incongruent people and autistic gender dysphoric/incongruent individuals, we found that both groups suffer from gender dysphoria to the same degree. Key stakeholders have also come to the conclusion that "the autistic transgender and gender-diverse youth sounded 'very much like typical trans youth' in terms of their gender urgency" (Strang, Powers, et al., 2018, p. 4050). Thus, the first and foremost clinical implication that arise from the findings presented in this thesis is the urgent need of increasing awareness among clinicians and healthcare professionals about the link between ASD and gender dysphoria/incongruence. This can be achieved by designing and offering trainings to

this group on this phenomenon. This is important to limit the number of cases in which ASD features in gender dysphoric/incongruent people or gender-related issues in autistic people are interpreted as secondary characteristics of either GD or ASD and as such being overlooked. When these issues are detected, it is vital that people are referred for further evaluation to specialised clinicians to get timely support and care (Strang, Meagher, et al., 2018).

The second major clinical implication that arises from the findings presented in this thesis is the need to provide psychoeducation about gender to autistic people. We found that autistic cisgender people have subclinical feelings of gender dysphoria, limited gender-typed behaviour recalled from childhood, and a weaker at least explicit gender self-concept. These findings underscore the need for autistic people to learn in a systematic and structured way about gender, how gender can be expressed, and the variations of gender. Psychoeducation about gender can be integrated to standard intervention programmes for this population aiming to minimise stressors associated with gender incongruence, enhance their understanding of their own self, and help them to develop their own identity so that they can live a more comfortable life.

Psychoeducation should also be provided to parents who raise autistic children to increase their knowledge and understanding on gender development and expression in ASD, reduce their stress levels if atypical expressions of gender are presented, and encourage them to report early signs of gender dysphoric feelings to healthcare professionals.

To our knowledge, this thesis provides the first evidence that a deficit in mentalising might contribute to the development of gender dysphoric feelings in ASD, and this is important from a theoretical perspective. Adopting a developmental

stance and examining how gender self-concept is developed and expressed in ASD may provide unique insights into the mechanisms that are involved in the normative development of gender self-concept. On this basis, it can be argued that the findings presented in this thesis provide supporting evidence to the hypothesis that mentalising is involved in the normative development of gender self-concept (Miller, 2007; Trautner et al., 2003; Zmyj & Bischof-Köhler, 2015). As already described in pervious chapters, this can occur in a number of ways. For example, gender self-concept/identity formulates and consolidates in a social context. From a young age, children start to internalise the traits that stereotypically define the gender associated with their birth-assigned sex (e.g., Coyne et al., 2016). To achieve this, children should be able to represent and impute mental states to others and to their own self. Furthermore, the emergence of self-conscious emotions, such as embarrassment and shame is thought to depend on mentalising (Hobson et al. 2006) and more specifically with the awareness of social norms violation and the ability to understand that one's own behaviour is observed and judged in a social context (Heerey et al., 2003). To avoid feelings of guilt and embarrassment that stem from violations to social norms, people tend to conform to social conditioning (e.g., Scheff 1988; Suhay 2015). On this basis, it is likely that in the process of gender self-concept/identity development, children conform to gender norms to avoid feelings of embarrassed and guilt that would be triggered as a result of others' reactions if they violated these norms. Without feeling a pressure to conform, autistic people are "free" to explore the variations of gender to a greater extent than non-autistic people.

Limitations and Future Directions

This thesis provides important insight into the high co-occurrence of ASD and gender dysphoria/incongruence, yet our findings should be interpreted in the

light of some limitations. First, it is important to mention that all the conclusions we made were based on evidence from the adult population and cannot be generalised to children. Given that the high co-occurrence of ASD and gender dysphoria/incongruence is also evident in the child population (e.g., Akgül et al., 2019; de Vries et al., 2010; Leef et al., 2019; van der Miesen, de Vries, et al., 2018), we strongly believe that in order to acquire a full understanding of such a complicated phenomenon, it is essential to study its manifestation in the course of development. Our aim was to partially address this issue in the current thesis by conducting a case-control study among autistic and neurotypical children. Unfortunately, due to unforeseen circumstances we were not able to proceed with our experiment.[17] Nonetheless, we hope that this thesis will provide impetus for a number of hypotheses related to this phenomenon to be tested in the child population.

For example, based on neurotypical transgender people's intact performance on the mentalising task, we argued that mentalising is not the shared underlying mechanism that could explain the high co-occurrence of ASD and gender dysphoria/incongruence. An alternative explanation would be, however, that neurotypical transgender people achieved an undiminished performance on the task by using "compensation" strategies (see Livingston & Happé, 2017). Research has shown that "typical" performance on explicit mentalising tasks might be achieved, albeit persisting, underlying difficulties in this ability (Lever & Geurts, 2016; Senju

[17] As part of this project, we had planned to conduct a study on gender development in autistic children. The study was approved by the University of Kent Psychology Research Ethics Committee (ID: 202015820420376361), and it was planned to begin in March, 2020. However, due to the outbreak of COVID-19 pandemic the experiment was aborted.

et al., 2009). To increase veracity in our findings, future research might usefully investigate mentalising in transgender children, and their parents and siblings. We could firmly reject the hypothesis that mentalising is the shared underlying mechanism that explains the link between ASD and gender dysphoria/incongruence, only if transgender children and their relatives show intact performance on implicit (e.g., anticipatory looking anticipatory eye movements during a false belief scenario; Senju et al., 2009) *and* explicit mentalising tasks (e.g. Strange Stories Films Task; Murray et al., 2017). It is also important to investigate mentalising ability in *clinically referred* neurotypical transgender people. Another topic that future research should examine is gender-related cognition in autistic children. Surprisingly, our knowledge on how autistic children formulate and consolidate a gender self-concept is nonexistent. In this thesis, we concluded that ASD seems to hinder the experience and expression of a strong explicit (and implicit at least among birth-assigned females) gender self-concept that matches people's birth-assigned sex. To verify our findings, future studies should examine whether autistic children show a developmental lag in the formation and consolidation of a gender self-concept, using implicit and explicit measures. Longitudinal studies can also provide important insight on the developmental trajectories of gender self-concept in ASD and identify factors that contribute to the development of diagnosable GD or gender incongruence only among a number of autistic children.

Second, we consider important to mention that the studies conducted as part of this project were mostly internet-based. The popularity of internet-based studies as a method of data collection in psychology has increased significantly the last years (Skitka, & Sargis, 2006). Compared to more traditional approaches of data collection (e.g., lab-based studies), internet-based studies are usually cheaper, more time

efficient, and give the opportunity to researchers to approach and examine participants from populations less easily accessible (Rubenstein & Furnier, 2020). This makes internet-based studies a particularly popular method for the investigation of the high co-occurrence of ASD and gender dysphoria/incongruence (e.g., Jones et al., 2012; Warrier et al., 2020) and the expression of gender in ASD more generally (e.g., George & Stokes, 2018b; Pecora et al., 2020). We should not overlook, however, the challenges of this method, when it comes to the analysis and interpretation of the findings of internet-based studies. The self-selection bias is considered as one of the main challenges of this method (Rubenstein & Furnier, 2020).

In the current thesis, we tried to minimise self-selection bias by providing a generic description of our studies, in order not to attract solely participants who were interested in gender-related topics or/and autism. Specifically, prospective participants were informed that they would take part in studies that explore self-concepts. Of course, to ensure transparency all the measures we used were described in detail in the information sheet of the respective studies. Thus, we cannot rule out entirely the possibility that self-selection bias did not have any effect on the effect estimates presented in this thesis. We should note, however, that findings in this thesis are in keeping with those from studies in which self-selection bias was addressed more rigorously (i.e., Warrier et al., 2020). This comparison was not always possible, thus replication of our findings awaits before strong conclusions can be drawn.

Lastly, we should note that in this thesis we used an implicit measurement technique to investigate gender-related cognition (i.e., Implicit Association Test). Although IATs are widely used in research, are less fakeable than self-reports (Nosek

et al., 2007), and have better psychometric properties that other implicit measures (Bosson et al., 2000; Teige et al., 2004), there is an ongoing debate about the mechanisms that underpin performance on this task. It has been suggested that IAT taps people's familiarity with environmental associations rather than automatic and spontaneous mental associations related to the concept of self (Karpinski & Hilton, 2001; Olson & Fazio, 2004). Specifically, Olson and Fazio (2004) argued that "the IAT is contaminated by extrapersonal associations—associations that are available in memory but are irrelevant to the perceived likelihood of personally experiencing a positive or negative outcome on interaction with the attitude object" (p. 653). If this is true, then high scores on the IATs we used might not indicate a strong female/male self-concept. Instead, they might indicate a person's familiarity with feminine/masculine stimuli. To address this potential limitation in future studies, researchers might adapt and used Olson and Fazio's (2004) "personalized" IAT to tap gender self-concept.

Conclusion

In sum, this thesis indicates that (a) the link between ASD and gender dysphoria/incongruence exists and is real, (b) a difficulty in mentalising ability might trigger subclinical gender dysphoric feelings in autistic cisgender people, and (c) ASD hinders the explicit (and implicit among birth-assigned females) development/expression of a strong gender self-concept in autistic cisgender people. We believe that our findings offer valuable insights into a phenomenon that despite its major clinical importance remains underresearched. Also, we look forward to additional research in the coming years that adds to and extends our findings. This is important because "we can provide good clinical support only to those whom we

understand and we can understand only those whom we acknowledge and study" (Strang, Janssen, et al., 2018, p. 886).

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Appendix A: Supplemental Material for Chapter 2

Meta-Analysis of Studies of ASD Traits in Gender Dysphoric/Incongruent People, Including Pasterski et al.'s (2014) Study

When we included Pasterski et al.'s (2014) study (g = 0.13) instead of Jones et al.'s (2012) study in the meta-analysis of studies of ASD traits in gender dysphoric/incongruent people results did not change substantively. Just as in the original meta-analysis, we used a random-effects model, and results revealed that the overall weighted effect size of the difference in the number of reported ASD traits between gender dysphoric/incongruent and neurotypical/population-based control participants was moderate, g = .66 (SE = 0.15, 95% CI 0.36 to 0.96, z = 4.28, p < .001). The Q-value was 603.94, df = 10, p < .001, indicating that the effect sizes included in the analysis were significantly different from each other. The I^2 statistic was 98.34, suggesting that 98.34% of the variance in the observed effects reflects variance in true effects rather than sampling error. The variance of true effects (Tau^2) was 0.25, the standard deviation of true effects (Tau) was 0.50, and the prediction interval was -0.52 to 1.83.

Meta-Analysis of Studies of ASD Traits in Gender Dysphoric/Incongruent People, Using Ruzich, Allison, Chakrabarti et al.'s (2015) Control Group for Kung's (2020) Study

When we conducted the meta-analysis of studies of ASD traits in gender incongruent/dysphoric people and included Kung's (2020) study with the control group from Ruzich, Allison, Chakrabarti, et al. (2015; g = 0.39) instead of the control group from Baron-Cohen et al. (2014), results did not change substantively. Just as in the original meta-analysis, we used a random-effects model, and results revealed that the overall weighted effect size of the difference in the number of reported ASD

traits between gender dysphoric/incongruent and neurotypical/population-based participants was moderate, $g = .64$ ($SE = 0.16$, 95% CI 0.34 to 0.95, $z = 4.14$, $p < .001$). The Q-value was 643.64, $df = 10$, $p < .001$, indicating that the effect sizes included in the analysis were significantly different from each other. The I^2 statistic was 98.45, suggesting that 98.45% of the variance in the observed effects reflects variance in true effects rather than sampling error. The variance of true effects (Tau^2) was 0.25, the standard deviation of true effects (Tau) was 0.50, and the prediction interval was -0.55 to 1.83.

Table A1

Studies Excluded from the Literature Review

	Reference	Reasons for Exclusion
1	Aldridge, Z., Patel, S., Guo, B., Nixon, E., Pierre Bouman, W., Witcomb, G. L., & Arcelus, J. (2020). Long-term effect of gender-affirming hormone treatment on depression and anxiety symptoms in transgender people: A prospective cohort study. *Andrology*.	Quantitative study (no relevant data)
2	Baker, P. & Shweikh, E. (2016). Autistic spectrum disorders, personality disorder and offending in a transgender patient: clinical considerations, diagnostic challenges and treatment responses. *Advances in Autism, 2*(3), 140-146.	Case report
3	Bejerot, S., Humble, M. B., & Gardner, A. (2011). Endocrine disruptors, the increase of autism spectrum disorder and its comorbidity with gender identity disorder–a hypothetical association. *International Journal of Andrology, 34,* Article e350.	Letter to the editor
4	Bennett, M., & Goodall, E. (2016). Towards an agenda for research for lesbian, gay, bisexual, transgendered and/or intersexed people with an Autism Spectrum Diagnosis. *Journal of Autism and Developmental Disorders*, *46*(9), 3190-3192.	Letter to the editor
5	Cain, L. K., & Velasco, J. C. (2020). Stranded at the intersection of gender, sexuality, and autism: Gray's story. *Disability & Society, 36*(3), 358-375.	Case study
6	Carlile, A. (2020). The experiences of transgender and nonbinary children and young people and their parents in healthcare settings in England, UK: Interviews with members of a family support group. *International Journal of Transgender Health, 21*(1), 16-32.	Qualitative study
7	Coleman-Smith, R. S., Smith, R., Milne, E., & Thompson, A. R. (2020). 'Conflict versus Congruence': A Qualitative Study Exploring the Experience of Gender Dysphoria for Adults with Autism Spectrum Disorder. *Journal of Autism and Developmental Disorders, 50,* 2643-2657.	Qualitative study
8	Davidson, J., & Tamas, S. (2016). Autism and the ghost of gender. *Emotion, Space and Society, 19,* 59-65.	Review
9	Di Ceglie, D. (2018). The use of metaphors in understanding atypical gender identity development and its psychosocial impact. *Journal of Child Psychotherapy, 44*(1), 5-28.	Theoretical/observational paper
10	Ehrensaft, D. (2018). Double helix rainbow kids. *Journal of Autism and Developmental Disorders*, *48*(12), 4079-4081.	Letter to the editor

	Reference	Reasons for Exclusion
11	Gallucci, G., Hackerman, F., & Schmidt, C. W. (2005). Gender identity disorder in an adult male with Asperger's syndrome. *Sexuality and Disability*, *23*(1), 35-40.	Case report
12	George R., Stokes M. (2016) "Gender Is Not on My Agenda!": Gender Dysphoria and Autism Spectrum Disorder. In: Mazzone L., Vitiello B. (eds) *Psychiatric Symptoms and Comorbidities in Autism Spectrum Disorder*. Springer.	Book chapter
13	George, R., & Stokes, M. A. (2018). A quantitative analysis of mental health among sexual and gender minority groups in ASD. *Journal of Autism and Developmental Disorders, 48*(6), 2052-2063.	Quantitative study (no relevant data)
14	Glidden, D., Bouman, W. P., Jones, B. A., & Arcelus, J. (2016). Gender dysphoria and autism spectrum disorder: A systematic review of the literature. *Sexual Medicine Reviews*, *4*(1), 3-14.	Literature review
15	Hall, J. P., Batza, K., Streed, C. G., Boyd, B. A., & Kurth, N. K. (2020). Health disparities among sexual and gender minorities with autism spectrum disorder. *Journal of Autism and Developmental Disorders*, 1-7.	Mixed-methods study (no relevant data)
16	Hill, S. A., Thorpe, A., Petrauskaite, R., & Wilson, S. (2020). Characteristics of patients with Gender Dysphoria admitted to a secure forensic adolescent hospital. *The Journal of Forensic Psychiatry & Psychology, 31*(6), 854-867.	Quantitative study (no relevant data)
17	Hillier, A., Gallop, N., Mendes, E., Tellez, D., Buckingham, A., Nizami, A., & OToole, D. (2020). LGBTQ+ and autism spectrum disorder: Experiences and challenges. *International Journal of Transgender Health*, *21*(1), 98-110.	Qualitative study
18	Jackson-Perry, D. (2020). The autistic art of failure? Unknowing imperfect systems of sexuality and gender. *Scandinavian Journal of Disability Research, 22*(1), 221-229.	Critical analysis
19	Jacobs, L. A., Rachlin, K., Erickson-Schroth, L., & Janssen, A. (2014). Gender dysphoria and co-occurring autism spectrum disorders: Review, case examples, and treatment considerations. *LGBT Health*, *1*, 277-282.	Case study
20	Jacobs, L. A., Rachlin, K., Erickson-Schroth, L., & Janssen, A. (2016). Response to Dr. Parkinson. *LGBT Health*, *3*, 175-176.	Letter to the editor
21	James, W. H., & Grech, V. (2020). Is exposure to high levels of maternal intrauterine testosterone a causal factor common to male sex, autism, gender dysphoria, and non-right-handedness?. *Early Human Development, 141*, Article 104872.	Review
22	Kraemer, B., Delsignore, A., Gundelfinger, R., Schnyder, U., & Hepp, U. (2005). Comorbidity of Asperger syndrome and gender identity disorder. *European Child & Adolescent Psychiatry*, *14*(5), 292-296.	Case report

	Reference	Reasons for Exclusion
23	Kaltiala-Heino, R., Sumia, M., Työläjärvi, M., & Lindberg, N. (2015). Two years of gender identity service for minors: overrepresentation of natal girls with severe problems in adolescent development. *Child and Adolescent Psychiatry and Mental Health, 9*(1), 1-9.	Quantitative study (no relevant data)
24	Kaltiala-Heino, R., Työläjärvi, M., & Lindberg, N. (2019). Sexual experiences of clinically referred adolescents with features of gender dysphoria. *Clinical Child Psychology and Psychiatry, 24*(2), 365-378.	Quantitative study (no relevant data)
25	Kuvalanka, K. A., Mahan, D. J., McGuire, J. K., & Hoffman, T. K. (2018). Perspectives of mothers of transgender and gender-nonconforming children with autism spectrum disorder. *Journal of Homosexuality, 65*(9), 1167-1189.	Qualitative study
26	Landén, M., & Rasmussen, P. (1997). Gender identity disorder in a girl with autism-a case report. *European Child & Adolescent Psychiatry, 6*, 170-173.	Case report
27	Lehmann, K., & Leavey, G. (2017). Individuals with gender dysphoria and autism: Barriers to good clinical practice. *Journal of Psychiatric and Mental Health Nursing, 24*(2-3), 171-177.	Essays and debates in mental health
28	Lemaire, M., Thomazeau, B., & Bonnet-Brilhault, F. (2014). Gender identity disorder and autism spectrum disorder in a 23-year-old female. *Archives of Sexual Behavior, 43*(2), 395-398.	Case report
29	Mukaddes, N. M. (2002). Gender identity problems in autistic children. *Child: Care, Health and Development, 28*(6), 529-532.	Case report
30	Naguy A. (2020). Autism and gender dysphoria: searching for the holy grail. *The Primary Care Companion for CNS Disorders, 22*(2), Article 19l02492.	Letter to the editor
31	Nordahl-Hansen, A., Cicchetti, D. V., & Øien, R. A. (2019). A Review Update on Gender Dysphoria and ASD and Response to Corrections. *Journal of Autism and Developmental Disorders, 49*(4), 1745-1748.	Commentary
32	Øien, R. A., Cicchetti, D. V., & Nordahl-Hansen, A. (2018). Gender dysphoria, sexuality and autism spectrum disorders: A systematic map review. *Journal of Autism and Developmental Disorders, 48*(12), 4028-4037.	Map review
33	Parkes, G., & Hall, I. (2006). Gender dysphoria and cross-dressing in people with intellectual disability: a literature review. *Mental Retardation, 44*(4), 260-271.	Literature review
34	Parkinson, J. (2014). Gender dysphoria in Asperger's syndrome: A caution. *Australasian Psychiatry, 22*(1), 84-85.	Case report
35	Parkinson, J. (2016). Gender dysphoria and autism spectrum disorders: A note of caution. *LGBT Health.*	Letter to the editor

	Reference	Reasons for Exclusion
36	Pecora, L. A., Hooley, M., Sperry, L., Mesibov, G. B., & Stokes, M. A. (2020). Sexuality and Gender Issues in Individuals with Autism Spectrum Disorder. *Child and Adolescent Psychiatric Clinics, 29*(3), 543-556.	Review
37	Perera, H., Gadambanathan, T., & Weerasiri, S. (2003). Gender identity disorder presenting in a girl with Asperger's disorder and obsessive compulsive disorder. *The Ceylon Medical Journal, 48*(2), 57-58.	Case report
38	Ristori, J., Cocchetti, C., Castellini, G., Pierdominici, M., Cipriani, A., Testi, D., Gavazzi, G., Mazzoli, F., Mosconi, M., Meriggiola, M. C., Cassioli, E., Vignozzi, L., Ricca, V., Maggi, M., & Fisher, A. D. (2020). Hormonal treatment effect on sexual distress in transgender persons: 2-year follow-up data. *The Journal of Sexual Medicine, 17*(1), 142-151.	Quantitative study (no relevant data)
39	Robinow, O. (2009). Paraphilia and transgenderism: a connection with Asperger's disorder? *Sexual and Relationship Therapy*, *24*(2), 143-151.	Literature review
40	Russell, I., Pearson, B., & Masic, U. (2021). A Longitudinal Study of Features Associated with Autism Spectrum in Clinic Referred, Gender Diverse Adolescents Accessing Puberty Suppression Treatment. *Journal of Autism and Developmental Disorders, 51*(6), 2068-2076.	Longitudinal study (no control group)
41	Saleem, F., & Rizvi, S. W. (2017). Transgender associations and possible etiology: A literature review. *Cureus, 9*(12), Article e1984.	Review
42	Selinger, D. (2018). Autism-What Does Gender Have to Do With It? *Journal of Infant, Child, and Adolescent Psychotherapy, 17*(3), 163-177.	Case report
43	Shapira, S., & Granek, L. (2019). Negotiating psychiatric cisgenderism-ableism in the transgender-autism nexus. *Feminism & Psychology, 29*(4), 494-513.	Qualitative study
44	Strang, J. F., Janssen, A., Tishelman, A., Leibowitz, S. F., Kenworthy, L., McGuire, J. K., Edwards-Leeper, L., Mazefsky, C. A., Rofey, D., Bascom, J., Caplan, R., Gomez-Lobo, V., Berg, D., Zaks, Z., Wallace, G. L., Wimms, H., Pine-Twaddell, E., Shumer, D., Register-Brown, K., Sadikova, E., Anthony, L. G., & Caplan, R. (2018). Revisiting the link: Evidence of the rates of autism in studies of gender diverse individuals. *Journal of the American Academy of Child & Adolescent Psychiatry*, *57*, 885-886.	Letter to the editor
45	Strang, J. F., Jarin, J., Call, D., Clark, B., Wallace, G. L., Anthony, L. G., Kenworthy, L., & Gomez-Lobo, V. (2018). Transgender youth fertility attitudes questionnaire: measure development in non-autistic and autistic transgender youth and their parents. *Journal of Adolescent Health, 62*(2), 128-135.	Mixed-methods study (no relevant data)

	Reference	Reasons for Exclusion
46	Strang, J. F., Klomp, S. E., Caplan, R., Griffin, A. D., Anthony, L. G., Harris, M. C., Graham, E. K., Knauss, M., & van der Miesen, A. I. R. (2019). Community-based participatory design for research that impacts the lives of transgender and/or gender-diverse autistic and/or neurodiverse people. *Clinical Practice in Pediatric Psychology*, *7*(4), 396-404.	Commentary
47	Strang, J. F., Knauss, M., van der Miesen, A., McGuire, J. K., Kenworthy, L., Caplan, R., Freeman, A., Sadikova, E., Zaks, Z., Pervez, N., Balleur, A., Rowlands, D. W., Sibarium, E., Willing, L., McCool, M. A., Ehrbar, R. D., Wyss, S. E., Wimms, H., Tobing, J., … Anthony, L. G. (2020). A clinical program for transgender and gender-diverse neurodiverse/autistic adolescents developed through community-based participatory design. *Journal of Clinical Child & Adolescent Psychology*, 1-16.	Mixed-methods study (no relevant data)
48	Strang, J. F., Meagher, H., Kenworthy, L., de Vries, A. L. C., Menvielle, E., Leibowitz, S., Janssen, A., Cohen-Kettenis, P., Shumer, D. E., Edwards-Leeper, L., Pleak, R. R., Spack, N., Karasic, D. H., Schreier, H., Balleur, A., Tishelman, A., Ehrensaft, D., Rodnan, L., E., S., Kuschner, Mandel, F., Caretto, A., Lewis, H. C., & Anthony, L. G. (2016). Initial clinical guidelines for co-occurring autism spectrum disorder and gender dysphoria or incongruence in adolescents. *Journal of Clinical Child & Adolescent Psychology, 47*(1), 105-115.	Mixed-methods study (no data)
49	Strang, J. F., Powers, M. D., Knauss, M., Sibarium, E., Leibowitz, S. F., Kenworthy, Sadikova, E., Wyss, S., Willing, L., Caplan, R., Pervez, N., Nowak, J., Gohari, D., Gomez-Lobo, V., Call, D., & Anthony, L. G. (2018). "They thought it was an obsession": Trajectories and perspectives of autistic transgender and gender-diverse adolescents. *Journal of Autism and Developmental Disorders*, *48*(12), 4039-4055.	Qualitative study
50	Tateno, M., Tateno, Y., & Saito, T. (2008). Comorbid childhood gender identity disorder in a boy with Asperger syndrome. *Psychiatry and Clinical Neurosciences, 62*(2), 238.	Letter to the editor
51	Tateno, M., Teo, A. R., & Tateno, Y. (2015). Eleven-year follow up of boy with Asperger's syndrome and comorbid gender identity disorder of childhood. *Psychiatry and Clinical Neurosciences*, *69*(10), 658-659.	Letter to the editor
52	Thrower, E., Bretherton, I., Pang, K. C., Zajac, J. D., & Cheung, A. S. (2020). Prevalence of Autism Spectrum Disorder and Attention-Deficit Hyperactivity Disorder Amongst Individuals with Gender Dysphoria: A Systematic Review. *Journal of Autism and Developmental Disorders, 50*(3), 695-706.	Literature review
53	Turban, J. L. (2018). Potentially reversible social deficits among transgender youth. *Journal of Autism and Developmental Disorders*, *48*(12), 4007-4009.	Letter to the editor

	Reference	Reasons for Exclusion
54	Turban, J. L., & van Schalkwyk, G. I. (2018). "Gender dysphoria" and autism spectrum disorder: Is the link real? *Journal of the American Academy of Child & Adolescent Psychiatry*, 57(1), 8-9.	Critical review
55	Turban, J. L., & van Schalkwyk, G. I. (2018). Drs. Turban and van Schalkwyk reply. *Journal of the American Academy of Child & Adolescent Psychiatry*, *57*(11), 887-889	Letter to the editor
56	van der Miesen, A. I. R., Cohen-Kettenis, P. T., de Vries, A. L. C. (2018). Is there a link between gender dysphoria and autism spectrum disorder? *Journal of the American Academy of Child & Adolescent Psychiatry*, 57(11), 884-885	Letter to the editor
57	Van Der Miesen, A. I., Hurley, H., & De Vries, A. L. (2016). Gender dysphoria and autism spectrum disorder: A narrative review. *International Review of Psychiatry*, *28*(1), 70-80	Narrative review
58	van Schalkwyk, G. I., Klingensmith, K., & Volkmar, F. R. (2015). Gender identity and autism spectrum disorders. *The Yale Journal of Biology and Medicine*, *88*(1), 81-83.	Literature review
59	Violeta, K. J., & Langer, S. J. (2017). Integration of desire, sexual orientation, and female embodiment of a transgender woman previously diagnosed with autism spectrum disorder: A case report. *Journal of Gay & Lesbian Mental Health*, *21*(4), 352-370.	Case report
60	Williams, P. G., Allard, A. M., & Sears, L. (1996). Case study: Cross-gender preoccupations in two male children with autism. *Journal of Autism and Developmental Disorders*, *26*(6), 635-642.	Case study
61	Zucker, K. J., & VanderLaan, D. P. (2018). Corrections to Øien, Cicchetti, and Nordahl-Hansen's (2018) "Gender dysphoria, sexuality and autism spectrum disorder: A systematic map review". *Journal of Autism and Developmental Disorders*, *48*, Article 4038.	Letter to the editor

Table A2

Quantitative Studies that Report Data on the Overlap Between ASD and Gender Dysphoria/Incongruence

Authors (year)	Targeted population	Design	Focus	Age group	Sample	Relevant measures	Relevant findings
de Vries et al. (2010)	GD/gender incongruence	Cross-sectional	ASD in children & adolescents with GD	Child & adolescent	Referred to gender clinic (N = 204) Children (n = 108) M_{age} = 8.06; SD = 1.82 Adolescents (n = 96) M_{age} = 13.92; SD = 2.29	Diagnostic tool: DISCO-10	7.8% of the sample had ASD 4.7% of individuals with GID had ASD 17.0% of individuals with GID-NOS had ASD
Jones et al. (2012)	GD/gender incongruence	Case-control	ASD traits	Adult	Transgender recruited online/gender clinic (n = 259) Transgender men (n = 61) M_{age} = 34.0; range = 19-52.7 Transgender women (n = 198) M_{age} = 45.1; range = 16-75 NT (Baron-Cohen et al., 2001; n = 174) M_{age} = 37; Range = 18.1-60.0 ASD (Wheelwright et al., 2006; n = 125) M_{age} = 37.6; range = 17.6-71.1	Screening tool: AQ-50 (cut-off scores & difference between group means) Self-reported ASD diagnosis	~ 30% of transgender men and 5% of transgender women scored in the medium or narrow autism phenotype range Transgender men scored significantly higher on the AQ than NT women and NT men. The difference in the AQ score between transgender women and either NT men or NT women was nonsignificant 2.7% of transgender individuals reported a diagnosis of ASD
Spack et al. (2012)	GD/gender incongruence	Retrospective chart review	Demographic & clinical data	Child, adolescent, & adult	Patients diagnosed with GID referred to a paediatric medical centre (N = 97) M_{age} = 14.8; SD = 3.4; Median = 16; range = 4-20	ASD diagnosis: Clinic notes & self/parent report	5.15% of patients had autism or PDD
Bejerot & Eriksson (2014)	ASD	Case-control	Sexuality & gender role	Adult	ASD female (n =24) M_{age} = 28.1; SD = 6.3 ASD male (n = 26) M_{age} = 31.8; SD = 7.8 NT female (n = 25) M_{age} = 27.7; SD = 6.7 NT male (n = 28) M_{age} = 32.9; SD = 7.4	Single item measure of gender identity and androgynous behaviour in childhood	Significantly more individuals with ASD than NT reported an atypical gender identity No difference was observed between males with ASD and NT males on being ‘a sissy in childhood’. Females with ASD rated themselves as being more tomboyish in childhood than NT females

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Quantitative Studies that Report Data on the Overlap Between ASD and Gender Dysphoria/Incongruence

Authors (year)	Targeted population	Design	Focus	Age group	Sample	Relevant measures	Relevant findings
Khatchadourian et al. (2014)	GD/gender incongruence	Retrospective chart review	Demographic & clinical data	Adolescent	Patients of a gender clinic diagnosed with GD (N = 84) M_{age} = 16.6, SD = 2.2; Median = 16.8; range = 11.4-22.5	ASD diagnosis: Clinic notes	7% of the sample had a diagnosis of PDD/ASD
Pasterski et al. (2014)	GD/gender incongruence	Case-control	ASD traits	Adult	Transgender diagnosed with GD or GID undertaking treatment at a gender clinic (n = 91) MtF (n = 63) M_{age} = 45.47 FtM (n = 28) M_{age} = 27.38 NT (Baron-Cohen et al., 2001; n = 174)	Screening tool: AQ-50 (cut-off scores & difference between group means)	5.5% of transgender people met the AQ threshold (≥ 32) There was no significant difference in the AQ score between transgender MtF and NT men. Transgender FtM scored higher than NT women on the AQ, but the between-group difference was nonsignificant
Strang et al. (2014)	ASD	Chart review	Gender variance	Child & adolescent	NT (n = 165) M_{age} = 11.87; SD = 3.31; range = 6-18 CBCL Normative (n = 1,605) M_{age} = 11.74; SD = 3.44; range = 6-18 Epilepsy/NF1 (n = 116) M_{age} = 10.12; SD = 2.88; range = 6-17 ADHD (n = 126) M_{age} = 9.77; SD = 2.95; range = 6-17 ASD (n = 147) M_{age} = 12.21; SD = 3.08; range = 7-18	CBCL item 110 = Wish to be the other binary gender	The item was endorsed by parents in 0% of NT participants, 0.7 % of normative nonreferred participants, 1.7 % of participants with a medical neurodevelopmental condition (epilepsy or NF1), 4.8% of participants with ADHD, and 5.4% of ASD participants Compared to the normative sample, parents of ASD participants were 7.59 times more likely to endorse the item and parents of ADHD participants were 6.64 times more likely to endorse the item

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Quantitative Studies that Report Data on the Overlap Between ASD and Gender Dysphoria/Incongruence

Authors (year)	Targeted population	Design	Focus	Age group	Sample	Relevant measures	Relevant findings
Kristensen & Broome (2015)	GD/gender incongruence	Cross-sectional	ASD traits	Adult	Online gender-variant sample (N = 446) Age range 18-75 (mostly)	Screening tool: AQ-10 (Cut-off scores) Self-reported ASD diagnosis	39% of the sample scored above the cut-off 6 14% of the sample reported a formal diagnosis of ASD
Shumer et al. (2015)	General	Cohort	Link between ASD traits & gender nonconformity	Adult	Children (n = 94) ASD (n = 19) Year of birth, median = 1985 NT (n = 75) Year of birth, median = 1985 Mothers (n = 198) Fathers (n = 269)	SRS, 4 items from the RCGI	Higher ASD traits in children or mothers were associated with higher degrees of gender nonconformity in children
Skagerberg et al. (2015)	GD/gender incongruence	Cross-sectional	ASD traits	Child & adolescent	Young people with GD attending a gender identity service (n = 166) M_{age} = 14.26; SD = 2.68; range = 5-18 Normative (Wigham et al., 2012; n = 500)	Screening tool: SRS (cut-off scores) ASD diagnosis: Patient files	27.1% of the GD group fell within the severe range for ASD 12.1% of the sample had a diagnosis of ASD
VanderLaan, Leef, et al. (2015a)	GD/gender incongruence	Retrospective chart review	ASD risk factors & ASD traits	Child	Patients at the outset of assessment for GD and outpatients who had been assessed for GD (N = 49) M_{age} = 7.19; SD = 2.71	Screening tool: SRS (cut-off scores)	44.9% of the sample fell within the clinical range for ASD
VanderLaan, Postema, et al. (2015b)	GD/gender incongruence	Retrospective chart review	Intense/Obsessional interests	Child	Age range = 3-12 Gender-referred (n = 534) Siblings (n = 419) CBCL clinic-referred sample (Achenbach, 1991; n = 1,201) CBCL nonreferred sample (Achenbach, 1991; n = 1,201)	Screening tool: CBCL items 9 (obsessions) & 66 (compulsions)	Gender-referred children were elevated compared to all the other groups for Item 9, and compared to siblings and nonreferred children for Item 66

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Quantitative Studies that Report Data on the Overlap Between ASD and Gender Dysphoria/Incongruence

Authors (year)	Targeted population	Design	Focus	Age group	Sample	Relevant measures	Relevant findings
Chen et al. (2016)	GD/gender incongruence	Retrospective chart review	Characteristics of referrals for GD	Child & adolescent	Patients in a paediatric clinic for GD, GID or gender identity (*N* = 38) M_{age} = 14.4; *SD* = 3.2	ASD diagnosis: chart review of referrals	13.1% of the sample had a diagnosis of ASD
Holt et al. (2016)	GD/gender incongruence	Cross-sectional	Demographics & associated difficulties	Child & adolescent	Referred to gender identity development service with features of GD (*N* = 218) M_{age} = 14; *SD* = 3.08; range = 5-17	ASD diagnosis: referral letters and clinician notes/reports	13.3% of the sample had a diagnosis of ASD
Janssen et al. (2016)	ASD	Retrospective chart review	Gender variance	Child & adolescent	CBCL nonreferred sample (Achenbach et al., 2001; *n* = 1,605) M_{age} = 11.74; *SD* = 3.44; range = 6-18 ASD (*n* = 492) M_{age} = 8.96; *SD* = 2.70; range = 3-17	CBCL item 110 = Wish to be the other binary gender	Compared to normative nonreferred sample, parents of ASD participants were 7.76 times more likely to endorse the item
Shumer et al. (2016)	GD/gender incongruence	Retrospective chart review	Evaluation of Asperger syndrome	Child, adolescent, & adult	Referred to a gender clinic (*N* = 39) M_{age} = 15.8; range = 8-20	Screening tool: ASDS (Cut-off scores) ASD diagnosis: Patient chart data	23.1% of the sample had possibly, likely or very likely Asperger syndrome 10.3% had a diagnosis of ASD
Peterson et al. (2017)	GD/gender incongruence	Retrospective chart review	Suicidality, self-harm, & body dissatisfaction	Adolescent & adult	Transgender who met diagnostic criteria for GD presenting at a gender clinic (*N* = 96) M_{age} = 17.1; *SD* = 2.3; range = 12-22	ASD diagnosis: chart review	3% of the sample had a diagnosis of ASD

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Quantitative Studies that Report Data on the Overlap Between ASD and Gender Dysphoria/Incongruence

Authors (year)	Targeted population	Design	Focus	Age group	Sample	Relevant measures	Relevant findings
Dewinter et al. (2017)	ASD	Case-control	Gender identity, sexual orientation, & romantic relationships	Adolescent & adult	ASD (n = 675) M_{age} = 43.2; SD = 13.5; range = 15-80 NT (n = 8,064) M_{age} = 42.64; SD = 15.9; Range = 15-70	Single item measure of gender identity	15.4% of the autistic participants reported trans, nonbinary, and other/unknown gender identities. Data were not available for the control group
May et al. (2017)	ASD	Case-control	Gender variance	Child & adolescent	ASD (n = 176) M_{age} = 10.5; SD = 2.6 CBCL Nonreferred (Achenbach & Rescorla, 2001; n = 1,605) M_{age} = 11.7; SD = 3.5; range = 6-18 CBCL Clinically referred (Achenbach & Rescorla, 2001; n = 1,605) M_{age} = 11.7; SD = 3.4; range = 6-18	CBCL item 110 = Wish to be the other binary gender	Compared to the normative nonreferred sample, parents of children with ASD were significantly more likely to endorse the item. No significant difference between children with ASD and clinically referred sample was observed
Nahata et al. (2017)	GD/gender incongruence	Retrospective medical record review	Mental health concerns & insurance denials	Child & adolescent	Patients with ICD 9/10 codes for GD referred to a paediatric endocrinology (N = 79) Median $_{age}$ = 15; range = 9-18	ASD diagnosis: Patient medical chart	6.3% of the sample had a diagnosis of ASD
Zucker et al. (2017)	GD/gender incongruence	Case control & cross validation study	Intense/obsessional interests	Child	Children referred to gender clinic service for GD (n = 386) M_{age} = 7.77; SD = 2.41 Clinically referred (Achenbach & Rescorla, 2001; n = 965) Nonreferred (Achenbach & Rescorla, 2001; n = 965)	Screening tool TRF items: 9 (obsessions) & 66 (compulsions)	Gender-referred children were elevated compared to all the other groups for Item 9, and compared to the nonreferred children for Item 66

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Quantitative Studies that Report Data on the Overlap Between ASD and Gender Dysphoria/Incongruence

Authors (year)	Targeted population	Design	Focus	Age group	Sample	Relevant measures	Relevant findings
Akgül et al. (2018)	GD/gender incongruence	Case-control	ASD traits & executive functions	Child & adolescent	Children satisfied DSM-5 criteria for GD (n = 25) M_{age} = 11.56; SD = 4.15 NT (n = 50) M_{age} = 11.42; SD = 3.91	Screening tool: SRS (cut-off scores & difference between group means)	68% of children with GD and 22% of NT children fell within the clinical range for ASD Children with GD had significantly more ASD traits than NT children
Becerra-Culqui et al. (2018)	GD/gender incongruence	Retrospective & prospective cohort study	Mental health	Child & adolescent	Age range = 3-17 Transgender/gender nonconforming in health care systems (n = 1,333) Reference (n = 26,300)	ASD diagnosis ICD-9 Code: 299.x electronic medical records	4.7% of the sample had a diagnosis of ASD
Cheung et al. (2018)	GD/gender incongruence	Retrospective audit of electronic medical records	Sociodemographic & clinical characteristics	Adolescent & adult	Transgender, nonbinary, and unassigned referred to a primary care and a secondary care gender clinic (N = 540) $Median_{age}$ = 27; range = 16-72	ASD diagnosis: Retrospective audit of electronic medical records	4.8% of the sample had a diagnosis of ASD
Chiniara et al. (2018)	GD/gender incongruence	Retrospective chart review	Demographic data, clinical characteristic, & mental health	Adolescent	Adolescents presenting to a transgender clinic (N = 203) AMAB (n = 47) M_{age} = 16.1; SD = 1.70 AFAB (n = 156) M_{age} = 16.3; SD = 1.63	Self-reported ASD diagnosis: Patient charts	5.40% of the sample reported a diagnosis of ASD

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Quantitative Studies that Report Data on the Overlap Between ASD and Gender Dysphoria/Incongruence

Authors (year)	Targeted population	Design	Focus	Age group	Sample	Relevant measures	Relevant findings
Cooper et al. (2018)	ASD	Case-control	Gender identity & social affiliation with gender groups	Adolescent & adult	Age range = 16-80 years ASD female (n = 101) M_{age} = 30.38; SD = 12.40 ASD male (n = 118) M_{age} = 33.2; SD = 12.53 NT female (n =153) M_{age} = 35.88; SD = 11.50 NT male (n = 114) M_{age} = 32.02; SD = 13.0	Single item measure of gender identity & gender transition	ASD participants were significantly more likely to be gender incongruent than NT participants ASD participants were significantly more likely to have or be planning a gender transition than NT participants
Fielding & Bass (2018)	GD/gender incongruence	Case note review	Pattern of referrals & characteristics	Adult	Individuals requested treatment for GD referred to clinician (N = 153) Referral period 2011-2013 M_{age} = 34.37; SD = 14.95 Referral period 2014-2016 M_{age} = 28.70; SD = 13.64	ASD diagnosis: Case notes	7.8% of the sample had a diagnosis of ASD
George & Stokes (2018b)	General & ASD	Case-control	Gender identity, gender dysphoric traits, & sexual orientation	Adult	ASD (n = 310) M_{age} = 31.01; SD = 11.37 NT (n = 261) M_{age} = 30.20; SD = 11.92	GIDYQ-AA, AQ-50, and self-reported gender identity and hormone replacement related to gender identity	ASD participants reported significantly more gender dysphoric traits than NT participants and a more diverse range of gender identities. They were also more likely to use hormone replacement therapy The rate of transgender people was 3.92% in the ASD sample and 1.92% in the NT sample Positive association between ASD traits and gender dysphoric feelings in NT adults who scored below 32 on AQ

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Quantitative Studies that Report Data on the Overlap Between ASD and Gender Dysphoria/Incongruence

Authors (year)	Targeted population	Design	Focus	Age group	Sample	Relevant measures	Relevant findings
Heylens et al. (2018)	GD/gender incongruence	Cross-sectional/ Retrospective chart review	Co-occurrence of ASD and GD	Adult	Patients consulted a gender clinic diagnosed with GD (n = 63) AMAB (n = 33) M_{age} = 31.3; SD = 14.7 AFAB (n = 30) M_{age} = 22.7; SD = 6.5 Files of patients diagnosed with GD or GID (n = 532) Normative (Constantino et al., 2012; n = 1,449)	Screening tools: SRS-A & AQ-50 (cut-off scores & difference between group means) ASD diagnosis: Medical records	27.11% of the patients scored > 60 on SRS-A indicating mild/moderate to severe difficulties in social responsiveness The GD group scored significantly higher on SRS-A, compared to the norm group 4.84% of GD scored above the 32 AQ cut-off point 6.02% of GD had a "certain" diagnosis of ASD
Nobili. Glazebrook, Bouman, et al. (2018)	GD/gender incongruence	Case-control	ASD traits	Adult	Transgender from a national transgender healthcare service (n = 661) M_{age} = 28.25; SD = 12.25 Cisgender (n = 656) M_{age} = 28.25; SD = 12.25	Screening tool: AQ-28 (cut-off scores & difference between group means)	33.2% of the cisgender group scored at or above 70, indicating possible ASC caseness, compared to 36.3% of the transgender group The transgender group scored significantly lower on AQ than the cisgender group
van der Miesen, de Vries, et al. (2018)	GD/gender incongruence	Case-control	ASD symptoms	Child & adolescent	Diagnosed with GID (n = 490) M_{age} = 11.1; SD = 3.73 NT (Hartman et al. 2006, 2015; n = 2,507) M_{age} = 10.1; SD = 3.73 ASD (Hartman et al. 2006, 2015; n = 196) M_{age} = 10.8; SD = 3.08	Screening tool: CSBQ (cut-off scores & difference between group means)	14.5% of the GD group had a threshold score of 38 or higher, potentially suggestive of an ASD diagnosis, compared to 3.5% in the NT sample The GD group showed elevated levels of autistic symptomatology compared to the NT group

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Quantitative Studies that Report Data on the Overlap Between ASD and Gender Dysphoria/Incongruence

Authors (year)	Targeted population	Design	Focus	Age group	Sample	Relevant measures	Relevant findings
van der Miesen, Hurley, et al. (2018)	ASD	Case-control	Gender variance	Adolescent & adult	ASD adolescent (n = 573) M_{age} = 15.98; SD = 1.85 ASD adult (n = 807) M_{age} = 32.14; SD = 12.86 YSR Nonreferred adolescent (Verhulst et al., 1997; n = 1,016) ASR Nonreferred adult (Achenbach, & Rescorla, 2003; n = 846) M_{age} = 29.9; SD = 9.5	YSR/ASR item 110: Wish to be the other binary gender	ASD adolescents were 2.12 times more likely to endorse the item, compared to nonreferred adolescents ASD adults were 2.46 times more likely to endorse the item, compared to nonreferred adults
Vermaat et al. (2018)	GD/gender incongruence	Case-control	ASD symptoms	Adult	Referred to a centre for GD (n = 326) M_{age} = 30.20; SD = 11.57 NT adults (Baron-Cohen et al., 2001; n = 174) M_{age} = 37.0; SD = 7.7 NT students (Baron-Cohen et al., 2001; n = 840) M_{age} = 21.0; SD = 2.9 Dutch validation study NT adults (Hoekstra et al., 2008; n = 302) M_{age} = 35.68; SD = 6.33	Screening tool: AQ-50 (cut-off scores & difference between group means)	2.1% of the sample referred for GD scored above the cut-off of 32 and 9.5% above the cut-off of 26. In comparison, 2.3% of the NT adults scored above the cut-off of 32 and 8.0% above 26, and 2.95% of the NT students scored above the cut-off of 32 and 11.85% above 26 For the Dutch total AQ score, 1.2% scored above the cut-off of 145 The mean AQ score in individuals referred for GD was similar to the NT samples
Walsh et al. (2018)	ASD	Cross-sectional analysis	Gender identity, ASD traits, & sensory differences	Adolescent & adult	ASD (N = 669) M_{age} = 44.67; SD = 12.63; range = 15.92-80.14	Single item measure of gender identity	15% of autistic people reported trans/nonbinary identities

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Quantitative Studies that Report Data on the Overlap Between ASD and Gender Dysphoria/Incongruence

Authors (year)	Targeted population	Design	Focus	Age group	Sample	Relevant measures	Relevant findings
Nabbijohn et al. (2019)	General & ASD	Case-control	Gender variance	Child	Age range = 6-12 NT (*n* = 2,004) M_{age} = 8.7; *SD* = 2.0 Clinical (*n* = 441) M_{age} = 9.4; *SD* = 1.9	GIQC/CSBQ	Positive association between characteristics of ASD and GV in the nonclinical subgroup of children GV was associated with parent-reported clinical diagnoses of ASD, SPD, and ODD. ASD, SPD, and ODD showed significantly higher levels of parent-reported GV than nonclinical children
Hisle-Gorman et al. (2019)	ASD	Retrospective case-cohort	GD	Child & adolescent	Age range = 2-18; M_{age} = 8.83; *SD* = 3.44 99.8% were matched on date of birth ASD (*n* = 48,762) NT (*n* = 243,810)	Health care records ICD-9-CM codes for gender identity disorder or transsexualism	Children with ASD (GD = 0.07%) were over four times more likely to have a diagnosis of GD compared to children without ASD (GD = 0.01%)
Leef et al. (2019)	GD/gender incongruence	Case-control	ASD traits	Child	Referred to gender identity service (*n* = 61) M_{age} = 7.97; *SD* = 2.46; range = 4.08-12.95 Clinic-referred (*n* = 40) M_{age} = 9.48; *SD* = 1.81; range = 6.49-12.93	Screening tools: SRS/SCQ (cut-off scores & difference between group means) ASD diagnosis (PDD in DSM–IV–TR or ASD in DSM–5): Medical records	21.7% of the gender-referred group had a clinical range score on the SCQ, compared to 2.5% of the clinically referred group. No significant differences were found between groups on the SRS The gender-referred group scored significantly higher than the clinically referred group on SCQ, but their scores on SRS groups were similar 21.3% of the gender-referred group had a diagnosis of ASD, compared to 0% of the clinically referred group

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Quantitative Studies that Report Data on the Overlap Between ASD and Gender Dysphoria/Incongruence

Authors (year)	Targeted population	Design	Focus	Age group	Sample	Relevant measures	Relevant findings
Mahfouda et al. (2019)	GD/gender incongruence	Retrospective chart review	Mental health correlates of ASD	Child & adolescent	Children/adolescents attending a service for gender diversity (N = 104) M_{age} = 14.62; SD = 1.72	Screening tool SRS (cut-off scores) Self-reported ASD diagnosis	Based on the SRS-2 DSM-5 subscale, 22.1% of the sample fell in the 'severe' range (indicated ASD) 9.62% of the sample reported a diagnosis of ASD
Stagg & Vincent (2019)	GD/gender incongruence	Cross-sectional	ASD traits	Adult	Online transgender/ nonbinary (n = 109) Transgender male M_{age} = 24; SD = 8 Transgender female M_{age} = 31; SD = 14 Nonbinary (AMAB) M_{age} = 29; SD = 14 Nonbinary (AFAB) M_{age} = 25; SD = 7 Online Cisgender (n = 68) Cis-male M_{age} = 32; SD = 16 Cis-female M_{age} = 23; SD = 7	Screening tool: AQ-50 (cut-off scores & difference between group means) Self-reported ASD diagnosis	28% of the transgender and nonbinary group met the AQ cut-off score and none of the cisgender participants met the AQ cut-off score of 32 Both transgender and nonbinary groups scored significantly higher on AQ compared to the cisgender group 14% of the transgender and nonbinary group and 4% of the cisgender group reported an ASD diagnosis
Kallitsounaki & Williams (2020)	General	Cross-sectional	Link between ASD traits & GD	Adult	General population (N = 101) M_{age} = 36.93; SD = 10.11; range = 22-70	AQ, GIDYQ-AA-AA, & RCGI	Positive association between ASD traits, one the one hand, and current gender dysphoric feelings and recalled childhood gender-typed behaviour, on the other hand

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Quantitative Studies that Report Data on the Overlap Between ASD and Gender Dysphoria/Incongruence

Authors (year)	Targeted population	Design	Focus	Age group	Sample	Relevant measures	Relevant findings
Pecora et al. (2020)	ASD	Case-control	Gender identity, sexual orientation & adverse sexual experiences in autistic females	Adult	Autistic females (n = 134) M_{age} = 26.2; SD = 8.7 Non-autistic females (n = 161) M_{age} = 22.0 years, SD = 4.6	Single item measure of gender identity	Autistic females (19.40%) were more likely to be gender incongruent than non-autistic females (8.70%)
Kung (2020)	GD/gender incongruence	Case-control	ASD traits, systemizing, empathizing, & theory of mind	Adult	Online transgender/nonbinary (n = 323) M_{age} = 35.83; SD = 15.69; range = 18-76 NT controls (Baron-Cohen et al., 2014; n = 3,906) NT controls (Ruzich, Allison, Chakrabarti, et al., 2015; n = 450,394)	Screening tool: AQ-50 (difference between group means	Transgender men > control females Nonbinary AFAB > control females Transgender women = control males Nonbinary AMAB = control males
Lehmann et al. (2020)	GD/gender incongruence	Cross-sectional	ASD traits	Adolescent & adult	People attending or previously attended specialist gender services (N = 123)	Screening tools: AQ-50/RAADS-14 (cut-off scores)	Based on AQ, the prevalence of ASD traits was 19.5%, while using the RAADS-14, the prevalence was 25.4%

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Quantitative Studies that Report Data on the Overlap Between ASD and Gender Dysphoria/Incongruence

Authors (year)	Targeted population	Design	Focus	Age group	Sample	Relevant measures	Relevant findings
Murphy et al. (2020)	GD/gender incongruence	Case-control	ASD & transgender identity	Adult	Online transgender (n = 124) M_{age} = 27.31; SD = 10.77 Online cisgender (n = 603) M_{age} = 28.30; SD = 11.44	Screening tool: AQ-50 (cut-off scores & difference between group means) Self-reported ASD diagnosis	In the cisgender group, 10.3% met cut-off (score > 32), in comparison to 40.3% in the transgender group. AQ scores were significantly higher in trans men, compared to cis women and cis men Trans women showed similar AQ scores to cis men and cis women. 20.2% of the transgender group and 6.1% of the cisgender group reported a diagnosis of ASD
Nobili et al. (2020)	GD/gender incongruence	Longitudinal	Stability of ASD traits	Adult	Transgender assessed at a transgender health service (N = 118) M_{age} = 27.95; SD = 13.11	Screening tool: AQ-28 (cut-off scores)	34.7% scored above the cut-off (>70) indicating clinically significant levels of ASD traits
Warrier et al. (2020)	GD/gender incongruence	Case control	Rates of ASD, other neurodevelopmental and psychiatric diagnoses, & ASD traits	Adolescent & adult	C4 age range 15-90: ASD cisgender (n = 27,251); NT cisgender (n = 484,038); ASD transgender/gender diverse (n = 668); NT transgender /gender diverse (n = 2,143) MU age range 18-88: ASD cisgender (n = 1,031); NT cisgender (n = 83,950) ASD transgender/gender diverse (n = 55); NT transgender/gender diverse (n = 634) IMAGE: ASD cisgender (n = 330); NT cisgender (n = 1,411); ASD transgender/gender diverse (n = 36) NT transgender/gender diverse (n = 26)	C4 Screening tool: AQ-10 Self-reported ASD diagnosis MU Self-reported ASD diagnosis IMAGE Screening tool: AQ-50 Self-reported and verified ASD diagnosis APHS Self-reported ASD diagnosis	In the C4, IMAGE, and LifeLines datasets, transgender and gender-diverse people scored significantly higher than cisgender males and females on the AQ Transgender and gender-diverse individuals had, on average, higher rates of ASD diagnoses than cisgender people

					APHS age range 16-90: ASD cisgender (*n* = 949); NT cisgender (*n* = 1,200); ASD transgender/gender diverse (*n* = 133); NT transgender/gender diverse (*n* = 29) LifeLines age > 18: ASD cisgender (*n* = 436); NT cisgender (*n* = 37,030); ASD transgender/gender diverse (*n* = 3); NT transgender/gender diverse (*n* = 50)	Life-Lines Screening tool: AQ-10 Self-reported ASD diagnosis	
Kallitsounaki et al. (2021)	General	Cross-sectional	Link between ASD traits & GD	Adult	General population (*N* = 126) M_{age} = 20.99; *SD* = 4.10; range = 18-45	AQ, GIDYQ-AA-AA, RCGI	Positive association between ASD traits, one the one hand, and current gender dysphoric feelings and recalled childhood gender-typed behaviour, on the other hand.

Note. AFAB = assigned female at birth; AQ = Autism-Spectrum Quotient; AMAB = assigned male at birth; ASC = autism spectrum conditions; ASD = autism spectrum disorder; ASDS = Asperger Syndrome Diagnostic Scale; ASR = Adult Self-Report; CBCL = Child Behaviour Checklist; CSBQ = Children's Social Behaviour Questionnaire; DISCO-10 = Diagnostic Interview for Social and Communication Disorders-10th revision; GD = gender dysphoria; GID = gender identity disorder; GID-NOS = gender identity disorder not otherwise specified; GIDYQ-AA = Gender Identity/Gender Dysphoria Questionnaire for Adolescents and Adults; GIQC = Gender Identity Questionnaire for Children; NF1 = neurofibromatosis 1; ODD = oppositional defiant disorder; PDD = pervasive developmental disorder; RAADS-14 = The Ritvo Autism Asperger Diagnostic Scale; RCGI = Recalled Childhood Gender Identity/Gender Role Questionnaire; SCQ = Social Communication Questionnaire; SPD = Sensory processing disorder; SRS (-A) = Social Responsiveness Scale (Adult Version); NT = neurotypical; TRF = Teacher's Report Form; YSR = Youth Self-Report.

Table A3

Overview of Characteristics of Studies Containing Data on the Prevalence of ASD Diagnoses in Gender Dysphoric/Incongruent Individuals

Author (year)	Gender dysphoric/incongruent (*N*)	Number of people with ASD	Mean age	Study design	% AMAB	Participants
de Vries et al. (2010)	204	16	10.8	Clinical	56.4	Gender-referred
Spack et al. (2012)	97	5	14.8	Clinical	44.3	Diagnosis GD/GID/GID-NOS
Khatchadourian et al. (2014)	84	6	16.6	Clinical	46.4	Diagnosis GD/GID/GID-NOS
Skagerberg et al. (2015)	166	20	14.3	Clinical	37.3	Gender-referred
Chen et al. (2016)	38	5	14.4	Clinical	42.1	Gender -referred
Holt et al. (2016)	218	29	14.0	Clinical	37.2	Gender -referred
Peterson et al. (2017)	96	3	17.1	Clinical	n.s.[c]	Diagnosis GD/GID/GID-NOS
Shumer et a. (2016)	39	4	15.8	Clinical	56.4	Gender -referred
Nahata et al. (2017)	79	5	13.5[a]	Clinical	35.4	Diagnosis GD/GID/GID-NOS
Becerra-Culqui et al. (2018)	1,333	63	10.0[a]	Population	44.1	Diagnosis GD/GID/GID-NOS
Chiniara et al. (2018)	203	11	16.3[b]	Clinical	23.2	Gender -referred
Leef et al. (2019)	61	13	8.0	Clinical	73.8	Diagnosis GD/GID/GID-NOS
Mahfouda et al. (2019)	104	10	14.6	Clinical	24.0	Gender -referred
Jones et al. (2012)	259	7	42.5[b]	Clinical & population	76.4	Gender incongruent
Kristensen & Broome (2015)	446	62	46.5[a]	Population	n.s.[c]	Gender incongruent
Cheung et al. (2018)	540	26	44.0[a]	Clinical	n.s.[c]	Gender -referred
Fielding & Bass (2018)	153	12	30.7[b]	Clinical	63.4	Gender -referred
Heylens et al. (2018)	532	32	n.s.	Clinical	66.0	Diagnosis GD/GID/GID-NOS
Stagg & Vincent (2019)	109	15	26.5[b]	Population	34.9	Gender incongruent

Author (year)	Gender dysphoric/incongruent (*N*)	Number of people with ASD	Mean age	Study design	% AMAB	Participants
Murphy et al. (2020)	124	25	27.3	Population	38.7	Gender incongruent
Warrier et al. (2020) C4	2,811	668	25.4	Population	n.s.[c]	Gender incongruent
Warrier et al. (2020) MU	689	55	22.4	Population	n.s.[c]	Gender incongruent
Warrier et al. (2020) IMAGE	62	36	29.7	Population	n.s.[c]	Gender incongruent
Warrier et al. (2020) APHS	162	133	35.2	Population	n.s.[c]	Gender incongruent
Warrier et al. (2020) LifeLines	53	3	47.9	Population	n.s.[c]	Gender incongruent

Note. All the studies included in the table were meta-analysed; GD = gender dysphoria; GID = gender identity disorder; GID-NOS = gender identity disorder not otherwise specified; AMAB = assigned male at birth; n.s. = not specified; Gender-referred = participants referred to clinics/services for gender-related issues (mainly gender dysphoria).

[a] Mean age was calculated by taking a midpoint between the minimum and maximum of the age range. [b] Combined mean age was calculated from the data provided by the authors. [c] Information about birth-assigned sex was not reported for the total number of participants who took part in the study.

Table A4

Overview of Characteristics of Studies Containing Data on the Prevalence of ASD Traits in Gender Dysphoric/Incongruent Individuals

Author (year)	Study design	Number of participants per group	Age group	Control group	Mean scores (*SD*)	Direction of effect (Hedges'*g*)
Skagerberg et al. (2015)	Clinical	n_{case} = 166 $n_{control}$ = 500	Child & adolescent	Secondary	M_{case} = 58.51 (37.58)[a] $M_{control}$ = 29.8 (16.7)	Cases > Controls (1.21)
Akgül et al. (2018)	Clinical	n_{case} = 25 $n_{control}$ = 50	Child & adolescent	Primary	M_{case} = 70.36 (16.72) $M_{control}$ = 49.78 (16.95)	Cases > Controls (1.22)
van der Miesen, de Vries, et al. (2018)	Clinical	n_{case} = 490 $n_{control}$ = 2,507	Child & adolescent	Secondary	M_{case} = 20.58 (15.71) $M_{control}$ = 11.69 (11.49)	Cases > Controls (0.72)
Jones et al. (2012)	Clinical & population	n_{case} = 259 $n_{control}$ = 174	Adult	Secondary	M_{case} = 18.15 (7.97)[a] $M_{control}$ = 16.4 (6.3)	Cases > Control (0.23)
Heylens et al. (2018)	Clinical	n_{case} = 58 $n_{control}$ = 1,449	Adult	Secondary	M_{case} = 52.53 (22.48) $M_{control}$ = 36.74 (22.66)	Cases > Controls (0.70)
Nobili, Glazebrook, Bouman, et al. (2018)	Clinical	n_{case} = 656 $n_{control}$ = 656	Adult	Primary	M_{case} = 65.77 (11.81) $M_{control}$ = 66.88 (8.48)	Cases < Controls (-0.11)
Stagg & Vincent (2019)	Population	n_{case} = 109 $n_{control}$ = 68	Adult	Primary	M_{case} = 28.72 (10.06)[a] $M_{control}$ = 18.41 (7.39)[a]	Cases > Controls (1.12)
Vermaat et al. (2018)	Clinical	n_{case} = 326 $n_{control}$ = 840	Adult	Secondary	M_{case} = 16.79 (6.96) $M_{control}$ = 17.6 (6.4)	Cases < Controls (-0.12)[b]
Murphy et al. (2020)	Population	n_{case} = 124 $n_{control}$ = 603	Adult	Primary	M_{case} = 28.04 (11.37)[a] $M_{control}$ = 19.70 (8.96)[a]	Cases > Controls (0.89)

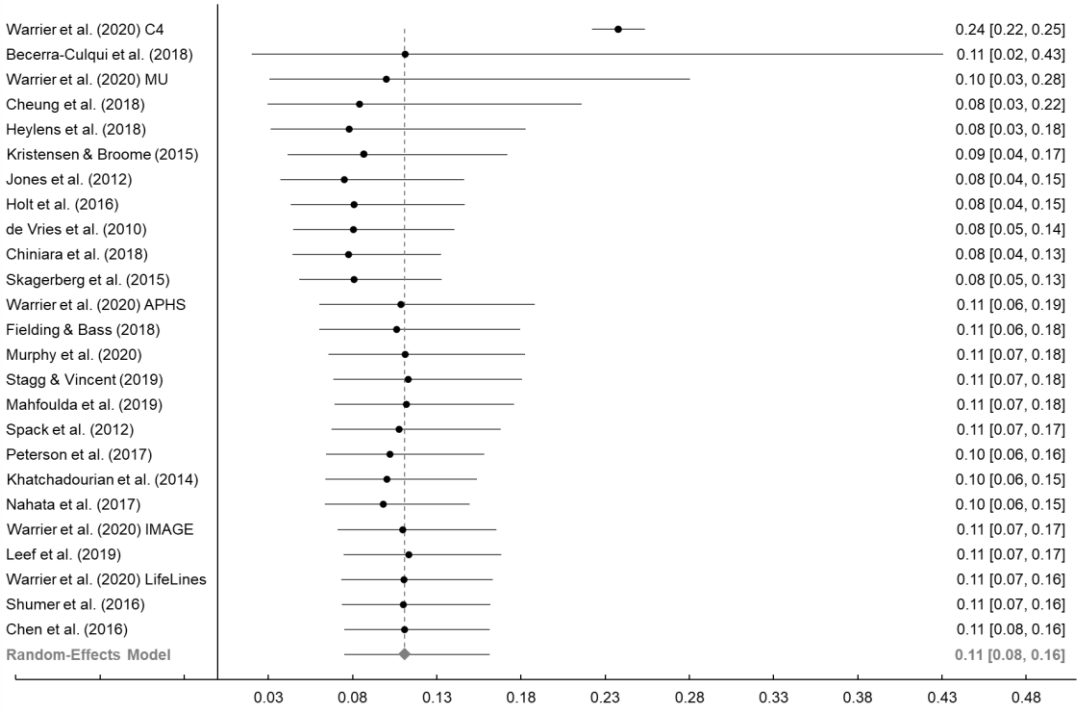
Author (year)	Study design	Number of participants per group	Age group	Control group	Mean scores (SD)	Direction of effect (Hedges'g)
Kung (2020)	Population	n_{case} = 308 $n_{control}$ = 3,906	Adult	Secondary	M_{case} = 23.20 (8.50)[a] $M_{control}$ = 18.20 (7.82)[a]	Cases > Controls (0.64)
Warrier et al. (2020) C4	Population	n_{case} = 2,143 $n_{control}$ = 484,038	Adult	Primary	M_{case} = 5.56 (2.69) $M_{control}$ = 3.32 (2.26)[a]	Cases > Controls (0.99)

Note. All studies included in the table were meta-analysed.

[a] Means and *SD*s were calculated from the data reported by the authors in the original publications. [b] Vermaat et al. (2019) reported that the difference between the sample referred for GD and the comparison group of NT students in AQ score was d = -0.28. When we calculated this effect from the means and *SD*s provided by the authors in the original publication, we found that the d was -0.12.

Figure A1

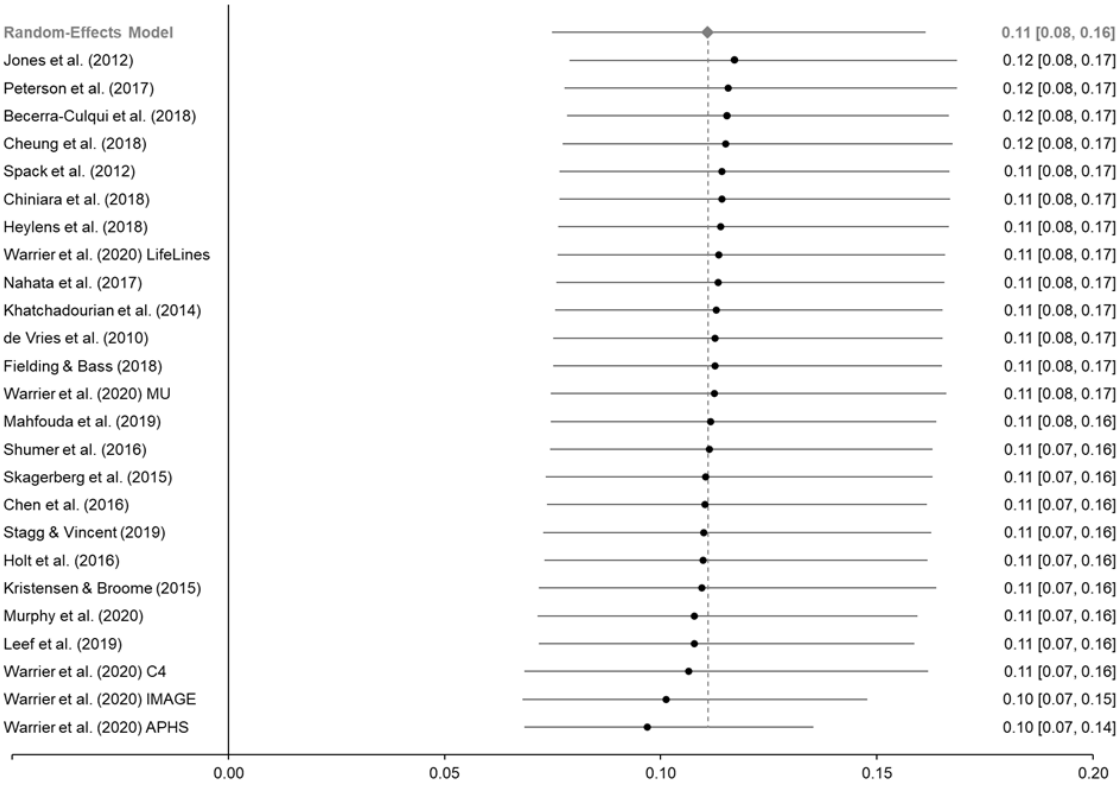
Cumulative Forest Plot of Studies Included in ASD Diagnosis Meta-Analysis



Note. Each row shows the pooled prevalence estimate of ASD diagnoses in gender dysphoric/incongruent people and its 95% confidence interval when that row's study was included in the meta-analysis. The grey diamond marks the pooled prevalence estimate and its 95% confidence interval when all studies were included in the meta-analysis.

Figure A2

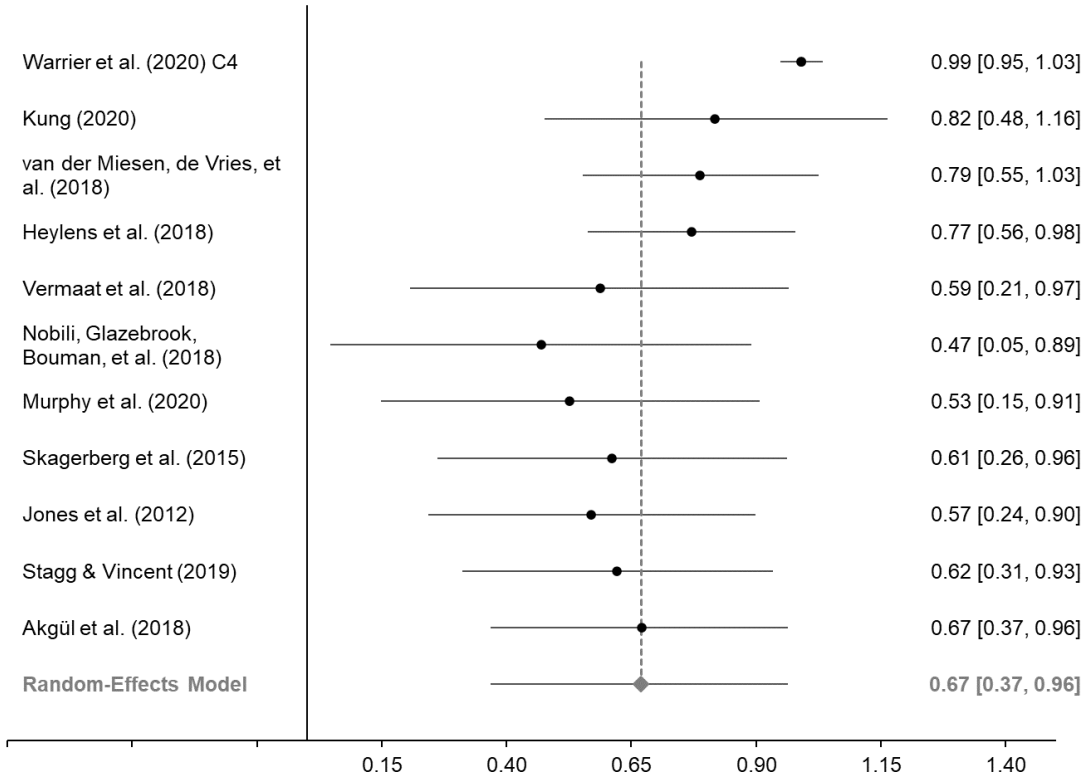
Forest Plot of Pooled Estimate Prevalence Estimates of ASD Diagnoses After One Study Removed



Note. Each row shows the pooled prevalence estimate of ASD diagnoses in gender dysphoric/incongruent people and its 95% confidence interval when that row's study was removed from the meta-analysis. The grey diamond marks the pooled prevalence estimate and its 95% confidence interval when all studies were included in the meta-analysis.

Figure A3

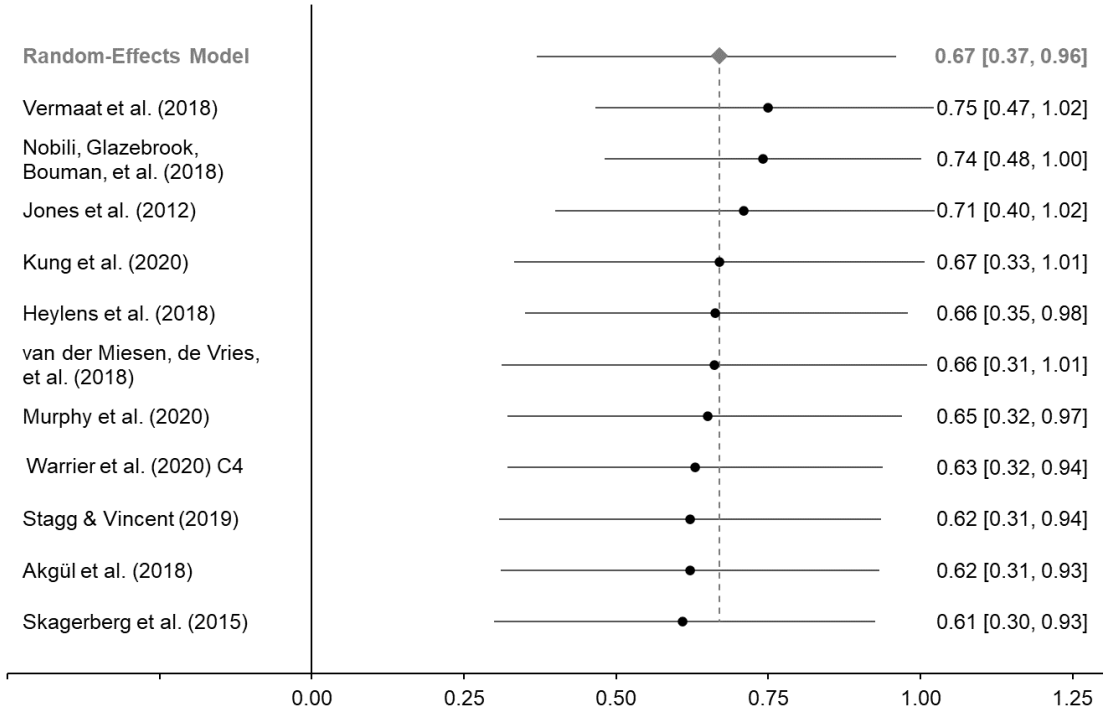
Cumulative Forest Plot of Studies Included in ASD Traits Meta-Analysis



Note. Each row shows the overall weighted effect size of the difference in the number of reported ASD traits between gender dysphoric/incongruent and neurotypical/population-based participants and its 95% confidence interval when that row's study was included in the meta-analysis. The grey diamond marks the pooled prevalence estimate and its 95% confidence interval when all studies were included in the meta-analysis.

Figure A4

Forest Plot of Hedges' g After One Study Removed



Note. Each row shows the overall weighted effect size of the difference in the number of reported ASD traits between gender dysphoric/incongruent and neurotypical/population-based participants and its 95% confidence interval when that row's study was removed from the meta-analysis. All *p* values were < .001. The grey diamond marks the mean weighted effect and its 95% confidence interval when all studies were included in the meta-analysis.

Appendix B: Supplemental Material for Chapter 3

Statistical Analyses Excluding Autistic Participants

When participants who reported possession of a formal diagnosis of ASD (n = 13) were excluded from the analysis, results did not change substantively except in one analysis. Just as in the full sample, AQ remained negatively and significantly correlated with both GIDYQ-AA, r = -.26, p = .017 and RCGI, r = -.30, p = .005, after the exclusion of these 13 participants. Moreover, RMIE remained positively and strongly associated with GIDYQ-AA, r = .59, p < .001. The interaction between AQ and RMIE predicted significantly GIDYQ-AA, b = .003, $t(84)$ = 2.38, p = .019. Simple slopes analysis showed that when performance on RMIE was low (-1SD), AQ score predicted negatively and significantly GIDYQ-AA, b = -0.04, $t(84)$ = -3.30, p = .001, whereas, when it was high (+1SD), AQ score did not predict GIDYQ-AA, b = -0.003, $t(84)$ = -0.31, p = .761.

The *only* result that changed substantively (i.e., from significant to nonsignificant) in this reduced sample of 88 participants was the RMIE × RCGI correlation. In the full sample, the correlation was moderate and statistically significant, whereas in the reduced sample it was small and nonsignificant, r = .14, p = .201. A power analysis using G*Power 3.1.9.2 indicated that to detect an association of .33 between RMIE and RGIQ on 80% of occasions (as recommended by Cohen, 1992), using two-tailed tests, 69 participants were required. Arguably, it seems likely that the correlation we found between RMIE and RGIQ in the full sample was inflated by the inclusion of autistic participants.

Correlation Analyses Between AQ Subscales, GIDYQ-AA, and RCGI

An anonymous reviewer of the published version of Chapter 3 made a constructive suggestion that we conduct additional analyses to explore the relation between current gender dysphoric traits and recalled gender-typed behaviour, on the one hand, and each of the individual AQ subscales, on the other hand. We have done this and reported the results in Table B1, below. However, we think it is important to make clear that these analyses are entirely post hoc, and we did not have any predictions about the results. Exploratory analyses that are not based on a specific prediction (following from a sound theory) risk contributing to an inflation bias (i.e., selective reporting/*p*-hacking; e.g., John et al., 2012; Kühberger et al., 2014; Masicampo & Lalande, 2012) in the field. If researchers regularly analyse variables they never intended to analyse, it will inevitably lead to the publication of false positives. In turn, if other researchers then make predictions based on results that are (conceptually and statistically) likely to reflect Type II errors, then the field becomes (further) biased toward the search for and belief in positive results. Therefore, we urge readers to be cautious when interpreting the results of these supplementary analyses. That being said, George and Stokes (2018b) did analyse the relation between GIDYQ-AA score and each subscale of the AQ (although it is not clear the analyses were based on specific hypotheses) and found that all of them were significantly associated with gender dysphoric feeling. The current results replicate the significant association between AQ communication subscale and GIDYQ-AA (note that partialling out the effect of AQ communication subscale, the GIDYQ-AA × AQ imagination subscale lost its significance, $r = -.14$, $p = .154$). This suggests that there *may* be a reliable connection between gender dysphoric feelings and communication ASD features, specifically.

Table B1

Bivariate Correlations among AQ subscales, GIDYQ-AA, and RCGI

Variable	1	2	3	4	5	6	7
1. AQ social skills	-						
2. AQ attention switching	.45***	-					
3. AQ attention to detail	.05	.16	-				
4. AQ communication	.64***	.44***	.21*	-			
5. AQ imagination	.29**	.09	-.05	.39***	-		
6. GIDYQ-AA	-.04	-.10	-.12	-.50***	-.31**	-	
7. RCGI	-.28**	-.29**	-.08	-.35**	-.04	.53***	-

Note. $N = 101$; AQ = Autism-Spectrum Quotient; GIDYQ-AA = Gender Identity/Gender Dysphoria Questionnaire for Adolescents and Adults (low scores = more gender dysphoric feelings); RCGI = The Recalled Childhood Gender Identity/Gender Role Questionnaire (low scores = less recalled gender-typed behaviour from childhood).

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

Appendix C: Supplemental Material for Chapter 4

Statistical Analyses Excluding Autistic Participants

When participants who reported possession of a formal diagnosis of ASD (n = 2) were excluded from the analysis, results did not change substantively (i.e., for significant to nonsignificant and vice versa). Just as in the full sample, AQ remained negatively and significantly correlated with both GIDYQ-AA, r = -.26, p = .004, BF_{10} = 6.46 and RCGI, r = -.35, p < .001, BF_{10} > 100, after the exclusion of these two participants. Moreover, RMIE remained positively and strongly associated with GIDYQ-AA, r = .49, p < .001, BF_{10} > 100, and negatively associated with AQ, r = -.28, p = .002, BF_{10} = 12.50. A mediation analysis was also conducted. Just as in the full sample, the indirect effect was significantly different from zero, b = -.01, SE = .01, 95% CI [-.02, -.001], indicating that mentalising mediated the significant relation between ASD traits and current gender dysphoric feelings. While the total effect between ASD traits and gender dysphoric feelings was negative and significant, b = -.02, SE = .01, p = .004, the direct effect between these variables was nonsignificant, b = -.01, SE = .01, p = .118.

Table C1

Comparison Between Replication and Original Sample (Kallitsounaki & Williams, 2020)

Characteristic	Current study (N = 126)	Original study (N = 101)	Comparison
Age in years	M = 20.99 (SD = 4.10)	M = 36.93 (SD = 10.11)	$t(126.38) = -14.89$, $p < .001$
Birth-assigned sex	Male: 23% Female: 77%	Male: 50.50% Female: 49.50%	$\chi^2 = 18.55$, $p < .001$
Native English speakers	76.20%	94.10%	$\chi^2 = 13.42$, $p < .001$
Autistic participants	1.60%	12.90%	$\chi^2 = 11.57$, $p = .001$

Appendix D: Supplemental Material for Chapter 5

Experiment 1

Statistical Analyses Excluding Autistic Participants

Bivariate Correlations.

When participants who reported possession of a formal diagnosis of ASD (n = 13) were excluded from the analysis results did not change substantively (i.e., from significant to nonsignificant and vice versa). AQ remained negatively and significantly correlated with PAQ femininity and PAQ masculinity scale scores, r = -.44, p < .001 (one-tailed) and r = -.35, p < .001 (one-tailed), respectively. Moreover, the correlation between AQ and *D* scores remained negative and significant, r = -.23, p = .014 (one-tailed).

Case-Control Analyses.

We also examined differences between a low-AQ group and a high-AQ group in the size of explicit and implicit gender self-concept. Participants who scored below the median score on the AQ (i.e., *Mdn* = 19.00) were assigned in the low AQ-group (n = 46), whereas participants who scored above the median were categorised in the high-AQ group (n = 42). The average femininity score was 3.10 (*SD* = 0.57) in the low-AQ group and 2.63 (*SD* = 0.83) in the high-AQ group, a difference that remained significant, $t(86) = 3.10$, p = .002 (one-tailed), d = 0.66. The average masculinity score was 2.72 (*SD* = 0.64) in the low-AQ group and 2.18 (*SD* = 0.80) in the high-AQ group, a difference that remained significant, $t(86) = 3.49$, p = .001 (one-tailed), d = 0.74. The average *D* score was 0.48 (*SD* = 0.38) in the low-AQ group and 0.31 (*SD* = 0.23) in the high-AQ group, a difference that remained significant, $t(75.46) = 2.62$, p = .006 (one-tailed), d = 0.55.

Alternative Way of Splitting Participants: AQ-50 Clinical Cut-Off Score

When we used the AQ threshold (i.e., the clinical cut-off score) of 26 to split participants in a high-AQ group (n = 21) and in a low-AQ group (n = 81), results remained essentially the same. The between-group difference in PAQ femininity scale score remained significant, with the high-AQ group (M = 2.29; SD = 0.70) scoring lower than the low-AQ group (M = 3.00; SD = 0.66), $t(99) = 4.33$, $p < .001$ (one-tailed), d = 1.04. Likewise, people in the high-AQ group (M = 2.09; SD = 0.66) scored significantly lower on PAQ masculinity scale than people in the low AQ group (M = 2.52; SD = 0.73), $t(99) = 2.47$, $p = .008$ (one-tailed), d = 0.63. In terms of the implicit measure of gender self-concept, the average D score (M = 0.29; SD = 0.26) in the high-AQ group was lower, relative to the average D score in the low-AQ group (M = 0.41; SD = 0.33) and that difference was marginally significant, $t(99) = 1.56$, $p = .061$ (one-tailed), d = 0.41.

Experiment 2

Statistical Analyses Excluding Autistic Participants

Bivariate Correlations.

When participants who reported possession of a formal diagnosis of ASD (n = 2) were excluded from the analysis results did not change substantively (i.e., from significant to nonsignificant and vice versa). AQ remained negatively and significantly correlated with PAQ femininity and PAQ masculinity scale scores, r = -.29, $p = .001$, BF_{10} = 35.71 (one-tailed) and r = -.44, $p < .001$, $BF_{10} > 100$ (one-tailed), respectively. Furthermore, the correlation between AQ and D scores remained nonsignificant, r = -.12, $p = .097$, BF_{10} = 0.47 (one-tailed).

Case-Control Analyses.

We also examined differences between a low-AQ group and a high-AQ group in the size of explicit and implicit gender self-concept. Participants who scored below the median score on the AQ (i.e., *Mdn* = 18.00) were assigned in the low AQ-group (*n* = 64), whereas participants who scored above the median were categorised in the high-AQ group (*n* = 60). The average femininity score was 2.98 (*SD* = 0.60) in the low-AQ group and 2.76 (*SD* = 0.65) in the high-AQ group, a difference that remained significant, $t(122) = 1.90$, $p = .030$, $d = 0.34$, $BF_{10} = 1.86$ (one-tailed). The average masculinity score was 2.52 (*SD* = 0.51) in the low-AQ group and 2.05 (*SD* = 0.65) in the high-AQ group, a difference that remained significant, $t(122) = 4.53$, $p < .001$, $d = 0.82$, $BF_{10} > 100$ (one-tailed). The average *D* score was 0.53 (*SD* = 0.35) in the low-AQ group and 0.48 (*SD* = 0.32) in the high-AQ group, a difference that remained nonsignificant, $t(119) = 0.82$, $p = .207$, $d = 0.15$, $BF_{10} = 0.41$ (one-tailed).

Alternative Way of Splitting Participants: AQ-50 Clinical Cut-Off Score

When we used the AQ threshold (i.e., the clinical cut-off score) of 26 to split participants in a high-AQ group (*n* = 14) and in a low-AQ group (*n* = 112), results remained the same. The between-group difference in PAQ femininity scale score remained significant, with the high-AQ group (*M* = 2.46; *SD* = 0.77) scoring lower than the low-AQ group (*M* = 2.92; *SD* = 0.60), $t(124) = 2.64$, $p = .005$, $d = 0.75$, $BF_{10} = 9.78$ (one-tailed). Likewise, people in the high-AQ group (*M* = 1.60; *SD* = 0.64) scored significantly lower on PAQ masculinity scale than people in the low AQ group (*M* = 2.37; *SD* = 0.57), $t(124) = 4.67$, $p < .001$, $d = 1.32$, $BF_{10} > 100$ (one-tailed). In terms of the implicit measure of gender self-concept, the difference in *D* score between the low-

AQ group (M = 0.50; SD = 0.33) and the high-AQ group (M = 0.55; SD = 0.36) was nonsignificant $t(121) = -0.57$, $p = .287$, $d = -0.17$, $BF_{10} = 0.20$ (one-tailed).

Appendix E: Supplemental Material for Chapter 6

Matching Participant Groups for Age and Statistical Analyses

To verify that the between-group differences on the key dependent variables resulted from differences in diagnostic status (autistic/neurotypical) and gender identity (transgender/cisgender), rather than from between-group differences in age, we matched participant groups closely on this variable. Following the recommendation of Mervis and Klein-Tasman (2004), groups were considered matched only if between-group differences in age were not statistically significant (i.e., $p \geq .500$). In order to achieve this matching, we gradually removed the oldest neurotypical cisgender participants and the youngest autistic cisgender participants. Then, we gradually removed the oldest neurotypical transgender participants and the youngest autistic transgender participants, and finally we gradually removed the oldest neurotypical and autistic cisgender participants and the youngest neurotypical and autistic transgender participants, until groups were matched for age (see Table E1). The number of birth-assigned females and males did not differ significantly between groups, χ^2 (3, N = 219) = 5.92, p = .115, φ = .16. As shown below, results remained essentially the same when groups were matched for age.

Table E1

Descriptive and Matching Statistics

Variable	Groups				Comparisons			
	NT cis *n* = 68	ASD cis *n* = 69	NT trans *n* = 52	ASD trans *n* = 30	*t*-tests	*p*	Cohen's *d*	95% CI
	M(*SD*)	*M*(*SD*)	*M*(*SD*)	*M*(*SD*)				
Age	28.84 (5.83)	29.16 (2.84)	28.50 (7.32)	28.93 (6.94)				
					NT cis = NT trans	.779	0.05	[-0.31, 0.41]
					NT cis = ASD cis	.683	-0.07	[-0.41, 0.27]
					NT cis = ASD trans	.944	-0.02	[-0.45, 0.41]
					NT trans = ASD cis	.540	-0.13	[-0.49, 0.24]
					NT trans = ASD trans	.793	-0.06	[-0.51, 0.39]
					ASD cis = ASD trans	.864	0.05	[-0.38, 0.48]

Note. NT = neurotypical; ASD = autism spectrum disorder; Cis = cisgender; Trans = transgender; 95% CI = 95% Confidence Intervals.

Performance on the Explicit Measure of Gender Self-Concept

A 2 (birth-assigned sex: male/female) × 2 (diagnostic category: neurotypical/autistic) × 2 (gender Identity: cisgender/transgender) ANOVA was conducted on participant scores from the explicit measure of gender self-concept. The three-way interaction remained significant, $F(1,211) = 10.51$, $p = .001$, $\eta_p^2 = .05$, and the results of the simple effects analysis did not change substantively (i.e., from significant to nonsignificant and vice versa). Just as in the unmatched sample, the simple effects analysis of birth-assigned sex within gender identity and diagnostic category indicated that the explicit measure of gender self-concept was sensitive to gender identity differences (all *p*s < .001, one-tailed; all $\eta_p^2 \geq .59$).

Next, a simple effects analysis of diagnostic category within birth-assigned sex and gender identity was conducted. Results of the analyses did not change substantively. Just as in the unmatched sample, autistic cisgender birth-assigned males (marginal $M = -3.78$, $SE = 0.27$) scored significantly higher on the task than neurotypical cisgender birth-assigned males (marginal $M = -4.82$, $SE = 0.22$), $F(1, 211) = 8.79$, $p = .001$ (one-tailed), $\eta_p^2 = .04$, and autistic cisgender birth-assigned females (marginal $M = 2.80$, $SE = 0.22$) scored significantly lower than neurotypical cisgender birth-assigned females (marginal $M = 5.01$, $SE = 0.26$), $F(1,211) = 42.97$, $p < .001$ (one-tailed), $\eta_p^2 = .17$. Among transgender participants, autistic birth-assigned males (marginal $M = 4.87$, $SE = 0.36$) scored significantly higher on the explicit task than neurotypical birth-assigned males (marginal $M = 3.85$, $SE = 0.23$), $F(1,211) = 5.30$, $p = .011$ (one-tailed), $\eta_p^2 = .03$, and no difference in the strength of the explicit gender self-concept was observed between autistic (marginal $M = -3.99$, $SE = 0.36$) and neurotypical birth-assigned females (marginal $M = -4.37$, $SE = 0.30$), $F(1,211) = 0.67$, $p = .207$ (one-tailed), $\eta_p^2 = .00$.

Performance on the Implicit Measure of Gender Self-Concept

A 2 (birth-assigned sex: male/female) × 2 (diagnostic category: neurotypical/autistic) × 2 (gender identity: cisgender/transgender) ANOVA was conducted on participant scores from the IAT. The three-way interaction remained nonsignificant, $F(1,184) = 0.99$, $p = .322$, $\eta_p^2 = .01$, and the results of the simple effects analysis did not change substantively. Just as in the unmatched sample, the simple effects analysis of birth-assigned sex within gender identity and diagnostic category indicated that the IAT was sensitive to gender identity differences (all *p*s < .001, one-tailed; all $\eta_p^2 \geq .08$).

Next, a simple effects analysis of diagnostic category within birth-assigned sex and gender identity was conducted. Just as in the unmatched sample, autistic cisgender females (marginal $M = 0.18$, $SE = 0.07$) achieved a significantly lower *D* score on the IAT than neurotypical cisgender females (marginal $M = 0.44$, $SE = 0.08$), $F(1,184) = 6.64$, $p = .006$ (one-tailed), $\eta_p^2 = .04$, and autistic cisgender males (marginal $M = -0.76$, $SE = 0.09$) achieved a significantly lower *D* score than neurotypical cisgender males (marginal $M = -0.48$, $SE = 0.07$), $F(1,184) = 6.87$, $p = .005$ (one-tailed), $\eta_p^2 = .04$. Furthermore, the size of the *D* score did not differ significantly either between autistic (marginal $M = -0.33$, $SE = 0.12$) and neurotypical transgender birth-assigned females (marginal $M = -0.47$, $SE = 0.09$), $F(1,184) = 0.89$, $p = .173$ (one-tailed), $\eta_p^2 = .01$, or between autistic (marginal $M = 0.30$, $SE = 0.12$) and neurotypical transgender birth-assigned males (marginal $M = 0.44$, $SE = 0.08$), $F(1,184) = 0.93$, $p = .168$ (one-tailed), $\eta_p^2 = .01$.

Performance on RMIE and Self-Report Measures

A series of 2 (birth-assigned sex: male/female) × 2 (diagnostic category: neurotypical/autistic) × 2 (gender identity: cisgender/transgender) ANOVAs was

conducted on RMIE, AQ, GIDYQ-AA, RCGI, and TAS-20 scores. Table E2 shows descriptive statistics for participant scores. Just as in the unmatched sample, none of the 3-way interactions were significant (all *p*s ≥ .349, all η_p^2s ≤ .005), and as shown in Table E3, results of the *t*-tests did not change substantively.

Table E2

Participant Characteristics and Mean (Standard Deviation) Performance on RMIE and Self-Report Measures

Groups	RMIE	GIDYQ [a,b]	RCGI [a,c]	AQ	TAS
	M (*SD*)	*M* (*SD*)	*M* (*SD*)	*M* (*SD*)	*M* (*SD*)
NT cis	26.29 (4.61)	4.78 (0.18)	3.93 (0.48)	20.75 (7.64)	46.47 (12.18)
NT trans	27.02 (4.23)	2.17 (0.33)	2.71 (0.61)	24.96 (9.29)	52.94 (12.54)
ASD cis	18.25 (8.20)	4.12 (0.76)	3.50 (0.59)	30.61 (6.76)	61.20 (9.40)
ASD trans	23.73 (5.18)	2.25 (0.49)	2.53 (0.66)	37.50 (6.21)	64.70 (10.52)

Note. NT = neurotypical; ASD = autism spectrum disorder; Cis = cisgender; Trans = transgender; RMIE = Reading the Mind in the Eyes; GIDYQ-AA = Gender Identity/Gender Dysphoria Questionnaire for Adolescents and Adults; RCGI =Recalled Childhood Gender Identity/Gender Role Questionnaire; AQ = Autism-Spectrum Quotient; TAS-20 = Toronto Alexithymia Scale.

[a] One neurotypical cisgender birth-assigned male completed the female version of the GIDYQ-AA and RCGI, and one autistic cisgender birth-assigned female completed the male version of the GIDYQ-AA and RCGI. Hence, their data has not been included in the analysis. [b] Low scores = more gender dysphoria. [c] Low scores = less recalled gender-typed behaviour from childhood.

Table S3

Planned and Post-Hoc Comparisons Between Groups

Measure	*t*-tests	Cohen's *d*	95% CI
GIDYQ-AA			
	NT cis > NT trans ***	10.06	[8.72, 11.39]
	NT cis > ASD cis ***	1.20	[0.83, 1.56]
	NT cis > ASD trans ***	8.22	[6.97, 9.45]
	NT trans < ASD cis ***	-3.19	[-3.73, -2.64]
	NT trans = ASD trans	-0.20	[-0.65, 0.26]
	ASD cis > ASD trans ***	2.73	[2.14, 3.30]
RCGI			
	NT cis > NT trans ***	2.27	[1.81, 2.73]
	NT cis > ASD cis ***	0.81	[0.46, 1.16]
	NT cis > ASD trans ***	2.59	[2.03, 3.15]
	NT trans < ASD cis ***	-1.32	[-1.72, -0.92]
	NT trans = ASD trans	0.29	[-0.16, 0.74]
	ASD cis > ASD trans ***	1.58	[1.10, 2.06]
AQ			
	NT cis < NT trans **	-0.50	[-0.87, -0.13]
	NT cis < ASD cis ***	-1.37	[-1.74, -0.99]
	NT cis < ASD trans ***	-2.31	[-2.85, -1.77]
	NT trans < ASD cis ***	-0.71	[-1.08, -0.34]
	NT trans < ASD trans ***	-1.51	[-2.01, -1.00]
	ASD cis < ASD trans ***	-1.04	[-1.49, -0.59]
RMIE			
	NT cis = NT trans	-0.16	[-0.52, 0.20]
	NT cis > ASD cis ***	1.21	[0.84, 1.57]
	NT cis > ASD trans **	0.54	[0.10, 0.97]
	NT trans > ASD cis ***	1.29	[0.90, 1.69]
	NT trans > ASD trans **	0.72	[0.25, 1.18]
	ASD cis < ASD trans ***	-0.74	[-1.18, -0.30]
TAS-20 [a]			
	NT cis < NT trans *	-0.53	[-0.89, -0.16]
	NT cis < ASD cis ***	-1.36	[-1.73, -0.98]
	NT cis < ASD trans ***	-1.56	[-2.04, -1.07]
	NT trans < ASD cis **	-0.76	[-1.13, -0.39]
	NT trans < ASD trans ***	-0.99	[-1.46, -0.52]
	ASD cis = ASD trans	-0.36	[-0.79, 0.07]

Note. 95% CI = 95% Confidence Intervals.

[a] The analysis of TAS-20 was exploratory, therefore symbols for statistical significance reflect results from two-tailed tests. Tukey HSD correction was applied. In all the other measures, symbols for statistical significance denote results from one-tailed tests, and following the preregistration no corrections have been applied.

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

Preregistered Hypotheses and Analyses not Included in Chapter 6

Block 1.3: Implicit Gender Self-Concept

Hypothesis 1. Among birth-assigned males and females, we expected neurotypical cisgender individuals to show a significantly stronger implicit gender self-concept than autistic cisgender, neurotypical transgender, and autistic transgender people. We also expected autistic cisgender people to show a significantly stronger implicit gender self-concept than both neurotypical and autistic transgender individuals. Lastly, we predicted that neurotypical transgender people would show a significantly stronger implicit gender self-concept than autistic transgender people (neurotypical cisgender > autistic cisgender > neurotypical transgender > autistic transgender).

Analysis.

A 2 (birth-assigned sex: male/female) × 2 (diagnostic category: neurotypical/autistic) × 2 (gender identity: cisgender/transgender) ANOVA was conducted on participant scores from the IAT (note: *D* scores were transformed to positive values so that the higher the score the stronger the implicit gender self-concept, regardless of whether it is male or female. Also, participants with error rate > 20% in the critical blocks of the task were excluded from the analysis). As shown in Table E4, the 3-way interaction was significant. To test our hypotheses, a series of planned *t*-tests was conducted. Results of the analyses are presented in Table E4.

Table E4

Implicit Measure of Gender Self-Concept Analysis of Variance Results and Planned t-Tests

Measure	Effect	*F*	*p*	η_p^2	Planned *t*-tests [a]	Cohen's *d*	95% CI
Implicit	Sex	3.83	.051	.01			
	Gender identity	8.18	.005	.03			
	Diagnostic category	2.77	.097	.01			
	Sex × Gender Identity	1.85	.175	.01			
	Sex × Diagnostic Category	6.04	.015	.02			
	Gender Identity × Diagnostic Category	0.06	.814	.00			
	Sex × Gender Identity × Diagnostic Category	14.22	<.001	.05			
					Birth-Assigned Males		
					NT cis = NT trans	0.08	[-0.36, 0.52]
					NT cis < ASD cis **	-0.50	[-0.91, -0.08]
					NT cis = ASD trans	0.39	[-0.09, 0.87]
					NT trans < ASD cis **	-0.55	[-1.01, -0.09]
					NT trans = ASD trans	0.30	[-0.21, 0.81]
					ASD cis > ASD trans ***	0.84	[0.33, 1.35]
					Birth-Assigned Females		
					NT Cis > NT Trans **	0.69	[0.26, 1.12]
					NT Cis > ASD Cis ***	1.01	[0.58, 1.43]
					NT Cis > ASD Trans **	0.70	[0.18, 1.22]
					NT Trans = ASD Cis	0.32	[-0.10, 0.75]
					NT Trans = ASD Trans	0.01	[-0.51, 0.54]
					ASD Cis = ASD Trans	-0.31	[-0.81, 0.20]

Note. AGAB = birth-assigned sex; NT = neurotypical; ASD = autism spectrum disorder; Cis = cisgender; Trans = transgender; 95% CI = 95% Confidence Intervals.

[a] Symbols for statistical significance denote results from one-tailed tests.

**$p < .01$. ***$p < .001$.

In contrast to predictions, autistic cisgender birth-assigned males (n = 43; M = 0.68; SD = 0.32) achieved a significantly higher score on the IAT than neurotypical cisgender birth-assigned males (n = 50; M = 0.53; SD = 0.29). This indicates a stronger implicit gender self-concept among autistic cisgender birth-assigned males than among neurotypical cisgender birth-assigned males. Also, a nonsignificant difference in the strength of the implicit gender self-concept was found between neurotypical cisgender birth-assigned males and neurotypical transgender birth-assigned males (n = 34; M = 0.51; SD = 0.31), and between neurotypical cisgender and autistic transgender birth-assigned males (n = 26; M = 0.41; SD = 0.32). As predicted, however, autistic cisgender birth-assigned males scored significantly higher on the task than both neurotypical transgender and autistic transgender birth-assigned males. Results indicate a stronger implicit gender self-concept among autistic cisgender birth-assigned males than among neurotypical transgender and autistic transgender birth-assigned males. Contrary to predictions, a nonsignificant difference in the strength of the implicit gender self-concept was found between neurotypical transgender and autistic transgender birth-assigned males.

In keeping with predictions, neurotypical cisgender birth-assigned females (n = 49; M = 0.63; SD = 0.28) scored significantly higher on the task than neurotypical transgender (n = 40; M = 0.44; SD = 0.28), autistic cisgender (n = 48; M = 0.35; SD = 0.29), and autistic transgender birth-assigned females (n = 22; M = 0.44; SD = 0.28), indicating a stronger implicit gender self-concept, compared to the rest of the groups. In contrast to predictions, a nonsignificant difference in the strength of the implicit gender self-concept was observed between autistic cisgender birth-assigned females and either neurotypical transgender or autistic transgender birth-assigned females. Lastly, we did not find a significant difference in the strength of the implicit gender

self-concept between neurotypical transgender and autistic transgender birth-assigned females.

Block 2.3 Explicit Gender Self-Concept

Hypothesis 1. Among birth-assigned males and females, we expected neurotypical individuals (cisgender and transgender) to show a significantly stronger explicit gender self-concept than autistic people (cisgender and transgender). A nonsignificant difference in the strength of explicit gender self-concept was expected between neurotypical cisgender and neurotypical transgender people, as well as between autistic cisgender and autistic transgender individuals (neurotypical cisgender = neurotypical transgender < autistic cisgender = autistic transgender).

Analysis.

A 2 (birth-assigned sex: male/female) × 2 (diagnostic category: neurotypical/autistic) × 2 (gender identity: cisgender/transgender) ANOVA was conducted on participant scores from the explicit measure of gender self-concept (note: scores were transformed to positive values so that the higher the score the stronger the explicit gender self-concept, regardless of whether it is male or female). As shown in Table E5, the 3-way interaction was nonsignificant. Nonetheless, to test our hypotheses a series of planned *t*-tests was conducted. Results of the analyses are presented in Table E5.

Table E5

Explicit Measure of Gender Self-Concept Analysis of Variance Results and Planned t-Tests

Measure	Effect	*F*	*p*	η_p^2	Planned *t*-tests [a]	Cohen's *d*	95% CI
Explicit	Sex	0.28	.600	.00			
	Gender identity	1.23	.269	.00			
	Diagnostic category	12.32	<.001	.04			
	Sex × Gender Identity	4.51	.034	.01			
	Sex × Diagnostic Category	9.48	.002	.03			
	Gender Identity × Diagnostic Category	35.10	<.001	.09			
	Sex × Gender Identity × Diagnostic Category	0.01	.934	.00			
					Birth-Assigned Males		
					NT cis > NT trans ***	1.01	[0.56, 1.45]
					NT cis > ASD cis ***	0.83	[0.43, 1.23]
					NT cis = ASD trans	0.39	[-0.07, 0.84]
					NT trans = ASD cis	0.04	[-0.39, 0.46]
					NT trans < ASD trans *	-0.53	[-1.02, -0.04]
					ASD cis < ASD trans *	-0.47	[-0.93, -0.01]
					Birth-Assigned Females		
					NT cis > NT trans *	0.36	[-0.05, 0.78]
					NT cis > ASD cis ***	1.41	[0.99, 1.83]
					NT cis > ASD trans *	0.57	[0.10, 1.05]
					NT trans > ASD cis ***	1.06	[0.63, 1.49]
					NT trans = ASD trans	0.21	[-0.28, 0.69]
					ASD cis < ASD trans ***	-0.84	[-1.31, -0.36]

Note. Sex = birth-assigned sex; NT = neurotypical; ASD = autism spectrum disorder; Cis = cisgender; Trans = transgender; 95% CI = 95% Confidence Intervals.

[a] Symbols for statistical significance denote results from one-tailed tests.

*p < .05. **p < .01. ***p < .001.

As expected, neurotypical cisgender birth-assigned males (n = 55; M = 4.84; SD = 0.74) scored significantly higher on the explicit task than neurotypical transgender (n = 37; M = 3.84, SD = 1.29) and autistic cisgender birth-assigned males (n = 50; M = 3.78; SD = 1.68). This indicates a stronger explicit gender self-concept among neurotypical cisgender birth-assigned males than among neurotypical transgender and autistic cisgender birth-assigned males. In contrast to predictions, a nonsignificant difference in the strength of the explicit gender self-concept was found between neurotypical cisgender and autistic transgender birth-assigned males (n = 29; M = 4.49; SD = 1.14). Also unexpectedly, neurotypical transgender birth-assigned males scored significantly lower on the task than autistic transgender birth-assigned males. Results suggest a weaker explicit gender self-concept among neurotypical transgender birth-assigned males than among autistic transgender birth-assigned males. A nonsignificant difference in the strength of the explicit gender self-concept was observed between neurotypical transgender birth-assigned males and autistic cisgender males. Furthermore, in contrast to predictions, neurotypical transgender birth-assigned males scored significantly lower on the task than neurotypical cisgender birth-assigned males, and autistic cisgender birth-assigned males scored significantly lower than autistic transgender birth-assigned males. Results indicate a weaker explicit gender self-concept among neurotypical transgender birth-assigned males than among neurotypical cisgender males and a weaker explicit gender self-concept among autistic cisgender males than among autistic transgender birth-assigned males.

As predicted, neurotypical cisgender birth-assigned females (n = 51; M = 4.89; SD = 0.94) scored significantly higher on the task than both autistic cisgender (n = 57; M = 2.98; SD = 1.64) and autistic transgender birth-assigned females (n = 27; M = 4.27; SE

= 1.32). This denotes a stronger explicit gender self-concept among neurotypical cisgender birth-assigned females than among autistic cisgender and autistic transgender birth-assigned females. Also in keeping with predictions, neurotypical transgender birth-assigned females (n = 41; M = 4.52; SD = 1.13) scored significantly higher on the explicit task than autistic cisgender birth-assigned females, indicating a stronger explicit gender self-concept among neurotypical transgender birth-assigned females. Yet, a nonsignificant difference in the strength of the explicit gender self-concept was found between neurotypical transgender birth-assigned females and the autistic transgender birth-assigned females. In contrast to predictions, autistic transgender birth-assigned females scored significantly higher than autistic cisgender birth-assigned females, and neurotypical cisgender birth-assigned females scored higher than neurotypical transgender birth-assigned females. Results indicate a stronger explicit gender self-concept among autistic transgender birth-assigned females than among autistic cisgender birth-assigned females, and among neurotypical cisgender birth-assigned females than among neurotypical transgender birth-assigned females.

Block 4: Association Analyses Between the Explicit and the Implicit Measures of Gender Self-Concept

Hypothesis 1. We expected the relation between explicit and implicit measures of gender self-concept to be less strong among transgender individuals (neurotypical and autistic) than among cisgender people (neurotypical and autistic).

Analysis.

A series of Fisher's Z tests was conducted to examine between-group differences in the magnitude of the association (the results of the association analyses are presented in Chapter 6). As expected, the association between the explicit and the implicit

measures of gender self-concept was stronger among neurotypical cisgender people than among neurotypical (z = 1.81, p = .035, one-tailed) and autistic transgender individuals (z = 1.80, p = .036, one-tailed). Contrary to predictions, there was not a significant difference in the magnitude of the association between autistic cisgender and either neurotypical (z = 0.77, p = .220, one-tailed) or autistic transgender individuals (z = 0.90, p = .183, one-tailed).

Block 5: Association Analyses Between Measures of Gender Self-Concept, and Current Gender Dysphoric Feelings and Recalled Gender-Typed Behaviour

Hypothesis 1. Within cisgender individuals (neurotypical and autistic), scores from the explicit and implicit measures of gender self-concept would be positively and significantly correlated with GIDYQ-AA score and RCGI score (stronger explicit and implicit identification with gender groups = fewer current gender dysphoric feelings and more recalled gender-typed behaviour).

Hypothesis 2. Within transgender individuals (neurotypical and autistic), scores from the explicit and implicit measures of gender self-concept would be negatively and significantly correlated with GIDYQ-AA score and RCGI score (weaker explicit and implicit identification with gender groups of birth-assigned sex = more current gender dysphoric feelings and fewer recalled gender-typed behaviour).

Hypothesis 3. The above described correlations would be less strong among transgender individuals (neurotypical and autistic) than among cisgender people (neurotypical and autistic).

Analysis.

In keeping with the preregistration, a series of correlation analyses was conducted examining the relations between the explicit and implicit measures of gender

self-concept, and current gender dysphoric feelings (measured using the GIDYQ-AA) and recalled gender-typed behaviour (measured using the RCGI). Please note that scores from the explicit and implicit measure of gender self-concept were transformed to positive values so that higher scores denote a stronger gender self-concept, regardless of whether it is male or female. As predicted, the strength of the explicit gender self-concept was positively and significantly related to GIDYQ-AA score, $r = .37$, $p < .001$ (one-tailed) and RCGI score, $r = .21$, $p = .017$ (one-tailed) among neurotypical cisgender people. The strength of the explicit gender self-concept was also positively and significantly correlated to GIDYQ-AA score, $r = .72$, $p < .001$ (one-tailed) and RCGI score, $r = .56$, $p < .001$ (one-tailed) among autistic cisgender people. Results suggest that the stronger the explicit identification of cisgender people (neurotypical and autistic) with the gender groups associated with their birth-assigned sex, the fewer their current gender dysphoric feelings tended to be and the more gender-typed behaviour they recalled from childhood.

We also found that among neurotypical transgender people, the strength of the explicit gender self-concept was negatively and significantly related to GIDYQ-AA score, $r = -.31$, $p = .003$ (one-tailed) and RCGI score, $r = -.22$, $p = .026$ (one-tailed). The correlation between the strength of the explicit gender self-concept and GIDYQ-AA score was also negative and significant among autistic transgender people, $r = -.44$, $p < .001$ (one-tailed). Contrary to predictions, the strength of the explicit gender self-concept was not significantly associated with RCGI score among autistic transgender people, $r = -.14$, $p = .161$ (one-tailed). Results suggest that the weaker the explicit identification of transgender people (neurotypical and autistic) with the gender groups associated with their birth-assigned sex, the more their current gender dysphoric feelings tended to be,

and the weaker the explicit identification of neurotypical transgender people with the gender groups associated with their birth-assigned sex, the less gender-typed behaviour they recalled from childhood.

A series of Fisher's *Z* tests was conducted to examine between-group differences in the magnitude of the associations. Contrary to predictions, neither the strength of the explicit gender self-concept × GIDYQ-AA correlation nor the strength of the explicit gender self-concept × RCGI correlation differ significantly between neurotypical cisgender and neurotypical transgender people ($z = 0.48$, $p = .314$, one-tailed and $z = -0.10$, $p = .459$, one-tailed, respectively). Also contrary to predictions, neither the strength of the explicit gender self-concept × GIDYQ-AA correlation nor the strength of the explicit gender self-concept × RCGI correlation differ significantly between neurotypical cisgender and autistic transgender people ($z = -0.45$, $p = .325$, one-tailed and $z = 0.43$, $p = .333$, one-tailed). As predicted, however, both the explicit gender self-concept × GIDYQ-AA correlation and the explicit gender self-concept × RCGI correlation were significantly larger among autistic cisgender people than among neurotypical transgender ($z = 3.89$, $p < .001$, one-tailed and $z = 2.66$, $p = .004$, one-tailed, respectively) and autistic transgender people ($z = 2.60$, $p = .005$, one-tailed and $z = 2.91$, $p = .002$, one-tailed, respectively).

Furthermore, as predicted, there was a positive and significant association between the strength of the implicit gender self-concept and GIDYQ-AA score, $r = .18$, $p = .042$ (one-tailed) among neurotypical cisgender individuals. Results suggest that the stronger the implicit identification of neurotypical cisgender people with the gender groups associated with their birth-assigned sex, the fewer their current gender dysphoric

feelings. In contrast to predictions, the relation between the strength of the implicit gender self-concept and RCGI score was nonsignificant, r = -.01, p = .446 (one-tailed).

In keeping with predictions, the strength of the implicit gender self-concept was also positively and significantly correlated with GIDYQ-AA score, r = .24, p = .012 (one-tailed) and RCGI score, r = .28, p = .004 (one-tailed), among autistic cisgender people. Nonetheless, a series of partial correlations revealed that the shared variance between the strength of the implicit gender self-concept, and GIDYQ-AA and RCGI can be attributed to the strength of the explicit gender self-concept (r = -.03, p = .760 and r = .12, p = .266, respectively). Contrary to predictions, neither among neurotypical transgender nor among autistic transgender individuals was there a significant association between the strength of the implicit gender self-concept, and GIDYQ-AA score and RCGI score (all ps ≥ .275, one-tailed). Given that only one of our predictions was confirmed, between-group differences in the magnitude of the associations were not examined.

Block 7: Association Analysis Between ASD Traits and Mentalising

Hypothesis 1. We expected RMIE score to be negatively and significantly correlated with AQ score (better mentalising = fewer ASD traits) within each group.

Analysis.

Contrary to predictions, performance on RMIE task did not correlate significantly with AQ score either among neurotypical cisgender people, r = .001, p = .495 (one-tailed) or among autistic transgender individuals, r = .02, p = .453 (one-tailed). A negative and significant correlation between RMIE and AQ was found among neurotypical transgender people, r = -.27, p = .008 (one-tailed), but when we accounted for the influence of TAS, the relation lost its significance, r = .01, p = .954. Contrary to

predictions, we also found a positive and significant relation between RMIE score and AQ score among autistic cisgender individuals, r = .27, p = .002 (one-tailed), suggesting that the better the mentalising ability of autistic cisgender people, the fewer their ASD traits. Given that results were mostly out of keeping with predictions, no further analyses were conducted.

Statistical Analyses Including Participants Whose Error Rate in the Critical Blocks of the IAT Exceeded 20%

All the analyses included in Chapter 6 were reconducted including all participants, regardless of their error rate in the critical blocks of the IAT. Results of the analyses all presented below.

Association Analysis Between AQ and Performance on the Implicit Measure of Gender Self-Concept

When all neurotypical cisgender people were included in the analysis, the relation between AQ and the strength of the implicit gender self-concept remained nonsignificant, r = -.05, p = .299 (one-tailed).

Performance on the Implicit Measure of Gender Self-Concept

A 2 (birth-assigned sex: male/female) × 2 (diagnostic category: neurotypical/autistic) × 2 (gender identity: cisgender/transgender) ANOVA was conducted on participant scores from the IAT. Just as in the reduced sample, significant main effects were detected for birth-assigned sex, $F(1,338) = 4.34$, p =.038, η_p^2 = .01, gender identity, $F(1,338) = 5.77$, p =.017, η_p^2 = .02, and diagnostic category, $F(1,338) = 11.78$, p =.001, η_p^2 = .03. The analysis also yielded a significant Birth-Assigned Sex × Gender Identity interaction, $F(1,338) = 250.51$, $p < .001$, η_p^2 = .43, a significant Gender

Identity × Diagnostic Category interaction, $F(1,338) = 4.37$ $p = .037$, $\eta_p^2 = .013$, and a nonsignificant Birth-assigned Sex × Diagnostic Category interaction, $F(1,338) = 0.20$, $p = .657$, $\eta_p^2 = .00$. The *only* result that changed substantively in the full sample was the 3-way interaction. In the reduced sample, the 3-way interaction was nonsignificant, whereas in the full sample it was significant.

Breaking down the three-way interaction, a simple effects analysis of birth-assigned sex within gender identity and diagnostic category indicated that just as in the reduced sample the IAT was sensitive to gender identity differences. That is, among neurotypical and autistic cisgender individuals, birth-assigned females scored significantly higher on the IAT than birth-assigned males, whereas among neurotypical and autistic transgender individuals, birth-assigned females scored significantly lower than birth-assigned males (all ps $< .001$, all $\eta_p^2 \geq .07$).

Next, we conducted a simple effects analysis of diagnostic category within birth-assigned sex and gender identity. Similar to the reduced sample, we found that autistic cisgender females achieved a significantly lower *D* score on the IAT than neurotypical cisgender females ($p < .001$, one-tailed, $\eta_p^2 = .07$), indicating a weaker implicit female self-concept. We also found that autistic and neurotypical transgender adults displayed an implicit gender self-concept that was in keeping with their experienced/reported gender, rather than birth-assigned gender, to the same degree (all ps $\geq .060$, all $\eta_p^2 \leq .01$). The only result that changed substantively in the full sample was the difference in *D* score between autistic and neurotypical cisgender birth-assigned males. In the reduced sample, autistic cisgender birth-assigned males scored significantly higher on the IAT

than neurotypical cisgender birth-assigned males, whereas a nonsignificant between-group difference was found in the full sample (p = .078, one-tailed, η_p^2 = .01).

Table E6

List and Explanation of Deviations from the Preregistration

Where?	What?	Why?
Sample size	*N* = 347 instead of 306, mainly because of the inclusion of a fourth participant group that was not planned at preregistration. Hence, neurotypical cisgender adults *n* = 106 instead of 102, neurotypical transgender adults *n* = 78 instead of 102, autistic cisgender adults *n* = 107 instead of 102, and autistic transgender adults *n* = 56 (not included in the preregistration).	After data collection begun, but *before* any statistical analyses were conducted, we decided to include as an independent group those autistic participants who identified as transgender and those transgender participants who reported a diagnosis of ASD rather than exclude them from the study. Due to changes in the data collection procedures (see below) preselection of participants based on specific criteria (diagnosis of ASD, gender identity etc.) was not always feasible. This partially explains why the total number of participants and the number of participants per group deviate from the preregistered numbers.
Hypotheses	Hypotheses about autistic transgender people were made before any statistical analyses were conducted. Therefore, *p* values for one-tailed tests are reported.	Due to the inclusion of autistic transgender people in the study as an independent group.
	Preregistration Block 5: Negative association between explicit/implicit gender self-concept, and GIDYQ-AA and RCGI among transgender people (autistic and neurotypical instead of positive.	Due to a typographical error in the preregistration.
Study design	2 (Birth-Assigned Sex: Male/Female) × 2 (Diagnostic Category: NT/ASD) × 2 (Gender Identity: Cisgender/Transgender) instead of 3 (Group: neurotypical/ autistic/ transgender) x 2 (Sex: male/female).	Due to the inclusion of an additional group (i.e., autistic transgender).

Where?	What?	Why?
Data collection procedures	Autistic participants were recruited through social media, Prolific Academic, and the Autism Research at Kent database. The City, University of London database was not used.	The planned collaboration with City, University of London did not proceed.
	Autistic participants (neurotypical and transgender) received £7.50 (i.e., the equivalent of £10 per hour) for their participation instead of £4.	The completion time of the study was increased to 45 minutes to meet the needs of autistic people.
	Although, one of the inclusion criteria was that participants must have English as their first language, three nonnative English speakers took part in the study	Preselection of participants based on their language was not always feasible during the data collection.
Data exclusion	Participants with error rate > 20% in the critical blocks of the IAT were excluded from the analysis.	We adopted the analytic strategy followed by Greenwald et al. (1998) to exclude the possibility that poor performance on the task had an effect on the results of the study. We decided to adopt this approach *before* conducting any inferential statistical analysis.
Analysis	To examine differences in mentalising, ASD traits, AND current and recalled gender dysphoria a series of 2 (Birth-Assigned Sex: Male/Female) × 2 (Diagnostic Category: NT/ASD) × 2 (Gender Identity: Cisgender/Transgender) ANOVAs were conducted instead of one-ways AVOVAs.	Due to the inclusion of an additional group (i.e., autistic transgender) and because it has become increasingly common to examine sex/gender-specific effects in studies on ASD and gender incongruence/dysphoria.
	Fisher's Z tests described in Blocks 5 and 7 of the preregistration were not conducted. The moderation analysis described in Block 7 was also not conducted.	Relations between scores from the implicit measure of gender self-concept, and GIDYQ-AA and RCGI were mostly out of keeping with predictions, so the results of Fisher's Z test would not be informative. Relations among AQ, GIDYQ-AA, and RMIE were mostly out of keeping with our predictions, so a moderation analysis could not be conducted and the results of Fisher's *Z* tests would not be informative.