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Links Between Autism, Childhood Gender Nonconformity, and Adult Sexual Orientation

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Abbreviated Title

Autism, Early Gender Nonconformity, and Adult Sexual Orientation

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Abstract

Background

Autism, gender diversity, and sexual orientation diversity can each be indexed categorically (autistic vs nonautistic; cisgender/gender incongruent; heterosexual/non-heterosexual) and dimensionally (continuous trait scores). Research has linked autism with both gender and sexual orientation diversity, but the basis of these associations remains unclear. Because childhood gender nonconformity is a robust dimensional correlate of adult sexual orientation in the general population, we examined whether recalled childhood gender nonconformity accounts for associations between autism and adult sexual orientation, and whether autism influences the childhood gender nonconformity $\times$ adult sexual orientation link.

Methods

Across two preregistered studies and two direct replications, we examined associations among autism, recalled childhood gender nonconformity, sexual attraction, and sexual identity in cisgender adults. Study 1 examined dimensional associations with autistic traits in 400 nonautistic adults and was replicated in an independent sample of 396 nonautistic adults. Study 2 compared 200 diagnosed autistic adults with 200 nonautistic adults and was replicated in an independent sample of 244 autistic and 229 nonautistic adults.

Results

Across all studies and samples, childhood gender nonconformity predicted adult sexual orientation. Study 1 showed no evidence that autism traits were associated with sexual attraction, sexual identity, or gender nonconformity. Autism traits did not moderate the association between childhood gender nonconformity and adult sexual orientation, and childhood gender nonconformity did not mediate these null associations. In contrast, Study 2 showed that autistic adults reported less heterosexual attraction, were more likely to identify as non-heterosexual, and recalled greater childhood gender nonconformity than nonautistic

adults. Childhood gender nonconformity mediated associations between autism diagnostic status and sexual orientation, whereas moderation effects were largely absent.

Conclusions

These findings suggest that autism-sexual orientation links emerge at the categorical level of autism diagnosis only and are accounted for statistically by childhood gender nonconformity.

Keywords

Autism, Autism Traits, Childhood Gender Nonconformity, Sexual Orientation, Gender Diversity, Cisgender Adults

Community brief

Why is this an important issue?

Childhood gender nonconformity (preferring activities and interests not stereotypically associated with one's biological sex) is strongly associated with sexual orientation diversity in adulthood. Also, autism is associated with sexual orientation and gender diversity in adults, but little is known about how autism relates to childhood gender nonconformity, or whether autism influences the link between childhood gender nonconformity and adult sexual orientation. This matters because some have speculated that gender diversity has an autism-specific basis in autistic people. If so, then the usual link between childhood gender nonconformity and adult sexual orientation may not hold in autistic people.

What was the purpose of this study?

To assess whether recalled childhood gender nonconformity accounts statistically for the association between autism and adult sexual orientation. We tested whether this association is found for autistic traits in nonautistic adults, autism diagnostic status, or both.

What did the researchers do?

We conducted two studies and repeated each study with a new group of participants ("replication studies"). Study 1 examined autistic traits, recalled childhood gender nonconformity, sexual attraction, and sexual identity in 400 cisgender nonautistic adults (and another 396 participants in the replication study). Study 2 compared 200 autistic and 200 nonautistic cisgender adults (and another 244 autistic and 229 nonautistic participants in the replication study) using the same measures.

What were the results?

Across studies, people who recalled more childhood gender nonconformity reported less heterosexual patterns of adult sexual attraction and were more likely to identify as non-heterosexual. In Study 1, autistic traits in nonautistic adults were not significantly related to adult sexual attraction or identity, or recalled childhood gender nonconformity. In Study 2, autistic adults reported greater sexual orientation diversity and recalled higher childhood gender nonconformity than nonautistic adults. Childhood gender

nonconformity partly explained the statistical association between autism diagnosis and adult sexual orientation. Findings from each study replicated closely. Importantly, autism (traits or diagnoses) did *not* influence the robust association between childhood gender nonconformity and adult sexual identity or attraction except in one instance.

What do these findings add to what was already known?

The research shows for the first time that (a) autistic traits among non-autistic people do not predict sexual orientation diversity, whereas autism diagnosis does, and (b) childhood gender nonconformity and adult sexual orientation are related in similar ways in autistic and nonautistic adults.

The results can't be used to predict any individual child's future adult sexual orientation or categorical gender identity, and gender nonconformity and non-heterosexuality shouldn't be conflated (they are not synonymous, even though they are related). As such, the observed association between childhood gender nonconformity and adult sexual orientation in our study shouldn't be used to dismiss any individual child's gender nonconformity as evidence they are "just gay", or used as a reason to discourage or attempt to change their gender presentation.

What are potential weaknesses in the study?

The studies were cross-sectional, so they cannot prove developmental or causal pathways. Childhood gender nonconformity was measured retrospectively (adults reported what they remembered about childhood).

How will these findings help autistic adults now or in the future?

These findings may help clinicians, educators, families, and researchers avoid interpreting autistic people's gender expression and sexuality as autism-specific simply because the person is autistic. Instead, they suggest autistic people's gender expression and sexual orientation should be understood as in the wider population, while ensuring that communication and support are accessible to autistic people. These results might provide useful context for some autistic adults when they reflect on their own childhood

gender nonconformity or adult sexuality, although experiences are individual and group-level results cannot explain any individual's identity.

Links Between Autism, Childhood Gender Nonconformity, and Adult Sexual Orientation

Background

Gender diversity (GD) refers broadly to variation in gender identity and gender-related behaviour relative to one's biological/birth-registered sex and associated cultural norms.¹ Notably, variation in gender identity can be operationalised and measured categorically or dimensionally.² For example, a person who identifies their gender as aligning with their sex-at-birth could be contrasted with a person who identifies their gender as something categorically other than their birth sex. The former person would be labelled as cisgender, whereas the latter referred to as gender incongruent. Even within these broad categories, however, there is dimensional variation in the degree to which a person identifies as, or acts in ways typical of, their gender. For example, a birth-/biological male might identify categorically as a male and thus be classified as cisgender, but nonetheless believe that they are not typical of other birth-males, or not act in ways that are stereotypically associated with males. Throughout this paper, we refer to gender congruence versus incongruence to distinguish categorical gender identity, and to degrees of gender conformity/nonconformity to distinguish dimensional differences in gender identity/expression. However, we directly *measure* only the latter dimensional operationalisation in the current studies and only among categorically cisgender participants.

Crucially, dimensional variation in childhood gender identity and behaviour is highly predictive of several adult outcomes. For example, developmental research consistently shows that childhood gender nonconformity predicts not only gender diversity in adolescence and adulthood,³⁻⁶ but more strikingly adult sexual orientation.⁷ Like gender identity, sexual orientation can be operationalised and measured categorically via sexual identity (e.g., heterosexual/non-heterosexual), or dimensionally via degree of sexual attraction to those of the same vs other sex/gender.^{8,9} For example, a significant minority of heterosexual-identifying adults nonetheless report some attraction to individuals of the same sex/gender, demonstrating that categorical sexual identities don't fully capture dimensional variation in sexual attraction.⁸ In the current manuscript, we measure sexual orientation categorically, as heterosexual versus non-heterosexual, and dimensionally, as individual differences in sexual attraction. When we refer to

“greater sexual orientation diversity”, or similar, we mean less exclusively heterosexual patterns of dimensional attraction and/or a greater likelihood of categorical non-heterosexual identity.

The link between childhood gender nonconformity and adult sexual orientation (across identity and attraction) has been demonstrated across retrospective, observational, and longitudinal studies, including prospective evidence from early childhood to adolescence and adulthood,^{6,10–13} with Hamer (p.166) describing the association as perhaps “the most consistent, well-documented, and significant finding in the entire field of sexual-orientation research and perhaps in all of human psychology”.¹⁴

Over the past decade, research has also documented an association between autism spectrum disorder (ASD) and GD. Like GD and sexual orientation, autism can be conceptualised categorically via diagnostic status (autistic vs nonautistic) and dimensionally via autism traits (continuous variation in autistic characteristics in non-autistic individuals). Compared to nonautistic adults, adults with diagnoses of autism report dimensionally weaker, less sex-typical gender self-concepts and less comfort with their assigned sex (i.e., elevated gender dysphoria traits), as well as a higher rate of categorical gender incongruence.^{15–19} Complementary qualitative research has focused on autistic gender diverse people’s lived experience and understanding of the relation between autism and gender.^{20,21}

Correspondingly, autistic adults also show higher rates of categorical non-heterosexual sexual orientation than nonautistic adults,^{22–24} and complementary qualitative research has explored how autistic young people understand their sexuality.²⁵ In adult general population samples, dimensional autism traits are associated significantly with adult gender nonconformity, as well as recalled levels of childhood gender nonconformity.^{26–28} It is unclear whether autism traits are also associated with adult *sexual orientation*, a gap in the literature that the current investigation was designed in part to fill. In the current manuscript, we measure autism categorically, via diagnostic status (autistic versus non-autistic), and dimensionally, via individual differences in autism traits in the non-autistic population.

To our knowledge, the only study to explore this matrix of relations between autism, GD, and sexual orientation was conducted by George and Stokes.¹⁷ While their study made a seminal and much-needed contribution to research on gender and sexual orientation in autism, several aspects of the design

complicate interpretation of its findings. Most notably, the autistic and nonautistic groups were combined in the key association/mediation analyses (creating a possible aggregation bias¹), the autistic group included disproportionately more gender incongruent participants than the nonautistic group, and “transgender” identity was coded as a non-heterosexual sexual orientation, potentially inflating group differences on (and associations between) these variables artificially². In addition, the study focused on *adult* GD (distress-related) traits as a mediator of the relation between autism traits and sexual orientation, but left unaddressed the influence of *childhood* gender nonconformity on the relation between autism and adult sexual orientation. It is this latter relation that has been the focus of the vast majority of research in the sexual orientation literature,^{6,11–13} but has so far been unexplored in autistic samples. The present studies were designed to address these issues directly.

Clarifying these associations has potential theoretical and practical importance. Some authors have suggested that GD in autistic people may, at least in some cases, be shaped by autism-related features such as restricted and stereotyped interests, and/or cognitive rigidity/concrete thinking.^{17,29,30} These accounts concern gender diversity rather than sexual orientation per se, and the current studies do not directly test any proposed mechanisms. Nonetheless, the current studies make a relevant contribution by asking whether recalled childhood gender nonconformity shows the well-established association with adult sexual orientation in autistic as well as nonautistic adults. A similar association across groups would not necessarily show that childhood gender nonconformity has the same basis in autistic and nonautistic people. However, it would suggest that childhood gender nonconformity in autistic people is not entirely autism-specific in its developmental correlates, but instead relates to adult sexual orientation in a way that

¹ Put simply, if groups X and Y differ significantly on variables A and B (as autistic and non-autistic participants did on both autism and GD traits in George and Stokes’ study), then collapsing the groups for correlation analyses can yield an overall association that is driven by between-group differences rather than within-group relations, thereby artificially inflating the correlation. A pooled mediation analysis is inappropriate where the associations differ across groups, because it assumes that the relations among the variables can be represented by a common set of paths in the pooled sample.

² The unusually high percentage of the autistic group classified as non-heterosexual under this coding scheme (rates of almost 70% vs 19%; 23%; 37%; and 41% in studies of a similar nature ^{22,23,38,57}), is consistent with this possibility. Note that rates of non-heterosexual identity were taken from George and Stokes²⁴, which involved the same sample as reported in George and Stokes¹⁷ (M. Stokes, personal communication).

is also observed in the wider population. This distinction matters practically because clinicians, educators, and families may be tempted to interpret autistic people's gender expression and sexuality exclusively through an autism-specific lens, and even possibly discount experiences of these as "just autism", when in reality they reflect the same phenomena in both autistic and non-autistic populations. A similar phenomenon has been reported in relation to gender identity, with some autistic individuals reporting that their gender-related concerns were explained away as a manifestation of their autism and therefore not something that should be supported directly.³¹

Study 1 used an individual-differences design in a large nonautistic adult sample to examine whether variation in autism traits is associated with sexual orientation, and whether recalled childhood gender nonconformity statistically accounts for (mediates), or moderates, this association at the dimensional/trait level. Sexual orientation was measured both dimensionally via sexual attraction (continuous attraction toward same- and other-gender/sex targets) and categorically via sexual identity (heterosexual versus non-heterosexual). Childhood gender nonconformity was measured via retrospective self-report about participants' gender-related identity and behaviour in childhood. Study 2 complemented this approach using a case-control design among cisgender autistic and cisgender nonautistic adults, using the same measures of childhood gender nonconformity and sexual orientation as in Study 1.

Based on the research on autism and sexual orientation, and literature on childhood gender nonconformity and adult sexual orientation, we predicted that autism (operationalised *dimensionally* as autism traits in Study 1 and *categorically* as diagnostic status in Study 2) would be associated with greater sexual orientation diversity (operationalised dimensionally as sexual attraction and categorically as non-heterosexual sexual identity) (Hypothesis 1), and that higher levels of childhood gender nonconformity would be associated with diminished heterosexual patterns of sexual attraction and sexual identity (Hypothesis 2). However, we made no *a priori* predictions about whether the association between childhood gender nonconformity and sexual orientation would differ as a function of autism traits (Study 1) and diagnosis (Study 2), nor whether any association between autism (traits/diagnostic status) and sexual orientation would be accounted for by childhood gender nonconformity (Hypothesis 3).

After completion of studies 1 and 2, we undertook direct replication studies using the same measures among an independent sample of cisgender, nonautistic adults (replication study 1; individual differences) and autistic vs non-autistic cisgender participants (replication study 2; case-control). We report the full details of the original Studies 1 and 2 here and summarise the results from their replications, reporting the full replication results in the supplementary materials.

Study 1: Method

Participants

Participants were 406 adults, of whom six were excluded for failing attention checks ($n = 5$) or reporting a gender-diverse identity ($n = 1$), yielding a final sample of 400 cisgender participants (50% female). The mean age was 45.77 years ($SD = 13.75$, range = 20–81). The sample was predominantly White (83.75%; 5.75% Asian; 5.50% Black; 4.50% Mixed; 0.50% Other ethnic backgrounds). None of the participants reported an autism diagnosis, being in the process of seeking a diagnosis, or identifying as autistic.

Participants were recruited via Prolific, provided informed consent, and completed the study online via Qualtrics. In each survey (across studies 1 and 2, and their replications), we included two attention checks to detect inattentive responding, a CAPTCHA item to screen for automated responses, and Qualtrics' reCAPTCHA score as an additional indicator of response validity. All participants included in the analyses passed both attention checks and the CAPTCHA item. Across all samples, only seven participants (out of 1669) had a reCAPTCHA score below 50. Although such scores can indicate automated responding, they are not definitive on their own. These participants were retained because they passed all direct quality checks, and results were unchanged when they were excluded.

Ethical approval was granted by the XXX (anonymised), and the study was preregistered on OSF (https://osf.io/2u3ex/overview?view_only=40c44924f93e4af18bd24aa5bcaf29e1).

Materials and Procedure

Autism Spectrum Quotient (AQ-50)

Autistic traits were measured using the AQ-50,³² a 50-item self-report questionnaire (scores = 0–50), with higher scores indicating greater autistic traits.

Recalled Childhood Gender Identity/Gender Role Questionnaire (RCGI)

Recalled childhood gender nonconformity was assessed using the 18-item Factor 1 of the RCGI,³³ assessing gender role behaviour and identity during the first 12 years of life (scores = 1–5), administered in male and female versions according to birth-assigned sex. Lower scores indicate *less* childhood gender-typical behaviour (i.e., more gender nonconformity).

Sexual Attraction: Sell Assessment of Sexual Orientation (SASO-Sexual Attraction Index)

Sexual attraction was assessed using two items from the Sell Assessment of Sexual Orientation,³⁴ measuring attraction to males and females over the past year via a 7-point scale. A continuous attraction index was computed as: $(Att_{same} - Att_{other}) / (Att_{same} + Att_{other})$, where Att_{same} and Att_{other} represent attraction to individuals of the same and other birth-assigned sex, respectively. The resulting scores range from –0.75 (exclusively other-sex attraction) to +0.75 (exclusively same-sex attraction). Thus, scores above zero indicated increasing degrees of same-sex attraction whereas scores below indicate increasing degrees of other-sex attraction. For example, a male participant who rated their attraction to women as 6 and to men as 2, would score –0.50 (moderately other-sex attracted): $(2 - 6) / (2 + 6) = -0.50$.

Sexual Identity

Participants were asked to report their sexual identity by selecting one of the following options: heterosexual, homosexual, bisexual, asexual, other, or prefer not to say, which were subsequently categorised as heterosexual (including only participants who identified as heterosexual) or non-heterosexual (including all participants who selected homosexual, bisexual, asexual, or other). Those who selected “prefer not to say” were excluded from the analyses involving sexual identity ($n = 4$).

Statistical Analysis

For Hypothesis 1, associations between autism traits and sexual orientation were examined using Pearson’s correlations for dimensional sexual attraction and point-biserial correlations for categorical sexual identity.

For Hypothesis 2, moderation analyses were conducted using PROCESS Model 1 (Version 5),³⁵ separately for sexual attraction and sexual identity. In these models, recalled childhood gender nonconformity (RCGI) was entered as the predictor (X), sexual orientation as the outcome (Y), and autism traits (AQ-50) as the moderator (W). Sexual attraction was analysed using ordinary least squares regression and sexual identity using logistic regression. Continuous predictors were mean-centred prior to computing interaction terms, such that a value of zero represents the average. This allowed regression coefficients to be interpreted as effects at average levels of the continuous predictors.

For Hypothesis 3, mediation analyses were conducted using PROCESS Model 4 (Version 5), separately for sexual attraction and sexual identity. Autism traits (AQ-50) were entered as the predictor (X), recalled childhood gender nonconformity (RCGI) as the mediator (M), and sexual orientation as the outcome (Y). In line with the preregistration, mediation was tested only when the corresponding moderation analysis was nonsignificant.

For the moderation and mediation models, bias-corrected bootstrap confidence intervals were based on 5,000 resamples (if a confidence interval for an effect does not include zero, the effect can be considered significant).

To clarify, the moderation analyses assess whether associations between individual differences in recalled childhood gender nonconformity and adult sexual orientation *differ* by level of autism traits (high/average/low). For example, it could be hypothetically that childhood gender nonconformity is associated with adult sexual identity/attraction among individuals with a high level of autism traits only.

In contrast, the mediation analyses assess whether associations between individual differences in autism traits and sexual identity/attraction are *statistically explained* by individual differences in recalled childhood gender nonconformity. Crucially, however, the planned mediation analyses are only appropriate when moderation analyses are nonsignificant. This is because mediation analysis assumes that the association between recalled childhood gender nonconformity and sexual orientation is equivalent across all levels of autism traits. Significant moderation would show that this assumption was not met, so it would be inappropriate to conduct a mediation analysis.

Finally, Bayesian analyses were used to supplement null-hypothesis significance testing where feasible; $BF_{10} > 1, > 3, > 10, > 30, > 100$ represent anecdotal, substantial, strong, very strong, and decisive evidence for H1, respectively, whereas $BF_{10} < 1, < 0.33, < 0.10, < 0.03$, and < 0.01 represent anecdotal, substantial, strong, very strong, and decisive evidence for H0.³⁶

Study 1: Results

Table 1 presents descriptive statistics and correlations. AQ-50 scores (autism traits) were not significantly associated with SASO score (sexual attraction) or sexual identity (heterosexual/non-heterosexual). In contrast, RCGI scores were significantly associated with both, such that lower RCGI (i.e., *more* childhood gender *nonconformity*) was linked to higher SASO scores (i.e., *more same-sex* attraction) and a greater chance of reporting a non-heterosexual identity.

Moderation Analyses

Sexual Identity

We next tested moderation for sexual identity (Y = heterosexual vs non-heterosexual; X = RCGI; W = AQ-50). The model was significant, $\chi^2(3) = 13.29, p = .004$, explaining 3.30% (Cox–Snell) to 6.15% (Nagelkerke) of variance. RCGI predicted sexual identity, $b = -0.76$, 95% bootstrap CI $[-1.22, -0.28]$, indicating greater odds of non-heterosexual identity with higher childhood gender nonconformity, independent of autism traits. AQ-50 did not predict identity, $b = 0.03$, 95% bootstrap CI $[-0.002, 0.07]$, and the $RCGI \times AQ-50$ interaction was nonsignificant, $b = 0.02$, 95% bootstrap CI $[-0.04, 0.08]$. Thus, autism traits did not moderate the association between recalled childhood gender nonconformity and categorical sexual identity in adulthood (i.e., childhood gender nonconformity predicted adult sexual identity similarly in adults with high, average, and low autism traits).

Sexual Attraction

The corresponding moderation model for sexual attraction (SASO score) was also significant, $F(3, 396) = 16.16, p < .001, R^2 = .11$. As with sexual identity, RCGI significantly predicted SASO score, $b = -0.18$, 95% bootstrap CI $[-0.25, -0.12]$, whereas AQ-50 did not, $b = 0.003$, 95% bootstrap CI $[-0.001, 0.01]$. The $RCGI \times AQ-50$ interaction was again nonsignificant, $b = 0.01$, 95% bootstrap CI

[−0.002, 0.01]. Thus, autism traits did not moderate the RCGI–sexual attraction association (i.e., childhood gender nonconformity predicted adult sexual attraction similarly in adults with high, average, and low autism traits).

Mediation Analyses

Although AQ-50 was not significantly associated with sexual attraction or sexual identity, we conducted the pre-registered mediation analyses because an indirect effect (i.e., mediation) can occur without a significant total effect/association (e.g., Hayes, 2009).

Sexual Attraction

In the first analysis, AQ-50 was the predictor (X), RCGI was the mediator (M), and SASO score was the outcome (Y). AQ-50 did not significantly predict RCGI, $b = -0.005$, $SE = 0.004$, $p = .150$, 95% CI [−0.013, 0.002], although RCGI significantly predicted SASO, $b = -0.18$, $SE = 0.03$, $p < .001$, 95% CI [−0.23, −0.12] such that higher childhood gender nonconformity was associated with less heterosexual patterns of attraction. The total effect of AQ-50 on sexual attraction was nonsignificant, $b = 0.004$, $SE = 0.002$, $p = .063$, 95% CI [−0.0002, 0.0083], as was the direct effect, $b = 0.003$, $SE = 0.002$, $p = .137$, 95% CI [−0.001, 0.007]. The indirect effect was also nonsignificant, $b = 0.001$, 95% bootstrap CI [−0.0003, 0.0024], providing no evidence of mediation.

Sexual Identity

In the second mediation model (X = AQ-50; M = RCGI; Y = sexual identity), AQ-50 again did not significantly predict RCGI, $b = -0.005$, $SE = 0.004$, $p = .156$, 95% CI [−0.013, 0.002], whereas RCGI significantly predicted sexual identity, $b = -0.73$, $SE = 0.23$, $p = .002$, 95% CI [−1.17, −0.28]. The direct effect was nonsignificant, $b = 0.03$, $SE = 0.02$, $p = .138$, 95% CI [−0.009, 0.07], as was the indirect effect, $b = 0.004$, 95% bootstrap CI [−0.001, 0.011], providing no evidence of mediation.

Direct Replication of Study 1

A direct replication of Study 1 in an independent sample of 396 nonautistic cisgender adults (50% female; $M_{\text{age}} = 45.77$ years, $SD_{\text{age}} = 13.63$) replicated the original pattern of findings (see Supplementary Materials and Table 2). Thus, all significant effects from Study 1 replicated, and all nonsignificant effects

remained nonsignificant. AQ-50 was not associated with sexual attraction or sexual identity, whereas recalled childhood gender nonconformity was associated with both. Fisher's Z tests indicated that the magnitudes of the correlations did not differ significantly between the original and replication samples, apart from SASO $\times$ Sexual Identity. As in the original study, autism traits did not moderate the associations between childhood gender nonconformity and sexual attraction or sexual identity, and childhood gender nonconformity did not mediate the (null) associations between autism traits and sexual attraction or identity.

Study 1: Discussion

Contrary to Hypothesis 1, autism traits were not directly associated with sexual orientation (attraction *or* identity) in this cisgender general population sample. Consistent with Hypothesis 2 and a substantial historical literature, however, recalled childhood gender nonconformity was a significant predictor of sexual orientation (both attraction and identity), with higher gender nonconformity associated with less heterosexual patterns of sexual orientation. Crucially, the analyses showed no reliable evidence that a) autism traits moderated the association between childhood gender nonconformity and sexual orientation, nor that b) childhood gender nonconformity mediated any association between autism traits and sexual orientation.

Together, these findings suggest a robust relation between childhood gender nonconformity and adult sexual orientation, that is largely independent of dimensional variation in autism traits in a cisgender nonautistic adult sample. Because individual-differences designs may not capture effects specific to clinically diagnosed autism, Study 2 employed a case-control design to provide a more stringent test of group differences in sexual orientation and underlying mechanisms.

Study 2: Method

Participants

Participants were 407 adults recruited via Prolific, provided informed consent, and completed the study online via Qualtrics, of whom seven were excluded for failing attention checks, yielding a final

sample of 200 autistic and 200 age- and birth-assigned sex-matched nonautistic adults (see Table 3). All participants identified as cisgender and predominantly White (78.50%; 7.25% Mixed; 7.00% Asian; 6.25% Black; 1.00% Other ethnic background). Groups did not differ significantly in ethnicity/ethnic background ($p = .321$; $BF_{10} = 0.08$). All autistic participants reported a formal ASD diagnosis made by a qualified clinician and, as expected, reported significantly higher autism traits than nonautistic participants (Table 3). Participants provided informed consent and completed the study online. Ethical approval was granted by the XXX and the study was preregistered on OSF (https://osf.io/2nva4/overview?view_only=2c9543cf428941c6941108d75fc14e66).

Measures and Procedure

Participants completed the same measures as in Study 1.

Statistical Analysis

Statistical analyses in Study 2 followed the same approach as in Study 1, except that autism was operationalised categorically as diagnostic status rather than dimensionally as AQ-50 score. Thus, in the moderation analyses, diagnostic status was entered as the moderator in place of autism traits, and in the mediation analyses it was entered as the predictor in place of autism traits.

Study 2: Results

Group Differences in Sexual Orientation

Table 3 presents group means for SASO (sexual attraction) and the percentage identifying as non-heterosexual (sexual identity). Groups differed significantly in sexual attraction (moderate effect size and a BF_{10} that strongly supported the alternative hypothesis; see Table 3)³. Groups also differed significantly in sexual identity, $\chi^2(1, N = 398) = 17.59, p < .001, \phi = .21, BF_{10} > 100$. Thus, as predicted, when operationalised categorically, autism was associated significantly with sexual orientation (attraction and identity).

³ Note that the between-group difference in SASO score remained significant after excluding participants who identified as categorically non-heterosexual ($p = .02, d = -0.27, BF_{10} = 1.81$).

Moderation Analyses

Sexual Identity

A moderation analysis was conducted (Y = heterosexual vs non-heterosexual; X = RCGI; W = diagnostic status). The overall model was significant, $\chi^2(3) = 66.19$, $p < .001$, explaining between 15.32% (Cox-Snell R^2) and 23.65% of the variance in sexual identity (Nagelkerke R^2). RCGI significantly predicted sexual identity, $b = -1.28$, 95% bootstrap CI $[-2.10, -0.67]$, such that higher recalled childhood gender nonconformity was associated with higher odds of non-heterosexuality, independent of diagnostic status. Diagnostic status was also a significant predictor, $b = 0.68$, 95% bootstrap CI $[0.11, 1.28]$, mirroring the Chi-square analysis in showing that autistic participants were more likely than nonautistic participants to identify as non-heterosexual. Crucially, the $\text{RCGI} \times \text{diagnostic status}$ interaction was nonsignificant, $b = 0.02$, 95% bootstrap CI $[-0.79, 1.01]$, indicating that childhood gender nonconformity predicted adult sexual identity similarly in autistic and nonautistic adults.

Sexual Attraction

The corresponding moderation model for sexual attraction was significant, $F(3, 396) = 38.81$, $p < .001$, $R^2 = .23$. As with sexual identity, RCGI significantly predicted SASO score, $b = -0.23$, 95% bootstrap CI $[-0.34, -0.14]$, whereas diagnostic status did not, $b = 0.07$, 95% bootstrap CI $[-0.004, 0.14]$. The $\text{RCGI} \times \text{diagnostic status}$ interaction was again nonsignificant, $b = -0.04$, 95% bootstrap CI $[-0.16, 0.10]$. This indicates that diagnostic status did not alter the association between childhood gender nonconformity and sexual attraction in adulthood. Thus, childhood gender nonconformity predicted adult sexual attraction similarly in autistic and nonautistic adults.

Mediation Analyses

Sexual Attraction

A mediation analysis was conducted, with diagnostic status as the predictor (X), RCGI as the mediator (M), and SASO score as the outcome (Y). Diagnostic status significantly predicted RCGI (a path), $b = -0.35$, $SE = 0.07$, $p < .001$, 95% CI $[-0.48, -0.22]$, such that autistic participants were more

likely to report higher childhood gender nonconformity than nonautistic participants. In turn, RCGI significantly predicted sexual attraction (b path), $b = -0.26$, $SE = 0.03$, $p < .001$, 95% CI $[-0.31, -0.21]$, such that higher childhood gender nonconformity was associated with less heterosexual patterns of attraction. The total effect of diagnostic status on sexual attraction via childhood gender nonconformity was also significant, $b = 0.157$, $SE = 0.04$, $p < .001$, 95% CI $[0.08, 0.23]$, whereas the direct effect of diagnostic status on sexual attraction was nonsignificant, $b = 0.07$, $SE = 0.04$, $p = .069$, 95% CI $[-0.005, 0.14]$. Yet crucially, the indirect effect was significant, providing evidence of full mediation, $b = 0.091$, 95% bootstrap CI $[0.05, 0.14]$, with 58% of the total effect of diagnostic status on sexual attraction operating through childhood gender nonconformity.⁴ Thus, group differences in sexual attraction were fully mediated by differences in recalled childhood gender nonconformity.

Sexual Identity

In the corresponding model for sexual identity (X = diagnostic status; M = RCGI; Y = sexual identity), diagnostic status significantly predicted RCGI, $b = -0.36$, $SE = 0.07$, $p < .001$, 95% CI $[-0.49, -0.23]$, and RCGI significantly predicted sexual identity, $b = -1.27$, $SE = 0.19$, $p < .001$, 95% CI $[-1.65, -0.89]$. The direct effect diagnostic status on sexual identity remained significant, $b = 0.68$, $SE = 0.28$, $p = .016$, 95% CI $[0.13, 1.23]$. The indirect effect was also significant, $b = 0.45$, 95% bootstrap CI $[0.25, 0.70]$, indicating partial mediation, with the indirect effect corresponding to a 57% increase in the odds of identifying as non-heterosexual⁵.

Direct Replication of Study 2

A direct replication of Study 2 in an independent sample of 244 autistic and 229 nonautistic adults (all cisgender) largely replicated the original pattern of findings (see Supplementary Materials and

⁴ To aid interpretation of the magnitude of the mediation effect, the proportion of the total effect accounted for by the indirect effect was calculated by dividing the indirect effect by the total effect.

⁵ Total effects are not available when the model includes a dichotomous outcome variable. Therefore, to aid interpretation of the magnitude of the indirect effect, the log-odds coefficient (b) of the indirect effect was converted into an odds ratio, which was then expressed as a percentage increase.

Table 2). Autistic participants again reported less heterosexual attraction, were more likely to identify as non-heterosexual, and recalled greater childhood gender nonconformity than nonautistic participants. The null moderation effect for sexual attraction and the mediation effect for sexual attraction were also replicated.

The only difference across the two concerned categorical sexual identity, where autism diagnostic status significantly moderated the recalled childhood gender nonconformity $\times$ sexual identity association in the replication study, unlike in the original. Crucially, the moderation showed that the association was significant in *both* groups but was weaker in autistic ($b = -0.90$) than non-autistic ($b = -2.06$) participants (see supplementary material for full details). In line with the preregistration, mediation was not tested for categorical sexual identity because significant moderation renders a simple mediation model uninterpretable.

Study 2: Discussion

Autistic cisgender adults reported less heterosexual sexual attraction, were more likely to identify as non-heterosexual, and recalled more childhood gender nonconformity than nonautistic adults. Diagnostic status did not moderate the association between recalled childhood gender nonconformity and either sexual attraction or categorical sexual identity. Recalled childhood gender nonconformity fully mediated the association between diagnostic status and sexual attraction, and partially mediated the association with categorical sexual identity in the original Study 2.

The only result from the original study that was not replicated in the replication study concerned the moderating role of diagnostic status in the relation between recalled childhood gender nonconformity and categorical sexual identity. Specifically, in the original study, diagnostic status did not moderate the RCGI $\times$ sexual identity association, whereas it did in the replication study. This would have been striking and potentially theoretically important if there was no, or only a minimal, association in autistic participants. However, the moderation effect actually reflected a significant association in *both* diagnostic groups, albeit one that was stronger in non-autistic than autistic participants. Moreover, the moderation

effect appeared descriptively to be driven more by an unusually large association among non-autistic participants than by an unusually small association in autistic participants (see S3 for further discussion).

The difference itself is unlikely to be explained by differences in the demographics of participants in the original and replication studies. The two studies had highly comparable samples, with demographics being very similar in terms of ratio of males to females, ethnic backgrounds, and gender identity. The only difference appeared to be in average age, which was around two to three years lower in the replication than original study. However, both samples of participants were in their 30s and there is no obvious reason to think the relatively minor age difference at this stage of adulthood would impact one result specifically. We are unclear why the association would be particularly strong among non-autistic than autistic participants in Study 2 (replication). However, the fact that it was directionally the same in both groups in both studies, and also that moderation was present in the replication study only, suggests that the difference may not be reliable and that, if it is reliable, it is relatively minor.

General Discussion

Across both studies (original and replication), we observed the well-established association between recalled childhood gender nonconformity and adult sexual orientation, indexed both dimensionally (sexual attraction) and categorically (sexual identity). Study 1 and its replication consistently indicated that autism traits did not moderate this association, however. In Study 2, the original study also found no evidence of moderation, and the replication study reproduced this for sexual attraction. For categorical sexual identity, however, the replication indicated that the association was present in both groups, but stronger in non-autistic than autistic participants. Overall, these findings suggest broadly similar links between recalled childhood gendered behaviour and adult sexual orientation in autistic and nonautistic individuals. However, in other respects, Studies 1 and 2 differed in important ways.

First, when autism was operationalised dimensionally in Study 1, autism traits were not significantly associated with recalled childhood gender nonconformity in either the original or replication

sample, which casts doubt on the reliability of previous findings of such an association in predominantly nonautistic adults.^{27,28} As we noted in the Introduction, previous studies in the general population that observed a significant association between autism traits and gender identity-related measures have included autistic and/or gender-incongruent participants in their samples.¹⁷ The risk of this is that any observed association between dimensionally-measured autism traits and gender identity-related traits could be artificially inflated and not reflective of the true association in the nonclinical population. Therefore, in Study 1 (both original and replication), we chose to include only non-autistic cisgender participants. It is notable, therefore, that the results from Study 1 differed from those in Study 2, where autism was operationalised categorically according to diagnostic status. In Study 2, autistic participants in both the original and replication samples recalled significantly higher levels of childhood gender nonconformity than nonautistic participants, with moderate effect sizes ($ds \geq 0.54$) and decisive support for the group difference ($BF_{10S} \geq 100$). A potential disadvantage of including only non-autistic cisgender adults in Study 1 is that the range of scores on the AQ-50 and/or RCGI might have been low, which would have reduced power to detect significant correlations between the measures. However, both the original and replication studies had sufficient power to detect small correlations, there was substantial variation in AQ-50 scores to detect associations, and there was sufficient variation in RCGI scores to observe associations with sexual identity and attraction. Thus, the null results are unlikely to be explained solely by insufficient variation in scores.

Second, and more importantly, Studies 1 and 2 differed in the extent to which autism was associated with sexual orientation and in whether recalled childhood gender nonconformity statistically accounted for that association. In Study 1, autism *traits* were not significantly associated with either sexual attraction or sexual identity, and recalled childhood gender nonconformity did not mediate these relations. In Study 2, however, autism *diagnostic status* was significantly associated with both sexual attraction and sexual identity, and recalled childhood gender nonconformity mediated the association between diagnostic status and sexual attraction in both the original and replication samples (as well as the association between diagnostic status and sexual identity in the original study).

The null association between autism traits and sexual orientation in Study 1 is potentially important. To our knowledge, only one other study has examined autism $\times$ sexual orientation associations using a trait measure,³⁸ which found that participants from a large population sample who scored above the AQ-10 clinical cut-off were less likely to identify as heterosexual than those scoring below it. However, because autism traits were dichotomised as above versus below a clinical cutoff score, Rudolph et al.'s approach was closer to a case-control design than the individual-differences design we used in Study 1. Taken together, the findings might imply that sexual orientation diversity is associated with categorical autism status, rather than with variation across the full autism-trait distribution (though we note that an above-threshold AQ score neither indicates diagnosis nor reliably predicts diagnostic outcome in adults undergoing full assessment for ASD³⁹).

Across studies 1 and 2, the findings imply that associations between autism and sexual orientation exist primarily at the level of clinical autism rather than across the full trait continuum. On this account, variation in autism traits among nonautistic individuals is insufficient to predict sexual orientation, even though childhood gender nonconformity itself robustly and consistently predicts sexual orientation. Instead, autism predicts sexual orientation only once autism-related characteristics reach a level typically associated with clinical diagnosis (but see ³⁸). This may help explain why autism $\times$ sexual orientation associations were observed in the case-control Study 2 (original and replication) but not in the individual-differences study (original or replication), and why mediation by childhood gender nonconformity was found only when autism was operationalised categorically.

Together, across the original and replication samples, the findings from Study 2 suggest that autism is associated with sexual orientation primarily indirectly, through its association with recalled childhood gender nonconformity. In both the original and replication Study 2, autism diagnostic status was associated with higher levels of recalled childhood gender nonconformity, which in turn was associated with greater sexual orientation diversity. This pattern suggests that autism may increase the likelihood of entering developmental trajectories associated with adult sexual orientation diversity by shifting the distribution of early gender nonconformity. Although the replication differed from the

original study in yielding significant moderation for categorical sexual identity, this reflected a *quantitative* difference in the strength of an association that was present in both groups, rather than evidence for a qualitatively distinct developmental pathway in autistic people.

It is important to be clear that the present data were cross-sectional rather than longitudinal and so the mediation analyses should be interpreted as statistical rather than causal, and the present findings do not establish developmental pathways. Nonetheless, the current findings are important not only because they shed light on sexual orientation diversity that is well-documented in autistic people, but also because they inform broader debates about whether gender diversity in autistic and nonautistic people reflects the same underlying phenomenon.^{40,41} If gender diversity shows similar associations across autistic and nonautistic groups, then this is *consistent with* (though not necessarily proof of) the view that it is tapping a comparable construct rather than an autism-specific variant of gender diversity. In the current studies, autism (traits/status) did not moderate the association between childhood gender nonconformity and adult sexual orientation (sexual identity or sexual attraction). Thus, childhood gender diversity (operationalised dimensionally) showed a similar link with adult sexual orientation in autistic and nonautistic adults (albeit in a cross-sectional design). This pattern supports the interpretation that dimensional gender nonconformity functions similarly across groups, which is consistent with the broader view that gender diversity in autism is not a distinct phenomenon.^{42–44} This has practical relevance.

If sexual orientation and/or gender diversity is conceptualised as having a different underlying basis in autistic versus nonautistic people, then clinicians, educators, and/or families might interpret autistic people's gender expression and sexuality as autism-specific. Such an interpretation is not supported in any way by the current studies. Practically, this implies that autistic people's gender identities and sexual orientations should be understood in the same way it is understood in the wider population, rather than as an autism-specific manifestation of these traits. This might help validate the experiences of gender and/or sexually diverse autistic people.

One thing to consider is how the use of retrospective self-reports of gender non-conformity, via the RCGI, could have influenced results. By their nature, retrospective reports rely on memory, which is

an inherently reconstructive and, therefore, fallible process. In general, errors in memory for childhood experiences would generally weaken, rather than produce, associations. However, it is possible that adult characteristics could systematically bias memories to produce unreliable associations between childhood experiences and adult outcomes. This has been suggested as an explanation for the well-established link between self-reported childhood gender non-conformity and adult non-heterosexual orientation that we also observed in the current studies.⁴⁵ However, as noted in the Introduction, the link between childhood gender non-conformity and adult non-heterosexual orientation has been observed reliably not only using retrospective self-report measures, but also in direct observational and longitudinal studies. For example, adult sexual orientation can be predicted from independent observations of home videos of children when they were of preschool age.¹¹ Perhaps most important, retrospective self-reports of childhood gender nonconformity were associated significantly with the independent observations of childhood behaviour, suggesting that self-reports can be a valid and reliable means of assessing gender in childhood. These findings mirror those from longitudinal studies that show a robust link between gender nonconformity measured during childhood and sexual orientation in adulthood.^{10,12,46} Although recall bias cannot be ruled out, therefore, the convergence of evidence across multiple different methods in previous studies, makes it unlikely that the childhood gender nonconformity $\times$ adult sexual orientation association is merely an artefact of retrospective reporting.

It is also worth considering the implications of including only non-autistic cisgender participants in Study 1, rather than a mixed general population sample that included a mixture of autistic and non-autistic people (and/or gender incongruent people). Were we to have included a general population sample that included a proportion of autistic (and/or gender incongruent) adults, any association in the pooled sample could have been driven partly or exclusively by differences between the diagnostic groups on the relevant measures. For example, we know that, by definition, autistic participants would have more ASD traits and therefore be almost certain to score substantially higher than non-autistic participants on the AQ-50. We also know from extensive previous findings that autistic adults tend to be less gender conforming and so would be likely to score lower than non-autistic participants on the RCGI. Under these

conditions, including both groups in a correlation analysis risks producing an AQ $\times$ RCGI association even if AQ and RCGI scores were not related in either group separately. This potential aggregation bias affects many previous dimensional studies of the relation between autism traits and gender identity-related traits¹⁶ and could have overestimated the extent to which autism traits are associated with weaker gender identity or atypical gender expression in non-autistic cisgender people. The current Study 1 samples were around four times larger than the samples in the only two previous studies to report a significant AQ-50 $\times$ RCGI association (combined N = 211 participants),^{27,28} suggesting that the earlier findings may not be reliable.

Finally, it is worth considering how the inclusion of participants who identified as asexual in the non-heterosexual identity category may have influenced results. Asexuality might be considered different from other forms of non-heterosexuality in that it relates to the absence of sexuality, rather than the sex/gender toward which sexuality is directed. There is relatively little research into the extent to which asexual people differ from other non-heterosexual people in terms of their gender identity. On the one hand, there is evidence that concurrent gender diversity traits (i.e., weaker identification and/or lower satisfaction with one's birth sex) are equivalent in asexual, homosexual, bisexual, and "other" non-heterosexual identities.¹⁷ On the other hand, there is also evidence that asexual people are more likely than people with other non-heterosexual identities to be categorically gender incongruent and female.⁴⁷ These issues are unlikely to have affected our results, because the rate of asexuality in participants was very low in each study ($\leq 1.7\%$) and all participants were categorically cisgender. Nonetheless, to confirm that the inclusion of asexual participants did not skew our results, we re-ran all experimental analyses in each study after excluding asexual participants. All results, apart from one⁶, were substantively identical

⁶ The only result that changed after exclusion of asexual participants was in the moderation analysis of sexual identity in Study 2 (original). In the primary analysis, the diagnostic status was a significant predictor of sexual identity *at the mean level of childhood gender nonconformity* (RCGI score) ($b = 0.68, p = .020, 95\% \text{ bootstrap CI} = 0.11-1.28$), whereas it was nonsignificant after the removal of asexual participants from the analysis, ($b = 0.56, p = .065, 95\% \text{ bootstrap CI} = -0.02-1.19$). To put this in context, the odds of being non-heterosexual at the mean RCGI score were 98% higher in autistic than non-autistic participants in the primary analysis ($b = 0.68$), whereas they were reduced slightly to 76% higher in the re-analysis. Importantly, the Chi-square analysis showing an overall unadjusted association between diagnostic status and sexual identity remained highly significant in the reanalysis (χ^2

after the exclusion of asexual participants. To clarify, apart from one exception, all results that were significant in the primary analyses continued to be significant, just as all that were non-significant continued to be non-significant, after excluding asexual participants. Thus, the inclusion of asexual participants in the non-heterosexual identity category did not meaningfully influence our results or interpretation of them.

Two potential limitations should be noted. First, although the AQ-50 was selected to facilitate comparison with earlier research (particularly George & Stokes, 2018¹⁷), it is limited as a measure of autism traits in the sense that it shows item-level gender non-invariance.⁴⁸ Future research should consider using other trait measures, such as the Comprehensive Autistic Trait Inventory (CATI), which have evidence to suggest measurement invariance of autism traits across genders.^{49,50} Second, both studies relied exclusively on participants recruited through Prolific who completed the measures online. Although Prolific has been shown to yield higher-quality responses than several other online platforms,⁵¹ the recruitment and testing methods likely produce samples that are not representative on several dimensions.⁵² Therefore, future studies would benefit from adopting a more diverse approach to recruitment and testing that generates a more representative sample than in the current study.

An important methodological strength of the present work is that both studies were preregistered and accompanied by direct replications. This is important because replication is central to theory building, yet many published findings do not reproduce cleanly when retested.^{53,54} Moreover, undisclosed flexibility in data collection, measurement, and statistical analysis can inflate false positive rates,⁵⁵ making it difficult to distinguish robust effects from artefacts of analytic choice. By preregistering hypotheses and analytic decisions in advance, the present studies provide a firmer basis for drawing meaningful conclusions.⁵⁶

Conclusion

= 14.68, $p < .001$, $\Phi = .19$), and the RCGI $\times$ diagnostic status interaction effect continued to be clearly nonsignificant ($b = -0.09$, $p = .83$, 95% bootstrap CI = -0.90, 0.82) showing that moderation was not present.

Taken together, these studies confirm the previously-reported link between recalled childhood gender nonconformity and adult sexual orientation. They call into question, however, the previously-observed association between individual differences in autism traits and gender nonconformity in predominantly non-autistic adults, and also find no evidence that autism traits in nonautistic cisgender adults are associated with sexual orientation. Crucially, though, the results show a robust link between autism diagnostic status (autistic/non-autistic) and both recalled childhood gender nonconformity and adult sexual orientation. Perhaps most importantly, the link between gender nonconformity and sexual orientation was broadly equivalent in both autistic and non-autistic cisgender participants. Because of the investigation's cross-sectional design and use of retrospective reports of gender nonconformity, it can't establish how these links develop or be used to predict any individual child's future identity. Nonetheless, the findings caution against treating sexual orientation or gender diversity in autistic adults as an autism-specific phenomenon.

Author contributions

David Williams and Aimilia Kallitsounaki contributed to all aspects (conceptualization, methodology, investigation, formal analysis, and original draft preparation. Matthew Fysh contributed to methodology, investigation, and writing (review and editing). Sophie Lind contributed to conceptualization, methodology, and writing (review and editing).

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Tables

Table 1

Descriptive Statistics and Correlations for Study 1 Variables

Variable	<i>M</i>	<i>SD</i>	1	2	3	4
1. AQ-50	18.43	7.96	-			
2. RCGI	3.99	0.59	-.07 ^a	-		
3. SASO	-0.53	0.34	.09 ^b	-.31*** ^d	-	
4. Sexual identity (% non-heterosexual;	12.87	-	.09 ^a	-.17*** ^c	.74*** ^d	-

Note. $N = 400$ (for analyses including sexual identity: $N = 396$). AQ-50 = Autism Spectrum Quotient-50; RCGI = Recalled Childhood Gender Identity; SASO = Self Assessment of Sexual Orientation – attraction index; Sexual identity: 0 = heterosexual / 1 = non-heterosexual.

*** $p < .001$.

^aBF₁₀ = 0.10–0.32. ^bBF₁₀ = 0.33–0.99. ^cBF₁₀ = 11–30. ^dBF₁₀ > 100.

Table 2*Summary of Original and Replication Findings Across Studies*

Analysis	Original study	Replication Study	Outcome
Study 1			
AQ-50 $\times$ sexual attraction	Nonsignificant	Nonsignificant	Overlap
AQ-50 $\times$ sexual identity	Nonsignificant	Nonsignificant	Overlap
RCGI $\times$ sexual attraction	Significant	Significant	Overlap
RCGI $\times$ sexual identity	Significant	Significant	Overlap
AQ-50 moderation of RCGI $\times$ sexual attraction	Nonsignificant	Nonsignificant	Overlap
AQ-50 moderation of RCGI $\times$ sexual identity	Nonsignificant	Nonsignificant	Overlap
RCGI mediation of AQ-50 $\times$ sexual attraction	Nonsignificant	Nonsignificant	Overlap
RCGI mediation of AQ-50 $\times$ sexual identity	Nonsignificant	Nonsignificant	Overlap
Study 2			
Sexual attraction group difference	Significant	Significant	Overlap
Sexual identity group difference	Significant	Significant	Overlap
RCGI group difference	Significant	Significant	Overlap
Diagnosis moderation of RCGI $\times$ sexual attraction	Nonsignificant	Nonsignificant	Overlap
Diagnosis moderation of RCGI $\times$ sexual identity	Nonsignificant	Significant	Divergence
RCGI mediation of diagnosis $\times$ sexual identity	Significant	N/A	N/A
RCGI mediation of diagnosis $\times$ sexual attraction	Significant	Significant	Overlap

Note. AQ-50 = Autism Spectrum Quotient-50; RCGI = Recalled Childhood Gender Identity.

Table 3*Participant Characteristics and t-Test Statistics in Study 2*

Variable	Nonautistic	Autistic	<i>t</i>	<i>p</i>	<i>d</i>	BF ₁₀
	50% female	50% female				
	<i>M (SD)</i>	<i>M (SD)</i>				
Age	37.98 (11.87)	38.01 (11.97)	−0.03	.977	−.003	0.11
AQ-50	19.38 (7.88)	34.02 (9.16)	−17.14	< .001	−1.71	> 100
RCGI	3.98 (0.58)	3.62 (0.73)	5.35	< .001	0.54	> 100
SASO	−0.55 (0.34)	−0.39 (0.43)	−4.05	< .001	−0.41	> 100
Sexual Identity (% non-heterosexual)	13.00	30.30				

Note. *N* = 400 (*N* = 398 for Sexual Identity). AQ-50 = Autism Spectrum Quotient-50; RCGI = Recalled Childhood Gender Identity; SASO = Sell Assessment of Sexual Orientation – attraction index.

Supplementary Materials

1. Direct Replication of Study 1

Participants

Participants were 403 adults, of whom seven were excluded for failing attention checks, yielding a final sample of 396 cisgender participants (50% female). The mean age was 45.77 years ($SD = 13.63$, range = 20–84). The sample was predominantly White (83.59%; 7.32% Black; 4.55% Mixed; 4.04% Asian; 0.51% Other ethnic backgrounds). None of the participants reported an autism diagnosis, being in the process of seeking a diagnosis, or identifying as autistic. Participants were recruited via Prolific, provided informed consent, and completed the study online.

Results

Correlation Analyses

Table S1

Descriptive Statistics and Correlations for Replication Study 1 Variables

Variable	<i>M</i>	<i>SD</i>	1	2	3	4
1. AQ-50	17.71	7.75	-			
2. RCGI	4.06	0.57	-.06 ^b	-		
3. SASO	-0.55	0.32	.06 ^b	-.29*** ^c	-	
4. Sexual identity (% non-heterosexual;	7.85	-	<.01 ^a	-.28*** ^c	.65*** ^c	-

Note. $N = 396$ (for analyses including sexual identity: $N = 395$). AQ-50 = Autism Spectrum Quotient-50; RCGI = Recalled Childhood Gender Identity; SASO = Self Assessment of Sexual Orientation – attraction index; Sexual identity: 0 = heterosexual / 1 = non-heterosexual.

*** $p < .001$.

^a $BF_{10} = 0.03$ – 0.09 . ^b $BF_{10} = 0.10$ – 0.32 . ^c $BF_{10} > 100$.

Table S2

Bivariate Correlations Differences Between Original and Replication Study 1

Association	Original Study	Replication Study	Fisher's Z test
RCGI $\times$ SASO	$r = -.31^{***}$	$r = -.29^{***}$	$Z = 0.31, p = .757$
RCGI $\times$ Sexual Identity	$r = -.17^{***}$	$r = -.28^{***}$	$Z = 1.63, p = .104$
RCGI $\times$ AQ-50	$r = -.07$	$r = -.06$	$Z = 0.14, p = .888$
SASO $\times$ AQ-50	$r = .09$	$r = .06$	$Z = 0.42, p = .672$
Sexual Identity $\times$ AQ-50	$r = .09$	$r < .01$	$Z = 1.12, p = .261$
SASO $\times$ Sexual Identity	$r = .74^{***}$	$r = .65^{***}$	$Z = 2.45, p = .014$

Note. AQ-50 = Autism Spectrum Quotient-50; RCGI = Recalled Childhood Gender Identity; SASO = Sell Assessment of Sexual Orientation – attraction index; Sexual identity: 0 = heterosexual / 1 = non-heterosexual.

*** $p < .001$.

Moderation Analyses

Sexual Attraction.

A moderation analysis was conducted, with SASO score as the outcome variable (Y), recalled childhood gender nonconformity (RCGI) as the predictor (X), and autism traits (AQ-50) as the moderator (W). The model was significant, $F(3, 392) = 12.37, p < .001, R^2 = .09$. As in the original sample, **(a)** RCGI significantly predicted SASO, $b = -0.16$, 95% bootstrap CI $[-0.23, -0.09]$, indicating less heterosexual attraction with higher childhood gender nonconformity; **(b)** AQ-50 did not predict SASO, $b = 0.002$, 95% bootstrap CI $[-0.002, 0.006]$, and **(c)** the RCGI $\times$ AQ-50 interaction was nonsignificant, $b = -0.003$, 95% bootstrap CI $[-0.009, 0.006]$. Thus, autism traits did not moderate the RCGI–sexual attraction association.

Sexual Identity.

We next tested moderation for sexual identity (Y = heterosexual vs non-heterosexual; X = RCGI; W = AQ-50). The model was significant, $\chi^2(3) = 24.65, p < .001$, explaining 6.05% (Cox–Snell) to

14.30% (Nagelkerke) of variance. As in the original sample, **(a)** RCGI predicted sexual identity, $b = -1.29$, 95% bootstrap CI $[-1.90, -0.69]$, indicating greater odds of non-heterosexual identity with lower RCGI score (i.e., higher childhood gender nonconformity), independent of autism traits, and **(b)** AQ-50 did not predict identity, $b = -0.02$, 95% bootstrap CI $[-0.08, 0.03]$, and; **(c)** the $\text{RCGI} \times \text{AQ-50}$ interaction was nonsignificant, $b = -0.02$, 95% bootstrap CI $[-0.09, 0.05]$. Thus, autism traits did not moderate the association between recalled childhood gender nonconformity and categorical sexual identity in adulthood.

Mediation Analyses

Sexual Attraction.

In the first analysis, AQ-50 was the predictor (X), RCGI was the mediator (M), and SASO score was the outcome (Y). As in the original sample, **(a)** AQ-50 did not predict RCGI (a path), $b = -0.004$, $SE = 0.004$, $p = .265$, 95% CI $[-0.011, 0.003]$, **(b)** RCGI predicted SASO (b path), $b = -0.16$, $SE = 0.03$, $p < .001$, 95% CI $[-0.22, -0.11]$, such that higher childhood gender nonconformity was associated with less heterosexual patterns of attraction, and; **(c)** the total effect of autism traits on sexual attraction was nonsignificant, $b = 0.003$, $SE = 0.002$, $p = .230$, 95% CI $[-0.002, 0.007]$. After accounting for RCGI score, the direct effect of AQ-50 on sexual attraction was nonsignificant, $b = 0.002$, $SE = 0.002$, $p = .359$, 95% CI $[-0.002, 0.006]$. The indirect effect was also nonsignificant, $b = 0.001$, 95% bootstrap CI $[-0.001, 0.002]$, providing no evidence of mediation.

Sexual Identity.

In the second analysis (X = AQ-50; M = RCGI; Y = sexual identity), as in the original sample, **(a)** AQ-50 did not significantly predict RCGI (a path), $b = -0.004$, $SE = 0.004$, $p = .249$, 95% CI $[-0.012, 0.003]$, **(b)** RCGI significantly predicted sexual identity (b path), $b = -1.32$, $SE = 0.26$, $p < .001$, 95% CI $[-1.84, -0.80]$, such that higher childhood gender nonconformity was associated with increased odds of non-heterosexuality, and **(c)** the direct effect of AQ-50 on sexual identity was nonsignificant, $b = -0.007$, $SE = 0.02$, $p = .783$, 95% CI $[-0.06, 0.04]$, as was the indirect effect, $b = 0.006$, 95% bootstrap CI $[-0.004, 0.02]$, providing no evidence of mediation.

2. Direct Replication of Study 2

Participants

Participants were 495 adults recruited via Prolific, of whom six were excluded for failing attention checks, one for reporting a gender diverse identity, and 15 due to overlap between the original and replication samples, yielding a final sample of 244 autistic and 229 age- and birth-assigned sex-matched nonautistic adults (see Table S3). All participants identified as cisgender and predominantly White (76.53%; 8.46% Black; 6.77% Asian; 6.13% Mixed; 2.11% Other ethnic background). All autistic participants reported a formal ASD diagnosis made by a qualified clinician and, as expected, reported significantly higher autism traits than nonautistic participants (Table S3). Participants provided informed consent and completed the study online.

Results

Moderation Analyses

Sexual Attraction.

Table S3

Participant Characteristics and t-Test Statistics in Replication Study 2

Variable	Nonautistic 52% female	Autistic 52% female	<i>t</i>	<i>p</i>	<i>d</i>	BF ₁₀
	<i>M (SD)</i>	<i>M (SD)</i>				
Age	35.70 (10.33)	35.88 (10.37)	−0.19	.848	−.02	0.10
AQ-50	18.52 (7.29)	32.74 (7.88)	−20.34	< .001	−1.87	> 100
RCGI	4.07 (0.54)	3.60 (0.68)	8.32	< .001	0.76	> 100
SASO	−0.56 (0.33)	−0.36 (0.43)	−5.68	< .001	−0.52	> 100
Sexual Identity (% non-heterosexual)	13.16	34.71				

Note. *N* = 473 (for analyses including sexual identity: *N* = 470). AQ-50 = Autism Spectrum Quotient-50; RCGI = Recalled Childhood Gender Identity; SASO = Self Assessment of Sexual Orientation – attraction index.

A moderation analysis was conducted, with SASO score as the outcome variable (Y), recalled childhood gender nonconformity (RCGI) as the predictor (X), and diagnostic status (autistic/nonautistic) as the moderator (W). The overall model was significant, $F(3, 469) = 41.97, p < .001, R^2 = .21$, and the inclusion of the RCGI $\times$ diagnostic status interaction explained an additional, non-significant 0.3% of the variance beyond the main effects. In contrast with the original sample, Diagnostic group was a significant predictor of SASO scores in the replication sample, $b = 0.08$, 95% bootstrap CI [0.007, 0.15], showing that autistic participants were more likely than nonautistic participants to show non-heterosexual patterns of sexual attraction, independent of recalled childhood gender nonconformity. As in the original sample, however, **(a)** RCGI significantly predicted SASO scores, $b = -0.29$, 95% bootstrap CI [-0.39, -0.18], such that higher recalled childhood gender nonconformity was associated with less heterosexual patterns of sexual attraction, independent of diagnostic status and; **(b)** the RCGI $\times$ diagnostic status interaction was nonsignificant, $b = 0.07$, 95% bootstrap CI [-0.07, 0.19]. This indicates that diagnostic status did not alter the association between childhood gender nonconformity and sexual attraction in adulthood.

Sexual Identity.

A second moderation analysis was conducted (Y = heterosexual vs non-heterosexual; X = RCGI; W = diagnostic status). The overall model was significant, $\chi^2(3) = 87.68, p < .001$, explaining between 17.02% (Cox-Snell R^2) and 25.41% of the variance in sexual identity (Nagelkerke R^2). As in the original sample, **(a)** RCGI significantly predicted sexual identity, $b = -2.06$, 95% bootstrap CI [-2.86, -1.34], such that higher recalled childhood gender nonconformity was associated with higher odds of non-heterosexuality, independent of diagnostic status and; **(b)** Diagnostic status was also a significant predictor, $b = 0.91$, 95% bootstrap CI [0.38, 1.47], showing that autistic participants were more likely than nonautistic participants to identify as non-heterosexual. In contrast with the original sample, the RCGI $\times$ diagnostic status interaction was significant, $b = 1.16$, 95% bootstrap CI [0.27, 2.07], indicating that childhood gender nonconformity predicted adult sexual identity differently in autistic and nonautistic adults in the replication sample. To break down the interaction, a simple main effects analysis showed the relationship between childhood gender nonconformity and adult sexual identity was significantly larger in

non-autistic, $b = -2.06$, $SE = 0.34$, $p < .001$, 95% CI $[-2.82, -1.30]$, than autistic participants, $b = -0.90$, $SE = 0.21$, $p < .001$, 95% CI $[-1.31, -0.49]$.

Mediation Analyses

Sexual Attraction.

A mediation analysis was conducted, with diagnostic status as the predictor (X), RCGI as the mediator (M), and SASO score as the outcome (Y). As in the original sample, **(a)** Diagnostic status significantly predicted RCGI (a path: $b = -0.47$, $SE = 0.06$, $p < .001$, 95% CI $[-0.58, -0.36]$), such that autistic participants were more likely to report higher childhood gender nonconformity than nonautistic participants; **(b)** RCGI significantly predicted sexual attraction (b path), $b = -0.24$, $SE = 0.03$, $p < .001$, 95% CI $[-0.30, -0.19]$, such that higher childhood gender nonconformity was associated with less heterosexual patterns of attraction, and; **(c)** the total effect of diagnostic status on sexual attraction was also significant, $b = 0.20$, $SE = 0.04$, $p < .001$, 95% CI $[0.13, 0.27]$. In contrast with the original sample, however, the direct effect of diagnostic status on sexual attraction was also significant in the replication sample, $b = 0.08$, $SE = 0.03$, $p = .016$, 95% CI $[0.02, 0.15]$. Yet crucially, the indirect effect was significant, providing evidence of partial mediation, $b = 0.11$, 95% bootstrap CI $[0.08, 0.15]$, with 55% of the total effect of diagnostic status on sexual attraction operating through childhood gender nonconformity.

3. Difference in moderation effects in Study 2 original and replication

The only result from the original study that diverged in the replication study concerned the moderating role of diagnostic status in the relation between recalled childhood gender nonconformity and categorical sexual identity. Specifically, in the original study, diagnostic status did not moderate the RCGI $\times$ sexual identity association, whereas it did in the replication study. The significant moderation effect in the replication Study 2 appears to have been driven more by an unusually steep slope in non-autistic participants ($b = -2.06$) than by an unusually shallow slope in autistic participants (-0.90), however. Table S4 shows the regression slopes and associated odds ratio for the RCGI $\times$ sexual identity association in each sample in the current studies. Across studies, the b in autistic participants in Study 2 (replication) is only slightly lower than the equivalent b in most of the other samples and is actually larger than the b among non-autistic participants in Study 1 (original). In contrast, the slope and odds ratio for non-autistic

participants in Study 2 (replication) are considerably larger than any others across studies, with participants having 7.86 times greater odds of being non-heterosexual for each one-point decrease in RCGI score. This odds ratio is more than double the size of the equivalent odds ratio in any other sample.

Table S4: Regression slopes (*b*) and associated odds ratio for the RCGI × sexual identity association in each sample

Study	Group	(<i>b</i>)*	Odds ratio**
Study 1 original	Non-autistic (at mean AQ)	−0.76	2.14
Study 1 replication	Non-autistic (at mean AQ)	−1.29	3.63
Study 2 original	Non-autistic	−1.28	3.61
Study 2 original	Autistic	−1.26	3.54
Study 2 replication	Non-autistic	−2.06	7.86
Study 2 replication	Autistic	−0.90	2.46

*all slopes were significant at $p \leq .001$, showing that the adjusted RCGI × Sexual identity association was significant in all samples; **Odds ratio expresses the increase in odds of non-heterosexual identity per 1-point RCGI decrease.