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Links between gender diversity and autism traits in non-autistic cisgender and transgender adults: Contributions of negative affect and alexithymia

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ABSTRACT

Background: Much of the literature on Autism–gender diversity (GD) relies on self-report Autism screening questionnaires (e.g. Autism-spectrum Quotient; AQ-50) that may tap constructs other than Autism.

Aims: In two studies, we tested whether links between GD traits or categorical gender modality (cisgender/transgender) and Autism traits in non-Autistic cisgender and transgender adults reflect variance shared with alexithymia/negative affect.

Methods: Study 1 ($N=285$ cisgender adults) measured Autism traits (AQ-50), alexithymia, negative affect, perceived discrimination, and dimensional GD. Study 2 ($N=208$; 104 transgender and 104 cisgender) measured primarily Autism traits, alexithymia, and negative affect. Hierarchical regressions tested whether dimensional GD (Study 1) and being transgender(Study 2) predicted AQ-50 scores before and after other measures were included. Additionally, (a) mediations examined the interplay between alexithymia, negative affect, and Autism traits; (b) regressions tested whether dimensional GD or being transgenderpredicted "Autism-specific" AQ-50 variance, and; (c) groups in Study 2 were matched on alexithymia and negative affect, with differences in AQ-50 tested using independent *t*-tests.

Results: Both dimensional GD indices (Study 1) and being transgender (Study 2) predicted higher Autism traits, but adding alexithymia and negative affect eliminated the predictive effects. Mediation analyses showed partial, bidirectional alexithymia-negative affect associations with Autism traits. Importantly, Autism-specific variance on the AQ-50 was not predicted by dimensional GD in Study 1 or transgender identity in Study 2. Moreover, the predictive effect of being transgender in Study 2 disappeared after matching groups on alexithymia and negative affect.

Discussion: In non-Autistic cisgender and transgender people, associations between dimensional GD or being transgender and self-reported Autism traits became nonsignificant after accounting for alexithymia and negative affect. These findings caution against inferring Autism-specific links from screener totals. Clinically, elevated Autism traits alone in non-Autistic transgender people should not guide decisions.

KEYWORDS
Autism screening tools; transgender; internalizing symptoms; emotional awareness; intersection

Introduction

Gender diversity (GD) encompasses identities/expressions that differ from sex-assigned-at-birth norms, including transgender and nonbinary identities (Coleman et al., 2022), and is sometimes accompanied by gender dysphoria (significant distress about the mismatch between one's sex assigned at birth and experienced gender; APA, 2022). Over the past decade, a body of research has supported the existence of a link between GD and Autism spectrum disorder (ASD), with a meta-analysis suggesting Autism diagnoses are ~11 times more common in the gender diverse than general population (Kallitsounaki & Williams, 2023a).

Though Autism and gender diversity are frequently discussed in *categorical* terms (that is, Autistic vs non-Autistic; cisgender vs gender diverse), these can also be measured *continuously/dimensionally* on the basis that traits associated with Autism and gender diversity are distributed throughout the population (e.g., Constantino & Todd, 2003; Ehrensaft, 2018).[1] Currently, a

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handful of measures of dimensional gender diversity exist, including the Gender Identity/Dysphoria Questionnaire (GIDYQ; Deogracias et al., 2007) and the Gender Self-Report (GSR; Strang et al., 2023), all of which are thought to index gender diversity as broadly defined. Similarly, multiple Autism screening measures exist to measure Autism traits, of which the most frequently used is the Autism-spectrum Quotient (AQ-50; Baron-Cohen et al., 2001), especially in adult case-control studies examining the GD-Autism intersection (11 out of 13 studies used the AQ). The AQ is a 50-item questionnaire designed to measure Autism traits in adults *via* self-report, and *via* parent-report in adolescents (Baron-Cohen et al., 2006), and children (Auyeung et al., 2008).

Studies using Autism screeners, such as the AQ, consistently report more Autism traits in children, adolescents, and adults referred to specialist gender clinics than in controls (e.g., Akgül et al., 2018; David et al., 2025; Heylens et al., 2018; Huisman et al., 2026; Leef et al., 2019; Nobili et al., 2018; Özel et al., 2025; Skagerberg et al., 2015; van der Miesen et al., 2018; Vermaat et al., 2018; but see Pasterski et al., 2014). Because Autism is overrepresented in gender clinic samples (Kallitsounaki & Williams, 2023a), most studies comparing gender-referred individuals with cisgender controls likely included Autistic participants in the gender-referred group. However, the majority of these studies did not report or control for *Autism diagnoses*. As a result, elevated mean Autism trait scores in gender-referred samples may be driven by the inclusion of Autistic individuals rather than reflecting higher Autism traits associated with gender diversity per se.

Likewise, community studies also find elevated Autism traits in transgender and nonbinary versus control groups (Kung, 2020; Stagg & Vincent, 2019), but often cannot rule out inclusion of *diagnosed* Autistic participants. Even so, work that controls for Autism diagnosis or samples exclusively non-Autistic GD participants still reports higher Autism traits than in non-Autistic cisgender groups (English et al., 2025; Hendriks et al., 2022; Kallitsounaki & Williams, 2022; Murphy et al., 2020; Warrier et al., 2020).

Complementary evidence from cisgender-only samples shows that current gender dysphoric traits correlate positively with Autism traits (Kallitsounaki et al., 2021; Kallitsounaki & Williams, 2020a, 2022 [Supplementary Information]).[2] In other words, cisgender people with elevated gender dysphoric traits also report an elevated number of Autism traits. Whilst such associations among Autism traits and gender diversity are potentially highly important, conclusions based on screening measures (e.g., AQ cutoffs or total scores) should be weighed carefully in the absence of appropriate gold-standard clinical assessments. This is because screening measures, such as the AQ, are sensitive to traits associated with other constructs and conditions, which in turn reduces their specificity to Autism.

On the one hand, Autism screeners (including the AQ) generally show adequate-to-excellent psychometric properties (e.g. internal consistency and test–retest reliability) and do separate Autistic from non-Autistic samples in broad comparisons (e.g., Lundqvist & Lindner, 2017; Pehlivanidis et al., 2025; Yoshinaga et al., 2023; for a review see Ruzich et al., 2015). On the other hand, precisely because they are screeners, their discriminant validity is often found to be insufficient to distinguish Autism from other conditions or from traits common in those conditions (e.g. Cholemkery et al., 2014; South et al., 2017). For example, focusing on the AQ, Ashwood et al. (2016) assessed 476 adults referred for an Autism diagnostic assessment and found that AQ scores did not predict whether patients received a consensus diagnosis of Autism. Instead, elevated AQ scores predicted diagnosis of anxiety disorder, leading the authors to conclude that "generalized anxiety disorder may 'mimic' Autism and inflate AQ scores, leading to false positives" (p. 2595). Other studies have found that the AQ also discriminates Autism poorly from schizophrenia-spectrum disorders (Wouters & Spek, 2011) and only modestly from attention deficit hyperactivity disorder (Sizoo et al., 2009), for example. Beyond clinical diagnosis, AQ scores also correlate robustly with anxiety and depressive symptoms (e.g., Galvin et al., 2022; Romano et al., 2014).

All of this causes a concern that the purported link between gender diversity and Autism traits (as indexed by screening measures) in non-Autistic populations may reflect a link between GD and traits/conditions that co-occur with Autism, such as anxiety/depression, rather than with Autism itself.

This concern is motivated not merely by awareness of statistical covariance between measures, but also by widely cited theoretical frameworks that make such links plausible. For example, minority stress theory proposes that exposure to stigma-related stressors contributes to elevated psychological distress and increased levels of anxiety and depression among gender (and sexual) minority groups (e.g., Chodzen et al., 2019; Meyer, 2003). Accordingly, when an Autism screening measure such as the AQ-50 is well known to covary with negative affect (e.g., Romano et al., 2014; White et al., 2023), it is theoretically plausible that associations between gender-related variables and AQ-50 could be driven, at least in part, by variance shared with internalizing symptoms rather than by Autism-specific trait variance. This can occur because AQ-50 items do not exclusively index Autism-specific mechanisms. For example, some items tap experiences and behaviors (e.g., social withdrawal/discomfort, heightened self-monitoring, and reduced social engagement) that can also be elevated by internalizing symptoms and chronic stress, increasing endorsement of items for reasons unrelated to Autism specifically. Indeed, Turban and van Schalkwyk (2018, p. 8) argued exactly this:

> "It is increasingly understood that such screening instruments can be nonspecific for ASD, with youth suffering from other emotional or behavioral problems having higher scores in the absence of ASD. Higher scores would be expected in youth with GD, because this population is known to have high rates of internalizing psychopathology."

The same logic motivated our inclusion of a measure of alexithymia. Alexithymia is a trait/condition that is characterized by difficulty with identifying and describing one's internal emotional states (Nemiah et al., 1976; Sifneos, 1973), and is associated with Autism (e.g., for a meta-analysis, see Kinnaird et al., 2019), but separable from it (e.g., Bernhardt et al., 2014; Cuve et al., 2022; Yorke et al., 2025). A number of studies have also observed an elevation of alexithymia traits among gender diverse adults (e.g., Kallitsounaki & Williams, 2023b; Kessler et al., 2006; Mazzoli et al., 2022). It is possible that attention to, and appraisal of, internal bodily cues is intertwined with gender dysphoria and trauma-related responses to minority stress in transgender and gender diverse people. Accordingly, when the AQ-50 is known to covary with alexithymia (e.g. Nicholson et al., 2019; Shah et al., 2016), it is theoretically plausible that associations between gender-related variables and AQ-50 score could be driven, at least in part, by variance shared with alexithymia-related processes rather than by Autism-specific trait variance. Once again, this is plausible because some AQ-50 items require self-referential emotional processing, and difficulties identifying and describing feelings could increase endorsement of certain items even when Autism-specific traits are not elevated. The current paper addresses these issues across two studies.

In Study 1, a community sample of 285 non-Autistic cisgender adults completed the AQ-50 (Baron-Cohen et al., 2001), as well as measures of anxiety, depression, and stress (the Depression Anxiety Stress Scale-21 [DASS-21; Henry & Crawford, 2005]), alexithymia (the Toronto Alexithymia Scale-20 [TAS-20; Bagby et al., 1994]), experiences of everyday discrimination (the Everyday Discrimination Scale [EDS; Williams et al., 1997]), and two dimensional measures of gender diversity: The GIDYQ (Deogracias et al., 2007) to index gender dysphoric traits, and the GSR (Strang et al., 2023) to measure binary and nonbinary experiences of gender diversity. The GIDYQ was selected primarily to allow comparability with the existing Autism-gender literature, as it is currently the most widely used measure of gender diversity in general population individual differences studies with adults (e.g., George & Stokes, 2018; Kallitsounaki & Williams, 2020a). However, the GIDYQ taps only one aspect of gender diversity, namely gender dysphoric traits. Importantly, not all people with experiences of gender diversity report distress related to their sex assigned at birth. Therefore, Study 1 also included the GSR, which captures both nonbinary and binary gender diversity experiences. Unlike the GIDYQ, the GSR focuses on gender-related traits, experiences, and goals that are affirming and bring comfort. The GSR was also developed using participatory methodology and has been validated across Autistic and non-Autistic individuals, as well as gender diverse and cisgender populations (Strang et al., 2023).

Why did we include a *non*-Autistic *cisgender* sample in Study 1 when our intention was to refine our understanding of the link between Autism and gender diversity? To reiterate the discussion above, a not-insubstantial portion of the evidence cited in support of a gender diversity-Autism intersection comes from individual-differences studies in cisgender or general-population samples, rather than from samples of transgender and gender diverse participants. In this literature, Autism is operationalized dimensionally (e.g., *via* AQ-50 scores). Likewise, "gender diversity" is usually operationalized dimensionally *via* continuous gender-related traits to reflect the fact that even categorically cisgender individuals vary in level of gender conformity/typicality across behavior, comfort, interests, or identity (e.g., Jacobson & Joel, 2018; Sandfort et al., 2021; Tabler et al., 2021). This dimensional *variation* in gender is itself associated with Autism traits (dimensional), such that less gender conformity/typicality is associated with more Autism traits (e.g., Kallitsounaki et al., 2021; Kallitsounaki & Williams, 2020a, 2020b; Munoz Murakami et al., 2022; van Der Miesen et al., 2024). Therefore, Study 1 was included to directly evaluate this portion of evidence for the gender diversity-Autism intersection under conditions designed to reduce potential confounds (i.e., inclusion of non-Autistic participants only, and key covariates measured and accounted for). Of course, care should be taken not to assume that results from studies of gender variation in cisgender people necessarily generalize to transgender and gender diverse people. For this reason, we employed a case-control design in Study 2 to address the same set of issues in transgender versus cisgender groups.

In Study 2, an independent community sample of 208 non-Autistic adults, half of whom identified as transgender (i.e., identified with a binary gender different from their sex assigned at birth), completed the AQ-50, the TAS-20, the DASS-21, and a self-report measure of gender dysphoric traits based on DSM-5 criteria for gender dysphoria (Kennedy et al., 2021).

Study 1: Method

Participants

Three hundred and forty-seven people completed this study. To be eligible, participants had to be located in the United Kingdom, report that they did not have a clinical diagnosis of Autism, and identify with the gender that corresponded with their sex assigned at birth. To ensure data quality, two attention checks were used. Participants who failed the attention checks ($n = 24$), reported an Autism diagnosis ($n = 26$), identified as gender diverse ($n = 9$), or did not report their gender modality ($n = 3$) were excluded. The final sample included 285 non-Autistic cisgender participants (85.96% assigned female at birth). Their mean age was 20.11 years ($SD = 4.84$, range = 17–64). In terms of ethnicity, 60% of participants were White, 20.35% Black, 11.58% Asian, 4.91% Mixed, and 3.17% were from other ethnic backgrounds.

Participants were recruited online through a Research Participation Scheme for undergraduate psychology students ($n = 266$) and from the general population ($n = 19$) by advertising the study on the social and professional networks of the researchers who conducted the data collection. Undergraduate students received course credit for partial fulfillment of their degree, and people from the general population did not receive any compensation for participation. All participants provided digital informed consent, and the study was approved by the Kent Psychology Research Ethics Committee (ID: 202417309076429326).

Measures and procedure

Gender identity/gender dysphoria questionnaire for adolescents and adults (GIDYQ)

The GIDYQ is a self-report measure of gender dysphoric traits (Deogracias et al., 2007). It includes 27 self-referential statements about participants' feelings, wishes, thoughts, and behaviors regarding their sex assigned at birth and their experienced gender (e.g., "In the past 12 months, have you felt unhappy about being a woman?"). Participants responded to each of the items using a 5-point scale, ranging from "never" to "always". The questionnaire has two analogous versions, one for individuals who were assigned male at birth and one for those who were assigned female at birth. Lower mean scores indicate more gender dysphoric traits.

Gender self report (GSR)

The GSR is a self-report measure of gender identity (Strang et al., 2023). It includes 30 self-referential statements (e.g., "I want people to see me as a male"), each of which participants rated on a 4-point scale, ranging from "never true" to "always true". The GSR comprises two factors: (a) the *Female–Male Continuum* (FMC) scale, which captures binary experiences of femaleness and maleness; and (b) the *Nonbinary Gender Diversity* (NGD) scale, which captures identification with neither, both, or another gender outside of binary categories. Higher scores on the FMC indicate greater identification with femaleness and lower identification with maleness, whilst lower scores on the FMC indicate lower identification with femaleness and higher identification with maleness. Higher scores on the NGD indicate stronger nonbinary identification.

Following Strang et al. (2023), FMC and NGD scores were transformed to range from 0 to 1. Then a *Binary Gender Diversity* (BGD) score was computed from FMC and sex assigned at birth. The BGD is the primary index of the distance between the participant's gender identity and their sex assigned at birth. Whereas the FMC provides directional information (femaleness vs maleness). Hence, for participants assigned female at birth, the BGD score equals 1 minus FMC; for participants assigned male at birth, the BGD score equals FMC. Thus, participants assigned male at birth with high BGD scores would have a high level of femaleness, and those assigned female at birth with high BGD scores would have a high level of maleness. To be clear, a higher BGD indicates greater alignment with characteristics of a binary gender different from one's sex assigned at birth, not that a participant necessarily identifies as that gender. For the purposes of this study, only the BGD and NGD scale scores were used as the primary GSR variables.

Autism-spectrum quotient (AQ-50)

The AQ-50 is a self-report measure of Autism traits (Baron-Cohen et al., 2001). It includes 50 self-referential statements (e.g., "I find it difficult to imagine what it would be like to be someone else"), which participants rated on a 4-point scale, ranging from "definitely agree" to "definitely disagree". Responses indicative of Autism were scored as 1, while non-indicative responses were scored as 0. Scores range from 0 to 50, with higher scores indicating more Autism traits.

Toronto alexithymia scale (TAS-20)

The TAS-20 is a self-report measure of alexithymia (Bagby et al., 1994). It includes 20 self-referential items regarding participants' awareness and understanding of, and ability to describe, their own bodily sensations and feelings (e.g., "I find it hard to describe how I feel about people"). Participants indicated their level of agreement with each statement using a 5-point scale that ranges from "completely disagree" to "completely agree". Total scores range from 20 to 100, with higher scores indicating greater alexithymia.

Depression anxiety stress scale-21 (DASS-21)

The DASS-21 is a self-report measure of negative affect (Henry & Crawford, 2005). It includes 21 items that tap on depression (e.g., "I felt that I had nothing to look forward to"), anxiety (e.g., "I felt scared without any good reason"), and stress (e.g., "I found it hard to wind down"). Participants rated the extent to which each statement applied to them over the past week using a 4-point scale ranging from "did not apply to me at all" to "applied to me very much, or most of the time". Following the original scoring guidelines, total sum scores were multiplied by two to match the original DASS-42 scale. Final total scores range from 0 to 126, with higher scores indicating more negative affect.

Everyday discrimination scale (EDS)

The EDS is a self-report measure of perceived discriminatory experiences (Williams et al., 1997). It includes nine items (e.g., "People act as if they think you are not smart"), with participants rating how often these events occurred on a 6-point scale ranging from "never" to "almost every day." Higher mean scores indicate more experiences of everyday discrimination.

All measures were completed online *via* Qualtrics.

Statistical analysis

Data were screened for missing data, and none were identified. For all the analyses below, the relevant assumptions were examined and were largely met. Where assumptions were violated, this is explicitly noted in the relevant analysis, together with how the issue was addressed.

Three separate hierarchical regressions were conducted. In each regression, AQ-50 total score was the outcome variable, age and sex assigned at birth were entered as predictor variables in Step 1, and TAS-20, DASS-21, and EDS scores were added as predictor variables in Step 3. What differed across the regressions was the measure of gender diversity used as a predictor variable at Step 2. Specifically, Regression 1 included the GIDYQ as the Step 2 predictor, Regression 2 included the BGD, and Regression 3 included the NGD. For each analysis, we reported explained variance (R^2), change in explained variance (ΔR^2), standardized (β) and unstandardized (*B*) coefficients, with standard errors and 95% confidence intervals. Standardized coefficients (β) are interpreted as Pearson's *r* correlation coefficients (values of .10, .30, .50 represent small, medium, and large effects, respectively) (Cohen, 1988).

To explore the interplay between alexithymia, negative affect, and Autism traits, two exploratory mediation analyses were conducted. In the first analysis, we examined whether *alexithymia* (M) mediates the relationship between negative affect (X) and Autism traits (Y). In the second analysis, we examined whether *negative affect* (M) mediates the relationship between alexithymia (X) and Autism traits (Y). Analyses were conducted using PROCESS v4.2 in SPSS, and unstandardized indirect effects were estimated using 5,000 bootstrap samples.

Next, we explored whether any associations between Autism traits (AQ-50) and gender-related measures (GIDYQ mean score; BGD score on the GSR; and NGD score on the GSR) identified in the initial regression analysis were independent of variance shared with alexithymia (TAS-20) and negative affect (DASS-21). To do this, total AQ-50 scores were regressed on TAS-20 and DASS-21 to gain the residuals that reflected Autism-specific variance unique to AQ-50, independent of variance shared with TAS-20 and DASS-21. This Autism-specific variance of the AQ was then used as the outcome variable in three separate linear regression, each of which has one of the gender-related variables as the predictor variable, alongside age and sex assigned at birth.

To be clear, by "Autism-specific variance", we mean the variance in AQ-50 scores after variance associated with these covariates has been partialled out. This does not guarantee that the remaining variance in AQ scores after this partialling process necessarily reflects uniquely and specifically Autism traits, but rather that measures AQ variance that is independent of variance shared with measures of related but separable constructs. Thus, in the current study, we estimated this Autism-specific variance in two ways. First, we produced regression models that showed the predictive effect of AQ score on gender-related outcome variables before and after including covariates. Second, we conducted additional regression analyses using a residualized AQ-50 score (i.e. AQ-50 variance that is independent of the covariates) as the predictor variable.

Bayesian analyses were also conducted to assess the relative evidence supporting the alternative hypothesis over the null (e.g., Dienes, 2014). Bayes factors ($BF_{10} > 1$) indicate increasing support for the alternative hypothesis ($BF_{10} > 1$, > 3, > 10, > 30, > 100 represent anecdotal, substantial, strong, very strong, and decisive evidence for H1, respectively), whereas those < 1 provide increasing evidence for the null hypothesis ($BF_{10} < 1$, < 0.33, < 0.10, < 0.03, and < 0.01 represent anecdotal, substantial, strong, very strong, and decisive evidence for H0, respectively). All Bayesian analyses were performed using R (version 4.3.3; R Core Team, 2024) and JASP 0.19.3 (JASP team, 2025).

Finally, at the request of an anonymous reviewer and for transparency, we report the results of exploratory sensitivity analyses after excluding ($n = 28$) participants who scored ≥ 32 on the AQ-50 (see Supplementary Materials), because any participant who scored above this threshold score is considered to have clinically significant levels of Autism traits (Baron-Cohen et al., 2001). However, we focus our interpretation on the results from full sample (before exclusion of those with AQ-50 scores ≥ 32) for several reasons. First, it is important to distinguish between Autism diagnosis and Autism trait scores. All participants in Study 1 reported that they did not have a formal diagnosis of Autism. Our critique of previous case-control studies concerned the failure to measure formal

Autism diagnoses in gender diverse clinic samples, where diagnosed Autism is overrepresented. In contrast, the AQ-50 is a continuous self-report measure of traits and above-threshold scores neither indicate a diagnosis, nor do they predict an Autism diagnosis in adults referred for a full diagnostic assessment (Ashwood et al., 2016). Therefore, excluding high AQ scorers would not be equivalent to excluding diagnosed Autistic participants. Second, such exclusion would truncate the trait distribution, which can weaken associations because of reduced statistical power (e.g., Zimmermann et al., 2017). For this reason, and in keeping with the dimensional approach of the study, we retained the full range of AQ-50 scores in the primary analyses, allowing an estimate of the relation between gender-related trait variance and the full continuum of Autism traits.

Table 1. Descriptive statistics for the key variables in Study 1.

Variable	*M*	*SD*
1. GIDYQ	4.85	0.20
2. BGD	0.07	0.08
3. NGD	0.03	0.08
4. AQ-50	21.16	7.33
5. TAS-20	52.91	12.16
6. DASS-21	45.59	27.01
7. EDS	1.51	0.87

Note. N = *285.* GIDYQ = Gender Identity/Gender Dysphoria Questionnaire; BGD = Binary Gender Diversity; NGD = Nonbinary Gender Diversity; AQ-50 = Autism-spectrum Quotient-50; TAS-20 = Toronto Alexithymia Scale-20; DASS-21 = Depression Anxiety Stress Scale-21; EDS = Everyday Discrimination Scale.

Study 1: Results

Main analyses

Descriptive statistics and correlations for the study variables are reported in Table 1 and Figure 1, respectively, and the results of each hierarchical regression are presented in Table 2. Each of the GD trait indices (GIDYQ, GSR-BGD, GSR-NGD) predicted higher AQ-50 at Step 2 in each regression (ΔR^2 = .01–.04; a 1–4% improvement across regressions). Adding TAS-20 and DASS-21 (and EDS) produced a far larger increment (ΔR^2 = .24–.26; a 24–26% improvement across regressions), and GD predictive effects were no longer significant in any of the regressions (Table 2). In standardized terms, a +1 *SD* change in DASS-21 or TAS-20 corresponded to +0.48 *SD* and +0.46 *SD* in AQ-50, respectively (total effects; see Figure 2).[3] Thus, once alexithymia and negative affect were included in the model, the associations of gender dysphoric traits, binary gender diversity, and non-binary gender diversity with Autism traits were no longer apparent.[4]

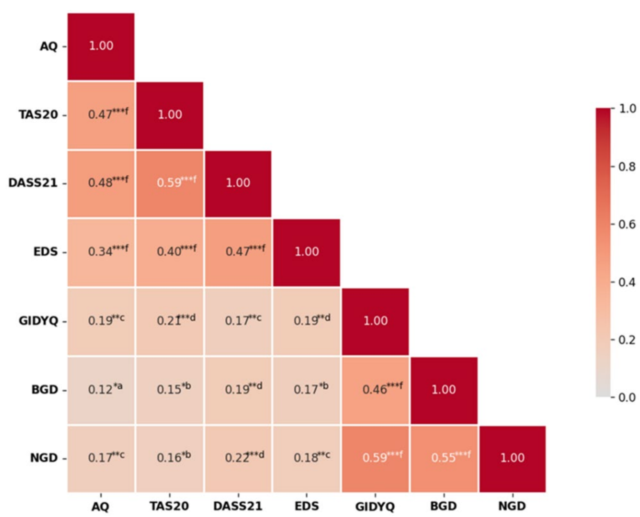

Figure 1. Correlation heatmap for *S*tudy 1.

Note. N = *285*. Note that DASS-21, GIDYQ, BGD, and NGD were not normally distributed. However, Pearson's *r* correlation is considered robust to violations of the normality assumption, particularly in larger samples (Edgell &Noon, 1984; Ghasemi & Zahediasl, 2012; Havlicek & Peterson, 1976). Therefore, no transformations or adjustments were made to the data. All correlations involving GIDYQ (low score = more gender dysphoric traits) were negative but are shown as positive in this figure for clarity of visualisation. GIDYQ = Gender Identity/Gender Dysphoria Questionnaire; BGD = Binary Gender Diversity; NGD = Nonbinary Gender Diversity; AQ-50 = Autism-spectrum Quotient-50; TAS-20 = Toronto Alexithymia Scale-20; DASS-21 = Depression Anxiety Stress Scale-21; EDS = Everyday Discrimination Scale.

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

$^{a}BF_{10} < 1$; $^{b}BF_{10} > 1$; $^{c}BF_{10} > 3$; $^{d}BF_{10} > 10$; $^{e}BF_{10} > 30$; $^{f}BF_{10} > 100$

Table 2. Hierarchical regression results for autism traits in Study 1.

	Regression 1 GD Predictor = GIDYQ						Regression 2 GD Predictor = BGD						Regression 3 GD Predictor = NGD					
	B	95% CI for *B*					*B*	95% CI for *B*					*B*	95% CI for *B*				
Predictor		*LL*	*UL*	*SE B*	β	BF_{10}		*LL*	*UL*	*SE B*	β	BF_{10}		*LL*	*UL*	*SE B*	β	BF_{10}
Step 1																		
Constant	26.51***	22.18	30.85	2.20			26.51***	22.18	30.85	2.20			26.51***	22.18	30.85	2.20		
Age	−0.20*	−0.38	−0.03	0.09	−.13	1.32	−0.20*	−0.38	−0.03	0.09	−.13	1.32	−0.20*	−0.38	−0.03	0.09	−.13	1.32
SAAB	−1.12	−3.58	1.34	1.25	−.05	0.33	−1.12	−3.58	1.34	1.25	−.05	0.33	−1.12	−3.58	1.34	1.25	−.05	0.33
Model Summary	$F(2, 282) = 3.24$, $p = .041$, $R^2 = .02$						$F(2, 282) = 3.24$, $p = .041$, $R^2 = .02$						$F(2, 282) = 3.24$, $p = .041$, $R^2 = .02$					
Step 2																		
Constant	60.90***	40.23	81.57	10.50			25.53***	21.12	29.94	2.24			25.75***	21.44	30.06	2.19		
Age	−0.19*	−0.37	−0.02	0.09	−.13	3.53	−0.22*	−0.39	−0.04	0.09	−.14	1.88	−0.19*	−0.36	−0.01	0.09	−.12	2.30
SAAB	−1.58	−4.01	0.85	1.24	−.08	1.08	−0.67	−3.15	1.82	1.26	−.03	0.39	−1.16	−3.59	1.27	1.24	−.06	0.66
GD	−7.02***	−11.15	−2.89	2.10	−.20	23.52	10.99*	0.41	21.57	5.38	.12	1.22	15.85**	4.78	26.91	5.62	.16	6.77
Model Summary	$F(3, 281) = 5.97$, $p < .001$, $R^2 = .06$, $\Delta R^2 = .04$, $p < .001$						$F(3, 281) = 3.58$, $p = .014$, $R^2 = .04$, $\Delta R^2 = .01$, $p = .042$						$F(3, 281) = 4.86$, $p = .003$, $R^2 = .05$, $\Delta R^2 = .03$, $p = .005$					
Step 3																		
Constant	22.57*	2.79	42.36	10.05			9.26***	3.89	14.63	2.73			9.34***	3.98	14.70	2.72		
Age	−0.05	−0.20	0.11	0.08	−.03	0.28	−0.05	−0.20	0.11	0.08	−.03	0.23	−0.04	−0.20	0.11	0.08	−.03	0.25
SAAB	−0.24	−2.42	1.94	1.11	−.01	0.25	−0.01	−2.21	2.19	1.12	< .01	0.20	−0.10	−2.27	2.07	1.10	−.01	0.23
GD	−2.59	−6.30	1.12	1.89	−.07	0.54	1.08	−8.26	10.42	4.74	.01	0.21	5.16	−4.69	15.01	5.00	.05	0.36
TAS-20	0.15***	0.07	0.22	0.04	.24	> 100	0.15***	0.08	0.23	0.04	.25	> 100	0.15***	0.08	0.23	0.04	.25	> 100
DASS-21	0.15***	0.08	0.22	0.04	.27	> 100	0.15***	0.08	0.22	0.04	.28	> 100	0.15***	0.07	0.22	0.04	.27	> 100
EDS	0.80	−0.16	1.76	0.49	.10	0.76	0.86	−0.10	1.82	0.49	.10	0.70	0.83	−0.13	1.79	0.49	.10	0.74
Model Summary	$F(6, 278) = 19.56$, $p < .001$, $R^2 = .28$, $\Delta R^2 = .24$, $p < .001$						$F(6, 278) = 19.13$, $p < .001$, $R^2 = .29$, $\Delta R^2 = .26$, $p < .001$						$F(6, 278) = 19.36$, $p < .001$, $R^2 = .30$, $\Delta R^2 = .25$, $p < .001$					

Note. All variance inflation factors (VIFs) < 2.5, indicating no multicollinearity concerns. CI = confidence interval; *LL* = lower limit; *UL* = upper limit; TAS-20 = Toronto Alexithymia Scale-20; DASS-21 = Depression Anxiety Stress Scale-21; EDS = Everyday Discrimination Scale. GIDYQ = Gender Identity/Gender Dysphoria Questionnaire; SAAB = sex assigned at birth; GD = gender diversity; BGD = Binary Gender Diversity; NGD = Nonbinary Gender Diversity.

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

Exploratory analyses

Mediation effects

Figure 2(a) shows the analysis of whether alexithymia (TAS-20) mediated the relationship between negative affect (DASS-21) and Autism traits (AQ-50). As per the main regression (above), the total effect of DASS-21 on AQ-50 was significant. DASS-21 predicted TAS-20 (a-path) and TAS-20 in turn predicted AQ-50 (b-path). When accounting for TAS-20, the effect of DASS-21 on AQ-50 remained significant (direct effect), with each 1 *SD* increase in DASS-21 being associated with a 0.33 *SD* increase in AQ-50 after adjusting for the mediating influence of TAS-20 (compared to +0.48 *SD* before adjustment). The indirect effect was also significant, $b = 0.04$, $SE = 0.01$, 95% CI [0.02, 0.07] indicating partial mediation. Overall, 31% of the effect of DASS-21 on AQ-50 was mediated by TAS-20.

Figure 2(b) shows whether negative affect (DASS-21) mediated the relationship between alexithymia (TAS-20) and Autism traits (AQ-50). The total effect of TAS-20 on AQ-50 was significant. TAS-20 predicted DASS-21 (a-path) and DASS-21 in turn predicted AQ-50 (b-path). When accounting for DASS-21, the effect of TAS-20 on AQ-50 remained significant (direct effect), with each 1 *SD* increase in TAS-20 being associated with a 0.28 *SD* increase in AQ-50 after adjusting for the mediating influence of DASS-21 (compared to +0.46 *SD* before adjustment). The indirect effect was also significant, $b = 0.11$, $SE = 0.03$, 95% CI [0.07, 0.17], indicating partial mediation. Overall, 39% of the effect of TAS-20 on AQ-50 was mediated by DASS-21.

The mediation analyses confirm that both TAS-20 and DASS-21 independently predict Autism traits, but clarify that part of their associations with AQ-50 reflect shared variance.

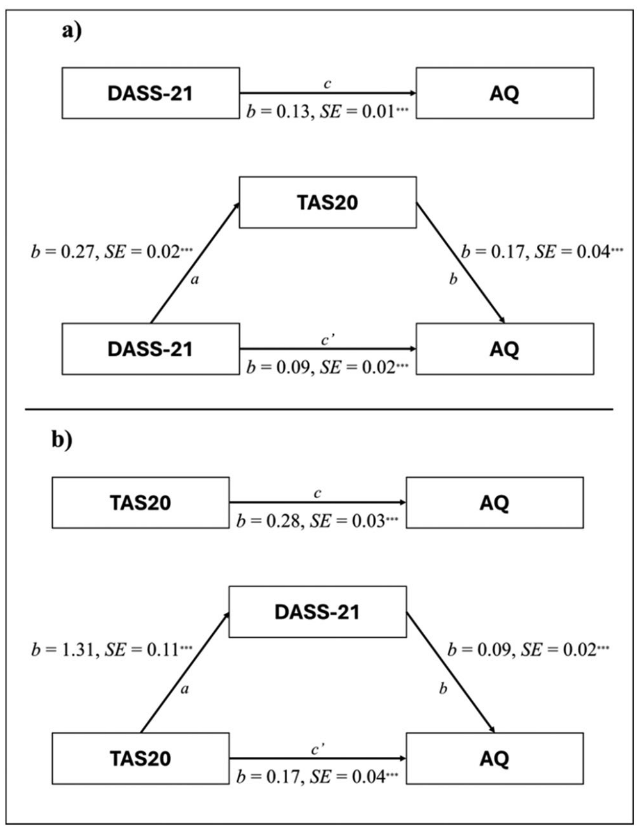

Figure 2. Results of mediation analyses for Study 1.
Note. a, b, c and c′ are path coefficients representing unstandardized regression coefficients (*b*) and standard errors (*SE*). The c path coefficient represents the total effect of DASS-21 (a) or TAS-20 (b) on AQ-50. The c-prime path coefficient refers to the direct effect of DASS-21 (a) or TAS-20 (b) on AQ-50. All analyzed paths were significant.
*** $p < .001$

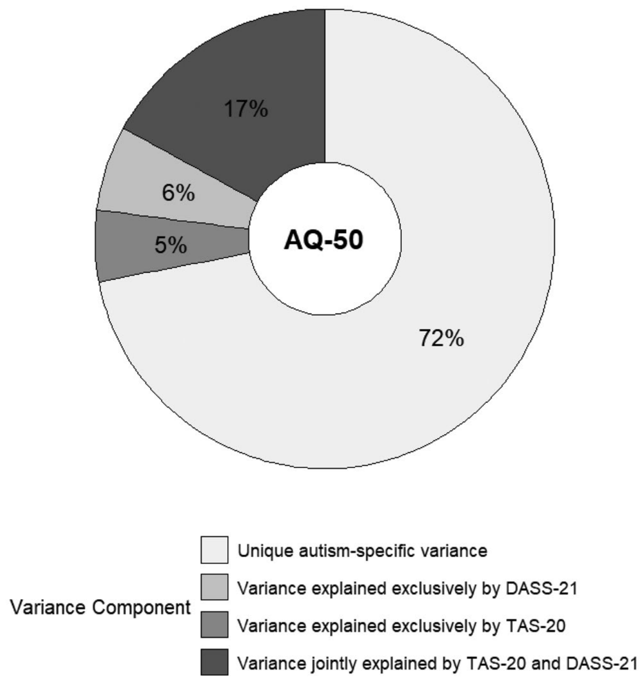

Figure 3. Breakdown of variance in AQ-50 score explained by TAS-20 and DASS-21 for Study 1.

Sensitivity analysis

Together, TAS-20 and DASS-21 explained 28% of the variance in AQ-50 scores, leaving 72% of the variance in AQ-50 specific to Autism traits independent of alexithymia and negative affect (see Figure 3). A series of linear regressions was conducted to examine whether any of the gender-related variables predicted this unique Autism-specific variance in AQ-50 independent of variance shared with TAS-20 and DASS-21. The analysis showed that none of the gender-related variables significantly predicted the AQ-50 residualized scores (GIDYQ: p = .138, BF_{10} = 0.38; BGD: p = .722, BF_{10} = 0.14; NGD: p = .263, BF_{10} = 0.25; for full results, see Table S2). Results suggest that once the AQ-50 variance shared with TAS-20 and DASS-21 was removed, the associations of gender dysphoric traits, binary gender diversity, and nonbinary gender diversity with Autism-specific variance were nonsignificant.

Study 2: Method

Participants

Two hundred and twenty-four adults participated in this pre-registered study. To be eligible, participants had to be located either in the United Kingdom or the USA, have English as their first language, and report that they did not have a clinical diagnosis of Autism, being in the process of receiving a diagnosis, or identify being on the Autism spectrum.[5] All participants were required to identify as men (including transgender men) or women (including transgender women). Additionally, for transgender participants, their current gender had to differ from their sex assigned at birth, whereas for cisgender participants, their current gender had to correspond with their sex assigned at birth. To ensure data quality, exceptionally fast submissions were automatically rejected by Prolific, and two attention checks and a Captcha Verification question were embedded within the survey. Participants who reported a nonbinary gender ($n = 9$) and those who provided inconsistent responses regarding their current gender and/or sex assigned at birth ($n = 7$) were excluded.[6]

The final sample included 208 participants, divided into two groups: cisgender ($n = 104$) and transgender ($n = 104$). Participant characteristics are presented in Table 3. Groups were matched for sex assigned at birth and age. In terms of ethnicity, 75.48% of participants were White, 11.06% Mixed, 7.21% Black, 5.77% Asian, and 0.48% were from other ethnic backgrounds. None of the participants reported having a formal diagnosis

Table 3. Sample characteristics and t-test statistics for age and GD in Study 2.

	Cisgender 47.12% AFAB	Transgender 47.12% AFAB				
Variable	*M* (*SD*)	*M* (*SD*)	*t*	*p*	*d*	BF_{10}
Age	31.67 (9.33)	31.53 (9.29)	0.11	.911	0.02	0.15
GD[a]	1.27 (0.48)	4.61 (0.51)	−48.45	< .001	−6.72	> 30
AQ-50	17.24 (8.05)	20.20 (7.63)	−2.72	.007	−0.38	4.71
TAS-20	44.57 (12.00)	48.29 (12.41)	−2.20	.029	−0.31	1.43
DASS-21[b]	26.13 (24.87)	41.75 (28.54)	−4.21	< .001	−0.58	> 30

Note. N = 208. AFAB = assigned female at birth; GD = DSM-5 criteria for gender dysphoria; AQ-50 = Autism-spectrum Quotient-50; TAS-20 = Toronto Alexithymia Scale-20; DASS-21 = Depression Anxiety Stress Scale-21.
[a]Due to violation of the normality assumption, results of a nonparametric Mann–Whitney U test are also reported: $U = 15.00$, $z = -12.59$, $p < .001$.
[b]Due to violation of the homogeneity of variances assumption, Welch's *t*-test results are reported.

of Autism, being in the process of receiving a diagnosis, or identified being on the Autism spectrum.

All participants were recruited online through the crowdsourcing platform Prolific Academic, which has been found to produce higher-quality data compared to other online platforms (e.g., Douglas et al., 2023; Esch et al., 2025) and has been used widely in research involving gender diverse populations (e.g., Boskey et al., 2023; Subedi et al., 2025). They all participated after providing digital informed consent, and received monetary compensation for their time and effort. The study was approved by the Kent Psychology Research Ethics Committee (ID: 2025175343711910069) and was preregistered on Open Science Framework (preregistration can be viewed here: https://osf.io/3p98r/overview?view_only=9cdbc3b4e1114473bb3010ff-faf4e43b).

Measures and procedure

All participants completed the AQ-50 (Baron-Cohen et al., 2001), the TAS-20 (Bagby et al., 1994), and the DASS-21 (Henry & Crawford, 2005) online *via* Qualtrics. Additionally, participants completed a 7-item questionnaire based on the DSM-5 criteria of gender dysphoria for adolescents and adults. This measure was originally designed as a parent-report measure (Kennedy et al., 2021), but for the purposes of this study a self-report adaptation of it was used. Participants responded on a series of statements about their feelings and experiences regarding their sex assigned at birth and experienced gender (e.g., "I express a desire to be a gender different from the one assigned to me at birth") using a 5-point scale ranging from "strongly disagree" to "strongly agree" (for the full item wording of the measure, see the Supplementary Materials).[7] Mean scores were calculated, with higher scores denoting greater gender dysphoria. Note that transgender participants reported significantly greater gender dysphoria than cisgender participants, in keeping with their gender diverse identity (see Table 3). Note that the EDS was not included in this study, as it did not predict Autism traits in any of the regressions conducted in Study 1.

Statistical analysis

Data were screened for missing data, and none were identified. For all the analyses below, the relevant assumptions were examined and were largely met. Where assumptions were violated, this is explicitly noted in the relevant analysis, together with how the issue was addressed.

Following Study 1, a hierarchical regression included AQ-50 total scores as the outcome variable, with age and sex assigned at birth as predictors in Step 1, gender modality status (cisgender/transgender) as a categorical predictor in Step 2 (cisgender = reference group), and TAS-20 and DASS-21 as predictors in Step 3.

Mirroring Study 1, we ran two exploratory mediation models: (i) alexithymia mediating the effect of negative affect on Autism traits, and (ii) negative affect mediating the effect of alexithymia on Autism traits. To assess robustness, we compared AQ-50 scores between transgender and cisgender participants after propensity-score matching on age, sex assigned at birth, TAS-20, and DASS-21 using the MatchIt package in R (Bang et al., 2020). This pairs each participant in the reference group with a participant in the comparison group with the closest propensity score. As common, balance was improved by applying a caliper width of 0.20 of the *SD* of the logit of the propensity score (e.g., Austin, 2011). We also computed AQ-50 residuals from TAS-20 and DASS-21 and regressed these "Autism-specific" residuals on gender modality. Lastly, at the request of an anonymous reviewer and for transparency, we report the results of exploratory sensitivity analyses after excluding participants who scored ≥ 32 on the AQ-50 (see Supplementary Materials). However, we focus our interpretation on the results from the full sample (before exclusion of those with AQ-50 scores ≥ 32) for reasons described in Study 1.

Study 2: Results

Main analyses

Table 3 shows the descriptive statistics for each group on each variable and associated inferential statistics. The results of the hierarchical regression analysis are presented in Table 4. In Step 1, age and sex assigned at birth were nonsignificant. Adding gender modality status (cisgender/transgender) in Step 2 improved model fit (ΔR^2 = .04), with being transgenderpredicting higher AQ-50 scores. In Step 3, inclusion of TAS-20 and DASS-21 produced a larger improvement (ΔR^2 = .33), with both being significant predictors of AQ-50, but gender-identity status no longer significant. Thus, once alexithymia and negative affect were included in the model, the association of gender modality with Autism traits was no longer apparent.[8]

Table 4. Hierarchical regression results for autism traits in Study 2.

Predictor	*B*	95% CI for *B* *LL*	*UL*	*SE B*	β	BF_{10}	R^2	ΔR^2
Step 1							< .01	< .01
Constant	18.02***	13.10	22.95	2.50				
Age	0.04	−0.08	0.16	0.06	.04	0.12		
SAAB	−0.30	−2.49	1.90	1.11	−.02	0.11		
Step 2							.04	.04**
Constant	16.51***	11.53	21.48	2.52				
Age	0.04	−0.08	0.15	0.06	.04	0.26		
SAAB	−0.30	−2.46	1.87	1.10	−.02	0.24		
gender modality	2.97**	0.82	5.12	1.09	.19	2.32		
Step 3							.33	.29***
Constant	2.03	−3.67	7.73	2.89				
Age	0.07	−0.03	0.17	0.05	.08	0.64		
SAAB	0.18	−1.65	2.00	0.93	.01	0.31		
gender modality	0.93	−0.95	2.82	0.96	.06	0.44		
TAS-20	0.24***	0.16	0.33	0.05	.38	> 100		
DASS-21	0.07***	0.03	0.11	0.02	.25	> 100		

Note. All variance inflation factors (VIFs) < 2.5, indicating no multicollinearity concerns.CI = confidence interval; *LL* = lower limit; *UL* = upper limit; SAAB = sex assigned at birth; TAS-20 = Toronto Alexithymia Scale-20; DASS-21 = Depression Anxiety Stress Scale-21; Gender identity = cisgender (0)/transgender (1).
**$p < .01$. ***$p < .001$.

Exploratory analyses

Mediation effects

Figure 4(a) shows the analysis of whether alexithymia (TAS-20) mediated the relationship between negative affect (DASS-21) and Autism traits (AQ-50). As per the main regression (above), the total effect of DASS-21 on AQ-50 was significant. DASS-21 predicted TAS-20 (a-path) and TAS-20 in turn predicted AQ-50 (b-path). When accounting for TAS-20, the effect of DASS-21 on AQ-50 remained significant (direct effect), with each 1 *SD* increase in DASS-21 being associated with a 0.27 *SD* increase in AQ-50 after adjusting for the mediating influence of TAS-20 (compared to +0.41 *SD* before adjustment). The indirect effect was also significant, $b = 0.06$, $SE = 0.01$, 95% CI [0.03, 0.08] indicating partial mediation. Overall, 46% of the effect of DASS-21 on AQ-50 was mediated by TAS-20.

Figure 4(b) shows whether negative affect (DASS-21) mediated the relationship between alexithymia (TAS-20) and Autism traits (AQ-50). The total effect of TAS-20 on AQ-50 was significant. TAS-20 predicted DASS-21 (a-path) and DASS-21 in turn predicted AQ-50 (b-path). When accounting for DASS-21, the effect of TAS-20 on AQ-50 remained significant (direct effect), with each 1 *SD* increase in TAS-20 being associated with a 0.37 *SD* increase in AQ-50, after the mediating influence of DASS-21 was accounted for (compared to +0.51 *SD* before adjustment). The indirect effect was also significant, $b = 0.09$, $SE = 0.03$, 95% CI [0.04, 0.16], indicating partial mediation. Overall, 27% of the effect of TAS-20 on AQ-50 was mediated by DASS-21.

Sensitivity analyses

As a follow-up sensitivity analysis, we matched groups for negative affect and alexithymia and conducted an independent *t*-test to examine

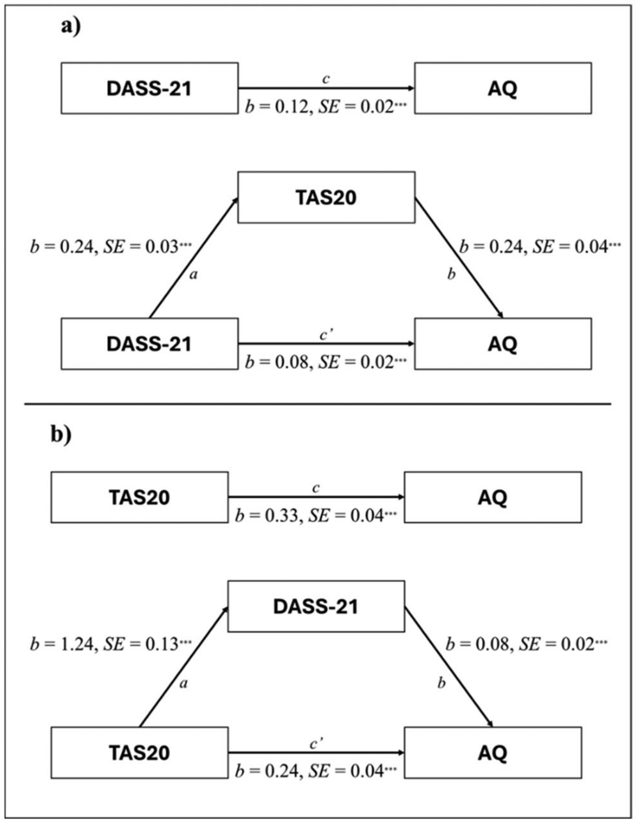

Figure 4. Results of mediation analyses for Study 2.
Note. a, b, c and c′ are path coefficients representing unstandardized regression coefficients (*b*) and standard errors (*SE*). The c path coefficient represents the total effect of DASS-21 (a) or TAS-20 (b) on AQ-50. The c-prime path coefficient refers to the direct effect of DASS-21 (a) or TAS-20 (b) on AQ-50. All analyzed paths were significant.
*** $p < .001$

whether the between-group difference in Autism traits observed in the unmatched sample persisted in the matched sample (see Table 5). Once matched for negative affect and alexithymia, the previously significant difference between transgender and cisgender participants in Autism traits ceased to be significant.

Next, a linear regression was conducted to examine whether gender modality (cisgender/transgender) predicted the residualized Autism-specific variance in AQ-50. Together, TAS-20 and DASS-21 explained 32% of the variance in AQ-50 scores, leaving 68% of the variance in AQ-50 specific to Autism traits independent of alexithymia and negative affect (very similar to the effects in Study 1; see Figure 5). The regression analysis showed that gender modality was not a significant predictor of residualized AQ-50 score (p = .334, BF_{10} = 0.23; for full results see Table S4), suggesting that once the variance shared with TAS-20 and DASS-21 was removed, cisgender and transgender participants showed similar levels of Autism traits.

General discussion

Across two studies, using independent samples and multiple operationalizations of gender diversity, we observed a consistent pattern of findings. In Study 1, we replicated the multiple studies showing an association in non-Autistic cisgender people between dimensional gender diversity traits and dimensional Autism traits. However, these associations were no longer evident once alexithymia and negative affect were accounted for. In Study 1 (cisgender community sample), each of the measures of gender diversity (the GIDYQ and GSR) predicted AQ-50 scores, after the effects of age and sex assigned at birth were accounted for (ΔR^2 = 1-4%). However, adding alexithymia (TAS-20) and negative affect (DASS-21), as well as perceived everyday discrimination (EDS), produced a much larger increase in explained

Table 5. Group comparisons on autism traits after matching for sex assigned at birth, age, negative affect, and alexithymia.

	Cisgender (n = 85) 47.06% AFAB[a]	Transgender (n = 85) 48.04% AFAB				
Variable	*M* (*SD*)	*M* (*SD*)	*t*	*p*	*d*	BF_{10}
Age[b]	32.46 (9.58)	31.19 (9.10)	0.89	.377	0.14	0.24
DASS-21	31.34 (24.34)	34.45 (25.68)	−0.81	.420	−0.12	0.23
TAS-20	46.18 (11.74)	46.96 (11.82)	−0.44	.663	−0.07	0.18
AQ-50	18.18 (8.21)	18.91 (7.24)	−0.61	.540	−0.09	0.20

Note. N = 170. AFAB = assigned female at birth; DASS-21 = Depression Anxiety Stress Scale-21; TAS-20 = Toronto Alexithymia Scale-20; AQ-50 = Autism-spectrum Quotient-50.
[a]Groups were also matched for sex assigned at birth (p = .878, φ = -0.01, BF_{10} = 0.19).
[b]Due to violation of the normality assumption, results of a nonparametric Mann–Whitney U tests are also reported for Age (U = 3.32, z = -0.90, p = .366), DASS-21 (U = 3.34, z = -0.84, p = .400), and TAS-20 (U = 3.46, z = -0.46, p = .643).

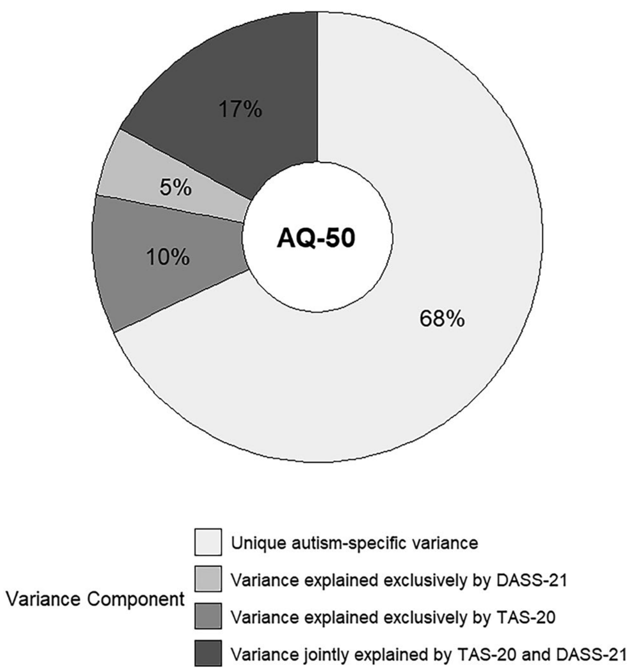

Figure 5. Breakdown of variance in AQ-50 score explained by TAS-20 and DASS-21 for Study 2.

variance (ΔR^2 = 24-26%). After accounting for this additional variance, each of the gender diversity trait predictors ceased to be significant predictors of AQ-50 score.

Study 2 replicated this pattern in groups of transgender versus age- and sex-assigned-at-birth-matched cisgender adults. After age and sex assigned at birth were accounted for, gender modality status (cisgender/transgender) was a significant predictor of AQ-50 score (ΔR^2 = 4%). Once again, however, this predictive effect disappeared after adding TAS-20 and DASS-21 (ΔR^2 = 33%). Mediation analyses in both studies indicated partial, bidirectional links between alexithymia and negative affect in relation to AQ-50 variance. Finally, "Autism-specific" variance in AQ-50 (estimated using residualized scores) was unrelated to any gender diversity indicator in either study and group differences in AQ-50 score were eliminated once groups were matched for negative affect and alexithymia, with Bayes factors consistently favoring the null.The large degree of variance in AQ-50 accounted for by alexithymia and negative affect (~28-31%), alongside the loss of gender diversity's predictive effects after accounting for this shared variance, suggests that the previously reported association between Autism traits and gender diversity traits in non-Autistic samples may have been artificially inflated by AQ measurement error (Hendriks et al., 2022; Kallitsounaki & Williams, 2022; Murphy et al., 2020; Warrier et al., 2020). This mirrors concerns about discriminant validity of Autism screeners and their sensitivity to emotion-related difficulties in addition to Autism. The current results indicate that, when Autism traits are measured in non-Autistic populations by self-report screeners, the observed link to gender diversity may reflect *non*-Autism-specific factors. We confirmed this in several ways.

First, we isolated "Autism-specific" AQ variance by residualizing AQ-50 onto TAS-20 and DASS-21. None of the gender diversity variables predicted this Autism-specific variance in AQ-50 (i.e., the residual) in Study 1, and neither did transgender status predict it in Study 2, with Bayes factors consistently favoring the null. If a specific overlap between gender diversity and Autism traits existed, then this association should persist when examining unique and isolated AQ-50 variance (i.e., that variance not shared with alexithymia or negative affect), but it did not in either study.

Second, we matched transgender and cisgender groups on TAS-20 and DASS-21, after which group differences in AQ-50 disappeared, again with Bayes factors strongly favoring the null. Again, if gender diversity was associated with Autism-*specific* traits in non-Autistic samples, then matching closely for alexithymia and negative effect should leave sufficient AQ-specific variance to detect differences between gender modality groups (cisgender/transgender) on AQ-50 score.

If elevated Autism screener scores among gender diverse non-Autistic people do not reflect an elevation of Autism-specific traits, then what do they reflect? There are two plausible answers. First, there could be a common underlying (biological or environmental) factor that contributes to Autism traits, alexithymia, and negative affect, and it is *this* latent factor that is linked to gender diversity. Second, there could be measurement error inherent in Autism screeners such that responses reflect not only Autism-specific traits, but also facets unrelated to Autism. This measurement error could take the form of (a) common method variance (e.g., same responder, measurement occasion, and/or response format), which induces covariation across measures through individual response biases, or (b) measure contamination, where items on the AQ-50 mistakenly tap alexithymia and/or negative affect rather than Autism-specific features.

Arguably, measurement error is the more parsimonious explanation of our findings. First, the fact that the same rater completed all measures in the current studies immediately risks measurement error through common method variance. Second, Autism screeners tend to be very broad, increasing the chance that some items will tap constructs that are not specific to Autism. For example, whilst endorsement of items 22 and 46 of the AQ-50 ("*I find it hard to make new friends*" and "*New situations make anxious*", respectively) may flag possible Autism, these items also describe psychological states that are likely to be common in *non*-Autistic people with internalizing difficulties such as anxiety and depression. In transgender samples, elevated minority stress exposure might further inflate the likelihood of scoring positively on these items without implying Autism-specific liability. However, it should be noted that in Study 2 we did not include a minority stress measure to directly test this hypothesis.

Much of the evidence concerning the gender diversity-Autism intersection has come from studies that have used Autism screeners in non-Autistic samples. Some studies have shown elevated total scores on Autism screeners among non-Autistic gender diverse people (Akgül et al., 2018; English et al., 2025; Kallitsounaki & Williams, 2022; Kennedy et al., 2025; Murphy et al., 2020; Warrier et al., 2020). Others have shown an association between gender diversity traits and Autism screener total scores in general population samples (e.g., Kallitsounaki et al., 2021; Kallitsounaki & Williams, 2020a, 2022; van der Miesen et al., 2024). Hence, the conclusions drawn from these studies, namely that gender diversity is specifically associated with Autism traits, are called into question by the findings of the current research.

Of course, these findings do *not* question that some gender diverse people are Autistic, or that there is an over-representation of valid Autism diagnoses in gender diverse populations. In clinical practice, diagnostic processes should involve multi-disciplinary assessment, using diagnostic tools in addition to screeners to inform expert judgment about the appropriate diagnosis. Reassuringly, studies have shown that gender diverse people with a formal diagnosis of Autism show a pattern of difficulties on these in-depth clinical measures, as well as screeners, that is entirely consistent their Autism diagnosis (Kallitsounaki et al., 2025a). Equally, other studies have shown that gender identity profiles in diagnosed Autistic people are not quantitatively or qualitatively different from gender identity profiles in non-Autistic people (Fischbach et al., 2025; Fysh et al., 2026; Kallitsounaki et al., 2025b). Therefore, our results provide *no reason* to question the validity of the gender diversity-Autism link in samples of people diagnosed with Autism. Rather, our findings emphasize the multi-faceted nature of behavior, and that it is possible for similar behavioral profiles to manifest from different developmental pathways, of which Autism is only one. In other words, whilst Autism screeners tap Autism traits, they also tap traits that *covary with* Autism traits but *are not* Autism-specific. It may be the inadvertently tapping of these covariates that has produced the consistently-reported association between gender diversity and Autism traits in non-Autistic populations.

The key takeaway from our results is, in our view, that elevated scores on Autism screeners (the AQ-50, specifically) do not necessarily index elevated Autism-specific traits/characteristics. Autism screeners are widely understood to be nonspecific for Autism and some variance in scores results from the screener inadvertently measuring traits that are not Autism traits themselves, but which may frequently covary with Autism. If no measurement of or statistical control of likely covariates is made, then scores on the AQ-50 will likely measure non-Autism-specific traits. It could be these non-Autism-specific traits, rather than Autism traits themselves are elevated in transgender adults and/or associated with gender-related variation in cisgender populations. The important thing is not to make a category mistake by attributing variance not attributable to Autism traits to Autism traits. In the current study, results imply that alexithymia and negative affect (internalizing symptoms) (a) covary with AQ-50 scores, and (b) are elevated in non-Autistic transgender adults and related to gender-related variation in non-Autistic cisgender adults. This may have led previous studies to conclude from gender × AQ associations that Autism traits are specifically related to gender variance and being transgender when, in fact, those associations reflect (at least in part) relations between gender variance and alexithymia and negative affect. Of course, we should note that the findings of Study 2 cannot necessarily be generalized to other gender groups (e.g. nonbinary individuals). Furthermore, the cross-sectional design of the current studies does not allow us to make any claims about the stability of the observed associations across the lifespan in non-Autistic transgender people. Future research could usefully investigate whether distinct Autistic trait profiles exist across different gender groups (e.g., nonbinary, agender), especially using a longitudinal design.

Could it be that unmeasured traits/characteristics/conditions could have influenced the results of the current studies and thereby invalidate the interpretation we make? This is theoretically possible, but highly unlikely. Once the gender × AQ associations are already attenuated to near-zero after controlling for TAS-20 and DASS-21 scores, adding further covariates might further reduce the magnitude of the gender × AQ-50 associations even closer to zero (or to zero), but this would not change our results substantively or their interpretation. In other words, including additional covariates is very unlikely to increase the gender × AQ associations and reinstate statistical significance. Only such an increase would alter results and conclusions substantively. To do this, the unmeasured covariate would need to be a "suppressor variable", defined by Conger (1974, p. 36-37) as "a variable which increases the predictive validity of another variable (or set of variables) by its inclusion in a regression equation". More specifically, the unmeasured variable would need to be related to gender-related predictors and to AQ-50 in such a way that omitting it causes the covariate-adjusted gender × AQ associations to be underestimated/suppressed. We are not aware of a plausible candidate for suppression in this context, but future research might reveal one.

Furthermore, what do the current results tell us about the relation of the covariates to gender diversity (categorically and/or dimensionally operationalized)? In Study 1, variation in gender-related traits, measured by the GIDYQ and GSR, was associated significantly with alexithymic traits and negative affect. Mirroring this, transgender participants in Study 2 had significantly higher negative affect and alexithymic traits than cisgender participants. These findings replicate the many others that observe elevated internalizing symptoms in transgender adults (e.g., Borgogna et al., 2019; Perez-Brumer et al., 2017), as well as the few studies that have investigated alexithymia in this population (Kallitsounaki & Williams 2023b; Kessler et al., 2006; Mazzoli et al., 2022). Elevated negative affect in transgender adults is plausibly attributed to elevated minority stress associated with their gender identity (Chodzen et al., 2019; Meyer, 2003). Likewise, elevated alexithymic traits could be a (partial, at least) consequence of gender diversity, maybe *via* minority stress. Perhaps, for example, distress relating to gender-related bodily functions (e.g., menstruation) shapes bodily awareness in some transgender people, which increases alexithymic traits scores. While these explanations are entirely plausible, the direction of effect could be opposite (cf. Bailey, 2020, regarding mental health in minority sexual groups). The current study does not speak to the issue of causality and nor need it to achieve the aim of establishing the specificity of relations between gender diversity (categorically and dimensionally operationalized) and Autism screener scores in non-Autistic people. Moreover, it is important to be clear that the relatively modest elevation in alexithymic trait scores among transgender participants in Study 2 could reflect measurement error in the TAS-20, rather than signaling a true elevation in alexithymia specifically. If anything should be taken away from the central findings of the current paper, it is that some caution should always be exercised when interpreting relations between variables when relevant covariates are unaccounted for. Further research dedicated to understanding these relations would be beneficial.

Future studies could investigate this in more depth by conducting regression analyses within a bifactor model. Bifactor models allow partitioning of the latent/general factor (common variance across AQ-50, TAS-20, DASS-21) from specific factors that uniquely tap Autism traits (AQ-specific), alexithymia (TAS-specific), and negative affect (DASS-specific). If gender diversity predicts the general factor after the specific factors are accounted for, then this would confirm the association operates *via* shared, non-Autism-specific variance. To distinguish a common underlying factor from measurement overlap, data from multiple informants could be modeled. If the link between the general factor and gender diversity holds in bifactor models of both informant-report and self-report data, then a substantive common factor would be implied. In contrast, if the link were confined to either self- or informant-reports (or if the link disappeared when a method variable was included in a single model), then measurement error would be suggested. Regardless, the current study strongly suggests that an association between gender diversity (traits or identity status) and Autism traits (indexed by screener total score) in non-Autistic populations does not show that gender diversity is related to Autism specifically.

It is also important to note that the AQ-50 has not yet been validated in transgender and gender diverse populations. It was selected as the Autism trait measure in Studies 1 and 2 primarily to ensure comparability with the existing gender-Autism literature, where it is by far the most commonly used measure of Autism traits in both predominantly cisgender/general population individual differences studies and case-control studies involving transgender and gender diverse participants. Indeed, in 11 of the 13 identified adult case-control studies, the AQ has been used to assess Autistic traits. Likewise, apart from the Gender Self-Report, which was used in one previous study (Thomas et al., 2025), no other measure has been used in general adult population individual differences studies. Using the AQ-50 therefore allowed us to position our findings directly in relation to prior work that has formed the basis for claims about a gender diversity-Autism link, and to test our central argument about the extent to which AQ total scores may reflect non-Autism-specific variance. Future research might usefully seek to replicate the present findings using measures that have been found to be psychometrically robust among transgender/gender diverse people (e.g., the Comprehensive Autistic Trait Inventory; English et al., 2025).

In relation to clinical practice, our findings highlight the importance of interpreting elevated scores on Autism screening measures (e.g., AQ-50) in transgender adults within a broader psychological and social context. More specifically, clinicians should avoid treating elevated Autism screener scores in isolation as evidence of Autism or Autism traits, particularly given the overlap between traits measured with Autism screening tools and experiences associated with anxiety, depression, alexithymia, and minority stress. Comprehensive assessment that includes a structured diagnostic evaluation, developmental history, and careful consideration of current mental health and psychosocial stressors, is essential when Autism is suspected. Equally important, elevated Autism traits in transgender adults should also prompt a thorough assessment of internalizing difficulties, broader mental health concerns, and potential trauma-related experiences. Such a trauma-informed and context-sensitive approach supports accurate differential diagnosis and ensures that individuals receive appropriate, tailored, and timely care and support.

Notes

1. Identity-first language (e.g., "Autistic person") is used throughout this paper because some Autistic adults prefer this and consider it less stigmatizing than person-first language (e.g., "person with Autism"; Kenny et al., 2016; Robertson et al., 2025; but see De Laet et al., 2025). However, we note that there is no clear consensus on this across the samples of Autistic people who have been surveyed (Schuck et al., 2025), so acknowledge that some have no preference and others prefer person-first language. We also use "Autistic" with a capital A. Similar to the Deaf community, many Autistic adults capitalize "Autistic" to emphasize belonging to a shared community and to acknowledge collective struggles for acceptance and social justice. However, we acknowledge that this preference will likely not apply to all Autistic individuals.
2. Kallitsounaki et al. (2021) did not make clear whether their sample included any gender diverse participants. However, we can confirm that only two participants in that sample reported a gender identity different from their sex assigned at birth. When these participants were excluded, the relationship between Autism traits and gender dysphoric traits remained the same as before their exclusion.
3. Δ AQ-50 (*SD* units) per +1 *SD* predictor is computed as b × ($SD_{predictor}/SD_{AQ}$). For example, in Study 1, SD_{AQ} = 7.33 and SD_{DASS} = 27.01 (see Table 1). Thus, for the total effect of DASS on AQ: 0.13 × (27.01/7.33) = 0.48 *SD* change.
4. The main analyses were rerun after excluding participants who scored ≥ 32 on the AQ-50. Results did not change substantively, apart from three bivariate correlations (i.e., AQ-50 × BGD; AQ-50 × NGD; and NGD × EDS) that lost their statistical significance in the reduced sample. However, *Z* tests revealed that none of these associations was significantly smaller in the reduced sample than in the full sample, suggesting a reduction in statistical power from truncation of AQ-50 variation and smaller sample size led to the losing of significance in the reanalyses. Full results are reported in the Supplementary Materials. Even if this interpretation is incorrect and bivariate associations in the full sample were driven by undiagnosed Autism cases, this would show only that our concern about composition bias in previous studies is reasonable/valid.
5. The inclusion criteria in Study 2 differed from Study 1 due to differences in the recruitment method. Participants in Study 2 were recruited through Prolific. Therefore, English as a first language was included as an eligibility criterion to ensure that participants could understand and complete the study measures. Recruitment was also extended to participants in the USA to ensure that the target sample size could be achieved.
6. The majority of studies examining Autism traits in transgender and gender diverse adults either did not distinguish between these groups and included anyone who identified with a gender different from their sex assigned at birth or did not examine transgender and nonbinary participants separately. Thus, it is unclear what drove the between-group differences observed in these studies. Therefore, in the current study we decided to focus exclusively on transgender people.
7. The seven-item self-report questionnaire based on DSM-5 criteria for gender dysphoria may not fully capture the lived experience of people with gender dysphoria. In this study, it was included solely as a descriptive measure to aid characterization of the groups. As specified in the pre-registration, it was not intended for any further analyses.
8. The main analyses were rerun after excluding participants who scored ≥ 32 on the AQ-50. (cisgender $n = 5$; transgender $n = 7$). Results did not change substantively. That is, no *p* value that was significant in the full sample results was nonsignificant in the reduced sample results, and vice versa, no *p* value that was nonsignificant in the full sample results was significant in the reduced sample results (see Supplementary Materials for full results).

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Ethical approval

All procedures performed in studies involving human participants were in accordance with the ethical standards of the Kent Psychology Research Ethics Committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

Informed consent

Informed consent was obtained from all individual participants included in the study.

Author contributions

CRediT: **Aimilia Kallitsounaki**: Conceptualization, Data curation, Formal analysis, Investigation, Methodology, Project administration, Visualization, Writing – original draft, Writing – review & editing; **David M. Williams**: Conceptualization, Funding acquisition, Methodology, Writing – original draft, Writing – review & editing; **Matthew C. Fysh**: Visualization, Writing – original draft, Writing – review & editing.

Disclosure statement

The authors declare that they have no conflict of interest.

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Data availability statement

The data necessary to reproduce the analyses presented here will be uploaded to OSF after publication.

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