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Latent profiles of eating disorder symptoms among transgender, gender diverse, and cisgender people with lived experience of eating disorders: associations with neurodivergent traits and gender dysphoria

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Abstract

Eating disorder (ED) research has historically overlooked trans and gender diverse (TGD) people, despite evidence of elevated ED symptoms in this group. TGD people often report co-occurring autistic and ADHD traits that may intersect with their ED. We examined whether distinct profiles of ED symptomatology differ by gender identity, and the extent to which gender dysphoria and neurodivergent traits are associated with profile assignment. We applied latent profile analysis to ED symptom data from 487 individuals with experience of an ED (303 TGD people; 18–62 years). A four-profile solution provided the best fit: (1) Avoidant eating without ED cognitions, 18.7%; (2) Low ED cognitions and behaviours, 23.8%; (3) Avoidant eating with ED cognitions, 28.1%; and (4) Binge eating with ED cognitions, 29.4%. The Avoidant eating without ED cognitions profile was most relevant to gender dysphoria (OR = 1.50, p = .010) and autistic (OR = 1.57, p = .006) and ADHD traits (OR = 1.58, p = .006). Autistic traits increased the odds of membership in profiles characterised by ED cognitions. These findings demonstrate heterogeneity in ED presentations and highlight the value of a transdiagnostic, data-driven approach. They highlight the need to consider dual diagnoses of ARFID and anorexia nervosa, particularly among TGD and neurodivergent groups.

Introduction

Eating disorders (EDs) are heterogenous conditions, with variation in presentation, treatment response, and diagnostic crossover [1, 2]. Individuals differ in patterns of ED cognitions (e.g., weight, shape, and eating concerns) and behaviours (e.g., restriction, binge eating), which may co-occur in patterns that cut across formal diagnostic boundaries [3, 4]. Data-driven approaches, such as latent profile analysis (LPA), are well-suited to identifying transdiagnostic ED symptom patterns and shared mechanisms across individuals, including groups whose experiences are underrepresented in clinical research and guidance [5].

ED diagnostic frameworks have primarily been based on clinical presentations observed in cisgender populations [6] and may not adequately capture the experiences of transgender and gender diverse (TGD) individuals, whose gender identity does not align with their sex assigned at birth. TGD people experience disproportionately high levels of ED symptoms [7–9], affecting 17.7% of binary trans individuals [i.e., trans women or trans men; 10], with even higher reported prevalence among gender expansive individuals (TGD people who do not identify with a binary or nonbinary identity e.g., bigender, genderqueer) [11]. TGD individuals are overrepresented in diagnoses such as Other Specified Feeding or Eating Disorder [OSFED; an ED diagnosis for individuals who have ED symptoms that cause distress or impairment but do not meet the specific criteria for other EDs; 12, 13, 14], suggesting ED symptom presentations that fall outside traditional diagnostic thresholds.

TGD people report a range of ED behaviours, although research has primarily focused on restrictive eating as seen in anorexia nervosa. Restrictive eating may function as a way of alleviating gender dysphoria, which is the distress associated with gender incongruence, by altering body shape or

suppressing pubertal changes in TGD people [7, 15, 16]. Emerging evidence also suggests TGD people present with elevated levels of binge eating [17] and avoidant/restrictive food intake disorder (ARFID) symptoms [18]. ARFID is characterised by restrictive eating unrelated to weight and shape concerns, such as sensory sensitivities, lack of interest in food, and/or fear of aversive consequences of food [19], but has historically been under researched and often not included in assessment batteries despite being prevalent [20]. Studies using the Eating Disorder Examination Questionnaire [21], a well-established measure of ED cognitions, further suggest that TGD people report higher levels of eating concern, weight concern, and shape concern than presumed cisgender men and women [22, 23]. These findings demonstrate the importance of better characterising transdiagnostic ED profiles in this group by profiling across multiple cognitive and behavioural domains.

TGD individuals also report high rates of co-occurring autism and attention deficit hyperactivity disorder [ADHD; 24, 25], with prevalence estimates 2–6 times higher than in cisgender populations [26]. Autistic people and those with ADHD are at increased risk for a range of EDs, including ARFID, anorexia nervosa, bulimia nervosa, and binge eating disorder [27–29]. Several mechanisms have been proposed, including cognitive differences, such as a preference for predictability and routine; emotional differences, including identifying, processing, and communicating emotions; and sensory differences, such as the intensity of bodily responses to sensory stimuli and interoception [30–32]. However, prior research has rarely examined autism and ADHD together, nor considered the full spectrum of ED behaviours.

Although limited, evidence suggests TGD people who are also autistic may be at greater risk of developing EDs compared to TGD people who are not autistic [33, 34]. However, research has not yet explored whether TGD people with ADHD are at greater risk for developing EDs compared to TGD people who do not have ADHD. One possibility for the heightened risk of EDs in autistic TGD people may be the intersection of autistic traits and gender dysphoria. For example, qualitative evidence with autistic TGD adults suggests sensory differences may exacerbate gender dysphoria by enhancing negative sensory experiences to bodily changes (e.g., menstruation, facial hair growth), known as ‘sensory dysphoria’ [35]. No studies to-date have quantitatively examined the unique and intersecting contributions of autism, ADHD, and gender dysphoria to different patterns of ED symptoms.

Importantly, understanding whether these patterns of ED symptoms, and their relations with autistic and ADHD traits, are specific to TGD populations or reflect broader transdiagnostic ED profiles that exist across gender identities, requires inclusion of cisgender individuals. It is also important to examine differences amongst TGD identities, as previous research has often considered TGD people as one homogenous group despite evidence suggesting they experience unique risk factors for EDs [36]. This approach enables clearer interpretation of how gender identity and gender dysphoria shape risk and presentation of ED symptoms.

The present study uses LPA to identify ED symptom profiles in a mixed sample of TGD and cisgender individuals with lived experience of an ED, inclusive of a range of neurotypes. We aim to examine differences in profile prevalence across gender identities and explore the unique and intersecting

contributions of gender dysphoria, autistic traits, and ADHD traits on these profiles. By considering both gender identity and gender dysphoria, this study aims to clarify the mechanisms sharing ED presentations beyond diagnostic labels, recognising that not all TGD people experience gender dysphoria [37] and that its intensity may vary across contexts.

Method

Participants

Five-hundred and forty-six participants provided consent and complete data for analyses (> 95% item completion). From these, 68 participants were excluded as they did not meet the eligibility criteria for the study (*n* = 38 were not based in the UK, *n* = 18 did not have lived experience of an ED, *n* = 2 were under the age of 18) and one participant was excluded due to malicious responses. This resulted in a final sample of 487 participants. This sample was formed of 303 TGD participants (*M* age = 27.9 years, *SD* = 8.3, ranged from 18–62 years), 173 cisgender participants (*M* age = 31 years, *SD* = 10.2, ranged from 18–62 years), and 11 participants who responded 'prefer not to say' when asked whether their gender aligned with their sex assigned at birth (*M* age = 28.2 years, *SD* = 9.9, ranged from 18–51 years). Previous research has suggested a sample size of around 500 participants is necessary for LPA [38, 39].

TGD participants were recruited in our first wave of recruitment, followed by a second wave inclusive of both TGD and cisgender participants. We used this approach to overrepresent TGD participants and ensure our sample represented the diversity of TGD identities, a common limitation of previous ED research [36]. Inclusion criteria for both groups required participants to have current or past experience of an ED, be 18 years or older, currently based in the UK, and fluent in English. Participants were required to report lived experience of an ED to be eligible, but a formal ED diagnosis was not required. This approach was intended to promote the inclusion of a representative sample with diverse ED presentations, acknowledging existing research that highlights substantial barriers to ED assessment within TGD populations [40].

Participants were recruited through social media (Instagram, Facebook, BlueSky, Reddit), national and local charities, support organisations, social groups, and LGBTQ+ events. Recruitment materials indicated that the study aimed to explore the relationships between EDs, gender identity, and neurodivergence.

The project received ethical approval from the Cardiff University School of Psychology Research Ethics Committee (EC.24.11.12.7066A) and all methods were performed in accordance with appropriate ethical guidelines. Informed consent was obtained online from all participants before completing the questionnaires. Demographic information is presented in Table 1.

Community Involvement

An advisory board formed of TGD and/or neurodivergent people with lived experience of an ED advised on the aims of the study, the question relevance, accessibility of the questionnaire, interpretations of the results, and reporting of the findings, e.g., the profile names and interpretations. The research team was formed of researchers, academics, and clinicians who are trans, nonbinary, and cisgender people, include both neurodivergent and neurotypical identities, with some members also having lived experience of an ED.

Table 1

Sample characteristics (N = 487)

Sex assigned at birth	**Frequency (%)**
Female	401 (82.1)
Male	84 (17.2)
Prefer not to say	3 (0.6)
Born with variation of sex characteristics[a]	
Yes	5 (1.0)
No	426 (87.5)
Unsure	54 (11.1)
Prefer not to say	2 (0.4)
Gender	
Agender	20 (4.1)
Cisgender man	17 (3.5)
Cisgender woman	156 (32.0)
Gender fluid	15 (3.1)
Genderqueer	10 (2.1)
Nonbinary	89 (18.3)
Trans man	86 (17.7)
Trans woman	48 (9.9)
Other TGD identity	35 (7.2)
Prefer not to say	11 (2.3)
Sexuality	
Asexual	44 (9.0)
Bisexual	157 (32.2)
Gay and/or lesbian	76 (15.6)
Heterosexual/Straight	84 (17.2)

Note. TGD = trans and gender diverse, ADHD = Attention Deficit Hyperactivity Disorder, AuADHD = co-occurring Autism and ADHD.

Sex assigned at birth	Frequency (%)
Other sexuality	121 (24.8)
Prefer not to say	5 (1.0)
Ethnic group or background	
Asian/Asian British	7 (1.4)
Black/African/Caribbean/Black British	4 (0.8)
Mixed/Multiple ethnic groups	26 (5.3)
Other ethnic group	7 (1.4)
White	440 (90.3)
Prefer not to say	3 (0.6)
Autism diagnosis	
Yes	207 (42.5)
No	272 (55.9)
Prefer not to say	8 (1.6)
ADHD diagnosis	
Yes	117 (24.0)
No	363 (74.5)
Prefer not to say	7 (1.4)
Self-identified neurodivergence[b]	
Neurodivergent	299 (71.9)
AuADHD	140 (33.7)
Autistic	256 (61.5)
ADHD	140 (33.7)
Note. TGD = trans and gender diverse, ADHD = Attention Deficit Hyperactivity Disorder, AuADHD = co-occurring Autism and ADHD.	

[a]An umbrella term used to describe differences in physical sex development from those generally expected of assigned female and male sex at birth. These differences may be chromosomal, hormonal, anatomical, or gonadal. Sometimes referred to as intersex [41].

[b]Participants were asked to select whether they self-identified with any of the four options provided. They could select more than one response so percentages may exceed 100%.

Procedure

Here we report on quantitative data collected as part of a larger mixed-methods survey to understand the presentation and experiences of EDs in TGD and neurodivergent people. This survey was developed and administered via an online platform [42; version July 2023]. Participants first completed a screening question designed to identify invalid or low-quality responses [43]. In this question, they were shown an image of a snow sculpture and asked to briefly describe what they predicted would happen to it on a warm, sunny day. We screened individual free-text responses to confirm that participants provided a plausible description consistent with snow melting.

Participants provided demographic information and details regarding gender transition, neurotype (e.g., autism, ADHD, neurotypical), ED history, and ED treatment experience, if applicable. They then completed a series of self-report questionnaires examining ED symptoms, autistic and ADHD traits, and gender dysphoria. These self-report questionnaires were presented in a random order to avoid order effects. After completing the questionnaires, participants were offered entry into a prize draw to win one of ten £50 monetary vouchers as compensation for their time. Participants' contact details were collected via a separate link to their data.

Measures

The Nine-Item ARFID Screen [NIAS; 44] is a self-report measure of ARFID symptoms comprised of three subscales: the picky eating subscale measuring selective eating behaviours, the appetite subscale where higher scores indicate less interest in food or eating, and the fear subscale measuring fear of aversive consequences of eating. Participants respond on a 6-point scale from 0 (Strongly disagree) to 5 (Strongly agree). We used the NIAS subscale scores in our analyses (Picky eating $\alpha = .901$; Appetite $\alpha = .890$; Fear $\alpha = .910$).

The Eating Disorder Examination Questionnaire [EDE-Q; 21] assesses ED behaviours and cognitions over the previous 28 days, with responses ranging from 0 (no days) to 6 (every day). Higher scores indicate a greater endorsement of ED symptoms. Three subscales measure ED cognitions: eating concern, shape concern, and weight concern. The restraint subscale is considered a measure of behavioural and cognitive aspects of dieting, specifically focusing on the intentional effort to restrict food intake, avoid certain foods, and follow dietary rules. Only the subscales were used in the current analyses (Eating concern $\alpha = .811$; Shape concern $\alpha = .902$; Weight concern $\alpha = .836$; Restraint $\alpha = .883$).

The Eating Pathology Symptoms Inventory [EPSI; 45] binge eating subscale is a self-report measure assessing binge eating symptoms. The subscale consists of eight items and participants respond on a 5-point Likert scale ("Never" to "Very often"), based on their experiences over the past 4 weeks. Item

scores are summed to create a total subscale score ranging from 0–32, with higher scores indicating greater severity of binge eating symptoms (α = .924).

The Gender Preoccupation and Stability Questionnaire – 2nd Edition [GPSQ-2; 46] is a 14-item self-report measure of gender dysphoria based on thoughts and feelings over the past two weeks. It covers two domains: gender preoccupation and gender identity stability. Participants respond on a 5-point Likert scale from 0 (Never) to 4 (All the time), with higher scores indicating higher levels of gender dysphoria. Only the total score was used in the current study (α = .928).

The Comprehensive Autistic Trait Inventory [CATI; 47] is a 42-item self-report measure designed to assess autistic traits across six domains: socialising, communication, social imagination, cognitive rigidity, attention to detail, and sensory sensitivity. For each item, participants respond on a 5-point Likert scale (“Strongly disagree” to “Strongly agree”). Higher scores indicating higher levels of autistic traits. Only the total score was used in this study (α = .918).

The Adult ADHD Self-report Scale [ASRS; 48] is an 18-item self-report measure of ADHD traits. For each item, the participant is asked to respond on a 5-point Likert scale (“Never” to “Very often”), based on how they have felt/behaved over the past 6 months. Items are scored as 0 or 1. Higher scores indicate higher levels of current ADHD traits (α = .922).

Statistical analysis

We conducted LPA in R version 4.5.2 using mclust [49] and tidyLPA [50]. All other analyses were conducted in SPSS version 29.0. We used the four subscales of the EDE-Q, three subscales of the NIAS, and the binge eating subscale of the EPSI as indicators (measures of ED behaviours and cognitions) for the LPA. Z-standardised scores were used for all subscales. We conducted 1–6 profile solutions based on previous ED research using LPA reporting between 2–5 profile models [51–54]. We selected the best-fit solution using common model-fit indices: Bayesian information criteria (BIC) [49], Akaike information criteria (AIC), the maximisation of entropy [55], size of profiles [56] and Bootstrap Likelihood Ratio Test (BLRT). Resulting profiles from the best-fit solution were compared on indicator variables to evaluate construct validity of the profiles and descriptive statistics were computed for each profile.

For examining demographic characteristics of each subgroup, gender identity was categorised into four groups: cisgender (gender and sex assigned at birth aligned), binary trans (gender was opposite to gender attributed to sex assigned at birth e.g., trans man, trans woman), nonbinary (participant reported nonbinary gender identity), and gender expansive (where identity could not be captured within a binary or nonbinary identity e.g., bigender, genderqueer).

Chi-square and analysis of variance were conducted to explore whether gender identity, gender dysphoria, autistic traits, and ADHD traits varied by assigned profile. A multinominal logistic regression was used to examine the relative contributions of gender dysphoria, autistic traits, ADHD traits, and their interactions, to profile membership. These continuous variables were standardised prior to analysis to

aid interpretation of odds ratios [57]. Interaction terms were computed as the product of standardised variables. To reduce family-wise error, Tukey's HSD adjustment was applied to examine post hoc pairwise comparisons.

Results

Aim 1: Profiles of ED symptoms in a mixed sample of TGD and cisgender adults with experience of an ED.

Model fit indices for latent profile analyses are listed in Table 2. On balance, a four-profile solution fitted the data best. We investigated the fit statistics for solutions with one to six profiles. We considered the two-profile solution because it showed high entropy (0.91). However, this solution was formed of profiles that only differed in the EDE-Q subscale scores and the AIC and BIC continued to decrease with additional profiles, indicating a better model fit to the data. Therefore, this solution only provided partial support for fit criteria and did not offer interpretations of theoretical interest. We then considered the four-profile solution, which also showed high entropy (0.88). The BLRT value continued to be significant as more profiles were added; however, entropy began to decrease for the 5-profile and 6-profile solutions. Finally, the four-profile solution showed qualitatively different profiles of theoretical interest and resonated with the lived experience of our community advisory board.

Table 2
Fit statistics for 1–6 profile solutions.

Number of profiles	LogLik	AIC	BIC	Entropy	BLRT (*p*)
1	-5524.18	11080.36	11147.37	1.000	-
2	-4930.95	9911.89	10016.60	0.909	1186.47 ($p < .01$)
3	-4815.27	9698.54	9840.94	0.848	231.35 ($p < .01$)
4	-4719.82	9525.65	9705.74	0.876	190.89 ($p < .01$)
5	-4573.88	9251.75	9469.54	0.857	291.89 ($p < .01$)
6	-4530.25	9182.50	9437.98	0.850	87.26 ($p < .01$)

Note. LogLik = Log-Likelihood, AIC = Akaike Information Criterion; BIC = Bayesian Information Criterion, BLRT = Bootstrap Likelihood Ratio Test.

Figure 1 presents the standardised means of the indicator variables for the selected four-profile solution. Table 3 details the mean scores of each indicator variable across the four profiles and differences between these profiles. The four profiles were identified as:

Profile 1: Avoidant eating without ED cognitions (n = 91, 18.7%), included individuals with relatively elevated picky eating behaviours, fear of aversive consequences of eating, and less appetite and interest

in food. This was alongside relatively low ED cognitions (restraint, eating, shape, and weight concerns) and binge eating behaviours.

Profile 2: Low ED cognitions and behaviours (n = 116, 23.8%), was marked by relatively low levels of eating, shape, and weight-related concerns, as well as fear. It was accompanied by low levels of restraint, binge eating behaviours, and picky eating behaviours.

Profile 3: Avoidant eating with ED cognitions (n = 137, 28.1%), comprised individuals with similarly high levels of picky eating behaviours, fear, and low appetite and interest in food as the *Avoidant eating without ED cognitions* group, but in this group ED cognitions were also relatively high.

Profile 4: Binge eating with ED cognitions (n = 143, 29.4%) profile, was characterised by relatively high levels of binge eating behaviours and restraint, as well as eating, shape, and weight-related concerns. They had relatively low levels of picky eating and fear and, of note, more interest in food and increased appetite.

Note

EPSI-Binge Eating: Eating Pathology Symptoms Inventory – Binge Eating subscale; ARFID: avoidant/restrictive food intake disorder. Higher scores indicate greater endoresement of eating disorder symptoms. For Appetite, higher scores indicate less interest in food and lower appetite.

Table 3

Mean scores (SD) across the four profiles for each of the indicator variables (N = 487).

Measure Subscale	Profile 1: Avoidant eating without ED cognitions	Profile 2: Low ED cognitions and behaviours	Profile 3: Avoidant eating with ED cognitions	Profile 4: Binge eating with ED cognitions		
	N = 91 (18.7%)	N = 116 (23.8%)	N = 137 (28.1%)	N = 143 (29.4%)	*F*	η^2
EDE-Q Eating concern	1.5 (1.0)	.8 (.7)	3.9 (1.1)	3.5 (1.1)	283.7	.638
EDE-Q Shape concern	3.0 (1.4)	2.4 (1.1)	5.4 (.6)	5.0 (.8)	292.1	.645
EDE-Q Weight concern	2.5 (1.2)	2.1 (1.2)	5.2 (.7)	4.7 (.9)	289.5	.643
EDE-Q Restraint	1.2 (1.3) †	1.1 (1.2) †	4.5 (1.3)	3.3 (1.7)	177.4	.524
EPSI Binge eating	7.5 (6.6) †	8.5 (5.7) †	8.0 (7.8) †	17.4 (8.4)	55.2	.255
NIAS Picky eating	9.4 (4.2) †	4.2 (4.0)	9.8 (4.1) †	5.7 (4.7)	48.8	.232
NIAS Appetite*	11.3 (2.7) †	3.4 (3.0)	10.5 (2.9) †	2.1 (2.2)	380.1	.702
NIAS Fear	6.8 (5.0) †	1.9 (2.8)	6.6 (4.7) †	2.2 (3.0)	56.0	.258

Note. All *p*s < .001. Tukey's HSD adjustment applied. ED: Eating Disorder; EDE-Q: Eating Disorder Examination Questionnaire; EPSI: Eating Pathology Symptoms Inventory; NIAS: Nine-Item Avoidant/Restrictive Food Intake Disorder Scale.

†Indicates groups that do not significantly differ from each other on indicator variables.

*Higher scores indicate less interest in food and lower appetite.

Aim 2: to examine differences in profile membership by gender identity.

Table 4 presents the characteristics of the four profiles, including gender identity, sex, ED diagnosis and treatment status, and neurodivergent diagnoses and self-identities. We found profile assignment was significantly associated with gender identity group: cisgender, binary trans, nonbinary, or gender expansive ($\chi^2(9, N = 476) = 18.43$, $p = .031$, $V = .114$). Post-hoc pairwise comparisons using Bonferroni-adjusted alpha (= .0083) were conducted to compare the association with profile assignment across gender identity groups. This was significant when comparing the gender expansive and cisgender

participants ($\chi^2(3, N = 253) = 14.12$, $p = .003$, $V = .236$). Examination of standardised residuals suggested that gender expansive participants were underrepresented in the *Avoidant eating with ED cognitions* profile (Z = -2.5) but overrepresented in the *Avoidant eating without ED cognitions* profile (Z = 1.7). Conversely, cisgender participants were overrepresented in the *Avoidant eating with ED cognitions* profile (Z = 1.7). All other pairwise comparisons were not statistically significant.

Table 4

Characteristics by profile in a sample (%) of 487 individuals with lived experience of eating disorders (EDs).

	Profile 1: Avoidant eating without ED cognitions	Profile 2: Low ED cognitions and behaviours	Profile 3: Avoidant eating with ED cognitions	Profile 4: Binge eating with ED cognitions
	N = 91 (18.7%)	**N = 116 (23.8%)**	**N = 137 (28.1%)**	**N = 143 (29.4%)**
Gender identity				
Cisgender	22 (24.7)	41 (36.3)	61 (46.2)	49 (34.5)
Binary trans	28 (31.5)	29 (25.7)	33 (25.0)	44 (31.0)
Nonbinary	20 (22.5)	21 (18.6)	27 (20.5)	21 (14.8)
Gender expansive	19 (21.3)	22 (19.5)	11 (8.3)	28 (19.7)
Sex assigned at birth				
Male	20 (22.0)	22 (19.3)	16 (11.7)	26 (18.3)
Female	71 (78.0)	92 (79.3)	121 (88.3)	116 (81.7)
ED diagnosis	49 (53.8)	62 (53.4)	98 (71.5)	71 (49.7)
Current ED treatment	7 (7.7)	9 (7.8)	50 (36.5)	19 (13.3)
Autism diagnosis[a]	47 (51.6)	35 (30.2)	67 (48.9)	58 (40.6)
ADHD diagnosis[a]	32 (35.2)	28 (24.1)	28 (20.4)	29 (20.3)
AuADHD diagnosis[a]	20 (22.0)	9 (7.8)	13 (9.5)	17 (11.9)
Neurodivergent self-identity[b]				
Neurodivergent	61 (72.6)	67 (71.3)	84 (73.7)	87 (70.2)
AuADHD	35 (41.7)	25 (26.6)	36 (31.6)	44 (35.5)
Autistic	53 (63.1)	52 (55.3)	79 (69.3)	72 (58.1)
ADHD	33 (39.3)	36 (38.3)	31 (27.2)	40 (32.3)

Note. ADHD = Attention Deficit Hyperactivity Disorder, AuADHD = co-occurring Autism and ADHD.

[a]Participants were asked whether they had received a clinical diagnosis of autism and ADHD, separately. They could answer 'yes', 'no' or 'prefer not to say'. Participants who only answered 'yes' to having an

autism diagnosis are counted in the Autism diagnosis category, while those who only reported an ADHD diagnosis are counted in the ADHD diagnosis category. Those participants who responded 'yes' to receiving both an autism and ADHD diagnosis were counted in the AuADHD diagnosis category.

[b]In addition to the question on diagnosis, participants were asked to select whether they self-identified with any of the four identities provided. This question was asked to everyone, irrespective of whether they reported a neurodivergent diagnosis or not. They could select more than one response so percentages may exceed 100%. Some participants reported an AuADHD identity but did not also report an Autistic and ADHD identity.

Aim 3: to explore the unique and intersecting contributions of gender dysphoria, autistic traits, and ADHD traits on profile membership.

We found levels of gender dysphoria, autistic traits, and ADHD traits differed significantly across profiles (Table 5). The *Low ED cognitions and behaviours* profile displayed the lowest levels of gender dysphoria, autistic traits, and ADHD traits. Gender dysphoria and ADHD traits were highest in the *Avoidant eating without ED cognitions* profile, whilst autistic traits were highest in the *Avoidant eating with ED cognitions* profile.

Table 5

Mean scores (SD) across the four profiles for gender dysphoria (GPSQ-2), autistic traits (CATI), and ADHD traits (ASRS).

	Profile 1: Avoidant eating without ED cognitions	**Profile 2: Low ED cognitions and behaviours**	**Profile 3: Avoidant eating with ED cognitions**	**Profile 4: Binge eating with ED cognitions**			
	N = 91 (18.7%)	**N = 116 (23.8%)**	**N = 137 (28.1%)**	**N = 143 (29.4%)**	***F***	***P***	**η^2**
Gender dysphoria	21.0 (12.1)	15.2 (10.8)	16.2 (13.8)	17.9 (13.3)	4.18	.006	.025
Autistic traits	163.1 (30.7)	144.1 (29.1)	167.6 (27.7)	158.7 (29.3)	14.71	< .001	.084
ADHD traits	12.9 (4.4)	10.1 (4.8)	12.1 (4.8)	11.5 (4.5)	7.14	< .001	.042

Note. ED: eating disorder; ADHD: Attention Deficit Hyperactivity Disorder; GPSQ-2: Gender Preoccupation and Stability Questionnaire – 2nd Edition; CATI: The Comprehensive Autistic Trait Inventory; ASRS: Adult ADHD Self-report Scale.

We examined the relative contributions of neurodivergent traits and gender dysphoria and their interactions to profile membership using a multinominal logistic regression. With the *Low ED cognitions and behaviours* profile as the reference, the overall model was significant, $\chi^2(15) = 72.52$, $p < .001$ (Nagelkerke $R^2 = .148$). Autistic traits made the largest unique contribution to distinguishing between the

four profiles ($\chi^2(3) = 36.06$, $p < .001$), followed by gender dysphoria ($\chi^2(3) = 15.00$, $p = .002$) and ADHD traits ($\chi^2(3) = 8.00$, $p = .046$). The interaction between gender dysphoria and autistic traits was significant overall ($\chi^2(3) = 8.93$, $p = .030$), whereas the gender dysphoria x ADHD traits interaction was not ($\chi^2(3) = 2.31$, $p = .511$).

Parameter estimates showed that higher autistic traits were associated with greater odds of membership in the *Avoidant eating without ED cognitions* (OR = 1.574, 95% CI [1.140, 2.173], $p = .006$), *Avoidant eating with ED cognitions* (OR = 2.580, 95% CI [1.847, 3.605], $p < .001$), and *Binge eating with ED cognitions* profiles (OR = 1.518, 95% CI [1.154, 1.997], $p = .003$), relative to the reference group. As continuous measures have been standardised, odds ratios represent change in odds associated with a one standard deviation increase in measure scores. Higher ADHD traits and higher gender dysphoria uniquely increased odds of membership in the *Avoidant eating without ED cognitions* profile (ADHD OR = 1.575, 95% CI [1.140, 2.176], $p = .006$; Gender dysphoria OR = 1.500, 95% CI [1.101, 2.044], $p = .010$), but not the *Avoidant eating with ED cognitions* or *Binge eating with ED cognitions* profiles (*ps* ≥ .178). The gender dysphoria x autistic traits interaction indicated that the positive association between autistic traits and the odds of the *Avoidant eating with ED cognitions* profile became stronger as gender dysphoria increased (OR = 1.493, 95% CI [1.096, 2.035], $p = .011$). There was no evidence of an interaction between gender dysphoria and ADHD traits (*ps* ≥ .473).

Discussion

The current study investigated latent profiles of ED symptoms in a mixed sample of TGD and cisgender people with lived experience of EDs. Using a data-driven approach, we stratified the heterogeneity of ED symptoms, creating transdiagnostic groups that cut across traditional diagnostic boundaries and differ meaningfully in gender identity, gender dysphoria, and autistic and ADHD traits. These findings provide novel insights into the complex intersectionality of EDs, gender identity, and neurodivergence, and support the use of a transdiagnostic dimensional approach when examining these intersections.

The profiles showed alignment with ED diagnostic categories [58]. The *Binge eating with ED cognitions* profile closely resembled features of binge eating disorder and bulimia nervosa, while the *Avoidant eating with ED cognitions* profile aligned with diagnoses like anorexia nervosa. The *Avoidant eating without ED cognitions* profile overlapped conceptually with ARFID. Lastly, the presence of a *Low ED cognitions and behaviours* profile is consistent with previous research looking at ED profiles [51] and may reflect individuals with remitted, fluctuating, or unendorsed symptoms at the time of assessment.

We found individuals with higher levels of gender dysphoria, autistic traits, and ADHD traits were more likely to report *Avoidant eating without ED cognitions*. This is in line with previous research from TGD and cisgender samples that has demonstrated associations between ARFID symptoms and both autism [29, 59, 60] and ADHD [59, 60]. Our findings provide useful insights into the types of mechanisms that might underpin avoidant eating in the absence of ED cognitions, such as sensory differences [60], executive functioning differences affecting food planning and preparation, or interoception differences, such as

hyposensitivity to hunger cues [32, 61]. For gender dysphoria, this pattern aligns with avoidant eating serving as a coping mechanism for gender-related distress in the absence of traditional weight and shape-related cognitions. Recent research by Cobbaert, Schweizer [59] proposed avoidant eating in TGD individuals may reflect broader stress responsivity to external stressors, such as minority stress and chronic threat exposure [62, 63]. This mechanism may be particularly prevalent for gender expansive individuals, who were overrepresented in this profile, and aligns with evidence that TGD people often present with atypical ED presentations, including restrictive behaviours not driven by weight and shape concerns [13, 60].

In addition to associations with the *Avoidant eating without ED cognitions* profile, higher autistic traits also increased the odds of membership in the other two ED-related profiles relative to the *Low ED cognitions and behaviours* profile. This is in line with previous research that finds autism to be associated with restrictive EDs and binge eating [31, 64]. These findings support qualitative research proposing autism-specific mechanisms for ED development, including cognitive, emotional, and sensory differences [30, 65]. Notably, ADHD traits were not associated with the *Binge eating with ED* cognitions profile, challenging assumptions that ADHD primarily confers risk for binge-type ED presentations [66].

We found the association between autistic traits and the *Avoidant eating with ED cognitions* was stronger at higher levels of gender dysphoria. This interaction is the first quantitative evidence of an intersectional effect and is consistent with qualitative accounts of sensory dysphoria among autistic TGD adults, whereby sensory differences interact with gender dysphoria and increase sensitivity to negative experiences of one's body [35]. Together, these findings suggest that gender dysphoria may intensify the expression of ED cognitions among autistic individuals, potentially as a means of managing or gaining control over body-related distress. Longitudinal and qualitative research is needed to clarify the temporal and experiential dynamics of these intersecting processes.

Clinical Implications

Our findings have important clinical implications for ED assessment and treatment in TGD neurodivergent people. The strong association between formal ED diagnosis and the *Avoidant eating with ED cognitions* profile likely reflects the diagnostic visibility of weight and shape concerns in current frameworks compared to avoidant eating only. The increased likelihood of the *Avoidant eating without ED cognitions* profile among individuals with elevated gender dysphoria and neurodivergent traits highlights the need for assessment models that extend beyond weight-centric definitions of EDs. Screening for avoidant eating and ARFID-related symptoms in individuals with gender dysphoria and/or neurodivergent traits may help identify those who require support but remain underserved by existing services. Clinicians should also be aware of access barriers to neurodevelopmental assessments and diagnostic overshadowing in marginalised groups, especially TGD participants [67, 68]. Furthermore, the presence of an *Avoidant eating with ED cognitions* profile indicates there is a group who report high levels of ARFID symptoms in addition to traditional ED cognitions, which currently do not fit DSM-5 ED diagnostic criteria [58]. This profile was more likely among individuals with higher levels of autistic traits, particularly those

who also had higher levels of gender dysphoria, indicating neurodivergent people and TGD people may benefit from dual diagnosis of ARFID with anorexia nervosa, and DSM-5 exclusion criteria may need to change.

Strengths and Limitations

The inclusion of a diverse sample of gender identities in our study is a notable strength not addressed in most ED research. LPA allows us to identify profiles based on symptom patterns rather than diagnostic labels, offering a person-centred approach that is critical for advancing inclusive and tailored ED interventions and care. However, the cross-sectional design prevents causal inference, which would be important for future research to address to understand directionality and stability of profile assignment across time. The sample was predominantly White, English speaking, and assigned female at birth, which may limit generalisability, particularly given evidence of elevated ED risk among trans people of colour [69]. Lastly, these quantitative insights should be integrated with qualitative research to understand the lived experiences underpinning these symptom profiles.

Conclusion

ED symptoms in our mixed TGD and cisgender sample arose through multiple pathways shaped by neurodivergence and gender dysphoria that may not be fully captured in current diagnostic frameworks. The ARFID-type profile in our study was most relevant to gender dysphoria, autistic traits, and ADHD traits, highlighting the importance of ARFID screening and formulation when supporting TGD and neurodivergent people with EDs. Overall, these findings reinforce the need for gender-affirming, neurodiversity-informed approaches that address the shared and profile-specific mechanisms underlying EDs in TGD and neurodivergent people.

Declarations

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Competing interests

The author(s) declare no conflicts of interest.

Author Contribution

KT: Conceptualisation; Data Curation; Formal Analysis; Funding Acquisition; Investigation; Methodology; Project Administration; Software; Visualisation; Writing – Original Draft Preparation. GW: Conceptualisation; Investigation; Methodology; Project Administration; Writing – Review & Editing. MK:

Conceptualisation; Investigation; Methodology; Project Administration; Writing – Review & Editing. HB: Conceptualisation; Methodology; Writing – Review & Editing. RB: Conceptualisation; Methodology; Writing – Review & Editing. JM: Conceptualisation; Methodology; Writing – Review & Editing. LEADING Lived Experience Advisory Group: Conceptualisation; Methodology; Writing – Review & Editing. MW: Conceptualisation; Methodology; Writing – Review & Editing. SC: Methodology; Software; Writing – Review & Editing. JH: Conceptualisation; Methodology; Writing – Review & Editing. KC: Conceptualisation; Methodology; Writing – Review & Editing; Supervision (equal). CJ: Conceptualisation; Methodology; Writing – Review & Editing; Supervision (equal).

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Data Availability

Data are available at https://osf.io/sazrc/

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Figures

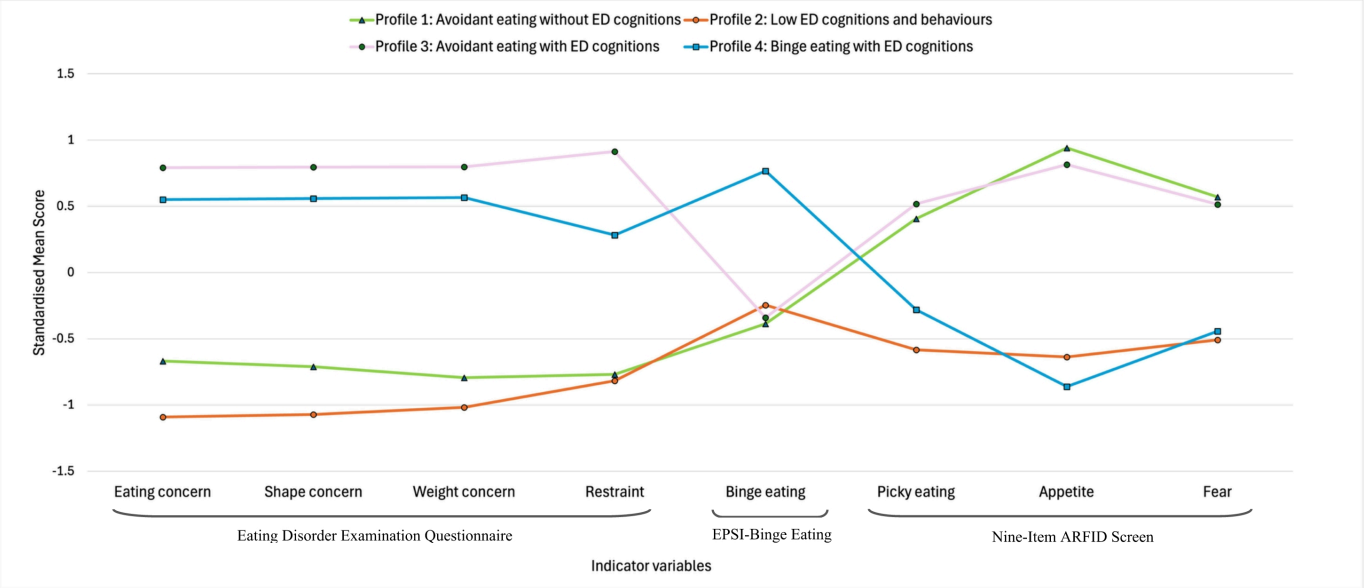


Figure 1

Standardised mean score of eating disorder (ED) symptoms for the four latent profiles (N = 487).

Note. EPSI-Binge Eating: Eating Pathology Symptoms Inventory – Binge Eating subscale; ARFID: avoidant/restrictive food intake disorder. Higher scores indicate greater endoresement of eating disorder symptoms. For Appetite, higher scores indicate less interest in food and lower appetite.