



Published in final edited form as:

*Eur Child Adolesc Psychiatry*. 2021 December ; 30(12): 1929–1938. doi:10.1007/s00787-020-01655-9.

## Prospective Associations between Childhood Social Communication Processes and Adolescent Eating Disorder Symptoms in an Epidemiological Sample

Katherine Schaumberg<sup>1</sup>, Stephanie C. Zerwas<sup>2</sup>, Cynthia M. Bulik<sup>2,3,4</sup>, Chiara Fiorentini<sup>5</sup>, Nadia Micali<sup>5,6,7</sup>

<sup>1</sup>Department of Psychiatry, University of Wisconsin – Madison, United States

<sup>2</sup>Department of Psychiatry, University of North Carolina at Chapel Hill, United States

<sup>3</sup>Department of Nutrition, University of North Carolina at Chapel Hill, United States

<sup>4</sup>Department of Medical Epidemiology and Biostatistics, Karolinska Institutet, Stockholm, Sweden

<sup>5</sup>Department of Psychiatry, Faculty of Medicine, University of Geneva, Switzerland

<sup>6</sup>Department of Psychiatry, Icahn School of Medicine at Mount Sinai, United States

<sup>7</sup>Institute of Child Health, University College London, United Kingdom

### Abstract

**Objective:** Deficits in social cognition and communication, the processes associated with human social behavior and interaction, have been described in individuals with eating disorder psychopathology. The current study examined whether social communication characteristics present in middle childhood (ages 8-14) were associated with eating disorder behaviors, cognitions, and diagnoses across adolescence (ages 14-18) in a large, population-based sample.

**Method:** Participants (N = 4864) were children enrolled in the Avon Longitudinal Study of Parents and Children (ALSPAC), a population-based, prospective study of women and their children. Regression methods tested prospective associations between social functioning using a facial emotion recognition task and parentally reported social communication symptoms (or difficulties), measured by the Social Communication Disorder Checklist (SCDC), with eating disorder symptoms and diagnoses.

**Results:** Misattribution of faces as sad or angry at age 8.5 was associated with purging and anorexia nervosa diagnosis at age 14, respectively, among girls. Further, autistic-like social communication difficulties during middle childhood were associated with bulimia nervosa symptoms during adolescence among both girls and boys.

---

**Correspondence to:** Katherine Schaumberg, University of Wisconsin – Madison, Department of Psychiatry, 6001 Research Park Blvd, Madison, WI, USA, 53719.

Conflicts of Interest:

Katherine Schaumberg, Nadia Micali, Stephanie Zerwas, and Chiara Fiorentini have no conflicts to report  
Cynthia Bulik: Shire (grant recipient, Scientific Advisory Board member); Idorsia (consultant); Pearson (author, royalty recipient).

**Conclusions:** Results did not support global associations between measured social communication deficits and eating disorder risk in this sample, but specific difficulties with facial emotion recognition and social communication may enhance risk for disordered eating behaviors.

### Keywords

ALSPAC; social communication; eating disorder

---

## Introduction

Social cognition and communication processes refer to the mental processes underlying human social behavior and interaction, including the ability to create mental representations of others' social experiences [1]. Many psychiatric conditions, and most notably autism spectrum disorder (ASD), are characterized by impairments in these processes [2]. Recently, there has been increasing interest the role of social cognition and social communication in the development of eating disorders [3], along with the overlap between eating disorder risk and ASD traits [4-6].

Some experimental studies have investigated social processes among individuals with eating disorders, and findings suggest that those with active eating disorders demonstrate deficits in social processing, including difficulties attending to positive facial expressions [7, 8] and attentional bias towards angry or rejecting faces [7, 8], which may persist after eating disorder recovery [9]. In response to socially frustrating situations, one study has also found that individuals with anorexia nervosa (AN) demonstrate greater aggression and less focus on social solutions [10]. Further, patients with eating disorders also self-report high levels of alexithymia -- difficulty identifying and describing emotions [11]. While the bulk of research on social processes among eating disorders has focused on AN, deficits in social processing appear to be present across the range of eating pathology, including among those with binge eating disorder [12] and bulimia nervosa (BN) [13, 14].

While clinical studies of individuals with eating disorders often demonstrate differences in social cognition and communication processes when comparing those with eating disorders to healthy individuals [15-17], the developmental course of these deficits remains unclear. As eating disorders often onset during adolescence [18, 19], and can disrupt social development and peer relationships as well as neurocognitive functioning, it is possible that social processing deficits among those with eating disorders are primarily a consequence of, rather than a precursor to, the disorder. Experiences of trauma and symptoms of anxiety and depression, which commonly co-occur with eating disorders, are also associated with social processing difficulties [20-22] and may account for social processing difficulties present in eating disorders.

To date, a few studies have examined social processes in eating disorders in population-based samples. Using a twin design, one investigation found greater difficulties in emotion recognition and regulation along with attentional bias to social threat among twins affected by an eating disorder compared to control twins [23]. Further, attentional biases to social threat were also present in unaffected co-twins of those with BN [23]. Another epidemiological investigation found greater presence of social cognitive difficulties among

children whose mothers had eating disorder psychopathology [24] suggesting potential genetic associations between social cognition and eating disorders. Further, a nationwide investigation of individuals in Denmark found increased autism spectrum disorder risk in families of AN probands, but indicated that this may be due to non-specific psychiatric risk aggregating in these families [25]. In sum, there is tentative support for associations between eating disorders and social processing difficulties in population studies, though the specificity and nature of this shared risk remains unclear.

Additional prospective, epidemiological research is needed to delineate developmental links between social cognition and eating disorder risk. To date, limited research has prospectively examined either social communication deficits or performance on relevant tasks in childhood as a predictor of eating disorders. One study on the ALSPAC sample indicated that social communication deficits across childhood associated with a composite measure of disordered eating behaviors at age 14. In this investigation, disordered eating behaviors were collapsed together and examined only at a single time point and eating disorder diagnoses were not evaluated [26], limiting conclusions related to more specific behavior- or disorder-level processes. Another prospective cohort study, focusing on middle childhood, identified that autistic traits at six years old were associated with picky eating and emotional eating at age 10, specifically in girls [27]. Further, a prospective twin study did not find elevated rates of parent-reported ASD or associated traits at age 9 among children who went on to develop AN; however, those with acute AN did display some ASD traits at age 18 [28], concluding that it is still in question whether social communication difficulties among those with AN are an epiphenomenon of AN or whether the eating disorder brings to light underlying ASD traits that were previously not captured.

The current study adds to this literature by examining whether task-based (facial emotion recognition) and parent-report measures of social communication in childhood and early adolescence associate with eating disorder symptoms and diagnoses across adolescence (at ages 14, 16, and 18 years) in a large, prospective epidemiological sample of children. As there are few existing studies that have examined these questions, the present study tests associations in a broad, exploratory manner. While we do not have specific hypotheses about every association to be tested, we hypothesize that a tendency to interpret faces as more negatively-valenced in middle childhood will associate with likelihood of AN and BN diagnoses and associated behaviors (compulsive exercise, fasting, binge eating, purging) during adolescence. Further, we expect that parent-reported social communication difficulties in middle childhood will associate specifically with AN diagnosis during adolescence.

## Method

### Participants

Participants were children enrolled in the Avon Longitudinal Study of Parents and Children (ALSPAC) study, an epidemiological, longitudinal study of mothers and their children [29-31]. Women expecting to deliver a child between 1<sup>st</sup> April 1991 and 31<sup>st</sup> December 1992 in Avon, UK were invited to take part in the study, and interested expectant mothers provided informed and written consent. Children ( $n = 14,062$ ) from 14,451 pregnancies

were enrolled. At seven years, 913 additional children were enrolled in the cohort [29]. The study website contains details of all the data that are available through a fully searchable data dictionary and variable search tool: <http://www.bris.ac.uk/alspac/researchers/our-data/>. Ethical approval for this study was obtained from the ALSPAC Ethics and Law Committee and the Local Research Ethics Committees and has therefore been performed in accordance with the ethical standards laid down in the 1964 Declaration of Helsinki and its later amendments. Informed consent for the use of data collected via questionnaires and clinics was obtained from participants following the recommendations of the ALSPAC Ethics and Law Committee at the time. Detailed phenotypic, exposure, and socio-demographic data were collected via self- and maternal-report, face-to-face assessments [29]. With regards to data relevant to the current study, 7,488 children attended face-to-face data collection waves at age 8 and 7,563 at age 10. A total of 5,938 children completed eating disorder questionnaire measures at age 14; 5,131 at age 16; and 3,372 at age 18.

## Measures

Assessments by age are presented in Supplementary Table 1.

**ED Behaviors**—All ED behaviors were self-reported and were measured at 14, 16, and 18 years of age. Questions about ED behaviors inquired about the previous year and were adapted from the Youth Risk Behavior Surveillance System (YRBSS) questionnaire [32] and validated [33]. We defined eating disorder behaviors as *present* or *absent* at each assessment period (for details, see [34]. In addition to a rating of presence or absence of ED behaviors at each assessment point, a lifetime variable was calculated for each behavior, such that individuals who endorsed engaging in that behavior at *any* age (14, 16, or 18) were coded as Yes (present = 1) or No (absent = 0).

**ED and Related Cognitions**—All of the following measures were self-reported at 14 years of age.

**Body dissatisfaction** was assessed using the Body Dissatisfaction Scale of the Satisfaction and Dissatisfaction with Body Parts Scale [35] This scale asks individuals to rate their satisfaction with nine body parts on a 5-point scale, from ‘extremely satisfied’ to ‘extremely dissatisfied,’ (Cronbach’s  $\alpha = 0.84$  in the current study). A continuous score was derived for this measure, with higher values indicating higher dissatisfaction.

**Fear of weight gain** was assessed through one item asking the degree to which participants have worried about gaining a little weight (0 = not at all, 1 = a little, 2 = a lot, 3 = all the time).

**Pressure to lose weight** was assessed through six items asking the degree to which participants feel pressure to lose weight and was adapted from the Perceived Sociocultural Pressure Scale [36].

**Emotional eating, external eating, and restrained eating** were assessed through using 25 items of the Dutch Eating Behaviors-Questionnaire [DEBQ; [37]], which were rated on a five-point Likert scale. The DEBQ raw scores are totaled into three subscale scores:

Emotional Eating (eating in reaction to emotions), External Eating (eating in reaction to external cues), and Restrained Eating (cognitively attempting to limit one's caloric intake). All emotional and external eating items were used in this study; only two items of the restraint subscale were used as a measure of restraint. Higher scores on each subscale indicate greater symptomatology.

**Thin ideal internalization** was assessed using the Ideal-Body Stereotype Scale-Revised [IBSS-R;[36]]. Questions were gender-specific; girls were asked five questions (Cronbach  $\alpha=0.56$ ) and boys six questions (Cronbach  $\alpha=0.71$  [38]).

**ED Diagnoses**—Eating disorder diagnoses (AN, BN, BED, and purging disorder [PD]) were derived using questionnaire data from the YRBSS from adolescents using DSM-5 diagnostic criteria [2, 18]. Body mass index (BMI) was an objective measure collected at face-to-face assessment (median ages 13.8, 15.5, and 17.8 years) and was included as a diagnostic criterion for AN. Underweight was determined using age, gender, and BMI-specific cutoffs based on UK reference data [39] corresponding to World Health Organization (WHO) grade 1 thinness, defined as curve passing through BMI of 18.5 (BMI Z-score  $\sim -1.00$ ) at age 18 years. Given that this was an epidemiological sample, we included both threshold and subthreshold BN and BED cases (e.g. binge eating between 1x/wk and 1x/month) in order to capture a greater range of eating pathology.

Parental report of AN symptoms was also used at ages 14 and 16 when formulating AN diagnoses as prior research has shown that parental report often aids in the diagnosis of AN in adolescents due to under-reporting of AN symptoms [40].

### **Social Cognition and Communication Processes**

**Social Communication Disorders Checklist (SCDC; [41]):** The SCDC is a 12-item questionnaire that is designed to be completed by parents and that measures social reciprocity and other verbal/nonverbal social communication difficulties, particularly those that are typical of autism spectrum disorders. SCDC items are gender neutral, which helps avoid gender bias and increases sensitivity of the measure. A higher SCDC score is indicative of more deficits in social communication. Studies have found the measure to have good internal consistency (0.93), high test-retest reliability (0.81), and high heritability in both genders (0.74) [41]. In addition, the SCDC has been found to be predictive of autism with a sensitivity of 0.88 and a specificity of 0.91, when using a score of 9 out of 24 [42]. In accordance with prior work in the ALSPAC sample, we dichotomized SCDC composite score at 9 to define social communication difficulties consistent with probable autism diagnosis [43].

**Facial Emotion Recognition: Diagnostic Analysis of Non-Verbal Accuracy (DANVA):** Facial emotion recognition was assessed using the faces subtest of the DANVA at age 8.5 [44]. This computerized task measures a child's ability to recognize emotion from facial cues. Participants were shown photographs of children expressing happiness, sadness, anger, or fear. Binary scores, indicating whether children made more (above cut-off) or less (below cut-off) errors/misattributions, have been developed based on the distribution of results in the cohort. The cut-offs were determined by ALSPAC in collaboration with the

creator of the task, and full details have previously been published [43]. For the current analyses, predictor variables included whether individuals were above or below cutoffs for errors within each of the emotion categories (e.g. number of faces that were angry but not correctly identified as such), as well as misattributions as those emotions (e.g. number of faces during the task which were incorrectly identified as angry when in fact they showed a different emotion).

### Confounders

Maternal education and BMI at age 10 were included as covariates, as these variables may associate with both social cognition and ED symptoms and cognitions. BMI was calculated from objectively-measured height and weight at 10 years of age, a time point prior to age of increased risk for eating disorder onset, and evaluated at an in-person assessment. Age and sex-adjusted BMI Z-scores were used in analyses. Maternal education was used as a proxy for socioeconomic status. Maternal educational level was obtained by questionnaire at enrollment, dichotomized into: (i) ordinary-level qualifications generally obtained at age 16 years or higher; (ii) certificate of secondary school education (lowest level qualifications generally obtained at age 16 years), vocational qualification or no qualifications.

### Data Analytic Plan

Participants were included if they completed at least one measure of social cognition along with a measure of ED behaviors at age 16 (N = 4864). Thirty percent of the sample had complete data, with an additional 18% of the sample missing only eating disorder measures at age 18. We used a maximum likelihood estimation procedure for analyses, a robust procedure for handling missing data [45]

Exclusion criteria were child being deceased, having no known address, or refusing participation in the study. Similar to other studies in the cohort, for multiple births, the older twin was included and younger twin excluded. As social communication difficulties and eating disorder risk are both known to vary by sex in the ALSPAC cohort [18, 43], all analyses were stratified by gender. Analyses included logistic regression to evaluate associations between neuropsychological and ED behavior variables, followed by ordinary least squares regression examining the relationship between neuropsychological predictors and ED risk at age 14. As fear of weight gain was measured with a limited number of ordinal response points, we used ordinal logistic regression to estimate effects for this variable. To account for multiple comparisons, a Benjamini-Hochberg correction was applied within each set of analyses (e.g. for each social cognition variable predicting the larger set of ED variables). Given the exploratory nature of the study, we proceeded with a false discovery rate set to 0.20. As there was a fair degree of stability in SCDC scores across ages (Pearson  $r^2$  range = 0.52-0.64) we followed-up on significant associations between SCDC predictors and ED variables by introducing SCDC scores at earlier ages as a covariate, in order to clarify the age-related specificity of these findings.



## Results

The percentage of boys and girls with DANVA errors and misattributions and SCDC scores above cutoff is presented in Tables 1a-1b. The percentage of girls and boys reporting different eating disorder behaviors and diagnoses at each age are presented in Tables 1c-1d. Results on the association between autism-like social communication deficits and eating disorder symptoms and diagnoses is presented in Table 2, and associations between DANVA errors and misattributions and eating disorder symptoms and diagnoses are presented in Supplemental Tables 2a-2b.

### SCDC.

Several associations were present between autism-like social communication difficulties on the parent-reported SCDC and eating pathology during adolescence. Social communication difficulties at age eight were associated with cognitive eating disorder symptoms at age 14 for girls—namely body dissatisfaction and dietary restraint. At age 10, autism-like social communication difficulties were associated with higher levels of lifetime fasting for girls ( $p = 0.004$ ), as well as greater desire to lose weight at age 14 ( $p = 0.01$ ). After accounting for age 8 difficulties, both effects (lifetime fasting,  $OR = 1.75$  (95% CI: 0.99, 3.09),  $p = 0.05$ ; age 14 desire to lose weight,  $b = 0.23$  (95% CI:  $-0.02, 0.48$ ),  $p = 0.13$ ) were consistent but attenuated.

When examined at age 14, these social communication difficulties were associated with several measures of concurrent and later adolescent eating pathology among girls, including concurrent (age 14) body dissatisfaction ( $p < 0.001$ ) and purging ( $p = 0.001$ ), greater likelihood of binge eating at age 16 ( $p < 0.001$ ) as well as lifetime binge eating by age 18 ( $p = 0.002$ ), and greater likelihood of a BN diagnosis at age 16 ( $p < 0.001$ ). After accounting for earlier social communication difficulties (ages 8 and 10), the associations between age 14 SCDC and these outcomes were consistent, with slightly diminished effect sizes. (body dissatisfaction  $b = 0.33$  (95% CI: 0.09, 0.57),  $p = 0.008$ ; age 16 binge eating  $OR = 1.95$  (95% CI: 1.06, 3.57),  $p = 0.03$ ; lifetime binge eating  $OR = 2.23$  (95% CI: 1.24, 4.00),  $p = 0.007$ ; age 16 BN diagnosis  $OR = 2.31$  (95% CI: 1.08, 4.94),  $p = 0.03$ ).

Given the low frequency of eating pathology among boys, several associations between social communication difficulties and eating disorder behaviors and diagnoses were not adequately powered for analyses. As such, we only evaluated outcomes for models that converged (see Table 2). Among boys, autism-like social communication difficulties at age 8 were not associated with adolescent eating pathology, though these difficulties at age 10 were associated with a greater likelihood of binge eating ( $p = 0.004$ ), purging ( $p < 0.001$ ), and a BN diagnosis ( $p = 0.002$ ) at age 16. After controlling for age 8 SCDC, the associations between age 10 SCDC and age 16 binge eating ( $OR = 3.71$  [95% CI: 1.50, 9.17],  $p = 0.004$ ), purging ( $OR = 4.72$  [95% CI: 1.16, 19.10],  $p = 0.029$ ), and BN diagnosis ( $OR = 6.55$  [95% CI: 2.02, 21.25],  $p = 0.002$ ) remained consistent. There were no associations for later (age 14) social communication difficulties and adolescent eating pathology among boys.

### DANVA Errors and Misattributions.

Overall, there were no significant associations between errors on the DANVA task at age 8 and eating disorder behaviors, cognitions, or diagnoses during adolescence. For DANVA misattributions, only two associations were significant after correcting for multiple comparisons. First, more misattributions of sad faces (identifying faces as sad incorrectly) was associated with a greater likelihood of purging for girls at age 14 ( $p = 0.005$ ). In addition, more misattributions of angry faces (identifying faces as angry incorrectly) was associated with a greater likelihood of an AN diagnosis for girls, also at age 14 ( $p = 0.001$ ). There were no associations between DANVA misattributions and adolescent eating disorder variables among boys.

### Discussion

The current study investigated relationships between task-based facial emotion recognition and parent-reported social communication processes in middle childhood (ages 8-14) and adolescent eating pathology (ages 14-18). Although clinical studies indicate consistent differences between those with and without eating pathology on measures of social processes [3], and one study in the ALSPAC cohort indicated higher levels of parent-reported social communication deficits across childhood and adolescence among children reporting weekly or monthly disordered eating behaviors at age 14 [26], results from the current investigation were not robust to suggest that deficits in social processing pre-date eating pathology based on a combination of task-based and parent-reported data in this large, epidemiological sample.

Some findings corroborated existing literature linking social cognitive biases and eating disorder risk. In particular, a tendency to misattribute non-angry faces as angry at age 8 was associated with higher odds of girls having a diagnosis of AN at age 14. This specific finding is in line with research suggesting an attentional bias towards angry faces among those with AN [10]. Attentional bias towards angry faces aligns with heightened levels of anxiety and perceived threat characteristic of AN [46], and may manifest in AN via increased rigidity and intolerance of uncertainty [47]. The finding that misattribution of sad faces was associated with purging at age 14 supports theories that high negative and low positive emotions may lead to uptake of disordered eating behaviors, and, more generally, supports an emotion regulation account of purging behavior [48]. Together, both of these findings are in line with existing studies suggesting that interpretation of interpersonal situations and others' emotions as more negative may predict eating pathology [9, 13, 49].

In addition, this study provided a next step in clarifying potential links between social communication difficulties and eating disorders. Our results did suggest some associations between autistic-like social communication difficulties and eating disorder risk. For boys, a specific link emerged between age 10 social communication difficulties and age 16 bulimic pathology. For girls, the links between parent-reported social communication difficulties and adolescent eating disorders (specifically BN) became strongest when the assessment point for social communication was closer to adolescence (age 14), suggesting a potential for reverse causation, in which eating disorder symptoms may precipitate social withdrawal and interrupt healthy social development. As binge eating and purging can be compulsive



behaviors that can serve emotion regulation functions, it is possible that those with autism-like traits are more likely to find these behaviors soothing or reinforcing [50]. Further, eating disorder psychopathology may exacerbate social communication deficits, strengthening this relationship over time. An additional interpretation as to why parent-reported social communication deficits in childhood and early adolescence may associate with binge eating and purging includes that attention-deficit hyperactivity disorder (ADHD) associates with both autism spectrum disorder [51] and BN [52, 53] and therefore may link social communication difficulties and these specific pathological eating behaviors.

The association between social communication difficulties and bulimic-spectrum pathology (binge eating, purging, BN diagnoses) is particularly intriguing as the majority of literature to-date has focused on links between social communication and AN, rather than BN. As individuals with AN may be overrepresented in more acute care settings, potential links between BN symptoms and poor social communication, both as a precipitate and consequence of the eating disorder, are less understood. The current study points to the need for additional study of the etiology of social processes in the context of risk for bulimic-spectrum eating disorders (BN, binge eating disorder, purging disorder, binge-purge subtype AN).

Strengths of the current study included a prospective, longitudinal design with a large cohort of children. This design allows for a robust test of whether differences at earlier ages associate with later eating pathology. An additional strength was multiple measures of social processing, including a task-based measure and a parent-report measure at multiple ages, assessment of eating disorder psychopathology at multiple ages, and assessment of risk for eating disorders among boys. Limitations of the current study include low power to detect certain effects due to a small percentage of children, particularly boys, endorsing eating pathology, and a relatively homogenous sample (96% Caucasian) from one geographic area. It is possible that associations in the current study do not generalize to other populations, and, as such, replication in other cohorts would bolster confidence in our findings. Further, although burgeoning research demonstrates associations between autism traits and behaviors characteristic of avoidant-restrictive food intake disorder [27, 54], we did not capture this phenotype of eating pathology in the current study. Given that avoidant-restrictive food intake disorder may demonstrate a younger age of onset compared with other eating disorders [55, 56], studies examining avoidant-restrictive eating patterns, specifically, may choose to focus on an earlier developmental window.

Overall, current findings clarify literature that demonstrates associations between eating disorders and social processes. As a whole, emotion recognition and social communication difficulties are not strongly associated with adolescent eating pathology among this sample. Though not explicitly tested in this study, this brings to light the possibility that research that recruits clinical samples of individuals with eating disorders may include an overrepresentation of those with symptoms of social communication disorders, as compared to rates found in the overall population. We did find evidence for specific associations between social communication difficulties and bulimic-spectrum pathology along with limited support for the hypothesis that bias toward negatively-valenced interpretation of social stimuli associates prospectively with eating disorder risk. Further longitudinal

research will aid in clarifying whether mechanisms of association between social processing deficits and eating disorder risk are unidirectional, bidirectional or influenced by broader underlying vulnerability that is differentially expressed across development.

## Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

## Acknowledgements:

We are extremely grateful to all the families who took part in this study, the midwives for their help in recruiting them, and the whole ALSPAC team, which includes interviewers, computer and laboratory technicians, clerical workers, research scientists, volunteers, managers, receptionists and nurses.

### Acknowledgement of Funding

This manuscript was supported by grants from the National Institute of Mental Health (R21MH109917). The UK Medical Research Council and Wellcome (Grant ref: 217065/Z/19/Z) and the University of Bristol provide core support for ALSPAC. This publication is the work of the authors and they will serve as guarantors for the contents of this paper. A comprehensive list of grants funding is available on the ALSPAC website (<http://www.bristol.ac.uk/alspac/external/documents/grant-acknowledgements.pdf>) This research was specifically funded by NIH 5R01MH073842.

## References

1. Adolphs R (2001) The neurobiology of social cognition. *Curr Opin Neurobiol* 11:231–239. 10.1016/S0959-4388(00)00202-6 [PubMed: 11301245]
2. American Psychiatric Association (2013) Diagnostic and Statistical Manual of Mental Disorders
3. Oldershaw A, Hambrook D, Stahl D, et al. (2011) The socio-emotional processing stream in Anorexia Nervosa. *Neurosci Biobehav Rev* 35:970–988. 10.1016/j.neubiorev.2010.11.001 [PubMed: 21070808]
4. Mandy W, Tchanturia K (2015) Do women with eating disorders who have social and flexibility difficulties really have autism? A case series. *Mol Autism* 6:6. 10.1186/2040-2392-6-6 [PubMed: 26056560]
5. Rhind C, Bonfili E, Hibbs R, et al. (2014) An examination of autism spectrum traits in adolescents with anorexia nervosa and their parents. *Mol Autism* 5:56. 10.1186/2040-2392-5-56 [PubMed: 25553237]
6. Zucker NL, Losh M, Bulik CM, et al. (2007) Anorexia Nervosa and Autism Spectrum Disorders: Guided Investigation of Social Cognitive Endophenotypes. *Psychol Bull* 133:
7. Cardi V, Matteo RD, Corfield F, Treasure J (2013) Social reward and rejection sensitivity in eating disorders: An investigation of attentional bias and early experiences. *World J Biol Psychiatry* 14:622–633. 10.3109/15622975.2012.665479 [PubMed: 22424288]
8. Cserjési R, Vermeulen N, Lénárd L, Luminet O (2011) Reduced capacity in automatic processing of facial expression in restrictive anorexia nervosa and obesity. *Psychiatry Res* 188:253–257. 10.1016/j.psychres.2010.12.008 [PubMed: 21208661]
9. Harrison A, Tchanturia K, Treasure J (2010) Attentional Bias, Emotion Recognition, and Emotion Regulation in Anorexia: State or Trait? *Biol Psychiatry* 68:755–761. 10.1016/j.biopsych.2010.04.037 [PubMed: 20591417]
10. Harrison A, Genders R, Davies H, et al. (2011) Experimental measurement of the regulation of anger and aggression in women with anorexia nervosa. *Clin Psychol Psychother* 18:445–452. 10.1002/cpp.726 [PubMed: 20859934]
11. Kessler H, Schwarze M, Filipic S, et al. (2006) Alexithymia and facial emotion recognition in patients with eating disorders. *Int J Eat Disord* 39:245–251. 10.1002/eat.20228 [PubMed: 16485269]

12. Aloï M, Rania M, Caroleo M, et al. (2017) Social Cognition and Emotional Functioning in Patients with Binge Eating Disorder. *Eur Eat Disord Rev* 25:172–178. 10.1002/erv.2504 [PubMed: 28211586]
13. DeJong H, Van den Eynde F, Broadbent H, et al. (2013) Social cognition in bulimia nervosa: A systematic review. *Eur Psychiatry* 28:1–6. 10.1016/j.eurpsy.2011.07.002 [PubMed: 21920709]
14. Dapelo MM, Surguladze S, Morris R, Tchanturia K (2017) Emotion Recognition in Face and Body Motion in Bulimia Nervosa. *Eur Eat Disord Rev* 25:595–600. 10.1002/erv.2554 [PubMed: 28960589]
15. Westwood H, Tchanturia K (2017) Autism Spectrum Disorder in Anorexia Nervosa: An Updated Literature Review. *Curr Psychiatry Rep* 19:41. 10.1007/s11920-017-0791-9 [PubMed: 28540593]
16. Ambwani S, Berenson KR, Simms L, et al. (2016) Seeing things differently: An experimental investigation of social cognition and interpersonal behavior in anorexia nervosa. *Int J Eat Disord* 49:499–506. 10.1002/eat.22498 [PubMed: 26712303]
17. Renwick B, Musiat P, Lose A, et al. (2015) Neuro- and social-cognitive clustering highlights distinct profiles in adults with anorexia nervosa. *Int J Eat Disord* 48:26–34. 10.1002/eat.22366 [PubMed: 25363476]
18. Micali N, Solmi F, Horton NJ, et al. (2015) Adolescent Eating Disorders Predict Psychiatric, High-Risk Behaviors and Weight Outcomes in Young Adulthood. *J Am Acad Child Adolesc Psychiatry* 54:652–659.e1. 10.1016/j.jaac.2015.05.009 [PubMed: 26210334]
19. Hudson JI, Hiripi E, Pope HG, Kessler RC (2007) The prevalence and correlates of eating disorders in the National Comorbidity Survey Replication. *Biol Psychiatry* 61:348–58. 10.1016/j.biopsych.2006.03.040 [PubMed: 16815322]
20. Cisler JM, Bush K, Scott Steele J, et al. (2015) Brain and behavioral evidence for altered social learning mechanisms among women with assault-related posttraumatic stress disorder. *J Psychiatr Res* 63:75–83. 10.1016/j.jpsychires.2015.02.014 [PubMed: 25769397]
21. Collin L, Bindra J, Raju M, et al. (2013) Facial emotion recognition in child psychiatry: A systematic review. *Res Dev Disabil* 34:1505–1520. 10.1016/j.ridd.2013.01.008 [PubMed: 23475001]
22. Wyer JrRS, Srull TK (2014) Social cognition and clinical psychology: Anxiety, depression, and the processing of social information. In: *Handbook of social cognition*. Psychology Press, pp 305–354
23. Kanakam N, Krug I, Raouf C, et al. (2013) Social and Emotional Processing as a Behavioural Endophenotype in Eating Disorders: A Pilot Investigation in Twins. *Eur Eat Disord Rev* 21:294–307. 10.1002/erv.2232 [PubMed: 23649701]
24. Kothari, Barona MMs, Treasure JOPFFrcp, Micali NP (2015) Social cognition in children at familial high-risk of developing an eating disorder. *Front Behav Neurosci* 9:. 10.3389/fnbeh.2015.00208
25. Koch SV, Larsen JT, Mouridsen SE, et al. (2015) Autism spectrum disorder in individuals with anorexia nervosa and in their first- and second-degree relatives: Danish nationwide register-based cohort-study. *Br J Psychiatry* 206:401–407. 10.1192/bjp.bp.114.153221 [PubMed: 25657359]
26. Solmi F, Bentivegna F, Bould H, et al. Trajectories of autistic social traits in childhood and adolescence and disordered eating behaviours at age 14 years: A UK general population cohort study. *J Child Psychol Psychiatry* n/a: 10.1111/jcpp.13255
27. van 't Hof M, Ester WA, Serdarevic F, et al. (2020) The sex-specific association between autistic traits and eating behavior in childhood: An exploratory study in the general population. *Appetite* 147:104519. 10.1016/j.appet.2019.104519 [PubMed: 31738945]
28. Dinkler L, Taylor MJ, Råstam M, et al. (in Press) Anorexia nervosa and autism: a prospective twin cohort study. *J Child Psychol Psychiatry*, 10.1111/jcpp.13265
29. Boyd A, Golding J, Macleod J, et al. (2013) Cohort Profile: the 'children of the 90s'—the index offspring of the Avon Longitudinal Study of Parents and Children. *Int J Epidemiol* 42:111–27. 10.1093/ije/dys064 [PubMed: 22507743]
30. Fraser A, Macdonald-Wallis C, Tilling K, et al. (2013) Cohort Profile: The Avon Longitudinal Study of Parents and Children: ALSPAC mothers cohort. *Int J Epidemiol* 42:97–110. 10.1093/ije/dys066 [PubMed: 22507742]

31. Golding J, Pembrey M, Jones R, ALSPAC ST (2001) ALSPAC—the Avon Longitudinal Study of Parents and Children. I. Study methodology. *Paediatr Perinat Epidemiol* 15:74–87 [PubMed: 11237119]
32. Kann L, Warren CW, Harris WA, et al. (1996) Youth risk behavior surveillance—United States, 1995. *J Sch Health* 66:365–77 [PubMed: 8981266]
33. Field AE, Taylor CB, Celio A, Colditz GA (2004) Comparison of self-report to interview assessment of bulimic behaviors among preadolescent and adolescent girls and boys. *Int J Eat Disord* 35:86–92. 10.1002/eat.10220 [PubMed: 14705161]
34. Schaumberg K, Brosos LC, Lloyd EC, et al. (2020) Prospective associations between childhood neuropsychological profiles and adolescent eating disorders. *Eur Eat Disord Rev* 28:156–169. 10.1002/erv.2721 [PubMed: 31994257]
35. Berscheid E, Walster E, Bohrnstedt G (1973) The Happy American Body: A Survey Report. *Psychol Today* 6:119–123
36. Stice E, Nemeroff C, Shaw HE (1996) Test of the dual pathway model of bulimia nervosa: Evidence for dietary restraint and affect regulation mechanisms. *J Soc Clin Psychol* 15:340–363
37. Van Strien T, Frijters JER, Bergers G, Defares PB (1986) The Dutch Eating Behavior Questionnaire (DEBQ) for assessment of restrained, emotional, and external eating behavior. *Int J Eat Disord* 5:295–315. 10.1002/1098-108X(198602)5:2<295::AID-EAT2260050209>3.0.CO;2-T
38. Calzo JP, Austin SB, Micali N (2018) Sexual orientation disparities in eating disorder symptoms among adolescent boys and girls in the UK. *Eur Child Adolesc Psychiatry*. 10.1007/s00787-018-1145-9
39. Cole TJ, Flegal KM, Nicholls D, Jackson AA (2007) Body mass index cut offs to define thinness in children and adolescents: international survey. *BMJ* 335:194. 10.1136/bmj.39238.399444.55 [PubMed: 17591624]
40. House J, Eisler I, Simic M, Micali N (2008) Diagnosing eating disorders in adolescents: a comparison of the eating disorder examination and the development and well-being assessment. *Int J Eat Disord* 41:535–41. 10.1002/eat.20528 [PubMed: 18433028]
41. Skuse DH, Mandy WPL, Scourfield J (2005) Measuring autistic traits: heritability, reliability and validity of the Social and Communication Disorders Checklist. *Br J Psychiatry* 187:568–572. 10.1192/bjp.187.6.568 [PubMed: 16319410]
42. Skuse DH, Mandy W, Steer CD, et al. (2009) Social communication competence and functional adaptation in a general population of children: preliminary evidence for sex-by-verbal IQ differential risk. *J Am Acad Child Adolesc Psychiatry* 48:128–137. 10.1097/CHI.0b013e31819176b8 [PubMed: 19106766]
43. Kothari, Skuse D, Wakefield J, Micali N (2013) Gender differences in the relationship between social communication and emotion recognition. *J Am Acad Child Adolesc Psychiatry* 52:1148–1157.e2. 10.1016/j.jaac.2013.08.006 [PubMed: 24157389]
44. Nowicki S, Duke MP (1994) Individual differences in the nonverbal communication of affect: The Diagnostic Analysis of Nonverbal Accuracy Scale. *J Nonverbal Behav*
45. Allison P (2001) Missing Data
46. Murray SB, Strober M, Craske MG, et al. (2018) Fear as a translational mechanism in the psychopathology of anorexia nervosa. *Neurosci Biobehav Rev* 95:383–395. 10.1016/j.neubiorev.2018.10.013 [PubMed: 30392878]
47. Brown M, Robinson L, Campione GC, et al. (2017) Intolerance of Uncertainty in Eating Disorders: A Systematic Review and Meta-Analysis. *Eur Eat Disord Rev*. 10.1002/erv.2523
48. Lavender JM, Wonderlich SA, Engel SG, et al. (2015) Dimensions of emotion dysregulation in anorexia nervosa and bulimia nervosa: A conceptual review of the empirical literature. *Clin Psychol Rev* 40:111–122. 10.1016/j.cpr.2015.05.010 [PubMed: 26112760]
49. Caglar-Nazali HP, Corfield F, Cardi V, et al. (2014) A systematic review and meta-analysis of ‘Systems for Social Processes’ in eating disorders. *Neurosci Biobehav Rev* 42:55–92. 10.1016/j.neubiorev.2013.12.002 [PubMed: 24333650]
50. Bishop SL, Hus V, Duncan A, et al. (2013) Subcategories of Restricted and Repetitive Behaviors in Children with Autism Spectrum Disorders. *J Autism Dev Disord* 43:1287–1297. 10.1007/s10803-012-1671-0 [PubMed: 23065116]

51. Antshel KM, Russo N (2019) Autism Spectrum Disorders and ADHD: Overlapping Phenomenology, Diagnostic Issues, and Treatment Considerations. *Curr Psychiatry Rep* 21:34. 10.1007/s11920-019-1020-5 [PubMed: 30903299]
52. Seitz J, Kahraman-Lanzerath B, Legenbauer T, et al. (2013) The Role of Impulsivity, Inattention and Comorbid ADHD in Patients with Bulimia Nervosa. *PLoS ONE* 8:e63891. 10.1371/journal.pone.0063891 [PubMed: 23700439]
53. Yao S, Kuja-Halkola R, Martin J, et al. (2019) Associations Between Attention-Deficit/Hyperactivity Disorder and Various Eating Disorders: A Swedish Nationwide Population Study Using Multiple Genetically Informative Approaches. *Biol Psychiatry* 86:577–586. 10.1016/j.biopsych.2019.04.036 [PubMed: 31301758]
54. Dovey TM, Kumari V, Blissett J, Mealtime Hostage Parent Science Gang (2019) Eating behaviour, behavioural problems and sensory profiles of children with avoidant/restrictive food intake disorder (ARFID), autistic spectrum disorders or picky eating: Same or different? *Eur Psychiatry J Assoc Eur Psychiatr* 61:56–62. 10.1016/j.eurpsy.2019.06.008
55. Kurz S, van Dyck Z, Dremmel D, et al. (2015) Early-onset restrictive eating disturbances in primary school boys and girls. *Eur Child Adolesc Psychiatry* 24:779–785. 10.1007/s00787-014-0622-z [PubMed: 25296563]
56. Nicely TA, Lane-Loney S, Masciulli E, et al. (2014) Prevalence and characteristics of avoidant/restrictive food intake disorder in a cohort of young patients in day treatment for eating disorders. *J Eat Disord* 2:. 10.1186/s40337-014-0021-3

**Table 1a.**

Percentage of boys and girls with DANVA scores above cutoff

	<b>Boys (n = 1555)</b>	<b>Girls (n = 2202)</b>
Happy Errors	25.9%	17.8%
Sad Errors	18.1%	15.4%
Angry Errors	19.4%	13.2%
Fearful Errors	17.6%	16.9%
Happy Misattributions	13.7%	11.8%
Sad Misattributions	19.2%	11.9%
Angry Misattributions	10.3%	11.5%
Fearful Misattributions	20.1%	18.1%

Note. DANVA – Diagnostic analysis of non-verbal accuracy.



**Table 1b.** Percentage of boys and girls scoring above clinical cutoff ( 9) on the Social Communication Disorders Checklist

	<b>Boys (n = 1815)</b>	<b>Girls (n = 2446)</b>
SCDC age 7	7.1%	4.5%
SCDC age 10	6.5%	4.1%
SCDC age 14	6.2%	4.6%

Note. SCDC = Social Communication Disorders Checklist

**Table 1c.** Percentage of boys and girls with eating disorder diagnoses at ages 14, 16, and 18

	Boys	Girls
BED 14	0.3%	0.5%
BED 16	0.6%	2.3%
BED 18	3.0%	7.8%
PD 14	0.1%	0.5%
PD 16	0.2%	2.3%
PD 18	0.4%	2.1%
BN 14	0.8%	1.9%
BN 16	1.5%	5.8%
BN 18	0.4%	2.0%
AN 14	1.5%	3.7%
AN 16	0.9%	2.5%
AN 18	1.0%	1.8%

Note. BED – binge eating disorder or subthreshold BED, PD – purging disorder, BN – bulimia nervosa or subthreshold BN, AN – anorexia nervosa; “14,” “16,” and “18” indicate age (in years) at assessment

**Table 1d.** Percentage of boys and girls endorsing eating disorder behaviors during adolescence

	Boys	Girls
Binge 14	3.5%	7.5%
Binge 16	4.8%	18.3%
Binge 18	7.5%	24.3%
Purge 14	0.6%	2.0%
Purge 16	1.3%	9.8%
Purge 18	1.5%	8.1%
Ex 14	6.3%	10.2%
Ex 16	11.1%	33.3%
Ex 18	9.8%	37.8%
Fast 14	2.0%	9.5%
Fast 16	1.2%	9.6%
Fast 18	1.0%	7.4%

Note. Binge = binge eating; Purge = purging; Ex = compulsive exercise; Fast = fasting; "14," "16," and "18" indicate age (in years) at assessment

**Table 2.**

Relationship between SCDC likely autism and eating disorder symptoms

		Eating Disorder Behaviors (Odds Ratios)					
		AGE 8		AGE 10		AGE 14	
		Boys	Girls	Boys	Girls	Boys	Girls
Binge 14		1.11(0.33, 3.67)	1.83(0.80, 4.17)	2.10(0.80, 5.53)	1.85(0.85, 4.02)	1.34(0.40, 4.47)	2.23(0.98, 4.64)
Binge 16		1.25(0.49, 3.23)	1.85(1.08, 3.18)	<b>3.08(1.50, 6.35) *</b>	1.82(1.06, 3.12)	2.36(1.03, 5.43)	<b>3.07(1.97, 5.04) *</b>
Binge 18		2.19(0.86, 5.59)	1.61(0.79, 3.30)	2.03(0.67, 6.18)	1.44(0.71, 2.92)	3.11(1.19, 8.14)	1.16(0.53, 2.53)
<b>Binge Lifetime</b>		1.50(0.74, 3.03)	1.41(0.82, 2.41)	1.52(0.73, 3.16)	1.37(0.80, 2.37)	2.14(1.05, 4.36)	<b>2.59(1.54, 4.34) *</b>
Purge 14		--	3.11(1.07, 9.06)	1.98(0.23, 17.17)	1.58(0.37, 6.75)	--	<b>4.83(1.80, 12.95) *</b>
Purge 16		4.09(1.32, 12.70)	1.76(0.95, 3.24)	<b>7.16(2.65, 19.33) *</b>	1.27(0.64, 2.51)	1.95(0.44, 8.57)	1.03(0.49, 2.17)
Purge 18		1.42(0.18, 11.46)	0.49(0.12, 2.05)	---	0.48(0.11, 2.00)	--	0.87(0.26, 2.85)
<b>Purge Lifetime</b>		1.89(0.65, 5.55)	1.36(0.75, 2.47)	2.60(0.97, 6.99)	0.84(0.4, 1.70)	0.51(0.07, 3.76)	1.46(0.79, 2.70)
Ex 14		1.07(0.62, 1.86)	1.91(1.14, 3.19)	0.75(0.41, 1.36)	0.98(0.58, 1.67)	1.23(0.69, 2.22)	1.13(0.67, 1.89)
Ex 16		1.04(0.53, 2.05)	1.05(0.64, 1.73)	1.21(0.62, 2.34)	0.88(0.53, 1.47)	0.57(0.22, 1.47)	1.03(0.62, 1.69)
Ex 18		0.75(0.25, 2.23)	0.71(0.32, 1.57)	2.16(0.86, 5.40)	1.05(0.50, 2.19)	1.62(0.51, 5.12)	1.04(0.48, 2.26)
<b>Ex Lifetime</b>		0.89(0.53, 1.49)	1.55(0.90, 2.68)	0.92(0.54, 1.56)	0.94(0.55, 1.59)	1.10(0.61, 1.97)	1.00(0.60, 1.68)
Fast 14		1.49(0.43, 5.12)	1.44(0.69, 3.00)	1.01(0.23, 4.42)	2.10(1.06, 4.14)	0.61(0.08, 4.60)	2.10(1.04, 4.24)
Fast 16		1.59(0.36, 7.08)	1.32(0.67, 2.61)	3.04(0.85, 10.88)	1.55(0.82, 2.91)	0.95(0.13, 7.29)	1.23(0.63, 2.43)
Fast 18		2.13(0.25, 18.16)	2.49(1.08, 5.74)	4.77(0.92, 24.81)	0.87(0.26, 2.87)	2.30(0.27, 19.51)	1.32(0.46, 3.79)
<b>Fast Lifetime</b>		1.47(0.55, 3.88)	1.80(1.08, 3.01)	1.66(0.62, 4.43)	<b>1.97(1.18, 3.27) *</b>	0.73(0.17, 3.12)	1.74(1.02, 2.95)
<b>Eating Disorder Symptoms at Age 14 (B)</b>							
<b>BD</b>		0.27(0.06, 0.48)	<b>0.39(0.17, 0.60) *</b>	0.24(0.03, 0.46)	0.25(0.02, 0.47)	0.28(0.06, 0.50)	<b>0.38(0.15, 0.60) *</b>
<b>Fear Wt Gain</b>		0.92(0.50, 1.68)	1.51(0.96, 2.22)	1.01(0.56, 1.83)	1.03(0.64, 1.65)	0.48(0.21, 1.07)	1.36(0.85, 2.16)
<b>Restraint/Dieting</b>		0.24(0.03, 0.44)	<b>0.37(0.16, 0.59) *</b>	0.09(-0.12, 0.30)	0.21(-0.01, 0.44)	-0.13(-0.34, 0.09)	0.22(-0.003, 0.45)
<b>Lose Wt</b>		0.11(-0.10, 0.31)	0.23(0.01, 0.44)	0.20(-0.01, 0.41)	<b>0.31(0.08, 0.54) *</b>	-0.10(-0.32, 0.12)	0.25(0.03, 0.48)
<b>Emotional Eating</b>		0.16(-0.07, 0.38)	0.30(0.06, 0.54)	0.23(0.003, 0.45)	0.25(-0.01, 0.50)	0.09(-0.15, 0.33)	0.26(0.01, 0.51)
<b>External Eating</b>		0.10(-0.14, 0.33)	0.08(-0.16, 0.32)	0.12(-0.13, 0.36)	0.05(-0.21, 0.31)	0.20(-0.05, 0.45)	0.14(-0.12, 0.40)

Eating Disorder Behaviors (Odds Ratios)						
	AGE 8		AGE 10		AGE 14	
	Boys	Girls	Boys	Girls	Boys	Girls
<b>TI Internalization</b>	0.21(-0.01, 0.43)	-0.01(-0.24, 0.23)	-0.06(-0.28, 0.17)	-0.10(-0.34, 0.14)	-0.11(-0.34, 0.13)	-0.12(-0.36, 0.12)
<b>Eating Disorder Diagnoses (Odds Ratios)</b>						
AN 14	--	1.61(0.57, 4.56)	0.85(0.11, 6.67)	2.35(0.86, 6.40)	--	1.75(0.59, 5.17)
AN 16	--	1.82(0.65, 5.06)	0.99(0.13, 7.74)	2.57(0.95, 6.95)	1.01(0.13, 7.95)	2.17(0.74, 6.38)
AN 18	--	2.91(0.76, 11.24)	--	4.64(1.24, 17.28)	--	1.59(0.20, 12.52)
<b>AN Lifetime</b>	--	1.12(0.45, 2.76)	0.42(0.06, 3.19)	1.89(0.83, 4.30)	0.35(0.05, 2.80)	1.54(0.62, 3.83)
BN 14	1.12(0.14, 8.76)	3.43(1.17, 10.09)	4.51(1.21, 16.80)	2.48(0.74, 8.37)	3.08(0.67, 14.16)	0.83(0.11, 6.20)
BN 16	1.21(0.28, 5.22)	2.16(1.09, 4.28)	<b>4.34(1.70, 11.03)*</b>	2.40(1.24, 4.65)	3.63(1.35, 9.80)	<b>3.58(1.98, 6.47)*</b>
BN 18	13.90(0.82, 235.65)	1.01(0.13, 7.70)	--	--	--	--
<b>BN Lifetime</b>	1.88(0.64, 5.51)	2.08(1.10, 3.94)	2.44(0.92, 6.51)	2.14(1.13, 4.06)	2.09(0.72, 6.07)	2.48(1.33, 4.63)
BED 14	--	--	--	2.20(0.28, 17.38)	--	2.69(0.33, 21.68)
BED 16	--	--	--	1.56(0.47, 5.15)	--	2.81(1.08, 7.32)
BED 18	1.78(0.52, 6.16)	0.98(0.34, 2.80)	2.15(0.62, 7.54)	2.29(1.04, 5.06)	2.63(0.75, 9.29)	1.08(0.38, 3.08)
<b>BED Lifetime</b>	1.21(0.36, 4.04)	0.58(0.21, 1.61)	1.43(0.43, 4.78)	2.02(1.04, 3.92)	1.73(0.51, 5.81)	1.62(0.79, 3.33)
PD 14	--	1.77(0.22, 14.01)	--	2.20(0.28, 17.50)	--	2.53(0.32, 20.12)
PD 16	--	1.71(0.52, 5.66)	--	1.55(0.47, 5.12)	--	1.04(0.25, 4.38)
PD 18	--	--	--	--	--	1.13(0.15, 8.59)
<b>PD Lifetime</b>	--	1.44(0.51, 4.07)	2.10(0.24, 18.18)	1.40(0.50, 3.96)	--	1.55(0.55, 4.38)

**Note.** Labels “4,” “16,” and “18” refer to age in years at assessment. SCDC = social communication disorders checklist. SCDC dichotomized at 9. Binge Eating, Purge = binge eating, purge = purging behavior, Ex = driven exercise, Fast = fasting behavior. BD = body dissatisfaction. Fear Wt Gain = fear of weight gain. Lose Wt = pressure to lose weight. TI Internalization = thin-ideal internalization. AN – anorexia nervosa, BN – bulimia nervosa, BED – binge-eating disorder, PD – purging disorder. Body Mass Index z-score at age 10 and socioeconomic status entered as covariates in analyses.

(\*): Bolded coefficients with reached significance after correcting for multiple comparisons with Benjamini-Hochberg correction. Odds ratios and coefficients are presented with standard errors in parentheses.