Running head: INFLEXIBLE INTERPRETATIONS AND RESTRICTIVE EATING

Biased and Inflexible Interpretations of Ambiguous Social Situations: Associations with Eating Disorder Symptoms and Socioemotional Functioning

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Abstract

Background: Research indicates that difficulties across multiple socioemotional functioning domains (in social emotion expression/regulation, response to social elicitors of emotion, etc.) and negatively biased interpretations of ambiguous social situations may affect eating disorder symptoms. The impact of inflexible interpretations of social situations on eating disorder symptoms is less clear. The present study therefore examined relations between inflexible and biased social interpretations, socioemotional functioning, and eating disorder symptoms.

Method: 310 participants from the general population, recruited online, completed measures of socioemotional functioning (e.g., rejection sensitivity, negative social exchange), eating disorder symptoms, and positive and negative interpretation bias and inflexibility on a single measurement occasion.

Results: Socioemotional functioning impairments (Pillai's trace=0.11, $p<.001$), but not negative ($\beta=.07$, $p=.162$) or positive ($\beta=-.01$, $p=.804$) interpretation bias or inflexible interpretations ($\beta=.04$, $p=.446$), were associated with eating disorder symptoms in multiple regression models. In network analyses controlling statistically for multiple markers of socioemotional functioning, eating disorder symptoms were directly associated with negative (but not positive) interpretation bias. Inflexible interpretations were indirectly linked to symptoms via co-dampening of positive emotions. Exploratory causal discovery analyses suggested that several socioemotional functioning variables (social anxiety, depression, negative social exchange) may cause eating disorder symptoms.

Conclusions: Consistent with cognitive-interpersonal models of disordered eating, our results suggest that less accurate (biased, inflexible) interpretations of social information contribute to patterns of cognition (anxious anticipation of rejection) and behavior (down-regulation of positive social emotion) thought to encourage disordered eating.

Keywords: Interpretation Bias, Interpretation Inflexibility, Restrictive Eating, GFCI, Socioemotional functioning

Availability of Data, Materials, and Code

Anonymized data and sample analysis code are available at: https://osf.io/x9fjn/. Materials (excluding the Emotional BADE Task) will be made available on reasonable request.

Conflicts of Interest

Conflicts of Interest: The authors have no conflicts to declare.

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Biased and Inflexible Interpretations of Ambiguous Social Situations: Associations with Eating Disorder Symptoms and Socioemotional Functioning

Cognitive-interpersonal models posit that eating disorder symptoms arise through the combination of emotional and interpersonal difficulties (Treasure et al., 2020; Treasure & Schmidt, 2013). In support of this possibility, interpersonal difficulties frequently precede (Anckarsäter et al., 2012) and predict (Haynos et al., 2016) disordered eating. This association may reflect the fact that negative emotions elicited by social strife become more salient when subjected to the particular patterns of emotion regulation that are more frequently used by individuals with disordered eating (for example, less self-reported use of effective emotion regulation strategies, such as reappraisal or reframing; Haynos et al., 2018; Lavender et al., 2015). Individuals with disordered eating may attempt to reduce aversive emotion-related physiological experiences through eating disorder symptoms, such as caloric restriction or binging and purging (Haynos et al., 2011; Turton et al., 2018). Certain eating disorder symptoms, including caloric restriction, may also, initially, elicit approving comments (e.g., those lauding weight loss; (Walsh, 2013). This social feedback may be particularly salient in the context of interpersonal difficulties that limit other opportunities for positive social interaction. Over time, however, eating disorder related behaviors may erode social cognition (Cardi et al., 2017; by, for example, reducing cortical volume in brain areas associated with social cognition, including the inferior frontal gyrus and right pars opercularis, see: Brodrick et al., 2021; King et al., 2015) and strain social relationships (by, for example, encouraging social withdrawal secondary to avoidance of social situations involving eating; Christensen & Haynos, 2020), leaving individuals increasingly isolated and reliant on disordered eating for emotion regulation (Arcelus et al., 2013).

These same cognitive-interpersonal models assert that the patterns of socioemotional functioning (a construct spanning social emotion expression and regulation, social elicitors of emotional responses, and social consequences of emotional reactions, see: Thompson & Virmani, 2012) that contribute to eating disorder symptoms may be caused, at least in part, by biased or less accurate interpretation of social information (Treasure et al., 2020; Treasure & Schmidt, 2013). Consistent with this assertion, negative interpretation bias, the tendency to systematically assign overly negative meanings to unclear situations (Loscalzo & Giannini, 2017), appears to increase rejection sensitivity in people with eating disorders (Cardi et al., 2017, 2019), which may prompt diminished supportiveness and hostility, perpetuating interpersonal difficulties (Downey & Feldman, 1996). Further, negative interpretation bias has been associated with emotion regulation strategies that upregulate negative emotion (like rumination; Everaert et al., 2020) in the general population. This bias may therefore exacerbate existing risk for emotion dysregulation among individuals with disordered eating (see: Haynos et al., 2018; Lavender et al., 2015). Biased negative interpretations may most strongly interfere with socioemotional functioning, and thereby encourage disordered eating, when they are inflexible (i.e., they do not adequately change as situations evolve). In the general population, there is a reliable association (controlling for interpretation bias) between inflexible negative interpretations and depression, social anxiety, and dampening of positive emotion (Everaert et al., 2018, 2020). Despite the association between these emotion-related constructs and disordered eating (Coniglio et al., 2019; Puccio et al., 2017; Swinbourne & Touyz, 2007), studies examining the effects of negative interpretation inflexibility (above and beyond the effects of interpretation bias) among individuals with disordered eating are lacking. Therefore, it remains unclear whether inflexible interpretations of social situations are associated with eating pathology via their effects on

socioemotional functioning, and, if so, whether this association persists when controlling for interpretation bias.

The Present Study

To help resolve the above ambiguities in the literature, the present study tested the following pre-registered (https://osf.io/trf5x) hypotheses in a general population sample of adults from the United States, recruited via an online marketplace (in these hypothesis statements, the term "continuous variation" refers to individual differences in total scores on a multi-item measure of eating disorder symptoms):

Hypothesis 1: Continuous variation in eating disorder symptoms will be associated with biased and inflexible interpretations.

Hypothesis 2: In cross-sectional partial correlation networks, inflexible and biased interpretations will be independently related to continuous variation in eating disorder symptoms via indices of socioemotional functioning

Hypotheses 3 & 4: Endorsement (vs. non-endorsement) of restrictive eating will be associated with impaired socioemotional functioning (H3) and biased and inflexible interpretations (H4).

As these hypotheses imply, both individual differences in eating disorder symptoms generally and restrictive eating specifically were of interest in the present study. Restrictive eating was probed as a specific outcome for two reasons: 1) nutritional effects of restrictive eating may directly reduce cognitive flexibility (Treasure & Schmidt, 2013), including in interpreting social situations and 2) much of the literature linking biased interpretation of social situations to eating disorder symptoms has employed participants with anorexia nervosa, a hallmark of which is restrictive eating.

In addition to these hypothesis tests, we conducted a pre-registered exploratory analysis using machine learning algorithms to probe potential causal pathways connecting eating disorder symptoms, socioemotional functioning, and interpretative processes.

Method

Participants and Recruitment

Participants (*n*=1308) ages 18+ living in the United States were recruited via MTurk, an online crowdsourcing platform. These participants completed a brief screening survey assessing the presence/absence of continuous eating disorder symptoms (using the Eating Pathology Symptoms Inventory [EPSI; Forbush et al., 2013) and restrictive eating behavior (using the Dietary Restriction Screener [DRS; Haynos & Fruzzetti, 2015). Responses were used to identify three groups of participants: below median EPSI score/denied restrictive eating, below median EPSI score/endorsed restrictive eating, and above median EPSI score/endorsed restrictive eating. We planned to recruit 150 individuals from each group for the main study to ensure sufficient variation in both restrictive eating and general eating disorder symptoms. However, lower than expected rates of participants in the "below median EPSI score/denied restrictive eating" group caused slight over-recruitment of the remaining groups. Note that because these groups were created to ensure sufficient variation in key outcomes, rather than clinically meaningful categories, group membership is not used in any analysis described below. The final sample for the main study (n=310, after all data quality exclusions) had an age range of 58 years (M = 45.90, SD = 13.42), and was 59% female (41% male). For more information on demographics and differential drop-out: see **SI Section S1**.

Open Science Practices

Hypotheses and analyses were pre-registered, and the dataset is public (Bronstein, 2021). For details, see: **SI Section S2**.

Protocol

This study was approved by the University of Minnesota Institutional Review Board. During study screening, participants completed demographic information, the EPSI, the DRS, and several attention checks. During the main study, participants then provided demographic information again and completed all of the measures listed in the **Measures** section. Measures were completed in randomized order. Payment was $6.

Measures

Eating Disorder Symptoms

The Eating Pathology Symptoms Inventory (EPSI; Forbush et al., 2013) was used to measure continuous variation in cognitive and behavioral eating disorder symptoms. Scores range from 0 to 220. This measure has strong psychometric properties, including high test-retest reliability and an invariant factor structure across development (Forbush et al., 2013; Richson et al., 2021). Higher scores reflect more frequent thoughts, feelings, and behaviors associated with eating disorders.

The Dietary Restriction Screener (DRS; Haynos & Fruzzetti, 2015) is a single-item measure assessing past-month restrictive eating. The DRS first clearly defines restrictive eating, provides examples, and asks participants to indicate whether they have engaged in restrictive eating in the past month. The DRS has been found to predict eating disorder symptoms, intended and actual food intake, and clinical severity in several studies (Fox et al., 2019; Wang et al.,

2018), including predicting reduced objective in vivo food intake better than other measures of restrictive eating (Haynos & Fruzzetti, 2015).

Depression/Social Anxiety

These measures were included because eating disorders and depression/social anxiety are highly comorbid (Godart et al., 2007), and because interpretation bias and inflexibility are associated with depression and social anxiety (Everaert et al., 2020).

The Physician's Health Questionnaire – Depression Module (PHQ-9; Kroenke et al., 2001) was used to measure depression symptom severity. Questionnaire items correspond to diagnostic criteria for depression from the Diagnostic and Statistical Manual of Mental Disorders (American Psychiatric Association, 2013). Scores range from 0 to 27.

Social Interaction Anxiety Scale (SIAS)/Social Phobia Scale (SPS) – Short Forms (Fergus et al., 2012). The SAIS and SPS cover two dimensions of social anxiety: generalized social interaction anxiety and specific fears of social scrutiny. Respondents rate items on five-point scales (1=Not at all true of me, 5=Very true of me). Short forms of these measures (11 items total) with favorable psychometric properties (Fergus et al., 2012) were used.

Interpretation Bias/Inflexibility

The Emotional Bias Against Disconfirmatory Evidence (BADE) Task (Everaert et al., 2018) **– Short Form** was used to measure positive and negative interpretation bias, as well as interpretation inflexibility (metric scoring: **SI Section S3**). Respondents are presented with ambiguous interpersonal scenarios (example: **SI Section S3**) with themes of social rejection and failure. These themes tap into the fear of negative evaluation and rejection frequently found in people with disordered eating, and are consistent with the adverse interpersonal experiences that frequently precede eating disorders (Cardi et al., 2019). Scenarios contain three statements. After

viewing each statement, participants rate the plausibility of four interpretations of scenario events using a 21-point scale (1="poor",21="excellent"). Across scenarios, interpretations can be grouped into Absurd, Lure, and True categories. Absurd interpretations are consistently implausible. Lure interpretations are most plausible initially, then become less so. True interpretations are moderately plausible initially, then become most plausible. Given this structure, optimal performance on the Emotional BADE Task requires participants to revise beliefs about the most plausible interpretation for a given scenario by integrating the disconfirmatory information provided by each of the latter two statements (Everaert et al., 2020).

The Emotional BADE Task includes scenarios that invite revision of negative interpretations in response to disconfirmatory positive information ("*disconfirming-the-negative scenarios*") or vice-versa ("*disconfirming-the-positive scenarios*"). Given past research suggesting that disconfirming-the-negative scenarios may be more strongly related to internalizing psychopathologies (e.g., depression) frequently comorbid with eating disorders (Everaert et al., 2018), we focus on results from these scenarios (results from *disconfirming-the-positive* scenarios: **SI Section S4**).

A short form of the Emotional BADE Task was constructed by selecting a subset of scenarios that produced bias/inflexibility metrics that correlated highly (rho>.80) with those from the full scenario set, and that produced findings consistent with those obtained in past research using the full task.

Socioemotional Functioning

These measures were selected to examine domains of functioning associated with eating disorders, including theory of mind (Leppanen et al., 2018), interpersonal styles that generate

interpersonal stress and may decrease social support (Cardi et al., 2017; Cooley et al., 2007; Lavender et al., 2015), and difficulty regulating positive emotions (Coniglio et al., 2019).

The Reading the Mind in the Eyes Test – Revised (Baron-Cohen et al., 2001) was used to measure theory of mind. This 36-item multiple-choice test asks participants to match eye regions to emotional experiences. This measure had poor internal consistency (ω_{total}=.59), and should be interpreted with caution.

The Depressive Interpersonal Relationships Inventory – Reassurance Seeking Subscale (DIRI-RS; Metalsky et al., 1991) was used to capture engagement in reassurance seeking and perceptions of other's reactions to this behavior. The four-item subscale asks respondents to rate how they usually act with close others. Subscale scores range from 4 to 28.

The Rejection Sensitivity Questionnaire (RSQ; Downey & Feldman, 1996) was used to assess expected rejection by close others (rated: 1=very unlikely to be accepted; 6=Very likely to be accepted) and associated anxiety (1=Very unconcerned, 6=Very Concerned). Scores are the product of anxiety ratings and reverse-scored expectations of acceptance.

The Test of Negative Social Exchange (TENSE; Finch et al., 1999) was used to measure anger, insensitivity, and interference in social relationships. Across 24 items, respondents rate the frequency of various instances of negative social exchange in their relationships in the past month (0=Not at all, 9=Frequently).

The Attitudes Toward Emotion Expression Scale (ATTE; Laghai & Joseph, 2000) was used to measure beliefs about emotional expression, including that expression should be controlled/suppressed, is a sign of weakness, and will prompt rejection. Scores range from 20 to 100. This measure was included because interpersonal difficulty in eating disorders is partially

fueled by avoidance of emotional expression and prioritization of others' feelings (Arcelus et al., 2013).

The Difficulties in Emotion Regulation Scale-Positive (DERS-Positive; Weiss et al., 2015) was used to measure dysregulation of positive emotions. Respondents rate (on a five-point scale: 1=Almost Never, 5=Almost Always) the frequency with which they had difficulties in goal-directed behavior, impulse-control, and emotion acceptance when experiencing positive emotion.

The Co-Dampening and Co-Enhancing Questionnaire – Co-Dampening Subscale (CO-DEQ; Bastin, Nelis, Raes, Vasey, & Bijttebier, 2018) was used to examine dampening of positive emotions in interpersonal relationships. Respondents rate nine items on a four-point scale (1=Almost Never, 4=Almost Always). Co-dampening occurs when repetitive patterns of discussion downregulate positive emotions (e.g., by reminding conversation partners that positive emotions are temporary and do not solve existing problems) (Bastin et al., 2018).

Internal Consistencies and Timeframes

All measures except the revised Reading the Mind in the Eyes Test had good-to-excellent internal consistencies, as evaluated using Omega Total (McDonald, 1999; see: **SI Section S5**). To align with the EPSI, ATTE, DERS-Positive, SIAS/SPS, and PHQ9 ratings were made with respect to experiences during the last month.

Analyses

For information on statistical test assumptions, see: **SI Section S6**.

Demographic Covariates

In all analyses, age, sex, and BMI were included as covariates. Age and sex were included because these variables are related to eating disorder symptoms (Forbush et al., 2014).

BMI was included as a proxy for factors (ex: malnutrition) that might create relations between eating disorder symptoms and performance on cognitive tasks (e.g., the Emotional BADE Task). Notably, results did not differ qualitatively when statistically controlling for sex vs. when stratifying the sample by sex.

Regression Models

The hypothesis that continuous variation in eating disorder symptoms would be associated with biased and inflexible interpretations (*Hypothesis 1*) *was* tested using a multiple regression model. EPSI scores were the criterion variable. Interpretation inflexibility/bias, BMI, age, sex, PHQ-9, and SPS/SIAS scores were predictors.

Hypothesis 4 was tested using a logistic regression model. DRS scores were the criterion variable. Predictors were identical to those in the linear regression model.

Multivariate Analysis of Covariance

Multivariate analysis of covariance (MANCOVA) was used to test *Hypothesis 3*. Criterion variables were negative social exchange, reassurance seeking, positive emotion dysregulation, co-dampening, attitudes to emotional expression, theory of mind, and rejection sensitivity. Independent variables were age, sex, BMI, and endorsement/non-endorsement of restrictive eating. Pillai's trace was used as the multivariate test statistic. Testing this hypothesis using MANCOVA, which probes for differences according to the linear combination of outcome variables, limits risk of Type I error.

Psychometric Network Analysis

Psychometric network analysis was used to test the hypothesis that inflexible and biased interpretations would be indirectly related to eating disorder symptoms via markers of socioemotional functioning (*Hypothesis 2*). Network analysis has several properties that make it

useful for identifying potential indirect pathways. Our partial-correlation network analysis identifies relations between variables that persist when controlling for many other variables of interest, including other markers of socioemotional functioning, making it more likely that the identified indirect pathways represent potential causal relations and are not due to the effects of third variables. Moreover, psychometric networks evaluate relations between variables simultaneously, reducing the chances of Type I error when identifying potential indirect pathways. For a more comprehensive introductions to network analysis accessible by unfamiliar readers, see Epskamp, Borsboom, Eiko, et al., 2018; Epskamp & Fried, 2018).

Edges were calculated using partial correlations, with regularization via the least absolute shrinkage and selection operator (LASSO; Tibshirani, 2016) using a tuning parameter (λ) that minimized the Extended Bayesian Information Criterion (EBIC; Chen and Chen, 2008). The EBIC hyper-parameter (γ) was set to 0.5, prioritizing avoidance of Type I errors (Foygel & Drton, 2010). Graphs were visualized using *R's qgraph* package, version *1.6.9* (Epskamp, 2020).

Network nodes were evaluated for redundancy using *R's networktools* package, version *1.2.3* (Jones, 2020). Variable pairs were considered redundant if (1) their zero-order correlation exceeded .70 (Elliott, Jones, and Schmidt, 2020) and (2) correlations between each member of the pair and all remaining variables in the network were not statistically significantly different in over 75% of cases (Marchetti, 2020; Meier et al., 2019).

For information about node centrality and predictability, network accuracy and stability, and exploratory difference tests, see **SI Section S7.**

Causal Discovery Analysis

Exploratory Causal discovery algorithms were used to investigate potential causal pathways involving inflexible and biased interpretations, socioemotional functioning, and

disordered eating. These algorithms, which exploit information (e.g., patterns of partial correlation) that indicates the plausibility of particular causal relations (**Figure 1**), can recover complex causal pathways from observational data (Shen et al., 2020).

The algorithm employed here-in, Greedy Fast Causal Inference (GFCI), searches the space of penalized likelihood scores of all possible acyclic causal relations among the measured variables to produce a preliminary assessment of likely causal pathways. This preliminary result is then iteratively refined by ruling out causal models that imply patterns of conditional independence inconsistent with the data. The output of this procedure is a partial ancestral graph (PAG), with the edge types (**Table 3**) varying depending on the set of directed edges that were present across all remaining plausible causal models (e.g., a directed edge [→] is present if, and only if, all models not containing that edge were removed during the steps outlined above). A particular strength of GFCI is its ability to detect unmeasured confounders, making it particularly well-suited to analyses of data from human research studies (where practical concerns, such as time limitations, constrain measurement of all relevant variables).

For information about analysis of graph stability, algorithm parameters, and exploratory analyses, see: **SI Section S8**. To provide an estimate of the potential causal effects identified by GFCI, a structural equation model containing edges corresponding to the potential causal effects suggested by GFCI was fit to the data. Model fits were examined using the following indices (and cutoffs for excellent fit): Comparative Fit Index (CFI; >.95), Root Mean Square Residual (RMSEA; <.06), Standardized Root Mean Square Residual (SRMR; <.08) (Hu and Bentler, 1999). Chi-square statistics were not considered because they tend to over-reject models in the presence of large sample sizes (Bentler and Bonett, 1980).

Outliers, Missing Data, and Data Quality

Univariate outliers were detected using the method of Hubert and Van Der Veeken (2008), as pre-registered. A total of 99 univariate outliers were detected and winsorized (Fuller, 1991). Given recent concerns about quality of data from online recruitment marketplaces (Burnette et al., 2021), multiple steps were taken to ensure high data quality, including use of attention checks (which were failed by less than 2% of our main study sample) and checks for response consistency. For additional information, see **SI Section S9**. The final sample contained no missing data.

Statistical Power

Sensitivity analyses suggest that with 310 individuals, the present study could detect even a small effect (f^2=0.02) of inflexibility or bias on continuous variation in eating disorder symptoms (H1). With this sample size, a small effect (odds ratio=1.40) of bias or inflexibility on the odds of endorsing restrictive eating could also be detected (H4). A similarly small global effect (f^2=0.05) of socioemotional functioning on endorsement of restrictive eating could also be detected (H3). Simulation studies suggest that ability to reject the null hypothesis that network edges do not differ from one another begins to plateau at around 250 participants. Thus, with 310 participants (Sacha Epskamp, Borsboom, & Fried, 2018), the present study is likely adequately powered to detect network edges of reasonable size that are statistically significantly different from zero (H4). A priori power analyses (based on the estimate of 360 participants) can be found in the pre-registration document.

Results

Descriptive Statistics

The sample's Eating Pathology Symptom Inventory scores (M=36.39, SD=21.70) were generally lower than those observed in previous studies (e.g., M=96.82 in college students; Forbush et al., 2013). However, almost half (45%) the sample reported engaging in restrictive eating behavior during the last month. As expected, EPSI scores were higher in women (M=40.63, SD=23.16), than in men (M=30.76, SD=17.95), $t(300)=4.22$, $p<.001$. The vast majority of participants (>96%) denied being ever diagnosed with or hospitalized for an eating disorder. For additional descriptive statistics, see **SI Section S1 and S5**.

Zero-order Correlations Between Study Variables

Eating disorder symptoms were moderately correlated with socioemotional functioning markers (including: negative social exchange [rho(308)=.51, $p<.001$], difficulty regulating positive emotions [rho(308)=.37, $p<.001$]) and negative interpretation bias (rho(308)=.19, $p<.001$), but were not associated with negative interpretation inflexibility (rho(308)=.08, $p=.138$) or positive interpretation bias (rho(308)=-.08, $p=.127$). Negative interpretation inflexibility was correlated with socioemotional functioning indices, including co-dampening of positive emotions (rho(308)=.13, $p=.022$) and reassurance seeking (rho(308)=.12, $p=.038$). For more information on zero-order correlations, see **SI Section S10**.

Hypothesis 1: Inflexible and Biased Interpretations are Associated with Eating Disorder Symptoms

The multiple regression model (**Table 2**) constructed to test the hypothesis that inflexible and biased interpretations are associated with continuous variation in eating disorder symptoms (*Hypothesis 1*) was significant $F(9,300)=15.60$, $p<.001$, and accounted for 30% of the variance

in eating pathology (adjusted R^2=.30). In contrast to *Hypothesis 1*, negative interpretation inflexibility and positive and negative interpretation bias were unrelated to eating disorder symptoms. Instead, symptoms of depression, β=0.28, 95%CI=[0.16 0.41], and social interaction anxiety, β=0.22, 95%CI=[0.07 0.37], were significant predictors of eating disorder symptoms. For results by subgroups who did vs. did not endorse restrictive eating, see: **SI Section S11**.

Hypothesis 2: Inflexible and Biased Interpretations Relate to Eating Disorder Symptoms via Socioemotional Functioning

The goldbricker function suggested that social phobia and interaction anxiety were redundant. They were therefore replaced with their linear combination ("social anxiety"). In the regularized network (**Figure 2**), eating disorder symptoms were strongly and directly associated with multiple markers of socioemotional functioning, including negative social exchange, depression, social anxiety, and co-dampening of positive emotions (a result consistent with *Hypothesis 3*, which posited that restrictive eating would be associated with lower scores on multiple measures of socioemotional functioning). Co-dampening of positive emotions, in turn, was associated with inflexible negative interpretations, forming an indirect pathway to eating disorder symptoms (as predicted by *Hypothesis 2*). When this indirect-only mediation pathway was formally tested via a structural equation model (which was an excellent fit to the data: CFI=1, RMSEA=0.00, SRMR=0.00), Monte Carlo simulation (with 1,000,000 replications) indicated it was significant, β=0.07, 95%CI=[0.02 0.11]. Positive interpretation bias was also only indirectly associated with eating disorder symptoms, via rejection sensitivity. Negative interpretation bias was both directly and indirectly (via rejection sensitivity, depression, and social anxiety) associated with eating disorder symptoms. Taken together, these results support

the hypothesis that inflexible and biased interpretations are associated with eating disorder symptoms indirectly, via markers of socioemotional functioning (*Hypothesis 2*).

For information on network accuracy/stability (which was acceptable) and node centrality, see **SI Section S7**.

Exploratory Causal Discovery Analyses

Causal discovery analysis was employed to examine whether pathways connecting biased and inflexible interpretations, socioemotional functioning, and eating disorder symptoms in this network likely reflected causal relations. The consensus PAG generated via causal discovery analysis is depicted in **Figure 3**. A structural equation model featuring the relations suggested by this PAG was an adequate fit to the data, RMSEA=.08, CFI=.90, SRMR=.10, and was therefore used to indicate the sizes of potential causal effects.

The PAG generated by GFCI identified negative social exchange and social interaction anxiety as potential direct causes of eating disorder symptoms. Depression and social phobia were related to eating disorder symptoms indirectly, via potential causal effects on social interaction anxiety. Other socioemotional functioning indices (e.g., rejection sensitivity, difficulty regulating positive emotion) were not causal ancestors of disordered eating.

GFCI also suggested that negative and positive interpretation bias were not causal ancestors of eating disorder symptoms. Instead, a less positive interpretation bias was a (potentially confounded) cause of rejection sensitivity, which was not causally upstream of symptoms. The algorithm did not identify negative interpretation bias as a cause or consequence of other variables in the model. Similarly, the consensus PAG suggested that inflexible negative interpretations were not related to eating disorder symptoms via socioemotional functioning (in contrast to *Hypothesis 2*, which posited the existence of these indirect pathways). Support was

found for the initial leg of this indirect pathway ("path a"): inflexible negative interpretations were identified as a (potentially confounded) cause of co-dampening of positive emotions. However, support for the second leg of this indirect causal pathway ("path b") was absent, despite theoretical and empirical work suggesting that positive emotion dysregulation causes eating disorder symptoms (Selby & Coniglio, 2020).

Hypothesis 3: Restrictive Eating is Associated with Impaired Socioemotional Functioning

The MANCOVA model (visualization: **SI Section S12**) indicated that individuals who endorsed (vs. denied) restrictive eating differed according to a linear combination of socioemotional functioning metrics (negative social exchange, excessive reassurance seeking, dysregulation of positive emotion, co-dampening, attitude to emotional expression, rejection sensitivity, and theory of mind), Pillai's trace=0.11, $F(7,299)=5.41$, $p<.001$. A permutational MANOVA (conducted using R's vegan package, without model covariates, to address traditional MANOVA assumption violations) confirmed the impact of restrictive eating on socioemotional functioning, $Z(1,308)=13.07$, $p=.001$. These results strongly suggest that restrictive eating is associated with impairment across multiple socioemotional functioning domains.

Hypothesis 4: Restrictive Eating is Associated with Inflexible and biased Interpretations

A logistic regression model (**Table 1**) was constructed to test the hypothesis that biased and inflexible interpretations would be associated with endorsement of restrictive eating (*Hypothesis 4*). Contrary to this hypothesis, inflexible and biased interpretations did not predict restrictive eating.

Discussion

This study investigated relations between interpretation bias and inflexibility in the context of ambiguous social situations, socioemotional functioning, and eating disorder symptoms. Consistent with our pre-registered hypotheses, endorsement (vs. non-endorsement) of restrictive eating was associated with a pattern of lower scores across multiple measures of socioemotional functioning. Mixed support was observed for our hypotheses regarding the relations between inflexible or biased interpretations and eating disorder symptoms. Inflexible and biased interpretations were not directly associated with eating disorder symptoms in our multiple regression models. However, as hypothesized, network analyses revealed indirect relations between eating disorder symptoms and inflexible and biased interpretations (via co-dampening and rejection sensitivity, respectively). Exploratory causal discovery analyses suggested that several socioemotional functioning variables (social anxiety, depression, and negative social exchange) may cause eating disorder symptoms. Causal discovery analysis also indicated that inflexible interpretations may cause co-dampening, although neither construct appeared to cause eating disorder symptoms.

These results accord with cognitive-interpersonal models positing that the patterns of socioemotional functioning that contribute to eating disorders may be caused, at least in part, by biased or less accurate interpretation of social information (Treasure et al., 2020; Treasure & Schmidt, 2013). Consistent with this notion, our causal discovery analysis suggested that a less positive interpretation bias increases the tendency to perceive and anxiously expect rejection. Although theory suggests that disordered eating may emerge as an attempt to regulate negative emotions, including those resulting from this tendency, and to increase social acceptance (Turton et al., 2018), rejection sensitivity was not a causal ancestor of eating disorder symptoms in the

present study. This result was unexpected given that rejection sensitivity encourages behaviors that perpetuate interpersonal difficulties (Downey & Feldman, 1996), which we identified as a causal ancestor of eating disorder symptoms. Accordingly, future research should re-examine whether bias toward less positive interpretations encourages disordered eating via its effects on rejection sensitivity and interpersonal strife, perhaps by employing cognitive bias modification paradigms (e.g., Cardi et al., 2019) in individuals with eating disorders.

The present study is also broadly consistent with cognitive-interpersonal models asserting that disordered eating impedes pleasurable social experience (Treasure & Schmidt, 2013), potentially encouraging a vicious cycle of disordered eating and difficulty experiencing positive emotions (see: Coniglio et al., 2019; Selby & Coniglio, 2020). This cycle may be accelerated by the deleterious effects of disordered eating on cognitive flexibility (Miles et al., 2020) and social cognition (Treasure & Schmidt, 2013), a possibility congruent with the potential causal relation between inflexible negative interpretations and co-dampening observed in this study. Admittedly, this account implies that inflexible negative interpretations and co-dampening should have emerged as potential causal ancestors of disordered eating in the present study. This expected result would have been consistent with previous research showing that inflexible negative interpretations are related to two likely causal ancestors of disordered eating in the present study – depression and social anxiety – via co-dampening (Everaert et al., 2020). The aforementioned account also implies that eating disorder symptoms should have emerged as a cause of inflexible negative interpretations. However, incorporation of both these missing causal pathways would have violated our causal discovery algorithm's assumption that causal graphs are acyclic (i.e., that there are no vicious/virtuous cycles of causation). Future research should

therefore re-examine whether inflexible interpretations cause eating disorder symptoms via their effects on co-dampening and internalizing psychopathology.

Finally, our results lend credence to cognitive-interpersonal models' assignment of a key role to criticism, hostility, and other negative interactions with close others in the formation and maintenance of eating disorder symptoms (Treasure & Schmidt, 2013). In support of this position, in the partial ancestral graph produced by our causal discovery analysis, negative social exchange was a potential direct cause of eating disorder symptoms and the causally primary node in an indirect pathway to eating disorder symptoms via psychopathologies involving negative affect (depression, social anxiety). This pathway accords with the notion that eating disorder behaviors, such as calorie restriction, may be employed to regulate negative emotions (Haynos et al., 2011; Turton et al., 2018), including those elicited by negative social interactions. Negative social exchange was also a causal ancestor of excessive reassurance seeking, rejection sensitivity, and the belief that emotion expression should be suppressed because it will prompt rejection, all of which are thought to encourage eating disorder symptoms (Cardi et al., 2017; Christensen & Haynos, 2020). Given the position of negative social exchange in this potential causal graph, future research should investigate whether treatments targeting interpersonal strife (e.g., family therapy) alleviate eating disorder symptoms by decreasing behaviors (e.g., excessive reassurance seeking) that may lead to expectations of (and actual) social rejection and by improving syndromes involving negative affect (depression, social anxiety).

The implications of the present study should be considered in the context of several limitations. Our sample's low symptom severity (see **Descriptive Statistics**) may explain why we found no association between eating disorder symptoms and inflexible interpretations. More severe symptoms may have nutritional sequalae that interfere with cognitive flexibility (Miles et

al., 2020), and neurological sequelae that interfere with social cognition (Brodrick et al., 2021). Moreover, more severe symptoms may elicit reactions from close others (anxiety/depression, accommodating behaviors; see Treasure et al., 2020) that perpetuate interpersonal stress and impact social emotion regulation in ways not captured by the present study. Future research should re-examine the relation between inflexible interpretations and eating disorder symptoms in people with clinically significant eating disorders. Alternatively, our use of a short form Emotional BADE Task may explain this null finding. While the present study conceptually replicated certain results obtained with the full task (e.g., the relation between [co-]dampening and inflexibility; Everaert et al., 2020), it failed to replicate others (e.g., the association between inflexible interpretations and depression/social anxiety, controlling for interpretation bias; Everaert et al., 2018, 2020). Given this inconsistency, future research should employ the full task until an improved short form is developed. Other potential limitations stem from our use of an MTurk sample, which may not be representative of nonclinical populations experiencing eating disorder symptoms. This limitation, which is mitigated by the congruence between our study and previous work (e.g., in implicating interpersonal strife as a cause of eating disorder symptoms; Cardi et al., 2018), could be addressed by replicating our study in a more representative sample. A final limitation of this study concerns its use of cross-sectional data for causal discovery. Casual discovery analyses may more accurately recover casual relations when temporal information is provided (Shen et al., 2020). Moreover, the set of contemporaneous causal relations between variables may differ from that unfolding across time. Future work should therefore use causal discovery analysis to examine the relations between interpretation processes, socioemotional functioning, and psychopathology in longitudinal datasets. While not a limitation per se, a final consideration is that Emotional BADE Task scenarios focus on themes of

interpersonal rejection/social failure. A stronger relation between disordered eating and interpretation processes might have been found using scenarios with more eating-disorder-specific content (Korn et al., 2020).

Conclusion

The present study deepens our understanding of the relations between socioemotional functioning and psychopathology by suggesting that negative social exchange increases depression, thereby promoting social anxiety and eating disorder symptoms. It also extends past work linking interpretation of ambiguous social situations to socioemotional functioning and disordered eating (e.g., Cardi et al., 2017) by identifying inflexible negative interpretations as a likely cause of co-dampening and implicating less positively biased interpretations as a likely cause of rejection sensitivity. These findings provide strong foundations for future research using the Emotional BADE Task to probe causal pathways to disordered eating in populations with higher symptom levels.

Author Contributions

M.V. Bronstein and J. Everaert developed the study design and concept, which S. Vinogradov and A. Haynos helped refine. M.V. Bronstein analyzed the data. E. Kummerfeld provided expertise surrounding causal discovery analyses. M.V. Bronstein drafted the manuscript. A. Haynos, J. Everaert, and S. Vinogradov provided critical revisions. All authors approved the final manuscript for submission.

References

American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders (5th ed.)*. American Psychiatric Association.

Anckarsäter, H., Hofvander, B., Billstedt, E., Gillberg, I. C., Gillberg, C., Wentz, E., & Råstam, M. (2012). The sociocommunicative deficit subgroup in anorexia nervosa: Autism spectrum disorders and neurocognition in a community-based, longitudinal study. *Psychological Medicine*, *42*(9), 1957–1967. https://doi.org/10.1017/S0033291711002881

Arcelus, J., Haslam, M., Farrow, C., & Meyer, C. (2013). The role of interpersonal functioning in the maintenance of eating psychopathology: A systematic review and testable model. *Clinical Psychology Review*, *33*(1), 156–167. https://doi.org/10.1016/j.cpr.2012.10.009

Baron-Cohen, S., Wheelwright, S., Hill, J., Raste, Y., & Plumb, I. (2001). The "Reading the Mind in the Eyes" Test revised version: A study with normal adults, and adults with Asperger syndrome or high-functioning autism. *Journal of Child Psychology and Psychiatry and Allied Disciplines*, *42*(2), 241–251. https://doi.org/10.1017/S0021963001006643

Bastin, M., Nelis, S., Raes, F., Vasey, M. W., & Bijttebier, P. (2018). Party Pooper or Life of the Party: Dampening and Enhancing of Positive Affect in a Peer Context. *Journal of Abnormal Child Psychology*, *46*(2), 399–414. https://doi.org/10.1007/s10802-017-0296-3

Bentler, P. M., & Bonett, D. G. (1980). Significance Tests and Goodness of Fit in the Analysis of Covariance Structures. In *Psychological Bulletin* (Vol. 88, Issue 3).

Brodrick, B. B., Adler-Neal, A. L., Palka, J. M., Mishra, V., Aslan, S., & McAdams, C. J. (2021). Structural brain differences in recovering and weight-recovered adult outpatient

women with anorexia nervosa. *Journal of Eating Disorders*, *9*(1), 1–12. https://doi.org/10.1186/s40337-021-00466-w

Bronstein, M. V. (2021). *Biased and Inflexible Interpretations of Ambiguous Social Situations: Associations with Restrictive Eating Behavior and Socioemotional Functioning.* osf.io/x9fjn

Burnette, C. B., Luzier, J. L., Bennett, B. L., Weisenmuller, C. M., Kerr, P., Martin, S., Keener, J., & Calderwood, L. (2021). Concerns and recommendations for using Amazon MTurk for eating disorder research. *International Journal of Eating Disorders*, *September*, 1–10. https://doi.org/10.1002/eat.23614

Cardi, V., Tchanturia, K., & Treasure, J. (2018). Premorbid and Illness-related Social Difficulties in Eating Disorders: An Overview of the Literature and Treatment Developments. *Current Neuropharmacology*, *16*(8), 1122–1130. https://doi.org/10.2174/1570159x16666180118100028

Cardi, V., Turton, R., Brazil, C., Harrison, A., Rowlands, K., Treasure, J., & Hirsch, C. (2019). Training Rejection Interpretation in Eating disordeRs (TRIER): Preliminary Findings of a Feasibility Study in Adolescents with Anorexia Nervosa. *Cognitive Therapy and Research*, *43*(6), 1109–1117. https://doi.org/10.1007/s10608-019-10034-x

Cardi, V., Turton, R., Schifano, S., Leppanen, J., Hirsch, C. R., & Treasure, J. (2017). Biased Interpretation of Ambiguous Social Scenarios in Anorexia Nervosa. *European Eating Disorders Review*, *25*(1), 60–64. https://doi.org/10.1002/erv.2493

Chen, J., & Chen, Z. (2008). Extended Bayesian information criteria for model selection with large model spaces. *Biometrika*, *95*(3), 759–771. https://doi.org/10.1093/biomet/asn034

Christensen, K. A., & Haynos, A. F. (2020). A theoretical review of interpersonal emotion regulation in eating disorders: Enhancing knowledge by bridging interpersonal and affective dysfunction. *Journal of Eating Disorders*, *8*(1), 1–10. https://doi.org/10.1186/s40337-020-00298-0

Coniglio, K. A., Christensen, K. A., Haynos, A. F., Rienecke, R. D., & Selby, E. A. (2019). The posited effect of positive affect in anorexia nervosa: Advocating for a forgotten piece of a puzzling disease. *International Journal of Eating Disorders*, *52*(9), 971–976. https://doi.org/10.1002/eat.23147

Cooley, E., Toray, T., Valdez, N., & Tee, M. (2007). Risk factors for maladaptive eating patterns in college women. *Eating and Weight Disorders*, *12*(3), 132–139. https://doi.org/10.1007/BF03327640

Downey, G., & Feldman, S. I. (1996). Implications of rejection sensitivity for intimate relationships. *Journal of Personality and Social Psychology*, *70*(6), 1327–1343. https://doi.org/10.1037//0022-3514.70.6.1327

Elliott, H., Jones, P. J., & Schmidt, U. (2020). Central Symptoms Predict Posttreatment Outcomes and Clinical Impairment in Anorexia Nervosa: A Network Analysis. *Clinical Psychological Science*, *8*(1), 139–154. https://doi.org/10.1177/2167702619865958

Epskamp, Sacha, Borsboom, · Denny, Eiko, ·, & Fried, I. (2018). Estimating psychological networks and their accuracy: A tutorial paper. *Behav Res*, *50*, 195–212. https://doi.org/10.3758/s13428-017-0862-1

Epskamp, Sacha, Borsboom, D., & Fried, E. I. (2018). Estimating psychological networks and their accuracy: Suplementary Materials. *Behavior Research Methods*, *50*(1), 195–212.

https://doi.org/10.3758/s13428-017-0862-1

Epskamp, Sacha, & Fried, E. I. (2018). A Tutorial on Regularized Partial Correlation Networks. *Psychological Methods*, *23*(4), 617–637. https://doi.org/10.1037/met0000167

Epskamp, Sasha. (2021). *qgraph* (1.6.9). http://sachaepskamp.com/qgraph

Everaert, J., Bronstein, M. V., Cannon, T. D., & Joormann, J. (2018). Looking Through Tinted Glasses: Depression and Social Anxiety Are Related to Both Interpretation Biases and Inflexible Negative Interpretations. *Clinical Psychological Science*, *6*(4), 517–528. https://doi.org/10.1177/2167702617747968

Everaert, J., Bronstein, M. V., Castro, A., Cannon, T. D., & Joormann, J. (2020). When negative interpretations persist, positive emotions don't! Inflexible negative interpretations encourage depression and social anxiety by dampening positive emotions. *Behaviour Research and Therapy*, *124*, 103510. https://doi.org/10.1016/j.brat.2019.103510

Fergus, T. A., Valentiner, D. P., McGrath, P. B., Gier-Lonsway, S. L., & Kim, H. S. (2012). Short forms of the social interaction anxiety scale and the social phobia scale. *Journal of Personality Assessment*, *94*(3), 310–320. https://doi.org/10.1080/00223891.2012.660291

Finch, J. F., Okun, M. A., Pool, G. J., & Ruehlman, L. S. (1999). A Comparison of the Influence of Conflictual and Supportive Social Interactions on Psychological Distress. *Journal of Personality*, *67*(4), 581–621. https://doi.org/10.1111/1467-6494.00066

Forbush, K. T., Wildes, J. E., & Hunt, T. K. (2014). Gender norms, psychometric properties, and validity for the Eating Pathology Symptoms Inventory. *International Journal of Eating Disorders*, *47*(1), 85–91. https://doi.org/10.1002/eat.22180

Forbush, K. T., Wildes, J. E., Pollack, L. O., Dunbar, D., Luo, J., Patterson, K., Petruzzi, L., Pollpeter, M., Miller, H., Stone, A., Bright, A., & Watson, D. D. of P. S. (2013). Development and validation of the eating pathology symptoms inventory (EPSI). *Psychological Assessment*, *25*(3), 859–878. https://doi.org/10.1037/a0032639

Fox, K. R., Wang, S. B., Boccagno, C., Haynos, A. F., Kleiman, E., & Hooley, J. M. (2019). Comparing self-harming intentions underlying eating disordered behaviors and NSSI: Evidence that distinctions are less clear than assumed. *International Journal of Eating Disorders*, *52*(5), 564–575. https://doi.org/10.1002/eat.23041

Foygel, R., & Drton, M. (2010). Extended Bayesian Information Criteria for Gaussian Graphical Models. In *Advances in neural information processing systems* (pp. 604–612).

Fuller, W. A. (1991). Simple Estimators for the Mean of Skewed Populations. *Statistica Sinica*, *1*(1), 137–158.

Godart, N. T., Perdereau, F., Rein, Z., Berthoz, S., Wallier, J., Jeammet, P., & Flament, M. F. (2007). Comorbidity studies of eating disorders and mood disorders. Critical review of the literature. In *Journal of Affective Disorders* (Vol. 97, Issues 1–3, pp. 37–49). Elsevier. https://doi.org/10.1016/j.jad.2006.06.023

Haynos, A. F., & Fruzzetti, A. E. (2015). Initial evaluation of a single-item screener to assess problematic dietary restriction. *Eating and Weight Disorders*, *20*(3), 405–413. https://doi.org/10.1007/s40519-014-0161-0

Haynos, A. F., Fruzzetti, A. E., Safer, D. L., & Chen, E. Y. (2011). Anorexia Nervosa as a Disorder of Emotion Dysregulation: Theory, Evidence, and Treatment Implications. *Clinical Psychology: Science and Practice*, *18*(3), 203–207. https://doi.org/10.1111/j.1468-

2850.2011.01251.x

Haynos, A. F., Wang, S. B., & Fruzzetti, A. E. (2018). Restrictive eating is associated with emotion regulation difficulties in a non-clinical sample. *Eating Disorders*, *26*(1), 5–12. https://doi.org/10.1080/10640266.2018.1418264

Haynos, A. F., Watts, A. W., Loth, K. A., Pearson, C. M., & Neumark-Stzainer, D. (2016). Factors Predicting an Escalation of Restrictive Eating During Adolescence. *Journal of Adolescent Health*, *59*(4), 391–396. https://doi.org/10.1016/j.jadohealth.2016.03.011

Hu, L. T., & Bentler, P. M. (1999). Cutoff criteria for fit indexes in covariance structure analysis: Conventional criteria versus new alternatives. *Structural Equation Modeling*, *6*(1), 1–55. https://doi.org/10.1080/10705519909540118

Hubert, M., & Van Der Veeken, S. (2008). Outlier detection for skewed data. *Journal of Chemometrics*, *22*(3–4), 235–246. https://doi.org/10.1002/cem.1123

Jones, P. (2020). *networktools: Tools for identifying important nodes in networks* (R package version 1.2.3). https://cran.r-project.org/package=networktools

King, J. A., Geisler, D., Ritschel, F., Boehm, I., Seidel, M., Roschinski, B., Soltwedel, L., Zwipp, J., Pfuhl, G., Marxen, M., Roessner, V., & Ehrlich, S. (2015). Global cortical thinning in acute anorexia nervosa normalizes following long-term weight restoration. *Biological Psychiatry*, *77*(7), 624–632. https://doi.org/10.1016/j.biopsych.2014.09.005

Korn, J., Dietel, F. A., & Hartmann, A. S. (2020). Testing the specificity of interpretation biases in women with eating disorder symptoms: An online experimental assessment. *International Journal of Eating Disorders*, *53*(3), 372–382.

https://doi.org/10.1002/eat.23201

Kroenke, K., Spitzer, R. L., & Williams, J. B. (2001). The PHQ-9: validity of a brief depression severity measure. *Journal of General Internal Medicine*, *16*(9), 606–613.

Laghai, A., & Joseph, S. (2000). Attitudes towards emotional expression: Factor structure, convergent validity and associations with personality. *British Journal of Medical Psychology*, *73*(3), 381–384. https://doi.org/10.1348/000711200160598

Lavender, J. M., Wonderlich, S. A., Engel, S. G., Gordon, K. H., Kaye, W. H., & Mitchell, J. E. (2015). Dimensions of emotion dysregulation in anorexia nervosa and bulimia nervosa: A conceptual review of the empirical literature. *Clinical Psychology Review*, *40*, 111–122. https://doi.org/10.1016/j.cpr.2015.05.010

Leppanen, J., Sedgewick, F., Treasure, J., & Tchanturia, K. (2018). Differences in the Theory of Mind profiles of patients with anorexia nervosa and individuals on the autism spectrum: A meta-analytic review. In *Neuroscience and Biobehavioral Reviews* (Vol. 90, pp. 146–163). https://doi.org/10.1016/j.neubiorev.2018.04.009

Loscalzo, Y., & Giannini, M. (2017). Social Anxiety Disorder and Interpretation Bias in Adolescents. *Adolescent Psychiatry*, *7*(14), 190–203.

Marchetti, I. (2020). Hopelessness: A network analysis. *Cognitive Therapy and Research*, *43*(3), 611–619.

McDonald, R. (1999). *Test Theory: A Unified Treatment*. L. Erlbaum Associates.

Meier, M., Kossakowski, J. J., Jones, P. J., Kay, B., Riemann, B. C., & McNally, R. J. (2019). Obsessive–compulsive symptoms in eating disorders: A network investigation.

International Journal of Eating Disorders, *53*(3), 362–371. https://doi.org/10.1002/eat.23196

Metalsky, G. I., Joiner, T. E., Potthoff, J., Pacha, L., Alfano, M. S., & Hardin, T. (1991). The depressive interpersonal relationships inventory. *Unpublished Manuscript*.

Miles, S., Gnatt, I., Phillipou, A., & Nedeljkovic, M. (2020). Cognitive flexibility in acute anorexia nervosa and after recovery: A systematic review. *Clinical Psychology Review*, *81*(August), 101905. https://doi.org/10.1016/j.cpr.2020.101905

Puccio, F., Fuller-Tyszkiewicz, M., Youssef, G., Mitchell, S., Byrne, M., Allen, N., & Krug, I. (2017). Longitudinal Bi-directional Effects of Disordered Eating, Depression and Anxiety. *European Eating Disorders Review*, *25*(5), 351–358. https://doi.org/10.1002/erv.2525

Richson, B. N., Forbush, K. T., Chapa, D. A. N., Gould, S. R., Perko, V. L., Johnson, S. N., Christensen, K. A., Swanson, T. J., & Tregarthen, J. (2021). Measurement invariance of the Eating Pathology Symptoms Inventory (EPSI) in adolescents and adults. *Eating Behaviors*, *42*(June), 101538. https://doi.org/10.1016/j.eatbeh.2021.101538

Selby, E. A., & Coniglio, K. A. (2020). Positive Emotion and Motivational Dynamics in Anorexia Nervosa: A Positive Emotion Amplification Model (PEAMP). *Psychological Review*. https://doi.org/10.1037/rev0000198

Shen, X., Ma, S., Vemuri, P., Simon, G., Weiner, M. W., Aisen, P., Petersen, R., Jack, C. R., Saykin, A. J., Jagust, W., Trojanowki, J. Q., Toga, A. W., Beckett, L., Green, R. C., Morris, J., Shaw, L. M., Khachaturian, Z., Sorensen, G., Carrillo, M., … Fargher, K. (2020). Challenges and Opportunities with Causal Discovery Algorithms: Application to Alzheimer's Pathophysiology. *Scientific Reports*, *10*(1), 1–12.

https://doi.org/10.1038/s41598-020-59669-x

Swinbourne, J. M., & Touyz, S. W. (2007). The co-morbidity of eating disorders and anxiety disorders: A review. *European Eating Disorders Review*, *15*(4), 253–274. https://doi.org/10.1002/erv.784

Thompson, R. A., & Virmani, E. A. (2012). Socioemotional Development. In V.S. Ramachandran (Ed.), *Encyclopedia of Human Behavior* (2nd ed., pp. 504–511). Academic Press.

Tibshirani, R. (2016). Regression Shrinkage and Selection via the Lasso. *Journal of the Royal Statistical Society. Series B (Methodological)*, *58*(1), 267–288.

Treasure, J., & Schmidt, U. (2013). The cognitive-interpersonal maintenance model of anorexia nervosa revisited: A summary of the evidence for cognitive, socio-emotional and interpersonal predisposing and perpetuating factors. *Journal of Eating Disorders*, *1*(1), 1–10. https://doi.org/10.1186/2050-2974-1-13

Treasure, J., Willmott, D., Ambwani, S., Cardi, V., Clark Bryan, D., Rowlands, K., & Schmidt, U. (2020). Cognitive Interpersonal Model for Anorexia Nervosa Revisited: The Perpetuating Factors that Contribute to the Development of the Severe and Enduring Illness. *Journal of Clinical Medicine*, *9*(3), 630. https://doi.org/10.3390/jcm9030630

Turton, R., Cardi, V., Treasure, J., & Hirsch, C. R. (2018). Modifying a negative interpretation bias for ambiguous social scenarios that depict the risk of rejection in women with anorexia nervosa. *Journal of Affective Disorders*, *227*(November 2017), 705–712. https://doi.org/10.1016/j.jad.2017.11.089

Walsh, B. T. (2013). The enigmatic persistence of anorexia nervosa. *American Journal of Psychiatry, 170*(5), 477–484. https://doi.org/10.1176/appi.ajp.2012.12081074

Wang, S. B., Pisetsky, E. M., Skutch, J. M., Fruzzetti, A. E., & Haynos, A. F. (2018). Restrictive eating and nonsuicidal self-injury in a nonclinical sample: Co-occurrence and associations with emotion dysregulation and interpersonal problems. *Comprehensive Psychiatry, 82*, 128–132. https://doi.org/10.1016/j.comppsych.2018.02.005

Weiss, N. H., Gratz, K. L., & Lavender, J. M. (2015). Factor Structure and Initial Validation of a Multidimensional Measure of Difficulties in the Regulation of Positive Emotions: The DERS-Positive. *Behavior Modification, 39*(3), 431–453. https://doi.org/10.1177/0145445514566504

Table 1.

Logistic Regression Model: Biased and inflexible Interpretations' Association with Restrictive Eating

Criterion	**Predictor**	***β***	***SE***	***z***	***p***	***95%CI***
Restriction (Y/N)	Negative Interpretation Inflexibility	0.11	0.13	0.87	.383	[-0.14 0.37]
	Negative Interpretation Bias	0.02	0.14	0.13	.899	[-0.25 0.29]
	Positive Interpretation Bias	0.09	0.13	0.70	.482	[-0.16 0.35]
	Depression	**-0.61**	**0.18**	**3.37**	**<.001**	**[-0.98 -0.27]**
	Social Phobia	-0.05	0.22	0.21	.836	[-0.49 0.39]
	Social Interaction Anxiety	-0.38	0.22	1.73	.083	[-0.82 0.04]
	BMI	-0.14	0.13	1.05	293	[-0.40 0.12]
	Age	0.11	0.14	0.77	.443	[-0.16 0.38]
	Sex	**-0.34**	**0.13**	**2.63**	**.009**	**[-0.60 -0.09]**

Note. Sex is dummy coded as the effect of being female (male=0). Restriction is coded as 1=No (Yes=0). **BOLD**=significant. Model intercept=0.17 (not significant).

Table 2.

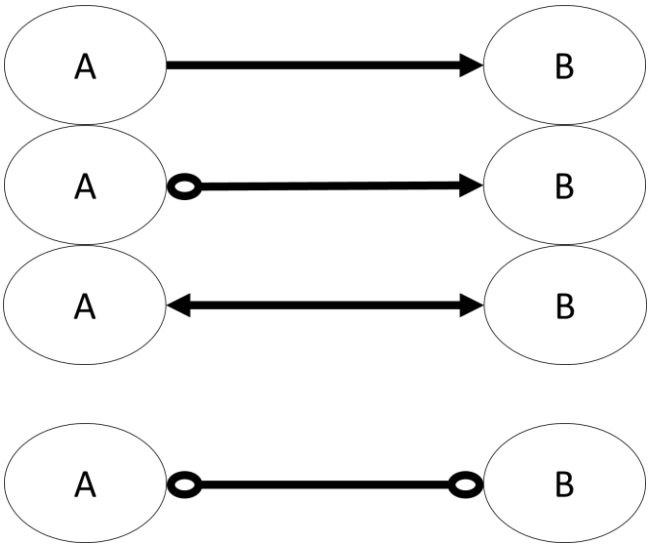
Multiple Linear Regression Model: Biased and Inflexible Interpretations' Association with Eating Disorder Symptoms

Criterion	**Predictor**	***β***	***SE***	***t***	***p***	***95%CI***
EPSI Scores	Negative Interpretation Inflexibility	0.04	0.05	<1	.446	[-0.06 0.13]
	Negative Interpretation Bias	0.07	0.05	1.40	.162	[-0.02 0.17]
	Positive Interpretation Bias	-0.01	0.05	<1	.804	[-0.11 0.08]
	Depression	**0.28**	**0.05**	**4.623**	**<.001**	**[0.16 0.41]**
	Social Phobia	0.08	0.08	<1	.346	[-0.08 0.24]
	Social Interaction Anxiety	**0.22**	**0.08**	**2.81**	**.005**	**[0.07 0.37]**
	BMI	0.05	0.05	1.09	.276	[-0.04 0.15]
	Age	0.08	0.05	1.48	.139	[-0.02 0.18]
	Sex	**0.13**	**0.05**	**2.56**	**.011**	**[0.03 0.22]**

Note. Sex is dummy coded as the effect of being female (male=0). **BOLD**=significant. Model statistics: $F(9,300)=15.60$, $p<.001$, Adjusted $R^2=.30$. Model intercept (00.00) was not significant ($p<.001$). All variables were z-scored prior to entry into the model.

Table 3.

Edge Types in a Partial Ancestral Graph Convey Information about Potential Causal Relations

Edge Type	Information Conveyed
	A is a direct or indirect cause of B. A and B are potentially confounded. B is not a cause of A. Either A is a cause of B or there is an unmeasured confounder of A and B, or both. B is not a cause of A. There is an unmeasured confounder (L) of A and B. There may be measured variables along the causal pathway from L to A or B. Exactly one of the following holds: 1. A is a cause of B 2. B is a cause of A 3. There is an unmeasured confounder of A and B 4. Both a and c 5. Both b and c

Note. In addition to the above, if an edge is **bold** (thickened), then the relation is definitely direct. Else, it is possibly indirect. If an edge is green, there is no latent confounder of the relation; if it is blue, there may be a latent confounder.

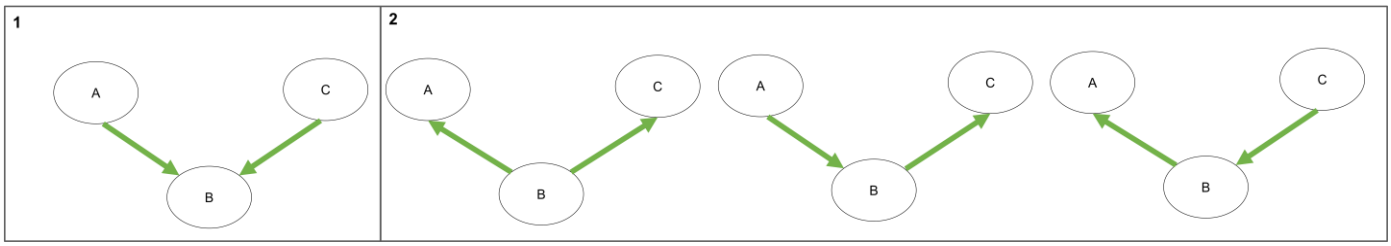


Figure 1. **Patterns of conditional relations convey information about causal orientations.** The absence of an arrow denotes the absence of a causal relation. Green arrows denote causal relations between variables (see **Table 3**). **Panel 1:** A "collider" graph (A and C directly cause B, no edge between A and C). A is unconditionally independent of C, and A is dependent on C conditional on B. **Panel 2:** However, in all other possible relations between A, B, and C (where no edge is present between A and C), a different pattern of conditional relations emerges: A is unconditionally dependent on C, and A is independent of C conditional on B. Given the differential pattern of conditional relations between the graphs in **Panel 1** and **Panel 2**, examining conditional relations can support inference about whether a collider or some other causal process generated the observed data. Greedy Fast Causal Inference uses cases like that illustrated above to determine the direction of causal edges and to rule in/out latent confounds of the relations between variables.

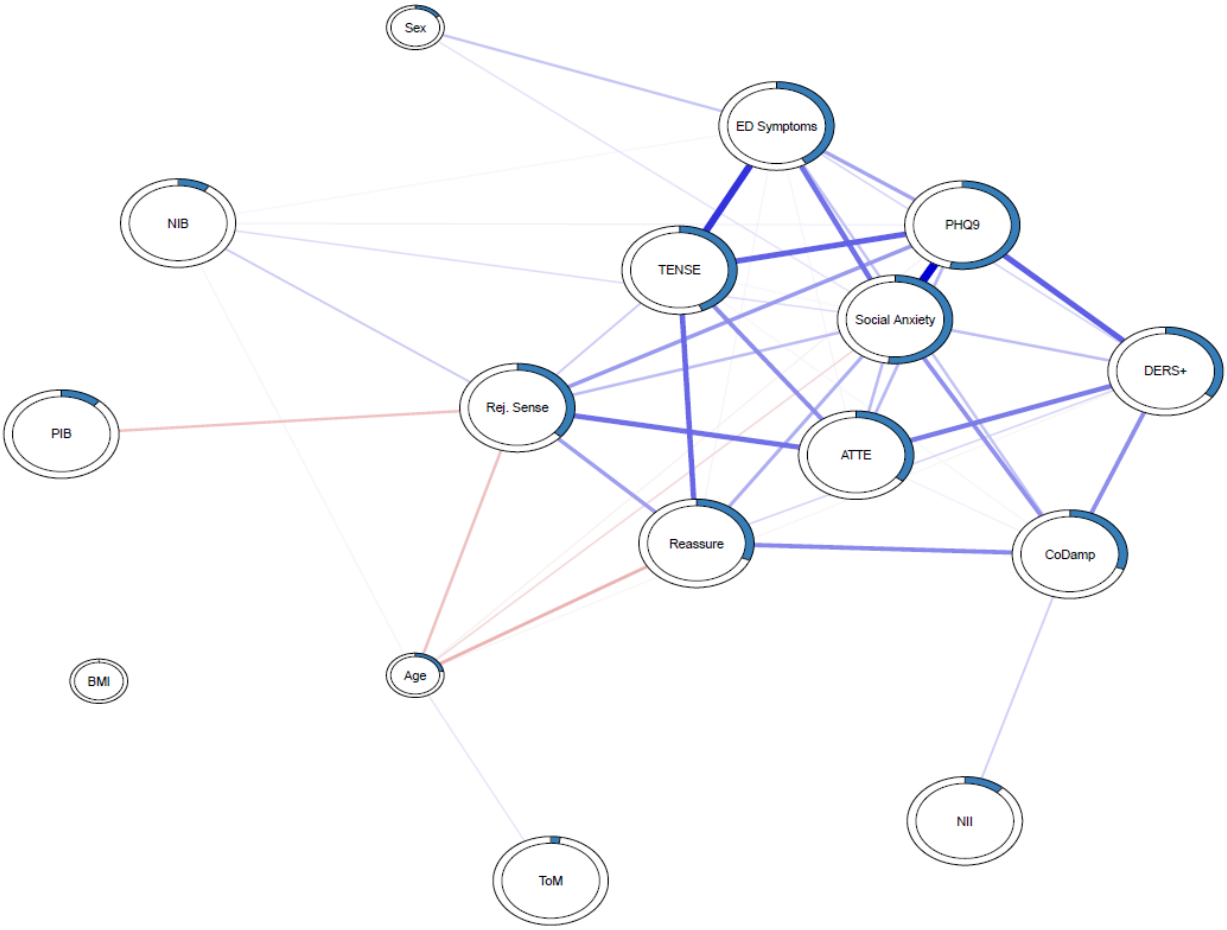


Figure 2. Regularized partial correlation network. Annulus surrounding each node denotes predictability (more filled=more predictable). Red=negative association. Blue=positive association. Smaller nodes are covariates. Sex is coded as the effect of being female. ED=eating disorder. PHQ9=depression, DERS+=difficulty regulating positive emotion. ATTE=attitudes to emotional expression. Rej. Sense=rejection sensitivity. Reassure=excessive reassurance seeking. TENSE=negative social exchange. CoDamp=co-dampening of positive emotion. ToM=theory of mind. NII=negative interpretation inflexibility. PIB=positive interpretation bias. NIB=negative interpretation bias.

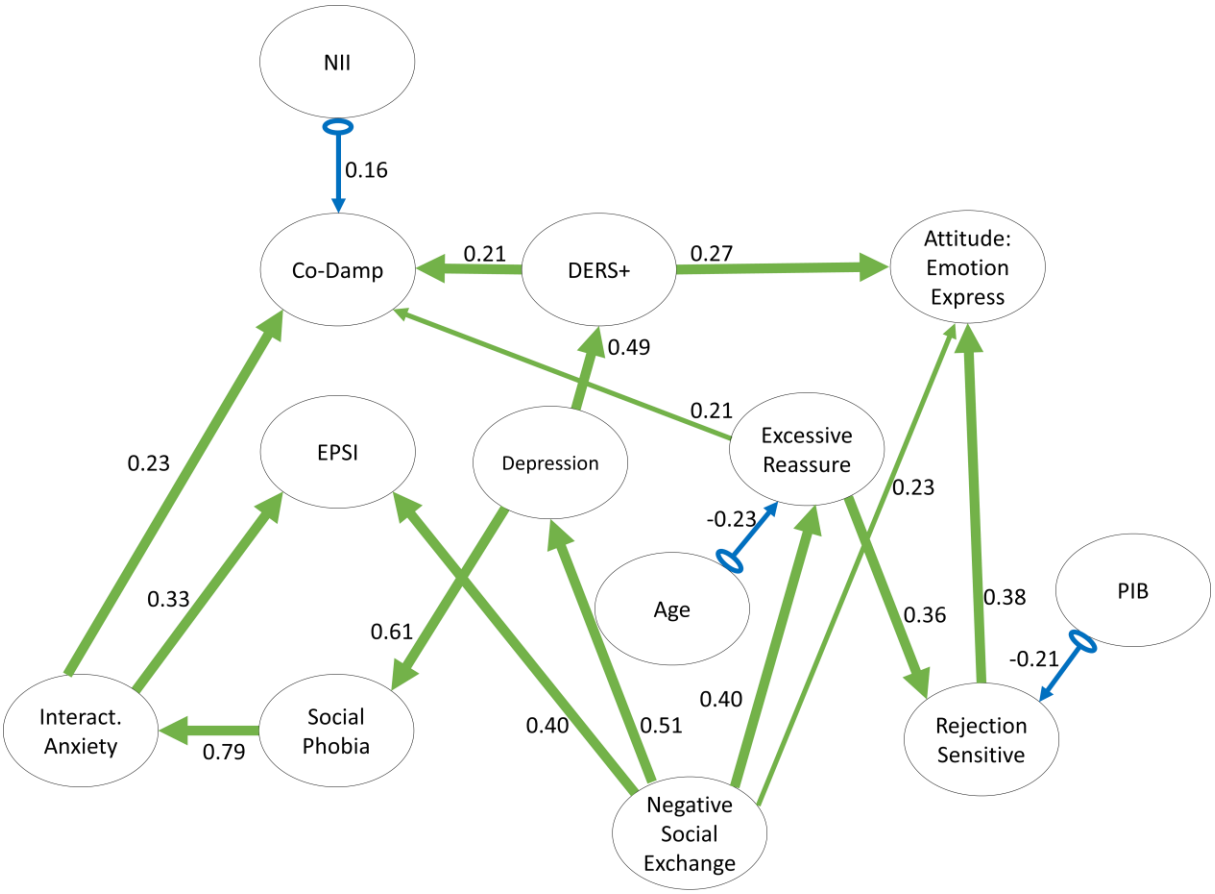


Figure 3. Directed Acyclic Graph suggested by the Greedy Fast Causal Inference (GFCI) causal discovery algorithm. See **Table 3** for a description of possible edge types. Variables are not depicted if GFCI could not determine a potential causal relation between them and another variable included in the analysis. Numbers adjacent to edges are standardized parameter estimates from a structural equation model of the causal structure suggested by GFCI. NII=Negative Interpretation Inflexibility. NIB=negative interpretation bias, PIB=positive interpretation bias, DERS+=difficulty regulating positive emotions. EPSI=Eating Pathology Symptoms Inventory.

Supplementary Material for:

Biased and Inflexible Interpretations of Ambiguous Social Situations: Associations with Restrictive Eating Behavior and Socioemotional Functioning

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Table of Contents

Section S1. Demographic Information

Table S1.
Demographic Information

Demographic	**Screener**	**Main Study**
N	1308	310
Age [*M* (*SD*)]	44.72 (13.47)	45.90 (13.42)
Gender Identity		
Male	517	127
Female	784	183
Non-binary	7	0
Sex Assigned at Birth		
Male	517	127
Female	791	183
Intersex	0	0
Race		
White/Caucasian	1062	254
Black/African American	108	24
Asian	81	21
Pacific Islander	2	0
Middle Eastern/North African	2	0
Native American/Alaska Native	2	0
Other	7	1
Hispanic	44	10
Years of Education [*M* (*SD*)]	15.54 (2.34)	15.47 (2.26)
Marital Status		
Single, Never Married	452	106
Married/Domestic Partnership	658	149
Widowed/Widower	38	10
Divorced	153	44
Separated	7	1
Restrictive Eating	Yes: 564 | No: 744	Yes: 140 | No: 170
BMI [*M* (*SD*)]	27.37 (7.31)	26.48 (6.01)
Diagnosed with an ED	--	Yes: 11 | No: 299
Hospitalized due to ED	--	Yes: 4 | No: 306

Note. Individuals could opt not to answer demographic questions, and were able to select more than one option for race/ethnicity. For the main study, demographic information refers to participants included in the final sample for analysis.

Differential Drop-out

Just over half of the individuals (66%) who were invited to participate in the main study did so. To examine the possibility of differential drop-out, individuals who were invited to participate in the main study and accepted that invitation were compared to individuals who were invited but declined on

variables recorded during the screening survey. People who accepted (vs. declined) did not differ in terms of age ($t(399.04) = 1.72$, $p = .086$), years of education ($t(398.01) = 1.27$, $p = .204$), BMI ($t(356.60) < 1$, $p = .451$), gender ($\chi^2(2) = 1.71$, $p = .426$), or continuous eating disorder symptoms ($t(393.84) = 1.08$, $p = .279$). However, people who accepted had a pattern of sex and endorsement of restrictive eating that differed from chance ($\chi^2(1) = 27.93$, $p < .001$, and $\chi^2(1) = 29.32$, $p < .001$, respectively): individuals who accepted were more likely than expected to deny restrictive eating (though the difference from expected values was very slight, at 3.50 individuals), and were more likely to be male (though again, the difference from expected values was small, at 7.66 individuals). Thus, our two-stage recruitment method may have systematically selected for men and people who deny restrictive eating, making the results of this study slightly less generalizable to women and people who endorse restrictive eating. However, these threats to generalizability appear incredibly minor, with significant test values likely driven by large sample size rather than large deviations from what would be expected by chance.

Section S2. Open Science Practices

Open Science Practices. The hypotheses and analysis plan for this study was pre-registered at:

https://osf.io/trf5x. Anonymized data are available at: https://osf.io/x9fjn/.

Section S3. Example Emotional BADE Task Scenario and Scoring

To aid readers in understanding the emotional BADE task, an example of a *disconfirming-the-negative* scenario is provided in the figure below. The statement numbers and explanation types are labelled in the figure. Lure explanations appear most plausible at first, and become less so as the scenario continues. Absurd explanations remain implausible throughout the scenarios. True explanations are moderately plausible at the start of the scenario, but become most plausible by its end. The -A and -B labels for the Lure explanations are arbitrary.

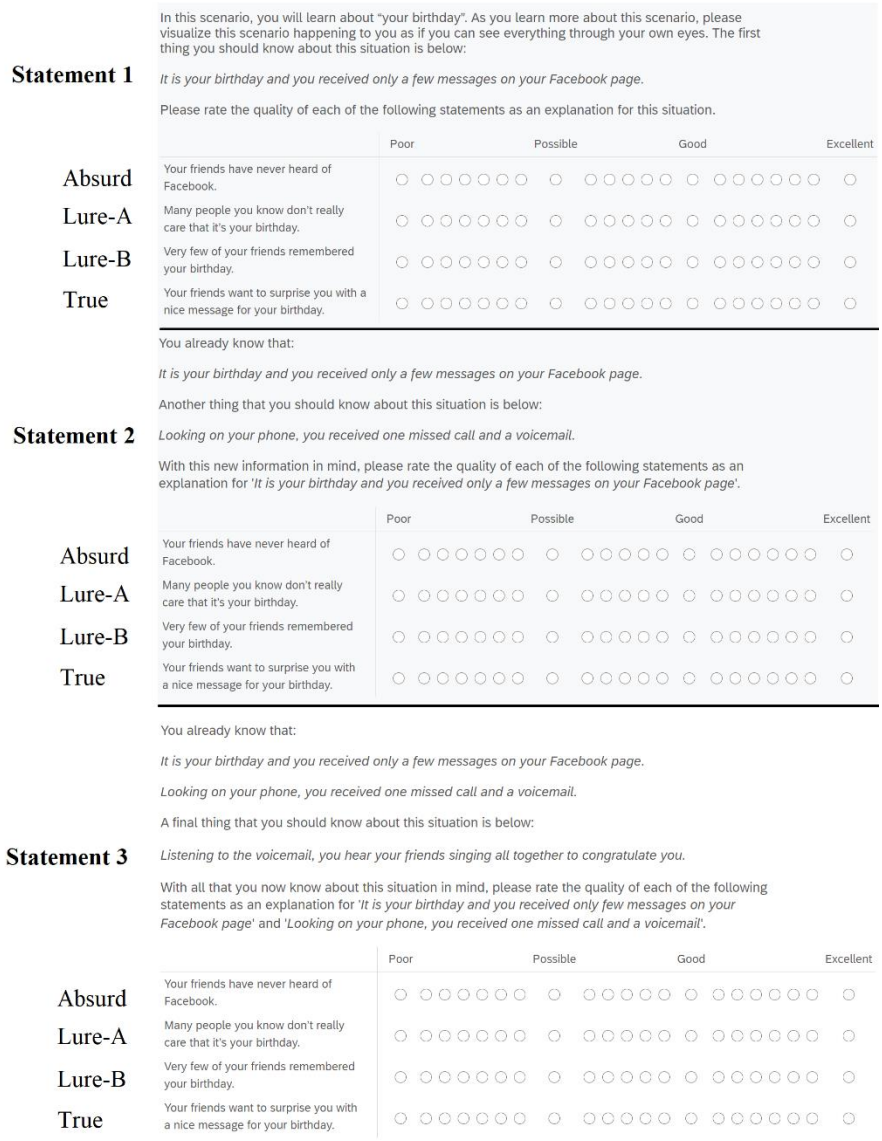


Figure S1. Example of an Emotional BADE Task scenario.

As in previous research (Everaert et al., 2021), task metrics were scored as follows: negative interpretation inflexibility (Absurd1+Absurd2+Absurd3+LureA3+LureB3-True3), positive interpretation bias (True1+True2+True3), and negative interpretation bias (LureA1+LureB1+LureA2+LureB2). In these formulas, numbers in variable names represent the statement after the rating was made, while the remainder of the name denotes the category of explanation being rated.

Section S4. Results Regarding Disconfirming the Positive Scenarios

Table S2.
Zero-order correlations

	PII	PIB	NIB
Eating Pathology Symptoms Inventory	.02	.08	.11
Mind in the Eyes	-.05	-.02	-.04
Reassurance Seeking	.11	. 09	.00
Social Phobia	-.06	-.06	.09
Social Interaction Anxiety	.04	.01	.03
Attitude to Emotional Expression	-.04	-.16*	.05
Difficulty Regulating Positive Emotion	.04	.03	.06
Depression (PHQ9)	-.07	-.06	.15*
Negative Social Exchange	-.03	.01	.18*
Dampening of Positive Emotions	.11^{+}	.12^{+}	.04
Rejection Sensitivity	-.06	-.25*	.16*

Note. All correlations are non-parametric (Spearman's rho), df= 308. $^{+}$=p<.05, *=p<.01. PII = Positive Interpretation Inflexibility. PIB = Positive Interpretation Bias. NIB = Negative Interpretation Bias.

Hypothesis 1: Inflexible/Biased Interpretations are Associated with Eating Disorder Symptoms

A multiple regression model with (continuous) eating disorder symptoms as the criterion variable and inflexible/biased interpretations, social phobia/interaction anxiety, and BMI, age, and sex as

predictors was constructed to test the hypothesis that continuous variation in eating disorder symptoms would be associated with inflexible and biased interpretations (*Hypothesis 1*). The overall model was significant $F(9,300)=16.70$, $p<.001$, and accounted for 31% of the variance in eating pathology (adjusted $R^2=.31$). Partial support for *Hypothesis 1* was found: A more positive interpretation bias predicted eating disorder symptoms above and beyond these other variables, $\beta=0.01$, $t(300)=2.49$, $p=.013$, *95%CI*=[0.00 0.03]. However, inflexible positive interpretations and negative interpretation bias were unrelated to these symptoms. Results were qualitatively identical when points flagged by regression diagnostics (dfbetas, cook's distance, studentized residuals, covariance rations) were removed. All *VIF*s were <2.97. For full model results, see **SI Table S3**.

Table S3.

Multiple Linear Regression Model: Biased/Inflexible Interpretations' Association with Eating Disorder Symptoms

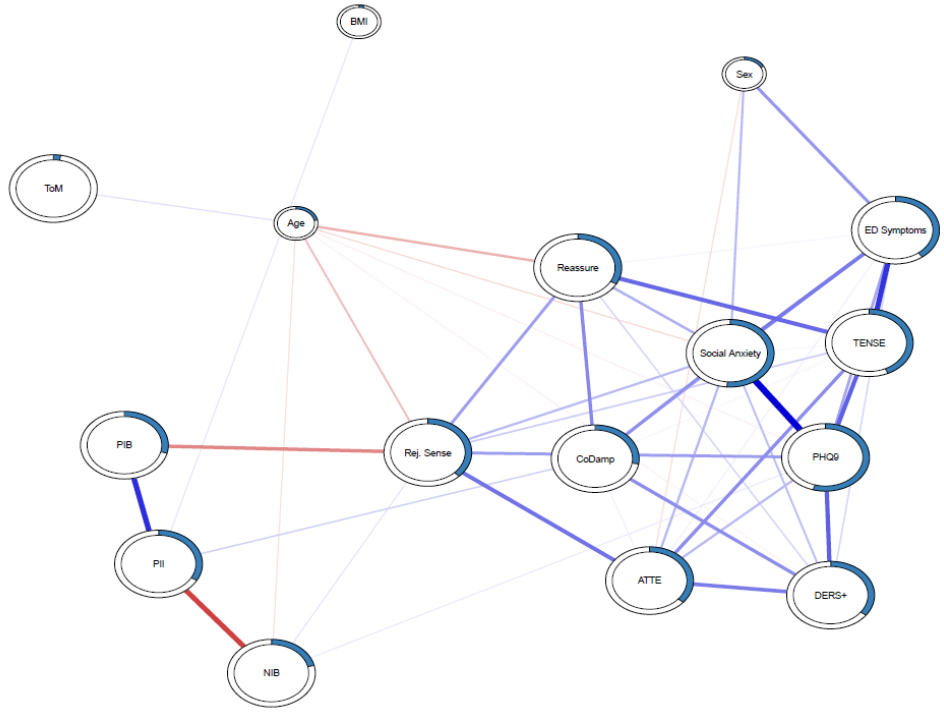
Criterion	**Predictor**	***β***	***SE***	***t***	***p***	***95%CI***
EPSI Scores	Positive Interpretation Inflexibility	0.00	0.01	<1	.627	[-0.08 0.01]
	Negative Interpretation Bias	0.01	0.01	<1	.764	[-0.01 0.02]
	Positive Interpretation Bias	**0.01**	**0.01**	**2.49**	**.014**	**[0.00 0.03]**
	Depression	**0.31**	**0.06**	**5.04**	**<.001**	**[0.19 0-.43]**
	Social Phobia	0.10	0.08	1.20	.231	[-0.06 0.26]
	Social Interaction Anxiety	**0.21**	**0.08**	**2.73**	**.007**	**[0.06 0. 36]**
	BMI	0.05	0.05	<1	.320	[-0.05 0.14]
	Age	0.07	0.05	1.32	.187	[-0.03 0.17]
	Sex	**0.12**	**0.05**	**2.43**	**.015**	**[0.02 0.22]**

Note. Sex is coded as the effect of being female (female=1,male=0). **BOLD** = significant. Model statistics: $F(9,300)=16.73$, $p<.001$, Adjusted $R^2=.31$.

Hypothesis 2: Inflexible/Biased Interpretations Relate to Eating Disorder Symptoms via Socioemotional Functioning

Network analysis including BADE metrics from disconfirming-the-positive scenarios yielded results (**SI Figure S2**) extremely similar to those for disconfirming-the-negative scenarios. Like negative interpretation inflexibility, positive interpretation inflexibility was indirectly associated with eating disorder symptoms via co-dampening of positive emotions. Less positive interpretation bias was again

associated with rejection sensitivity. Negative interpretation bias also continued to be associated with rejection sensitivity, but was no longer directly associated with eating disorder symptoms.



SI Figure S2. Regularized partial correlation network. Annulus surrounding each node denotes predictability (more filled=more predictable). Red=negative association. Blue=positive association. Smaller nodes are covariates. Sex is coded as the effect of being female. ED = eating disorder. PHQ9=depression, DERS+=difficulty regulating positive emotion. ATTE=attitudes to emotional expression. Rej. Sense = rejection sensitivity. Reassure=excessive reassurance seeking. TENSE=negative social exchange. CoDamp=co-dampening of positive emotion. ToM = theory of mind. PII=positive interpretation inflexibility. PIB=positive interpretation bias. NIB=negative interpretation bias.

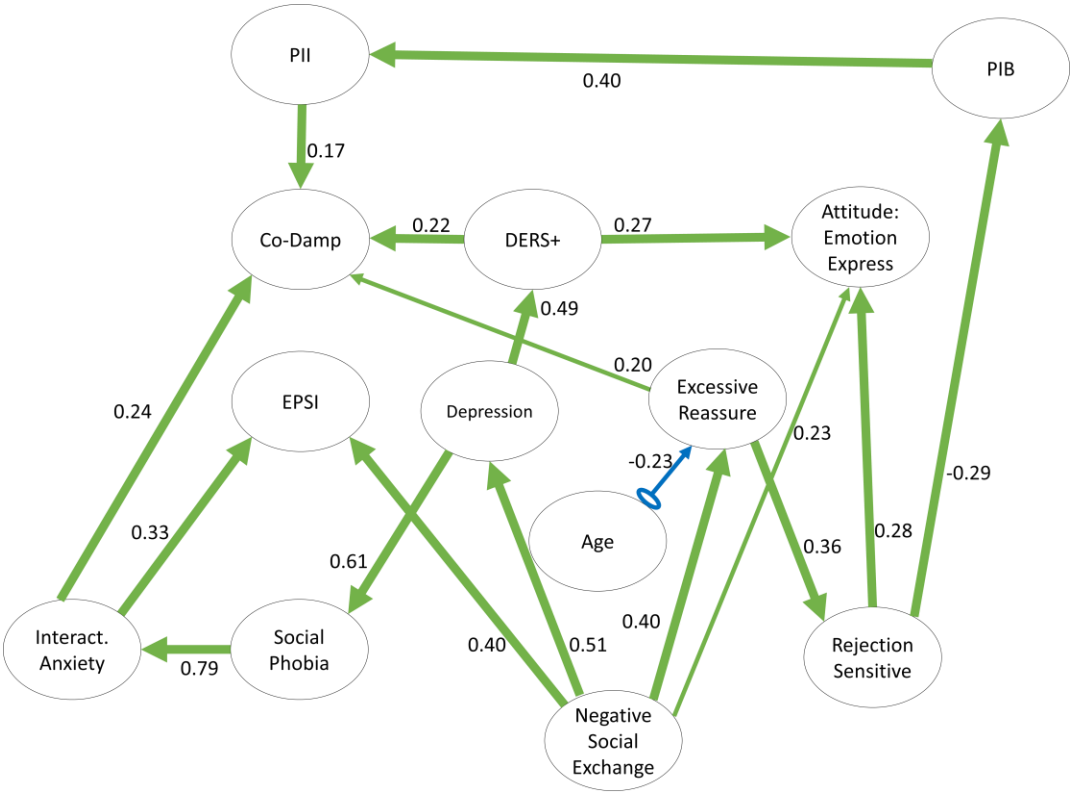
Exploratory Causal Discovery Analyses

The results of the causal discovery analysis involving metrics generated from disconfirming-the-positive scenarios were generally similar to those obtained for disconfirming-the-negative scenarios (the primary difference was with respect to the role of positive interpretation bias in the graph). The consensus PAG generated via causal discovery analysis is depicted in **SI Figure S3**. A structural equation model

featuring the relations suggested by this PAG was an adequate fit to the data, RMSEA=.09, CFI=.89, SRMR=.12, and was therefore used to indicate the sizes of potential causal effects.

The PAG generated by GFCI identified negative social exchange and social interaction anxiety as potential direct causes of eating disorder symptoms. Depression and social phobia were related to symptoms indirectly, via potential causal effects on social interaction anxiety. Other markers of socioemotional functioning, including rejection sensitivity, attitudes to emotional expression, and difficulty regulating positive emotions were not involved in causal pathways leading to eating disorder symptoms.

GFCI also suggested that negative and positive interpretation bias were not causal ancestors of eating disorder symptoms. While a less positive interpretation bias was identified as a consequence of rejection sensitivity, this construct was not a causal ancestor of eating pathology. Similarly, the consensus PAG suggested that inflexible positive interpretations were not related to eating disorder symptoms via a causal pathway involving socioemotional functioning. Instead, inflexible interpretations were a cause of co-dampening, which was not a causal ancestor of eating disorder symptoms.



SI Figure S3. Directed Acyclic Graph suggested by the Greedy Fast Causal Inference (GFCI) causal discovery algorithm. See **Table 1 (main text)** for a description of possible edge types. Variables are not depicted if GFCI could not determine a potential causal relation between them and another variable included in the analysis. Numbers adjacent to edges are standardized parameter estimates from a structural equation model of the causal structure suggested by GFCI. PII=Positive Interpretation Inflexibility. NIB=negative interpretation bias, PIB=positive interpretation bias, DERS+=difficulty regulating positive emotions. EPSI=Eating Pathology Symptoms Inventory.

Hypothesis 4: Restrictive Eating is Associated with Inflexible/Biased Interpretations

A logistic regression model (**SI Table S4**) with endorsement vs. denial of restrictive eating as the criterion variable and inflexible/biased interpretations, social phobia/interaction anxiety, and BMI, age, and sex as predictors was constructed to test the hypothesis that restrictive eating is associated with lower scores across multiple measures of socioemotional functioning (*Hypothesis 4*). Contrary to this hypothesis, inflexible/biased interpretations did not predict restrictive eating. However, being female was associated with a 28% increase in the conditional odds of endorsing restrictive eating. The coefficients

also suggested that a one standard deviation increase (decrease) in depression above (below) its mean would portend a 12% increase (9% decrease) in the conditional odds of endorsing restrictive eating.

Table S4.

Logistic Regression Model: Biased/Inflexible Interpretations' Association with Restrictive Eating

Criterion	**Predictor**	***β***	***SE***	***z***	***p***	***95%CI***
Restriction (Y/N)	Positive Interpretation Inflexibility	0.00	0.01	0.18	.859	[-0.02 0.02]
	Negative Interpretation Bias	-0.02	0.02	1.05	.296	[-0.06 0.02]
	Positive Interpretation Bias	-0.01	0.02	0.56	.575	[-0.04 0.02]
	Depression	**-0.59**	**0.18**	**3.28**	**.001**	**[-0.96 -0.25]**
	Social Phobia	-0.06	0.22	0.29	.771	[-0.50 0.37]
	Social Interaction Anxiety	-0.37	0.22	1.69	.091	[-0.81 0.05]
	BMI	-0.11	0.13	0.86	.392	[-0.37 0.14]
	Age	0.09	0.14	0.69	.491	[-0.18 0.37]
	Sex	**-0.33**	**0.13**	**2.49**	**.013**	**[-0.59 -0.07]**

Note. Sex is coded as the effect of being female (dummy coded; male=0). Restriction is coded as 1=No (0=yes). **BOLD** = significant. Model intercept =1.22 (not significant).

Section S5. Internal consistency and descriptive statistics of study measures

Table S5.
Internal Consistency and Descriptive Statistics of Study Measures

Measure	*M* (*SD)*	Omega Total
Eating Pathology Symptoms Inventory	36.59 (21.70)	.94
Mind in the Eyes	84.23 (4.36)	.52
Reassurance Seeking	7.85 (5.36)	.95
Social Phobia	10.94 (4.62)	.88
Social Interaction Anxiety	8.87 (4.30)	.88
Attitude to Emotional Expression	50.07 (14.99)	.95
Difficulty Regulating Positive Emotion	15.85 (4.20)	.93
Depression (PHQ9)	13.99 (5.14)	.93
Negative Social Exchange	43.36 (39.81)	.97
Dampening of Positive Emotions	12.24 (3.54)	.88
Rejection Sensitivity	86.10 (44.47)	.87
Negative Interpretation Inflexibility (DNEG)	0.35 (10.52)	--
Negative Interpretation Bias (DNEG)	45.49 (12.07)	--
Positive Interpretation Bias (DNEG)	29.14 (5.84)	--
Positive Interpretation Inflexibility (DPOS)	9.90 (11.08)	--
Positive Interpretation Bias (DPOS)	33.52 (7.21)	--
Negative Interpretation Bias (DPOS)	38.36 (9.35)	--

Note. Descriptive statistics and internal consistencies for outlier filtered data. DNEG = disconfirming the negative scenarios. DPOS = disconfirming the positive scenarios.

Section S6. Note on Statistical Test Assumptions

Examination of Test Assumptions. MANOVA: The assumptions of MANOVA model were tested as follows: hetereogeneity of covariances – Box's M test, multivariate normality – Henze-Zirkler test, multicollinearity – correlations between criterion variables were not allowed to exceed .90, linear relation between dependent variables – visual inspection of scatterplots. Multivariate outliers were checked for using Mahalanobis distances. Multiple Regression: Assumptions were checked using R's *car* package version 3.0.11. Multicolinearity was examined by calculating variance inflation factors (VIFs). Influential values were checked for using Cook's distance, studentized residuals, DFBetas, and covariance ratios. For logistic regression, scatterplots were also inspected to ensure that the relations between predictors and the logit of the criterion were linear.

Two assumptions of MANCOVA were violated: covariance matrices were not homogeneous (Box's M: $\chi(28)=96.48$, $p<.001$) and multivariate normality was not achieved (Henze-Zirkler=2.62, $p<.001$). To address these issues, Pillai's trace (which is robust to violations of homogeneity, Tabachnick & Fidell, 2007), was employed as the test statistic, and permutational MANOVA was used to supplement the traditional MANCOVA. Ten multivariate outliers were identified using mahalanobis distances. Removing these outliers did not alter test conclusions.

In multiple regression models, results were qualitatively identical when points flagged by regression diagnostics were removed. All *VIF*s were <3.03.

Section S7. Network Stability, Accuracy, and Difference Tests

Method Information:

Node centrality and predictability were examined for exploratory purposes. Node predictability was computed using *R's mgm* package, version *1.2.12*. Predictability indexes how much variance in a given node is explained by the nodes that are connected to it in the network (Haslbeck and Fried, 2017), and can be understood as an upper bound on controllability (Fried et al., 2018).

Node centrality was measured using one-step expected influence (in networks with positive and negative edges, this measure of centrality may be preferable; see: Robinaugh, Millner, and Mcnally, 2016). One-step expected influence was calculated as the sum of the value of all edges extending from a given node, taking the mathematical sign of each edge into account. Expected influence was calculated using *networktools* (Jones, 2020).

Because it is possible that unequal variances of the nodes in the network affect their centrality estimate, thereby influencing the observed network structure (Terluin, De Boer, and De Vet, 2016), correlations between expected influence values and standard deviations (SDs) of the individual nodes were examined. The correlations between expected influences and means of individual nodes were also examined to better ensure that differences in severity did not explain node centrality or network structure.

R's *bootnet* package, version *1.4.3* (Epskamp, 2020) was used to conduct exploratory statistical tests of the differences between edge weights and node centrality measures. Data were bootstrapped 1000 times for this purpose. If the 95% confidence interval for the difference between two edge weights or node centrality metrics did not overlap with zero, the difference was considered statistically significant.

Network Accuracy and Stability

Network accuracy and stability were also estimated using *bootnet* and 1000 bootstrapped re-samples. The accuracy of edge weights was quantified using the 95% confidence intervals generated from this bootstrapping procedure. Stability of centrality measures was quantified using the correlation stability coefficient (CS-coefficient; Epskamp, Borsboom, and Fried, 2018), which denotes the proportion of cases that can be dropped such that the set of stability measures obtained using the full and reduced data-sets are correlated above a certain threshold (.70 in the present study) with 95% probability. Metrics were considered stable if the CS-coefficient exceeded .25 (Epskamp et al., 2018).

Results:

Network Inference. Significant differences were observed between nodes' expected influence (**SI Figure S6: bottom**). Social anxiety, depression, and negative social exchange were the nodes with the strongest expected influence. Each of these variables had a stronger expected influence than over half the

other network nodes. Eating disorder symptoms had the next strongest expected influence. Negative interpretation inflexibility, negative interpretation bias, and positive interpretation bias had a smaller expected influence than many other nodes in the network.

There was no correlation between the standard deviation of a node and its expected influence, rho(14)=-.14, p=.605, 95%CI=[-.74 .47]. There was also no correlation between node means and their expected influences, rho(14)=.20, p=.456, 95%CI=[-.46 .85]. Thus, the relative average levels of socioemotional functioning/eating disorder symptoms and differential node informativeness were not capable of explaining the centrality of nodes in the network.

Variance in nodes was generally well explained by their neighbors. The average predictability across nodes was .27, indicating that just over one quarter of variance in the network could be accounted for. However, 63% of the variance in the network remained unexplained and may be attributable to unmeasured variables. The three best explained nodes in the network were depression (predictability=.54), social anxiety (predictability=.52), and negative social exchange (predictability=.44). The predictability of eating disorder symptoms was .41.

Network Accuracy and Stability

Bootstrapped confidence intervals suggested that the precision of network edges was acceptable (**SI Figure S4**). Edges and one-step expected influences were sufficiently stable, with a correlation-stability coefficient (.44) exceeding the minimum cutoff (.25 [but not the preferred .50 cutoff]; Epskamp et al., 2018). Consequently, findings regarding these metrics could be interpreted.

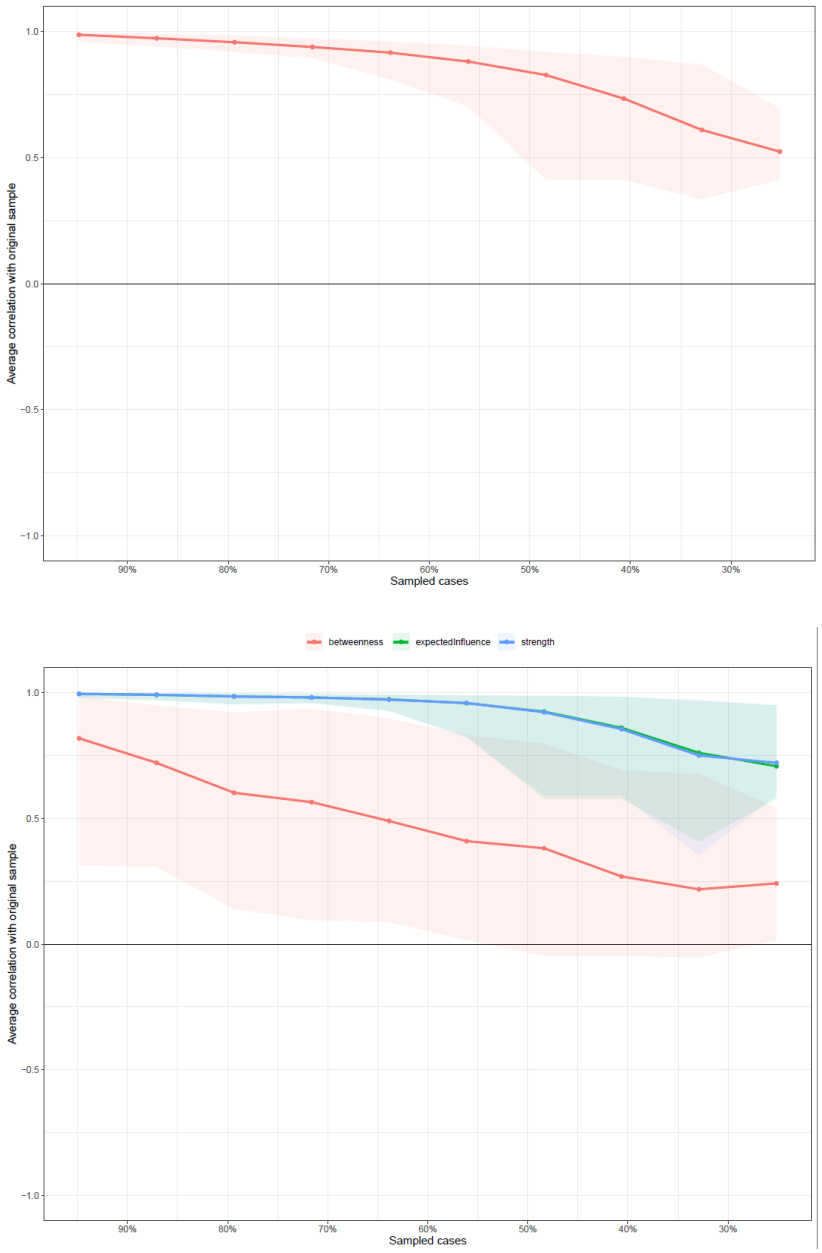


Figure S4. Stability of edge weights and centrality metrics, computed using a case-drop bootstrapping procedure. Clouds represent 95% CIs.

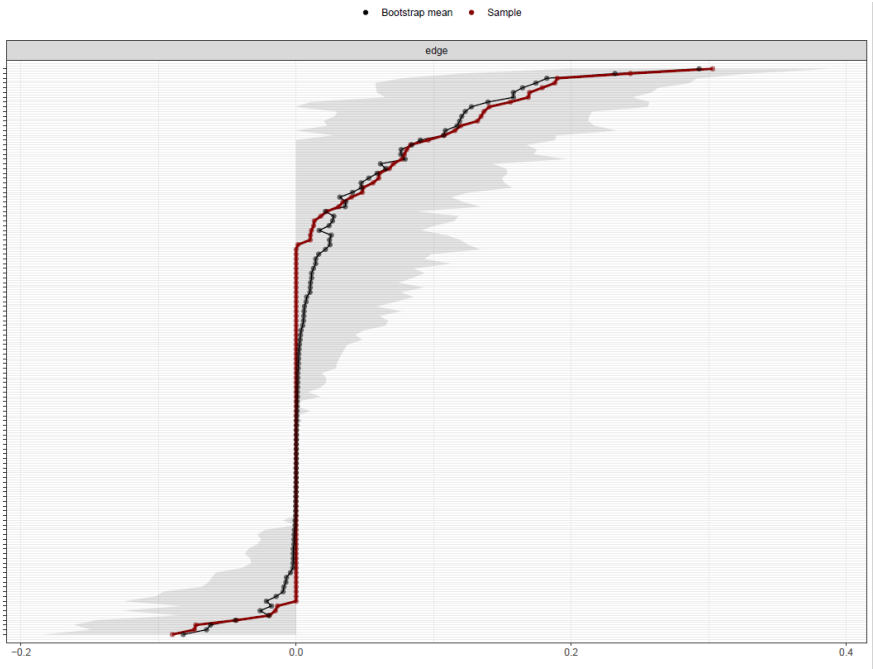


Figure S5. Bootstrapped confidence intervals for edge weights. Red = sample means, black = bootstrapped means. Grey = 95% CI. Edges are ordered by weight, with ties broken by bootstrapped sample means. Y-axis labels omitted as is customary.

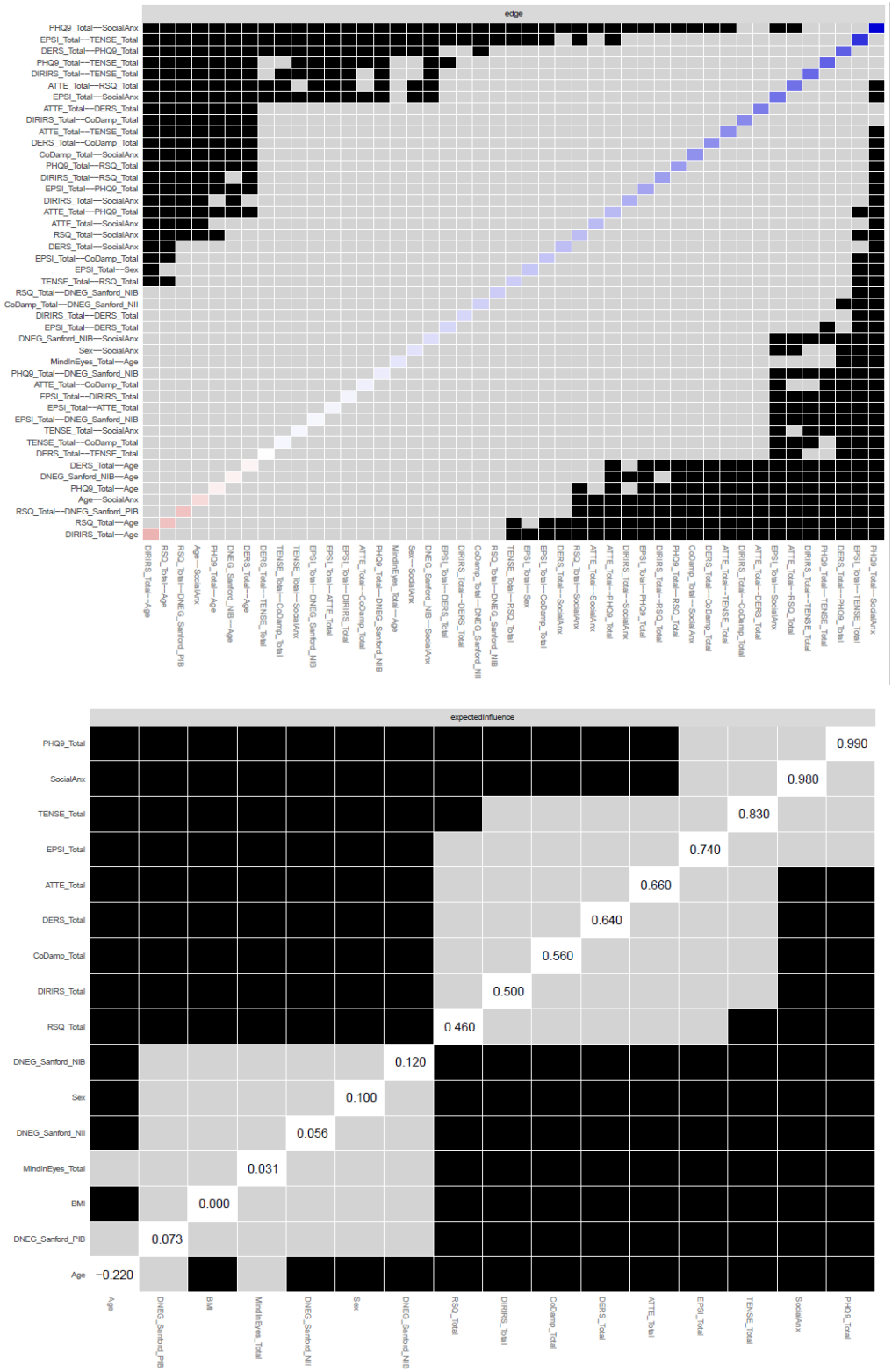


Figure S6. Bootstrapped difference tests (α = .05) between non-zero edge weights (top) or expected influences (bottom) in the estimated network. Black = parameters are significantly different for those

edges. Grey = no significant difference. Each number in a white box represents the value of the tested parameter for that node. Darker colors on the diagonal of the edge-weight plot represent more positive (blue) or negative (red) edge weights.

In order to explore whether the pattern of partial correlations in the estimated network differed by biological sex, the estimated networks for male and female participants were compared using R's NetworkComparisonTest package 2.2.1. In an omnibus test, the networks for participants of different sexes did not differ according to conventional significant thresholds, M = .30, p = .137. Both networks are visualized below for readers to inspect. The regularized graphs are relatively similar to one another, supporting the test's conclusion. Note that the present study is likely underpowered to detect sex differences; these results are preliminary and for exploratory purposes.

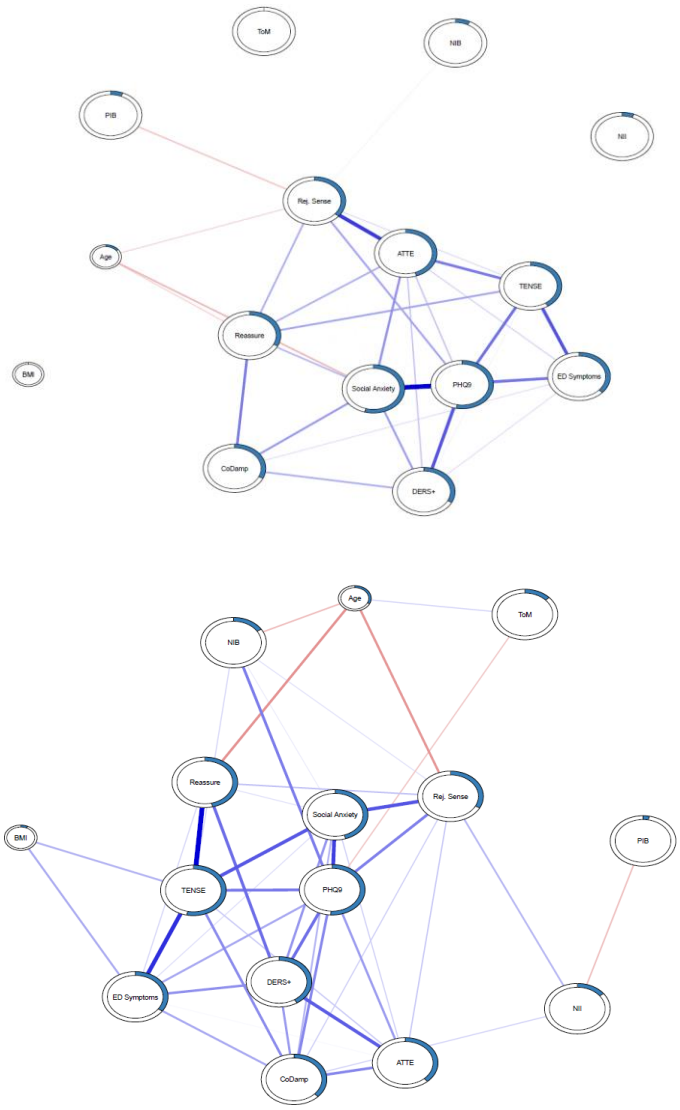


Figure S7. The regularized partial correlation networks for females (left) and males (right) are presented separately in the figure above, for exploratory purposes. Annulus surrounding each node denotes predictability (more filled = more predictable). Smaller nodes = covariates. ED = eating disorder. PHQ9=depression, DERS+=difficulty regulating positive emotion. ATTE=attitudes to emotional expression. Rej. Sense = rejection sensitivity. Reassure=excessive reassurance seeking. TENSE=negative social exchange. CoDamp=co-dampening of positive emotion. ToM = theory of mind. NII=negative interpretation inflexibility. PIB=positive interpretation bias. NIB=negative interpretation bias.

The network for individuals with EPSI scores at or above the sample median was visualized because of recommendations that one should examine the network in those higher in the variables of interest when conducting network analyses on skewed data (e.g., because item variability can influence network centrality; de Beurs, 2017). This network was relatively similar to that in the full sample. However, smaller edges, including those for inflexible/biased interpretations, were absent, likely due to the reduced power of greatly reduced sample size. For the same reason, the networks of individuals who endorsed (vs. denied) restrictive eating on the DRS were examined. These did not significantly differ, M=0.22, p=.450. Taken together, these results point toward similarity between the networks of people with higher vs. lower eating disorder symptom levels. However, future research should continue to examine the relation between inflexible/biased interpretations, socioemotional functioning, and eating disorder symptoms in groups with higher symptom burden.

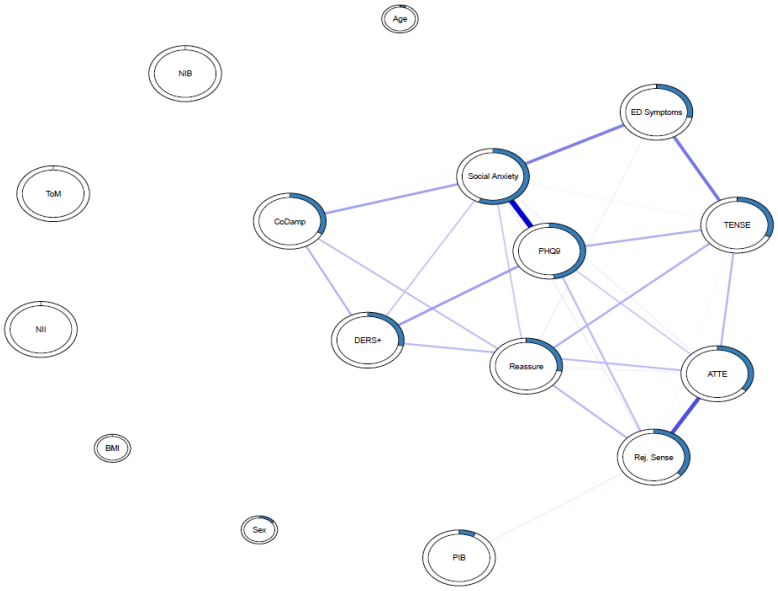


Figure S8. The regularized partial correlation network with only those scoring at or above the sample median on the EPSI is presented above. Annulus surrounding each node denotes predictability (more filled = more predictable). ED = eating disorder. PHQ9=depression, DERS+=difficulty regulating positive emotion. ATTE=attitudes to emotional expression. Rej. Sense = rejection sensitivity. Reassure=excessive reassurance seeking. TENSE=negative social exchange. CoDamp=co-dampening of positive emotion. ToM = theory of mind. NII=negative interpretation inflexibility. PIB=positive interpretation bias. NIB=negative interpretation bias.

Section S8. Additional Information on Causal Discovery Analyses

To better ensure graph stability, the GFCI algorithm was repeated on 10000 jackknifed re-samples of the study data. The original dataset was included as an additional re-sample. Results were aggregated into a single, consensus PAG by depicting the edge type (including: "no edge") and orientation most commonly present in the PAGs created from the jackknifed re-samples. The full FCI rule set was employed. Default values for remaining parameters were used. For example, the penalty discount (c) used for generating the initial likelihood scores (BIC) was set to 1, the alpha value used in conjunction with Fisher's *z* tests to determine conditional independence and refine the preliminary results was set to .010, and one-edge faithfulness was not assumed. Because causal discovery algorithms recover causal pathways more effectively when they are provided with prior knowledge (Shen et al., 2020), the algorithm was told that age and sex could not be caused by any other variable.

To provide information about the size of potential causal effects identified by GFCI, structural equation models featuring the edges GFCI suggested were fit to the data (using lavaan; Rosseel, 2019). Standardized structure coefficients were then added to the PAG. In cases where the voting ensemble preferred one edge orientation (→) to its opposite (←) by a slim margin (< 10%), or when an edge orientation did not match that suggested by prior literature, Vuong's test (Vuong, 1989) was used to compare the model suggested by GFCI to that with the edge in question reversed. Significant results

indicate support for a particular edge orientation (GFCI's suggestion or the alternative). If Vuong's test could not resolve the ambiguity, and the edge connected two skewed variables, the Linear Non-Gaussian Acyclic Model (LiNGAM; Shimizu et al., 2006)) was used to identify a preferred edge direction. Rather than relying on a pattern of partial correlations to identify potential causal relations, LiNGAM leverages information resulting from skewed distributions to identify potential causal relations. It is therefore capable of resolving edge direction with as few as two variables (unlike GFCI).

Table S6.
GFCI Ensemble Voting Shares

Var1	Var2	0->	<-0	0-0	←pdpl	←pdnl	←ddnl	←→	→pdpl	→pdnl	→ddnl	none
CoDamp	SIAS	--	.02	--	.04	.02	--	--	--	--	**.51**	.41
CoDamp	NII	--	**.56**	.04	--	--	--	--	--	--	.20	.17
CoDamp	DERS+	.03	--	--	.03	.04	**.49**	--	.02	.01	.35	.03
ATTE	DERS+	.10	.01	--	.01	.04	**.38**	.06	.02	.01	.30	.05
CoDamp	DIRIRS	.03	--	--	.01	**.32**	.21	--	.01	.03	.31	.08
Age	DIRIRS	**.51**	--	--	--	--	--	--	.41	--	--	.08
ATTE	RSQ	.03	.03	.04	.01	.02	**.50**	--	.07	.04	.26	--
ATTE	TENSE	.08	--	--	.04	**.31**	.19	.01	.01	--	.16	.18
EPSI	SIAS	--	.02	--	.09	.05	**.78**	--	--	--	--	.05
DERS+	PHQ9	--	.08	.03	.06	.27	**.54**	--	--	--	--	--
DIRIRS	RSQ	--	--	--	.04	.04	.02	--	--	.23	**.43**	.23
PIB	RSQ	.75	--	.08	--	--	.11	--	--	--	.02	.03
EPSI	TENSE	--	.04	--	--	.36	**.50**	--	.04	--	.03	--
DIRIRS	TENSE	--	.04	--	.04	.04	**.49**	--	--	--	.38	--
SIAS	SPS	--	--	.12	--	--	**.82**	--	--	--	.05	--
PHQ9	SPS	.03	--	.10	--	.01	.30	--	.02	.05	**.42**	.06

PHQ9	TENSE	.08	.02	.06	--	--	.35	.02	.03	.04	**.36**	.04

Note. Var=variable. Pd=possibly direct. Dd=definitely direct. Pl=possibly latent. Nl=no latent. NIB=negative interpretation bias. NII=negative interpretation inflexibility. PIB=positive interpretation bias. DIRIRS=excessive reassurance seeking. RSQ=rejection sensitivity. ATTE=attitude to emotional expression. DERS+=difficulty regulating positive emotion. SIAS=social interaction anxiety. SPS=social phobia. EPSI=eating pathology symptoms inventory. CoDamp=Co-dampening of positive emotion. TENSE=negative social exchange. Numbers represent the fraction of the ensemble that voted for a particular edge type. Among edges where significant ambiguity existed, Vuong's test did not provide support for either the edge preferred by the ensemble or the reversed edge (PHQ9-TENSE: z=0, p=.500; CoDamp-DIRIRS: z=-.82, ATTE-DERS+: *p*=.793; z=1.12, *p*=.132.). LiNGAM supported the ensemble-preferred edge DIRIRS → CoDamp, but suggested that PHQ9 was caused by TENSE, rather than vice-versa. Therefore, the edge preferred by LiNGAM (rather than the ensemble) was inserted into the final PAG.

Section S9. Outliers and Data Quality

Outliers. Summary scores for measures of interest were checked for univariate outliers using the method of Hubert and Van Der Veeken (2008), which is designed for filtering skewed data. A total of 99 univariate outliers were detected across the following varisables: BMI (1), average plausibility ratings on the BADE task (84), and BADE Task metrics of inflexibility/bias (14). Multivariate outliers were checked for by visualizing bivariate relations between key variables of interest and by examining Mahalanobis

distances. No such outliers were observed. The results of the present study did not differ qualitatively when outliers were included vs. not included in the dataset.

Data Quality. Several pre-registered steps were taken to ensure high data quality. Screening survey respondents were required to have a history of good-quality responses (at least 99% approval of past submissions on the MTurk platform, across at least 1000 submissions [1000, rather than 3000, as pre-registered, was used due to the options available in the CloudResearch platform). Respondents were not invited to complete the main study if they failed at least one of five attention checks (items placed at random intervals to detect inattentive or low-effort responding), reported a birth date inconsistent with their age, completed the screening survey at a speed more than three standard deviations from the mean, or had a person-total correlation on the EPSI more than three standard deviations from the mean (M =.50, SD = .19). Main survey respondents who finished at an especially slow/fast speed (more than 3 SDs from the mean completion speed, M = 40.77 minutes, SD = 24.39 minutes), failed at least one of four attention checks, reported a birth date inconsistent with their age, or provided demographic information (race, sex, gender, or education level) inconsistent with their screening survey answers were excluded. We also conducted checks on self-reported height (which was required to be within 2 inches in both surveys) and weight (which was required to be within 10 pounds in both surveys; larger changes were deemed implausible given the ~1 week period between surveys). Finally, a captcha was included in both surveys to deter "participation" by computer programs.

In addition to these pre-registered criteria, individuals with EPSI scores in the screener and main study that differed (in absolute value) by three standard deviations more than the sample average were excluded; large differences in EPSI scores across measurement occasions just days apart were deemed indicative of low-quality responding because the measure captures one month of behavior. Notably, the correlation between EPSI scores across measurement occasions was very high (rho=.89,p<.001), supporting the validity of our data. We also excluded individuals with implausible self-reported heights (>100 inches) or weights (> 600lbs). Finally, we used CloudResearch, an online platform that provides additional services for researchers using MTurk, to select for participants who passed data quality checks

("CloudResearch Approved Participants"), block duplicate IP addresses, block suspicious geolocations, and ensure that IP addresses were consistent with a US location. In accordance with recent recommendations (Burnette et al., 2021), we also checked for implausible BMI values (< 14; none were present in our final sample), and examined the rate of eating disorder hospitalization and diagnosis in our sample to ensure these were plausible (both were).

Our strategy for ensuring high-quality data closely follows the recommendations of data-quality researchers (Chandler et al., 2020; Chandler & Shapiro, 2016). Our previous studies of interpretative processes following recommendations by these researchers has produced replicable results (example: compare: (Everaert et al., 2018, 2020).

Table S7.

Data Quality Screening Summary

Survey	Reason For Removal	Number Removed
Screener		
	Failed too many attention checks	36
	Completion speed > +/-3SD from mean	2
	Person-total correlation > +/- 3SD from mean	11
	Implausible Weight or Height	6
Main		
	Failed too many attention checks	5
	Completion speed > +/-3SD from mean	5
	Person-total correlation > +/- 3SD from mean	1
	Implausible Weight or Height	1
	Inconsistent Demographics across surveys	75

Note. Number of removed participants is for each criterion individually, without considering other criteria, so that future researchers can better evaluate the effectiveness of each measure taken.

Section S10. Zero-order correlations

Table S8.

Zero-order correlations

	2	3	4	5	6	7	8	9	10	11	12	13	14
1	-.08	.26*	.44*	.45*	.29*	.37*	.48*	.51*	.32*	.19*	.08	.19*	-.09
2		-.02	-.08	-.13	-.05	-.10	-.13^{+}	.02	-.02	-.08	-.04	-.06	.05
3			.29*	.32*	.24*	.31*	.27*	.42*	.36*	.35*	.12^{+}	.11^{+}	.05
4				.81*	.39*	.39*	.58*	.45*	.33*	.40*	.06	.21*	-.13^{+}
5					.35*	.38*	.51*	.38*	.34*	.36*	.06	.16*	-.09
6						.35*	.42*	.43*	.30*	.41*	.08	.04	-.09

7		.53*	.42*	.39*	.23*	.09	.13^{+}	-.02
8			.53*	.37*	.42*	.07	.24*	-.11
9				.39*	.35*	.12^{+}	.20*	-.01
10					.23*	.13*	.16*	.08
11						.15^{+}	.18*	-.16*
12							-.09	-.18*
14								.02

1=Eating Pathology Symptoms Inventory, 2=Mind in the Eyes, 3=Reassurance Seeking, 4=Social Phobia, 5=Social Interaction Anxiety, 6=Attitude to Emotional Expression, 7=Difficulty Regulating Positive Emotion, 8=Depression, 9=Negative Social Exchange, 10=Co-dampening of Positive Emotions, 11=Rejection Sensitivity, 12=Negative Interpretation Inflexibility, 13=Negative Interpretation Bias, 14=Positive Interpretation Bias. All correlations are non-parametric (Spearman's rho), df=308. $^{+}$=p<.05, *=p<.01. All BADE task metrics are from disconfirming-the-negative scenarios.

This system of zero-order correlations is consistent with the notion that eating disorder symptoms (such as restrictive eating) are associated with impaired socioemotional functioning and biased interpretations, but provides less support for a relation between these symptoms and inflexible interpretations.

Section S11. Additional Pre-registered Analyses

Do the Variables That Predict Continuous Eating Disorder Symptoms Differ in Individuals Who Do Vs. Do Not Endorse Restrictive Eating?

As pre-registered, this regression model in main text with continuous eating disorder symptoms as the criterion variable was re-examined in subgroups segmented by their responses to the dietary restriction screener. For people who endorsed restrictive eating, depression (at a trend level), but not social interaction anxiety, continued to predict eating disorder symptoms. Negative interpretation bias also predicted eating disorder symptoms at a trend level in these individuals (β=0.15, t(130)=1.83, p=.070, *95%CI*=[-0.01 0.30]). For people who denied restrictive eating, inflexible negative interpretations (at a trend level: β=0.12, t(160)=1.71, p=.089, *95%CI*=[-0.02 40.27]), depression (β=0.30, t(160)=3.75, p<.001, *95%CI*=[0.14 0.45]), and social interaction anxiety (β=0.29, t(160)=2.72, p=.007, *95%CI*=[0.08 0.50]),) predicted eating disorder symptoms.

Which Symptoms Might Contribute to the Comorbidity Between Anxiety, Depression, and Eating Pathology?

Anxiety disorders (e.g., social anxiety) and affective disorders (e.g., depression) are both frequently comorbid with eating disorders (Godart et al., 2007; Swinbourne & Touyz, 2007). Research exploring the comorbidity between depression, anxiety, and eating disorder symptoms has begun to

employ psychometric network analysis, with the goal of identifying bridge symptoms that may link these conditions (Forrest et al., 2019; Levinson et al., 2018; Sahlan et al., 2020). Identifying links between these forms of pathology in the general population may inform treatment development for individuals with comorbid eating and mood disorders, which is important because psychiatric comorbidities in individuals with eating disorders are associated with worse outcomes, including suicide attempts (Pisetsky et al., 2013) and reduced weight gain during treatment (Eskild-Jensen et al., 2020).

Accordingly, exploratory network analyses were conducted to identify symptoms bridging depression, social anxiety, and eating disorder symptoms. The bridge one-step expected influence was sufficiently stable (**SI Figure S9**). The nodes with the strongest bridge centrality in the network included PHQ item 8 (psychomotor agitation/retardation), PHQ item 5 (excessive/depressed appetite), and SPS item 1 (people staring while you walk down the street). PHQ item 8 was strongly connected to use of diet pills (EPSI 17) and vomiting to lose weight (EPSI 27). PHQ item 5 was related to meal skipping (EPSI 43), eating on autopilot (EPSI 44), and binge eating (EPSI 45), and evening snacking without awareness (EPSI 9). SPS item 1 (people staring while you walk down the street) was associated with EPSI 41 (exercised until exhausted), EPSI 7 (muscle building supplements), EPSI 19 (ate until uncomfortably full), and EPSI 28 (did not notice how much you ate). The EPSI items with the strongest bridge centralities were items 39 (overeating until feeling ill) and 44 (eating on autopilot) and 32 (muscles too small). EPSI item 39 was connected to SIAS 1 (tense up when meeting an acquaintance on the street), SPS 5 (tense when speaking in public), and PHQ 5 (excessive/depressed appetite). EPSI item 44 was strongly connected to several PHQ items, including PHQ 5 and PHQ4 (low energy), and PHQ 8 (psychomotor agitation/retardation). EPSI item 32 was strongly related to several PHQ items (e.g., PHQ 8) and social anxiety items (e.g., SPS 5, tense when public speaking and SPS 3, tense on public transit).

These results are broadly consistent with those of previous research using network analysis to examine relations between eating disorder symptoms and anxiety/depression. For example, in the present study a relatively major bridge node, eating without awareness (EPSI 9), was associated with feeling bad about oneself (PHQ 6), which parallels the relation between feeling inadequate and fear of losing control

over eating in past research (Forrest et al., 2019). Future research could expand upon this exploratory analysis by examining how symptoms of depression, social anxiety, and eating disorders are related to one another across time, with the goal of identifying symptoms that may contribute to the formation and maintenance of comorbidities between these psychopathologies.

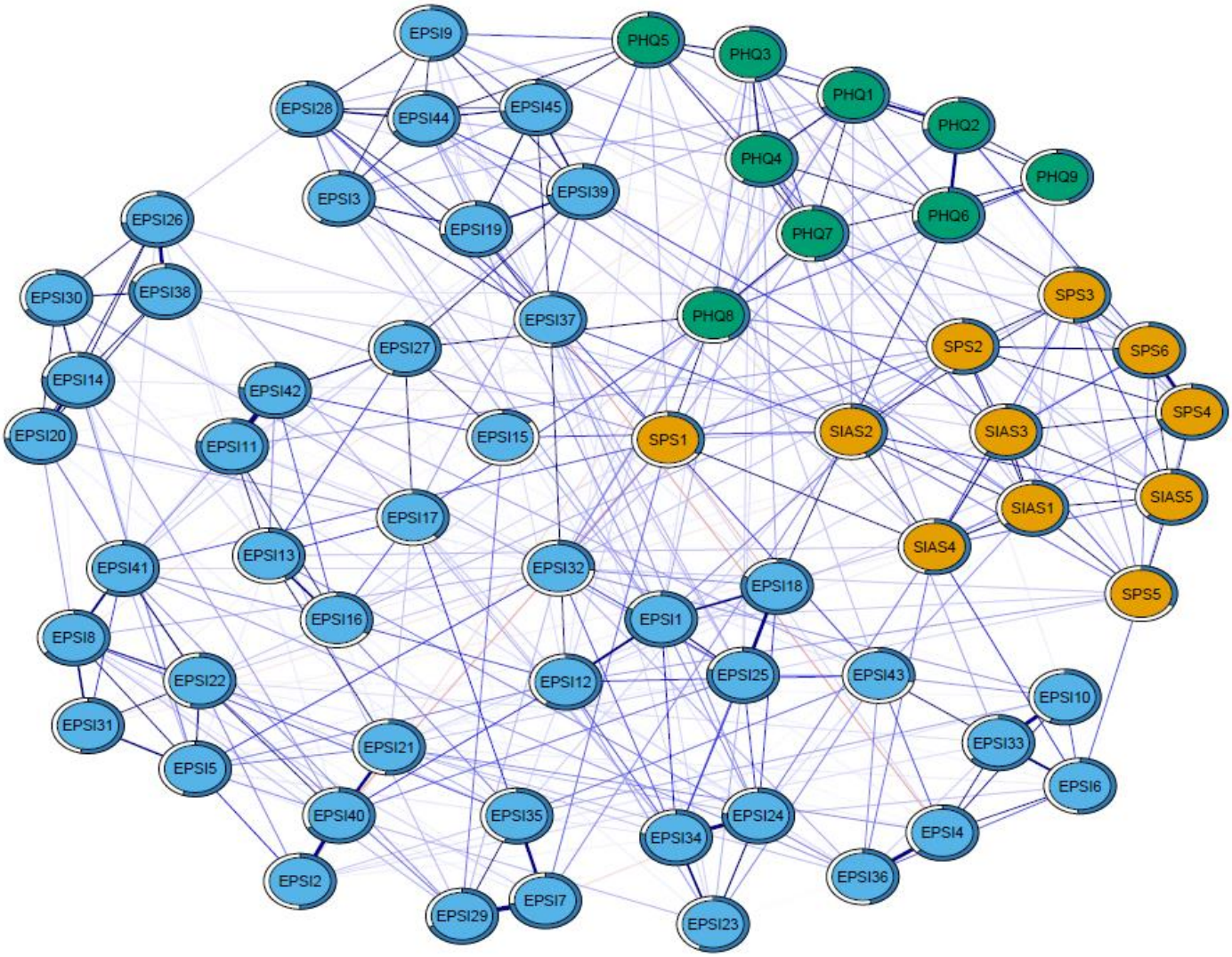


Figure S9. Regularized partial correlation network. Annulus surrounding each node denotes predictability (more filled=more predictable). Red=negative association. Blue=positive association. Smaller nodes are

covariates. EPSI1=dislike clothing fit. EPSI2=exclude unhealthy food. EPSI3=ate when not hungry. EPSI4=told not eating much. EPSI5=drive to exercise daily. EPSI6=how little I eat would surprise others. EPSI7=used muscle building supplements. EPSI8=exercise hard. EPSI9=evening snacking sans awareness. EPSI10=full more easily than others. EPSI11=diuretics to lose weight. EPSI12=dislike many outfits. EPSI13=believed laxatives are good way to lose weight. EPSI14=obese people lack self control. EPSI15=thought about steroids. EPSI16=used diet tea. EPSI17=diet pills. EPSI18=disliked body appearance. EPSI19=ate until uncomfortable. EPSI20=felt overweight people lazy. EPSI21=counted calories. EPSI22=planned around exercise. EPSI23=butt too big. EPSI24=disliked thighs. EPSI25=wished body change. EPSI26=disgusted by overweight people. EPSI27=vomit to lose weight. EPSI28=did not notice consumption volume. EPSI29=considered muscle building supplement. EPSI30=overweight people unattractive. EPSI31=exercise 5+ days/week. EPSI32=muscles too small. EPSI33=full after eating little. EPSI34=dissatisfied with hips. EPSI35=protein supplements. EPSI36=people said eat more. EPSI37=could not resist offered food. EPSI38=disgusted by obese people. EPSI39=ate until sick. EPSI40=avoided caloric food. EPSI41=exercised to exhaustion. EPSI42=diuretics for weight loss. EPSI43=skipped 2+ meals straight. EPSI44=ate on autopilot. EPSI45=binge eating. PHQ1=anhedonia. PHQ2=low mood. PHQ3=sleep disturbance. PHQ4=low energy. PHQ5=appetite disturbance. PHQ6=feeling bad about self. PHQ7=trouble concentrating. PHQ8=psychomotor disturbance. PHQ9=suicide ideation. SPS1=nervous about people staring when walking down street. SPS2=fear of blushing. SPS3=tense on public transit. SPS4=self-conscious when public eating. SPS5=tense when public speaking. SPS6=worry head will nod/shake. SIAS1=tense if meet acquaintance. SIAS2=tense when alone with one person. SIAS3=nervous mixing with unfamiliar people. SIAS4=worry about being ignored in groups. SIAS5=tense in groups. Blue nodes=eating disorder symptoms. Orange nodes=anxiety symptoms. Green nodes=depression symptoms.

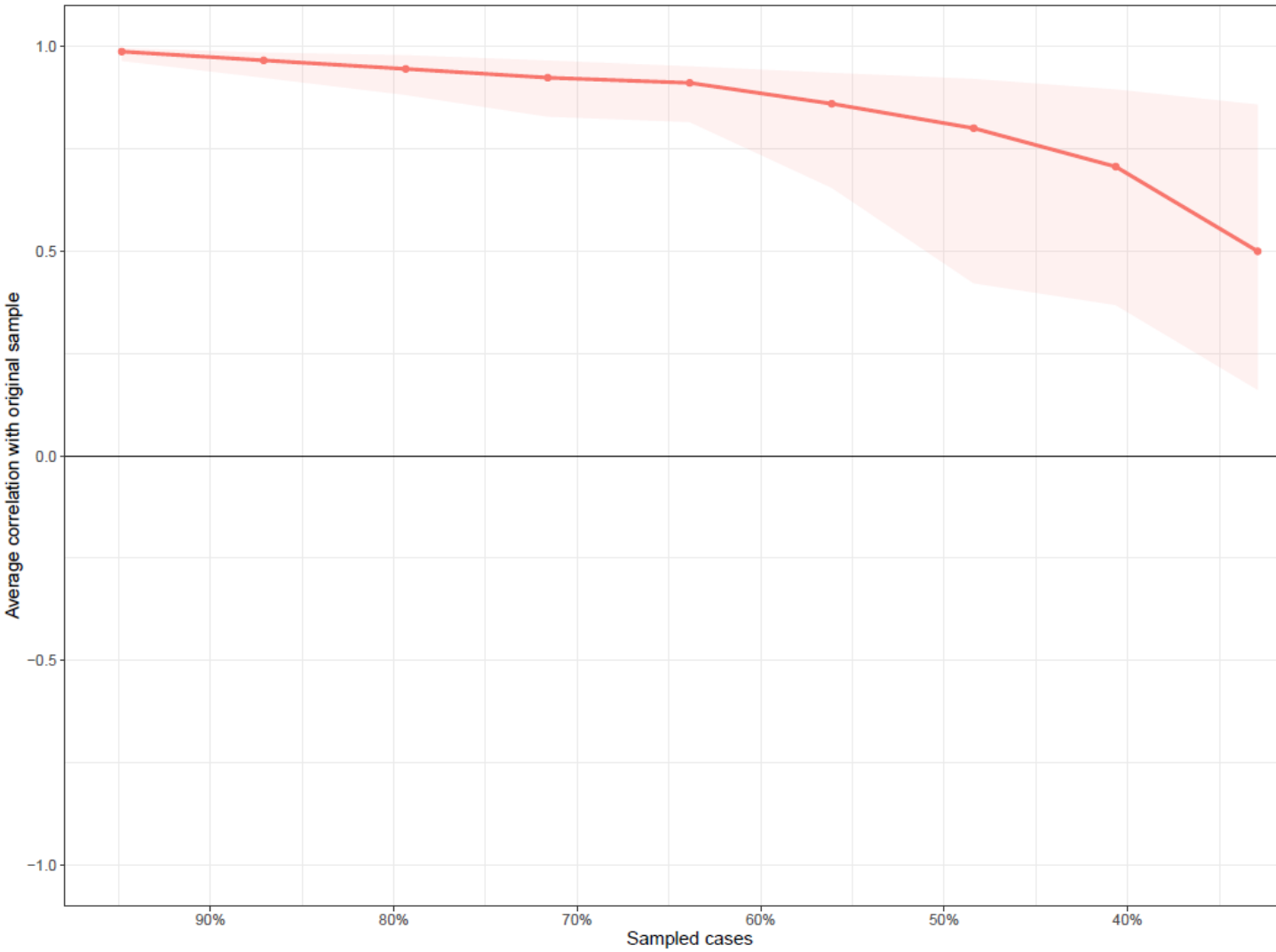


Figure S10. Stability of bridge one-step expected influence, computed using a case-drop bootstrapping procedure. Cloud represents 95% CIs. CS-Coefficient=.44, which is above the minimum .25 threshold, but not above the preferred .50 threshold.

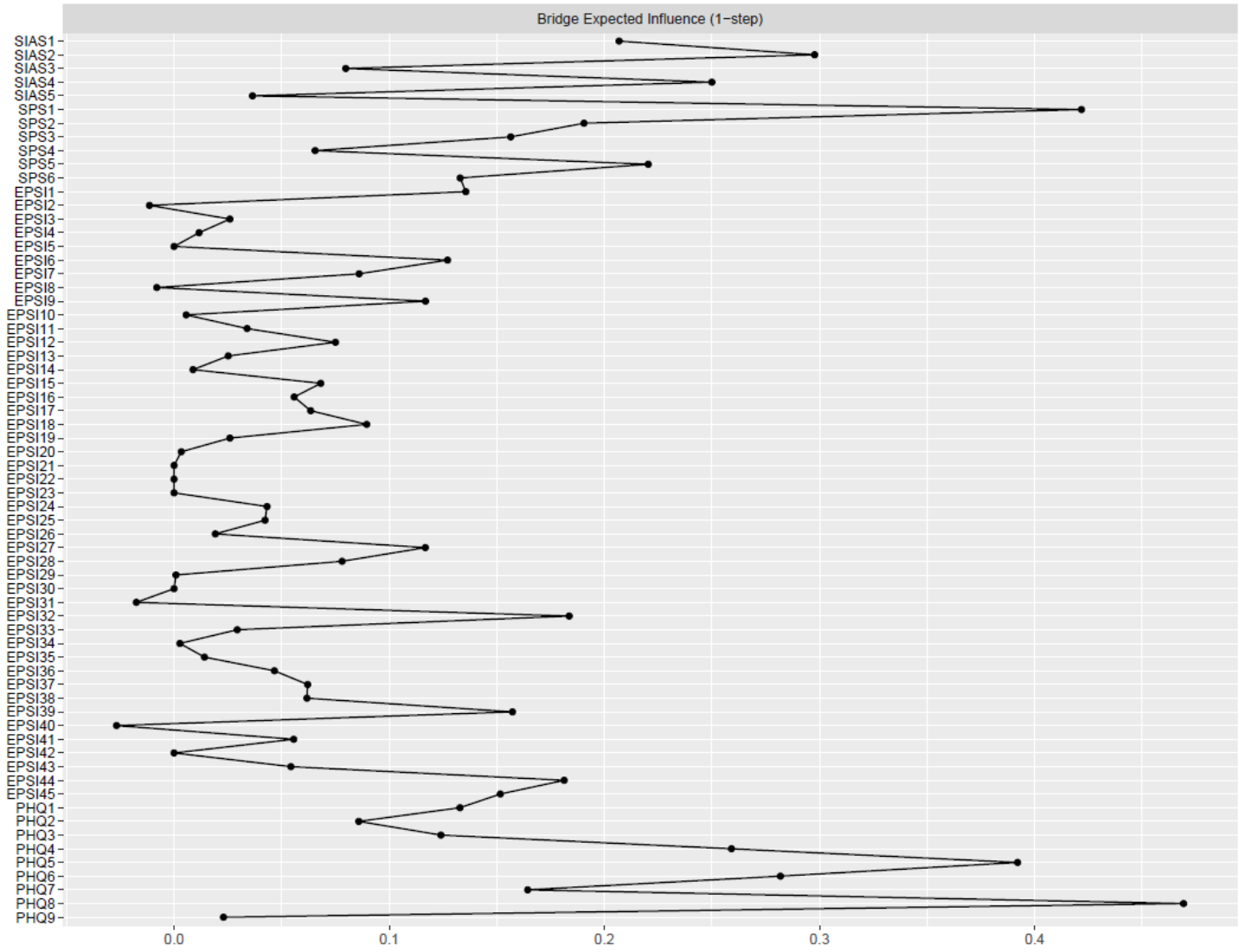


Figure S11. Bridge one-step expected influence for each network node.

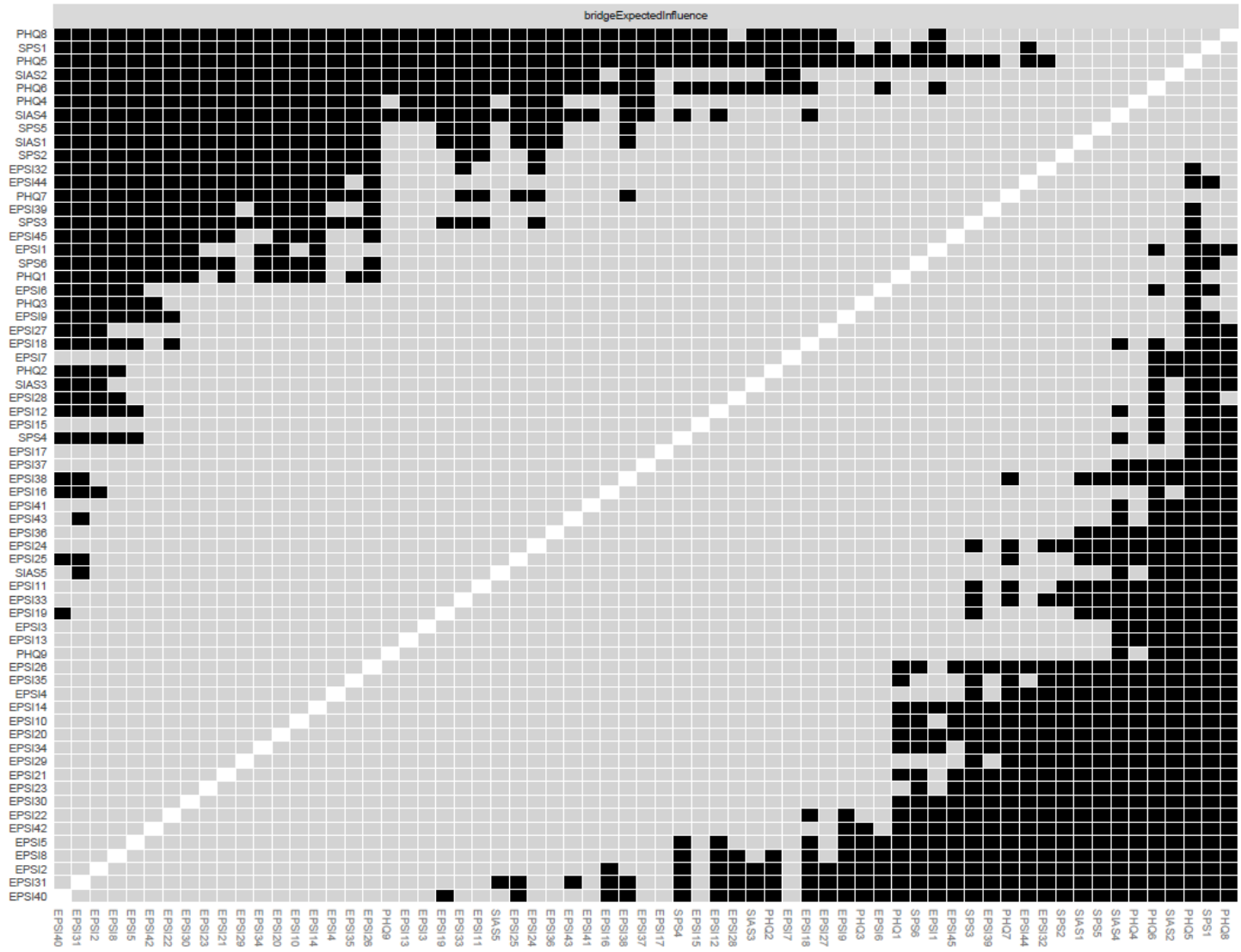


Figure S12. Bootstrapped difference tests (α = .05) between bridge one-step expected influences in the estimated network. Black = parameters are significantly different for those edges. Grey = no significant difference. Each number in a white box represents the value of the tested parameter for that node.

Section S12. Visualization of MANCOVA Results

This section presents a visualization of the MANCOVA results.

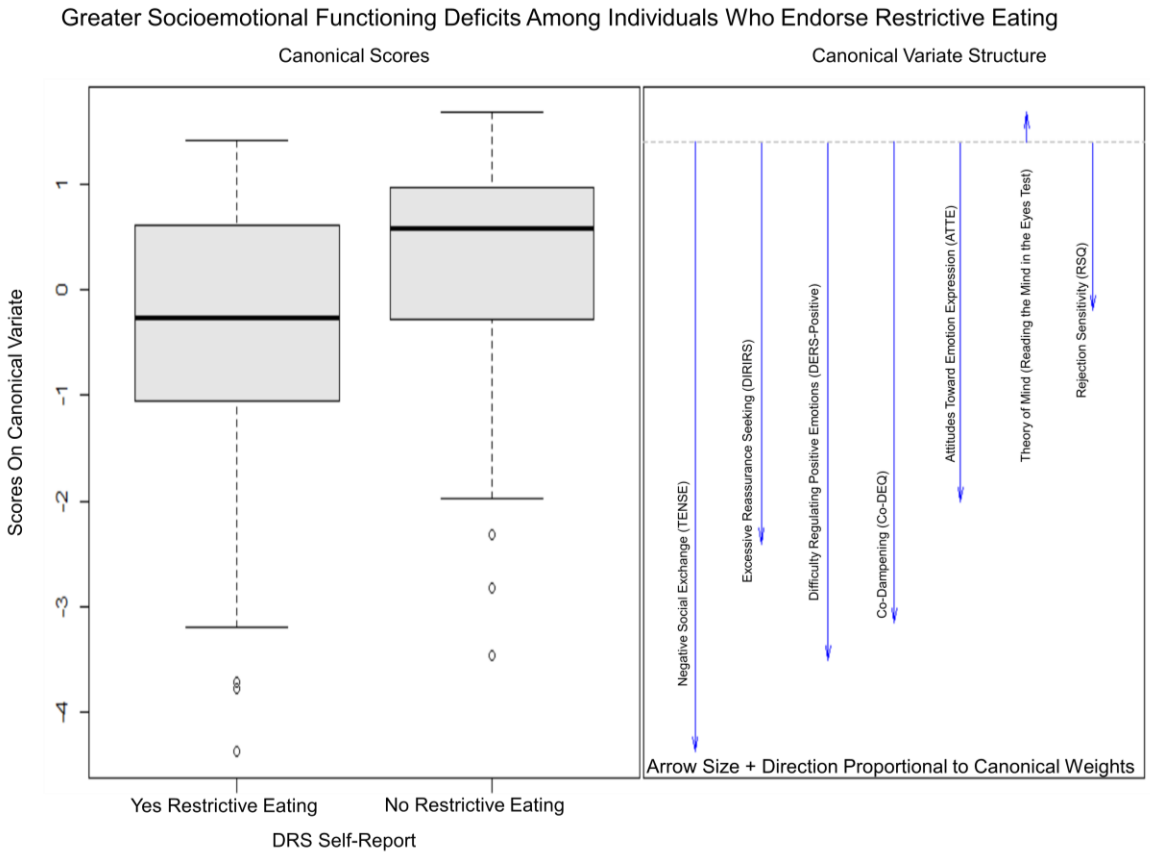


Figure S13. The left panel provides boxplots of scores on a canonical discriminant (the linear combination of response variables that contributes most to the multivariate effect) for the DRS predictor. The right panel depicts the contribution of each response variable to the discriminant; arrows that point upward represent positive structure coefficients; those that point downward represent negative coefficients. Arrow length is proportional to the absolute value of the structure coefficient. Note that because of the structure of the variate, lower scores = more impairment.

References

Burnette, C. B., Luzier, J. L., Bennett, B. L., Weisenmuller, C. M., Kerr, P., Martin, S., Keener, J., & Calderwood, L. (2021). Concerns and recommendations for using Amazon MTurk for eating disorder research. *International Journal of Eating Disorders*, *September*, 1–10. https://doi.org/10.1002/eat.23614

Chandler, J., & Shapiro, D. (2016). *Conducting Clinical Research Using Crowdsourced Convenience Samples. 12*, 53–81. https://doi.org/10.1146/annurev-clinpsy-021815-093623

Chandler, J., Sisso, I., & Shapiro, D. (2020). Participant carelessness and fraud: Consequences for clinical research and potential solutions. *Journal of Abnormal Psychology*, *129*(1), 49–55. https://doi.org/10.1037/abn0000479

de Beurs, D. (2017). Network analysis: A novel approach to understand suicidal behaviour. *International Journal of Environmental Research and Public Health*, *14*(3). https://doi.org/10.3390/ijerph14030219

Epskamp, S. (2020). *bootnet* (1.4.3). https://cran.r-project.org/web/packages/bootnet/index.html

Epskamp, S., Borsboom, D., & Fried, E. I. (2018). Estimating psychological networks and their accuracy: A tutorial paper. *Behavior Research Methods*, *50*(1), 195–212. https://doi.org/10.3758/s13428-017-0862-1

Eskild-Jensen, M., Støving, R. K., Flindt, C. F., & Sjogren, M. (2020). Comorbid depression as a negative predictor of weight gain during treatment of anorexia nervosa: A systematic scoping review.

European Eating Disorders Review, *January*, 1–15. https://doi.org/10.1002/erv.2787

Everaert, J., Bronstein, M. V., Cannon, T. D., & Joormann, J. (2018). Looking Through Tinted Glasses: Depression and Social Anxiety Are Related to Both Interpretation Biases and Inflexible Negative Interpretations. *Clinical Psychological Science*, *6*(4), 517–528. https://doi.org/10.1177/2167702617747968

Everaert, J., Bronstein, M. V., Castro, A., Cannon, T. D., & Joormann, J. (2020). When negative interpretations persist, positive emotions don't! Inflexible negative interpretations encourage depression and social anxiety by dampening positive emotions. *Behaviour Research and Therapy*, *124*, 103510. https://doi.org/10.1016/j.brat.2019.103510

Everaert, J., Bronstein, M. V., Klonsky, E. D., Cannon, T. D., & Joormann, J. (2021). Inflexible Interpretations of Ambiguous Social Situations: A Novel Predictor of Suicidal Ideation and the Beliefs that Inspire It. *Clinical Psychological Science*.

Forrest, L. N., Sarfan, L. D., Ortiz, S. N., Brown, T. A., & Smith, A. R. (2019). Bridging eating disorder symptoms and trait anxiety in patients with eating disorders: A network approach. *International Journal of Eating Disorders*, *52*(6), 701–711. https://doi.org/10.1002/eat.23070

Fried, E. I., Eidhof, M., Palic, S., Costantini, G., Huisman-van Dijk, H., Bockting, C., Engelhard, I., Armour, C., Nielsen, A., & Karen-Inge, K. (2018). Replicability and Generalizability of Posttraumatic Stress Disorder (PTSD) Networks: A Cross-Cultural Multisite Study of PTSD Symptoms in Four Trauma Patient Samples. *Clinical Psychological Science*, *6*(3), 335–351. https://doi.org/10.1177/2167702617745092

Godart, N. T., Perdereau, F., Rein, Z., Berthoz, S., Wallier, J., Jeammet, P., & Flament, M. F. (2007). Comorbidity studies of eating disorders and mood disorders. Critical review of the literature. *Journal of Affective Disorders*, *97*(1–3), 37–49. https://doi.org/10.1016/j.jad.2006.06.023

Haslbeck, J. M. B., & Fried, E. I. (2017). *How predictable are symptoms in psychopathological networks? A reanalysis of 18 published datasets*. https://doi.org/10.1017/S0033291717001258

Jones, P. (2020). *networktools: Tools for identifying important nodes in networks* (R package version 1.2.3). https://cran.r-project.org/package=networktools

Levinson, C. A., Brosof, L. C., Vanzhula, I., Christian, C., Jones, P., Rodebaugh, T. L., Langer, J. K., White, E. K., Warren, C., Weeks, J. W., Menatti, A., Lim, M. H., & Fernandez, K. C. (2018). Social anxiety and eating disorder comorbidity and underlying vulnerabilities: Using network analysis to conceptualize comorbidity. *International Journal of Eating Disorders*, *51*(7), 693–709. https://doi.org/10.1002/eat.22890

Pisetsky, E. M., Thornton, L. M., Lichtenstein, P., Pedersen, N. L., & Bulik, C. M. (2013). Suicide attempts in women with eating disorders. *Journal of Abnormal Psychology*, *122*(4), 1042–1056. https://doi.org/10.1037/a0034902

Robinaugh, D. J., Millner, A. J., & Mcnally, R. J. (2016). Identifying Highly Influential Nodes in the Complicated Grief Network. *Journal of Abnormal Psychology*, *125*(6), 747–757. https://doi.org/10.1037/abn0000181

Rosseel, Y. (2019). *lavaan* (0.6.5). https://cran.r-project.org/web/packages/lavaan/index.html

Sahlan, R. N., Williams, B. M., Forrest, L. N., Saunders, J. F., Fitzsimmons-Craft, E. E., & Levinson, C. A. (2020). Disordered eating, self-esteem, and depression symptoms in Iranian adolescents and young adults: A network analysis. *International Journal of Eating Disorders*, *February*, 1–16. https://doi.org/10.1002/eat.23365

Shen, X., Ma, S., Vemuri, P., Simon, G., Weiner, M. W., Aisen, P., Petersen, R., Jack, C. R., Saykin, A. J., Jagust, W., Trojanowki, J. Q., Toga, A. W., Beckett, L., Green, R. C., Morris, J., Shaw, L. M., Khachaturian, Z., Sorensen, G., Carrillo, M., … Fargher, K. (2020). Challenges and Opportunities

with Causal Discovery Algorithms: Application to Alzheimer's Pathophysiology. *Scientific Reports*, *10*(1), 1–12. https://doi.org/10.1038/s41598-020-59669-x

Shimizu, S., Hoyer, P. O., Hyvärinen, A., & Kerminen, A. (2006). A linear non-gaussian acyclic model for causal discovery. *Journal of Machine Learning Research*, *7*, 2003–2030.

Swinbourne, J. M., & Touyz, S. W. (2007). The co-morbidity of eating disorders and anxiety disorders: A review. *European Eating Disorders Review*, *15*(4), 253–274. https://doi.org/10.1002/erv.784

Tabachnick, B. G., & Fidell, L. S. (2007). *Using Multivariate Statistics*. Pearson.

Terluin, B., De Boer, M. R., & De Vet, H. C. W. (2016). Differences in connection strength between mental symptoms might be explained by differences in variance: Reanalysis of network data did not confirm staging. *PLoS ONE*, *11*(11), 1–12. https://doi.org/10.1371/journal.pone.0155205

Vuong, Q. H. (1989). Likelihood Ratio Tests for Model Selection and Non-Nested Hypotheses. *Econometrica, Vol. 57, No. 2*, *57*(2), 307–333.