


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 (e.g., social emotion expression/regulation, response to social elicitors of emotion) 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: A total of 310 participants from the general population, recruited from an online crowdsourcing platform, 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 emotion regulation (down-regulation of positive social emotion) thought to encourage disordered eating.

Public Significance: This study suggests that less accurate interpretations of ambiguous social information encourage anxious anticipation of rejection and down-regulation of positive social emotions, both of which are thought to promote eating disorder symptoms. Knowledge provided by this study about the likely relations between interpretive processes, social/emotional functioning, and eating disorder symptoms may help inform treatments for eating disorders, particularly those that attempt to modify patterns of interpretation.

KEYWORDS

GFCI, interpretation bias, interpretation inflexibility, restrictive eating, socioemotional functioning

1 | INTRODUCTION

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, Watts, Loth, Pearson, & Neumark-Stzainer, 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 (e.g., less self-reported use of effective emotion regulation strategies, such as reappraisal or reframing; Haynos, Wang, & Fruzzetti, 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 binge eating and purging (Haynos, Fruzzetti, Safer, & Chen, 2011; Turton, Cardi, Treasure, & Hirsch, 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; 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 (for example, encouraging social withdrawal secondary to avoidance of social situations involving eating; Christensen & Haynos, 2020). This process would leave individuals with eating disorder tendencies increasingly isolated and reliant on disordered eating for emotion regulation (Arcelus, Haslam, Farrow, & Meyer, 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). Furthermore, negative interpretation bias has been associated with emotion regulation strategies that increase negative emotion (like rumination; Everaert, Bronstein, Castro, Cannon, & Joormann, 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 between inflexible negative interpretations and depression, social anxiety, and dampening of positive emotion, even when statistically controlling for interpretation bias (Everaert et al., 2020; Everaert, Bronstein, Cannon, & Joormann, 2018). Despite the association between these emotion-related constructs and disordered eating (Coniglio, Christensen, Haynos, Rienecke, & Selby, 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.

2 | THE PRESENT STUDY

To help resolve the above ambiguities in the literature, the present study tested the following preregistered (<https://osf.io/trf5x>) hypotheses in a general population sample of adults from the United States, recruited via an online marketplace. In the following hypotheses, 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.*

Hypothesis 3. *Endorsement (vs. non-endorsement) of restrictive eating will be associated with impaired socioemotional functioning.*

Hypothesis 4. *Endorsement (vs. non-endorsement) of restrictive eating will be associated with biased and inflexible interpretations.*

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: (a) nutritional effects of restrictive eating may directly reduce cognitive flexibility (Treasure & Schmidt, 2013), including in interpreting social situations and (b) 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 preregistered exploratory analysis using machine learning algorithms to probe potential causal pathways connecting eating disorder symptoms, socioemotional functioning, and interpretative processes.

3 | METHOD

3.1 | Participants and recruitment

Participants ($n = 1,308$) 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, in order to ensure sufficient variation in both restrictive eating and general eating disorder symptoms. We planned to recruit 150 individuals from each group for the main study. 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 Appendix S1, Section S1.

3.2 | Open science practices

Hypotheses and analyses were preregistered, and the dataset is public (Bronstein, 2021). For details, see Section S2.

3.3 | Protocol

This study was reviewed and 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 Section 3.4. Measures were completed in randomized order. The payment was \$6.

3.4 | Measures

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

3.4.2 | 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 Physica's Health Questionnaire—Depression Module (PHQ-9; Kroenke, Spitzer, & Williams, 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, Valentiner, McGrath, Gier-Lonsway, & Kim, 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.

3.4.3 | 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: Section S3). Respondents are presented with ambiguous interpersonal scenarios (for example, 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*: 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.

3.4.4 | Socioemotional functioning

These measures were selected to examine domains of functioning associated with eating disorders, including theory of mind (Leppanen, Sedgewick, Treasure, & Tchanturia, 2018), interpersonal styles that generate interpersonal stress and may decrease social support (Cardi et al., 2017; Cooley, Toray, Valdez, & Tee, 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, Wheelwright, Hill, Raste, & Plumb, 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 ($\omega_{\text{total}} = .59$), 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, Okun, Pool, & Ruhlman, 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, Gratz, & Lavender, 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).

3.4.5 | 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: 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.

3.5 | Analyses

For information on statistical test assumptions, see Section S6.

3.5.1 | 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, Wildes, & Hunt, 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 (see, e.g., Figure S7).

3.5.2 | 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.

3.5.3 | 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/nonendorsement 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.

3.5.4 | 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. This approach makes 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, 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 & 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, 2021).

Network nodes were evaluated for redundancy using the goldbricker function in R's *networktools* package, version 1.2.3 (Jones, 2020). Variable pairs were considered redundant if (a) their zero-order correlation exceeded .70 (Elliott, Jones, & Schmidt, 2020) and (b) 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 Section S7.

3.5.5 | Causal discovery analysis

Exploratory Causal discovery algorithms (implemented using the Rcausal package, version 1.2.1) 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 indicate the plausibility of particular causal relations (Figure 1), can recover complex causal pathways from observational data (Shen et al., 2020).

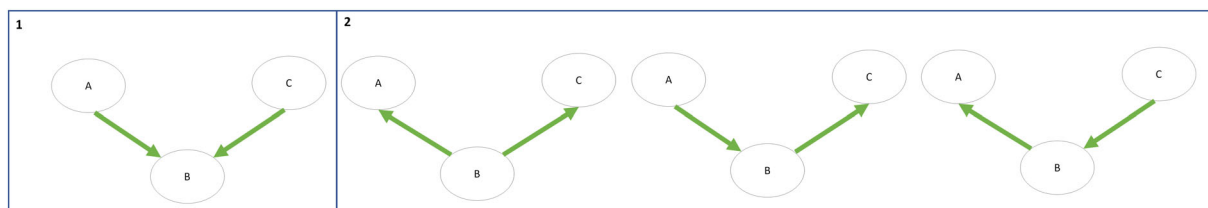
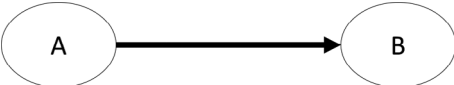

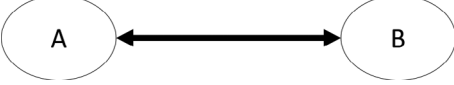



FIGURE 1 Patterns of conditional relations convey information about causal orientations. The absence of an arrow denotes the absence of a causal relation. Green arrows denote causal relations between variables (see Table 3). (a) A “collider” graph (A and C directly cause B, no edge between A and C). A is unconditionally independent of C, and A is dependent on C conditional on B. (b) 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 (a) and (b), 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

TABLE 1 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

Notes: 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.

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 1) varying depending on the set of directed edges that were present across all remaining plausible causal models (e.g., a directed edge \rightarrow 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 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 & Bentler, 1999). Chi-square statistics were not considered because they tend to over-reject models in the presence of large sample sizes (Bentler & Bonett, 1980).

3.5.6 | 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 Section S9. The final sample contained no missing data

3.6 | 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.

4 | RESULTS

4.1 | 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%) of 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 Sections S1 and S5.

4.2 | 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 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, $\beta = 0.28$, 95% CI = [0.16 0.41], and social interaction anxiety, $\beta = 0.22$, 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 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-

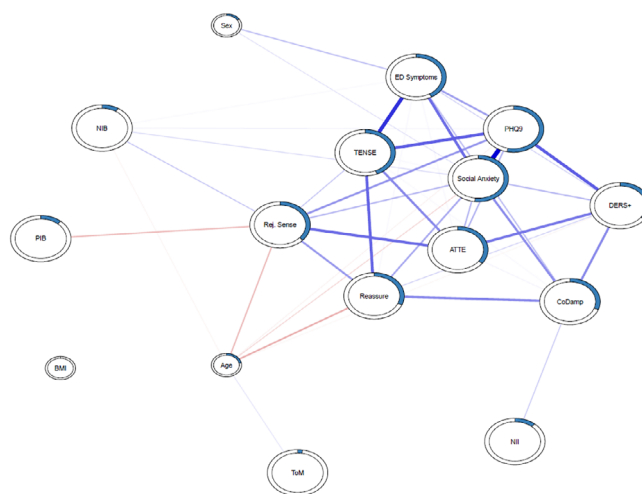


FIGURE 2 Regularized partial correlation network. Annulus surrounding each node denotes predictability (more filled = more predictable). Red = negative association. Blue = positive association. Smaller nodes are covariates. Sex is dummy coded as the effect of being female (male = 0). ATTE, attitudes to emotional expression; CoDamp, co-dampening of positive emotion; DERS, difficulty regulating positive emotion; ED, eating disorder; NIB, negative interpretation bias; NII, negative interpretation inflexibility; PHQ9, depression; PIB, positive interpretation bias; Rej. Sense, rejection sensitivity; Reassure, excessive reassurance seeking; TENSE, negative social exchange; ToM, theory of mind

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]
	MI	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]

Notes: Sex is dummy coded as the effect of being female (male = 0). Significant values are in bold. Model statistics: $F(9, 300) = 15.60$, $p < .001$, Adjusted $R^2 = .30$. Model intercept (00.00) was not significant. All variables were z-scored prior to entry into the model. Abbreviations: BMI, Body mass index; CI, confidence interval; EPSI, Eating Pathology Symptoms Inventory.

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, $\beta = 0.07$, 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 Section S7.

4.3 | 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

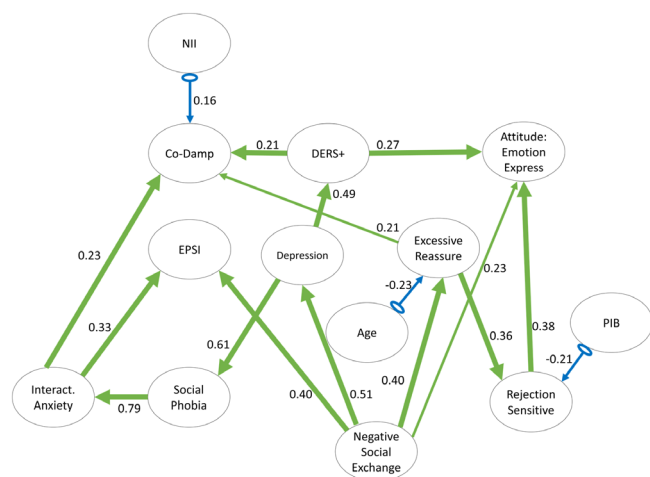


FIGURE 3 Directed Acyclic Graph suggested by the Greedy Fast Causal Inference (GFCI) causal discovery algorithm (see Table 1 for a description of possible edge types). Variables are not depicted if GFCI could not determine a potential causal relation between them and another variable included in the analysis. Numbers adjacent to edges are standardized parameter estimates from a structural equation model of the causal structure suggested by GFCI. DERS+, difficulty regulating positive emotions; EPSI, Eating Pathology Symptoms Inventory; NII, negative interpretation inflexibility; NIB, negative interpretation bias, PIB, positive interpretation bias,

TABLE 3 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]
	Body mass index	-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]

Notes: Sex is dummy coded as the effect of being female (male = 0). Restriction is coded as 1 = No (Yes = 0). Significant values are in bold. Model intercept = 0.17 (not significant).

Abbreviations: BMI, Body mass index; CI, confidence interval.

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: 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 3) 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.

5 | 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 preregistered hypotheses, endorsement (vs. nonendorsement) 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 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, Gnat, Phillipou, & Nedeljkovic, 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 Section 4.1) may explain why we found no association between eating disorder symptoms and inflexible interpretations. More severe symptoms may have nutritional sequelae that interfere with cognitive flexibility (Miles et al., 2020), and neurological sequelae that impact 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. Another 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 further 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, Dietel, & Hartmann, 2020). 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, Tchanturia, & Treasure, 2018), could be addressed by replicating our study in a more representative sample. Replicating our study in a non-crowdsourced sample would also be useful given recent concerns about the validity of

MTurk data for eating disorders research (Burnette et al., 2021), despite our use of a rigorous data quality assessment regime (see Section S9).

6 | 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.

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CONFLICT OF INTEREST

The authors have no conflicts to declare.



AUTHOR CONTRIBUTIONS

Michael V. Bronstein and Jonas Everaert developed the study design and concept, which Sophia Vinogradov and Ann Haynos helped refine. Michael V. Bronstein analyzed the data. Erich Kummerfeld provided expertise surrounding causal discovery analyses. Michael V. Bronstein drafted the manuscript. Ann Haynos, Jonas Everaert, and Sophia Vinogradov provided critical revisions. All authors approved the final manuscript for submission.

DATA AVAILABILITY STATEMENT

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.

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