Examining The Bi-Directional Association between Peer Stress and Disordered Eating: The Influence of Individual Traits and Behaviour

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Abstract

Peer stress has been identified as an important correlate of disordered eating and it is hypothesized that the association is bidirectional – not only does peer stress influence disordered eating, but it is thought that eating pathology and associated characteristics, in turn, can elicit negative evaluations from others, creating a cycle of increasing interpersonal problems and worsening eating pathology. To date, however, the nuances of these associations have yet to be fully elucidated and several key gaps remain in the literature. The aim of my dissertation work was to examine individual traits and behavioural patterns that may help explain the bidirectional association between peer stress and disordered eating.

In Article 1, I sought to examine whether two forms of eating pathology – binge eating and dietary restriction – were associated with behavioural responses to peer stress. Specifically, in a sample of 132 undergraduate women, I examined whether each form of eating pathology was associated with the tendency to retaliate following rejection by peers during competition. Participants were asked to complete an online "Survivor"-type game in which they voted to either accept or reject computerized coplayers, while also receiving acceptance or rejection feedback from others across several rounds. I examined the association between eating pathology and participants' tendency to reject coplayers who voted to reject them in the previous round. While I did not find an association between either form of eating pathology and the tendency to retaliate when rejected, we did find that dietary restriction was associated with a greater tendency to reject coplayers, *regardless of how coplayers voted for them*. This finding suggests that individuals with dietary restriction may push others away, regardless of how others treat them. Although I did not collect data that allowed me to examine why this might occur, I theorized that individuals with dietary restriction may be more sensitive to the possibility of interpersonal rejection and engage misguided attempts at pre-empting rejection by rejecting others.

In Article 2, I sought to build upon the findings of Article 1 by examining whether the trait of rejection sensitivity – the tendency to be overly sensitive to negative social evaluation – was associated with eating pathology via increased interpersonal stress. That is, whether rejection sensitivity was associated with negative reactions from others, which in turn were associated with increased eating pathology. Using both cross sectional and longitudinal self-report data collected from two samples of women – 189 first-year undergraduate students and 77 community women with binge eating – I examined whether rejection sensitivity was indirectly associated with eating pathology via two forms of interpersonal stress – ostracism and peer victimization. I did not find any indirect associations between rejection sensitivity and eating pathology via interpersonal stress in either sample. I did, however, find that rejection sensitivity was *directly* associated with eating pathology in both samples at the same time point. This finding suggests that the anticipation or perception of rejection may be a more important predictor of concurrent disordered eating than actual experiences of peer stress.

In Articles 1 and 2, eating pathology was associated with maladaptive interpersonal traits and behaviour, regardless actual experiences of peer stress. Contrary to extant theories and empirical evidence, these findings brought into question the role of interpersonal stress in disordered eating. Therefore, the goal of Article 3 was to determine if such an association may be better elucidated by considering an additional indirect pathway via which peer stress might influence eating pathology. When individuals experience chronic interpersonal stress, peer relationships may become less rewarding and pleasurable, leading individuals to seek sources of reward elsewhere (e.g., via binge eating or controlling their appearance). As such, in Article 3, I sought to examine whether peer stress was indirectly associated with eating pathology via decreased responsiveness to social reward (i.e., social anhedonia). Using longitudinal self-report data from a sample of 189 undergraduate women, I did not find an indirect association between two forms of peer stress (ostracism and victimization) and eating pathology via social anhedonia. However, I did find that victimization (but not ostracism) was directly associated with eating pathology across time. These findings suggest that a history of peer victimization is associated with worsening eating pathology over time.

Overall, the findings from these three studies add to the extant literature in two ways: 1) they suggest the need to reconsider the role of interpersonal stress in eating pathology and suggest that additional factors, such as rejection sensitivity, may be more important predictors and better targets for intervention; and 2) they highlight the importance of examining distinct forms of peer stress and suggest that victimization may be more predictive of vulnerability to worsening eating pathology over time than ostracism.

Résumé

Le stress social a été identifié comme un corrélatif important de l'alimentation désordonnée. Selon une hypothèse, cette association serait bidirectionnelle: le stress social influence l'alimentation désordonnée qui peut ensuite susciter d'avantages d'évaluations négatives par les autres. Cependant, les nuances de cette association doivent toujours être pleinement élucidées et il y a plusieurs lacunes à combler dans la littérature sur le sujet. Le but de ma thèse était d'étudier des caractéristiques individuelles aidant à expliquer l'association bidirectionnelle entre le stress social et l'alimentation désordonnée.

Dans l'Article 1, j'ai étudié si l'hyperphagie boulimique et la restriction alimentaire étaient associées avec la tendance à se venger à la suite du rejet par les pairs. Les participants devaient compléter un jeu en ligne dans lequel ils votaient pour accepter ou rejeter des joueurs virtuels tout en étant informés de leur acceptation ou rejet de la part d'autres joueurs lors de plusieurs tours. J'ai étudié l'association entre la pathologie alimentaire et la tendance des participants à rejeter les joueurs qui les avaient précédemment rejetés. Bien que je n'aie pas trouvé une association entre la pathologie alimentaire et la tendance à se venger, j'ai trouvé qu'une restriction alimentaire plus sévère était associée avec une plus grande tendance à rejeter les autres joueurs, et ce indépendamment des votes des autres joueurs à leur égard. Ceci suggère que les individus pratiquant la restriction alimentaire repoussent les autres indépendamment de comment les autres les traitent. J'ai théorisé que les individus pratiquant la restriction alimentaire sont potentiellement plus sensibles à la possibilité d'être rejetés et cherchent à devancer le rejet en rejetant les autres.

Dans l'Article 2, j'ai élaboré sur ces résultats en étudiant si la sensibilité au rejet (SR), c'est-à-dire la tendance à être très sensible au rejet social, était associée avec la pathologie

alimentaire par le biais de l'augmentation du stress social. Je prévoyais que la SR serait associée aux réactions négatives d'autrui, ce qui prédirait une augmentation de la pathologie alimentaire à son tour. J'ai étudié cette question en utilisant des données longitudinales obtenues d'étudiantes de premier cycle et de femmes dans la communauté souffrant d'hyperphagie boulimique. Je n'ai trouvé aucune association indirecte entre la SR et la pathologie alimentaire via le stress social dans les deux groupes. Cependant, j'ai trouvé que la SR était directement reliée avec la pathologie alimentaire à un moment particulier, ce qui suggère que l'appréhension ou la perception du rejet pourraient être des corrélatifs plus importants de l'alimentation désordonnée que le stress social réel.

Les résultats des Articles 1 et 2 ont mis en question le rôle du stress social dans l'alimentation désordonnée. Il est possible que l'association entre le stress social et l'alimentation désordonnée s'explique mieux par des mécanismes indirects. Le but de l'Article 3 était d'étudier un nouveau mécanisme indirect par lequel le stress social pourrait influencer la pathologie alimentaire. Le stress interpersonnel chronique peut mener à la difficulté d'éprouver du plaisir dans les relations interpersonnelles et les individus pourraient alors chercher ailleurs des sources de récompense (par exemple, les aliments ou l'apparence personnelle). Dans l'Article 3, j'ai étudié si l'ostracisme et la victimisation par les pairs étaient indirectement reliés avec la pathologie alimentaire par le biais de l'anhédonie sociale. Ayant recours à des données longitudinales, je n'ai trouvé aucune association indirecte entre le stress social et la pathologie alimentaire par le biais de l'anhédonie sociale. Cependant, j'ai trouvé que la victimisation (et non l'ostracisme) était directement reliée avec des changements de la pathologie alimentaire au fil du temps, suggérant qu'un historique de victimisation est relié à une détérioration diachronique de la pathologie alimentaire. Dans l'ensemble, mes résultats suggèrent le besoin de réexaminer le rôle du stress interpersonnel dans la pathologie alimentaire et d'indiquer que certains facteurs tels que la SR sont potentiellement des corrélatifs plus pertinents et de meilleures cibles d'intervention. De plus, mes résultats soulignent l'importance d'étudier les différentes formes de stress social et suggèrent que la victimisation pourrait être plus prédictive que l'ostracisme d'une détérioration de l'alimentation désordonnée au fil du temps.

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Contributions of Authors

I am the primary author on all three articles included in this dissertation. For Article 1, I was responsible for contributing to the design of the larger study from which my data were collected, conceptualizing the research question explored in Article 1, data cleaning and analysis, preparing the introduction, results, discussion, and figures for the manuscript, and making necessary revisions during the review process. Iulia Banica (second author) was responsible for contributing to the design of the larger study, administrative duties related to running the larger study, and drafting the method section of the manuscript. Dr. Anna Weinberg (third author), the principal investigator of the larger study, offered input on my research question and analytical approach and reviewed each iteration of the manuscript prior to submission/re-submission. Dr. Sarah Racine (fourth author) was responsible for supervising all aspects of the study, from inception to manuscript preparation and revision. All authors read, edited, and approved the final version of the manuscript published in the *International Journal of Eating Disorders*.

For Article 2, I designed and conducted the study from which the undergraduate data were collected. The clinical participant data were obtained from a larger study, for which Dr. Sarah Racine was the primary investigator. That study was conducted by a large team of graduate and undergraduate students, including myself. I conceptualized the research question examined in the manuscript, analyzed the data from both samples, and prepared the manuscript for publication. Dr. Sarah Racine supervised and provided input on all aspects of the study and manuscript preparation and approved the final version currently under revision at *Appetite*.

I was responsible for all aspects of Article 3, including study conceptualization and administration, data collection and analysis, and manuscript preparation. Dr. Sarah Racine

provided supervision and input for the entire process, from study inception to manuscript preparation.

Original Contributions to Knowledge

In Article 1 of my dissertation, I examined associations between core eating disorder symptoms and the tendency to retaliate in response to interpersonal rejection by peers during competition. To my knowledge, this was the first study to examine how disordered eating is associated with interpersonal behaviour in response to rejection. Results of this study indicated that, while eating pathology was not associated with undergraduate women's tendency to reject coplayers who previously rejected them, participants higher in dietary restriction were more likely to reject coplayers *overall* (i.e., regardless of how coplayers voted). This finding contributes to the larger body of literature on interpersonal functioning in EDs by highlighting a maladaptive behavioural tendency associated with dietary restriction – the tendency to push others away – that may contribute to the deterioration of interpersonal relationships.

In Article 2, I examined whether the trait of RS was indirectly associated with eating pathology via increased peer stress. Specifically, in samples of undergraduate women and women with binge eating, I sought to investigate whether RS was associated with greater lifetime history of ostracism and peer victimization, and whether these forms of peer stress were then associated with increased eating pathology both concurrently and across time. I did not find indirect associations between RS and eating pathology via peer stress in either sample. However, I did find that RS was *directly* associated with concurrent binge eating and weight/shape concerns. These findings contribute to the literature by suggesting that, in relation to eating pathology, actual experiences of peer stress might not be as important as individuals' tendency to be overly attuned and sensitive to the possibility of social threat.

The aim of Article 3 was to examine whether peer stress was indirectly associated with eating pathology via decreases responsiveness to social reward. I aimed to contribute to the

literature on interpersonal stress in EDs by considering whether a lifetime history of ostracism and victimization were associated with increased social anhedonia, and whether social anhedonia was subsequently associated with compensatory reward seeking via binge eating and dietary restriction. Further, I examined whether these indirect associations would be stronger in individuals who held positive beliefs about eating and having a favourable appearance. While my hypotheses were not supported, my findings revealed that ostracism and victimization might be associated with different negative outcomes. I found that ostracism, but not victimization, was associated with higher levels of social anhedonia, but increased social anhedonia was not subsequently related to disordered eating. Conversely, victimization was not associated with social anhedonia but was directly associated with increases in eating pathology across the first semester of university. These findings inform future research by highlighting the importance of examining distinct forms of interpersonal stress in the context of disordered eating and by identifying peer victimization as a particularly impactful form of peer stress that may represent a risk factor for chronicity of eating pathology. Furthermore, to my knowledge, this was the first study to examine social anhedonia in relation to continuous measures of disordered eating symptoms – specifically, binge eating and dietary restriction – rather than diagnostic categories.

General Introduction

Eating disorders (EDs) are debilitating and costly psychological disorders (Simon et al., 2005). These disorders are associated with significant morbidity and mortality (Jon Arcelus et al., 2011; Franko & Keel, 2006), and the economic burden of EDs is estimated to be substantial (Simon et al., 2005). Without proper treatment, many individuals with EDs develop chronic, recurrent courses, with the burden of disability increasing over time. This suggests a need to intervene prior to the onset of these disorders or soon after their development. However, the causes and maintaining factors of EDs are currently not well understood, and even the best existing treatments for EDs are only moderately effective (Wilson et al., 2007). Identification of vulnerable individuals and the development of more effective individualized treatment approaches depend on our ability to identify factors that precede and maintain disordered eating.

One factor that appears to be particularly relevant to eating pathology is interpersonal stress. Interpersonal difficulties have consistently been shown to influence cognitive, emotional, and behavioural symptoms of EDs (Monteleone et al., 2018), although the mechanisms by which interpersonal stress influences disordered eating are not fully understood. Additionally, it is thought that the association between interpersonal stress and ED symptoms is bidirectional. Not only do interpersonal stress experiences predict later disordered eating, but it is hypothesized that both ED symptoms and maladaptive interpersonal styles characteristic of individuals with EDs, can, in turn, elicit negative evaluations from others (Arcelus et al., 2013; Nielsen & Bará-Carril, 2003; Rieger et al., 2010; Schmidt & Treasure, 2006). This can create a cycle of increasing interpersonal problems and worsening ED symptoms. Research on the influence of ED symptoms on interpersonal stress is scarce, however, and relies primarily on self-report data. Elucidating the mechanisms by which interpersonal stress and ED symptoms influence one

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another over time may contribute to improved treatment efficacy, particularly since interpersonal stress is associated with poorer treatment response in individuals with EDs (Agras et al., 2000; Hartmann et al., 2010).

The research studies described in this dissertation aimed to identify potential mechanisms for the development and maintenance of eating pathology by investigating individual characteristics that may influence the bidirectional relationship between interpersonal stress and eating pathology. Across three studies using varied methodologies and populations, I examined the following: 1) whether core symptoms of EDs are associated with maladaptive behavioural responses to negative social evaluation, 2) whether the trait of rejection sensitivity influences eating pathology by way of increased social stress, and 3) whether altered social reward responsiveness explains the association between interpersonal stress and disordered eating.

Literature Review

Eating Disorders

Categorical Definitions

As defined in the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5), feeding and eating disorders are characterized by chronic disturbances in eating and/or eating-related behaviours that affect the consumption or absorption of food and lead to impairments in physical or psychosocial functioning (American Psychiatric Association, 2013). Of the eight feeding and eating disorders included in the DSM-5, only the five classified as EDs - anorexia nervosa (AN), bulimia nervosa (BN), binge eating disorder (BED), other specified feeding and eating disorder (OSFED) and unspecified feeding and eating disorder (UFED) – will be described for the purpose of this dissertation. AN is characterized by significant caloric restriction resulting in abnormally low weight, fear of weight gain and/or deliberate attempts to prevent weight gain, and disturbances to the perception of one's own body weight or shape. Additionally, there is excessive influence of body weight or shape on self-evaluation and/or lack of recognition of the impact of low body weight. BN is defined by recurrent episodes of binge eating (i.e., eating abnormally large amounts of food within a short amount of time, accompanied by a feeling of lack of control over eating) and compensatory behaviours aimed at preventing weight gain (e.g., self-induced vomiting, excessive exercise) occurring at least once per week for three months. Similar to AN, the self-evaluation of individuals with BN is overly influenced by body weight/shape. BED is characterized by recurrent episodes of binge eating, occurring at least once per week for three months, in the absence of regular compensatory behaviours. Binge eating episodes cause significant distress and are associated with features such as nonhomeostatic eating (i.e., abnormally rapid eating, eating far past satiety, or eating when not

physically hungry), eating in secret due to embarrassment, and/or feelings of disgust, depression, or guilt following the episode. OSFED and UFED were added to the *DSM-5* to replace the *DSM-IV* diagnosis of eating disorder not otherwise specified (EDNOS). A diagnosis of OSFED is given in cases where symptoms are similar to those of the above EDs and cause clinically significant distress and/or impairment, but do not meet full diagnostic criteria. Examples include atypical AN (all criteria for AN are met except for low weight), BN or BED of low frequency/duration (all criteria are met except binge eating and/or compensatory behaviours occur less than once per week and/or for less than three months), and purging disorder (recurrent use of purging behaviour to control weight/shape without binge eating). A diagnosis of UFED applies in cases where symptoms of a feeding and eating disorder are present and cause clinically significant distress or impairment, but individuals do not fit into a main ED or OSFED category.

Dimensional Approach

While the diagnostic classification systems, such as the DSM, describe EDs as being discrete disorders, there is evidence that a dimensional approach may more accurately reflect the nature of EDs (Williamson et al., 2005). Evidence suggesting that a categorical approach may not be ideal includes the high rate of cross-over among ED diagnostic categories (Allen et al., 2013; Steinhausen & Weber, 2009; Tozzi et al., 2005), the similarity in clinical impairment across individuals with full and sub-threshold EDs (Fairweather-Schmidt & Wade, 2014; Mangweth-Matzek et al., 2014; Wade & O'Shea, 2015), and the fact that the most commonly diagnosed ED is OSFED (40-60% of cases; Keel et al., 2011; Stice et al., 2013).

Taxometric analyses, designed to examine whether a given construct is categorical or dimensional, have shown that cognitive symptoms of EDs (e.g., dietary restraint, body image concerns, drive for thinness) are dimensional – that is, they exist along a continuum with

normality (for reviews, see Gordon et al., 2007; Williamson et al., 2005). However, findings are less clear when examining behavioural symptoms of EDs, such as binge eating and dietary restriction. Some studies have shown that EDs characterized by binge eating are qualitatively different from those characterized by dietary restriction and do not exist along a continuum with normative behaviour (Gordon et al., 2007; Williamson et al., 2005), although one taxometric study found that EDs characterized by binge eating and dietary restriction may in fact exist along a common continuum (Olatunji et al., 2012).

Recent studies by Forbush and colleagues examined the utility of a novel dimensional model of EDs comprised of two broad internalizing factors – distress and fear/avoidance – under which were nested several ED symptoms and associated features (Forbush et al., 2017, 2018). Specific ED symptoms included were body dissatisfaction and binge eating (nested under distress) and restricting, purging, dietary restraint, excessive exercise, and negative attitudes towards obesity (nested under fear/avoidance). This model was shown to account for almost 70% of the variance in ED-related impairment, as compared to DSM-5 diagnostic categories which predicted 11% of the variance (Forbush et al., 2017). The dimensional model was also a better predictor of ED outcomes (e.g., recovery, restoration of body weight, reduction in binge eating and compensatory behaviours, decreased body dissatisfaction) across six months, accounting for 60% of the variance in outcomes, as compared to DSM-5 diagnoses which accounted for 36% of the variance (Forbush et al., 2018). Taken together, these findings suggest that research investigating factors associated with EDs may benefit from measuring core symptoms of EDs (e.g., binge eating, dietary restriction, purging, weight/shape concerns) continuously rather than relying on diagnostic categories.

Prevalence

Prevalence refers to the percentage of the population that meets diagnostic criteria for a specified disorder during a defined period of time (e.g., lifetime, 12-month). A recent systematic review concluded that EDs are on the rise, with the global point prevalence (i.e., at a specific moment rather than during a defined length of time) of all EDs estimated to have increased from 3.5% to 7.8% between 2000 and 2018 (Galmiche et al., 2019). Current lifetime prevalence rates estimated in two recent systematic reviews ranged from 2.6% to 8.4% in women and 0.7% to 2.2% for men (Galmiche et al., 2019; Qian et al., 2022). With regards to specific diagnoses, lifetime prevalence rates were estimated to be 0.6% to 1.4% in women and 0.04% to 0.2% in men for AN, 1.2% to 1.9% in women and 0.4% to 0.6% in men for BN, 2.4% to 2.8% in women and 1.0% to 1.2% in men for BED, and 4.3% in women and 3.6% in men for EDNOS. Point prevalence estimates are 2.8% in women and 0.3% in men for AN, 1.5% in women and 0.1% in men in BN, 2.3% in women and 0.3% in men for BED, and 10.1% in women and 0.9% in men for EDNOS (Galmiche et al., 2019). Taken together, these estimates suggest that EDs are far more common in women and that not otherwise specified diagnoses account for the majority of cases.

Cross-culturally, EDs appear to be most common in Western countries, with prevalence rates approximately 8.5 times higher than non-Western countries for all EDs combined (Qian et al., 2022). However, prevalence rates do appear to be increasing in non-Western countries and ethnic minority populations, which is thought to be due, in part, to globalization and exposure to Western media (Becker et al., 2011; Eddy et al., 2007; Nasser, 2009). In the US, BN is in fact more prevalent in Latinos and African Americans, compared to non-Latino whites, whereas rates of AN and BED have been found to be similar (Marques et al., 2011).

Studies investigating the epidemiology of EDs in LGBTQ+ populations are scarce; in particular, those involving individuals who are transgender or gender non-conforming. Based on available data, rates of EDs in gay and bisexual men appear to be 10 times higher than those in straight men (Strong et al., 2000), whereas the literature on rates in lesbian and bisexual women is less clear. Some studies show that lesbian and bisexual women have lower levels of eating pathology compared to straight women (Lakkis et al., 1999; Strong et al., 2000), whereas others have not found any differences between groups (Feldman & Meyer, 2007; Heffernan, 1996), and some have found higher rates among lesbian and bisexual women (Davids & Green, 2011; Hadland et al., 2014; Heffernan, 1996; Jones et al., 2019). To date, there do not appear to be any studies of prevalence or incidence in transgender or non-binary individuals, although there is modest evidence that there may be an association between gender-identity disturbances and eating pathology (Khoosal et al., 2009; Silverstein et al., 1990; Witcomb et al., 2015).

As evidenced by the preceding prevalence estimates, full-threshold EDs are relatively uncommon in the general population. However, rates of core behavioural and cognitive eating disorder symptoms are estimated to be significantly higher. Across various samples ranging from early adolescence to mid-adulthood, binge eating has been reported by 8.4% to 31.1% of women and 1.7% to 26.8% of men (Croll et al., 2002; Neumark-Sztainer et al., 2011; Simone et al., 2022). Notably, US studies find that binge eating is most prevalent among Hispanic/Latino individuals (Croll et al., 2002; Neumark-Sztainer et al., 2011; Simone et al., 2022). Dietary restriction has been reported by 17.9% to 42.6% of women and 14.0% of men, with the highest rates found among American Indian and White women (Croll et al., 2002; Reba-Harrelson et al., 2009). Other extreme weight control behaviours, such as vomiting and use of laxatives and diet pills, have been reported by 1.9% to 40.2% of women and 1.6% to 7.3% of men and are most frequent among American Indian and White women (Croll et al., 2002; Neumark-Sztainer et al., 2011; Reba-Harrelson et al., 2009). Concerns about weight and shape, a core cognitive component of EDs, appear to be present in a majority of women (74.5%; Reba-Harrelson et al., 2009) and 2.7% to 10.0% of men (Glazer et al., 2021) and are most common among White and Asian women (Reba-Harrelson et al., 2009).

Age of Onset and Chronicity

Age of onset and chronicity of EDs is difficult to specify due to issues defining when onset happens (e.g., at first appearance of symptoms vs. when full diagnostic criteria are met) and high rates of migration between diagnostic categories (Allen et al., 2013; Anderluh et al., 2009; Castellini et al., 2011; Steinhausen & Weber, 2009; Tozzi et al., 2005). Across several studies, estimates for the age of onset of EDs, defined as meeting full diagnostic criteria, range from 17.8 to 18.9 for AN, 19.6 to 21.2 for BN, 23.2 to 25.7 for BED, and from 20.0 to 23.1 for OSFED and subthreshold BED (Favaro et al., 2018; Hudson et al., 2007; Mohler-Kuo et al., 2016). Notably, one study of a large sample of adolescents showed much lower ages of onset for all EDs – 12.3 for AN, 12.4 for BN, 12.6 for BED and subthreshold BED (Swanson et al., 2011). The average duration of an EDs across all diagnostic categories has been estimated to be eight years (Fairburn et al., 2009, 2015). However, average duration appears to vary depending on diagnosis, with estimates of the duration of single episodes to be 1.7 years for AN, 8.3 years for BN, and 8.1 years for BED (Hudson et al., 2007). Rates of recovery also vary across diagnoses. A study examining long-term recovery rates of AN and BN found that, at nine years after initial assessment, 31.4% of individuals with AN and 68.2% of individuals with BN were considered to have recovered, whereas rates at a 22-year follow-up were similar (62.8% for AN and 68.2% for BN; Eddy et al., 2017). Other studies have found that 46.9% of individuals with AN eventually

fully recover, while 33.5% may show partial improvement, compared to 66.6% and 28.4%, respectively, in BN (Steinhausen, 2002; Steinhausen & Weber, 2009). A meta-analysis examining rates of abstinence from binge eating in individuals receiving psychological treatment for BED found rates of abstinence to range from 42.3% to 50.9% at post treatment and follow-up (Linardon, 2018), whereas another review found a wider range of recovery, from 19% to 65% (Smink et al., 2013).

Comorbidity

Individuals with EDs often meet criteria for at least one comorbid psychiatric disorder. Findings from the National Comorbidity Replication Survey indicate that 56.2% of individuals with AN, 94.5% of individuals with BN, and 78.9% of individuals with BED meet criteria for another DSM diagnosis at some point in their lives (Hudson et al., 2007). The most common lifetime comorbidities appear to be depression (39.1% in AN, 50.1% in BN, and 32.3 in BED), anxiety disorders (47.9% in AN, 80.6% in BN, 65.1% in BED, most commonly specific phobia and social phobia), impulse control disorders (30.8% in AN, 63.8% in BN, and 43.3% in BED, most commonly ADHD in adults and oppositional defiant disorder in adolescents), and substance use (27.0% in AN, 36.8% in BN, 23.3% in BED, most commonly abused substances are tobacco, caffeine, and alcohol; Bahji et al., 2019; Hudson et al., 2007; Swanson et al., 2011). Other comorbidities identified in the National Comorbidity Replication Survey include bipolar disorder (3.0 - 17.7% across all EDs), obsessive compulsive disorder (0.0% - 17.4%), and posttraumatic stress disorder (12.0 - 45.4%; Hudson et al., 2007). Two classes of psychiatric conditions that were not examined in the National Comorbidity Replication Survey, but that appear to be highly comorbid with EDs are personality disorders (PDs) and autism spectrum disorder (ASD). A meta-analysis examining comorbid PDs in individuals with AN and BN found that, on average, roughly 50% of individuals with AN or BN also met criteria for a PD. The most common comorbid PDs were borderline personality disorder (19% of individuals with AN and 25% of individuals with BN) and avoidant personality disorder (23% of individuals with AN and 20% of individuals with BN). Individuals with different ED diagnoses did not differ significantly in their rates of comorbid PDs with the exception of OCPD, which was higher among individuals with AN (23%) versus BN (12%; Martinussen et al., 2017). Rates of ASD in individuals with EDs have been found to range from 8% to 37%, which is significantly higher than the rate of ASD in the general population (approximately 1% in adults; Brugha et al., 2011; Huke et al., 2013). The majority of studies have examined the co-occurrence between AN and ASD, with rates varying widely from 4.0 to 52.5% (Carpita et al., 2022), but the prevalence of ASD has also been found to be elevated in individuals with BN and BED (Karjalainen et al., 2016; Wentz & Lacey, 2005).

Impact

EDs are associated with significant negative consequences and impairment across multiples life domains:

Health. Individuals with EDs often have medical complications implicating the gastrointestinal, neurological, and cardiovascular systems that directly result from their EDs (e.g., from extreme low weight or frequent binge eating and/or purging; Mitchell & Crow, 2006). Medical complications from AN can be particularly severe, as evidenced by the fact that AN is the psychiatric disorder with the highest mortality rate, with the majority of deaths due to causes directly related to the illness rather than suicide (Arcelus et al., 2011).

Psychological Functioning. As outlined in the section on comorbidity, the majority of individuals with an ED will meet criteria for at least one other psychiatric disorder, with many

meeting criteria for two or three additional disorders (Hudson et al., 2007). Furthermore, individuals with EDs have elevated rates of suicide (Bulik et al., 2008; Zerwas et al., 2015), with the highest risk appearing to be among individuals with AN, followed with those with BN, whereas adults with BED appear to be at relatively low risk for suicide (Preti et al., 2011).

Occupational Functioning. Compared to women without EDs, women with AN and BN show poorer adjustment at work (Harrison et al., 2014), and those with EDs characterized by binge eating show poorer occupational functioning across multiple domains (Kessler et al., 2014; Ling et al., 2017; Striegel et al., 2012).

Interpersonal Functioning and Relationships. Social dysfunction has been well documented in individuals with EDs across multiple types of relationships (e.g., family, peers; (Arcelus et al., 2013; Swanson et al., 2011), as will be elaborated upon later in this literature review. Some of the strain on interpersonal relationships may come from the impact of individuals' ED symptoms on others: close others (e.g., caregivers, family members) have been found to experience increased psychological distress, decreased quality of life, and increased marital dysfunction as a result of their loved one's ED (Anastasiadou et al., 2014; Espina et al., 2003; Norre et al., 2006; Zabala et al., 2009).

Societal Burden. EDs are associated with significant healthcare utilization and economic burden (Agh et al., 2015; Simon et al., 2005; Streatfeild et al., 2021). Although it can be difficult to quantify the exact cost to society, a recent study estimated that, in the US, the total cost of EDs is estimated at 64.7 billion, with EDNOS contributing to 35% of the cost, followed by BED (30%), BN (18%), and AN (17%). Furthermore, costs associated with reduced wellbeing and functioning were estimated to be an additional 326.5 billion per year (Streatfeild et al., 2021).

As shown throughout the preceding sections, core ED symptoms such as binge eating, weight-control behaviours (e.g., dietary restriction, compensatory behaviours), and concerns about weight and shape are prevalent in the general population, despite relatively low rates of diagnosable EDs. Core ED symptoms are reported by individuals of diverse ethnic/racial groups and are substantially more prevalent in women, compared to men (Croll et al., 2002; Glazer et al., 2021; Neumark-Sztainer et al., 2011; Reba-Harrelson et al., 2009; Simone et al., 2022). EDs typically begin in adolescence and early adulthood (Favaro et al., 2018; Hudson et al., 2007; Mohler-Kuo et al., 2016), follow a chronic course with modest treatment outcomes (Eddy et al., 2017; Fairburn et al., 2009, 2015; Linardon, 2018; Smink et al., 2013; Steinhausen, 2002; Steinhausen & Weber, 2009), and are associated with a wide range of comorbidities and negative outcomes (Arcelus et al., 2013; Hudson et al., 2007; Mitchell & Crow, 2006; Swanson et al., 2011). As such, research in the field of disordered eating would likely benefit from examining specific symptoms of EDs, rather than diagnostic groups, in diverse samples of women. Focusing on disordered eating processes early in the development of symptoms may be helpful in informing efforts aimed at mitigating symptom chronicity and associated outcomes.

Interpersonal Functioning in Individuals with Disordered Eating

Individuals with EDs experience elevated interpersonal stress, both prior to and during their illness. Interpersonal dysfunction has been observed across multiple domains of life, with research showing that individuals with EDs or disordered eating experience elevated difficulties in relationships with family members (Tetley et al., 2014), romantic partners (Arcelus et al., 2012), and peers (e.g., Day et al., 2021; Lee & Vaillancourt, 2019). Individuals with EDs retrospectively report social difficulties beginning in childhood that predate the onset of illness. Specifically, individuals report having limited social interactions and few close friends, spending

significant time engaged in solitary activities, and experiencing bullying (Cardi et al., 2018; Fairburn et al., 1997, 1998, 1999; Krug et al., 2012). During the acute phase of illness, individuals report significant social isolation and lack of social support from both family and peers (Brochu et al., 2018; Cardi et al., 2018; Grissett & Norvell, 1992; Tiller et al., 1997; Troop et al., 1997).

Not only do women with EDs report having poor social function, but interpersonal stress is associated with the frequency and severity of eating pathology across diagnostic categories. In a large sample of women with various EDs, social difficulties were associated with higher levels of concurrent ED psychopathology (i.e., dietary restraint and eating, shape, and weight concerns) across diagnostic categories (Ivanova, Tasca, Proulx, et al., 2015). In a cross-sectional study of women with BED, interpersonal stress was associated more frequent binge eating episodes, increased dietary restraint, and greater eating, shape, and weight concerns (Ivanova, Tasca, Hammond, et al., 2015). Using an experience sampling method in women with BN, Steiger and colleagues found that binge eating episodes tended to be preceded by social interactions perceived to be negative (Steiger et al., 1999).

Associations between interpersonal stress and increased eating pathology have also been demonstrated in non-clinical samples. For example, in a sample of adult women from the community, interpersonal problems were concurrently associated with more frequent binge eating episodes and loss of control over eating, as well as greater dietary restraint and eating, shape, and weight concerns (Ansell et al., 2012). In college women, low perceived social support was related to greater eating pathology (Jackson et al., 2005), and interpersonal stressors related to friendships and romantic relationships predicted increases in dietary restraint across time (Cain et al., 2010). One study in children found that social difficulties were concurrently

associated with increased loss of control eating, suggesting that interpersonal stress may impact disordered eating beginning at an early age (Elliott et al., 2010).

The association between interpersonal stress and eating pathology is thought to be bidirectional. That is, not only is interpersonal stress expected to lead to increased eating pathology, but it is hypothesized that disordered eating symptoms then further exacerbate interpersonal difficulties. In individuals with AN, starvation can lead to difficulties in social cognition, such as difficulties with nonverbal communication and emotional reciprocity (Cardi et al., 2015; Davies et al., 2016), which can lead to aversive reactions from others and subsequent interpersonal rejection (Schneider et al., 2013; Szczurek et al., 2012). Furthermore, the severity of symptoms can lead to anxiety, criticism, and hostility from others (Anastasiadou et al., 2014; Treasure et al., 2009), which may lead to the deterioration of relationships. There is also qualitative evidence that ED symptoms lead to worsening interpersonal functioning across diagnostic categories. A sample of women with various EDs were asked to describe difficulties with their transition to university, and qualitative analysis identified a theme of ED symptoms leading to social isolation on campus (Goldschen et al., 2019). Evidence of the detrimental effect of ED symptoms on interpersonal functioning has also been obtained from non-clinical samples. Among adolescents, disordered eating behaviours were found to prospectively predict instances of bullying one year later (Lee & Vaillancourt, 2019). Further, in a sample of undergraduate students, dietary restraint prospectively predicted negative life events, including those of a personal nature (Dodd et al., 2014).

Influence of Interpersonal Stress on Treatment Outcome

Interpersonal stress appears to be related to poor ED treatment outcomes. According to a systematic review by Jones and colleagues (2015), pre-treatment interpersonal problems are

often associated with poorer treatment response in individuals with AN, BN, and EDNOS. Specifically, some studies have found evidence that general interpersonal problems, social avoidance, social phobia/anxiety, poor social adjustment, problems with dominance, quality of social relationships and caretaker expressed emotion are associated with poorer treatment outcomes such as minimal change in symptoms, treatment dropout, and relapse. Notably, however, a small number of studies found no effect of pre-treatment interpersonal functioning on treatment outcomes (for review, see Jones et al., 2015). These findings suggest that, even in therapies in which interpersonal functioning is not the main target, adjunct modules to address interpersonal difficulties may offer additional benefit.

Peer-Related Stressors

Given that the age of onset of most eating disorders is during adolescence or early adulthood (Favaro et al., 2018; Hudson et al., 2007; Mohler-Kuo et al., 2016; Swanson et al., 2011), a time during which peer relationships may be particularly important and influential, research has examined associations between specific forms of peer stress, particularly victimization and social exclusion, and disordered eating.

Peer Victimization. Victimization by peers can take several forms, including bullying, manipulation, and verbal or physical aggression. Several reviews of the literature have highlighted an association between peer victimization and disordered eating. One recent systematic review found that, among adolescents, both non-specific and weight-related teasing and bullying were associated with concurrent binge eating, dietary restraint, unhealthy weight control behaviours, body image disturbances, and global eating pathology (Day et al., 2021). Non-specific victimization was additionally associated with dietary restriction. Notably, these associations were primarily observed cross-sectionally, whereas longitudinal associations were

less consistent. Another recent study also failed to find consistent longitudinal associations between victimization and eating pathology - in a large sample of adolescents, peer victimization was associated with weight and shape concerns concurrently, but not one year later (Trompeter et al., 2022). Finally, in emerging adults, a history of childhood victimization was associated with current dietary restriction and binge eating (Markou et al., 2021). Taken together, these findings suggest that peer victimization may play a role in eating pathology, although the nature of that role is difficult to elucidate given inconsistent findings when examining longitudinal associations and a lack of investigation of potential pathways via which victimization might impact disordered eating.

Social Exclusion. Experiences of being ostracized (ignored or excluded) or rejected by peers have been shown to be associated with eating pathology, primarily using experimental methodologies. In the one study using self-report data, college women were asked to report on their daily social interactions, and it was found that daily experiences of rejection were associated with increased dietary restriction on the same day (Beekman et al., 2017). Experimental studies using social exclusion paradigms have consistently found associations between being excluded and maladaptive eating outcomes. Specifically, ostracism has been found to lead to increased motivation to earn food in adolescents (Salvy et al., 2012) and increased consumption of palatable food in undergraduate students (Baumeister et al., 2005; Oaten et al., 2008) and adult women (Hayman et al., 2015). Two of these studies also identified potential factors that might moderate the association between social exclusion and unhealthy eating. First, Hayman and colleagues (2015) demonstrated that black women who were ostracised by white women ate more potato chips than black women who were ostracised by other black women, suggesting that perceptions of discrimination might influence the association

between social exclusion and eating behaviour. Second, in a sample of undergraduate students, those who were ostracised ate more cookies than students who were not ostracised. However, this effect only persisted across time for students who were more socially anxious, suggesting that anxiety about being excluded may influence the extent to which actual exclusion impacts eating behaviour (Oaten et al., 2008). In summary, these findings suggest that social exclusion may play a role in disordered eating. However, most studies examining this association have relied on experimental paradigms (i.e., induced exclusion in the laboratory) and have focused on eating outcomes that may be maladaptive, but not necessarily pathological (e.g., overconsumption of palatable food). As such, more research is needed using varied methodology (i.e., self-report measures) and examining more pathological forms of eating (e.g., binge eating, dietary restriction).

Interpersonal Models of Eating Disorders.

As discussed in the preceding sections, it has been established that disordered eating is associated with interpersonal dysfunction, including two forms of peer-related stressors (social exclusion and victimization) that may be pertinent during the typical period during which eating pathology develops. However, in order to be useful in guiding prevention and treatment efforts, it is important to understand *why* such an association exists. Below is a discussion of theoretical models developed to explain how interpersonal stress and eating pathology are related to one another, along with evidence supporting each model.

Interpersonal Psychotherapy Model of Eating Disorders (IPT-ED)

IPT, originally developed to treat depression, has been used in the treatment of eating disorders for several decades and, in 2003, Wilfley and colleagues developed and tested a specific form of IPT for BN (Wilfley et al., 2003). However, there lacked a disorder-specific

rationale explaining the efficacy of IPT in treating EDs and guiding practice in ED populations (Rieger et al., 2010; Wilfley et al., 2003). Accordingly, in 2010, Rieger and colleagues developed a model to explain the role of interpersonal stressors in the maintenance of EDs and to identify appropriate targets of treatment (Rieger et al., 2010).

Originally developed to explain the association between interpersonal stress and binge eating, and later expanded to other ED symptoms, the basic IPT-ED model posits that interpersonal stress, specifically negative social evaluation, leads to low self-esteem and associated negative affect, and that ED symptoms develop as an attempt to restore sense of self and reduce negative affect (Rieger et al., 2010). Furthermore, the authors suggest that ED symptoms further exacerbate social difficulties via processes such as conflict related to eating, mood changes in the individual that elicit negative reaction from others, etc., creating a selfperpetuating cycle. As interpersonal stress increases, individuals are less likely to seek out social support to help manage their negative affect, relying on ED behaviours instead. The model also identifies several vulnerability factors thought to influence the development and maintenance of EDs, namely shape- and weight-based self-worth; positive expectancies about the outcome of eating, weight, and shape; perfectionism, and rejection sensitivity.

The basic premise of IPT-ED (i.e., that interpersonal stress influences disordered eating via negative affect) has been empirically validated in relation to binge eating in both clinical and community samples. For example, interpersonal stress has been found to be indirectly associated with concurrent binge eating and associated cognitions via negative affect in women with BED (Ivanova, Tasca, Hammond, et al., 2015). Using an experience sampling method, Steiger and colleagues (1999) found that interpersonal stressors and negative affect often preceded binge episodes in women with BN. In non-clinical samples, negative affect has been found to mediate

the association between interpersonal problems and concurrent binge eating and eating pathology (e.g., restraint, weight/shape concerns) in healthy adult women (Ansell et al., 2012), and between interpersonal problems and concurrent loss of control eating in healthy children and adolescents (Elliott et al., 2010). While the IPT-ED model has primarily been applied to binge eating, there is evidence for its validity across ED symptoms and diagnostic subtypes. In a large sample of treatment-seeking women, interpersonal problems were associated with concurrent dietary restraint and weight/shape concerns via negative affect in women with BED, BN, AN – restrictive eating subtype, and EDNOS (Ivanova, Tasca, Proulx, et al., 2015).

Interpersonal Psychotherapy for EDs. IPT developed specifically for EDs focuses on current, rather than past, functioning and circumstances across five domains: grief, interpersonal role disputes, role transitions, life goals, and interpersonal deficits (Murphy et al., 2012; Wilfley et al., 2003). The hypothesized mechanism of action is improvement in self-evaluation and self-esteem (i.e., that improved social functioning leads to more positive self-view, with in turn leads to reductions in ED symptoms). A course of therapy typically consists of 16 to 20 sessions across four to five months. Interpersonal domains identified as problematic are examined and addressed using techniques such as decision analysis, communication analysis, and role play. The therapist typically takes a non-directive stance and allows the patient to explore his or her feelings about interpersonal issues. Given that interpersonal functioning is given primacy in this form of therapy, ED symptoms are generally not assessed or discussed throughout most of the therapy.

Compared to enhanced cognitive behavioural therapy for eating disorders (CBT-E; Fairburn, 2008), the gold-standard ED treatment, IPT-ED demonstrates less efficacy at the end of treatment, although IPT-ED leads to comparable outcomes at long-term follow-ups for BN, BED, and AN (Agras et al., 2000; Carter et al., 2011; Fairburn et al., 1991, 1993; McIntosh et al., 2005; Wilfley et al., 1993, 2002). In a transdiagnostic sample of people with EDs, IPT led to lower rates of remission at the end of treatment, compared to CBT-E (33.3% vs. 65.5%, respectively; Fairburn et al., 2015). However, there was improvement in symptomatology in the IPT group across time, such that the difference in 60-week follow-up outcomes (i.e., global eating disorder symptoms) was statistically non-significant. It is notable that IPT-ED appears to be comparable to CBT-E in the long-term, given that IPT-ED does not explicitly address ED symptoms. It is thought that, because CBT-E addresses ED symptoms directly, its effects are more immediate, whereas IPT is theorized to indirectly influence ED symptoms via improvement in social functioning, explaining why the effect of IPT is only seen across a longer time frame.

Cognitive Interpersonal Maintenance Model of Anorexia Nervosa

Developed in 2006 by Schmidt and Treasure, the cognitive interpersonal maintenance model of AN draws from cognitive behavioural and evolutionary theories to identify factors involved in the maintenance (rather than development) of AN (Schmidt & Treasure, 2006). The model describes several stages of the disorder and highlights both intra- and interpersonal factors that may be implicated in the maintenance of symptoms, including perfectionism/cognitive rigidity, experiential and social avoidance, positive beliefs about AN symptoms, and reactions from close others. In 2013, the model was updated to include difficulties with set shifting and excessive attention to detail, bias towards social threat, and impaired social cognition as additional vulnerability factors (Treasure & Schmidt, 2013).

During the early stages of AN, the model posits that symptoms are maintained by an initial increase in positive affect, a sense of control that arises from successfully managing food intake, and subsequent development of positive beliefs about dietary restriction. The authors

highlight that during the early stages, individuals are unlikely to seek treatment due to a perceived lack of suffering. Furthermore, symptoms may be maintained at this stage by compliments about appearance following initial weight loss, which reinforce the motivation to restrict food intake. Later in the course of the disorder, eating becomes physically uncomfortable, leading to negative expectancies about the consequences of eating. Furthermore, individuals develop rigid rules about eating, and breaking these rules leads to negative affect and feelings of failure. Once individuals reach the point of starvation, their natural drive to eat increases and they become preoccupied with thoughts of food and eating, the effects of which are twofold. First, preoccupation with food and eating increases vigilance and rigidity in order to avoid breaking food-related rules and subsequent feelings of failure. Second, preoccupation with food and eating shifts focus away from unpleasant emotions and may come to be viewed as a helpful means of avoiding negative affect. From an interpersonal standpoint, worsening symptoms of AN may elicit care, attention, and enabling/accommodating behaviours from loved ones for some, while for others, worsening symptoms may elicit criticism and lead to social withdrawal. In both cases, the reactions from others serve to perpetuate symptoms.

Two studies have tested the validity of the basic premise of the cognitive-interpersonal maintenance model – that AN symptoms elicit reactions from close others, which then serve to perpetuate the disorder. In a sample of women with EDs characterized by dietary restriction (AN and EDNOS) and their primary carers, the cognitive-interpersonal maintenance model was partially supported (Goddard et al., 2013). Specifically, the authors found that carers' responses to ED symptoms (i.e., expressed emotion and psychological control) were associated with increased distress in both carers and patients, the latter of which was then associated with increased ED symptoms. While the Goddard et al. study did not find support for the role of

enabling and accommodating in the maintenance of AN symptoms, evidence supporting this part of the model was obtained in a sample of treatment-seeking adolescents with AN and their parents. In this sample, higher levels of accommodating behaviour by parents were associated with poorer treatment outcome, with the worst outcomes found in patients for whom both parents were accommodating of the disorder (Salerno et al., 2016).

Maudsley Model of Anorexia Nervosa Treatment for Adults (MANTRA). Based on the cognitive-interpersonal maintenance model of AN, MANTRA is a brief form of therapy (20-30 sessions) designed for both patients with AN and their caregivers (Schmidt et al., 2014). Modules are designed to target factors identified in the cognitive-interpersonal maintenance model of AN. Specifically, separate modules exist to target emotion regulation, issues

surrounding social identity, thinking styles, and perfectionism.

Compared to supportive therapy, MANTRA appears to lead to comparable weight restoration in individuals with AN at end of treatment (Schmidt et al., 2015). At 6- and 12month follow ups, MANTRA produces more favourable outcomes, with 50% of patients in the MANTRA group considered recovered compared to 14% in supportive therapy group (Schmidt et al., 2016). Additionally, MANTRA has been rated as more acceptable and credible than supportive therapy (Allen et al., 2016). Notably, the effectiveness of MANTRA has only been examined in individuals with AN and has not been compared to well-established treatments (e.g., CBT-E and IPT-ED).

Interpersonal Maintenance Model of Eating Disorders

In 2013, Arcelus and colleagues conducted a systematic review on interpersonal functioning in individuals with EDs and identified key areas of interpersonal functioning that may contribute to ED pathology; namely, difficulties with interpersonal problem-solving,

negative attitudes towards emotional expression, fear of negative evaluation, fear of intimacy/interpersonal distrust, and negative social comparisons (Arcelus et al., 2013). Based on their findings, they developed a model to explain how interpersonal difficulties maintain disordered eating symptoms.

The general model posits that interpersonal sensitivity, to which individuals with EDs are thought to be predisposed, contributes to low self-esteem and subsequent social anxiety. Social anxiety then leads individuals to be more socially inhibited, thus limiting the availability of social support. The central tenet of the model is that this lack of social support is what maintains disordered eating. The authors expand the model by describing unique variations of these pathways in individuals with eating pathology characterized by dietary restriction versus binge eating/purging symptoms. In individuals with dietary restriction, fear of negative evaluation, social comparison, and negative attitudes towards emotional expression are thought to interact to influence interpersonal sensitivity, self-esteem, and social anxiety. In individuals with binge eating/purging symptoms, interpersonal sensitivity, self-esteem, and social anxiety are instead hypothesized to be influenced by interpersonal distrust, fear of intimacy, lack of effective interpersonal problem-solving skills, and conflict with others.

To date, only one study appears to have directly examined the validity of the interpersonal maintenance model of EDs. Specifically, this study sought to test one of the key assumptions of the model – that interpersonal difficulties would differ across types of eating pathology – in a sample of individuals with AN, BN, and EDNOS (Raykos et al., 2014). Results partially supported the interpersonal maintenance model: individuals with AN had more difficulty with socialization and competitiveness/assertiveness, compared with individuals with BN and EDNOS, although there were no differences with regards to problems with

independence. Additionally, while individuals with BN reported more aggressiveness compared to those with AN, average levels of aggressiveness were normative in the BN sample, indicating that they are not, in fact, aggressive, as predicted by the interpersonal maintenance model.

Interpersonal Characteristics of Individuals with Disordered Eating

Although there are numerous studies examining personality traits and interpersonal tendencies of individuals with EDs (for a review, see Arcelus et al., 2013), two specific traits that have implications for interpersonal functioning will be described for the purpose of this dissertation: rejection sensitivity and social anhedonia.

Rejection Sensitivity

Rejection sensitivity (RS) is defined as the tendency to anxiously expect, readily perceive, and overreact to real or perceived rejection (Downey & Feldman, 1996). RS is thought to arise in response to early experiences of rejection by close others: when children are repeatedly rejected by people who are important to them, such as family members and peers, they may come to expect that such experiences will continue to happen and become hypervigilant to cues of social threat (Downey & Feldman, 1996; Pietrzak et al., 2005). RS has been shown to be more prevalent in women compared to men, younger versus older adults, and sexual minorities compared to heterosexual individuals (Maoilatesi et al., 2022). In adolescence and adulthood, RS has been found to be associated with a range of concurrent difficulties, such as depression, anxiety, borderline personality disorder, body dysmorphic disorder, loneliness, social withdrawal, victimization, aggression, and decreased functioning in romantic relationships (Downey & Feldman, 1996; Gao et al., 2017, 2021; Garthe et al., 2020; Watson & Nesdale, 2012). RS also predicts increases in depression, anxiety, loneliness, social withdrawal, victimization, and aggression across time (Gao et al., 2017, 2021; Gardner et al., 2020).

As outlined in the description of interpersonal models of EDs in the preceding section, RS, along with related constructs such as interpersonal sensitivity and fear of negative evaluation, has been hypothesized to be a risk factor for the development and maintenance of eating pathology. In support of these models, and as outlined in further detail in Manuscript 2, elevated RS has been found in individuals with EDs compared to healthy controls, as evidenced by attentional biases towards rejecting faces, the tendency to form negative evaluations of ambiguous social situations, and greater negative affect in response to social threat (Cardi et al., 2013, 2017; Monteleone et al., 2018; Rowlands et al., 2021). Furthermore, there is evidence that RS may be trait-like, rather than a consequence of disordered eating symptoms. In a sample of women with AN, women recovered from AN, and healthy controls, biases to social threat were similar in women with acute and recovered AN, compared to healthy controls (Harrison et al., 2010). In non-clinical samples, RS and related constructs have been found to be associated with concurrent disordered eating cognitions and behaviours (Atlas, 2004; De Paoli, Fuller-Tyszkiewicz, Halliwell, et al., 2017; Gilbert & Meyer, 2005) as well as increases in ED cognitions (e.g., cognitive restraint, weight/shape concerns) and body dissatisfaction across time (Bondü et al., 2020; Gilbert & Meyer, 2005).

Social Anhedonia

Social anhedonia refers to a trait-like (i.e., relatively stable) lack of interest or pleasure in social activities and connections. Across diverse populations, social anhedonia has been found to be associated with numerous negative outcomes related to psychological and social functioning. For example, social anhedonia has been linked to increased negative affect and decreased positive affect (Blanchard et al., 2011; Brown et al., 2007; Gooding et al., 2002) as well as decreased social support, poor social skills, and increased family conflict (Blanchard et al., 2011;

Llerena et al., 2012). Furthermore, social anhedonia has been associated with psychotic disorders, depression, post-traumatic stress disorder, autism spectrum disorder, and EDs (for review, see Barkus & Badcock, 2019).

Unlike RS, social anhedonia is not included in any of the major interpersonal models of EDs described in the preceding section. This is somewhat surprising given that general anhedonia has been found to be elevated across all ED subtypes (for review, see Dolan et al., 2022). Nonetheless, as outlined in Manuscript 3, a small body of research has examined whether individuals with EDs have deficits in social hedonic processing. Compared to healthy controls, individuals with EDs self-report higher levels of social anhedonia, with scores comparable to those of individuals with schizophrenia and depression (Deborde et al., 2006; Harrison et al., 2014; Tchanturia et al., 2012). Furthermore, in laboratory studies using an econometric choice task and eye-tracking, women with AN have been found to not find faces rewarding, and women with both AN and BN show attentional biases away from accepting faces (Cardi et al., 2013; Watson et al., 2010). In women with AN and BN, social anhedonia has been found to positively correlate with drive for thinness and fear of swallowing a sucrose solution (Eiber et al., 2002).

While social anhedonia appears to be associated with eating pathology, the direction of this association is unclear due to a lack of longitudinal investigation. Although there is evidence that the effects of disordered eating, particularly dietary restriction, can lead to social anhedonia (Keys et al., 1950), there is some theoretical discussion and indirect evidence suggesting that the reverse may also be true. For example, Belangee (2006) suggests that, from an individual psychology perspective, lack of social interest prevents individuals from forming meaningful relationships, with efforts being directed instead at disordered eating behaviour (Belangee, 2006). Furthermore, there is empirical evidence that constructs related to social anhedonia are

risk factors for the development of eating pathology. In a study of sister pairs, girls with an ED were found to have higher levels of social withdrawal prior to the onset of their ED, compared to their unaffected sisters (Adambegan et al., 2012). In a retrospective study using data collected across several European countries, individuals with an ED reported spending more time engaged in solitary activities during childhood, compared to healthy controls (Krug et al., 2012).

Gaps in the Literature and the Present Work

The preceding literature review highlights strong support for the role of interpersonal functioning in the development and maintenance of eating pathology. While theories and research have increased our understanding of the nature and nuances of the association between interpersonal stress and eating pathology, several notable gaps in the literature remain.

First, while a growing body of literature has examined how eating pathology relates to emotional, cognitive, and eating-related responses to interpersonal stress, there has not been any investigation into interpersonal reactions to social stressors. That is, how do individuals with eating pathology respond to those who reject, ostracize, or victimize them? The answer to this question would help explain how interpersonal problems are maintained in EDs, beyond the direct effect of ED pathology on interpersonal functioning and the reactions that symptoms elicit from others.

Second, while RS has been identified as a vulnerability factor in major interpersonal theories of eating pathology, research has only begun to examine its precise role. For example, some studies provide support for RS as a direct predictor of eating pathology (Bondü et al., 2020; Cardi et al., 2013) while others consider RS an intermediate variable in the association between other risk variables (e.g., attachment style, borderline personality disorder) and eating pathology (Al-Salom & Boylan, 2019; De Paoli, Fuller-Tyszkiewicz, & Krug, 2017; De Paoli, FullerTyszkiewicz, Halliwell, et al., 2017). Furthermore, to my knowledge, no studies have sought to examine indirect pathways through which RS may influence eating pathology. Doing so would help inform interventions aimed at mitigating the impact of RS on eating pathology by identifying the outcomes associated with RS that are most likely to impact disordered eating and tailoring interventions accordingly. For example, interventions may differ depending on whether they are targeting RS directly (e.g., positive interpretation bias training, Cardi et al., 2019) or are aimed at mitigating the outcomes of RS (e.g., mood regulation strategies for negative affect, social skills training to manage increased interpretsonal stress).

Third, negative affect is widely implicated as the pathway via which interpersonal stress influences eating pathology. However, it is likely that other pathways exist, especially since interpersonal stress does not always lead to negative affect (Blackhart et al., 2009), and disordered eating sometimes occurs in the context of positive mood states (Haynos et al., 2017). Social anhedonia may represent an alternate pathway, given that chronic interpersonal stress likely contributes to social anhedonia (Cacioppo et al., 2009; Hawkley et al., 2003, 2007; Rappaport et al., 2019), and social anhedonia is associated with eating pathology (Eiber et al., 2002; Harrison et al., 2014; Tchanturia et al., 2012; Watson et al., 2010). Examining this possibility would not only help identify an additional pathway via which interpersonal stress impacts eating pathology, but it would contribute to the extant literature on social anhedonia and eating pathology.

Lastly, there exist two smaller gaps that may have implications for the broad applicability of findings on interpersonal stress in eating pathology. First, with the exception of one study on the association between rejection and dietary restriction using daily-diary methodology (Beekman et al., 2017), the majority of research on social exclusion has relied on experimentally-manipulated ostracism in the laboratory and has examined eating outcomes that, while possibly maladaptive, are not necessarily pathological (e.g., increased consumption of palatable food, increased rate of consumption). As such, research examining both rejection and ostracism in relation to pathological forms of eating behaviours and cognitions using varied methodology is warranted. For example, research may benefit from examining associations between social exclusion and core ED symptoms, such as binge eating, dietary restriction, and weight/shape concerns using not only laboratory-based social exclusion paradigms but selfreport measures as well. Second, studies investigating the association between social anhedonia and eating pathology have exclusively examined samples of patients with diagnosed EDs. As outlined in the section on dimensional approaches to EDs, diagnostic categories may not accurately reflect the true nature of eating pathology, and many individuals who do not meet full criteria for AN, BN, or BED still suffer from clinically-significant eating pathology. As such, this area of research would likely benefit from studies examining associations between social anhedonia and continuously measured indices of eating pathology (e.g., binge eating, dietary restriction).

I sought to address these gaps across a series of three studies using varied methodology and diverse samples. In Article 1, I examined associations between core symptoms of EDs and maladaptive interpersonal responses to negative social evaluation. Specifically, I investigated whether binge eating and dietary restriction were associated with the tendency to retaliate following rejection by peers during competition. Based on findings suggesting that women with binge eating exhibit a more hostile and confrontational interpersonal style, whereas women with dietary restriction are submissive and avoidant of conflict (Arcelus et al., 2013), I predicted that binge eating would be associated with a greater tendency to retaliate against perpetrators of rejection, whereas dietary restriction would be associated with a lower tendency to retaliate following rejection. To test these hypotheses, I analyzed data from undergraduate women who were asked to complete an online "Survivor"-type game in which they voted to keep or reject "coplayers" (who, unknown to participants, were computerized) across several rounds until six winners remained. Participants created profiles (including photographs and biographical information) to be shown to "coplayers" and were also shown the profile of each of their "coplayers". In each round, participants voted to either keep or reject each coplayer. After entering their vote for a particular coplayer, they were shown how that coplayer voted for them. I examined the association between ED symptoms and participants' tendency to reject coplayers who voted to reject them in the previous round.

In Article 2, I sought to determine whether RS had an indirect effect on eating pathology via peer stress. In other words, whether RS was associated with increased ostracism and victimization, which in turn were associated with increased eating pathology. I examined this model with data from samples of undergraduate women and women with full-threshold and subthreshold EDs characterized by binge eating. Using both cross-sectional and longitudinal mediation models, I examined whether rejection sensitivity had an indirect effect on eating pathology (i.e., binge eating and shape/weight concerns) via peer victimization and ostracism. I hypothesized that RS would be concurrently associated with increased peer stress, which would then be associated with higher levels of eating pathology, both concurrently and over time.

In Article 3, I investigated whether peer stress, specifically victimization and ostracism, was indirectly associated with core ED symptoms (i.e., binge eating and dietary restriction) via altered social reward responsiveness across two time points. Specifically, I predicted that peer stress at Time 1 would be associated with disordered eating at Time 2 via social anhedonia.

Furthermore, I hypothesized that the pathway from social anhedonia to eating pathology would be moderated by positive expectancies about eating (for binge eating) and appearance-related reward responsiveness (for dietary restriction). To test these hypotheses, I collected longitudinal self-report data from first-year undergraduate women across three time points; although due to low retention, I only analyzed data from the first two time points (the beginning of the Fall and Winter semesters, respectively).

Article 1

Hunger Games: Associations between Core Eating Disorder Symptoms and Responses to Rejection by Peers during Competition¹

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Article 1 Abstract

Objective: Individuals with eating disorder (ED) symptoms are sensitive to social threat and report maladaptive interpersonal styles that may contribute to and exacerbate negative evaluation from others. Research in this area has relied primarily on self-report. The current study examined associations between behavioral responses to social threat and core ED symptoms using a behavioral paradigm. Based on previous findings that individuals with binge eating report being more reactive and confrontational, whereas individuals with dietary restriction tend to be more submissive and avoidant of conflict, we hypothesized that binge eating would be associated with a greater tendency to retaliate against rejection perpetrators, whereas dietary restriction would be associated with a lower tendency to retaliate when rejected.

Method: Undergraduate women (N = 132) completed a self-report measure of ED symptoms and participated in an online "Survivor"-type game in which they voted to either accept or reject computerized coplayers, while also receiving acceptance or rejection feedback from others. **Results:** Neither ED symptom was associated with how often participants retaliated against coplayers who rejected them. However, dietary restriction was related to more rejection votes *overall* (i.e., the tendency to reject others regardless of how others voted). **Discussion:** Findings suggest that individuals with dietary restriction may rely on a maladaptive defensive strategy aimed at pre-empting rejection, or alternatively, have difficulty shifting from habitual self-isolating behavior that results from over-involvement with restricting symptoms. Interventions targeting hypersensitivity to social threat or interpersonal flexibility may help reduce interpersonal stress and mitigate its impact on restricting symptoms.

Keywords: Eating Disorder Symptoms; Binge Eating; Dietary Restriction; Eating Disorders; Interpersonal Stress; Interpersonal Behavior

Hunger Games: Associations between Core Eating Disorder Symptoms and Responses to Rejection by Peers during Competition

Interpersonal stress, particularly negative social evaluation, has been shown to influence symptoms of eating disorders (EDs; see Monteleone et al., 2018 for a review). ED symptoms, in turn, can elicit negative evaluations from others, creating a cycle of increasing interpersonal stress and worsening symptoms (Nielsen & Bará-Carril, 2003; Rieger et al., 2010; Schmidt & Treasure, 2006). Additionally, individuals with EDs may exhibit maladaptive interpersonal biases and cognitions, particularly with regards to social threat (i.e., challenges to social inclusion or connectedness, such as rejection and ostracism; Rieger et al., 2010; Schmidt & Treasure, 2006). Specifically, women with EDs show attentional biases towards angry and rejecting faces and report increased negative affect following interpersonal stressors, compared to controls (Cardi et al., 2013; Harrison et al., 2010; Monteleone et al., 2018). However, little research has examined how individuals with EDs respond to people who are sources of social threat (i.e., those who evaluate them negatively). Examining how individuals with ED symptoms react towards others when negatively evaluated might further our understanding of the bidirectional association between interpersonal difficulties and ED symptoms.

To our knowledge, few studies have examined how individuals with ED symptoms respond towards people who judge them negatively. Studies using the Means-Ends Problem Solving Procedure (MEPS; Platt & Spivack, 1975), in which participants are asked to generate solutions to hypothetical interpersonal problems (e.g., rejection), have found that women with binge eating disorder (BED) and anorexia nervosa (AN) demonstrate less effective interpersonal problem solving, compared to healthy controls, and that drive for thinness is associated with ineffective interpersonal problem solving in undergraduate women (Ridout et al., 2015; Sternheim et al., 2020; Svaldi et al., 2011). In another study using hypothetical scenarios, women with AN and healthy controls were shown videos of hypothetical job supervisors providing critical feedback, ranging in tone from warm to cold (Ambwani et al., 2016). Participants then selected among possible behavioral responses to the feedback, also ranging from warm (e.g., "be warm or cooperative") to cold (e.g., "act unsociable or emotionally inhibited"; Erickson et al., 2009). Participants with AN selected significantly more cold behavioral responses, even when they perceived the supervisor's feedback to be warm. These findings suggest that, when faced with negative evaluation, women with ED symptoms may behave in ways that further exacerbate social difficulties.

Research on interpersonal style, anger expression, and hostility in individuals with EDs can also inform hypotheses, as individuals' responses to social threat are likely influenced by general interpersonal dispositions. For example, individuals who tend to be angry, hostile, and antagonistic are more prone to retaliating and acting aggressively when provoked with interpersonal conflict, compared to individuals lower on these traits (for a review, see Bettencourt et al., 2006). Research on interpersonal styles in EDs has shown that ED symptoms are associated with maladaptive dispositions that may influence how individuals respond to social threat, and these dispositions appear to differ across specific EDs and core symptoms (Arcelus et al., 2013).

When examining diagnostic groups, individuals with AN tend to be more conflict averse, are less hostile, and prioritize others' feelings, compared to individuals with BN and controls (Arcelus et al., 2013; Carter et al., 2012; Geller et al., 2000; Tiller et al., 1995). Conversely, individuals with bulimia nervosa (BN) score higher on measures of interpersonal conflict, hostility, and outwardly expressed anger, compared to individuals with AN and healthy controls (Arcelus et al., 2013; Fassino et al., 2001; Tiller et al., 1995). It is unclear whether these characteristics are associated with a particular symptom of BN (i.e., binge eating, compensatory behavior) or the entire symptom profile. Research on individuals with BED suggests that these patients are more outwardly angry, domineering, and cold, but also more submissive, compared to overweight and non-overweight controls, although there does not appear to be any research directly comparing individuals with BED to those with AN or BN (Brugnera et al., 2018, 2019; Fassino et al., 2003).

Only a few studies have examined associations between continuously measured ED symptoms and interpersonal styles in non-clinical samples. There is some support for the aforementioned findings, specifically that binge eating (though not purging) is associated with outwardly expressed anger and that oral control (similar to dietary restriction) is associated with lower hostility (MacLaren & Best, 2009; Peñas-Lledó et al., 2004). However, one study found that scores on a composite measure of bulimic symptoms were not associated with hostility, while another found that these scores were associated with anger suppression rather than expression (MacLaren & Best, 2009; Milligan & Waller, 2000).

Overall, available evidence suggests that individuals with binge eating may be more prone to conflict, expressing anger, and hostility. Based on this, we can hypothesize that these individuals may retaliate when faced with negative evaluation. Conversely, individuals with dietary restriction appear to avoid conflict and upsetting others, and may be less likely to retaliate. Notably, these patterns are clearer when examining diagnostic groups, whereas there is more variability in research examining continuously-measured symptoms. Further, findings on individuals with AN contradict the study by Ambwani et al. (2016), who found that individuals with AN favoured conflict-supportive rather than conflict-avoidant behavior. Ambwani and colleagues suggest that this discrepancy may be due to differences in measurement – it is possible that in hypothetical scenarios (such as the Ambwani and colleagues scenarios and the MEPS), participants behave differently than they do in real life, and that the more commonly used self-reports (e.g., The Inventory of Interpersonal Problems, Horowitz et al., 2003; The State-Trait Anger Expression Inventory; Spielberger, 1996) better reflect typical behavior. However, self-report measures are vulnerable to desirable responding and inaccurate recall of past events. Examining actual interpersonal behavior in response to social threat using a non-hypothetical social interaction paradigm is warranted and may extend our understanding of the associations between interpersonal difficulties and ED symptoms.

The current study examined associations between responses to rejection, a particularly salient form of social threat shown to have negative emotional and behavioral consequences (Gerber & Wheeler, 2009), and core ED symptoms (i.e., binge eating and dietary restriction). We used a novel interpersonal paradigm in which participants competed against coplayers (who were, unknown to participants, computer generated) in a realistic, computerized "Survivor"-type game (i.e., one in which participants vote to keep or oust one another from the game). Unlike self-report measures and hypothetical scenarios, this task allowed us to examine objective behavioral responses to subjectively "real" rejection. Our task also included a measure of participants' liking of coplayers, allowing us to examine effects independent of social anhedonia, which may affect behavioral responses to rejection and is found to be elevated in individuals with EDs (Harrison et al., 2014; Tchanturia et al., 2012).

Given findings suggesting that individuals with BN, BED, and binge eating may be more conflict-prone, hostile, and outwardly angry, whereas individuals with AN and dietary restriction may be more avoidant of conflict and upsetting others (e.g., Arcelus et al., 2013; Peñas-Lledó et al., 2004; Tiller et al., 1995), we hypothesized that binge eating would be associated with a greater tendency to retaliate against rejection perpetrators, whereas dietary restriction would be associated with a lower tendency to retaliate when rejected. Specifically, we hypothesized that binge eating would moderate the association between the feedback participants received from coplayers in the previous round (i.e., whether or not coplayers rejected them) and how participants voted for coplayers on the subsequent round, such that higher binge eating scores would be associated with a greater likelihood of participants voting to reject coplayers who rejected them in the previous round. Conversely, we hypothesized that dietary restriction would moderate the associated with a lower likelihood of participants voting, such that higher dietary restriction scores would be associated with a lower likelihood of participants voting to reject coplayers voting to reject coplayers who rejected them in the previous round.

Method

Participants

Participants were 132 women who were either students enrolled at a North American university or members of the local community. Participants ranged in age from 18 to 29 years (M= 20.69, SD = 2.24) and came from a range of ethnic/racial backgrounds: 49.6% identified as Caucasian, 15.7% as Chinese, 3.1% as South Asian, 3.1% as Arab, 2.4% as Japanese, 1.6% as Korean, 1.6% as Latin American, 0.8% as Black, 0.8% as Southeast Asian, 0.8% as West Asian, 14.2% as multiracial, 3.1% as other, and 2.4% preferred not to indicate ethnicity. Participants were recruited via a research participant pool organized by the authors' institution, posters around campus and the community, and online advertisements for a study on how brain activity relates to emotion and personality. Inclusion criteria were being over 18 years, female, and able to understand English. Students were offered either course credit or financial compensation for participation and community members were offered financial compensation. Five participants were excluded: two for incorrectly responding to an attention-check question, two due to prior familiarity with the social interaction task used, and one for not completing the questionnaires. Thus, the final sample size was 127 participants.

Procedure

Data for this manuscript were obtained from a larger study examining neural processing of social, monetary, and food reward. Prior to their laboratory visit, participants completed online questionnaires. At the start of the laboratory visit, participants were given information about the tasks to be completed and informed consent was obtained. Participants' profiles for the Island Getaway task were created (see below) and electroencephalogram (EEG) sensors were attached (n.b. EEG data were not analysed for the purpose of this study). Following task completion, participants rated the believability of the Island Getaway task and were debriefed about the computerized nature of the task.

Only study components relevant to the current research question are described in this manuscript. Data obtained from these components have not been included in other analyses or publications stemming from this study.

Island Getaway Task (modified from task described in Kujawa et al., 2014).

The Island Getaway task is a social interaction paradigm in which participants are told they are playing a "Survivor" type game with 11 peers in other laboratories across Canada and the United States who are, in fact, computer-generated. The code for the original task can be found at <u>http://arfer.net/projects/survivor</u>. The modified version of the task differs from the original in several ways, including the modification of coplayer profiles to better represent the target age group, the inclusion of "ally" and "enemy" coplayers (see below), and the addition of liking ratings of coplayers.

Before starting, participants are told that they will be playing a game consisting of six rounds in which players travel across Hawaiian islands. Each round, players vote to indicate whether they would like each coplayer to continue on the journey or be "kicked out" of the game. Participants are told that whoever receives the most "kick out" votes after each round will be eliminated, and that the goal of the game is to be one of six players to arrive at the final island. In reality, the task is programmed so that participants always make it to the final island.

Prior to beginning the task, participants have a headshot picture taken, answer demographic questions to create a player profile, and are shown their coplayers' profiles. Coplayers are six men and five women, and coplayers' photographs and information are randomly combined each time the task is run, in order to reduce any systematic effects of coplayer on participant behavior.

Each round, when shown a coplayer's profile, participants vote to keep or kick out that coplayer. They are told that they are voting simultaneously with the coplayer displayed on their screen and will see the coplayer's vote once both finish voting. Rejection or acceptance feedback from the coplayer is then displayed and participants are asked to enter liking ratings for that coplayer. In the first round, participants are required to vote to kick out at least three coplayers and to keep at least three coplayers. In subsequent rounds, they may vote however they choose.

After participants complete voting for a specific round, they are shown the coplayer who received the most "kick out" votes and are told this player was eliminated. With each round, participants expand their profiles by responding to a free-response question (e.g., "Who do you

most admire?") and can view coplayers' responses to these questions. In total, there are 51 voting/feedback trials across six rounds and task completion takes approximately 25 minutes.

To increase believability, four coplayers vote reciprocally with participants – that is, two coplayers vote to accept the participants on the first two rounds ("allies"), and two vote to reject on the first two rounds ("enemies"). All four coplayers then vote reciprocally on all subsequent rounds. The remaining coplayers' votes are programmed so that participants receive approximately half acceptance and half rejection feedback. In the current study, participants received an average of 24 "kick out" votes and 27 "keep" votes.

Measures

Coplayer Liking Ratings

After entering their vote for a given coplayer, participants are asked to use a visual analogue scale to rate how much they like that coplayer (1 = not at all, 9 = extremely).

Believability Rating

Prior to debriefing, participants were verbally asked "On a scale from 1 to 5, where 1 is "not at all" and 5 is "completely", how much did you believe you were playing against other lab participants in real time?".

Eating Pathology Symptoms Inventory (EPSI; Forbush et al., 2013)

The EPSI is a 45-item self-report questionnaire designed to measure ED symptoms via eight subscales. Using a five-point scale (0 = never to 4 = very often), participants rate the frequency of various symptoms over the preceding four weeks. The 8-item Binge Eating and 6-item Restricting subscales were used. In student samples, the Binge Eating and Restricting subscales have demonstrated adequate internal consistency ($\alpha s = .83$), good test-retest reliability over two-to-four weeks (rs = .71 - .75), and excellent convergent and discriminant validity

(Forbush et al., 2013). Internal consistency estimates in the current study were good for Binge Eating ($\alpha = .82$) and Restricting ($\alpha = .87$) subscales.

Body Mass Index (BMI)

BMI was calculated based on participants' self-reported height and weight using the standard formula ((weight in pounds)/(height in inches²))*703.

Statistical Analyses

We used generalized linear mixed models (GLMM) to examine whether ED symptoms moderated associations between the feedback participants received during the previous round of the Island Getaway task and their vote to accept or reject coplayers during the following round. Voting response (level 1) was nested within the 127 participants as well as the six rounds of the Island Getaway task. Participant and round were crossed at level 2, since every participant voted in every round. Participants' voting behavior was entered as the dichotomous outcome variable (0 = accept, 1 = reject). Feedback received during the previous round (level 1), ED symptom (i.e., binge eating or restricting scores; level 2), and the interaction between feedback and symptom were entered as fixed effects. Intercepts and slopes for participant and round were entered as random effects. We included liking of coplayers during the previous round as a covariate in order to examine effects independent of participants' general tendency to like others (results are described with and without liking as a covariate). Only data from rounds 2 to 6 were used as there was no previous round feedback associated with round 1 voting behavior. Separate models were constructed for binge eating and dictary restriction in SPSS (24.0; SPSS Inc.).

Results

Descriptive Statistics

Based on guidelines proposed by Kim (2013) regarding cutoffs for standardized skewness scores, and results of Kolmogorov-Smirnov (KS) tests, a square-root transformation was used to correct for the positive skew of binge eating ($Z_{skewness} = 4.03$; KS = 0.10, p = .004) and restricting ($Z_{skewness} = 3.69$; KS = 0.12, p < .001) scores. All continuous variables were standardized prior to analyses.

Descriptive statistics and zero-order correlations for variables included in our main analyses are presented in Table 1. On average, participants voted to reject coplayers 39% of the time. Binge eating and restricting scores were comparable to established norms for undergraduate women (Binge Eating: $M_{norm} = 8.92$, SD = 4.52; t(278) = 0.34, p = .74; Restricting: $M_{norm} = 5.55$, SD = 4.47; t(278) = 1.44, p = .15; Forbush et al., 2013). Participant BMIs ranged from underweight to obese (M[SD] = 21.40 [3.08]; range = 15.33 - 33.25).

Participants reported moderate belief that they were playing against real peers (M = 2.97; SD = 1.30). We chose not to exclude participants based on belief ratings, given previous research demonstrating that social exclusion paradigms evoke negative reactions even when participants know that they are interacting with a computer or trained confederates (Zadro et al., 2004).

GLMM Results

Table 2 presents GLMM results. Participants were more likely to vote to reject coplayers with each successive round, more likely to vote to reject a coplayer who had rejected them in the previous round, and less likely to vote to reject coplayers whom they liked in the previous round. Binge eating scores were not significantly associated with the likelihood of voting to reject coplayers, and the interaction between voting feedback received during the previous round and binge eating was non-significant. The pattern of results did not differ when previous round liking was removed as a covariate.

In contrast, there was a main effect of restricting scores on voting behavior, such that higher restricting scores were associated with a greater likelihood of voting to reject coplayers. The interaction between feedback received during the previous round and restricting was nonsignificant. When previous round liking was removed as a covariate, there was no longer a statistically significant main effect of dietary restriction, although the change in effect size was small (partial R^2 decreased from 0.09 to 0.07).

Given that binge eating and dietary restriction frequently co-occur, we examined a model in which both symptoms, along with their two-way interactions with coplayer voting, were entered simultaneously in order to investigate the effect of each symptom independently of the other. The pattern of results did not differ from those described above: there was a main effect of restricting scores (b = 0.15, SE = 0.07, p = .047), but not binge eating scores (b = 0.11, SE =0.07, p = .12), on voting behavior.

Post-hoc Analyses

To rule out alternative explanations (i.e., explore whether other factors influenced the association between ED symptoms and voting behavior), post-hoc analyses examined: 1) the effect of ED symptoms on the association between coplayer voting and participants' liking of coplayers (i.e., do ED symptoms influence the extent to which previous voting is associated with coplayer liking?); 2) whether the effect of ED symptoms on voting behavior or liking ratings differed as a function of coplayer gender (i.e., are effects more pronounced for coplayers of a given gender?); 3) whether results reflect social learning difficulties (i.e., did participants higher in ED symptoms not understand how to appropriately play the game or differentiate between allies and enemies?); 4) whether BMI influenced our findings (i.e., do results differ based on participants' weight status?); and 5) whether the extent to which participants believed that they

were playing against real coplayers influenced results (i.e., do results differ for participants who did not believe the task?).

The only analysis that changed our pattern of results was the inclusion of BMI as a covariate (see Supplemental Material). Including BMI as a covariate strengthened the effect of binge eating (change in Partial R^2 from 0.02 to 0.05) and attenuated the effect of dietary restriction (change in Partial R^2 from 0.09 to 0.05), on the tendency to reject co-players.

Discussion

The current study examined associations between core ED symptoms and interpersonal behavior following rejection by "peers" in a computerized, "Survivor"-type game. We hypothesized that binge eating would be associated with a greater tendency to retaliate in response to rejection, whereas dietary restriction would be associated with a lower retaliation tendency. We found that neither binge eating nor dietary restriction moderated the association between previous coplayer votes (i.e., how coplayers voted for participants in the preceding round) and participant voting (i.e., how participants voted for coplayers in the current round), indicating that the tendency to retaliate against coplayers did not differ based on these ED symptoms. Interestingly, however, restricting scores were associated with a greater tendency to reject coplayers overall, regardless of how coplayers previously voted. Thus, individuals with dietary restriction may engage in maladaptive interpersonal behavior that is independent of how they are treated by others.

Contrary to hypotheses and previous findings suggesting that reactions to social threat may differ across ED symptoms (e.g., Arcelus et al., 2013; Fassino et al., 2001; Tiller et al., 1995), the extent to which rejection was associated with retaliation did not vary based on ED symptom severity. The discrepancy between our findings and those derived from self-reports of interpersonal styles may be explained by inaccurate self-reporting. Individuals with dietary restriction may describe their interpersonal styles as more socially desirable than they actually are, and individuals with binge eating may describe their interpersonal styles as more problematic. However, it is unclear why participants with dietary restriction would present themselves more positively compared to participants with binge eating, given that both groups exhibit high levels of self-presentation and socially-prescribed perfectionism (which could influence positive self-descriptions; Bardone-Cone, 2007; Hewitt et al., 1995; Stoeber et al, 2017) as well as high levels of self-criticism (which could influence negative self-descriptions; Duarte et al., 2017; Fennig et al., 2008). Alternatively, the online nature of the Island Getaway task may have influenced participants' behavior, such that they behaved differently than they would face-to-face. Indeed, individuals are less inhibited and display more antisocial behavior online compared to in-person (e.g., Lapidot-Lefler & Barak, 2012; Suler, 2004). Given the recent rise in social media use, research investigating whether maladaptive interpersonal styles traditionally associated with EDs are consistent across modalities (i.e., in-person vs. online) is warranted. Finally, the majority of the extant research used to formulate our hypotheses was conducted using clinical samples with distinct EDs, whereas we measured ED symptoms continuously in a non-clinical sample. It may be that interpersonal behavior differs at higher levels of ED symptoms and/or that individual ED symptoms relate differently to interpersonal behavior than ED diagnoses. As such, our ability to extend our findings beyond individual symptoms to broader diagnostic groups is limited.

Interestingly, while we found that neither ED symptom was associated with the likelihood of retaliation, dietary restriction was associated with a greater tendency to reject coplayers, regardless of how coplayers voted. In other words, the behavior of participants with dietary restriction appeared to be independent of, and not always in line with, feedback received from coplayers. This finding is similar to that of Ambwani et al. (2016) where women with AN were more likely to select cold responses to interpersonal feedback, even when the feedback was perceived as warm. It is also in line with, and offers a possible explanation for, findings from studies examining social networks in relation to ED symptoms, which have shown that dietary restriction and restraint, but not binge eating, are associated with fewer friendships in young adults and adolescents (Forney et al., 2019; Rayner et al., 2013). Of note, covarying BMI attenuated the effect of dietary restriction, and strengthened the effect of binge eating, on the tendency to reject coplayers. This suggests that BMI may partially account for the effect of ED symptoms on the tendency to reject others, which is surprising given findings that, on its own, BMI is not associated with interpersonal problems (Blomquist et al., 2012; Carr & Friedman, 2006). However, prior studies have indicated that individuals with higher BMIs are likely to experience weight stigma (Puhl & Brownell, 2006), and these experiences of stigma are associated with increased expectations of interpersonal rejection (McClure Brenchley & Quinn, 2016). Future studies using this paradigm might explicitly assess experience with stigmatization and expectations of rejection to better understand the association between weight status and responses to rejection.

Although our results do not allow us to draw conclusions about mechanisms linking dietary restriction and the tendency to reject others, we can speculate based on previous research. This finding might be explained by the association between dietary restriction and rejection sensitivity (i.e., being hyper-attuned and emotionally reactive to rejection; Cardi et al., 2013, 2017; Monteleone et al., 2018). Rejection-sensitive individuals are thought to demonstrate a maladaptive defensive strategy in which they attempt to prevent rejection by shunning others

(Romero-Canyas & Downey, 2005). Given the nature of the Island Getaway task (i.e., the explicit possibility of being rejected by coplayers), individuals with higher levels of dietary restriction may have become hyper-attuned to the threat of rejection, leading them to reject others before others could reject them. The tendency to push others away to avoid rejection may contribute to the significant interpersonal difficulties thought to influence the onset and maintenance of EDs (Cardi et al., 2018; Monteleone et al., 2018; Rieger et al., 2010; Wilfley et al., 2003) Although binge eating has also been associated with rejection sensitivity (Monteleone et al., 2018), there is some evidence that rejection sensitivity and interpersonal problems are more pronounced among individuals with AN (Cardi et al., 2013; Raykos et al., 2014).

An alternate explanation may be that individuals with dietary restriction exhibit maladaptive interpersonal habits related to their symptoms. In AN, as symptoms become more established, individuals become increasingly socially withdrawn, as their focus centers more on their ED and less on valued goals in other domains (Mulkerrin et al., 2016; Wildes & Marcus, 2011). Further, individuals with AN tend to exhibit rigid behavior and inflexiblity across situations (Steinglass & Walsh, 2006; Tchanturia et al., 2004). It may be that individuals with dietary restriction are used to behaving in ways that isolate them from others, and that in the Island Getaway Task, participants had difficulty shifting from this behavioral pattern. It is unclear, however, why this pattern would not also be observed for binge eating, given that both BN and BED have been associated with social withdrawal and lack of flexibility (Lobera et al., 2009; Roberts et al., 2007). One possibility, although entirely speculative, is that the discrepancy lies in social cognition. Whereas individuals with AN have difficulties considering the internal experiences of others (Russell et al., 2009), individuals with BN and BED, who show less pronounced deficits in social cognition (Aloi et al., 2017; DeJong et al., 2013), may be able to override their tendency to withdraw by considering the impact of their behavior on others.

The use of a computerized, "Survivor"-type game was a notable strength of our study. It allowed us to examine interpersonal responses to rejection in association with ED symptoms using a realistic behavioral paradigm, increasing the likelihood that results reflected participants' typical behavior. Additionally, the use of the Island Getaway task allowed us to examine retaliatory behavior as it occurred, rather than relying on retrospective reports that may be inaccurate. Given that coplayer profiles were randomized (i.e., participants did not all play against the same set of coplayers), we can also be confident that participants' behavior was associated with coplayer feedback (i.e., voting) rather than individual coplayer characteristics.

There are also limitations to our methodology. First, the computerized task may not have adequately represented real-life stressors associated with ED symptoms. Participants competed against unknown "peers", and research in non-ED samples suggests that individuals react differently to rejection when perpetrated by strangers versus people they know, although there is conflicting evidence as to which has a more negative impact (Leary et al., 1998; Snapp & Leary, 2001; Sommer et al., 2009). Further, while forming and maintaining alliances can be viewed as beneficial to succeeding in the Island Getaway task, alliances formed during competition might not be an ideal proxy for meaningful interpersonal relationships. Future research should examine associations between ED symptoms and retaliation towards close individuals (e.g., friends, family, romantic partners). Second, the task was computerized rather than in-person. While emotional responses to rejection appear to be consistent across modalities (Filipkowski & Smyth, 2012), behavioral responses to rejection may differ with increased anonymity. Future research should aim to replicate the current findings using a less anonymous version of the task, for

example, by having participants play the game on computers, but in the same room as coplayers. Third, we examined a non-clinical, student sample. It is possible that maladaptive interpersonal styles are not present in individuals with less severe symptoms, as evidenced by inconsistent findings on the association between interpersonal styles and ED symptoms in non-clinical samples (MacLaren & Best, 2009; Milligan & Waller, 2000). Replicating the current study in a clinical sample might help clarify the inconsistency between the current findings and extant literature. Finally, we did not ask participants to report their reasons for rejecting coplayers. Elucidating the motivation behind rejection would enhance our understanding of how characteristics of individuals with EDs promote interpersonal stress.

This study was, to our knowledge, the first to examine the association between core ED symptoms and interpersonal responses to rejection using a realistic behavioral paradigm. The finding that dietary restriction was associated with a greater tendency to reject others suggests two possible avenues for intervention. First, modifying sensitivity to social threat may be helpful in reducing interpersonal stress in individuals with dietary restriction. A recent study demonstrated that positive interpretation bias training (i.e., increasing positive/realistic interpretations of ambiguous scenarios) lead to a reduction in negative interpretations of social scenarios and increased self-esteem in adolescents with AN (Cardi et al., 2019). Second, targeting rigidity might be helpful in promoting adaptive interpersonal behavior, rather than inflexible withdrawal patterns. A randomized control trial of cognitive remediation therapy in individuals with AN demonstrated that it was effective in increasing cognitive flexibility (Brockmeyer et al., 2014), but whether this treatment can impact social behavior is unclear. While our hypotheses were not supported, we believe that our findings provide insight into how interpersonal problems may develop and persist in individuals with dietary restriction. By

rejecting others, individuals with dietary restriction may limit the possibility for development and growth of interpersonal relationships.

Article 1 References

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Table 1

Descriptive Statistics and Zero-order Correlations

	1	2	3	4
1. Proportion of Rejection Votes	-			
2. Coplayer Liking	34**	-		
3. Binge Eating	.07	.10	-	
4. Restricting	.25**	.03	.07	-
Mean	0.39	0.11	9.12	6.41
SD	0.11	0.90	5.45	5.55
Range	0.06 - 0.67	1.98 - 9.00	0.00 - 29.00	0.00 - 22.00

Note. Proportion of rejection votes includes voting behavior across all six rounds.

Table 2

The Moderating Effect of ED Symptoms on the Association between Island Getaway Feedback

Predictor	Estimate	SE	р	Partial R ²	95% CIs
Model 1: Binge eating as moderator					
Intercept	-0.74	0.09	<.001	0.62	-0.93, -0.54
Coplayer liking previous round	-0.72	0.04	<.001	0.07	-0.81, -0.63
Previous round feedback	0.56	0.12	.001	0.66	0.29, 0.82
Binge eating	0.12	0.07	.11	0.02	-0.03, 0.27
Feedback x binge eating	-0.08	0.09	.40	0.01	-0.27, 0.11
Model 2: Dietary restriction as moderator					
Intercept	-0.74	0.09	<.001	0.64	-0.94, -0.55
Coplayer previous liking	-0.70	0.04	<.001	0.08	-0.79, -0.62
Previous round feedback	0.55	0.12	.001	0.65	0.29, 0.82
Dietary restriction	0.16	0.07	.03	0.09	0.01, 0.30
Feedback x dietary restriction	0.05	0.10	.58	0.002	-0.14, 0.24

and Participant Voting Behavior

Note. Previous round feedback and participant voting are coded as 0 = accept and 1 = reject.

Article 1 Supplemental Material

The Moderating Effect of Eating Pathology on the Association Between Current Round

Predictor	Estimate	SE	р	Partial R ²	95% CIs
Model 1: Binge eating as moderator					
Intercept	6.27	0.09	<.001	0.99	6.09, 6.45
Current round feedback	-1.55	0.13	<.001	0.88	-1.82, -1.29
Binge eating	0.10	0.08	.215	0.01	-0.06, 0.26
Feedback x binge eating	-0.06	0.10	.527	0.003	-0.25, 0.13
Model 2: Dietary restriction as moderator					
Intercept	6.28	0.09	<.001	0.99	6.10, 6.46
Current round feedback	-1.57	0.13	<.001	0.88	-1.84, -1.29
Dietary restriction	0.06	0.08	.441	0.005	-0.10, 0.22
Feedback x dietary restriction	-0.08	0.10	.436	0.005	-0.27, 0.12

Feedback and Coplayer Liking

Note. Feedback coded as 0 = accept and 1 = reject.

Predictor	Estimate	SE	р	Partial R ²	95% CIs
Model 1: Binge eating					
Intercept	-0.76	0.10	<.001	0.56	-0.97, -0.56
Coplayer liking previous round	-0.71	0.04	<.001	0.07	-0.79, -0.62
Previous round feedback	0.55	0.12	<.001	0.64	0.29, 0.80
Coplayer gender	0.06	0.08	.477	0.004	-0.10, 0.22
Binge eating	0.04	0.07	.539	0.02	-0.09, 0.18
Coplayer gender x binge eating	0.08	0.08	.346	0.01	-0.08, 0.24
Model 2: Dietary restriction					
Intercept	-0.77	0.10	<.001	0.58	-0.97, -0.57
Coplayer previous liking	-0.69	0.04	<.001	0.08	-0.78, -0.61
Previous round feedback	0.54	0.12	<.001	0.64	0.28, 0.80
Coplayer gender	0.07	0.08	.436	0.01	-0.10, 0.23
Dietary restriction	0.14	0.07	.039	0.10	0.01, 0.27
Coplayer gender x dietary restriction	0.10	0.08	.248	0.01	-0.07, 0.26

The Moderating Effect of Coplayer Gender on the Association Between Eating Pathology and

Voting Behavior

Note. Coplayer gender coded as 0 = male and 1 = female. Coplayer liking and feedback received

in the previous round were entered as covariates. Feedback coded as 0 = accept and 1 = reject.

Predictor	Estimate	SE	р	Partial R ²	95% CIs
Model 1: Binge eating					
Intercept	6.23	0.09	<.001	0.99	6.05, 6.41
Current round feedback	-1.52	0.13	<.001	0.88	-1.80, -1.25
Coplayer gender	0.05	0.05	.326	0.02	-0.05, 0.15
Binge eating	0.09	0.08	.241	0.01	-0.06, 0.25
Coplayer gender x binge eating	0.002	0.05	.961	0.02	-0.09, 0.10
Model 2: Dietary restriction					
Intercept	6.24	0.09	<.001	0.99	6.06, 6.43
Current round feedback	-1.54	0.13	<.001	0.88	-1.81, -1.26
Coplayer gender	0.05	0.05	.364	0.02	-0.06, 0.15
Dietary restriction	0.06	0.08	.488	0.004	-0.10, 0.22
Coplayer gender x dietary restriction	-0.01	0.05	.877	0.0002	-0.11, 0.09

The Moderating Effect of Coplayer Gender on the Association Between Eating Pathology and

Coplayer Liking

Note. Gender coded as 0 = male and 1 = female. Current found feedback included as covariate

and coded as 0 = accept and 1 = reject.

Predictor	Estimate	SE	р	Partial <i>R</i> ²	95% CIs
Model 1: Binge eating as moderator					
Intercept	-0.99	0.11	<.001	0.21	-1.21, -0.77
Coplayer liking previous round	-0.71	0.04	<.001	0.07	-0.80, -0.63
Previous round feedback	0.56	0.10	<.001	0.17	0.36, 0.76
Round	0.07	0.02	.005	0.02	0.02, 0.11
Binge eating	0.13	0.10	.189	0.005	-0.07, 0.33
Round x binge eating	-0.01	0.02	.541	0.001	-0.06, 0.03
Model 2: Dietary restriction as moderator					
Intercept	-0.99	0.11	<.001	0.24	-1.22, -0.77
Coplayer previous liking	-0.70	0.04	<.001	0.08	-0.78, -0.61
Previous round feedback	0.55	0.10	<.001	0.17	0.35, 0.75
Round	0.07	0.02	.005	0.02	0.02, 0.11
Dietary restriction	0.12	0.10	.249	0.003	-0.08, 0.32
Round x dietary restriction	0.02	0.02	.454	0.001	-0.03, 0.06

The Moderating Effect of Eating Pathology on the Association Between Round and Voting

Behavior

Note. Coplayer liking and feedback received in the previous round were entered as covariates.

Feedback coded as 0 = accept and 1 = reject.

Predictor	Estimate	SE	р	Partial R ²	95% CIs
Model 1: Binge eating as moderator					
Intercept	6.32	0.09	<.001	0.98	6.15, 6.49
Coplayer = Unknown	-0.81	0.07	<.001	0.80	-0.96, -0.67
Coplayer = Enemy	-1.53	0.13	<.001	0.88	-1.81, -1.25
Binge eating	0.10	0.08	.215	0.01	-0.06, 0.26
Unknown x binge eating	-0.03	0.07	.658	0.001	-0.16, 0.10
Enemy x binge eating	-0.07	0.10	.483	0.004	-0.27, 0.13
Model 2: Dietary restriction as moderator					
Intercept	6.32	0.09	<.001	0.98	6.15, 6.50
Coplayer = Unknown	-0.82	0.07	<.001	0.80	-0.96, -0.67
Coplayer = Enemy	-1.53	0.14	<.001	0.87	-1.82, -1.25
Dietary Restriction	0.11	0.08	.170	0.01	-0.05, 0.28
Unknown x dietary restriction	-0.11	0.07	.119	0.02	-0.24, 0.03
Enemy x dietary restriction	-0.13	0.10	.219	0.01	-0.33, 0.08

The Moderating Effect of Eating Pathology on the Association Between Ally/Enemy Status

and Coplayer Liking

Note. Ally/enemy status coded as 0 =ally, 1 = unknown, and 2 = enemy.

Predictor	Estimate	SE	р	Partial R ²	95% CIs
Model 1: Binge eating as moderator					
Intercept	-0.71	0.09	<.001	0.53	-0.90, -0.52
Coplayer liking previous round	-0.74	0.05	<.001	0.07	-0.83, -0.65
BMI	-0.03	0.06	.63	0.003	-0.15, 0.09
Previous round feedback	0.52	0.12	<.001	0.56	0.26, 0.78
Binge eating	0.18	0.08	.02	0.05	0.02, 0.33
Feedback x binge eating	-0.13	0.10	.21	0.01	-0.32, 0.07
Model 2: Dietary restriction as moderator					
Intercept	-0.71	0.09	<.001	0.55	-0.90, -0.52
Coplayer previous liking	-0.71	0.05	<.001	0.08	-0.80, -0.62
BMI	0.06	0.06	.30	0.01	-0.05, 0.17
Previous round feedback	0.52	0.12	<.001	0.57	0.25, 0.78
Dietary restriction	0.17	0.07	.02	0.05	0.03, 0.32
Feedback x dietary restriction	0.04	0.10	.71	0.001	-0.16, 0.24

Tests of Main Hypotheses with the Inclusion of BMI as a Covariate

Note. Previous round feedback and participant voting are coded as 0 = accept and 1 = reject.

BMI = Body Mass Index.

Predictor	Estimate	SE	р	Partial R ²	95% CIs
Model 1: Binge eating as moderator					
Intercept	-0.77	0.16	<.001	0.35	-1.09, -0.45
Coplayer liking previous round	-0.72	0.04	<.001	0.07	-0.81, -0.63
Believability	0.01	0.04	.78	0.001	-0.08, 0.10
Previous round feedback	0.56	0.12	<.001	0.66	0.29, 0.82
Binge eating	0.12	0.07	.11	0.02	-0.03, 0.27
Feedback x binge eating	-0.08	0.09	.40	0.01	-0.27, 0.11
Model 2: Dietary restriction as moderator					
Intercept	-0.82	0.16	<.001	0.40	-1.13, -0.51
Coplayer previous liking	-0.70	0.04	<.001	0.08	-0.79, -0.62
Believability	0.03	0.04	.54	0.004	-0.06, 0.11
Previous round feedback	0.55	0.12	<.001	0.65	0.28, 0.82
Dietary restriction	0.16	0.07	.03	0.09	0.01, 0.30
Feedback x dietary restriction	0.05	0.10	.58	0.002	-0.14, 0.24

Tests of Main Hypotheses with the Inclusion of Task Believability as a Covariate

Note. Previous round feedback and participant voting are coded as 0 = accept and 1 = reject.

Bridge to Article 2

In Article 1, I examined whether core eating disorder symptoms were associated with maladaptive behavioural responses to rejection by peers during competition. I used an experimental paradigm in which participants played a "Survivor"-type game that required them to vote to keep or reject computerized coplayers across several rounds, while simultaneously receiving acceptance or rejection feedback from coplayers. I examined whether binge eating and dietary restriction were associated with participants' tendencies to reject (i.e., retaliate against) coplayers who previously voted to reject them. While I did not find that either form of disordered eating was associated with an increased tendency to reject coplayers overall, regardless of how coplayers voted. I hypothesized that individuals with dietary restriction may be overly sensitive to the possibility of rejection and possibly attempt to pre-empt rejection by rejecting others. Such misguided attempts could have the paradoxical effect of increasing peer stress, and thus perpetuate disordered eating.

The goal of Article 2 was to expand on the aforementioned hypothesis by examining whether RS would be associated with eating pathology via elevated peer stress. Using data collected from undergraduate women and women with binge eating, I sought to investigate whether the tendency to be hypersensitive to social threat was associated with more frequent experiences of ostracism and victimization, and whether these forms of peer stress were in turn associated with increased eating pathology both concurrently and across time.

Article 2

Reconsidering the Role of Interpersonal Stress in Eating Pathology: Sensitivity to Rejection

Might be More Important than Actual Experiences of Peer Stress²

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² Article currently under review at *Appetite*.

Article 2 Abstract

Rejection sensitivity (i.e., the tendency to anxiously expect, readily perceive, and overreact to real or perceived rejection) is theorized to play a role in the onset and maintenance of disordered eating. Although rejection sensitivity has repeatedly been associated with eating pathology in clinical and community samples, the pathways through which this psychological trait influences eating pathology have been not fully established. The current study investigated peer-related stress, which can be influenced by rejection sensitivity and is associated with eating pathology, as a mechanism linking these constructs. In two samples of women -189 first-year undergraduate students and 77 community women with binge eating – we examined whether rejection sensitivity was indirectly associated with binge eating and weight/shape concerns via ostracism and peer victimization, both cross-sectionally and longitudinally. Our hypotheses were not supported: there were no indirect associations between rejection sensitivity and eating pathology via interpersonal stress in either sample. However, we did find that rejection sensitivity was *directly* associated with weight/shape concerns in both samples and with binge eating in the clinical sample in cross-sectional (but not longitudinal) analyses. Our findings suggest that the association between rejection sensitivity and disordered eating is not dependent on actual experiences of interpersonal stress. That is, simply anticipating or perceiving rejection may be sufficient to play a role in eating pathology. As such, interventions targeting rejection sensitivity may be helpful in the treatment of eating pathology.

Keywords: Rejection Sensitivity, Ostracism, Victimization, Binge Eating, Weight Concerns, Shape Concerns

Reconsidering the Role of Interpersonal Stress in Eating Pathology: Sensitivity to Rejection Might be More Important than Actual Experiences of Peer Stress

Rejection sensitivity (RS) – the tendency to anxiously expect, readily perceive, and overreact to real or perceived rejection (Downey & Feldman, 1996) – has been proposed as a vulnerability factor for eating pathology (Rieger et al., 2010). Indeed, RS and related constructs have been associated with eating disorders (EDs) and their symptoms in both clinical and nonclinical samples. For example, compared to healthy controls, individuals with EDs show an attentional bias towards rejecting faces and tend to form negative interpretations of ambiguous social situations, such as noticing the conversation stopping when they enter a room (Cardi et al., 2013, 2017). Further, the presence of an ED is associated with greater negative affect in response to social threat (Monteleone et al., 2018). In non-clinical samples, RS and a fear of negative evaluation have been positively associated with concurrent disordered eating cognitions and attitudes (Atlas, 2004; De Paoli et al., 2017; Gilbert & Meyer, 2005; Wolfe et al., 2009), although there is less evidence for concurrent associations with ED behaviours, such as binge eating and purging (Atlas, 2004; De Paoli et al., 2017; Gilbert & Meyer, 2005). RS may, however, be implicated in the maintenance and worsening of eating pathology over time. In a large sample of adolescents and young adults, anxious rejection sensitivity predicted increases in overall eating pathology across three years (Bondü et al., 2020). Similarly, in a sample of undergraduate women, fear of negative evaluation predicted worsening binge eating and purging across a 33-week period (Gilbert & Meyer, 2005). Despite extant findings supporting a link between RS and ED symptoms, the mechanism(s) through which RS may influence eating pathology are not clear. Uncovering pathways by which RS contributes to eating pathology could inform interventions aimed at mitigating its impact on the development and course of ED

symptoms.

One potential mechanism linking RS and ED symptoms is decreased interpersonal functioning (Arcelus et al., 2013; Rieger et al., 2010; Treasure & Schmidt, 2013). Individuals high in RS are not only overly attuned to and emotionally reactive to negative social evaluation (Downey & Feldman, 1996), but they tend to behave in ways that perpetuate interpersonal difficulties. In response to cues of perceived social threat, individuals high in RS may respond in a variety of maladaptive ways. For example, in both children and adults, RS has been associated with social withdrawal in situations of actual or possible rejection (Downey et al., 1998; London et al., 2007; Watson & Nesdale, 2012; Zimmer-Gembeck & Nesdale, 2013). In addition, some individuals high in RS exhibit more externalizing behavior when faced with potential rejection; for example, responding in a hostile or aggressive manner toward acquaintances, peers, and romantic partners (Downey et al., 1998, 2004; London et al., 2007; Purdie & Downey, 2000; Watson & Nesdale, 2012; Zimmer-Gembeck & Nesdale, 2013). While these behaviors are aimed at avoiding rejection, they have the paradoxical effect of increasing negative social evaluation and interpersonal difficulties (Romero-Canyas & Downey, 2005). It may be that individuals with eating pathology, in an attempt to avoid rejection by others, behave in ways that elicit negative reactions from others and promote interpersonal problems. Indeed, a recent study found that when the potential for rejection was salient, greater levels of dietary restriction were associated with a tendency to reject others, regardless of the other's behavior (Schell et al., 2021).

Individuals with eating pathology do indeed experience elevated levels of interpersonal difficulties (for a review, see Wilfley et al., 2003). In cross-sectional studies, interpersonal stress such as being teased or victimized by peers, having difficulty with communication and affiliation, and being mistrustful of others, has been associated with various forms of eating

pathology. Specifically, in both clinical and non-clinical populations, these forms of interpersonal stress have been associated with binge eating, loss of control eating, purging, dietary restriction, and body image disturbances (Ansell et al., 2012; Day et al., 2021; Elliott et al., 2010; Ivanova, Tasca, Hammond, et al., 2015; Ivanova, Tasca, Proulx, et al., 2015). Further, interpersonal difficulties predict increases in eating pathology over time and precede disordered eating episodes within an individual. For example, peer victimization was longitudinally associated with increases in disordered eating across a four-year period in adolescents (Lee & Vaillancourt, 2019), and interpersonal stress related to peers and romantic partners predicted greater dietary restraint over the course of an academic term in undergraduate women (Cain et al., 2010). Using daily diary methodology, it has been shown that negative social experiences precede episodes of binge eating in women with bulimia nervosa (Steiger et al., 1999) and engagement in restrictive eating in college women (Beekman et al., 2017). These findings suggest that interpersonal stress likely contributes to the development and maintenance of disordered eating.

Taken together, the aforementioned findings suggest that RS may be associated with eating pathology via interpersonal stress. That is, RS may lead to increased interpersonal difficulties (due to maladaptive behaviors aimed at preventing rejection), which may then contribute to eating pathology. While similar theoretical models have been proposed (e.g., that RS leads to social maladjustment and subsequent disordered eating; Arcelus et al., 2013; Rieger et al., 2010; Treasure & Schmidt, 2013), to our knowledge, there is no empirical evidence supporting these models. There is evidence, however, that the hypothesized model is applicable to depression, which is highly comorbid with EDs (Blinder et al., 2006; Touchette et al., 2011). In a study of adults with a history of depression, RS predicted increased depressive symptoms over a four-month period, and this association was mediated by dependent stressors (including those of an interpersonal nature, such as difficulties with family, friends, and romantic partners) experienced during the four-month period (Liu et al., 2014). Determining whether this model is also applicable to disordered eating is thus warranted.

The aim of the current study was to examine whether the association between RS and ED symptoms is explained by two types of peer-related stress: ostracism (i.e., being ignored or excluded by peers) and peer victimization (e.g., physical/verbal aggression, manipulation). Specifically, we sought to examine whether RS would be indirectly associated with eating pathology via ostracism and peer victimization. We hypothesized that RS would be associated with greater ostracism and victimization, which in turn would be associated with heightened eating pathology, specifically binge eating and weight/shape concerns. We examined this model using both cross-sectional and longitudinal data collected from undergraduate women (Study 1) and women with binge eating (Study 2).

Study 1

Method

Participants

Participants were 189 first-year female undergraduate students enrolled in a psychology course at a large North American university. Participants ranged in age from 18 to 32 years (M = 19.06, SD = 1.23) and primarily identified as White (73.01%), with 11.11% identifying as Chinese, 4.23% as Japanese, 2.12% as South Asian, 2.12% as Arab, 1.58% as Southeast Asian, 1.06% as Black, 1.06% as Latin American, 0.53% as West Asian, 0.53% as Korean, 0.53% as Filipino, and 2.12% as multi-racial. Most participants identified as heterosexual (80.95%), with 13.76% identifying as bisexual, 2.64% as lesbian, 1.06% as asexual, and 1.59% preferring not to

identify their sexual orientation. Participants represented a range of economic backgrounds, with 12.17% having an annual parental income of less than \$50,000, 19.05% with an annual parental income of \$51,000 to \$100,000, 26.45% with an annual parental income of \$101,000 to \$200,000, and 12.70% with an annual parental income of over \$200,000. Fifty-six participants (29.63%) did not provide data on annual household income.

Procedures

Inclusion criteria were being over the age of 18, being a first-year undergraduate student, identifying as a woman, and being able to understand English. After providing informed consent, participants completed an online survey at three time points throughout the academic year: the beginning of the Fall semester (T1), the beginning of the Winter semester (T2), and the end of the Winter semester (T3). Participants were offered course credit for the first time point and either course credit or entry into a cash draw for the second and third time points. Of note, due to very low retention at T3 (33.67%), only data from T1 and T2 were used.

Measures

Rejection Sensitivity Questionnaire – Personal 8-Item Version (RSQ; Downey &

Feldman, 1996). The 8-item RSQ is a modified version of the original 18-item RSQ (Downey & Feldman, 1996) and assesses respondents' concerns and expectations about interpersonal rejection. Participants are presented with eight hypothetical interpersonal scenarios (e.g., "You ask your boyfriend/girlfriend if he/she really loves you") and asked to use a 6-point scale to rate the extent to which they would be concerned about the outcome (1 = very unconcerned to 6 = very concerned) and how likely they would anticipate being rejected in the scenario (1 = very unconcerned to 6 = very likely). An overall score is derived by multiplying the concern score by the inverse of the anticipated rejection score for each item and taking the mean of the products. The

original RSQ has been shown to have good internal consistency ($\alpha = .83$) and test-retest reliability over a period of two-to-three weeks (r = .83; Downey & Feldman, 1996). Internal consistency in the current study was acceptable at T1 and good at T2 ($\alpha s = .77$ and .84, respectively). The RSQ also demonstrated good test-retest reliability over the four-month period between T1 and T2 (r = .76).

Ostracism Experience Scale for Adolescents (OES-A; Gilman et al., 2013). The OES-A is an 11-item self-report measure that assesses the frequency of respondents' experiences with being ignored and excluded. Respondents are asked to rate how often they have experienced scenarios such as "others have treated me as if I'm invisible" and "others have invited me to join them for weekend activities" (reverse scored) using a 5-point scale (1 = never to 5 = always). We modified the original instructions so that at T1, participants reported their lifetime experience with ostracism (i.e., "In general, throughout my life, others have...") and, at T2, their experience with ostracism during the previous two months (i.e., "In general, over the past two months, others have..."). The OES-A has been shown to have good construct and convergent validity in a sample of high-school students, as evidenced by correlations between ostracism scores and measures of victimization and social stress (rs = .24 - .66; Gilman et al., 2013). Although originally designed to be used in adolescent populations, the OESA has been used in samples of undergraduate students and was shown to have excellent internal consistency in this population $(\alpha s = .85 - .91; Cole et al., 2017)$. In the current study, the internal consistency estimate for the OESA total scale was good at both T1 and T2 (α s = .89 & .87, respectively).

Multidimensional Peer Victimization Scale (MPVS; Mynard & Joseph, 2000). The MPVS is a 16-item self-report measure designed to measure respondents' experiences with four types of peer victimization: physical victimization, verbal victimization, social manipulation, and

attacks on personal property. Sample items include "others have beat me up" and "others made fun of me for some reason". We modified the frequency rating scale to obtain a more nuanced estimated of victimization. Specifically, we replaced the original response options (0 = not at all, 1 = once, and 2 = more than once) with a 5-point scale (0 = never to 4 = always). We also modified the original instructions to obtain a lifetime estimate of victimization at T1 (i.e., "In general, throughout my life, others have...") and an estimate for the previous two months at T2 (i.e., "In general, over the past two months, others have..."). Although the MPVS includes a subscale for each type of victimization, only the total score was used in the current study. In a sample of adolescents, internal consistency was found to be adequate-to-good ($\alpha s = .73 - .85$) and convergent validity of the MPVS was evidenced by higher victimization scores for selfnominated victims compared to non-victims (Mynard & Joseph, 2000). In the current sample, internal consistency was good at T1 ($\alpha = .87$) and adequate at T2 ($\alpha = .76$).

Eating Pathology Symptoms Inventory (EPSI; Forbush et al., 2013). The EPSI is a self-report questionnaire designed to assess ED symptoms over the preceding four weeks. Participants are asked to provide frequency ratings for 45 items describing various ED symptoms using a five-point scale (0 = never to 4 = very often). The current study used the 8-item Binge Eating subscale. The Binge Eating subscale has demonstrated good internal consistency ($\alpha = .83$), good test-retest reliability over two-to-four weeks (r = .71), and excellent convergent and discriminant validity in student samples (Forbush et al., 2013). Internal consistency was good in the current study ($\alpha = .88$ for binge eating at both T1 and T2) and test-retest reliability between T1 and T2 was r = .72.

Eating Disorders Examination Questionnaire (EDE-Q; Fairburn & Beglin, 1994). The EDE-Q is a self-report version of the Eating Disorders Examination (Fairburn & Cooper, 1993) and assesses the frequency and severity of eating disorder psychopathology over the preceding four weeks. To capture the cognitive symptoms of EDs, the current study used the EDE-Q Weight Concern (5 items) and Shape Concern (8 items) subscales. Given that items from these subscales have been found to load on to a single factor (Allen et al., 2011; Peterson et al., 2007; Rand-Giovannetti et al., 2020), we averaged these subscales into a combined Weight/Shape concern score. In undergraduate women, the EDE-Q Weight Concern and Shape Concern subscales have demonstrated good-to-excellent internal consistency ($\alpha s = .89 - .93$) and good test-retest reliability over a two-week period (rs = .92 & .94, respectively; Berg et al., 2012). The EDE-Q is able to discriminate between participants with and without eating disorders, and correlates with other measures of eating pathology (rs = .31 - .79; Berg et al., 2012). In the current study, the combined Weight/Shape Concern subscale demonstrated excellent internal consistency at both T1 and T2 ($\alpha s = .94 & .95$, respectively) as well as good test-retest reliability from T1 to T2 (r = .80).

Body Mass Index (BMI). BMI was calculated based on participants' self-reported height and weight using the standard formula ((weight in pounds)/(height in inches²))*703. Mean BMI was in the normal weight range (M[SD] = 21.60 [3.20]; range = 15.26 – 39.54), with 9.04% of participant BMIs in the underweight range (BMI < 18.5), 79.26% in the normal weight range (BMI = 18.5-24.99), 9.57% in the overweight range (BMI = 25.0-29.99), and 2.13% in the obese range (BMI ≥ 30.0).

Statistical Analyses

All hypotheses and analyses were specified prior to data collection. Each survey included three attention check questions that instructed participants to select a specific response among the possible responses (e.g., "For this question, select 'very unlike me'). Ten participants (5.03%) were excluded from analyses for either incorrectly answering attention check questions or responding to the survey in a questionable manner (i.e., completing the survey in an unreasonably short amount of time or giving overly repetitive responses). Of the remaining 189 participants, 139 participants (73.54%) completed the second survey and 67 (33.67%) completed the third survey. Due to very low retention for T3, only data from T1 and T2 were used. The average time elapsed between questionnaire completion at T1 and T2 was 3.71 months. Chi-square tests and independent samples t-tests were conducted to determine whether participants with and without T2 data differed on demographic and baseline measures.

All variables were assessed for normality prior to analyses. All variables were found to be normally distributed, with the exception of BMI which was found to be leptokurtic (kurtosis = 6.61, SE = .35). An arctangent transformation was performed on BMI data prior to main analyses. Pearson's correlations were calculated using SPSS Statistical software (Version 24) to examine zero-order associations among RS, interpersonal stress, eating pathology, and BMI. Cross-sectional and longitudinal multiple-mediator path analyses were conducted in Mplus statistical software (Version 8; Muthén & Muthén). Missing data were accounted for in all path analysis models using full-information maximum likelihood estimation. Data from T1 were used in the cross-sectional model to examine the indirect effect of RS on binge eating and weight/shape concerns via ostracism and victimization. The longitudinal model examined the indirect effect of T1 RS on T2 binge eating and weight/shape concerns via T2 ostracism and victimization. T1 BMI was entered as a covariate in each model to examine effects independent of weight status. The longitudinal model additionally controlled for T1 eating pathology and T1 interpersonal stress. The significance of the indirect effect of rejection sensitivity on eating pathology via interpersonal stress was evaluated using 95% confidence intervals calculated from

5000 bootstrapped samples. Indirect effects were considered statistically significant if the 95% confidence intervals did not cross zero.

Results

Zero-order correlations and descriptive statistics are presented in Table 1. There were no significant differences between participants with and without T2 data on demographic and baseline measures, except for T1 binge eating scores. Specifically, participants who did not respond to the second survey had significantly higher T1 binge eating scores than participants who did respond to the T2 survey ($t_{186} = 2.17, p = .031$). As such, the range of binge-eating severity at T2 might not accurately reflect that of our initial sample (i.e., the upper end of the severity range might be under-represented in our T2 data).

The level of eating pathology in our sample differed somewhat from norms and previous samples. Specifically, whereas the mean baseline binge eating score in our sample was not significantly different from a previously established norm for undergraduate women ($t_{188} = 0.19$, p = .849; Forbush et al., 2013), scores were significantly higher for weight ($t_{187} = 2.05$, p = .042) and shape ($t_{187} = 2.45$, p = .015) concerns, compared to established norms for undergraduate women (Luce et al., 2008). Using the clinical cut-off of 4.0 for EDE-Q subscales (Luce et al., 2008), 20.21% of participants had clinically significant weight/shape concerns at T1.

At both time points, RS showed small-to-moderate positive correlations with both forms of interpersonal stress and small positive correlations with weight/shape concerns. RS was not significantly related to binge eating. There were small positive correlations between T1 ostracism and T1 weight/shape concerns, and between T2 ostracism and T2 weight/shape concerns, whereas ostracism was not associated with binge eating at either time point. Victimization showed small positive correlations with T2 binge eating and T1 weight/shape concerns, but there were no statistically significant associations between victimization and T1 binge eating or T2 weight/shape concerns. There were small-to-large positive correlations between ostracism and victimization across and within time points, with the exception of T1 ostracism and T2 victimization. BMI demonstrated small-to-moderate positive correlations with each form of eating pathology at both time points, but not with rejection sensitivity or with either form of interpersonal stress.

The results of the path analyses of direct and indirect effects for both the cross sectional and longitudinal samples are presented in Figures 1 and 2, respectively. The cross-sectional analysis revealed a significant direct effect (i.e., effect of RS controlling for interpersonal stress; $\beta_{direct} = 0.19, 95\%$ CI [0.01 – 0.31]) and significant total effect (i.e., the sum of direct and indirect effects; $\beta_{total} = 0.26, 95\%$ CI [0.10 – 0.36]) of RS on weight/shape concerns. The direct and total effects of RS on binge eating were not significant ($\beta_{direct} = 0.09, 95\%$ CI [-0.10 – 0.24]; $\beta_{total} = 0.12, 95\%$ CI [-0.02 – 0.25]). The specific indirect effects (i.e., indirect effects of each individual form of interpersonal stress on the association between RS and eating pathology) and the total indirect effect (i.e., the sum of both specific indirect effects) of RS on eating pathology via interpersonal stress were non-significant (see Figure 1). Results of the longitudinal analyses did not reveal significant direct or total effect of RS on weight/shape concerns ($\beta_{direct} = 0.04, 95\%$ CI [-0.07]; $\beta_{total} = 0.06, 95\%$ CI [-0.05 – 0.18]) or binge eating ($\beta_{direct} = -0.04, 95\%$ CI [-0.17 – 0.07]; $\beta_{total} = -0.05, 95\%$ CI [-0.16 – 0.06]). There were also no significant specific indirect or total indirect or total indirect effects of RS on significant specific indirect or total indirect or total specific indirect specific indirect specific indirect = -0.04, 95\% CI [-0.17 – 0.07]; $\beta_{total} = -0.05, 95\%$ CI [-0.16 – 0.06]). There were also no significant specific indirect or total indirect or total indirect effects of RS on significant specific indirect or total indirect or total specific indirect specific indirect or total specific indirect specific indirect or total specific indirect or total specific indirect specific indirect or total specific indirect specific indirect specific indirect or total specific indirect specific indirect specific indirect or total specific indirect specific indirect

Discussion

Study 1 examined the indirect effect of RS on eating pathology via interpersonal stress in a sample of first-year undergraduate women. At baseline, RS was directly associated with weight/shape concerns but not binge eating, although this finding was not replicated longitudinally. The association between RS and concurrent weight/shape concerns but not binge eating is in line with previous research (Atlas, 2004; Gilbert & Meyer, 2005). As suggested by Gilbert & Meyer (2005), it may be that individuals high in RS are initially concerned with improving their appearance in order to protect their social standing, although if this strategy fails, they may switch to binge eating and purging over time in order to cope with their negative affect. Contrary to our hypotheses, ostracism and/or peer victimization did not account for the effects of RS on eating pathology concurrently or longitudinally. In fact, there were no concurrent associations between interpersonal stress and eating pathology when controlling for RS and BMI. In the longitudinal model, while T1 RS was associated with ostracism experienced over the subsequent two months, this increased interpersonal stress was not associated with changes in eating pathology. Furthermore, controlling for baseline levels of eating pathology, neither form of eating pathology at T2 was associated with baseline RS or T2 interpersonal stress. The lack of association between interpersonal stress and eating pathology is surprising given extant literature supporting this association in undergraduate women (e.g., Beekman et al., 2017; Cain et al., 2010). It is possible that the lack of association between interpersonal stress and eating pathology is due to the use of a non-clinical sample. It may be that the hypothesized associations exist only in individuals with higher levels of eating pathology. Additionally, the lack of association across time may be due to minimal change in eating pathology across a short four-month period (see Measures). Study 2 sought to examine these possibilities by testing the indirect effect of RS on eating pathology via interpersonal stress using data collected from a sample of women with binge eating who were assessed twice across a longer time frame (i.e., approximately one-to-two years apart).

Study 2

Method

Participants

Participants were 77 women with binge eating who were recruited as part of a larger study on reward reactivity and binge eating. Of these women, 28 met diagnostic criteria for bulimia nervosa, 16 met criteria for binge eating disorder, one met criteria for anorexia nervosa binge eating/purging subtype, and 32 exhibited subthreshold (i.e., <1/week) binge eating. Participants ranged in age from 18 to 59 years (M = 24.57 years, SD = 8.46) and primarily identified as White (57.14%), with 10.38% identifying as Chinese, 5.19% as South Asian, 3.90% as Black, 3.90% as Arab, 2.60% as Korean, 2.60% as Latin American, 1.30% as West Asian, 1.30% as Japanese, 3.90% as 'Other Race', and 2.60% as multi-racial. Four participants (5.19%) did not provide information on racial background. Most participants identified as heterosexual (76.62%), with 16.88% identifying as bisexual, 2.60% as lesbian, 1.30% as asexual. Two participants (2.60%) did not identify their sexual orientation. Most participants (76.62%) indicated that they were students, 22.08% were employed non-students, and 1.30% reported that they were unemployed. Participants represented a range of economic backgrounds, with 35.06% having an annual household income of less than \$50,000, 22.07% with an annual household income of \$51,000 to \$100,000, 5.20% with an annual household income of \$101,000 to \$200,000, and 11.69% with an annual household income of over \$200,000 (n.b. participants identifying as students were asked to report parental income rather than their own household income). Twenty participants (25.98%) did not provide data on annual household income. **Procedures**

To determine initial eligibility, participants completed an online screening questionnaire assessing binge eating, mood, substance use, and psychotic symptoms as well as additional experimental inclusion criteria (e.g., normal or corrected-to-normal vision and hearing). Based on their responses, participants were contacted to undergo a telephone screening to further assess eligibility. Participants were eligible if they reported one or more objective binge eating episodes (i.e., consumption of an abnormally large amount of food in a short period of time accompanied by a loss of control) over the past 3 months. Participants experiencing a current major depressive or manic episode, current psychotic symptoms, or a current moderate-severe substance use disorder were ineligible.

As part of the larger study, eligible participants were invited to attend two laboratory sessions. In the first session, participants had their height and weight measured, tasted and rated several foods to be used for a task in the second session, and underwent structured clinical interviews administered by trained graduate students and research coordinators. The second session, approximately one week later, began with a standardized meal, followed by relaxation and imagery training, electrode attachment for facial myography and skin conductance, listening to imagery scripts, and completion of picture rating and button-pressing tasks while having physiology measured. Participants were asked to complete a battery of online questionnaires between laboratory visits (T1) and again approximately 12 to 24 months following their initial participation (T2). Only data from the questionnaires and the height and weight assessments are being used in the current study.

Measures

RSQ – Personal 8-Item Version (Downey & Feldman, 1996). The same RS measure used in Study 1 was used in Study 2. This measure has demonstrated good internal consistency

in a sample of women with disordered eating and a large mixed sample of women with and without disordered eating (α s = .85; De Paoli, Fuller-Tyszkiewicz, & Krug, 2017; De Paoli, Fuller-Tyszkiewicz, Halliwell, et al., 2017). In the current sample, the RSQ demonstrated good internal consistency (α = .80).

OESA (Gilman et al., 2013). Similar to the version we used for the student sample, we modified the original instructions so that at T1, participants reported their lifetime experience with ostracism (i.e., "In general, throughout my life, others have..."). It was our intention to modify the instructions at T2 so that participants were instructed to report on their experiences since T1. However, due to an error inputting the questionnaire into the online survey platform, the T2 version also asked about lifetime experiences. For this reason, only T1 OESA data were used. In the current study, the internal consistency estimate for the OESA total scale was good ($\alpha = .89$).

MPVS (Mynard & Joseph, 2000). Like the version used in the student sample, we replaced the original rating scale with a 5-point scale (0 = never to 4 = always) and modified the original instructions so that at T1, participants reported their lifetime experience with victimization. As with the OESA, it was our intention to modify the T2 instructions so that participants reported on victimization since T1, although the instructions were erroneously entered to ask about lifetime. As such, only T1 data were used. In the current sample, internal consistency of the MPVS total score was excellent ($\alpha = .91$).

Binge Eating Scale (BES; Gormally et al., 1982). The BES is a 16-item self-report questionnaire designed to assess behavioral, emotional, and cognitive symptoms of binge eating. The BES has previously demonstrated good internal consistency ($\alpha = .88$) and test-retest reliability across a one-month period (r = .84; Duarte et al., 2015). The BES has shown excellent sensitivity and specificity when comparing BES scores to interviewer-assessed binge eating in a sample of undergraduate women (Duarte et al., 2015), and BES scores correlate with frequency of objective and subjective binge episodes in women with binge eating (rs = .29 - .48; Timmerman, 1999). The BES demonstrated excellent internal consistency in the current sample at Time 1 and Time 2 ($\alpha s = .95$ & .94, respectively) and moderate stability across time points (r = .64).

Eating Disorders Examination Questionnaire (EDE-Q; Fairburn & Beglin, 1994).

Like Study 1, the current study used the Weight Concern and Shape Concern subscales and averaged these into a combined Weight/Shape Concern score. According to a psychometric review, the EDE-Q has demonstrated acceptable-to-excellent internal consistency ($\alpha s = .72 - .83$; Peterson et al., 2007) in a clinical sample and good test-retest reliability over a two-week period in individuals with binge-type eating disorders ($\rho s = .66 - .71$; Reas et al., 2006). In the current study, the combined Weight/Shape Concern subscale demonstrated excellent internal consistency at both time points ($\alpha s = .95 \& .96$) and moderate stability from T1 to T2 (r = .69).

Body Mass Index (BMI). In the first study session, height was measured using a wallmounted stadiometer and weight was measured with a digital scale. Weight was measured twice to ensure accurate measurement. If the two readings did not agree, additional measurements were conducted until two consistent readings were obtained. BMI was calculated based on these measurements using the standard formula described in Study 1. Mean BMI was in the overweight range (M[SD] = 25.63 [5.84]; range = 17.20 – 46.40), with 3.95% of participant BMIs in the underweight range (BMI < 18.5), 56.58% in the normal weight range (BMI = 18.5-24.99), 21.05% in the overweight range (BMI = 25.0-29.99), and 18.42% in the obese range (BMI ≥ 30.0).

Statistical Analyses

Data were examined for questionable responding (i.e., responding to questionnaires in an overly repetitive manner on in an unreasonably short period of time) and it was determined that all participants responded in an acceptable manner. We only used data from participants who had the opportunity to complete the questionnaires at both time points (i.e., those whose initial participation was at least one year prior to preparation of this manuscript). Retention at the second time point was adequate, with 58 participants (75.32%) completing the second battery of questionnaires. Average time between assessments was 18.94 months. Chi-square tests and independent samples t-tests were conducted to determine whether participants with and without T2 data differed on demographic and baseline measures.

All variables were assessed for normality and outliers prior to analyses and were found to be normally distributed. All hypotheses and analyses were specified prior to data collection and were identical to those performed in Study 1.

Results

Zero-order correlations and descriptive statistics are presented in Table 1. Participants with and without T2 data were not found to differ significantly on demographic or baseline measures. Baseline binge eating scores were, on average, significantly higher than population norms ($t_{75} = 18.66$, p < .001; Duarte et al., 2015) and similar to the mean score of treatmentseeking women from the original BES validation study ($t_{75} = -0.48$, p = .635; Gormally et al., 1982). Based on previously established cut-off scores (Marcus et al., 1985), 17.11% of our sample exhibited minimal binge eating (score ≤ 17), 44.73% of our sample exhibited mild to moderate binge eating (score of 18 - 26) and 38.16% exhibited severe binge eating (score ≥ 27) at baseline. T1 scores were significantly lower than those from a previous sample of community women with eating disorders (Mond et al., 2004) for both weight concerns ($t_{74} = -5.01$, p < .001) and shape concerns ($t_{74} = -5.06$, p < .001). Using a cut-off of 4.0, 28.00% of our sample exhibited clinically significant weight/shape concerns.

RS showed small-to-moderate positive correlations with both forms of interpersonal stress and both measures of eating pathology at T1 and T2. There was a small positive association between ostracism and T1 binge eating, but not T2 binge eating or weight/shape concerns at either time point. Victimization showed small-to-moderate positive correlations with binge eating at each time point and T2 weight/shape concerns. Ostracism and victimization were strongly correlated in a positive direction, as were binge eating and weight/shape concerns, at both time points. BMI demonstrated small positive correlations with binge eating at both time points and a moderate positive correlation with weight/shape concerns at T2, but not T1. BMI was not associated with rejection sensitivity or either of the interpersonal stress variables.

The results of the cross-sectional path analysis are presented in Figure 3. The crosssectional analysis revealed significant direct effects (i.e., the associations between RS and eating pathology, controlling for interpersonal stress) and total effects (i.e., the sum of all direct and indirect effects) of RS on weight/shape concerns ($\beta_{direct} = 0.35, 95\%$ CI [0.12 - 0.56]; $\beta_{total} =$ 0.36, 95% CI [0.16 - 0.54]) and binge eating ($\beta_{direct} = 0.40, 95\%$ CI [0.19 - 0.59]; $\beta_{total} = 0.42$, 95% CI [0.23 - 0.58]). The total indirect and specific indirect effects of RS on eating pathology via interpersonal stress were non-significant.

Results of the longitudinal analysis are presented in Figure 4. Analyses revealed a significant total effect of RS on T2 weight/shape concerns ($\beta_{total} = 0.26, 95\%$ CI [0.02 - 0.42]), but no direct effect ($\beta_{direct} = 0.22, 95\%$ CI [-0.09 - 0.48]). There were neither direct ($\beta_{direct} = 0.10, 95\%$ CI [-0.20 - 0.39]) nor total ($\beta_{total} = 0.10, 95\%$ CI [-0.15 - 0.32]) effects of RS on T2 binge

eating. Additionally, there were neither specific indirect nor total indirect associations between RS and either form of eating pathology at T2.

Discussion

Study 2 examined whether RS is indirectly associated with eating pathology via interpersonal stress in a sample of women with binge eating. Our hypotheses were not supported in either cross-sectional or longitudinal models – that is, there were no indirect associations between RS and eating pathology via either form of interpersonal stress. At baseline, however, RS was directly associated with both binge eating and weight/shape concerns. In other words, RS was associated with eating pathology, independent of interpersonal stress. While this finding was not replicated longitudinally, there was a total effect of RS on T2 weight/shape concerns, meaning that RS was associated with future weight/shape concerns via the combination of all possible pathways (i.e., the sum of all direct and indirect pathways). This suggests that RS may need to act via multiple pathways (i.e., directly and via interpersonal stress) in order to influence future eating pathology. As in Study 1, there were no associations between ostracism or rejection and eating pathology in path models, which contradicts previous research demonstrating a link between interpersonal stress and disordered eating in community and clinical samples (Ansell et al., 2012; Ivanova, Tasca, Hammond, et al., 2015; Ivanova, Tasca, Proulx, et al., 2015; Steiger et al., 1999). This finding brings into question the role of peer stress in eating pathology and highlights the importance of considering variables, such as RS, that might account for previously observed associations between peer stress and disordered eating.

General Discussion

Previous research has identified RS as a potential maintenance factor for eating pathology (Bondü et al., 2020; Gilbert & Meyer, 2005), although the mechanisms through which RS is

associated with eating pathology have not yet been established. The goal of this study was to examine interpersonal stress (i.e., ostracism, peer victimization) as a possible mechanism linking RS and eating pathology. Given previous research showing that individuals high in RS tend to behave in ways that promote interpersonal problems (Downey et al., 1998, 2004; London et al., 2007; Purdie & Downey, 2000; Watson & Nesdale, 2012; Zimmer-Gembeck & Nesdale, 2013), and that interpersonal stress is associated with eating pathology (Wilfley et al., 2003), we hypothesized that RS would be associated with more frequent experiences of ostracism and victimization, which in turn would be associated with elevated eating pathology (i.e., binge eating and weight/shape concerns). Our primary hypothesis was not supported in samples of undergraduate students and women with binge eating, although other notable findings emerged: RS was directly associated with weight/shape concerns in both samples and with binge eating in women with this form of disordered eating.

The finding that interpersonal stress was not a pathway via which RS is associated with eating pathology appears to be driven by a lack of association between interpersonal stress and eating pathology within path models. In both samples, there were some small significant bivariate correlations between interpersonal stress and eating pathology, both within and across time points. However, within path models, these associations were not significant, suggesting that when RS is accounted for, the association between interpersonal stress and eating pathology with both victimization and ostracism. Ostracism has been associated with increased consumption of palatable food by undergraduate students in laboratory studies (Baumeister et al., 2005; Hayman et al., 2015; Oaten et al., 2008), whereas peer victimization has been found to be associated with binge eating in samples of children and emerging adults (Copeland et al., 2015; Markou et al.,

2021), and with weight/shape concerns in adolescents (Day et al., 2021; Trompeter et al., 2022). Importantly, only two of the aforementioned studies included RS in their analyses. Oaten and colleagues (2008) examined dysregulated eating in response to induced ostracism in the laboratory and found that, while all ostracised participants initially ate more palatable food compared to non-ostracised participants, only those who reported a fear of negative evaluation continued to demonstrate dysregulated eating 45 minutes later. Trompeter and colleagues (2022) found that, concurrently, RS attenuated the association between victimization and weight/shape concerns (i.e., the association was weaker at higher levels of RS) and that, over time, there was no association between peer victimization and increases in weight/shape concerns when controlling for RS. Taken together with our results, these findings bring into question the role of peer stress in disordered eating and suggest that RS may be a more important predictor of eating pathology.

In both samples, RS was directly associated with weight/shape concerns at T1. This finding is consistent with the extant literature (e.g., Calogero et al., 2010; Lavell et al., 2014; Linardon et al., 2017; Park, 2007) and further suggests that the anxious anticipation of interpersonal stress is associated with weight/shape concerns regardless of actual experiences of ostracism or victimization. This is in line with previous findings in which the effect of victimization on body dysmorphic symptoms was either attenuated or no longer significant after controlling for appearance-based RS (Lavell et al., 2014; Trompeter et al., 2022). Taken together, it may be that individuals become preoccupied with their appearance to pre-empt interpersonal stress rather than recover from it. That is, rather than attempting to repair social connections following ostracism or victimization, individuals high on rejection sensitivity may take a proactive stance and become preoccupied with their appearance to increase social worth as

a means of garnering acceptance. This suggestion is similar to that proposed by the authors of a study examining associations between fear of negative evaluation and eating disorder symptoms in undergraduate women. This study found that fear of negative evaluation was associated with concurrent, but not future, drive for thinness, and with future, but not concurrent bulimic symptoms (Gilbert & Meyer, 2005). The authors suggest that initially, individuals may become preoccupied with controlling their appearance as a means of increasing social status, but if this strategy does not work, individuals shift to alternative coping mechanisms (i.e., binge eating and purging) over time. This latter point may help to explain why we did not find a longitudinal association between RS and weight/shape concerns in either sample, although we also did not find a longitudinal association with binge eating.

The finding that, cross-sectionally, there was a direct effect of RS on binge eating in the clinical sample adds to previous research linking RS with binge eating in community women, students, and adolescents (Linardon et al., 2017; Selby et al., 2010; Yoon et al., 2014). Theory and evidence suggest that binge eating episodes follow experiences of negative affect and that individuals who engage in binge eating may do so because they lack more adaptive ways of regulating their affect (for a review, see Dingemans et al., 2017). Women who have established patterns of binge eating may rely on binge eating episodes to cope with the negative affect that stems from anticipated social threat although, notably, this finding was not replicated longitudinally and therefore we cannot speak to the direction of the association between RS and binge eating. An alternative explanation is that women who binge eat are more rejection sensitive as a result of their binge eating, which might occur if they experience or perceive negative social evaluation based on their eating behavior or appearance (Albano et al., 2019).

In general, we did not detect prospective associations between RS, interpersonal stress,

and disordered eating, with the exception of a combined effect of RS and interpersonal stress on changes in weight/shape concerns in our clinical sample. It is possible that we were not able to observe most associations detected in the cross-sectional analysis due to there being minimal change in eating pathology from T1 to T2 in both the student (rs = .72 - .80) and clinical (rs =.64 - .69) samples. That is, when controlling for T1 eating pathology, there was very little variance left to be explained by RS and interpersonal stress, particularly in the student sample. Given the numerous changes and stressors associated with starting university, we expected to see more variability in eating pathology across time points in the student sample. However, previous studies have also found stability in eating pathology among undergraduate women over periods of seven months to three years (Cooley & Toray, 2001a, 2001b). With regards to the clinical sample, the lack of longitudinal associations could also have been influenced by the onset of the COVID-19 pandemic and subsequent lockdown measures that occurred between measurement points, during which participants may not have been not exposed to the types of social stressors examined in this study. It may be that their disordered eating symptoms were associated with other social stressors during this time, such as loneliness or lack of social support (Racine et al., 2022; Rodgers et al., 2020).

To our knowledge, this was the first study to examine the indirect effect of RS on eating pathology via interpersonal stress. Strengths include the examination of two forms of interpersonal stress, two forms of eating pathology, and both an undergraduate and clinical sample, providing additional confidence in our pattern of results. Additionally, our sample was relatively diverse with regards to ethnicity, sexual orientation, and economic status, thus increasing the generalizability of our findings. Our results should also be considered in light of some limitations. First, while we used both cross-sectional and longitudinal data, we were only able to use data collected at two time points in the student sample, and only collected two waves of data in the clinical sample, thus not allowing us to examine a full mediation model (which requires three times points) or make inferences about the direction of causality. Furthermore, in Study 2, we only had a measure of lifetime interpersonal stress, making it impossible to determine the direction of the association between these variables with RS or eating pathology. Second, our sample was exclusively female and therefore we cannot speak to the applicability of our model to individuals who do not identify as female. Males and gender-diverse individuals may differ in their experiences of and reactions to interpersonal stress, as well as in their presentations of RS and eating pathology (Carbone-Lopez et al., 2010; Gordon et al., 2021; Rudolph, 2002; Strother et al., 2012; Wells et al., 2020) and thus replicating our findings in these populations is warranted.

Findings from our study help elucidate the association between RS and eating pathology and may have implications for clinical practice. Specifically, they highlight the importance of RS in concurrent eating pathology, suggesting that interventions targeting RS may be beneficial additions to eating disorder treatment. Several types of interventions exist that may mitigate the direct impact of RS on eating pathology. First, positive interpretation bias training, which involves increasing positive or realistic interpretations of ambiguous scenarios, has been shown to reduce negative interpretations of social scenarios and increase self-esteem in individuals with anorexia nervosa (Cardi et al., 2019). Second, interventions aimed at improving self-esteem may be used to counteract the effects of RS and offer more adaptive ways to foster social confidence. A recent meta-analysis identified cognitive behavioral therapy, art therapy, and interventions based in mindfulness and relaxation to be most effective at increasing self-esteem in adults (Niveau et al., 2021). Finally, social skills training may help increase confidence in social interactions, thus mitigating the impact of RS. Effective social skills have been shown to be protective against disordered eating (Uzunian & Vitalle, 2015), and interventions aimed at increasing social skills have been identified as effective components of eating disorder treatments (Cardi et al., 2018). In summary, RS appears to be associated with eating pathology in student and clinical samples, and further investigation into the nuances of this association may benefit our understanding of and approaches to treatment of disordered eating.

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Article 2 Tables and Figures

Table 1

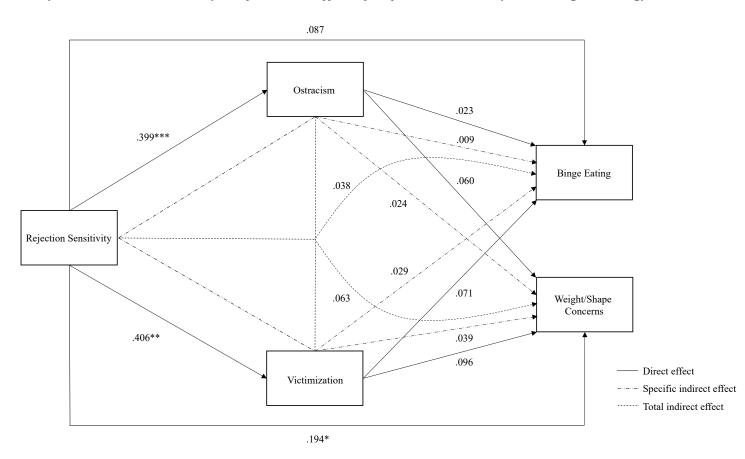
Study 1 Zero-Order Correlations and Descriptive Statistics

Variable	1	2	3	4	5	6	7	8	9	10
1. Rejection Sensitivity	-									
2. T1 Ostracism	.40***	-								
3. T2 Ostracism	.47***	.68***	-							
4. T1 Victimization	.41***	.38***	.39***	-						
5. T2 Victimization	.20*	.15	.21*	.51***	-					
6. T1 Binge Eating	.13	.05	.10	.10	.14	-				
7. T2 Binge Eating	.10	.16	.13	.19*	.18*	.72***	-			
8. T1 Weight/Shape Concerns	.27***	.16*	.15	.19*	.18*	.54***	.41***	-		
9. T2 Weight/Shape Concerns	.22**	.16	.22**	.16	.17	.46***	.52***	.80***	-	
10. BMI	.10	01	08	.04	.01	.17*	.24**	.40***	.35***	-
Mean	9.23	2.13	2.16	12.75	4.18	9.71	8.65	2.40	2.17	21.60
SD	4.06	0.70	0.70	7.40	4.10	6.49	6.22	1.65	1.57	3.20

 $\overline{Note. ***p < .001, **p < .01, *p < .05; T1 = Time 1, T2 = Time 2.}$

Figure 1

Study 1 Cross-Sectional Analysis of Indirect Effect of Rejection Sensitivity on Eating Pathology via Peer Stress

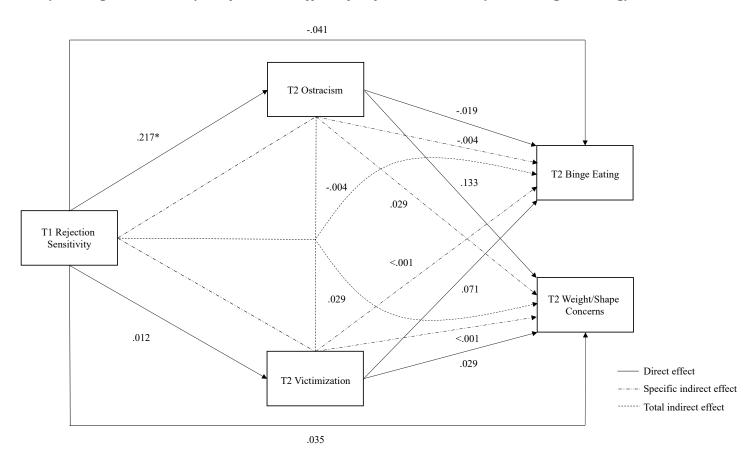


Note. Parameters displayed are standardized estimates of the direct and indirect effect of each pathway at Time 1. Body mass index included as a covariate in all paths.

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

Figure 2

Study 1 Longitudinal Analysis of Indirect Effect of Rejection Sensitivity on Eating Pathology via Peer Stress



Note. Parameters displayed are standardized estimates of the direct and indirect effects of each pathway. T1 = Time 1, T2 = Time 2. Body mass index, Time 1 interpersonal stress, and Time 1 eating pathology variables were included as covariates in all paths. ***p < .001, **p < .01

Table 3

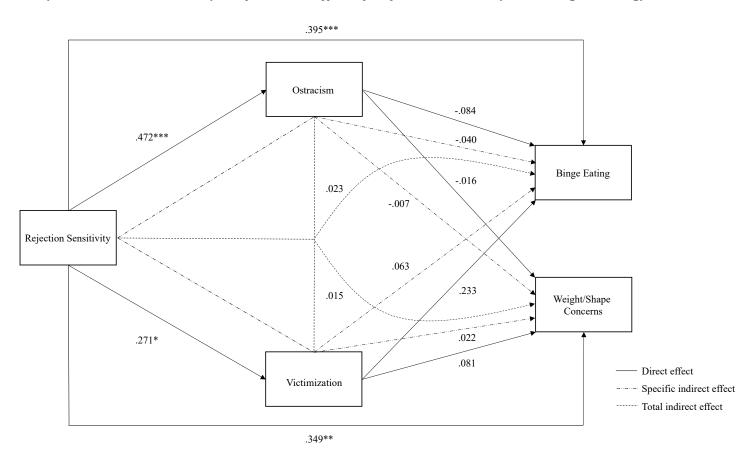
Study 2 Zero-Order	Correlations an	nd Descriptive Statistics
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Study Variable	1	2	3	4	5	6	7	8
1. Rejection Sensitivity	-							
2. Ostracism	.45***	-						
3. Victimization	.28*	.57***	-					
4. Time 1 Binge Eating	.45***	.25*	.31**	-				
5. Time 2 Binge Eating	.27*	.17	.26*	.64***	-			
6. Time 1 Weight/Shape Concerns	.38**	.21	.20	.72***	.62***	-		
7. Time 2 Weight/Shape Concerns	.34*	.23	.30*	.58***	.83***	.69***	-	
8. BMI	.09	.08	.08	.25*	.29*	.20	.32*	-
Mean	9.67	2.38	0.80	25.14	20.68	2.95	3.18	25.63
SD	4.52	0.62	0.55	8.50	9.54	1.47	1.60	5.84

Note. ****p* < .001, ***p* < .01, **p* < .05

Figure 3

Study 2 Cross-Sectional Analysis of Indirect Effect of Rejection Sensitivity on Eating Pathology via Peer Stress

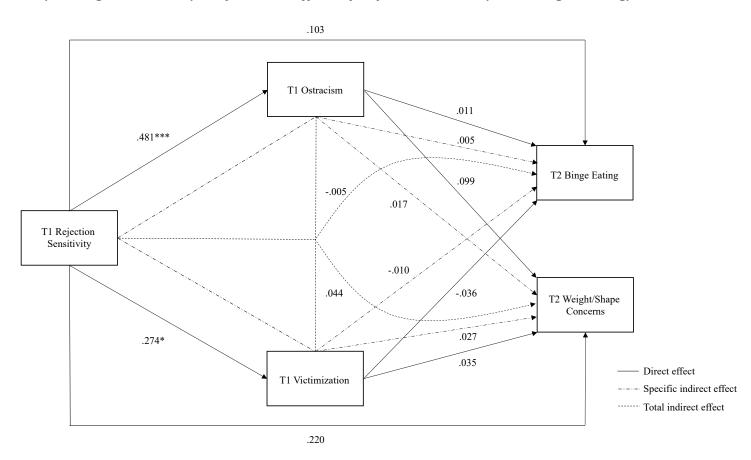


Note. Parameters displayed are standardized estimates of the direct and indirect effects of each pathway at Time 1. Body mass index included as a covariate in all paths.

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

Figure 4

Study 2 Longitudinal Analysis of Indirect Effects of Rejection Sensitivity on Eating Pathology via Peer Stress



Note. Parameters displayed are standardized estimates of the direct and indirect effects of each pathway. T1 = Time 1, T2 = Time 2. Body mass index and Time 1 eating pathology variables were included as covariates in all paths.

****p* < .001, ***p* < .01

Bridge to Article 3

In Article 2, I sought to investigate whether RS was associated with disordered eating via elevated peer stress in samples of undergraduate women and women with binge eating. That is, I expected that being hypersensitive to social threat would be associated with elevated rates of lifetime ostracism and victimization, and that this elevated peer stress would in turn be associated with increased binge eating and weight/shape concerns, both concurrently and across time. I did not find that RS was indirectly associated with eating pathology via peer stress, either concurrently or across time, in either sample. However, I found that RS was *directly* associated with concurrent eating pathology in both samples. My findings suggested that RS may be a more important correlate of concurrent eating pathology than actual experiences of peer stress.

Taken together with the results of Article 1 in which eating pathology was associated with maladaptive interpersonal behaviour regardless of actual experiences of rejection, these findings brought into question the role of interpersonal stress in disordered eating. I considered that the association between peer stress and eating pathology may be best elucidated by examining indirect, rather than direct pathways. As such, the goal of Article 3, was to investigate a novel indirect pathway through which peer stress may be associated with eating pathology. Specifically, I sought to test the indirect association between peer stress with binge eating and dietary restriction via social anhedonia and the possibility that this association would be more prominent in individuals who had positive beliefs about the outcomes of eating or how others view their appearance.

Article 3

Examining Altered Social Reward Processing as a Mediator of the Association Between

Interpersonal Stress and Disordered Eating

Article 3 Abstract

Poor interpersonal functioning has been implicated in the etiology and maintenance of eating pathology. According to the Interpersonal Psychotherapy Model for Eating Disorders, interpersonal stress leads to negative self-evaluation and associated negative affect, which then lead to disordered eating behaviours as an attempt to regulate negative affect. While there is empirical evidence supporting this model, interpersonal stress does not always lead to negative affect, and mood states other than negative affect are associated with disordered eating. As such, it is likely that negative affect is not the only pathway via which interpersonal stress influences eating pathology. The goal of this study was to consider an additional possible pathway: altered responsiveness to social reward. Chronic experiences of interpersonal stress may lead to a decrease in the anticipatory reward or pleasure that individuals experience from peer relationships, leading individuals to seek reward from other sources. As such, the current study examined whether peer stress was indirectly associated with eating pathology via social anhedonia. Using longitudinal self-report data from a sample of 189 undergraduate women, there was no indirect association between two forms of peer stress (ostracism and victimization) and eating pathology via social anhedonia. However, victimization (but not ostracism) was *directly* associated with changes in eating pathology across the first semester of university, suggesting that a history of peer victimization is associated with worsening eating pathology over time. Results also highlight the importance of examining distinct forms of peer stress, as they appear to differentially relate to eating pathology.

Examining Altered Social Reward Processing as a Mediator of the Association Between Interpersonal Stress and Disordered Eating

Among the various social and emotional influences associated with eating disorders (EDs), poor interpersonal functioning has been implicated as a key contributing and maintaining factor to disordered eating behaviour, including binge eating and dietary restriction (e.g., Monteleone et al., 2018; Rieger et al., 2010). Research across various populations and methodologies has shown that interpersonal stressors precede both over-consumption of food (e.g., Hayman et al., 2015; Monteleone et al., 2018; Salvy et al., 2012) and dietary restriction (Beekman et al., 2017; Cain et al., 2010; Cardi et al., 2018). Elucidating the mechanisms through which interpersonal stress influences disordered eating may contribute to improved treatment efficacy, particularly since interpersonal stress is also associated with poorer treatment response in individuals with EDs (Agras et al., 2000; Hartmann et al., 2010).

One model that offers an explanation of how interpersonal stress leads to disordered eating is the Interpersonal Psychotherapy Model for Eating Disorders (IPT-ED; Rieger et al., 2010). Mainly applied to binge eating disorder (BED), the IPT-ED model posits that interpersonal stress leads to negative self-evaluation and associated negative affect, which then leads to disordered eating behaviours as a means of regulating negative affect. Results of several studies in both clinical and community samples have provided support for the IPT-ED model in relation to binge eating. In a cross-sectional study of women with BED, increased interpersonal stress was indirectly associated with greater binge eating and related psychopathology (e.g., ED cognitions and motivations) via negative affect (Ivanova et al., 2015). Using experience sampling methodology, Steiger and colleagues found that, in women with bulimia nervosa (BN), binge eating episodes were often preceded by negative social interactions (e.g., interactions that were cold, hostile, rejecting, etc.) and negative affect (Steiger et al., 1999). Negative affect has also been found to mediate the association between interpersonal stress and binge eating in healthy adult women (Ansell et al., 2012), and between interpersonal stress and loss of control eating in healthy children and adolescents (Elliott et al., 2010). Although the interpersonal model has been primarily examined in relation to binge eating, a study in a large sample of treatmentseeking individuals found support for its validity across eating disorder subtypes (BED, BN, anorexia nervosa (AN) – restrictive eating subtype (AN-R), and eating disorder not otherwise specified; Ivanova et al., 2015). Specifically, interpersonal stress was indirectly associated with general ED psychopathology (i.e., dietary restraint and concerns about eating, shape, and weight) via negative affect across diagnostic subtypes.

Taken together, the aforementioned findings suggest that negative affect plays an important role in the association between interpersonal stress and disordered eating. However, there is some evidence to suggest that interpersonal stress is not always associated with negative affect, and that non-negative mood states are also associated with disordered eating. First, a meta-analysis on the effect of interpersonal rejection on mood found that rejection is more likely to lead to a shift towards a neutral mood state (i.e., low levels of both positive and negative affect; Blackhart et al., 2009). Second, it has been shown that, in addition to negative affect, positive affect also fluctuates around eating episodes. For example, feeling self-assured (a facet of positive affect) has been found to increase following restrictive eating in individuals with AN-R, and to increase prior to, and remain stable following, restrictive eating in individuals with AN-D binge eating-purging subtype (AN-BP; Haynos et al., 2017). These findings suggest that negative affect may not be the only pathway through which interpersonal stress leads to

disordered eating symptoms. In line with this, Treasure and colleagues have suggested that, for some, disordered eating may instead be aimed at increasing positive affect when faced with stressors (Treasure et al., 2018). Specifically, these authors posit that when other domains of life are chronically stressful and fail to provide the opportunity for pleasurable or rewarding experiences, eating may be used as a compensatory source of reward.

In an interpersonal context, there is evidence that repeated stress might not only represent a decrease in the quality of an individual's social experiences, but may in fact also lead to a reduction in that individual's ability to anticipate future positive social experiences or to enjoy social experiences that might otherwise be rewarding or pleasurable (i.e., social anhedonia). For example, compared to non-lonely participants, lonely participants showed less activation in brain areas associated with reward processing when viewing positive social stimuli (Cacioppo et al., 2009) and described their social experiences as less positive, independent of depressed mood (Hawkley et al., 2003, 2007). Similarly, a history of peer victimization has been associated with reduced neural responding to peer acceptance in late adolescence and emerging adulthood (Rappaport et al., 2019). The inability to anticipate or experience pleasure from social experiences may further motivate individuals to seek sources of reward elsewhere, such as through binge eating or restrictive eating.

Disordered eating has been shown to be associated with indices of altered social reward. Compared to healthy controls, patients with AN find faces less rewarding (Watson et al., 2010), show an attentional bias away from accepting faces and towards rejecting faces (Cardi et al., 2013), and show less activation in brain areas involved with social processing when receiving positive, but not negative, social feedback (Via et al., 2015). Patients with both AN and BN selfreport higher levels of social anhedonia compared to healthy controls (Harrison et al., 2014; Tchanturia et al., 2012) and, in a mixed sample of patients with EDs, higher social anhedonia scores were associated with greater drive for thinness (Eiber et al., 2002). Of note, one study in a sample of female undergraduate students produced mixed evidence for associations between responsiveness to social positive social feedback and disordered eating. Specifically, dietary restraint and binge eating/purging symptoms were both associated with *increased* self-reported sensitivity to positive social feedback. However, when using a button-pressing task that assessed willingness to work for positive social feedback, dietary restraint was associated with increased with increased with decreased willingness to work for social feedback, whereas binge eating/purging symptoms were associated with decreased willingness to work for social feedback (Fussner et al., 2018). As evidenced by this study, different ED features and presentations may be differentially related to social reward responsiveness, pointing to the importance of examining individual ED symptom dimensions in the context of social anhedonia.

If social experiences cease to be rewarding or pleasurable, and individuals begin seeking alternate sources of reward in other domains of life, they may engage in behaviours that they believe are likely to be rewarding based previous experience or expectancies. In the case of individuals with disordered eating, two such candidates are binge eating and dietary restriction. For example, if individuals have the expectation that consuming palatable food during binge eating episodes or controlling appearance via restricting food intake will be pleasurable or rewarding, they may be more likely to engage in those behaviours to make up for the reward deficit left by unsatisfactory social interactions. Conversely, individuals who do not have such expectancies may seek sources of reward elsewhere, for example by using drugs and alcohol or gambling. With regards to binge eating, there is evidence that using eating as a source of reward is associated with binge eating and related constructs in both clinical and non-clinical samples. In one study, patients with BN and BED reported greater eating to enhance reward than healthy weight controls and overweight controls, respectively (Leslie et al., 2018). Eating for reward enhancement is also associated with disordered eating symptoms in undergraduate samples. Specifically, eating to enhance reward has been associated with higher levels of binge eating (Boggiano et al., 2014; Burgess et al., 2014; Schell et al., 2019), food addiction (Burgess et al., 2014), emotional eating (Boggiano et al., 2015a), and eating palatable food in the absence of hunger (Boggiano et al., 2015b).

To our knowledge, there is no research on the association between dietary restriction and reward enhancement. It is unlikely that eating would be viewed as a potential source of reward in individuals with eating pathology primarily characterized by dietary restriction. However, it is possible that other aspects of eating pathology, such as the effect of dietary restriction on weight or shape, may be viewed as positive or rewarding by these individuals. There is indeed evidence that positive expectancies about the outcome of dieting and thinness (e.g., on self-esteem, selfimprovement) are associated with restrictive eating patterns. In undergraduate women, these expectancies are associated with dietary restraint both concurrently (Hohlstein et al., 1998) and prospectively (Stojek & Fisher, 2013), and women with AN have been found to endorse these expectancies more than psychiatric or healthy controls (Hohlstein et al., 1998). Additionally, there is evidence to suggest that the outcome of dietary restriction on body weight/shape (i.e., extreme thinness) is rewarding to women with EDs. For example, when shown pictures of emaciated women, adolescents with AN demonstrated an appetitive response (indicated by a reduced startle reflex), in contrast to healthy controls who showed an aversive response (Reichel et al., 2014). Women with AN have also been found to experience greater activation in the ventral striatum (a brain area associated with reward processing) and to provide more positive

ratings when viewing underweight versus normal weight bodies, whereas the reverse pattern was found in healthy women (Fladung et al., 2010). Finally, compared to healthy women, female patients with AN demonstrate an attentional bias away from faces and towards bodies, particularly emaciated bodies (Watson et al., 2010).

Taken together, this review of the literature suggests that an interesting area for investigation is examining whether changes in reward responsiveness mediate the association between interpersonal stress and disordered eating – that is, whether chronic interpersonal stress is associated with lower responsiveness to social reward (i.e., social anhedonia), which then leads to disordered eating as a compensatory reward seeking strategy. The goal of the current study was to examine this gap in the literature within the following overarching model: we hypothesized that interpersonal stress (i.e., experiences of ostracism and victimization) would be associated with core symptoms of EDs (i.e., binge eating and dietary restriction) via decreased responsiveness to social reward. Furthermore, we hypothesized that this indirect association would be moderated by indices of reward related to eating and appearance. Specifically, we hypothesized that the pathway from social anhedonia to binge eating would be moderated by the expectancy that eating is rewarding (eating reward expectancy), such that the association would be stronger at higher levels of this eating expectancy. We also hypothesized that the association between social anhedonia and dietary restriction would be moderated by the tendency to experience positive affect in response to verbal commentary on one's weight and shape (appearance reward), such that the association would be stronger at higher levels of appearance reward.

Method

Participants & Procedure

Participants were 189 undergraduate women aged 18 to 32 years (M = 19.06, SD = 1.23) enrolled in a psychology course at a North American university (see Table 1 for additional demographic information). In order to take part in the study, participants had to be over the age of 18 years, identify as a woman, be enrolled in their first year of undergraduate studies, and be able to understand English. Participants provided informed consent and were invited to complete a battery of online questionnaires at three time points: the beginning of the Fall semester (T1), the beginning of the Winter semester (T2), and the end of the Winter semester (T3). Participants were offered course credit for the first time point, and either course credit or entry into a cash draw for the second and third time points. Due to inadequate retention at T3 (33.67%), only data from T1 and T2 were used.

Measures

Eating Pathology Symptoms Inventory (EPSI; Forbush et al., 2013)

The EPSI is a 45-item self-report questionnaire designed to measure ED symptoms during the past four weeks. Participants are asked to rate how frequently they experience various ED symptoms using a five-point scale (0 = never to 4 = very often). The 8-item Binge Eating and 6-item Restricting subscales were used for the current study: example items include "I stuffed myself with food to the point of feeling sick" and "I skipped two meals in a row", respectively. The Binge Eating and Restricting subscales have demonstrated good internal consistency in both student ($\alpha s = .71 - .83$) and clinical samples ($\alpha s = .86 - .93$), and good test-retest reliability over two-to-four weeks in a student sample (rs = .71 - .75; Forbush et al., 2013). These subscales have also shown excellent convergent and discriminant validity (Forbush et al., 2013). Internal consistency estimates in the current study were good ($\alpha = .88$ for both binge eating at T1 and T2; $\alpha s = .83$ & .85 for T1 and T2 restricting, respectively).

Ostracism Experience Scale for Adolescents (OES-A; Gilman et al., 2013)

The OES-A includes 11 items measuring the frequency of respondents' lifetime experiences with ostracism across two domains: being ignored and being excluded. Using a 5point scale (1 = never to 5 = always), respondents are asked to rate statements such as: "others have ignored me during conversations" and "others have invited me to go out to eat with them" (reverse scored). The original validation study provided evidence of good construct and convergent validity in a sample of high-school seniors, as shown by correlations between the Ignored and Excluded subscales with measures of social stress and victimization (Gilman et al., 2013). The original validation paper did not report estimates of internal consistency, although in a study of undergraduate students, Cronbach's alphas ranged from .85 to .91. (Cole et al., 2017). The current study used the OES-A total score, and the internal consistency estimate was good (α =.89).

Multidimensional Peer Victimization Scale (MPVS; Mynard & Joseph, 2000)

The MPVS is a 16-item self-report measure designed to measure respondents' experiences with peer victimization across four domains: physical victimization, social manipulation, verbal victimization, and attacks on personal property. Respondents are asked to report the frequency with which they have experienced specific forms of peer victimization (e.g., "others have refused to talk to me" and "others have called me names") throughout their lives, using a 5-point scale (0 = never to 4 = always). In a sample of adolescents, internal consistency estimates ranged from adequate-to-good ($\alpha s = .73 - .85$), and convergent validity was demonstrated by significantly higher scores on each subscale for self-nominated victims compared to non-victims (Mynard & Joseph, 2000). In the current sample, internal consistency for the MPVS total scale was good ($\alpha = .87$).

Revised Social Anhedonia Scale (RSAS; Eckblad, Chapman, Chapman, & Mishlove, 1982)

The RSAS is a 40-item measure designed to assess lack of pleasure from social experiences using a true/false response format. Example items include: "Making new friends isn't worth the energy it takes" and "People often expect me to spend more time talking with them than I would like". Although originally designed to assess social anhedonia in individuals with schizotypal personalities, the RSAS has also been used in research on EDs (e.g., Tchanturia et al., 2012). The RSAS has been found to have adequate internal consistency in undergraduate students ($\alpha = .79$; Eckblad et al., 1982) and participants with EDs ($\alpha = .83$; Tchanturia et al., 2012). The RSAS correlates with measures of friendship, social engagement, and social discomfort, providing evidence for construct validity (Mishlove & Chapman, 1985). Internal consistency in the current sample was good ($\alpha = .82$).

Eating Expectancies Scale (EEI; Hohlstein, Smith, & Atlas, 1998)

The EEI is a 34-item self-report measure designed to assess expectancies about the outcome of eating using a 7-point scale (1 = *completely disagree* to 7 = *completely agree*). The 6-item Eating is Pleasurable and Useful as a Reward subscale was used for the current study and includes items such as "Eating is fun and enjoyable" and "Eating is a good way to celebrate". This subscale has previously demonstrated adequate internal consistency (α = .78), correlates with measures of ED symptoms, and distinguishes between individuals with and without an ED diagnosis (Hohlstein et al., 1998). Internal consistency of the Eating is Pleasurable and Useful as a Reward subscale in the current sample was acceptable at T2 (α = .79).

Verbal Commentary on Physical Appearance Scale (VCOPAS; Herbozo & Thompson, 2006)

The VCOPAS is a 21-item self-report measure designed to assess how often respondents received specific verbal comments about their appearance (e.g., "I wish I had a body like yours"

and "You are pretty") during the past two months, using a 5-point scale (1 = never to 5 = always). If participants endorse receiving a particular comment on their appearance, they are asked to rate how it made them feel using a 5-point scale (1 = very positive to 5 = very negative). Therefore, it provides both frequency and reaction scores across three subscales: Negative Weight and Shape, Positive Weight and Shape, and Positive General Appearance. Only the frequency score for the Positive Weight and Shape subscale was used in the current study. In samples of undergraduate students, this subscale has demonstrated acceptable internal consistency ($\alpha = .72$), excellent test-retest reliability over a two-week period (r = .91), and convergent validity as demonstrated by significant correlations with measures of appearance feedback and body image (Herbozo & Thompson, 2006). Internal consistency of the Positive Weight and Shape was good at T2 ($\alpha = .82$).

Body Mass Index (BMI)

BMI was calculated based on participants' self-reported height and weight using the standard formula ((weight in pounds)/(height in inches²))*703.

Statistical Analyses

Prior to analyses, data integrity was assessed by examining responses to attention check questions (e.g., "For this question, select 'very unlike me') and patterns of responding. Ten participants (5.03%) were found to have incorrectly answered the attention check questions or to have responded in a questionable manner (i.e., giving overly repetitive responses or completing the measures in an unreasonably short time). Of the remaining 189 participants, 139 participants (73.54%) provided data at T2 and 67 (33.67%) responded at T3. Due to inadequate retention at T3, only data from T1 and T2 were used. The average time elapsed between questionnaire completion at T1 and T2 was 3.71 months. Chi-square tests and independent samples t-tests were

conducted to evaluate whether participants with and without T2 data differed on demographic and baseline measures. Of note, measures for the moderator variables – The EEI and VCOPAS – were only added to the questionnaire package once half of the sample had already responded to the T1 questionnaires. As such, we used moderator variables scores from the T2 assessment.

All variables were assessed for normality prior to analyses. Appearance reward was found to be leptokurtic (kurtosis = 4.16, SE = .45), as was BMI (kurtosis = 6.61, SE = .35). As such, an arctangent transformation was performed on these variables. All other variables were found to be normally distributed. Pearson's correlations were calculated using SPSS Statistical software (Version 24) to examine zero-order associations between interpersonal stress, eating reward expectancy, appearance reward, and eating pathology.

Longitudinal moderated-mediation path analyses were conducted in Mplus statistical software (Version 8; Muthén & Muthén). Missing data were accounted for in all path analysis models using full-information maximum likelihood estimation. We examined the indirect effect of interpersonal stress on binge eating and dietary restriction via social anhedonia. The expectation that eating is rewarding was entered as a moderator of the social anhedonia – binge eating pathway, whereas appearance reward was entered as a moderator of the social anhedonia – binge and T1 data were used for interpersonal stress and social anhedonia, and T2 data were used for eating pathology and moderator variables. In both models, T1 eating pathology and T1 BMI were entered as covariates. Unfortunately, we were unable to control for T1 moderators as approximately half of our sample was missing data on those variables.

Results

Descriptive statistics and zero-order correlations are presented in Table 2. Participants with and without T2 data did not differ significantly on demographic and baseline measures,

with the exception of baseline eating pathology. Specifically, participants who did not provide data at T2 had significantly higher T1 binge eating ($t_{186} = 2.17$, p = .031) and dietary restriction ($t_{185} = 2.53$, p = .012) scores, compared to participants who did respond to the T2 survey. Therefore, the upper end of disordered eating severity range is likely under-represented in our T2 data.

Baseline EPSI scores were similar to established norms for female university students for both binge eating ($t_{188} = 0.19$, p = .849) and dietary restriction ($t_{186} = -0.01$, p = .989; Forbush et al., 2013). Participant BMIs ranged from underweight to obese (range = 15.26 – 39.54), with 9.04% of participant BMIs in the underweight range (BMI < 18.5), 79.26% in the normal weight range (BMI = 18.5-24.99), 9.57% in the overweight range (BMI = 25.0-29.99), and 2.13% in the obese range (BMI ≥ 30.0).

Each form of interpersonal stress showed small-to-moderate positive correlations with one another, social anhedonia, and T2 eating pathology. There was a small negative correlation between social anhedonia and the expectancy that eating is rewarding, and a small positive correlation between social anhedonia and T2 dietary restriction. At T2, the expectancy that eating is rewarding showed small-to-moderate negative correlations with dietary restriction at and a small positive correlation with BMI. Appearance reward was not correlated with any other variables. Binge eating was strongly correlated across time points and showed small positive correlations with baseline BMI at each time point. Dietary restriction was moderately correlated across time points and showed small negative correlations with baseline BMI at each time point.

Results of the longitudinal path analysis are presented in Figure 1. There were significant direct associations between victimization, but not ostracism, and each form of eating pathology at T2, such that victimization predicted changes in eating pathology, controlling for social

anhedonia. There were also significant total effects (i.e., sum of direct and indirect effects) of victimization on T2 eating pathology. There was a direct association between ostracism and social anhedonia, but not victimization and social anhedonia. The indirect associations between interpersonal stress and eating pathology via social anhedonia were non-significant. Furthermore, there was no moderation of the pathway between social anhedonia and binge eating by the expectancy that eating is reward, or between social anhedonia and restrictive eating by positive responses to comments on appearance.

Discussion

The goal of this study was to examine whether lifetime peer stress, specifically ostracism and victimization, was indirectly associated with binge eating and dietary restriction via social anhedonia. In other words, we were interested in whether chronic experiences of peer stress result in decreases in the ability to experience pleasure from social interactions, leading individuals to engage in disordered eating. Furthermore, we sought to examine whether this indirect association, particularly the pathway from social anhedonia to eating pathology, was moderated by reward variables related to eating and appearance. We hypothesized that the association between social anhedonia and binge eating would be moderated by the expectancy that eating is rewarding, and that the association between social anhedonia and restrictive eating would be moderated by the tendency to have positive emotional responses to positive comments on one's shape and weight. Results did not support our hypotheses. There was no indirect association between peer stress and eating pathology via social anhedonia at any level of the moderators. The expectancy that eating is rewarding did not moderate the association between social anhedonia and binge eating and appearance reward did not moderate the association between social anhedonia and dietary restriction.

The finding that there was no indirect association between peer stress and eating pathology via social anhedonia is surprising given previously observed associations between interpersonal stress and social anhedonia (Cacioppo et al., 2009; Hawkley et al., 2003, 2007; Rappaport et al., 2019) and between social anhedonia and eating pathology (Eiber et al., 2002; Harrison et al., 2014; Tchanturia et al., 2012; Watson et al., 2010). A possible explanation for this is that different types of peer stressors may impact social reward versus eating pathology. In our sample, when controlling for all other variables in the longitudinal path model, there was an association between ostracism, but not victimization, and social anhedonia. Conversely, we found an association between victimization, but not ostracism, and later eating pathology. It appears that, in our undergraduate sample, the types of peer stressors that are associated with social anhedonia may not be the same ones that contribute to eating pathology. For example, the experience of ostracism may relate to lower reward from social experiences, which may then be associated with difficulties other than eating pathology, such as depression (Barkus & Badcock, 2019; Gandhi et al., 2022). Similarly, victimization may predict changes in eating pathology via mechanisms other than social anhedonia, such as negative affect, emotion dysregulation, or identity disturbance (Cook-Cottone et al., 2016; Lee & Vaillancourt, 2018; Markou et al., 2022; Mukherjee & Hussain, 2022). Of note, the majority of previous research demonstrating an association between social anhedonia and eating pathology has been conducted in clinical samples, and the one study that used an undergraduate sample found mixed results across assessment method and disordered eating symptom (Fussner et al., 2018). Taken together, these findings highlight the need for continued research in this area using specific indices of peer stress in varied populations.

We did not find that the expectancy that eating is rewarding moderated the indirect

association between interpersonal stress and binge eating via social anhedonia, nor did we find that appearance reward moderated the indirect association between interpersonal stress and dietary restriction via social anhedonia. While we theorized that binge eating and dietary restriction may serve as compensatory rewards when social interactions fail to be rewarding, it is possible that the response to interpersonal stress and subsequent social anhedonia is not to find alternative sources of reward, but rather to try to improve the quality of one's social interactions (Williams, 2009). For example, participants who are excluded in the laboratory are more motivated to re-establish social connection, to make new friends, and to work with others, compared to non-excluded participants (Maner et al., 2007; Molden et al., 2009). If this is indeed the case, we would not expect social anhedonia to lead to binge eating, regardless of how rewarding someone expects eating to be. While controlling one's appearance may be viewed as a means of improving social standing (Hohlstein et al., 1998), the motivation to do so may not depend on how much reward individuals derive from their appearance, but rather the extent to which they believe others value and judge them based on their appearance.

Contrary to previous research demonstrating an association between ostracism and eating pathology (Baumeister et al., 2005; Hayman et al., 2015; Oaten et al., 2008), we did not observe such an association in either of our models. While ostracism was correlated with dietary restriction at both time points, this association was no longer significant when victimization was included in the path models, suggesting that peer victimization might be a more important predictor of disordered eating than ostracism. Notably, the majority of previous studies have examined the effect of experimental manipulations of ostracism on motivation to eat and/or consumption of palatable food. Not only did these studies examine relatively normative eating behaviours (vs. disordered eating), they did so in controlled laboratory settings. Specifically, these studies used the Cyberball paradigm (Williams et al., 2000), in which participants are excluded in a computerized ball-tossing game. It may be that this paradigm does not accurately reflect the type of social exclusion that individuals encounter in their daily lives or their typical responses to social exclusion (e.g., Williams et al., 2002). One recent study that examined the association between ostracism and disordered eating using the same self-report dataset used in the current study did not find a direct association between ostracism and eating pathology, but rather indirect associations through negative affect and feeling fat (Trolio et al., 2021). This suggests that the association between ostracism and eating pathology might be nuanced and studies examining only a direct association using laboratory paradigms may fail to accurately assess the association.

In line with previous research showing that experiences of peer victimization are associated with future disordered eating (Copeland et al., 2015; Day et al., 2021), we did find a direct association between victimization and both forms of eating pathology at T2. Given that we controlled for T1 eating pathology in our longitudinal model, our results can be interpreted as suggesting that a history of victimization predicted a change in eating pathology over the course of the first semester of university. It may be that the transition to university presents challenges, including those of a social nature, that more greatly impact individuals with a history of peer victimization. Indeed, previous research has shown that individuals with a history of peer victimization experience more psychological difficulties during important transitional periods, such as depression, anxiety, anger, and aggression (Leadbeater et al., 2014; Sheppard et al., 2019).

Despite largely non-significant results, we believe this study adds to the extant literature. First, to our knowledge, this is the first study to examine the impact of lifetime peer stress on eating pathology during the transition to university. Other studies have examined the impact of peer stress at a specific time-point (e.g., during childhood or adolescence) in relation to disordered eating Duarte et al., 2017; Gattario et al., 2020; Lee & Vaillancourt, 2019; Markou et al., 2021) or the effect of concurrent interpersonal stress on disordered eating during the transition to university (Barker & Galambos, 2007; Howard et al., 2020). However, to our knowledge, no studies have looked at *lifetime* peer stress specifically during the transition to university. Furthermore, we included two specific indices of peer stress, including ostracism, which has not been as extensively studied in the context of EDs as other forms of peer stress (e.g., teasing, bullying; for review see Day et al., 2021; Lee & Vaillancourt, 2018)). Our findings should also be considered in light of several limitations. First, we examined an undergraduate sample rather than a sample of people with EDs. In addition to having lower levels of disordered eating, the students in our sample reported lower levels of social anhedonia (M = 9.55) compared to samples of women with EDs from previous studies (M = 15.26 - 17.20; Harrison et al., 2014; Tchanturia et al., 2012). It may be that the hypothesized processes are not present in individuals with low(er) levels of eating pathology and/or social anhedonia. For example, the students in our relatively healthy sample may have more adaptive coping strategies to manage the effects of interpersonal stress. Future studies examining similar processes may wish to include participants with higher levels of eating pathology and social anhedonia. Second, due to low retention at T3, we were unable to test our full mediation model. It is possible that with more data, over a longer time period, we might have observed some of our hypothesized associations.

While our hypotheses were not supported, we believe that our literature review suggests that examining mechanisms other than negative affect that might mediate the association between interpersonal stress and eating pathology is worthy of future consideration. This is supported by the findings of Trolio and colleagues (2021) showing that certain associations may only be elucidated when additional intermediate variables are considered. Furthermore, our findings highlight the importance of examining specific, rather than general indices of peer stress, given that ostracism and victimization may differentially relate to eating pathology. Finally, our results revealed a history of peer victimization as a potential risk factor that may be helpful in identifying individuals at risk for increasing eating pathology during the transition to university.

Article 3 References

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Article 3 Tables and Figure

Table 1

Demographic Information

Demographic Variable	N (%)	
Race		
White	138 (73.01)	
Chinese	21 (11.11)	
Japanese	8 (4.23)	
South Asian	4 (2.12)	
Arab	4 (2.12)	
Southeast Asian	3 (1.58)	
Black	2 (1.06)	
Latin American	2 (1.06)	
West Asian	1 (0.53)	
Korean	1 (0.53)	
Filipino	1 (0.53)	
Other	0 (0.00)	
Multi-Racial	4 (2.12)	
Missing	0 (0.00)	
Sexual Orientation		
Heterosexual	153 (80.95)	
Bisexual	26 (13.76)	
Lesbian	5 (2.64)	

Asexual	2 (1.06)
Missing	3 (1.59)
Annual Income	
<\$50,000	23 (12.17)
\$51,000 - \$100,000	36 (19.05)
\$101,000 - \$200,000	50 (26.45)
>\$200,000	24 (12.70)
Missing	56 (29.63)

Note. Participants were asked to report parental annual income rather than personal annual

income.

Table 2

Zero-Order Correlations and Descriptive Statistics

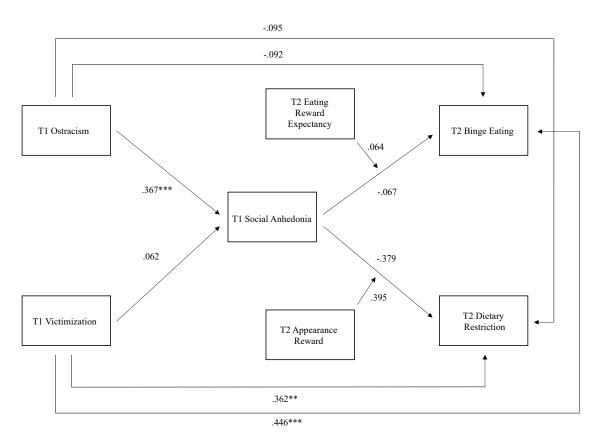
Variable	1	2	3	4	5	6	7	8
1. Ostracism	-							
2. Victimization	.376***	-						
3. Social Anhedonia	.455***	.254***	-					
4. Eating Reward Expectancy	045	090	197*	-				
5. Appearance Reward	.001	020	.053	106	-			
6. Binge Eating	.158	.190*	.007	.089	.183	-		
7. Dietary Restriction	.180*	.214*	.122	274**	.042	.009	-	
8. BMI	006	.041	037	.213	.185*	.243**	140	-
Mean	2.13	12.75	9.55	31.14	2.15	8.65	4.78	21.60
SD	0.70	7.40	6.08	6.81	1.03	6.22	4.94	3.20

 $\overline{Note. ***p < .001, **p < .01, *p < .05}$

Figure 1

Longitudinal Analysis of Indirect Effect of Peer Stress on Eating Pathology via Social

Anhedonia



Note. Parameters displayed are standardized estimates of the direct and indirect effects of each pathway. T1 = Time 1, T2 = Time 2. Body mass index and Time 1 eating pathology variables were included as covariates in all paths.

****p* < .001, ***p* < .01

General Discussion

Interpersonal stress is theorized to be an important risk and maintenance factor for disordered eating (e.g., Arcelus et al., 2013; Rieger et al., 2010). Disordered eating tends to develop during adolescence and early adulthood (Favaro et al., 2018; Hudson et al., 2007; Mohler-Kuo et al., 2016; Swanson et al., 2011), which are times that peer relationships can be particularly impactful. As such, previous research has aimed to determine the influence of peerrelated stressors on eating pathology, and in turn, how disordered eating and related cognitions impact peer relationships (e.g., Duarte et al., 2017; Gattario et al., 2020; Goldschen et al., 2019; Lee & Vaillancourt, 2019; Markou et al., 2021). However, several key gaps remain in the literature, and the nuances of the bidirectional association between peer stress and eating pathology has yet to be fully elucidated. The aim of this dissertation was to address these gaps by examining individual traits and behaviour that may help explain how peer stress and disordered eating relate to one another.

In Article 1, I examined whether core ED symptoms – binge eating and dietary restriction – were associated with maladaptive behavioural reactions to rejection by peers. Using an experimental paradigm in which participants played a "Survivor"-type game against computerized coplayers, I tested whether binge eating and dietary restriction were associated with participants' tendency to reject coplayers who had previously rejected them. Contrary to my hypotheses – that binge eating would be associated with increased retaliation and dietary restriction would be associated with lower retaliation – disordered eating was not associated with participants' tendency to retaliate following rejection. However, I found that dietary restriction was associated with an increased tendency to reject coplayers *overall*, regardless of how coplayers previously voted for them. This finding suggested that women with dietary restriction may behave in ways that push others away, although I did not collect data that allowed me to examine why this might be. One hypothesis I put forth was that individuals with dietary restriction may be hypersensitive to the possibility of rejection and misguidedly attempt to preempt rejection by rejecting others. Such behaviour could have the paradoxical effect of increasing peer stress, which may then perpetuate disordered eating.

In Article 2, I sought to expand on the aforementioned hypothesis by examining whether RS was associated with disordered eating via elevated peer stress. That is, whether being overly attuned and sensitive to potential or actual social threat was associated with more frequent experiences of ostracism and victimization, and whether these forms of peer stress were in turn associated with increased eating pathology. In both undergraduate women and women with binge eating, I did not find that RS was associated with eating pathology via increased peer stress, either concurrently or across time. I did, however, find that RS was *directly* associated with concurrent disordered eating behaviour and cognitions in both samples. My results suggested that the anticipation or perception of interpersonal rejection may be a more important predictor of concurrent eating pathology than actual experiences of peer stress.

In Articles 1 and 2, eating pathology was associated with maladaptive interpersonal traits and behaviour, regardless of actual experiences of peer stress. Surprisingly, these findings contradict both theory and empirical evidence by bringing into question the role of interpersonal stress in eating pathology. I considered whether it was possible, however, that the association between peer stress and eating pathology may be better elucidated by considering a novel indirect pathway via which peer stress may be associated with disordered eating. The goal of Article 3, therefore, was to determine whether peer stress was indirectly associated with binge eating and dietary restriction via decreased responsiveness to social reward, and whether this association was more likely to be found in individuals who had positive beliefs about the outcomes of eating or how others view their appearance. Using longitudinal data from a sample of undergraduate women, my hypothesis – that chronic experiences of ostracism and victimization would be associated with decreased reward from social experiences and subsequent attempts to increase reward via disordered eating behaviours – was not supported, regardless of participants' expectancies about the outcomes of these behaviours. I did, however, find that peer victimization (but not ostracism) was directly associated with increased eating pathology across the first semester of university. These findings suggested that a history of peer victimization may be a risk factor for worsening eating pathology during the transition to university.

Taken together, the findings from these three studies contribute to the topic of interpersonal stress and eating pathology in two ways. First, they bring into question the role of interpersonal stress in disordered eating and suggest that additional factors, such as RS, may be more important predictors than actual experiences of victimization and ostracism. Second, they suggest that the association between peer stress and eating pathology is nuanced and that not all forms of peer stress relate equally to ED symptoms, highlighting the importance of examining distinct forms of peer stress. The following is a discussion of these two points along with an examination of methodological issues related to this dissertation, suggestions for future research, and a discussion of the clinical implications of my findings.

Questioning the Role of Interpersonal Stress in Disordered Eating

In Articles 1 and 2, eating pathology was associated with maladaptive interpersonal traits and behaviour, independent of actual experiences of peer stress. Specifically, in Article 1, participants who self-reported more dietary restriction were more likely to reject coplayers in a computerized game, regardless of whether coplayers accepted or rejected them. I hypothesized that RS may explain why women higher in dietary restriction tended to reject others at a higher rate than other participants and regardless of whether they were accepted or rejected by others. In Article 2, when included in the same model, RS was associated with concurrent eating pathology, whereas ostracism and victimization were not, suggesting that RS may indeed be a more important correlate of disordered eating than actual peer stress. These findings indicate that subjective aspects of interpersonal functioning (i.e., an individual's beliefs, perceptions, predictions, and reactions to interpersonal situations) may be more strongly related to eating pathology than objective interpersonal stressors. Of note, these findings emerged in samples of women for whom RS and eating pathology were likely already established. It could be that interpersonal stress plays an important role earlier in development (e.g., during childhood and early adolescence) by contributing to risk factors that may influence the development or progression of eating pathology, such as RS (Rieger et al., 2010). Chronic experiences of peer stress and negative social evaluation have indeed been hypothesized as a risk factor for RS, and there is some evidence to support these associations (Downey & Feldman, 1996; Rosenbach & Renneberg, 2014). It may be that once RS has developed, actual peer stressors do not have as much of an influence on eating pathology as the individuals' beliefs about and perceptions of their social standing. Future research would benefit from employing longitudinal designs beginning earlier in development to investigate this possibility.

Nuances in the Association between Peer Stress and Eating Pathology

Unlike in Articles 1 and 2, I did find evidence for an association between peer stress and disordered eating in Article 3. However, this association appeared to be nuanced and differed across type of peer stress. Specifically, when both ostracism and victimization were included in the same model, only victimization was associated with worsening binge eating and dietary

restriction during the transition to university. Ostracism, conversely, was not associated with eating pathology, although it was related to social anhedonia, such that individuals with a history of ostracism were more likely to report being less responsive to social reward. These findings suggest that while different forms of peer stress are associated with negative outcomes, it may be that only more overt peer stressors, such as victimization, are related to increases in disordered eating during early adulthood. Alternatively, it is possible that victimization is more directly associated with the progression of eating pathology, whereas ostracism might exert an effect only through indirect pathways. Indeed, a recent study using the same dataset as Articles 3 found that, when both forms of peer stress were included in the same model, a lifetime history of victimization was directly associated with concurrent dietary restriction and worsening binge eating over time, whereas ostracism was not directly associated with eating pathology at any time point (Trolio et al., 2021). Conversely, ostracism demonstrated indirect associations, sequentially via negative affect and feeling fat, with both binge eating and dietary restriction concurrently, and with increases in binge eating during the first semester of university. This suggests not only a need to examine specific forms of peer stress, but to also consider how these forms of peer stress might relate to disordered eating. In the case of ostracism, future research may benefit from the inclusion of additional intermediate variables (e.g., negative affect, selfesteem) rather than simply examining direct associations between ostracism and eating pathology.

Methodological Considerations

Measures of Interpersonal Stress

While a strength of this dissertation was the use of both objective and subjective indices of interpersonal stress, it remains possible that the paradigm and measures used did not

adequately capture the nature of peer stressors that participants experienced in real life and/or that may have an influence on disordered eating. The Island Getaway Task used in Article 1 involves competing with, and receiving acceptance or rejection feedback from, computerized strangers. It is possible that the pattern of results obtained would have differed if coplayers were known to participants. Indeed, previous research has shown that reactions to rejection differ based on whether perpetrators of rejection are strangers versus known individuals (Leary et al., 1998; Snapp & Leary, 2001; Sommer et al., 2009). In Articles 2 and 3, ostracism and victimization were assessed via self-report measures. In addition to the inherent bias associated with self-report measures, a particular issue with the use of self-report measures of peer stress in ED research is the influence of RS on the recall of interpersonal stressors. Individuals with eating pathology, who demonstrate elevated levels of RS and are prone to making negative interpretations of ambiguous social scenarios (Cardi et al., 2013, 2017), may be likely to overreport instances of peer stress. Unfortunately, obtaining an objective measurement of peer stressors in adults is likely more difficult than doing so in children and adolescents, for whom corroborating reports can be obtained from parents, teachers, and classroom peers. As such, the combined use of both ecologically valid laboratory paradigms and self-report measures is likely the most useful approach for future research. In addition to issues of biased responding, the selfreport measures of peer stress used in this dissertation, the Ostracism Experiences Scale for Adolescents (Gilman et al., 2013) and the Multidimensional Peer Victimization Scale (Mynard & Joseph, 2000), were originally designed for use in children and adolescents. Although both scales have been used in young adult populations (Cosgrove et al., 2017; Lee et al., 2017; Niu et al., 2022; Ren & Evans, 2021), it is possible that they only capture the types of peer stressors

experienced earlier in life and fail to assess experiences of social exclusion and victimization that may be specific to adulthood (e.g., interpersonal workplace stressors; Cichy et al., 2007).

Measure of Social Anhedonia

The self-report measure of social anhedonia used in Article 3, the Revised Social Anhedonia Scale (RSAS; Eckblad et al., 1982), is a broad measure of responsiveness to social reward that was originally designed for use in individuals with schizophrenia-spectrum disorders. Although a small number of studies has used the RSAS to examine associations between social anhedonia and eating pathology (Harrison et al., 2014; Tchanturia et al., 2012), it is possible that it is not an ideal measure for research in the area of disordered eating. A recent review suggested that social anhedonia may be best viewed as a multifaceted construct that can be parsed into specific deficits related to anticipating enjoyment ("wanting") and deriving pleasure ("liking") from social interactions, and that the degree of impairment in these two facets may vary across psychiatric diagnoses (Barkus & Badcock, 2019). While previous research on reward responsiveness to food in individuals with eating pathology has differentiated between "wanting" and "liking" (e.g., Cowdrey et al., 2013; Dalton & Finlayson, 2014), the same has not been done for responsiveness to social reward. It is possible that only one of these aspects of reward responsiveness to social stimuli is impaired in individuals with disordered eating and that a broad measure such as the RSAS may fail to capture such nuances. For example, extant research suggests that loneliness is prevalent in individuals with eating pathology, suggesting that the "wanting" aspect of social reward responsiveness may be preserved (for review, see Levine, 2012). As such, future research should use more detailed measures of social anhedonia that differentiate between the "wanting" and "liking" aspects of reward responsiveness. To my knowledge, available options for assessing the "wanting" and "liking" aspects of social reward

that have been used in previous research include costly and time-consuming physiological measures, such as fMRI and facial electromyography, or single-item self-report ratings (e.g., Kohls et al., 2012; Korb et al., 2020). As such, future research may benefit from the development of a detailed self-report measure of anticipatory and consummatory pleasure from social rewards.

Timeframe of Assessment

In Article 1, I examined momentary reactions to acute peer stress using the Island Getaway task. While the use of this task allowed me to consider reactions to interpersonal stress in a relatively objective manner, it did not allow for an assessment of how *chronic* peer stress may influence individuals' behaviour towards others, nor did it allow for a more global assessment of interpersonal behaviour across unique situations. As such, the results of Article 1 did not provide a complete understanding of how behavioural responses to peer stress may contribute to the bidirectional association between peer stress and eating pathology. In Articles 2 and 3, the average timing between Time 1 and 2 assessment points in the undergraduate sample was 3.7 months, whereas the clinical participants in Article 2 completed assessments, on average, 18.9 months apart. It may be that, while these timeframes were long enough to detect the small-to-moderate changes in eating pathology observed, the processes hypothesized in this dissertation play out over longer intervals. Furthermore, the age of participants included in this series of studies was slightly older than the typical age of onset of disordered eating (Favaro et al., 2018; Hudson et al., 2007; Mohler-Kuo et al., 2016; Swanson et al., 2011). As such, I was unable to examine how peer stress, RS, and social anhedonia contribute to the *development* of eating pathology. Examining the influence of peer stress and eating pathology on one another over several years beginning in early adolescence may be more likely to capture the processes described in this dissertation and inform theory about the onset of eating pathology.

Diversity of Samples

Several characteristics of the samples included in this dissertation may limit the generalizability of my findings. First, participants across all three articles identified exclusively as female. While women are disproportionately affected by EDs (Galmiche et al., 2019), research on the association between peer stress and disordered eating in men is warranted, particularly given that responses to interpersonal stress may differ across genders. For example, women have been found to respond with more internalizing and physiological reactions (e.g., depression, anxiety, increased cortisol) and behave in ways that then further generate interpersonal stress, whereas men may be more prone to externalizing reactions (Rajchert et al., 2018; Rudolph, 2002; Stroud et al., 2002). Second, while my samples were relatively diverse with regards to ethnic background and sexual orientation (with 27-50% identifying as non-white and 19-23% identifying as non-heterosexual), I did not have adequate power to examine differences in results across specific ethnic and sexual minority groups. Further, while I specifically recruited participants identifying as women, I did not explicitly ask about biological sex and therefore could not draw any conclusions about the gender identity of participants. As such, it is difficult to determine whether the same pattern of results would have been obtained in ethnically diverse and LGTBQ+ populations. Previous research suggests that individuals belonging to minority groups may experience unique social and interpersonal stressors that are not adequately captured in research on majority groups, such as discrimination, prejudice, internalized negative stereotypes, and pressure to conform to appearance ideals of the majority group (Cheng, 2014; Convertino et al., 2021; Kwan et al., 2018; Parker & Harriger, 2020; Pullmer et al., 2021). As such, replicating the models tested in this dissertation using peer stressors that are specific to minority populations is warranted.

Future Directions

The preceding general discussion sections highlight several avenues for future research, related to both theory and methodology. With regards to theory, my results suggest that a focus on subjective aspects of peer stress (e.g., beliefs, expectations, perceptions, emotional reactions) may be warranted, both as predictors of eating pathology and as intermediate variables in the association between objective peer stress and disordered eating. Further, the preceding discussion suggests that measurement of interpersonal stress and related factors (e.g., social anhedonia) ought to be as specific as possible, as different facets of interpersonal constructs may differentially relate to eating pathology. From a methodological standpoint, future research would benefit from examining interpersonal models over longer time frames, beginning earlier in development (i.e., late childhood or early adolescence), using both ecologically valid laboratory paradigms and age-appropriate self-report measures, and including diverse samples and indices of minority-specific peer stressors.

Clinical Implications

The findings of this dissertation have implications for clinical practice in the field of EDs. First, the results of Article 3 suggest that when assessing psychosocial history, obtaining a detailed account of the types of peer stressors encountered by an individual may be useful for identifying risk for worsening eating pathology during major life transitions. Second, findings from Articles 1 and 2 highlight the importance of addressing subjective aspects of interpersonal functioning that may not be directly associated with actual experiences of interpersonal stress. In particular, interventions targeting RS may be especially beneficial, either within the framework of the most commonly-used treatment approaches (i.e., CBT-E and IPT) or as adjunct therapies used to compliment typical treatments. For example, within the framework of CBT-E (Fairburn, 2008; Fairburn et al., 2015), which primarily targets maladaptive eating behaviours, dietary restraint, and concerns about weight, shape, and eating, cognitive restructuring techniques could be employed to challenge patients' beliefs about the probability and outcome of interpersonal rejection and how they relate to body image and eating behaviour. My results also suggest that, when using an IPT approach (Agras et al., 2000; Rieger et al., 2010), a focus on RS or other subjective aspects of interpersonal functioning is important and may be more beneficial than focusing primarily on specific interpersonal stressors (e.g., role disputes or transitions). In addition to incorporating RS into existing treatment frameworks, the addition of RS-specific interventions may be beneficial. One promising intervention is positive interpretation bias training, which involves training individuals to make positive or realistic interpretations of ambiguous social situations. Positive interpretation bias training has been studied in individuals with AN and was shown to both reduce negative interpretations of ambiguous social scenarios and increase self-esteem (Cardi et al., 2015, 2019; Turton et al., 2017). Based on the findings from this dissertation, future research should assess whether positive interpretation bias training is also an appropriate intervention for individuals with EDs characterized by binge eating.

Concluding Statement

The present dissertation, using data collected via varied methodology and in different samples, expands upon previous research examining the role of peer-related stressors in disordered eating. Overall, results of my three studies suggest that interpersonal stress may not play as important or direct a role as previously theorized and that not all forms of peer stress are equally implicated in eating pathology. Specifically, my findings highlight RS and peer victimization as constructs warranting further investigation in the context of the self-perpetuating cycle of peer stress and disordered eating.

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