EXAMINING THE CORRELATES AND ANTECEDENTS OF SUBJECTIVE BINGE EATING EPISODES IN FEMALE UNDERGRADUATES

by

Molly E. Atwood

BA (Hons), Ryerson University, 2012

A thesis

presented to Ryerson University

in partial fulfillment of the

requirements for the degree of

Master of Arts

in the Program of

Psychology

Toronto, Ontario, Canada, 2015

AUTHOR'S DECLARATION FOR ELECTRONIC SUBMISSION OF A THESIS

I hereby declare that I am the sole author of this thesis. This is a true copy of the thesis, including any required final revisions, as accepted by my examiners.

I authorize Ryerson University to lend this thesis to other institutions or individuals for the purpose of scholarly research.

I further authorize Ryerson University to reproduce this thesis by photocopying or by other means, in total or in part, at the request of other institutions or individuals for the purpose of scholarly research.

I understand that my thesis may be made electronically available to the public.

Abstract

Examining the Correlates and Antecedents of Subjective Binge Eating Episodes in Female Undergraduates

Master of Arts, 2015

Molly E. Atwood

Psychology

Ryerson University

Binge eating is a core diagnostic feature of several eating disorders; however, controversy exists regarding the extent to which the size of an eating episode is important in the definition of a binge. The present study examined the relationship between subjective binge eating episodes (SBEs: experiencing loss of control while eating relatively small amounts of food) and eating disorder pathology, general pathology, and eating disorder-specific and general cognitive distortions in female undergraduate students (N=116) via self-report measures. In addition, negative affect and stress were examined as proximal antecedents of SBEs using naturalistic prospective monitoring. Findings indicated SBEs are associated with broad markers of eating disorder pathology and aspects of general pathology, and that eating disorder-specific cognitive distortions mediate the relationship between dietary restraint and SBE frequency. In addition, higher levels of negative affect were found to precede SBEs; however, stress was not identified as a statistically significant proximal antecedent. Findings are interpreted in light of methodological limitations, and clinical implications are discussed.

Acknowledgements

First and foremost, I would like to extend my thanks to my advisor, Dr. Stephanie Cassin. I am extremely grateful for the time, support, and encouragement she provided throughout the completion of this project. Her invaluable feedback and guidance over the past 2 years have strengthened the quality not only of this project, by of my research and clinical skills. I also wish to thank Dr. Michelle Dionne for her insightful feedback on this document, and Dr. Kristin Vickers for kindly agreeing to serve on my defense committee.

I would like to thank my fellow lab mates, Aliza and Lauren. It has been a pleasure collaborating with and getting to know these exceptional women over the past two years, and I greatly appreciate both their professional and interpersonal support. I also would like to thank Aiden Mehak who aided with data collection and entry. This project simply could not have been completed on time without her assistance.

Last, but certainly not least, I would like to express my most heartfelt thanks to my wonderful family for feigning interest in my scholarly pursuits, and for providing their unconditional and unwavering support in everything I do.

Table of Contents

List of Tables

List of Figures

List of Appendices

Examining the Correlates and Antecedents of Subjective Binge Eating Episodes in Female Undergraduates

Binge Eating as a Diagnostic Construct

Binge eating is a problematic eating pattern associated with significant clinical impairment and distress, as well as psychological and medical comorbidity (American Psychiatric Association [APA], 2013; Bulik, Sullivan & Kendler, 2002; Striegel-Moore, Wilfley, Pike, Dohm & Fairburn, 2000). The 5th edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-5; APA, 2013) defines a binge eating episode by two criteria: 1) eating an amount of food that is objectively larger than what most individuals would eat in a similar circumstance, within a discrete period of time, and 2) experiencing a sense of loss of control during the eating episode (e.g., feeling that one cannot stop eating or control what one is eating). Recurrent binge eating is a core diagnostic feature of both bulimia nervosa (BN) and binge eating disorder (BED), and also is common among individuals with anorexia nervosa binge-purge subtype (AN-B/P; APA, 2013). Thus, binge eating spans the eating disorder (ED) diagnostic spectrum.

Although the definition of a binge eating episode is consistent in BN and BED, the associated phenomena of binge eating in these disorders vary. For example, unlike BN the binge eating characteristic of BED must also be accompanied by at least three of the following five criteria: eating more quickly than normal, eating until uncomfortably full, eating a large amount of food despite not feeling hungry, eating alone due to embarrassment about how much one is eating, and feeling depressed, guilty or disgusted with oneself after eating (APA, 2013). Unlike BED, the binge eating characteristic of BN is followed by recurrent compensatory behaviours such as self-induced vomiting, laxative or diuretic misuse, and excessive exercise as a means to control shape or weight (APA, 2013). For AN-B/P subtype, no definition of binge eating is

provided in the DSM.

Binge eating was first introduced into the DSM nomenclature when BN was included as a diagnostic category in the third edition of the manual (DSM-III, 1980). Since the inclusion of BN in DSM-III, the criteria for this disorder have been the subject of much debate and empirical scrutiny (Palmer, 2004). As such, several diagnostic issues have been highlighted and subject to revision. For example, in the DSM-IV-TR (APA, 2000), individuals whose symptoms met all diagnostic criteria for BN except that they engaged in recurrent binge eating at a lower frequency (e.g., once per week) than required (i.e., twice per week), or engaged in recurrent binge eating without compensatory behaviours (i.e., BED), were deemed to be "subthreshold" presentations. As these individuals fell short of a full diagnosis, they were consequently relegated to the catchall diagnostic category of eating disorder not otherwise specified (EDNOS; Mond, 2013). This was highly problematic as EDNOS was an overinclusive diagnosis that made differentiating clinically severe, yet highly variable symptom profiles difficult (Fairburn et al., 2007; Rockert, Kaplan, & Olmsted, 2007).

BED has since been recognized as a separate diagnostic category in the DSM-5 (APA, 2013), following a large body of research demonstrating its merit as a distinct and clinically significant disorder (Devlin, Goldfein & Dobrow, 2003; Wilfley, Wilson & Agras, 2003**;** Wonderlich, Gordon, Mitchell, Crosby & Engel, 2009). Furthermore, the current nosology of both BN and BED requires that individuals engage in binge eating at a frequency of once per week for a period of 3 months. The decision to lower the frequency of binge eating was similarly supported by empirical data, which demonstrated that individuals who engage in binge eating at the once per week diagnostic threshold exhibit no meaningful differences on measures of core eating disorder pathology or associated pathology when compared to those who engage in binge eating at the twice per week threshold (Crow, Agras, Halmi, Mitchell & Kraemer, 2002;

MacDonald, McFarlane & Olmstead, 2012; Spoor, Stice, Burton & Bohon, 2007).

These changes were purported to improve the clinical utility of eating disorder diagnoses by reducing the prevalence of EDNOS (renamed "other specified feeding or eating disorder" in DSM-5; APA, 2013; Mochado, Goncalver & Hoek, 2013). Historically, EDNOS diagnoses have been perceived to be less severe or subthreshold ED cases, and this has hindered the acquisition of appropriate treatment for individuals whose symptoms are given this label. Thus, a "diagnostic shift" from EDNOS to BN and BED was thought to increase the clinical attention paid to clinically significant, yet previously overlooked, ED presentations (Mond, 2013; Walsh & Garner, 1997).

Validity of the Current Definition of Binge Eating

Despite efforts to amend the nosological classification of eating disorders as the state of the empirical ED literature continues to develop, diagnostic issues remain. Indeed, with each revision of the DSM (APA, 1987, 1994, 2000), what has remained consistent in the characterization of binge eating episodes is the consumption of an unusually large amount of food within a relatively brief period of time. However, whether the consumption of an *unusually* large amount of food accurately captures all patient reported binge eating patterns is questionable.

The characterization of binge eating as involving an unusually large amount of food can be traced back to early case study descriptions, wherein binges of 15,000 to 20,000 calories were reported (Russell, 1979). Subsequent laboratory research using experimental test meal paradigms confirmed that binge eating episodes, on average, do tend to involve the consumption of a large amount of calories; however, reported averages were far lower than what was originally proposed (e.g., Hadigan, Kissileff & Walsh, 1989; Kaye et al., 1992). For example, in a review of 6 early laboratory studies, Mitchell and colleagues (1998) noted that the mean caloric intake during binge episodes in individuals with BN ranged from 3,031 to 4,479 calories. Research examining

average caloric intake during binge episodes using prospective and retrospective self-report (e.g., food diaries and questionnaires) have found slightly lower estimates still, in the range of approximately 1,000 to 3,000 calories (Wolfe et al., 2009).

Additional research has demonstrated wide variability in the within-subject distribution of caloric intake during binge eating episodes. For example, Rossiter and Agras (1990) examined the caloric consumption of 343 self-reported binges obtained from 7-day food records completed by participants diagnosed with BN. No formal definition of binge eating (i.e., loss of control or excessive consumption parameters) was provided; thus, the participants' identification of binge episodes was a purely subjective appraisal. The authors found that approximately one third of self-reported binge episodes consisted of less than 500 calories, with an additional third consisting of between 501 and 1,000 calories. Furthermore, there was a wide range of caloric intake within subjects' binges (mean range of 1905 kcal), with only 3 of the 32 participants exhibiting a range of less than 850 calories. The authors concluded that the amount of food consumed during binge eating episodes varies substantially among individuals with BN and also within individuals, as most participants experienced both large (objective) and small (subjective) binges (Rossiter & Agras, 1990).

Rosen and colleagues (1986) examined the caloric content of 199 self-reported binge eating episodes and 440 nonbinge eating episodes obtained from 2-week food diaries completed by 20 women with BN. Again, participants were not provided with a formal definition of binge eating. The authors found that, although the caloric content of binge eating episodes was generally higher than that of nonbinge episodes, there was considerable overlap. That is, the majority of binge eating episodes (65%) could not be distinguished from nonbinge eating episodes on the basis of the amount of food consumed. Although approximately 27% of binge episodes involved the consumption of 2,000 calories or more, one-third of binge episodes contained 600 calories or

less. Furthermore, binge eating episodes ranged from 45 to 5,138 calories. The authors concluded that a significant proportion of binge eating episodes fall below the "large" cutoff (Rosen, Leitenber, Fisher & Khazam, 1986).

Similar results have been obtained in BED and AN-B/P participant samples. For example, Rossiter and colleagues (1992) examined the caloric content of 225 binge eating episodes reported on 1-week food diaries in a sample of 22 individuals with BED. On average, binge episodes consisted of 602 calories, with a range of 25 to 6,048 calories (Rossiter, Agras, Telch & Bruce, 1992). Although there is a paucity of research exploring binge eating in individuals with AN-B/P in general, one recent study by Burd and colleagues (2009) assessed the average caloric intake of eating episodes in women diagnosed with AN-B/P, AN restricting subtype (AN-R) or subsyndromal AN (i.e.. either DSM-IV amenorrhea, body image disturbance, fear of weight gain, or BMI criteria were not met). The caloric content of all eating episodes, including binge eating episodes, was recorded using ecological momentary assessment and 24-hour dietary recalls. The authors found that caloric intake on days when both binge eating and purging episodes occurred was significant higher than on days when binge episodes or purging episodes alone occurred, or days when no binge or purge episodes occurred. In addition, there was no difference in caloric intake on binge only and binge/purge free days. Furthermore, eating episodes ranged from 0 calories (i.e., diet soft drink) to approximately 15,000 calories, suggesting that some binge episodes do not resemble objectively large binges (Burd et al., 2009).

In sum, findings from both laboratory and naturalistic self-monitoring studies have demonstrated that some binge eating episodes are indeed quite large (e.g., Mitchell et al., 1998). However, several studies have demonstrated that binge size varies dramatically both between and within individuals, and that binge episodes that are quite *small* in size also commonly occur (e.g., Rosen, Leitenber, Fisher & Khazam, 1986; Rossiter & Agras, 1990). Furthermore, these findings

have been obtained in several ED diagnostic groups. This line of research highlights that an unusually large amount of food need not be consumed in order for a binge to be classified as such by individuals.

How, then, do we achieve specificity in defining and operationalizing binge eating? Several researchers have attempted to establish caloric parameters for objective binge eating episodes, for example, as the consumption of greater than 500 (Grilo, Schiffman & Carter-Campbell, 1994) or 1000 calories (Mitchell et al., 2012). Yet, a meaningful caloric cutoff continues to elude the field. Inherently problematic in the attempt to categorize binge eating episodes as those that include the consumption of 500 or 1000 calories or more, is that these caloric definitions are discrepant with the diagnostic definition of unusually large. As Rosen et al. (1986) noted, a binge of 600 calories is approximately equivalent to eating a double cheeseburger, which cannot reasonably be considered an unusually large amount.

Indeed, the above-reviewed studies have demonstrated that the amount of food consumed during a self-declared binge often is similar to that of nonbinge eating episodes. Thus, the size of an eating episode may not be the best indicator of whether that episode will be experienced as a binge. As a result, many investigators have argued that perhaps the more salient feature of binge eating is the experience of loss of control, regardless of the amount of food consumed. Among individuals with bulimic-type eating disorders the experience of loss of control over eating, more than binge size, has been found to be a robust predictor of impairment, psychiatric comorbidity and eating and general pathology (e.g., Vannucci et al., 2013). On the basis of this body of literature, one can conclude that binge eating as it is currently defined in the diagnostic nomenclature might not be an accurate or valid depiction of how this eating pathology manifests and presents in all patients. An important implication of focusing on loss of control over eating as the defining characteristic of binge eating episodes is that binge size might then be used for more

of a descriptive purpose, rather than a requisite feature, providing nuanced information on varying ED presentations.

Distinguishing Objective and Subjective Binge Eating

Over the past several decades a distinction has been drawn in the literature between two types of binge eating episodes with associated loss of control: those that fall in accordance with the DSM definition, termed objective binge episodes (OBEs), and those that are perceived by an individual as involving the consumption of more food than they should have eaten but the amount consumed is not objectively large, termed subjective binge episodes (SBEs). Fairburn and Cooper **(**1993**)** have long provided a conceptualization of, and assessment framework for, subjective binge episodes in the context of the Eating Disorder Examination (EDE), considered to be the gold standard in eating disorders assessment. Yet, historically, the assessment of SBEs has predominantly been for descriptive purposes, as the presence of SBEs does not inform diagnostic status. That is, although they are relevant to BN, BED and AN-B/P and can occur in combination with OBEs to meet DSM diagnostic requirements, the presence of SBEs alone is insufficient to warrant an ED diagnosis.

SBEs also appear to be relevant to a variant bulimic-type eating disorder currently captured within the DSM-5 "other specified feeding or eating disorder" sub-threshold diagnostic category. This variant, purging disorder (PD), is defined by the use of compensatory weight-control behaviours in the absence of binge eating (APA, 2013). This definition is problematic as individuals with PD do engage regularly in binge eating behaviour, albeit subjective as opposed to objective binging (Keel et al., 2005). Empirical evidence has begun to accumulate supporting the clinical significance of this eating disorder presentation (Keel, Haedt & Edler, 2005; Keel, Wolfe, Gravener & Jimerson, 2008); nevertheless, PD falls below the diagnostic threshold for an eating disorder diagnosis due to the fact that objective binge eating does not regularly occur.

Consequently, PD places a misguided emphasis on purging behaviour, as opposed to the combination of subjective binge eating and purging. In fact, it is this combination, as opposed to purging alone, that might be responsible for the impairment and distress observed in these individuals (Mond & Hay, 2010). Thus, whether the objective binge eating requirement for ED diagnostic status is clinically useful, or whether it hinders the identification of problematic and clinically meaningful ED presentations is a pressing research question.

A growing body of literature has begun to address this question by focusing on SBEs in several ways. For example, some research has explored group differences between individuals who engage regularly in only SBEs and individuals who engage regularly only in OBEs, on measures of eating disorder and general pathology. Keel, Mayer and Harnden-Fischer (2001) found that when comparing women from the community whose symptoms met diagnostic criteria for DSM-IV BN (OBN group) or whose symptoms met criteria for DSM-IV BN except that they engaged in subjective, as opposed to objective binge eating (SBN group), no significant differences were found on self-report measures of eating pathology (Revised Restraint Scale, Bulimia Test-Revised, and the Three-Factor Eating Questionnaire) or general pathology (Beck Depression Inventory-II [BDI-II], State-Trait Anxiety Inventory [STAI], Michigan Alcoholism Screening Test, and the Drug Abuse Screening Test). However, significant group differences were obtained for binge/purge frequency and levels of impulsivity (as measured by the Barratt Impulsiveness Scale-11 [BIS-11]). Specifically, the OBN group exhibited higher levels of impulsivity and higher frequency of both bingeing and purging compared to the SBN group. The authors concluded that while both OBN and SBN groups experience similar pathology, lower levels of impulse control seen in those with OBN might contribute to the consumption of larger amounts of food (Keel et al., 2001).

In another study, Mond and colleagues (2010) recruited a sample of two mutually exclusive groups of women drawn from a large-scale epidemiological study: those with 1) regular (i.e., at least once per week) SBEs but no regular (i.e., less than once per week) OBEs and 2) regular OBEs but no regular SBEs. The authors also did not find significant group differences on measures of eating disorder pathology (assessed via the EDE interview and Eating Disorder Examination-Questionnaire [EDE-Q]), general psychological distress (Kessler Psychological Distress Scale), or functional impairment (Medical Outcomes Study Short-Form; Mond, Latner, Hay, Owen & Rogers, 2010).

In a more recent study, Watson and colleagues (2013) examined OBEs and SBEs in a clinical sample including individuals whose symptoms met diagnostic criteria for AN-R, AN B/P, OBN, or SBN. No significant differences between the OBN and SBN groups were obtained on measures of perfectionism and impulse control (as measured by the Eating Disorder Inventory [EDI] subscales), negative affect (Depression Anxiety Stress Scale-42 [DASS-42]), interpersonal difficulties (Interpersonal Problems-32), quality of life (Quality of Life Enjoyment and Satisfaction Questionnaire-Short Form), self-esteem (Rosenberg Self-Esteem Scale), and most facets of eating disorder pathology (EDE). However, the authors did find that the SBN group exhibited higher levels of dietary restraint as measured by the EDE (Watson, Fursland, Bulik & Nathan, 2013).

A few studies have further explored the comparative relationship of objective and subjective binge eating frequency to a broad range of pathology using a continuous analytic approach. For example, Latner and colleagues (2007) found that in a community sample of women with a range of eating disorders, including BED and BN, subclinical eating disorders, and no eating disorders, the frequency of both OBEs and SBEs were significantly correlated with all facets of eating disorder pathology (assessed via the EDE interview and the EDI) and negative

affect (DASS-42). In addition, there were no differences between SBEs and OBEs with respect to the magnitude of the correlations. Furthermore, the frequency of both SBEs and OBEs accounted for significant unique variance in eating disorder pathology; however, only OBEs accounted for a significant amount of unique variance in negative affect.

Lastly, Brownstone and colleagues (2013) explored the relationship of OBEs and SBEs to eating disorder symptomatology, negative affect, and personality dimensions in a community sample of women whose symptoms met DSM-5 criteria for OBN or SBN. When comparing these groups, the authors found no significant differences on measures of eating disorder pathology (EDE) or negative affect (BDI-II and STAI). However, the SBN group was found to exhibit significantly greater levels of attentional impulsivity and cognitive distortions (measured via the Barrett Impulsiveness Scale-11 and the Dimensional Assessment of Personality Problems-Basic Questionnaire, respectively). Using a continuous data analytic approach, the authors further found that only the frequency of SBEs (and not OBEs) accounted for a significant amount of unique variance in weight and shape concern, anxious and depressive symptomatology, attentional impulsivity and cognitive distortions (Brownstone et al., 2013).

Given this evidence highlighting the clinical severity of SBEs in clinical and community samples, Luce, Crowther and Pole (2008) noted an important finding regarding the prevalence rates of these types of binge episodes in an undergraduate sample. Concernedly, these authors found that regular SBEs occur at a much higher rate in undergraduates than do OBEs (16.7% versus 6.4%, respectively). Jenkins and colleagues (2012) obtained slightly different rates in a separate sample of female college students (11.8% endorsed the occurrence of SBEs, 15.3% OBEs, and 13.6% the occurrence of both binge types); nevertheless, SBEs appear to be fairly common in this particular subset of the population (Jenkins, Conley, Hoste, Meyer & Blissett, 2012).

Only one study, to the best of the author's knowledge, has examined the clinical correlates of SBEs in an undergraduate sample. In this study, Jenkins and colleagues (2012) grouped 339 undergraduate women into the following five categories based on their responses on the EDE-Q: nonbinge eating (NBE), objective over eating (OOE; i.e., eating a "large amount" of food, without the experience of loss of control), OBE, SBE, and a mixed group of both OBE and SBE. The authors found that the OBE, SBE and mixed groups all reported higher scores on measures of eating disorder pathology (EDE-Q) and general psychopathology (Brief Symptom Inventory) than the NBE and OOE groups. Furthermore, NBE and OOE groups also exhibited better eating disorder related quality of life (based on a measure assessing the level of impairment experienced as a result of eating problems) than did the OBE, SBE and mixed OBE and SBE groups (Jenkins et al., 2012).

Taken together, this collection of findings suggests that SBEs are associated with a broad range of pathology and clinical markers of impairment and distress, and that individuals who engage only in SBEs exhibit comparable pathology to those who engage only in OBEs. Despite this fact, as Keel and colleagues (2001) note, individuals who engage only in SBEs are undertreated. That is, in their study, women with SBN were found to have received significantly less lifetime psychological treatment than those with OBN (39.1% versus 77.8% respectively) (Keel, et al, 2001). Several authors have suggested that this treatment discrepancy may reflect a tendency to study and treat what we define, rather than a true indication that greater pathology is associated with OBEs (Mond, 2013). It is imperative then, that researchers continue to appraise whether the DSM binge size distinction generates clinical utility. The continued exploration of meaningful pathology associated with variant (i.e. SBE) eating disorder pathology may improve our understanding and treatment of eating disorder presentations that are currently being overlooked.

Potential Pathology Associated with Subjective Binge Eating

Cognitive Distortions. Despite the fact that individuals with regular SBEs and those with regular OBEs exhibit comparable pathology on clinical correlates examined thus far, what remains somewhat unclear is why some individuals experience a sense of loss of control during eating episodes in which only a small amount of food is consumed, and thus, subjectively appraise such episodes as a binge. Several authors have speculated that cognitive distortions might be associated with SBEs. More specifically, these authors have posited that maladaptive cognitions about food and weight or shape (e.g., "If I have one chocolate bar it will make me obese") might contribute to the consumption of a small amount of food triggering feelings of loss of control (Kerzhnerman & Lowe, 2002; Niego, Pratt & Agras, 1997; Watson et al., 2013).

Support for the role of cognitive distortions in subjective binge eating might be inferred from the findings of several treatment studies in which SBEs have been included as an outcome measure. These studies have found that, in contrast to OBEs, SBEs are slower to respond to and tend to persist following cognitive behavioural therapy (CBT; Niego et al., 1997; Smith, Marcus & Kaye, 1992). Furthermore, higher frequency of SBEs at pretreatment has been found to predict lower rates of recovery in both BN and BED individuals at posttreatment (Castellini et al., 2011). Niego and colleagues (1997) posited that this differential treatment response might be related to the focus of CBT strategies aimed at reducing binge eating. Such strategies, including self-monitoring of binge eating episodes and the establishment of regular eating patterns, are largely behavioural (Apple & Agras, 2008). In addition, many CBT treatment protocols for binge eating-related disorders incorporate cognitive restructuring towards the end stages of treatment, and in a limited way (i.e., one or two sessions; Apple & Agras, 2008; Cooper & Fairburn, 2010; Murphy, Straebler, Cooper & Fairburn, 2010). Although behavioural strategies may be effective in diminishing binge size (i.e., OBEs), they might not adequately address the cognitive appraisal of

loss of control over eating. Thus, individuals might continue to experience a sense of loss of control when only small amounts of food are consumed (i.e., SBEs; Watson et al., 2013). Furthermore, Hildebrandt and Latner (2006) have demonstrated that the basic treatment strategy of self-monitoring binge eating episodes decreases the prevalence of OBEs; however, at the expense of increasing SBEs. This "binge drift" might infer the importance of cognitive mechanisms in maintaining SBEs (Hildebrandt & Latner, 2006).

Several researchers have further posited that maladaptive cognitions might play a role in the relationship between dietary restraint and SBEs. For example, Kerzhnerman and Lowe (2002) found that in a sample of treatment seeking women whose symptoms met criteria for either AN-B/P, BN or EDNOS, dietary restraint was significantly related to subjective, but not objective, binge episodes. As noted above, Watson and colleagues (2013) obtained a similar finding; whereas, Latner and colleagues (2007) found OBEs and SBEs to be equally associated with levels of dietary restraint. These authors independently concluded that the relationship between dietary restraint and SBEs indicates that cognitions related to restrained eating are associated with the feeling of being out of control while eating small amounts of food. That is, although the amount of food consumed during a SBE does not escalate to the level of an objective binge, individuals may compare what they have eaten with their rigid beliefs about food restriction and, if this amount is perceived to be in excess, might appraise this as a loss of control (Kerzhnerman & Lowe, 2002). This hypothesis might be best understood within the context of cognitive behavioural theories of eating disorders. For example, Fairburn, Cooper and Shafran's (2003) transtheoretical model proposes that the overevaluation of shape and weight is central to the maintenance of eating disorders, and that a consequence of this core pathology is the attempt to control shape and weight by adhering to strict and highly specific dietary rules and behaviours. For objective binge episodes, this appears to manifest as dietary or caloric *restriction* (e.g., eating

fewer meals per day and fasting; Elran-Barak et al., 2015). When even minor dietary slips inevitably occur, these are interpreted as evidence of a loss of control, which results in the temporary abandonment of dietary restriction and, thus, overeating ensues (Fairburn, Cooper & Shafran, 2003). Binge eating then serves to magnify preexisting concerns about shape, weight, and eating, leading to renewed efforts to restrict and thereby perpetuating the binge cycle: a phenomenon frequently termed the "abstinence violation effect" (Marlatt & Gordon, 1984). For subjective binge episodes, it is speculated that cognitions associated with dietary restraint (as opposed to actual restriction) are the more pertinent mechanism, whereby small violations of rigid dietary rules (e.g., the consumption of forbidden/fattening foods) leads to the appraisal of loss of control, but not necessarily a complete abandonment of control over food intake. This conceptualization might help to explain why some authors have observed the persistence of SBEs following CBT, despite the normalization of eating patterns (i.e., dieting).

Despite these assertions, none of the above-cited studies have specifically measured cognitions. Therefore, an exploration of the relationship between cognitive distortions and SBEs might prove useful in understanding why some individuals subjectively appraise as a binge the consumption of only a small amount of food. Although Brownstone et al. (2013) found a significant association between SBEs, as opposed to OBEs, and cognitive distortions, the self-report measure used to assess this construct in their study was a general measure of personality dimensions rather than cognitive distortions per se. Use of an ED specific measure of cognitive distortions, as well as a measure of general cognitive errors (e.g., all-or-nothing thinking or 'should' statements) may provide additional insight into the association of this pathology with SBEs.

Proximal Antecedents. An understanding of the function and maintenance of objective binge eating has been greatly advanced by empirical and theoretical work aimed at identifying

the immediate precipitants of this maladaptive eating pattern. It is now well established that although distal factors, such as dietary restraint, body dissatisfaction, and shape, weight and eating concerns contribute to the etiology and exacerbation of objective binge eating (Fairburn, Cooper & Shafran, 2003), binge episodes are influenced by acute emotional antecedents. Several theoretical models have been put forth to explain the maintenance of binge eating in relation to these antecedents, including the affect regulation model (Haedt-Matt & Keel, 2011), escape theory (Heatherton & Baumeister, 1991), and restraint theory (Polivy & Herman, 1985). These theories are unanimous in the postulate that acute changes in negative affect and distress contribute to binge eating, although they diverge somewhat in their conceptualization of the function of binge eating. For example, the affect regulation model suggests that binge eating serves to regulate or alleviate negative affect, whereas escape theory would suggest that binge eating serves to distract an individual from negative affect and distress stemming from aversive self-awareness.

Multiple studies have provided support for the role of negative affect in binge eating using several different methodologies. A large proportion of participants with BN and BED retrospectively report negative affect as a trigger of binge eating on self-report measures (e.g., Abraham, & Beumont, 1982; Arnow, Kenardy & Agras, 1992). In addition, laboratory studies have shown that experimentally inducing negative mood in participants with BED increases both the desire to binge and actual overeating (Chua, Touyz, & Hill, 2004; Cools, Schotte & McNally, 1992; Heatherton, Striepe & Wittenberg, 1998). Further, naturalistic studies employing daily food diaries and ecological momentary assessment have demonstrated that negative affect increases immediately prior to objective binge episodes (Haedt-Matt & Keel, 2011**).**

Stress has also been implicated as a precursor to binge eating. Studies examining binge eating episodes using food diaries have found that binge eating tends to occur more on days when

stressors occur, and that stressors are rated as more distressing in individuals who binge eat than in those who do not binge eat (Crowther, Snaftner, Bonifazi, & Shepherd, 2001). Furthermore, studies using ecological momentary assessment have found that greater levels of self-reported stress immediately precede binge episodes, as opposed to nonbinge episodes (Rydin-Gray, 2007**).** Lastly, experimentally induced stress in laboratory studies has been linked to increased urges to binge eat (Tuschen-Caffier & Vögele, 1999).

Thus, empirical data supports the notion that negative affect and stress are proximal triggers for objective binge episodes. As such, these findings have been incorporated into efficacious therapeutic approaches for binge eating, including CBT (Apple & Agras, 2008). Yet, despite the accumulation of evidence validating the role of negative affect and stress in objective binge eating, it is unclear as of yet whether these factors also contribute to subjective binge eating. It is possible that negative affect may contribute to individuals' perceptions of the amount of food eaten instead of, or in addition to, it affecting their actual eating behavior. Indeed, previous research has demonstrated that the perception of having overeaten is influenced, in part, by negative mood (Gleaves, Williamson & Barker, 1993). Furthermore, in a recent study, Rydin-Gray (2007) found that in a sample of undergraduate women with BED, negative affect and stress did not differ between binge episodes that were large in size, and those that were small in size. However, this study did not specifically recruit participants who engaged in only SBEs, nor was a nonbinge eating control group included as a comparison. There may be between group differences that this study failed to capture by recruiting a homogenous sample of individuals based on the presence of OBEs.

The Present Study

Aims

The present study seeks to address some of the gaps in the limited literature on subjective binge eating by exploring the clinical correlates and antecedents of this disordered eating pattern in an undergraduate sample. The primary aim of this study is twofold: 1) to examine the continuous relations between SBEs and OBEs and measures of eating disorder pathology including weight, shape, and eating concern; dietary restraint; emotional eating and uncontrolled eating; negative affect; and eating disorder specific and general cognitive distortions, and; 2) to examine group differences between those who engage regularly in SBEs (SBE group) and those who do not engage in eating binges (nonbinge eating controls) on the same clinical variables. Findings derived from this study will contribute to a growing debate regarding the validity and clinical utility of DSM defined binge eating, highlighting that disordered eating presentations that include only SBEs warrant increased clinical attention. As the majority of the above-reviewed studies, with the exception of Jenkins et al. (2012), have focused on SBE/OBE group comparisons, this study will also contribute to the negligible literature that has compared those who engage in SBEs with those who do not experience a sense of loss of control while eating. In addition, the inclusion of measures of eating disorder specific and general cognitive distortions is a novel contribution of this study to the literature, as this specific form of pathology has yet to be examined in relation to SBEs. If cognitive distortions are indeed highly related to SBEs, this finding might hold implications for modifying existing treatment strategies for this particular type of binge eating.

The secondary aim of this project is to explore the proximal antecedents of SBEs using naturalistic prospective monitoring in the form of daily food diaries. Specifically, this project aims to better understand whether acute changes in negative affect and stress contribute to SBEs. An understanding of the immediate triggers contributing to the misperception of having binged in the moment may highlight targets for intervention.

Hypotheses

In line with previous research findings, and literature speculating at additional pathology associated with SBEs, it is hypothesized that:

H1a: Both OBEs and SBEs will be significantly associated with eating disorder and general pathology.

H1b: SBEs will be more strongly associated with eating disorder-specific and general cognitive distortions than will OBEs.

H2: Individuals who engage in regular SBEs will exhibit greater levels of eating disorder and general pathology, and eating disorder specific and general cognitive distortions compared to nonbinge-eating controls.

Given the paucity of literature examining the relationship between cognitive distortions and SBEs, as well as the proximal antecedents of SBEs, the following will be examined as speculative hypotheses:

H3: The relationship between self-reported dietary restraint and SBE frequency will be mediated and moderated by cognitive distortions. With respect to mediation, it is expected that both eating disorder-specific and general cognitive distortions will help account for the relationship between dietary restraint and SBE frequency. With respect to moderation, it is predicted that individuals higher on either type of cognitive distortions will demonstrate a stronger relationship between dietary restraint and SBE frequency.

H4: It is hypothesized that greater levels of negative affect and stress will precede SBE episodes than nonbinge eating episodes in individuals who engage in regular SBEs. In addition, it is hypothesized that levels of negative affect and stress will be comparable preceding nonbinge episodes in the SBE group and nonbinge control group.

Method

Participants

Participants were undergraduate women ($N = 116$) enrolled in an Introductory Psychology course at Ryerson University. The age of participants ranged from 17 to 30 years ($M = 19.73$, $SD = 2.64$). Demographic information is presented in Table 1. Participants were recruited through the Ryerson University Psychology Department's online SONA system, a cloud-based subject pool management software. Brief, initial descriptions of all psychology studies being conducted at Ryerson are accessible to introductory students through this system, as a means of ensuring equal selection opportunity. For the purposes of this study, women were invited to participate in a two-part study examining the relationship between eating patterns and psychological wellbeing. As per the guidelines set by the Psychology Ethics Committee and the Department of Psychology at Ryerson University, participants in this study received 1% course credit towards their Introductory Psychology final grade in exchange for each hour of participation. As the present study required a total of 2 hours of participation (described below in Procedures section), participants were credited up to a maximum of 2% for full completion of the study. Participants were not compensated financially. The present study limited participants to females (self-declared), as gender differences in binge eating and associated correlates have been noted in previous studies (e.g., Kelly-Weeder, Jennings & Wolfe, 2012; Lewinsohn, Seeley, Moerk & Striegel-Moore, 2002). Ryerson University's Research Ethics Board approved this study for use in a human population.

Table 1.

Participant Demographics (N = 116)

	n or *M* (% or *SD*)
Ethnicity	
African Canadian	5 (4.3)
Caribbean Canadian	4 (3.4)
European Canadian	40 (34.5)
Latin/Central/South American Canadian	4 (3.4)
East/South East Asian Canadian	18 (15.5)
South Asian Canadian	14 (12.1)
West Asian/Arab/Maghrebi Canadian	8 (6.9)
Pacific Islander Canadian	1 (0.9)
Other	21 (18.1)
Relationship Status	
Single	78 (67.2)
Dating but neither married nor common-law	33 (28.7)
Married	3 (2.6)
Common-law	1 (0.9)
Education Level	
Graduate high school	71 (61.2)
Completed some college/university	39 (33.6)
Bachelor's degree	4 (3.4)
Professional degree	1 (0.9)
BMI	21.7 (3.11)

Measures

Eating Disorders Examination Questionnaire (EDE-Q; Fairburn & Beglin, 1994, 2008; Appendix A). The EDE-Q is a widely used self-report measure that corresponds to the Eating Disorders Examination interview (EDE; Fairburn & Cooper, 1993). The EDE is considered the gold standard in eating disorders assessment; however, the lengthy administration time (i.e., 30 – 60 minutes) and extensive training required for administration often preclude its use for research purposes. The EDE-Q is considered to be a viable alternative to the EDE, and has been found to have high internal consistency and test-retest reliability (Berg, Peterson, Frazier, & Crow, 2011). As individuals who binge and/or purge may be less inclined to disclose these behaviours via interview, the self-report nature of the EDE-Q is an additional noted strength (Mond, Latner, Hay, Owen & Rogers, 2010).

The EDE-Q contains a total of 36-items that assess for both the severity of eating disorder pathology as well as the frequency of eating disorder behaviours (e.g., subjective and objective binge eating, self-induced vomiting, laxative and diuretic misuse, excessive exercise) occurring over the course of the previous 28 days. The EDE-Q yields scores on four subscales (Restraint, Shape Concern, Weight Concern, and Eating Concern) that are derived from 22 items assessing the core attitudinal features of eating disorder pathology. Items are scored on a 7-point Likert scale, ranging from 0 to 6, with higher scores indicating higher eating disorder symptoms. Separate scores are obtained for each subscale, and a global EDE-Q score is obtained by averaging the four subscales. Cronbach's alpha in the present study was 0.94 for the global scale, 0.8 for the restraint subscale, 0.87 for the shape concern subscale, 0.8 for the weight concern subscale, and 0.72 for the eating concern subscale. The EDE-Q also includes items assessing height and weight, which are used to calculate body mass index (BMI; kg/m^2).

Several studies have examined the level of agreement between the EDE and EDE-Q. In general, agreement for subscales has been shown to be high, while agreement for binge eating behaviour has been shown to be low (Berg et al., 2011). This latter finding might reflect definition difficulties, as terms such as "unusually large amount of food" and "loss of control over eating" are ambiguous and may be difficult for participants to respond to accurately via self-report. However, providing participants with additional instructions when administering the EDE-Q has been shown to increase agreement between these measures (Goldfein, Devlin & Kamenetz, 2005). Thus, in the present study, the primary investigator or a trained research assistant provided participants with written definitions of loss of control and unusually large amount of food, as well as examples of OBEs and SBEs prior to completion of the EDE-Q in order to increase reliability. These definitions and examples were based on a combination of the probing questions delineated in the EDE interview, and an adaptation of the written instructions outlined by Goldfein et al. (2005; see Appendix B).

Lastly, as one of the primary aims of the study was to compare those with and without regular SBEs on clinical variables, participants were classified in this respect based on their responses on the EDE-Q. Specifically, those endorsing SBEs at last once per week, on average, over the past 28 days were grouped as subjective binge eaters. Due to the low prevalence of SBEs in the complete absence of OBEs, participants in the SBE group who reported OBEs were not excluded unless the frequency of OBEs met or exceeded the once per week threshold for regular occurrence. This grouping approach is equivalent to that used by Brownstone et al. (2013) and Mond et al. (2010). Those participants who reported an absence of binge eating episodes over the past 28 days were grouped as nonbinge eaters.

The Three Factor Eating Questionnaire-Revised 18 items (TFEQ-R18; Karlsson, Persson, Sjöström, & Sullivan, 2000; Appendix C). The TFEQ-R18 is a revised and shortened version of the original 51-item TFEQ (Stunkard & Messick, 1985). It measures three separate aspects of eating behaviour: cognitive dietary restraint, emotional eating, and uncontrolled eating. The uncontrolled eating subscale, comprised of 9 items, measures the tendency to eat more than is usual as a result loss of control over intake (e.g., "Sometimes when I start eating, I just can't seem to stop"); the emotional eating subscale, comprised of 3 items, measures the tendency to eat in response to negative emotions (e.g., "When I feel blue, I often overeat"); and the cognitive dietary restraint scale, comprised of 6 items, measures the conscious intention to restrict food intake in an attempt to control body weight (e.g., "I do not eat some foods because they make me fat"). TFEQ-R18 items are rated on a 4-point Likert scale, ranging from 1 (*definitely false, almost never, unlikely)* to 4 (*definitely true, almost always, very likely*). Scores on items corresponding to each subscale are summed to obtain total scale scores. Higher scale scores are indicative of greater cognitive dietary restraint, uncontrolled eating, or emotional eating. Although the TFEQ-R18 was first derived using a sample of obese individuals, the factor structure has since been validated in a sample representative of the general population (de Lauzon et al., 2004). Internal consistency estimates for the TFEQ-R18 are adequate (Karlsson et al., 2000). Internal consistency estimates in the current study were $\alpha = 0.82$, $\alpha = 0.88$, and $\alpha = 0.78$ for the cognitive dietary restraint, uncontrolled eating, and emotional eating subscales, respectively.

Depression Anxiety Stress Scales (DASS-21; Lovibond & Lovibond, 1995; Appendix D). The DASS-21 is a self-report measure designed to assess the unrelated aspects of the commonly co-occurring states of anxiety and depression, as well features common to both. The DASS-21 consists of a subset of items from the original 42-item version, and is comprised of three scales: depression, anxiety, and stress. Each subscale has seven items. Participants are asked

to indicate the extent to which each item applied to them during the previous week, on a scale ranging from 0 (*not at all*) to 3 (*very much or most of the time*). Factor analyses of the three-scale structure of the DASS have yielded strong support for the identified scales in clinical (Lovibond & Lovibond, 1995), non-clinical (Henry & Crawford, 2005) and combined clinical and non-clinical samples (Antony, Bieling, Cox, Enns & Swinson, 1998). The internal consistency estimates of the DASS-21 scales are excellent (e.g., Cronbach's α = .94, .87 and .91 for the Depression, Anxiety and Stress scales, respectively; Antony et al., 1998). Cronbach's alpha in the current study was 0.91 for the depression scale, 0.80 for the anxiety scale, and 0.85 for the stress scale.

Thought-Shape Fusion Questionnaire-Short Form (TSF; Coelho, Baeyens, Purdon, Shafran, Roulin & Bouvard, 2013; Appendix E). The TSF is an 18-item self-report measure used to assess cognitive distortions related to the overevaluation of the importance of thoughts about food. This measure assesses three aspects of TSF: 1) the belief that simply thinking about eating certain "forbidden" foods increases the likelihood of gaining weight or changing shape, 2) thoughts that eating a forbidden food is morally equal to actually eating that food, and 3) the experience of simply thinking about eating a forbidden food increasing feelings of fatness. Four items on this scale assess the frequency, impact, uncontrollability and importance of suppression of these cognitive distortions. Participants are asked to indicate the extent to which each statement is true of them in general, on a scale of 0 (*not at all*) to 4 (*totally/always*). The first 14 items can be summed to obtain a factor score for the concept-related domains (i.e., likelihood, moral and feeling domains). The last four items can be summed to obtain a factor score for the clinically relevant thoughts (i.e., frequency, impact etc.). Thus, the measure yields a total score, and two factor scores. The short form was derived through factor analysis of the original 34-item

measure (Shafran, Teachman, Kerry, & Rachman, 1999) in order to decrease the measure's length and, thus, facilitate its use in research and clinical settings. Preliminary support for the psychometric properties of the short form TSF has demonstrated high internal consistency (total Cronbach's $\alpha = .951$, concept-related domain Cronbach's $\alpha = .954$, and clinically relevant Cronbach's $\alpha = .927$; Coelho et al., 2013). Scores on the TSF have been shown to discriminate between a clinical eating disorder sample and healthy controls (Coelho et al., 2013). Furthermore, TSF scores also have been shown to correlate with eating disorder symptomatology in an undergraduate sample (Shafran et al., 1999). In the present study, Cronbach's $\alpha = 0.94$ for the total scale, 0.92 for the concept-related domain subscale, and 0.84 for the clinically relevant subscale. The total scale score was used in all analyses in the current study.

The Cognitive Distortions Scale (CDS; Covin, Dozois, Ogniewicz & Seeds, 2011; Appendix F). The CDS is a 20-item self-report measure designed to assess the frequency with which an individual engages in 10 common cognitive distortions, including mindreading, catastrophizing, all-or-nothing thinking, emotional reasoning, labeling, mental filtering, overgeneralization, personalization, should statements, and minimizing or disqualifying the positive. Participants are asked to indicate, on a scale ranging from 1 (*never*) to 7 (*all the time*), the frequency with which they tend to engage in each cognitive distortion in both social situations (e.g., with friends, partners or family) and achievement situations (e.g., at work or school). The CDS has demonstrated excellent internal consistency (Cronbach's $\alpha = .91$), as well as discriminant and convergent validity in a sample of undergraduate students (Covin et al., 2011). Cronbach's $\alpha = 0.93$ in this study sample.

6-Day Food Diary (Appendix G). Participants were provided with 6 days of paper food diary forms along with detailed instructions on how to monitor and record all foods and liquids

consumed throughout each day. Participants were instructed to specify as accurately as possible the quantity of the foods and liquids consumed during each eating episode, and a description of the food consumed. Participants also were asked to indicate whether the eating episode was a meal or a snack and the time of day the meal/snack was consumed. Participants were further instructed to indicate whether they considered each eating episode to be a binge or nonbinge and whether they perceived the amount eaten to be "unusually large." They also were asked to indicate on a 7-point Likert scale the degree of loss of control experienced during each eating episode. Participant determination of "unusually large" and ratings of loss of control were aided by provided definitions of each, consistent with those outlined in the EDE-Q instructions. Lastly, participants were asked to rate their levels of negative affect and stress immediately preceding each eating episode. Levels of negative affect and stress were indicated on a 7-point Likert scale, ranging from 1 (*low negative affect or stress*) to 7 (*high negative affect or stress*). A training session took place in the laboratory, wherein the primary investigator or trained research assistant reviewed these instructions with each participant. The importance of completing the food diaries directly before and after each eating episode was stressed during the training session in order to reduce retrospective recall bias. Participants were emailed once per day, at approximately 8 am, a reminder to complete the food diary.

Procedure

Upon arrival at the Healthy Eating and Lifestyle laboratory, all participants first completed an informed consent form**.** The primary investigator or research assistant then reviewed with participants the instructions for completion of the EDE-Q created for the purposes of this study (Appendix B), in order to ensure adequate understanding and accurate indication of objective and subjective binge eating episodes. Next, participants completed the self-report

measures battery, including a demographic questionnaire (see Appendix H), the EDE-Q, TFEQ-R18, DASS-21, TSF and CDS. All questionnaires were completed online using Qualtrics survey software. Qualtrics is hosted on secure servers by Qualtrics, Inc. in the United States.

Following the completion of questionnaires, all participants were provided with a detailed description of how to complete the 6-day food diaries. All participants returned to the laboratory approximately 7 days following the initial session to return their food diaries. Participants completed a brief, 4-item exit questionnaire that queried about adherence to instructions when completing the food diaries (see Appendix I). They were then verbally debriefed, as well as provided with a debriefing form outlining the purpose of the study and including a brief list of resources for managing eating disorder symptoms. The entire study, including informed consent, completion of questionnaires, training on food diaries, completion of food diaries, and debriefing, took approximately 2 hours. Data were collected from September through November and January through March, in accordance with the SONA system's recruitment period.

Data Analysis

All statistical analyses were run using SPSS for Macintosh version 20.0 (IBM Corp., 2011, Armonk, NY). Significance level (alpha) was set at $p = 0.05$ for all statistical tests. Hypotheses 1a and 1b were tested via bivariate correlations between SBE and OBE frequency and measures of eating disorder pathology, general pathology and cognitive distortions. Significant bivariate associations between the aforementioned clinical variables and either SBEs, OBEs, or both were followed up with multiple regression analyses**.** For each regression analysis, the continuous variables of OBE and SBE frequency were entered simultaneously to determine variance accounted for by each binge type above and beyond the other, and controlling for body mass index (BMI = kg/m^2). BMI was entered as a covariate because previous research indicates a

positive association between BMI and eating disorder pathology (e.g., Angle et al., 2009; van der Merwe, 2007).

Hypothesis 2 was tested using multivariate analyses of variance (MANOVA) for conceptual groupings of dependent variables. Specifically, three separate MANOVAs were conducted comparing the SBE group and nonbinge controls on measures of: eating disorder pathology, as measured by the EDE-Q and TFEQ subscales; negative affect, as measured by the DASS-21 Depression, Anxiety and Stress scales; and cognitive distortions, as measured by the TSF and CDS. Any significant multivariate findings were followed up with univariate analyses of variance (ANOVA)**.** Effect sizes were calculated using an online effect size calculator (DeFife, 2009, "Effect Size Calculator," accessed from http:// web.cs.dal.ca/~anwar/ds/Excel4.xlsx).

Bootstrapping mediation analyses (Hayes, 2009) and hierarchical multiple regression analyses were utilized to test hypothesis 3. The bootstrapping mediation method was selected as the statistical procedure of choice as it offers advantages over the traditional mediation method proposed by Baron and Kenny (1986). For example, bootstrapping has more statistical power associated with it, and is a nonparametric test; thus, it does not require the assumption of normality to be met and is ideally suited for small sample sizes. Bootstrapping analyses were conducted using Preacher and Hayes' (2004, 2008) INDIRECT macro for SPSS**.**

Hypothesis 4 was tested by aggregating ratings of negative affect and stress preceding both binge episodes and nonbinge episodes on the daily food diaries for each participant within the SBE group and preceding nonbinge episodes within the nonbinge control group. Paired-sample t-tests and independent t-tests were used to compare mean levels of negative affect and stress both within and between groups, respectively. Effect sizes were again calculated using an online effect size calculator.

Results

Preliminary Analyses

Missing data. Four (3.4%) participants did not report their age, 1 (0.8%) participant their ethnicity, 1 participant their education level, and 1 participant their relationship status. In addition, 1 participant did not report either the presence or absence of OBEs or SBEs on the EDE-Q. A total of 8 (6.8%) participants did not report their height and/or weight and, thus, were missing data for BMI. One participant's BMI score was removed due to the high likelihood of the participant incorrectly entering their height and weight, resulting in a value of 14.34 kg/m^2. With respect to self-report measures, 36.21% of participants had at least one missing data point. In cases with random missing data (i.e., < 10% missing on any of the questionnaires), imputation with the within-participant mean response for completed items was used to prorate the subscale or total scale score. However, participants who were missing substantial questionnaire data (i.e., > 10% missing on any of the questionnaires) were excluded from all analyses. Three participants (2.5% of sample) were excluded for substantial missing data on the EDE-Q, and two (1.7%) for missing data on the CDS, which resulted in a final sample of 111 participants.

Assumptions of normality. Prior to conducting the primary analyses, the data were screened for violations of normality. An inspection of the distribution of the mean scores on the majority of the key variables suggested that the distributions approximated normal, and that skewness and kurtosis values were within the acceptable ranges of |2| and |7|, respectively (West, Finch, & Curran, 1995). The distributions of OBE frequency scores did not fall within these parameters, and indicated positive skewness and platykurtosis. Scores were not transformed; nonparametric tests were used where appropriate when this variable was included in analyses.

Descriptive Characteristics of Study Sample

Overall, 74.7% of the sample endorsed some type of binge eating episode over the previous 28 days. The majority of the sample reported engaging in SBEs only (32.4%, n=36), or both SBEs and OBEs (33.3%; n=37). Approximately 24% (n=27) of the sample reported no binge eating episodes over the previous 28 days, and 9% (n=10) reported engaging in OBEs only. The frequency of SBEs over the previous 28 days ranged from 0 – 25 (*M* = 4.42, *SD* = 5.56). The frequency of OBEs ranged from 0 – 15 (*M* = 1.59, *SD* = 2.92). A small proportion of participants endorsed self-induced vomiting (6.3%; n=7; *M* = 0.24, *SD* = 1.27), laxative use (3.6%; n=4; *M* = 0.14, *SD* = 1), or diuretic use (1.8%; n=2; *M* = 0.1, *SD* = 0.95) within the previous 28 days as a means of controlling shape or weight. In addition, 45% (n=50) of the sample endorsed exercising hard (*M* = 3.9, *SD* = 6.4) as a means of controlling their shape or weight within the previous 28 days. The mean BMI in the sample was 21.7 kg/m^2 (*SD* = 3.11), which falls within the healthy range.

Descriptive statistics for each of the self-report measures are presented in Table 2. The means for the EDE-Q global score and restraint, eating concern, shape concern, and weight concern subscales were all higher than published norms for undergraduate women (Luce et al., 2008), indicating that the sample as a whole demonstrated elevated eating pathology; albeit the frequency of most compensatory behaviours was lower (see Table 3).

Table 2.

Means, Standard Deviations, and Cronbach's Alphas for Self-Report Measures

	M	*SD*	*Alpha*
EDE-Q Global	2.37	1.3	0.94
EDE-Q: Restraint	2.09	1.53	0.80
EDE-Q: Shape	3.27	1.45	0.87
EDE-Q: Weight	2.56	1.54	0.80
EDE-Q: Eating	1.55	1.3	0.72
TFEQ: Cognitive Dietary Restraint	16.43	4.8	0.82
TFEQ: Uncontrolled	22.31	5.61	0.88
TFEQ: Emotional	7.85	2.51	0.78
DASS-A	11.07	8.91	0.80
DASS-D	11.59	9.75	0.91
DASS-S	15.47	9.81	0.85
TSF	21.15	15.57	0.94
CDS	86.84	23.09	0.93

Note. EDE-Q Global = Total score on the Eating Disorder Examination Questionnaire; EDE-Q Restraint = Restraint subscale; EDE-Q Shape = Shape Concern subscale; EDE-Q Weight = Weight Concern subscale; EDE-Q Eating = Eating Concern subscale; TFEQ-Restraint = Cognitive Dietary Restraint Subscale of the Three Factor Eating Questionnaire; TFEQ-Uncontrolled = Uncontrolled Eating Subscale; TFEQ-Emotional = Emotional Eating Subscale; DASS-A = Anxiety subscale of the Depression Anxiety Stress Scales -21; DASS-D = Depression subscale; DASS-S = Stress subscale; TSF = Thought Shape Fusion Questionnaire; CDS = Cognitive Distortions Scale

Table 3.

Comparison Between Study Sample and Undergraduate Norms on EDE-Q Variables

	Present Sample		Undergraduate Norms[a]	
	M	*SD*	*M*	*SD*
EDE-Q Global	2.37	1.30	1.74	1.30
EDE-Q Restraint	2.09	1.53	1.62	1.54
EDE-Q Shape	3.27	1.45	2.27	1.54
EDE-Q Weight	2.56	1.54	1.97	1.56
EDE-Q Eating	1.55	1.30	1.11	1.11
	Any Occurrence (%)	Regular Occurrence (%)	Any Occurrence (%)	Regular Occurrence (%)
OBE	42.3	14.4	21.3	6.4
SBE	65.7	38.7	32.1	16.7
Vomiting	6.3	1.8	8.8	4
Laxative Use	3.6	0.9	8.3	3.1
Diuretic Use	1.8	0.9	6.6	3.6
Hard exercise	45	3.6[b]	30.8	5.9

Note: EDE-Q Global = Total score on the Eating Disorder Examination Questionnaire; EDE-Q Restraint = Restraint subscale; EDE-Q Shape = Shape Concern subscale; EDE-Q Weight =

Weight Concern subscale; EDE-Q Eating = Eating Concern subscale; OBE = Objective binge episode; SBE = Subjective binge episode. For the majority of behaviours, regular occurrence was defined as once per week, on average, over the previous 28 days. Frequencies for OBEs and SBEs exceed a total of 100%, as some participants endorsed both.

[a] Luce, K. H., Crowther, J. H., & Pole, M. (2008). Eating disorder examination questionnaire (EDE-Q): Norms for undergraduate women. *International Journal of Eating Disorder, 41,* 273-276.

[b] For hard exercise, regular occurrence was defined as an average of at least five times per week over the past 28 days, as per Luce et al. (2008).

Primary Analyses

Correlations Between SBEs, OBEs and Self-Report Measures

Table 4 presents Spearman's correlations between SBE and OBE frequency, eating pathology, negative affect, and cognitive distortions. The correlation between frequency of SBEs and OBEs was 0.29 ($p = 0.002$) in this sample, indicating that they are related but distinct constructs**.** Both types of binge eating episodes were significantly associated, in the small to moderate range, with facets of eating disorder pathology as measured by the EDE-Q global score and subscales (ps < 0.001), and the TFEQ subscales (ps < 0.05). Regarding compensatory behaviours, neither SBEs nor OBEs were significantly associated with frequency of laxative or diuretic use (ps > 0.05). Only frequency of OBEs was significantly associated with frequency of self-induced vomiting ($r = 0.2$, $p = 0.033$). In contrast, only SBEs were significantly associated with hard exercise ($r = 0.21$, $p = 0.032$). Interestingly, neither binge type was significantly correlated with depressive symptoms (ps > 0.05), and only frequency of SBEs was significantly associated with anxiety ($r = 0.24$. $p = 0.012$), and stress ($r = 0.33$. $p = 0.001$). Furthermore, only frequency of SBEs was significantly correlated with general cognitive distortions ($r = 0.2$, $p = 0.033$), whereas frequency of both OBEs ($r = 0.37$, $p = 0.000$) and SBEs ($r = 0.37$, $p = 0.000$) demonstrated a moderate positive association with eating disorder-specific cognitive distortions. Lastly, OBEs ($r = 0.31$, $p = 0.002$), but not SBEs, were significantly correlated with BMI.

Table 4.

Correlations Among SBEs, OBEs, and Self-Report Measures

Measure	*1*	*2*	*3*	*4*	*5*	*6*	*7*	*8*	*9*	*10*	*11*	*12*	*13*	*14*	*15*	*16*	*17*	*18*	*19*	*20*
1. SBE	--	**.29****	**.44****	**.38****	**.43****	**.35****	**.41****	.13	-.11	.01	**.21***	**.31****	**.36****	**.23***	**.24***	.17	**.33****	**.37****	**.20***	.08
2. OBE		--	**.45****	**.38****	**.41****	**.39****	**.48****	**.20***	-.02	-.03	.08	**.23***	**.52****	**.39****	.12	.18	.15	**.37****	.02	**.31****
3. EDE-Q Global			--	**.85****	**.9****	**.92****	**.87****	**.34***	-.01	.03	**.32****	**.67****	**.38****	**.20***	**.40****	**.38****	**.36****	**.76****	**.27****	**.27****
4. EDE-Q Restraint				--	**.63****	**.68****	**.70****	**.31****	-.01	.08	**.39****	**.74****	**.21***	.07	**.27****	**.23***	**.22***	**.68****	.12	**.24***
5. EDE-Q Shape					--	**.86****	**.71****	**.28****	.03	.03	**.26****	**.55****	**.39****	.19	**.38****	**.38****	**.37****	**.67****	**.34****	**.31****
6. EDE-Q Weight						--	**.75****	**.32****	-.04	.02	**.29****	**.52****	**.36****	**.24***	**.37****	**.38****	**.33****	**.66****	**.29****	**.39****
7. EDE-Q Eating							--	**.29****	.05	.06	**.24***	**.57****	**.43****	**.28****	**.42****	**.38****	**.38****	**.74****	**.26****	**.27****
8. Vomiting								**--**	-.05	-.04	.11	**.23***	**.2***	.10	.11	.15	.07	**.20***	.06	.17
9. Laxatives									**--**	**.7****	.18	.00	-.08	.00	.15	.11	.02	.04	.09	-.13
10. Diuretics										**--**	.19	.08	-.16	-.00	.13	.09	-.00	.04	.07	-.17
11. Exercise											**--**	**.39****	-.03	-.06	.08	-.00	-.06	**.34****	.01	.19
12. TFEQ Restraint												--	.07	-.01	**.28****	.15	**.19***	**.65****	.07	**.24***
13. TFEQ Uncontrolled													--	**.55****	**.29****	**.30****	**.27****	**.31****	**.25****	.17
14. TFEQ Emotional														--	**.22***	**.24***	**.27****	.06	**.25****	.13
15. DASS-A															--	**.64****	**.75****	**.46****	**.51****	-.12

16. DASS-D	--	**.74****	**.33****	**.54****	-.01
17. DASS-S		--	**.34****	**.46****	-.11
18. TSF			--	**.32****	**.21***
19. CDS				--	-.11
20. BMI					--

Note. SBE = Subjective binge episode frequency; OBE = Objective binge episode frequency; EDE-Q Global = Total score on the Eating Disorder Examination Questionnaire; EDE-Q Restraint = Restraint subscale; EDE-Q Shape = Shape Concern subscale; EDE-Q Weight = Weight Concern subscale; EDE-Q Eating = Eating Concern subscale; Vomiting = Frequency of self-induced vomiting; Laxatives = Frequency of laxative use; Diuretics = Frequency of diuretic use; Exercise = Frequency of hard exercise; TFEQ-Restraint = Cognitive Dietary Restraint Subscale of the Three Factor Eating Questionnaire; TFEQ-Uncontrolled = Uncontrolled Eating Subscale; TFEQ-Emotional = Emotional Eating Subscale; DASS-A = Anxiety subscale of the Depression Anxiety Stress Scales -21; DASS-D = Depression subscale; DASS-S = Stress subscale; TSF = Thought Shape Fusion Questionnaire; CDS = Cognitive Distortions Scale; BMI = Body Mass Index. * $p < .05$, ** $p < .01$

Regression Analyses

Hierarchical multiple regression analyses were performed to investigate the ability of OBE and SBE frequencies to predict levels of each criterion variable, after controlling for BMI. For each analysis, BMI was entered as a predictor into the first step of the model. Both OBE and SBE frequencies were entered into the second step of the model. For all variables in each model, the Tolerance values were > 0.10 and the Variance Inflation Factor (VIF) values were < 10.0, which excluded the presence of multicollinearity (Myers, 1990).

Eating Disorder Pathology. A regression analysis examined the simultaneous relative contributions of OBEs and SBEs to global eating disorder pathology (EDE-Q Global), controlling for BMI. The first step of the model was significant (F[1, 99] = 12.418, p = 0.001), with BMI accounting for 11.1% of the variance in global eating pathology. In step 2, the addition of OBEs and SBEs significantly added to the prediction of global eating pathology (ΔR^2 = 0.254, p = 0.000; F[3, 97] = 18.613; p = 0.000); the model accounted for 36.5% of the variance. SBEs (β = 0.322, p = 0.000), OBEs (β = 0.298, p = 0.001), and BMI (β = 2.58, p = 0.002) each accounted for a significant amount of unique variance in this variable (see Table 5)**.** Comparable results were obtained for regression analyses examining the ability of OBE and SBE frequency to predict EDE-Q Shape Concern, Weight Concern, and Eating Concern subscales (see Tables 6 through 9). For EDE-Q Restraint, BMI was again entered into Block 1, and the model was significant, R^2 = 0.042, F(1, 99) = 4.33, p = 0.04. In Block 2, frequency of SBEs and OBEs significantly added to the prediction of dietary restraint (ΔR^2 = 0.185, p = 0.000; F[3, 97] = 9.467, p = 0.000), which accounted for 22.6% of the variance. In this case, both SBEs (β = 0.270, p = 0.006) and OBEs (β = 0.259, p = .009), but not BMI accounted for a significant amount of unique variance in restraint.

Table 5.

Hierarchical Multiple Regression Analysis Predicting Global EDE-Q From SBE and OBE Frequency, Controlling for BMI

Predictor	Model Outcome						
	ß	*p*	R^2	ΔR^2	F	ΔF	*p* of model
Step 1			0.111		12.418		0.001**
BMI	0.334	0.001**					
Step 2			0.365	0.254	18.613	19.403	0.000***
BMI	0.258	0.002**					
OBE	0.298	0.001**					
SBE	0.322	0.000***					

Note. BMI = Body Mass Index; OBE = Objective binge episodes; SBE = Subjective binge episodes. *$p < 0.05$, **$p < 0.01$, *** $p < 0.001$

Table 6.

Hierarchical Multiple Regression Analysis Predicting EDE-Q Dietary Restraint From SBE and OBE Frequency, Controlling for BMI

Predictor	ß	*p*	Model Outcome R^2	ΔR^2	F	ΔF	*p* of model
Step 1			0.042		4.333		0.04*
BMI	0.205	0.04*					
Step 2			0.226	0.188	9.467	11.572	0.000***
BMI	0.139	0.131					
OBE	0.259	0.009**					
SBE	0.27	0.006**					

Note. BMI = Body Mass Index; OBE = Objective binge episodes; SBE = Subjective binge episodes.

*$p < 0.05$, **$p < 0.01$, ***$p < 0.001$

Table 7.

Hierarchical Multiple Regression Analysis Predicting EDE-Q Shape Concern From SBE and OBE Frequency, Controlling for BMI

			Model Outcome				
Predictor	ß	*p*	R^2	ΔR^2	F	ΔF	*p* of model
Step 1			0.092		10.07		0.002**
BMI	0.304	0.002**					
Step 2			0.304	0.211	14.107	14.729	0.000***
BMI	0.238	0.007**					
OBE	0.254	0.007**					
SBE	0.310	0.001**					

Note. BMI = Body Mass Index; OBE = Objective binge episodes; SBE = Subjective binge episodes.

*$p < 0.05$, **$p < 0.01$, *** $p < 0.001$

Table 8.

Hierarchical Multiple Regression Analysis Predicting EDE-Q Weight Concern From SBE and OBE Frequency, Controlling for BMI

			Model Outcome				
Predictor	ß	*p*	R^2	ΔR^2	F	ΔF	*p* of model
Step 1			0.173		20.732		0.000***
BMI	0.416	0.000***					
Step 2			0.345	0.172	17	12.698	0.000***
BMI	0.354	0.000***					
OBE	0.244	0.008**					
SBE	0.265	0.003**					

Note. BMI = Body Mass Index; OBE = Objective binge episodes; SBE = Subjective binge episodes.

*$p < 0.05$, **$p < 0.01$, *** $p < 0.001$

Table 9.

Hierarchical Multiple Regression Analysis Predicting EDE-Q Eating Concern From SBE and OBE Frequency, Controlling for BMI

			Model Outcome				
Predictor	ß	*p*	R^2	ΔR^2	F	ΔF	*p* of model
Step 1			0.06		7.356		0.008**
BMI	0.263	0.008**					
Step 2			0.329	0.26	15.847	18.773	0.000***
BMI	0.184	0.034*					
OBE	0.316	0.001**					
SBE	0.312	0.001**					

Note. BMI = Body Mass Index; OBE = Objective binge episodes; SBE = Subjective binge episodes

*$p < 0.05$, **$p < 0.01$, *** $p < 0.001$

Regarding compensatory behaviours, when BMI was entered as a predictor in the first step of the model, there was a significant effect on self-induced vomiting, F (1,99) = 14.431, p = 0.000; 12.7% of the variance was accounted for. The addition of OBE and SBE frequency in the second step of the model did not account for a significant amount of additional variance (R^2 = 0.153, ΔR^2 = 0.025, p = 0.238), although the model remained significant, F[3, 97] = 5.826, p = 0.001 (see Table 10). In this analysis, BMI emerged as the only significant predictor of unique variance in self-induced vomiting (β = 0.323, p = 0.001). For hard exercise, neither Block 1 (R^2 = 0.029, F[1, 98] = 2.918, p = 0.091), nor Block 2 (R^2 = 0.044, ΔR^2 = 0.016, p = 0.46, F[3, 96] = 1.49, p = 0.222) were significant (see Table 11).

Table 10.

Hierarchical Multiple Regression Analysis Predicting Frequency of Self-Induced Vomiting From SBE and OBE Frequency, Controlling for BMI

			Model Outcome				
Predictor	ß	*p*	R^2	ΔR^2	F	ΔF	*p* of model
Step 1			0.127		14.431		0.000***
BMI	0.357	0.000***					
Step 2			0.153	0.025	5.826	1.457	0.001**
BMI	0.323	0.001**					
OBE	0.173	0.092					
SBE	-0.074	0.459					

Note. BMI = Body Mass Index; OBE = Objective binge episodes; SBE = Subjective binge episodes.

*$p < 0.05$, **$p < 0.01$, *** $p < 0.001$

Table 11.

Hierarchical Multiple Regression Analysis Predicting Frequency of Hard Exercise From SBE and OBE Frequency, Controlling for BMI

			Model Outcome				
Predictor	ß	*p*	R^2	ΔR^2	F	ΔF	*p* of model
Step 1			0.029		2.918		0.091
BMI	0.17	0.091					
Step 2			0.044	0.016	1.490	0.782	0.222
BMI	0.162	0.115					
OBE	0.015	0.893					
SBE	0.119	0.266					

Note. BMI = Body Mass Index; OBE = Objective binge episodes; SBE = Subjective binge episodes.

A series of regression analyses also tested the extent to which frequency of OBEs and SBEs predicted cognitive dietary restraint, uncontrolled eating, and emotional eating, as measured by the TFEQ (see Tables 12 through 14). For the dependent variable of cognitive dietary restraint, BMI was again entered as a covariate into the model in Block 1, and there was a significant effect on the model, $F(1, 99) = 4.119$, $p = 0.030$; 4.6% of the variance was accounted for. In Block 2, frequency of OBEs and SBEs were entered into the model, which accounted for significant additional variance, $R^2 = .111$, $\Delta R^2 = 0.073$, $p = 0.021$; $F(3, 97) = 4.4$, $p = 0.006$. In this analysis, only frequency of SBEs ($\beta = 0.236$, $p = 0.023$) accounted for a significant amount of unique variance in dietary restraint. In contrast, while both OBEs and SBEs accounted for a significant amount of variance in emotional eating scores after controlling for BMI ($R^2 = .116$; $\Delta R^2 = 0.085$, $p = 0.012$***;*** $F[3, 97] = 4.229$, $p = 0.007$), OBEs ($\beta = 0.210$, $p = 0.046$), but not SBEs, accounted for significant unique variance in this construct. With respect to the criterion variable of uncontrolled eating, when BMI was entered into the first step of the model, there was again a significant effect, $R^2 = 0.061$, $F(1, 99) = 6.475$, $p = 0.012$). Frequencies of OBEs and SBEs accounted for a significant amount of additional variance in this variable ($R^2 = .304$, $\Delta R^2 = 0.243$, $p = 0.000$; $F[3, 97] = 14.118$, $p = 0.000$); in this case, both SBEs ($\beta = 0.242$, $p = 0.009$) and OBEs ($\beta = 0.361$, $p = 0.000$) accounted for significant unique variance.

Table 12.

Hierarchical Multiple Regression Analysis Predicting TFEQ Dietary Restraint From SBE and OBE Frequency, Controlling for BMI

			Model Outcome				
Predictor	ß	*p*	R^2	ΔR^2	F	ΔF	*p* of model
Step 1			0.046		4.819		0.030*
BMI	0.215	0.030*					
Step 2			0.12	0.073	4.4	4.042	0.006**
BMI	0.19	0.055					
OBE	0.077	0.462					
SBE	0.236	0.023*					

Note. BMI = Body Mass Index; OBE = Objective binge episodes; SBE = Subjective binge episodes.

*$p < 0.05$, **$p < 0.01$

Table 13.

Hierarchical Multiple Regression Analysis Predicting TFEQ Uncontrolled Eating From SBE and OBE Frequency, Controlling for BMI

			Model Outcome				
Predictor	ß	*p*	R^2	ΔR^2	F	ΔF	*p* of model
Step 1			0.061		6.475		0.012*
BMI	0.248	0.012*					
Step 2			0.304	0.243	14.118	16.9	0.000***
BMI	0.162	0.065					
OBE	0.361	0.000***					
SBE	0.242	0.009**					

Note. BMI = Body Mass Index; OBE = Objective binge episodes; SBE = Subjective binge episodes; *$p < 0.05$, **$p < 0.01$, *** $p < 0.001$

Table 14.

Hierarchical Multiple Regression Analysis Predicting TFEQ Emotional Eating From SBE and OBE Frequency, Controlling for BMI

			Model Outcome				
Predictor	ß	*p*	R^2	ΔR^2	F	ΔF	*p* of model
Step 1			0.03		3.114		0.081
BMI	0.175	0.081					
Step 2			0.088	0.085	4.229	4.671	0.007**
BMI	0.124	0.207					
OBE	0.21	0.046*					
SBE	0.147	0.152					

Note. BMI = Body Mass Index; OBE = Objective binge episodes; SBE = Subjective binge episodes. *$p < 0.05$, **$p < 0.01$

Negative Affect. Two regression analyses were conducted to test the effect of OBE and SBE frequency on measures of anxiety and stress. When entered as a predictor in the first step of the models, BMI did not have a significant effect on either anxiety ($R^2 = 0.023$; $F[1, 99] = 2.328$, $p = 0.130$), or stress scores, ($R^2 = 0.017$; $F[1, 99] = 1.71$, $p = 0.194$). The frequencies of OBEs and SBEs accounted for significant variance in anxiety, $R^2 = .114$, $\Delta R^2 = 0.091$, $p = 0.009$; $F(3, 97) = 4.167$, $p = 0.008$; however, only frequency of SBEs ($\beta = .244$, $p = .019$) accounted for a significant amount of unique variance. Similarly, frequencies of OBEs and SBEs significantly accounted for variance in stress, $R^2 = .171$, $\Delta R^2 = 0.154$, $p = 0.000$; $F(3, 97) = 6.68$, $p = 0.000$. Again, only frequency of SBEs ($\beta = .314$, $p = .002$) emerged as a significant predictor of unique variance in stress. Results of these analyses are presented in Tables 15 and 16.

Cognitive Distortions. Regarding general cognitive distortions, when BMI was once again entered into Block 1 of the model, there was no significant effect on CDS scores, $R^2 = 0.013$, $F(1, 99) = 1.28$, $p = 0.26$. When OBE and SBE frequency were entered into Block 2, these variables accounted for a significant amount of variance, $R^2 = 0.087$, $\Delta R^2 = 0.074$, $p = 0.023$; $F(3, 97) = 3.063$, $p = 0.032$. An inspection of the standardized beta coefficients revealed that frequency of SBEs emerged as the only significant predictor of unique variance in general cognitive distortions ($\beta = 0.290$, $p = 0.006$). For eating disorder-specific cognitive distortions, Block 1 of the model also was nonsignificant, $R^2 = 0.016$, $F(1, 99) = 1.577$, $p = 0.212$. Together, frequencies of OBEs and SBEs accounted for a significant amount of variance in eating disorder-specific cognitive distortions, $R^2 = 0.198$, $\Delta R^2 = 0.182$, $p = 0.000$; $F(3, 99) = 7.988$, $p = 0.000$. In this case, both SBEs ($\beta = 0.293$, $p = 0.003$) and OBEs ($\beta = 0.231$, $p = 0.022$) predicted a significant amount of unique variance in this construct. These results are presented in Tables 17 and 18.

Table 15.

Hierarchical Multiple Regression Analysis Predicting DASS-Anxiety From SBE and OBE Frequency, Controlling for BMI

Predictor	ß	*p*	Model Outcome R^2	ΔR^2	F	ΔF	*p* of model
Step 1			0.023		2.328		0.13
BMI	-.0152	0.13					
Step 2			0.144	0.091	4.167	4.992	0.008**
BMI	-0.186	0.061					
OBE	0.115	0.275					
SBE	0.244	0.019*					

Note. BMI = Body Mass Index; OBE = Objective binge episodes; SBE = Subjective binge episodes. *$p < 0.05$, **$p < 0.01$

Table 16.

Hierarchical Multiple Regression Analysis Predicting DASS-Stress From SBE and OBE Frequency, Controlling for BMI

			Model Outcome				
Predictor	ß	*p*	R^2	ΔR^2	F	ΔF	*p* of model
Step 1			0.017		1.71		0.194
BMI	-0.13	0.194					
Step 2			0.171	0.154	6.68	9.027	0.000***
BMI	-0.175	0.067					
OBE	0.154	0.129					
SBE	0.314	0.002**					

Note. BMI = Body Mass Index; OBE = Objective binge episodes; SBE = Subjective binge episodes. *$p < 0.05$, **$p < 0.01$, *** $p < 0.001$

Table 17.

Hierarchical Multiple Regression Analysis Predicting General Cognitive Distortions From SBE and OBE Frequency, Controlling for BMI

			Model Outcome				
Predictor	ß	*p*	R^2	ΔR^2	F	ΔF	*p* of model
Step 1			0.013		1.283		0.26
BMI	-.113	0.260					
Step 2			0.087	0.074	3.063	3.194	0.032*
BMI	-0.103	0.303					
OBE	-0.103	0.334					
SBE	0.29	0.006**					

Note. BMI = Body Mass Index; OBE = Objective binge episodes; SBE = Subjective binge episodes.

*$p < 0.05$, **$p < 0.01$

Table 18.

Hierarchical Multiple Regression Analysis Predicting Eating Disorder-Specific Cognitive Distortions From SBE and OBE Frequency, Controlling for BMI

Predictor	ß	*p*	Model Outcome R^2	ΔR^2	F	ΔF	*p* of model
Step 1			0.016		1.577		0.212
BMI	0.125	0.212					
Step 2			0.198	0.182	7.988	11.033	0.000***
BMI	0.065	0.489					
OBE	0.231	0.022*					
SBE	0.293	0.003**					

Note. BMI = Body Mass Index; OBE = Objective binge episodes; SBE = Subjective binge episodes. *$p < 0.05$, **$p < 0.01$, *** $p < 0.001$

Comparison between Regular Subjective Binge Eaters and Nonbinge Controls on Eating and General Pathology, and Cognitive Distortions

In order to examine differences in eating and general pathology and cognitive distortions between individuals who reported engaging in regular subjective binge eating and those who did not report engaging in any binge eating episodes, participants were grouped according to their responses on the EDE-Q. A total of 31 participants were categorized as regular subjective binge eaters (i.e., SBE frequency of at least once per week, on average, over the previous 28 days; OBEs did not meet the once per week threshold). As previously stated, 27 participants reported no binge eating episodes over the previous 28 days and, thus, comprised the nonbinge control group. Independent sample t-tests for continuous variables or chi-square tests for categorical variables were conducted in order to examine group differences on demographic variables (Table 19). The SBE and nonbinge control groups did not differ with respect to age (t[37.7] = -1.56, p = 0.12), or BMI (t[52] = 1.66, p = 0.103). Using Fisher's exact test (FET), groups also did not differ on ethnicity (p = 0.18), or education (p = 0.15). However, there was a significant difference between groups with respect to relationship status (p = 0.027, FET). An examination of the contingency table revealed that a greater proportion of individuals in the SBE group were single (74.2%), compared to nonbinge controls (44.4%); 7.4% of nonbinge controls were either married or in common-law relationships, compared to 0% in the SBE group. As relationship status was not significantly associated with any of the outcome variables, this variable was not included as a covariate in subsequent MANOVAs.

Table 19.

Comparison of Regular Subjective Binge Eaters and Nonbinge Controls on Demographic Variables

Variable	Regular SBE (n = 31) *M* or n *(SD* or %*)*	Nonbinge Controls (n = 27) *M* or n *(SD* or %*)*	*t* or FET [a]	*p value*
Age	19.16 (1.7)	20.16 (2.79)	-1.569 [b]	0.125
BMI	22.7 (4.9)	20.83 (2.99)	1.66	0.103
White/European	15 (48.4)	7 (25.9)	3.597	0.18
Single	23 (74.2)	12 (44.4)	5.956	0.027*
Graduated High school	22 (71)	13 (48.1)	4.657	0.149

Note. BMI = Body Mass Index; FET = Fisher's Exact Test

[a] Expected values were below 5% in at least 20% of cells, necessitating the use of Fisher's Exact Test

[b] Levene's test for equality of variances was significant for this analysis, *df* = 37.704.

*$p < 0.05$

For each analysis, assumptions were checked. Box's test of equality of covariance matrices was nonsignificant for each MANOVA (*ps* > 0.05), indicating that the assumption of homogeneity of variance was met. Levene's test also was nonsignificant for each dependent variable in each analysis, indicating that the error variance of the dependent measures were equal across groups.

Eating Disorder Pathology. Using Pillai's Trace, the omnibus test revealed a significant effect of binge status on eating disorder pathology, $V = 0.312$, $F(7, 50) = 3.241$, $p = 0.007$. Follow-up univariate ANOVAs revealed a significant difference between groups on each of the dependent variables (see Table 20): the SBE group demonstrated significantly higher mean scores than nonbinge controls on EDE-Q restraint ($F[1,56] = 8.437$, $p = 0.005$; $d = 0.778$), shape concern ($F[1,56] = 16.17$, $p = 0.000$; $d = 1.077$), weight concern ($F[1,56] = 6.767$, $p = 0.012$; $d = 0.697$), and eating concern ($F[1,56] = 6.32$, $p = 0.015$; $d = 0.673$), TFEQ cognitive dietary restraint ($F[1,56] = 5.697$, $p = 0.02$; $d = 0.639$), uncontrolled eating ($F[1,56] = 10.865$, $p = 0.002$; $d = 0.883$), and emotional eating ($F[1,56] = 6.145$, $p = 0.016$; $d = 0.664$). According to Cohen's (1977) classification, the effect sizes were moderate to large.

Negative Affect. Using Pillai's Trace, the MANOVA testing for group differences on the three dependent measures of negative affect was not statistically significant ($V = 0.059$, $F[3, 54] = 1.131$, $p = 0.345$), indicating that the SBE group and nonbinge controls did not differ on measures of depression, anxiety or stress symptomatology (see Table 21). Because the omnibus test was not significant, no follow-up ANOVAs were conducted.

Cognitive Distortions. In the final MANOVA, there also was no significant effect of binge status on cognitive distortions, $V = 0.078$, $F(2, 55) = 2.328$, $p = 0.107$ (see Table 22). No follow-up ANOVAs were conducted.

Table 20.
Group Differences on Measures of Eating Disorder Pathology

	Regular Subjective Binge Eaters (n=31) *M* *(SD)*	Nonbinge Controls (n=27) *M* *(SD)*	*F value*	*(df)*	*p value*
Eating Pathology			3.241	7,50	0.007**
EDE-Q Restraint	2.41 (0.25)	1.35 (0.27)	8.47	1, 56	0.005**
EDE-Q Shape	3.57 (0.19)	2.43 (0.21)	16.17	1, 56	0.000***
EDE-Q Weight	2.63 (0.22)	1.77 (0.24)	6.77	1, 56	0.012*
EDE-Q Eating	1.57 (0.19)	0.84 (0.21)	6.32	1, 56	0.015*
TFEQ Restraint	17.45 (0.84))	14.52 (0.89)	5.7	1, 56	0.020*
TFEQ Uncontrollec	22.81 (0.91)	18.41 (0.98)	10.87	1, 56	0.002**
TFEQ Emotional	7.9 (0.42)	6.37 (0.45)	6.15	1, 56	0.016*

Note. EDE-Q Global = Total score on the Eating Disorder Examination Questionnaire; EDE-Q Restraint = Restraint subscale; EDE-Q Shape = Shape Concern subscale; EDE-Q Weight = Weight Concern subscale; EDE-Q Eating = Eating Concern subscale; TFEQ-Restraint = Cognitive Dietary Restraint Subscale of the Three Factor Eating Questionnaire; TFEQ-Uncontrolled = Uncontrolled Eating Subscale; TFEQ-Emotional = Emotional Eating Subscale. * $p < .05$, ** $p < .01$, ** $p < .001$

Table 21.

Group Difference on Measures of General Pathology

	Regular Subjective Binge Eaters (n=31) *M* *(SD)*	Nonbinge Controls (n=27) *M* *(SD)*	*F value*	*(df)*	*P value*
Negative Affect			1.131	3,54	0.345
DASS D	11.42 (1.51)	8.3 (1.62)			
DASS A	11.74 (1.48)	9.63 (1.59)			
DASS S	16.65 (1.53)	12.67 (1.64)			

Note. DASS = Depression Anxiety Stress Scales – 21 item version; DASS A = Anxiety subscale; DASS D = Depression subscale; DASS S = Stress subscale.

Table 22.

Group Differences on Measures of Eating Disorder-Specific and General Cognitive Distortions

	Regular Subjective Binge Eaters (n=31) *M* *(SD)*	Nonbinge Controls (n=27) *M* *(SD)*	*F value*	*(df)*	*P value*
Cognitive Distortions			2.328	2,55	0.107
CDS	92.48 (4.14)	84.33 (4.44)			
TSF	21.32 (2.48)	13.85 (2.66)			

Note. TSF = Thought Shape Fusion Questionnaire; CDS = Cognitive Distortions Scale. * $p < .05$

Mediation Analyses

Two separate bootstrapping mediation analyses were utilized in order to determine whether general and eating disorder-specific cognitive distortions indirectly account for the relationship between dietary restraint and SBE frequency. In the first analysis, dietary restraint (as measured by the TFEQ Cognitive Dietary Restraint scale) was entered as the predictor variable and SBE frequency (as measured by the EDE-Q) was entered as the dependent variable. The indirect effect of general cognitive distortions (as measured by the CDS) on the relation between dietary restraint and SBE frequency was tested using a confidence level of 95 resampled 10 000 times. The indirect effect was nonsignificant (Bias Corrected Confidence Intervals = -0.028 to 0.113, standard error = 0.033; see Figure 1). In the second analysis, the indirect effect of eating disorder-specific cognitive distortions, as measured by the TSF, on the relationship between dietary restraint and SBE frequency was tested. In this case, an indirect effect of TSF on the relation between dietary restraint and SBE frequency was observed (Bias Corrected Confidence Interval = 0.061 to 0.531; standard error = 0.119; $p = 0.0001$)**.** These results are displayed in Figure 2.

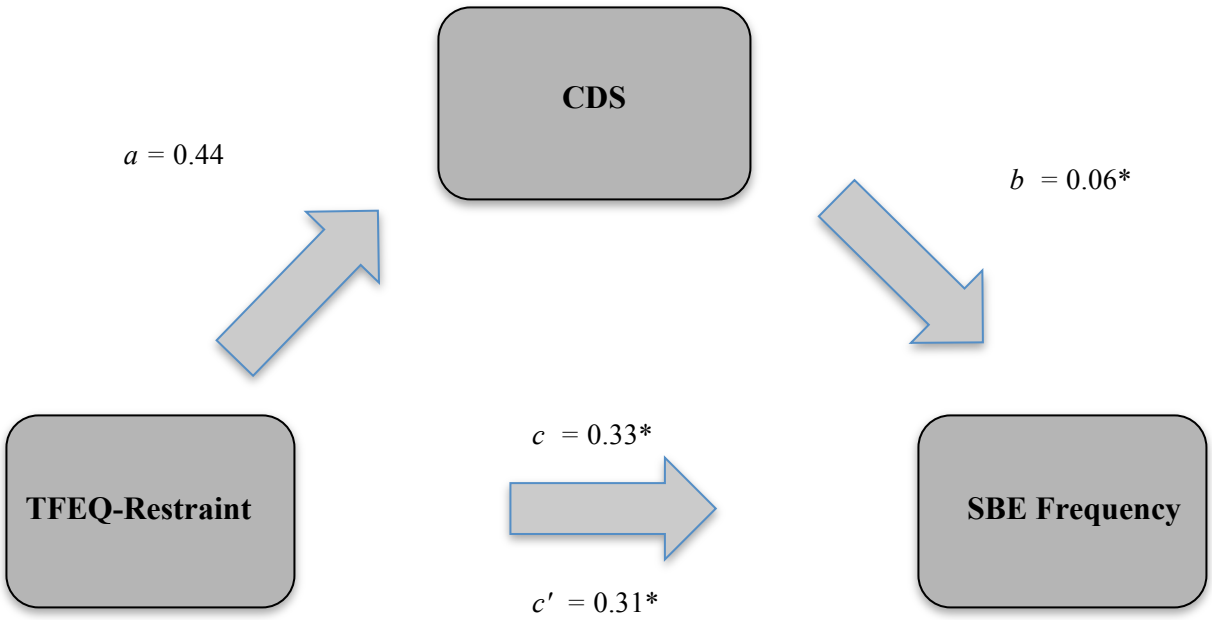

Figure 1. Indirect effect of CDS on the relation between TFEQ-Restraint Subscale and SBE Frequency. TFEQ-Restraint = Cognitive Dietary Restraint subscale of the Three Factor Eating Questionnaire; CDS = Cognitive Distortions Questionnaire; SBE = subjective binge eating. Coefficients for each path listed beside corresponding arrow; *$p < .05$.

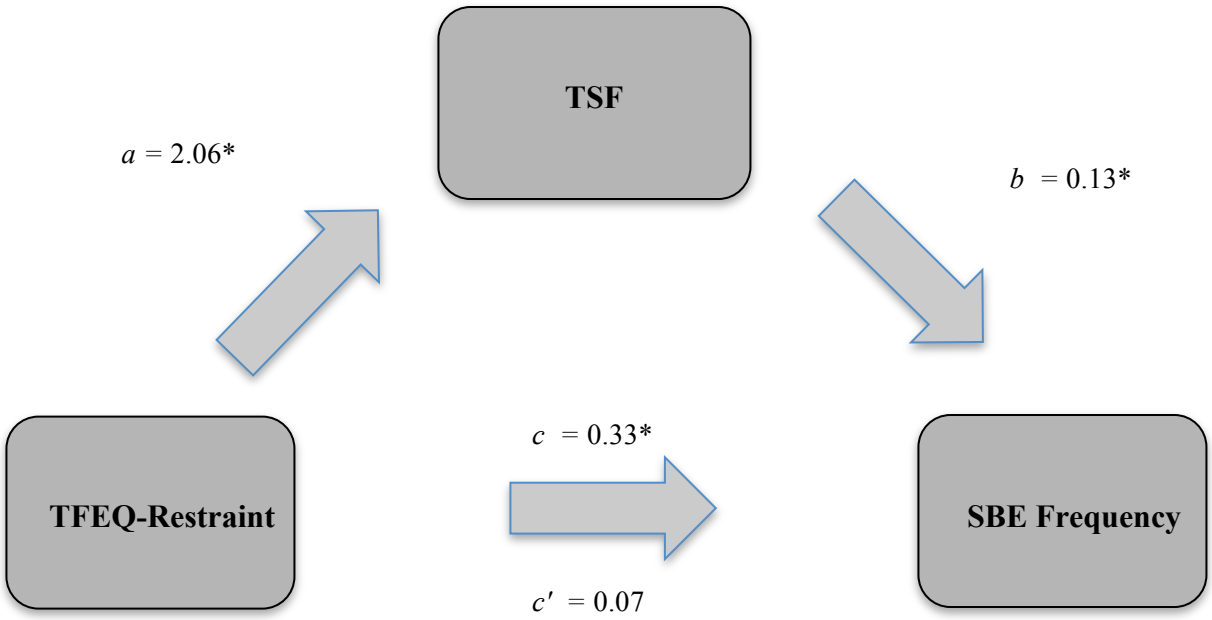

Figure 2. Indirect effect of TSF on the relation between TFEQ-Restraint Subscale and SBE Frequency; TFEQ-Restraint = Cognitive Dietary Restraint subscale of the Three Factor Eating Questionnaire; TSF = Thought Shape Fusion Questionnaire; SBE = subjective binge eating. Coefficients for each path listed beside corresponding arrow; *$p < .05$.

Moderation Analyses

To test the hypothesis that eating disorder-specific and general cognitive distortions moderate the relationship between dietary restraint and SBE frequency, two separate hierarchical multiple regression analyses were conducted. For each analysis, dietary restraint and the moderator variable were added into the first step of the model. The interaction term between dietary restraint and the moderator variable was entered into the second step. All predictor variables were centered before creating the interaction terms, to avoid potentially problematic multicolinearity (Aiken & West, 1991).

In the first analysis, general cognitive distortions (CDS) and dietary restraint (TFEQ-Cognitive Dietary Restraint scale) were entered into the first step of the model; together, these variables accounted for a significant amount of variance in the criterion variable of SBE frequency, $R^2 = .135$, $F(2, 107) = 8.328$, $p = 0.000$. Next, the interaction term between general cognitive distortions and dietary restraint was added to the regression model, which did not account for significantly more variance than cognitive distortions and dietary restraint alone, $R^2 = 0.144$, $\Delta R^2 = 0.006$, $p = 0.412$; $F(3, 106) = 5.762$, $p = 0.001$. Both CDS ($\beta = 0.243$, $p = 0.009$) and TFEQ ($\beta = 0.259$, $p = 0.005$) scores significantly predicted SBE frequency, whereas the interaction term did not, indicating that moderation had not occurred (see Table 23). Similar nonsignificant findings were obtained when eating disorder-specific cognitive distortions (TSF) were examined as a moderator of the relationship between dietary restraint and SBE frequency **(**see Table 24). When the predictor variables were entered into the first step of the model, they accounted for 15.8% of the variance in SBE frequency, $F(2, 107) = 9.957$, $p = 0.000$. When the interaction between eating disorder-specific cognitive distortions and dietary restraint was added to the regression model, no significant additional variance was accounted for, $R^2 = 0.158$, $\Delta R^2 =$

0.001, p = 0.176; $F(3, 106) = 6.614$, $p = 0.000$. In this analysis, only TSF scores ($\beta = 0.344$, p = 0.006) significantly predicted SBE frequency.

Table 23.

Moderation Analysis Predicting Subjective Binge Eating Frequency by Dietary Restraint and General Cognitive Distortions

			Model Outcome				
Predictor	ß	*p*	R^2	ΔR^2	F	ΔF	*p* of model
Step 1			0.135		8.328		0.000***
TFEQ R	0.264	0.004**					
CDS	0.233	0.011*					
Step 2			0.140	0.006	5.762	0.68	0.001**
TFEQ R	0.259	0.005**					
CDS	0.243	0.009**					
TFEQ R x CDS	0.75	0.412					

Note. TFEQ R = Cognitive Dietary Restraint scale of the Three Factor Eating Questionnaire;

CDS = Cognitive Distortions Scale; *$p < 0.05$, **$p < 0.01$, ***$p < 0.001$

Table 24.

Moderation Analysis Predicting Subjective Binge Eating Frequency by Dietary Restraint and Eating Disorder Specific Cognitive Distortions

			Model Outcome				
Predictor	ß	*p*	R^2	ΔR^2	F	ΔF	*p* of model
Step 1			0.157		9.957		0.000**
TFEQ R	0.058	0.614					
TSF	0.357	0.002*					
Step 2			0.158	0.001	6.614	0.097	0.000**
TFEQ R	0.066	0.579					
TSF	0.344	0.006*					
TFEQ R x TSF	0.029	0.756					

Note. TFEQ R = Cognitive Dietary Restraint scale of the Three Factor Eating Questionnaire; TSF = Thought Shape Fusion Questionnaire; *$p < 0.01$, **$p < 0.001$

Daily Food Diaries

Descriptive Characteristics of Eating Episodes. A total of 5 participants did not complete the diary portion of the study, resulting in an overall completion rate of 95.7%. Seventeen participants rated their negative affect and/or stress using an inappropriate scale and, thus, their diaries were excluded from the analyses. This resulted in four participants in the nonbinge control group being excluded from diary analyses (final n=23). All 31 participants in the SBE group submitted complete 6-day food diaries. The average number of eating episodes recorded on the diaries over the 6-day period in the SBE group was 23.33 (*SD* = 4.29; range = 15 - 32). Eighteen of these participants labeled a total of 64 recorded eating episodes as binges; of these, 2 binge eating episodes were excluded from analyses due to context (i.e., binges occurred at an all you can eat buffet, and thus were not considered to be an unusually large amount of food given the circumstance). The average number of eating episodes recorded during the 6-day monitoring period in the nonbinge control group was 24.05 (*SD* = 4.25; range = 15 – 32). Interestingly, five of these participants labeled a total of 14 recorded eating episodes as binges**.**

Binge eating episodes reported by both the SBE group and nonbinge controls were coded as either subjective or objective by two independent raters, including the first author and a trained research assistant. The interrater reliability for the raters was found to be Kappa = 0.701, p = 0.000, indicating substantial agreement (Landis & Koch, 1977). All discrepancies between raters were resolved via group discussion with both raters and the first author's supervisor, resulting in a total of 59 binges classified as SBEs in the SBE group and 13 SBEs in the nonbinge control group. As participants were asked to indicate whether they considered each binge episode to be "unusually large," level of agreement also was calculated between participants and each rater. Agreement between the first author and study participants was found to be Kappa = 0.153, p =

0.016, indicating slight agreement. There was fair agreement between the ratings of the trained research assistant and participants, Kappa = 0.266, $p = 0.001$.

Self-reported Adherence to Food Diary. Overall, the majority of the total sample reported completing ratings of negative affect and stress immediately before eating "every time" (24.3%; n=27) or "most of the time" (41%; n = 46); only 1.8% (n=2) of the sample reported never completing ratings immediately before eating. Similarly, most participants reported recording the amount and type of food they consumed immediately after eating "every time" (27%; n=30) or "most of the time" (25.2%; n=28); 3.6% (n=4) reported "none of the time." However, regarding how often participants completed the entire food record at the end of the day (as opposed to throughout the day), 12.6% (n=14) of the sample reported completing the entire diary at the end of the day on each of the 6 days. Only 15.3% (n=17) reported completing the diary throughout the day on each of the 6 days. Lastly, only 0.9% (n=1) and 1.8% (n=2) of participants reported recording a smaller amount of food than they actually ate, or omitting a food item "every time" or "most of the time" they completed the diary, respectively. Approximately 77% (n=85) of participants indicated that they never omitted a food item, or recorded a smaller quantity than was actually eaten. Together these results suggest that while retrospective recall bias and inaccurate information might be present, it is likely to be minimal.

Chi square analyses were conducted to evaluate differences between the SBE group and nonbinge control group for each item on the Exit Questionnaire. There was no significant difference between groups on any of the questions.

Comparison of Negative Affect and Stress Between Binge and Nonbinge Episodes Within Regular Subjective Binge Eater Group.

Two paired sample t-tests were conducted in order to determine whether greater mean ratings of negative affect and stress preceded SBEs compared to nonbinge eating episodes within

the SBE group. Due to unequal sample sizes (ratings of negative affect were available for only 18 participants who reported binge episodes, versus 31 who recorded negative affect prior to nonbinge episodes), paired sample t-tests were conducted using n =18. Prior to conducting the analyses, the distributions of the difference scores were inspected for violations of normality: none were found. The paired sample t-test revealed that the mean level of negative affect was significantly higher preceding SBEs (M = 3.43, SD = 1.36) than nonbinge eating episodes (M = 2.44, SD = 0.67), $t(17) = 2.805$, $p = 0.012$, $d = 1.36$. However, a second paired sample t-test revealed no significant difference between mean levels of stress preceding subjective binge episodes and nonbinge eating episodes (see Table 25). Effect sizes were large for both analyses.

Comparison of Negative Affect and Stress Between Nonbinge Episodes Across Regular Subjective Binge Eater and Nonbinge Control Groups.

Two independent samples t-tests were conducted to examine whether negative affect and stress prior to nonbinge episodes were comparable in the SBE and nonbinge control group (Table 26). Levene's test was significant for the analysis examining differences in negative affect (p = 0.018), indicating that the variances between groups was unequal. This was corrected for by reporting the t-statistic utilizing the unpooled estimate for the error term, and adjusting the degrees of freedom using the Welch-Satterthwaite method. Levene's test was nonsignificant for the analysis examining differences in stress; thus, equal variances could be assumed in this case. Results revealed that mean negative affect did not differ preceding nonbinge eating episodes between the nonbinge (M = 2.29, SD = 1.03) and SBE (M = 2.16, SD = 0.68) groups, $t(33.626)$ = 0.512, $p = 0.612$. Mean stress ratings also did not differ preceding nonbinge eating episodes between nonbinge controls (M = 2.67, SD = 0.99) and the SBE group (M = 2.75, SD = 0.86), $t(52) = 0.282$, $p = 0.779$. Effect sizes were small for these analyses.

Table 25.

Mean Negative Affect and Stress Coefficients for Binge and Nonbinge Episodes in Regular Subjective Binge Eater Group

	Eating Episode							
	Subjective Binge		Nonbinge					
	M	SD	M	SD	*t*	*df*	*p*	*d*
Negative Affect	3.43	1.36	2.44	0.67	2.805	17	0.012*	1.36
Stress	3.61	1.36	2.87	0.93	1.715	17	0.104	0.83

Note. d = Cohen's d, a measure of effect size. *$p < 0.05$

Table 26.

Mean Negative Affect and Stress Coefficients for Nonbinge Episodes in Regular Subjective Binge Eater and Nonbinge Control Groups

	Group							
	SBE Group		Nonbinge Group					
	M	SD	M	SD	*t*	*df*	*p*	*d*
Negative Affect	2.16	0.68	2.29	1.03	0.512[a]	33.626	0.612	0.14
Stress	2.75	0.86	2.67	0.99	0.282	52	0.779	0.079

Note. d = Cohen's *d*, a measure of effect size.

[a] Levene's Test was significant for this analysis

Discussion

Summary of Main Findings

The primary aim of the present study was to examine the clinical correlates of subjective binge eating episodes in a sample of female undergraduate students. This was accomplished using two approaches: 1) by examining the continuous relations between SBEs and OBEs and measures of eating disorder pathology, general pathology, and eating disorder-specific and general cognitive distortions, and; 2) by examining group differences between individuals who reported engaging in regular SBEs and those who did not report binge eating on the same clinical variables. The mediating and moderating influence of eating disorder-specific and general cognitive distortions on the relationship between dietary restraint and frequency of SBEs also was examined. The secondary aim of this project was to explore whether negative affect and stress are proximal antecedents of SBEs. Overall, findings suggest that a large proportion of female university students experience frequent episodes of subjective binge eating, and that, as expected, this disordered eating behaviour is associated with broad markers of eating disorder pathology and aspects of general pathology. Eating disorder-specific cognitive distortions were found to mediate the relationship between dietary restraint and SBE frequency, although general cognitive distortions did not. Neither was found to be a moderator of this relationship. In addition, for women reporting regular SBEs, higher levels of negative affect were found to precede SBEs using naturalistic self-monitoring data; however, stress was not identified as a statistically significant proximal antecedent. The specific results, limitations of the current study, and clinical implications will be discussed in detail.

Comparison of Objective and Subjective Binge Episodes on Clinical Variables

The present findings demonstrate that SBEs and OBEs appear to be similarly associated with multiple facets of eating disorder pathology, including shape, weight, and eating concerns,

as well as dietary restraint and uncontrolled and emotional eating. Further indication that SBEs and OBEs are each associated with core eating disorder pathology was supported in regression analyses, which demonstrated that both binge types significantly and independently predicted global and subscale scores on the EDE-Q, as well as uncontrolled eating on the TFEQ. These results suggest that SBEs are as strong a predictor of well-validated measures of eating disorder symptomatology as are OBEs, and that these associations are independent of the relationship between SBEs and OBEs. These findings corroborate those of a burgeoning literature, wherein the comparable association of these binge types with similar clinical variables has been demonstrated in clinical (Brownstone et al., 2013), community (Mond et. al., 2010; Latner et al., 2007), and undergraduate samples (Jenkins et al., 2012).

Also concordant with some prior research (Kerzhnerman & Lowe, 2001; Watson et al., 2013), SBEs emerged as a unique predictor of cognitive dietary restraint. However, interestingly, in the present study only OBEs uniquely predicted the emotional eating subscale of the TFEQ. This subscale is a brief (i.e., 3 item) measure of the tendency to eat in response to the negative emotions of anxiety, sadness, and loneliness. One potential explanation is that the differential ability of OBEs, as opposed to SBEs, to predict emotional eating is reflective of the unique function of OBEs as an emotion regulation strategy. That is, OBEs might be used to manage or escape from momentary distressing affect, whereas SBEs stem more from the cognitive aspects of dieting**.** Alternatively, it is possible that utilizing a more comprehensive measure might have elucidated nuanced differences in association between these binge types and aspects of emotional eating. For example, The Emotional Eating Scale (Arnow, Kenardy & Agras, 1995) is a well-validated measure, which assesses the urge to eat in response to a variety of emotions, grouped into three subscales: anger/frustration, depression, and anxiety. Using this scale, Castellini and colleagues (2011) demonstrated that SBEs exhibited a significant relationship with

anger/frustration in patients with BN and with depression in patients with BED. Thus, it is possible that the association between emotional eating and SBEs is dependent on the dimension of affect assessed. In addition, such associations might be further dependent on diagnostic status. Future research would benefit from further study of the relationship between OBEs and SBEs and multiple facets of emotional eating, in order to clarify the function of each binge type.

Several additional findings discrepant with the extant literature emerged from the data. For example, neither SBEs nor OBEs predicted frequency of self-induced vomiting or excessive exercise. The finding regarding self-induced vomiting in particular is in contrast to that of Brownstone et al. (2013), who found that OBEs and SBEs each significantly predicted unique variance in this compensatory behaviour. However, the mean frequencies of vomiting episodes over the previous 28 days in their study (M = 14.64 for the OBE group, M = 20.18 for the SBE group) were substantially higher than the mean frequency reported in the present sample (M = 0.24 for the total sample). Indeed, Brownstone et al. (2013) specifically recruited a clinical sample of individuals who endorsed the regular occurrence of either binge type in conjunction with purging. In contrast, the current study did not employ these inclusion criteria, and only 7 participants (approximately 6% of the sample) endorsed the occurrence of self-induced vomiting at any frequency over the previous 28 days. Nevertheless, given the relationship between SBEs and clinically relevant indicators of eating pathology in the present study, results suggests that even SBEs in the absence of regular purging behaviour should be the focus of increased clinical attention.

In contrast to our hypotheses, OBEs were not associated with elements of general pathology, including depression, anxiety, and stress, while SBEs were significantly related to anxiety and stress only. These results are surprising, given that previous research has demonstrated a robust association between depressive and anxiety symptoms and binge eating,

regardless of the amount of food consumed (e.g., Colles et al., 2008; Keel et al., 2001; Latner et al., 2007; Watson et al., 2013). It is possible that the discrepant results can be explained by the use of different samples. More specifically, the abovementioned studies recruited treatment seeking or community participants who fulfilled diagnostic criteria for an eating disorder or some subthreshold variant (e.g., purging disorder), whereas diagnostic status was not assessed in the present study. Perhaps SBEs operate differently in an undergraduate, nonclinical population than in a clinical one, though these results await replication.

Both SBEs and OBEs accounted for significant variance in eating disorder-specific cognitive distortions. On the other hand, SBEs uniquely predicted general cognitive distortions. The significant association between general cognitive distortions and SBEs lends partial support to our hypothesis regarding the unique importance of cognitive mechanisms in the maintenance of SBEs. Over the past several decades, an extensive body of literature has accumulated highlighting the importance of cognitive disturbance related to food, eating, shape, and weight in relation to disordered eating behaviour, and which has provided the basis for cognitive theories of eating disorders (see Cooper, 1997, 2005 for a review). The measure of eating disorder-specific cognitive distortions (i.e., the TSF) employed in the present study contains many items reflecting these specific themes. Thus, its relevance to both subjective and objective binge episodes is in line with existing theory and research. In contrast, the CDS is a broader measure of the tendency towards general negative styles of thinking. It is possible that while both types of binge eating (i.e., SBEs and OBEs) are associated with distorted thought content specific to eating disorder psychopathology, SBEs are more strongly characterized by a broader indicator of distorted thinking beyond that related to food, eating, and body concerns. The role that such distortions play in the etiology and maintenance of SBEs is as of yet uncertain, and requires elucidation by prospective enquiry.

Overall, these results replicate past findings indicating that SBEs and OBEs are equivalently related to a broad range of eating disorder pathology, although a few differences between binge types did emerge. It was further found that SBEs accounted for more variance in anxiety and stress symptomatology, as well as general cognitive distortions, than did OBEs. Although the nuanced distinctions between SBEs and OBEs might be clinically useful, the results of the current study suggest that this distinction does not merit emphasis in the diagnostic nomenclature.

Comparison Between Regular Subjective Binge Eaters and Nonbinge Controls

The hypothesis that participants who reported regular SBEs would exhibit elevated pathology on the clinical variables examined relative to participants who did not report binge eating was only partially supported. Indeed, significant between group differences emerged only for facets of eating disorder pathology. This latter finding is in keeping with previous results obtained from an undergraduate sample (Jenkins et al., 2012), indicating that individuals who exhibit loss of control eating in the absence of consuming an unusually large amount of food report greater levels of global eating pathology as measured by the EDE-Q than do nonbinge eating controls, and extends these findings to include cognitive restraint, disinhibited and emotional eating. Although the present sample size was small, the large effect sizes obtained suggest that these findings are relatively robust.

We had expected a significant between-group difference on measures of general pathology because previous research has demonstrated a consistent association between binge eating and depression and anxiety, as cited above. It is worth noting, however, that the mean DASS-21 scores obtained for the nonbinge control group were elevated in comparison to published nonclinical community norms (e.g., Antony et al., 1998; Sinclair, Siefert, Slavin-Mulford, Stein, Renna & Blais, 2012). One explanation is that negative affect tends to generally

be elevated in undergraduates students (Beiter et al., 2015; Mahoud, Staten, Hall & Lennie, 2012), who are entering a transitional period fraught with many challenges and stressors (particularly around midterm and exam time) that could negatively affect their mental health. Although mean scores were slightly higher in the SBE group, 18.5%, 62.9%, and 18.5% of the nonbinge control group demonstrated depression, anxiety, and stress scores in the moderate to severe range, respectively. Thus, elevated levels of general pathology in the control group might have limited the ability to detect between group differences on this measure.

The present study also did not reveal a significant between group difference on measures of general or eating disorder-specific cognitive distortions. It is plausible that the inability to distinguish individuals who endorsed disordered eating behaviour from those who did not on these variables is reflective of the commonality of general cognitive distortions in the broader population. To the best of the author's knowledge, no publications examining the prevalence or degree of general cognitive distortions in university samples exists; however, in an unpublished dissertation Roberts (2015) found that in a nonclinical, community sample, individuals aged 18-29 demonstrated significantly higher levels of cognitive distortions than those aged 41-85. Further, females were found to endorse higher levels of cognitive distortions than males. Although Roberts (2015) utilized the Inventory of Cognitive Distortions (Yurica & DiTomasso, 2002), the specific distortions included in this measure are comparable to those of the CDS. Thus, one might expect a sample of undergraduate women to generally evidence elevated levels of this particular pathology, making it difficult to differentiate subjective binge eaters from nonbinge controls with respect to this construct. In contrast, cognitive distortions related to eating pathology have been shown to differentiate those with disordered eating from those without (Coelho et al., 2013). Indeed, the nonsignificant MANOVA finding was surprising, particularly given the large perceived difference in mean scores on the TSF between the SBE and nonbinge

control groups. This study was small in scale, and therefore the sample size for each group was relatively small. A post hoc power analysis conducted using G*Power Software (version 3.1) revealed that, given the medium obtained effect size (V = 0.078) and α of 0.05, 126 participants would have been required to obtain power of 0.8 to detect a statistically significant effect. Perhaps the recruitment of a larger sample would further clarify whether there is specificity for measures of eating disorder-specific cognitive distortions in differentiating individuals who report engaging in subjective binge eating from those who do not.

Mediator and Moderator Analyses

Results of the mediation analyses revealed that eating disorder-specific cognitive distortions indirectly accounted for the relationship between dietary restraint and SBE frequency. Specifically, it appears likely that high levels of dietary restraint might contribute to greater maladaptive cognitions related to food, shape, and weight that, consequently, result in increased frequency of SBEs. For example, if a restrained eater endorses the belief "If I eat a forbidden or fattening food I will gain weight," this could result in even small lapses in restraint being appraised as a loss of control. The finding that such beliefs are common in restrained eaters is not in and of itself novel; however, this study is the first to show that they account for the documented relationship between dietary restraint and SBEs. This finding is noteworthy, as it further highlights the importance of cognitive factors in the maintenance of SBEs**,** and might hold implications for clinical practice. For example, Safer, Agras, Lowe and Bryson (2004) have reported that dietary restraint as measured by the TFEQ has not been shown to reduce significantly following CBT for BN. However, in the same study, dietary restraint as measured by the EDE-Q did decrease significantly. The authors concluded that the TFEQ appears to be less useful with respect to capturing changes in dietary restraint from pre to posttreatment. Yet, it is important to consider that items on the EDE-Q restraint scale place heavy emphasis on the

measurement of dieting, whereas items on the TFEQ restraint scale measures more the tendency towards cognitive restraint, regardless of actual caloric restriction. It is possible that the inability to detect changes on the TFEQ in their study was due to the continued presence of SBEs following treatment; albeit, this binge type was not assessed. Taken together with findings from the current study, this finding might suggest that placing an increased emphasis on targeting rigid beliefs about food, shape, and weight, as opposed to actual dieting behaviour, is important for patients who engage in SBEs.

In contrast to the abovementioned finding, general cognitive distortions did not explain the relationship between dietary restraint and frequency of SBEs. Thus, it appears that although a general tendency towards negative thinking styles contributes to the occurrence of SBEs, it is not related to dietary restraint. In addition, regarding tests of moderation, there was no evidence of an interaction between cognitive distortions and dietary restraint in the prediction of SBE frequency. This absence of moderation was observed for both general and eating disorder-specific cognitive distortions. As such, the relationship between dietary restraint and SBE frequency does not appear to be dependent on the level of either general or eating disorder-specific cognitive distortions.

Negative Affect and Stress as Proximal Antecedents of Subjective Binge Episodes

Given that a plethora of prior research has identified increases in negative affect and stress as triggers for OBEs, the present study examined whether these constructs also serve as triggers for SBEs. Findings derived from daily diary data revealed that higher levels of negative affect preceded SBEs, as compared to nonbinge eating episodes, within the SBE group. However, stress did not emerge as a statistically significant proximal antecedent to SBEs. Consistent with our hypotheses, there was no observed difference between negative affect and stress preceding nonbinge episodes between the SBE and nonbinge control groups.

The finding regarding negative affect is consistent with previous research employing ecological momentary assessment (EMA), which has shown that mood tends to be worse prior to the occurrence of binge episodes (see Haedt-Matt & Keel, 2011). In the objective binge eating literature, such findings have been explained most readily in the context of the affect regulation model of binge eating. This model proposes that increases in negative emotions trigger binge episodes, which function to alleviate negative affect via comfort and distraction (Hawkins & Clement, 1984). However, to date, support for this model has been inconsistent, as several studies have shown mood to actually *worsen* subsequent to a binge (Johnson & Larson, 1982; Stein, Kenardy, Wiseman, Dounchis, Arnow & Wilfley, 2007; Stickney & Miltenberger, 1999). Thus, it is as of yet unclear what maintains binge eating, and several competing explanations cannot be ruled out. For example, restraint theory (Herman & Polivy, 1980) posits that negative affect serves to disrupt or disinhibit cognitive control over attempts at dietary restriction, which reduces ones ability and/or desire to maintain dietary control. However, this model does not suggest that binge episodes are maintained through reductions in negative affect, but via a post-binge recommitment to dietary restraint in order to compensate for the binge, thereby perpetuating the dieting-binge eating cycle. Yet another model is escape theory (Heatherton & Baumeister, 1991), which proposes that binge eating episodes serve to reduce negative affect through the narrowing of cognitive attention away from aversive self-awareness (particularly relating to failure to meet high personal standards) to the more present environment (i.e., the act of eating). Similar to the affect regulation model and restraint theory, the escape model contends that increases in negative affect would precede binge eating episodes. In contrast, however, escape theory proposes that reductions of negative affect occur *during* binge eating due to lowered self-awareness, and that negative affect increases upon completion of a binge episode when self-awareness returns. As the present study did not assess for the trajectory of negative affect during and following a subjective

binge episode, we are unable to comment on how these results do or do not support the various maintenance theories of binge eating. Regardless, the present findings would suggest that future research should incorporate SBEs into the continued study of binge antecedents and consequences. In particular, the literature would benefit from a direct comparison of SBEs and OBEs, in order to delineate the similar versus unique functions of these two binge types.

It is interesting that negative affect was found to precipitate SBEs, in light of the fact that only OBEs emerged as a unique predictor of emotional eating via self-report. However, as noted above, one of the limitations of this measurement tool is that it assesses only three emotional states. As the present study measured negative affect more broadly, it in unclear what specific type of negative emotion might have been driving the propensity to engage in subjective binge eating in the current sample. Future research would benefit from the use of a more comprehensive list of negative emotions from which participants can chose when completing the daily food diary.

In contrast to the above results, and to our expectation, stress was not identified as a proximal trigger for subjective binge episodes. Previous studies using EMA have found stress to increase prior to binge episodes in patients with BN (Smyth et al., 2007), as well as to be greater prior to both binge episodes and regular meals than prior to random prompts in patients with BED (Le Grange et al., 2001), suggesting that stress may act as an immediate trigger of objective binge eating in these patient groups. Further, Rydin-Gray (2007) also found that in a sample of undergraduate women with BED, stress was greater prior to binges than regular meals and random prompts, and that levels of stress did not differ between binge episodes that were large in size, and those that were small in size. However, given the fact that, to date, stress has not been examined as a proximal antecedent of SBEs in a sample of participants characterized exclusively by the presence of regular subjective binge eating in the absence of regular objective binge

eating, it is possible that stress is not a relevant trigger for individuals with this specific disordered eating pattern.

Yet, similar to that for negative affect, the effect size for the difference in levels of stress preceding SBEs and nonbinge episodes was large, despite a nonsignificant *p* value. Although a statistical test of significance is standard in psychological research, and aids in interpretation of results, it does not provide information about the magnitude of the difference between two means (i.e., the practical significance), nor can it easily be compared across studies (Sullivan & Feinn, 2012). Furthermore, when examining effects using small samples, significance testing can be misleading as it conflates sample size and effect size, and is subject to type II error (Sullivan & Feinn, 2012). The effect size, in contrast, is independent of sample size. The fact that the effect size for stress was large ($d = 0.83$, approximately $8/10^{th}$ of a standard deviation) might suggest that elevated stress is relevant as an antecedent of subjective binge episodes, but that further research employing a larger sample is required.

In this study, participants were asked to rate their level of stress immediately preceding eating episodes on the daily food diary; however, participants were not provided with a comprehensive definition of stress. Indeed, stress is a complex construct that lacks a concise and universally accepted definition. Previous studies in which stress has been shown to be an antecedent of OBEs have attempted to narrow and make more explicit an operational definition by utilizing a subset of items from validated questionnaires, such as the Daily Stress Inventory (DSI; Brantley, Waggoner, Jones & Rappaport). A noted strength of the DSI includes its high convergent validity with endocrine measures of stress (e.g., Brantley, Dietz, McKnight, Jones, & Tulley, 1988). Furthermore, this measure includes subscales assessing stress related to daily hassles as well as interpersonal difficulties. Given that interpersonal problems have been linked to binge eating (Fairburn, 1997; Wilfley et al., 2002), and loss of control eating more broadly

(Ansell, Grilo & White, 2012; Brownstone et al., 2013), utilizing select items from the DSI in future studies examining antecedents of SBEs might prove fruitful, in order to elucidate specific types of events appraised as stressful that contribute to this binge type.

Of note, one interesting and unexpected finding that emerged from the diary data was that some participants in the regular SBE group did not record binge episodes during the 6-day monitoring period, whereas several participants in the nonbinge control group did. One might presume that the eating episodes recorded on the diary by individuals in the SBE group may not be fully representative of these participants' normative eating patterns, due to the potential reactive or therapeutic effect of self-monitoring. However, previous research highlighting the ineffectiveness of self-monitoring on the reduction of SBEs in particular would warn against this interpretation (Hildebrandt & Latner, 2006**).** Nevertheless, this possibility is difficult to examine statistically, and should be acknowledged when drawing conclusions. To add to the perplexity, in a study conducted by Grilo et al. (2001), the authors found prospective self-monitoring to result in higher rates of SBEs than those reported on the EDE-Q in a sample of 82 participants with BED. The self-monitoring period in their study spanned 4 weeks, however, whereas in the present study participants monitored their eating for only 6 days. Given logistical restraints, as well as the desire to limit participant burden and, thus, enhance study completion rates, a longer monitoring period was deemed imprudent. However, it is possible that the 6-day monitoring period was not sufficient to capture some participant binges. That is, the EDE-Q requires participants to report the total number of subjective binge episodes occurring over the previous 28 days. Though participants in the regular SBE group reported an *average* of one binge episode per week, the entirety of these binge episodes could conceivably all occur within a relatively brief period across the 28-day timeframe. Future research using a longer monitoring period might be beneficial, in order to increase the number of participant recorded binges.

Regarding SBEs reported by participants in the nonbinge control group, a similar finding also has been reported in studies employing EMA methods to examine binge eating in patients with BED and nonbinge controls. For example, le Grange, Gorin, Cately & Stone (2001) found in their sample that there was no difference in the rates of binge eating in BED and non-BED women over a 2-week monitoring period, despite conducting diagnostic interviews to confirm the absence of binge eating in the control group. Similarly, Greeno and colleagues (2000) found that two-thirds of women in their non-BED control group reported binge eating at a comparable frequency during their 6-day monitoring period. Interestingly, in their study, worse mood preceded binge episodes for women with BED, but not binge episodes for women without BED. Due to the small number of participants who recorded subjective binge episodes (n = 5) in the current study, a comparison of antecedents between SBEs in the SBE group and nonbinge control group was not conducted. Nevertheless, it does appear that SBEs occur in individuals without eating pathology; albeit, they might not serve the same function as they do for individuals who exhibit regular binge eating.

Study Limitations

Several limitations warrant consideration when interpreting the results of this study. First, the reported findings are based on a relatively small sample. This issue likely limited statistical power to find significant group differences between the SBE and nonbinge control groups on self-report measures, as well as antecedents on the diary data. Thus, it is important to note that null findings might have been influenced by low as opposed to meaningful group similarities. Furthermore, consistent with prior reports (Jenkins et al., 2013), in the current study the prevalence of OBEs was much lower than that of SBEs, which precluded the possibility of including OBE-only and mixed OBE and SBE presentations in the group comparison analyses. There is a paucity of research examining the clinical correlates of SBEs and OBEs in individuals

who report the regular occurrence of both binge types, but there is some data to suggest that such individuals demonstrate elevated levels of pathology as compared to those reporting the regular occurrence of just one binge type (Jenkins et al., 2012; Mond et al., 2010). This study would have benefited from the inclusion of these comparison groups, which might have been accomplished with a larger sample, in order to help further distinguish the clinical severity of SBE-only presentations.

Another important limitation of this study is that the assessment of SBEs was carried out via self-report. It has been well documented that the EDE-Q does not assess SBEs as reliably as it does OBEs (Reas, Grilo, & Masheb, 2004). This is likely due to the fact that the EDE-Q relies on participants' judgment alone of whether an amount of food is objectively large or not. In the present study, participants were provided with a definition of an "unusually large" amount of food prior to completing the EDE-Q, in an effort to increase accurate reporting of SBEs. However, agreement between raters and participants regarding the labeling of self-reported binge episodes as either SBEs or OBEs on the diary data was poor, which suggests that participants still had difficulty accurately identifying an unusually large amount of food. Thus, this study might have been strengthened by the use of the EDE-Interview, which allows the clinician to determine the presence of SBEs.

This study was the first of its kind to examine the proximal antecedents of subjective binge episodes in a group of participants selected specifically for endorsing only regular SBEs. The use of prospective daily food diaries is a noted strength of the study design, as this method of data collection reduces the likelihood of memory bias, and inspires confidence in an ecologically valid representation of the relationship between negative affect, stress, and eating episodes. However, due to the paper and pencil assessment of these variables, exact time points at which participants recorded their responses could not be examined. Although responses on the Exit

questionnaire suggest that the majority of participants completed ratings of negative affect and stress directly prior to eating, and recorded food consumption immediately following an eating episode, it would have been beneficial to corroborate this self-report with a method of objective assessment. This could have been accomplished using electronic devices (e.g., hand-held computers or phone applications) that allow for online data recording with time stamps. In addition, as previously mentioned there also are limitations regarding the measurement of binge eating antecedents. More specifically, the present study focused on the measurement of overall negative affect and stress proposed to contribute to the occurrence of SBEs. Participants were not asked to identify the specific negative or positive emotional states or nuanced aspects of the stress construct that preceded eating episodes; the assessment of which might have provided more fine-grained detail about SBE triggers. Furthermore, this study did not assess the trajectory of negative affect occurring during and following a binge and, thus, conclusions cannot be drawn regarding the function of SBEs.

Finally, results obtained from the daily diary must be considered in light of statistical limitations. More specifically, as Garson (2013) notes, uncorrelated error is an important assumption of statistical analyses within the general linear model family (e.g., t-tests). This assumption is often violated when error terms are not independent but instead cluster by one or more grouping variable over repeated observations, as in the present study (i.e., observations of antecedents clustered within type of eating episodes, and type of eating episode clustered within participant group). Applying traditional general linear model analysis to such data could potentially lead to misinterpretation of the magnitude of effects. In addition, this analytic approach can reduce power (Garson, 2013). Future research would benefit from the use of a more sophisticated analytic technique that holds the potential to overcome these limitations.

Clinical Implications

Despite noted limitations of this research, results from the current study add to a mounting body of literature, which suggests that SBEs are a clinically meaningful disordered eating behaviour that warrant inclusion in future revisions of the DSM eating disorder diagnostic criteria. In some respects, the results of this study corroborate the convincing argument that the prominent feature of binge eating is the presence of loss of control, as opposed to the size of a binge eating episode. If there were no meaningful differences in clinical correlates between binges that are objectively large in size, and those that are relatively small in size, accordant changes to eating disorder diagnostic criteria might consist only of expanding the size criterion so that SBEs are recognized within diagnoses other than OSFED, as previous authors have proposed (Mond et al., 2010; Watson et a., 2013). However, specific results of this and prior research (e.g., Brownstone et al., 2013; Keel et al., 2001) suggest that there might be utility in distinguishing between binge types based on the size of the eating episode. That is, some nuanced differences have emerged regarding the association between SBEs, OBEs and clinical correlates examined thus far, although arguably the only somewhat consistent finding has been the relationship between SBEs, as opposed to OBEs, and cognitive dietary restraint. If research continues to identify reliable differences in clinical correlates, as well as differential response to treatment, a more substantial modification to eating disorder diagnostic criteria might be defensible.

As Mond et al. (2010) notes, one attractive option to inspire such research is to include variants of binge eating-related disorders characterized by recurrent SBEs, but not OBEs, as provisional diagnoses in need of further study in the next iteration of the DSM, as was done with PD in DSM-5. Indeed, research on PD has increased in recent years, and has provided preliminary support for its distinctiveness from BN and its merit as a separate eating disorder diagnosis. Yet, it is worth noting that the inclusion of PD as a distinct eating disorder diagnosis

would not capture those individuals who engage in regular SBEs in combination with non-purging compensatory behaviors (e.g., excessive exercise), or who report the absence of any compensatory behaviours. Indeed, there is a notable paucity of research examining the prevalence and clinical correlates of variant BED presentations characterized by SBEs (Mond et al., 2010). As only a small minority of participants in the present study endorsed self-induced vomiting, our findings might speak to such a variant; however, further research in this area is needed as these results are preliminary.

Results from the present study also underscore the importance of increased attention to the monitoring and targeting of SBEs in treatment. Smith et al. (1992) and Niego et al. (1997) have postulated that the reason objective binge eating decreases more rapidly during CBT is that the experience of loss of control over eating takes longer to treat than does the regular consumption of objectively large amounts of food. In the initial stages of CBT for binge eating-related disorders, a heavy emphasis is placed on establishing normative eating patterns. This serves to reduce the pattern of dietary restriction followed by eventual disinhibition around food intake, which contributes to the perpetuation of objective binge eating (Cooper & Fairburn, 2010; Murphy, Straebler, Cooper & Fairburn, 2010). In contrast, for individuals with pure SBE presentations, or perhaps mixed SBE and OBE presentations, cognitive restructuring may need to be introduced earlier in treatment, either separate from or in conjunction with behavioural strategies. Further, given the present study's finding that SBEs seem to coincide with broader negative thinking styles, treatment for individuals who report regular SBEs may be improved by increased emphasis on targeting negative cognitive appraisals in broader domains, as opposed to only those relevant to eating, shape, and weight. On the other hand, findings derived from the diary data suggest a link between momentary negative affect and SBEs. This suggests that, similar to individuals characterized by regular OBEs, aiding individuals with SBEs in managing

distressing affect using more adaptive strategies might also be an important component of efforts to reduce this binge type. Future research would benefit from regularly including SBEs as an outcome variable in treatment studies, in order to determine whether novel or adapted treatment strategies are effective in targeting this particular type of binge episode.

Appendix A: EDE-Q

The following questions are concerned with the PAST FOUR WEEKS (28 days) only. Please read each question carefully and circle the appropriate number. Please answer all questions.

On how many of the past 28 days…	No days	1-5 days	6-12 days	13-15 days	16-22 days	23-27 days	Every day
1. Have you been deliberately trying to limit the amount of food you eat to influence your shape or weight (whether or not you have succeeded)?	0	1	2	3	4	5	6
2. Have you gone for long periods of time (8 waking hours or more) without eating anything at all in order to influence your shape or weight?	0	1	2	3	4	5	6
3. Have you tried to avoid eating any foods which you like in order to influence your shape or weight?	0	1	2	3	4	5	6
4. Have you tried to follow definite rules regarding your eating (for example, a calorie limit, a set amount of food, or rules about what or when you should eat) in order to influence your shape or weight (whether or not you have succeeded)?	0	1	2	3	4	5	6
5. Have you wanted your stomach to be empty?	0	1	2	3	4	5	6
6. Has thinking about food, eating or calories made it much more difficult to concentrate on things you are interested in (for example, reading, watching TV, or following a conversation)?	0	1	2	3	4	5	6
7. Have you been afraid of losing control over eating?	0	1	2	3	4	5	6
8. Have you had an episode of binge eating?	0	1	2	3	4	5	6
9. Have you eaten in secret? (Do not count binges.)	0	1	2	3	4	5	6
10. Have you definitely wanted your stomach to be flat?	0	1	2	3	4	5	6

11. Has thinking about shape or weight made it very difficult to concentrate on things you are interested in (for example, reading, watching TV, or following a conversation)?	0	1	2	3	4	5	6
12. Have you had a definite fear that you might gain weight or become fat?	0	1	2	3	4	5	6
13. Have you felt fat?	0	1	2	3	4	5	6
14. Have you had a strong desire to lose weight?	0	1	2	3	4	5	6

OVER THE PAST FOUR WEEKS (28 days)…

15. On what proportion of times that you have eaten have you felt guilty because of the effect on your shape or weight? (Do not count binges.)

    (Circle the number which applies.)

    0 – None of these times
    1 – A few of these times
    2 – Less than half of these times
    3 – Half of these times
    4 – More than half of the times
    5 – Most of the times
    6 – Every time

16. Over the past four weeks (28 days), have there been any times when you have felt that you have eaten what other people would regard as an unusually large amount of food given the circumstances? (Please circle YES or NO and indicate the appropriate number on the line.) YES NO

17. How many such episodes have you had over the past four weeks? _____

18. During how many of these episode of overeating did you have a sense of having lost control over your eating? _____

19. Have you had other episodes of eating in which you have had a sense of having lost control and eaten too much, but have not eaten an unusually large YES NO

amount of food given the circumstances?		_____
20. How many such episodes have you had over the past four weeks?		
21. Over the past four weeks have you made yourself sick(vomited) as a means of controlling your shape or weight?	YES	NO
22. How many times have you done this over the past four weeks?		_____
23. Have you taken laxatives as a means of controlling your shape or weight?	YES	NO
24. How many times have you done this over the past four weeks?		_____
25. Have you taken diuretics (water tablets) as a means of controlling your shape or weight?	YES	NO
26. How many times have you done this over the past four weeks?		_____
27. Have you exercised hard as a means of controlling your shape or weight?	YES	NO
28. Have many times have you done this over the past four weeks?		_____

Over the PAST FOUR WEEKS (28 DAYS).. (please circle the number which best described your behaviour)	**Not at all**		**Slightly**		**Moderately**		**Markedly**
29. Has your weight influenced how you think about (judge) yourself as a person?	0	1	2	3	4	5	6
30. Has your shape influenced how you think about (judge) yourself as a person?	0	1	2	3	4	5	6
31. How much would it have upset you if you had to weigh yourself once a week (no more or less often) for the next four weeks?	0	1	2	3	4	5	6
32. How dissatisfied have you been with your weight?	0	1	2	3	4	5	6
33. How dissatisfied have you been with your shape?	0	1	2	3	4	5	6
34. How uncomfortable have you felt seeing your body (for example, seeing your shape in the mirror, in a window reflection, while undressing, or taking a bath or shower)?	0	1	2	3	4	5	6

35. How uncomfortable have you felt about <u>others</u> seeing your shape or figure (for example, in communal changing rooms, when swimming, or wearing tight clothes)?	0	1	2	3	4	5	6

What is your weight at present? (Please give your best estimate) _____

What is your height? (Please give your best estimate) _____

Appendix B: EDE-Q instructions

Some items on this questionnaire will ask you about any times when you have eaten what most people would regard as an unusually large amount of food given the circumstances, while feeling a sense of having lost control over your eating. Different people mean different things by an "unusually large" amount of food. However, for the purposes of answering this questionnaire, an unusually large amount of food is something that most people would feel is more than a large meal.

A sense of having lost control while eating might be experienced as feeling driven or compelled to eat; not being able to stop eating once you have started; not being able to keep yourself from eating large amounts of, or certain kinds of, food in the first place; or giving up on even trying to control your eating because you know that, no matter what, you are going to overeat.

Other questions will ask you about any times in which you have experienced a sense of having lost control and eaten too much, but have **not** actually eaten an unusually large amount of food. This question is referring to times when you have felt that you have eaten too much at one time but others might not agree that it was too much.

Here are some examples:

"After work one evening, Marie ate three pieces of chicken, a 16-ounce package of frozen vegetables, three cups of rice, three fourths of a coffee cake, and a piece of fruit. This is an unusually large amount of food. While she ate Marie felt completely out of control, like she could not stop herself from continuing to eat."

"For lunch one day, Joseph had a ham and cheese sandwich with mayonnaise on a roll, a candy bar, and a diet coke. This meal was not unusually large. However, Joseph felt out of control because he had planned to have turkey on whole wheat with lettuce and tomato plus a piece of fruit for dessert, but changed his mind at the last minute while ordering his sandwich. Joseph may have perceived that he had eaten a larger amount of food than he should have, but others might not agree that he ate an unusually large amount of food."

Marie and Joseph both experienced binge eating episodes, as they both experienced a sense of loss of control, even thought Joseph ate much less food than the unusually large amount that Marie consumed.

Appendix C: TFEQ-R18

INSTRUCTIONS: Below is a set of statements describing feelings or experiences you may have had, or are familiar with because you have had them for a long time. Please read each statement, and select from the multiple choice options the answer that indicates the frequency with which you find yourself feeling or experiencing what is being described in the statements below.

1. When I smell a delicious food, I find it very difficult to keep from eating, even if I have just finished a meal.

   Definitely true (4)/ mostly true (3)/ mostly false (2)/ definitely false (1)

2. I deliberately take small helpings as a means of controlling my weight.

   Definitely true (4)/ mostly true (3)/ mostly false (2)/ definitely false (1)

3. When I feel anxious, I find myself eating.

   Definitely true (4)/ mostly true (3)/ mostly false (2)/ definitely false (1)

4. Sometimes when I start eating, I just can't seem to stop.

   Definitely true (4)/ mostly true (3)/ mostly false (2)/ definitely false (1)

5. Being with someone who is eating often makes me hungry enough to eat also.

   Definitely true (4)/ mostly true (3)/ mostly false (2)/ definitely false (1)

6. When I feel blue, I often overeat.

   Definitely true (4)/ mostly true (3)/ mostly false (2)/ definitely false (1)

7. When I see a real delicacy, I often get so hungry that I have to eat right away.

   Definitely true (4)/ mostly true (3)/ mostly false (2)/ definitely false (1)

8. I get so hungry that my stomach often seems like a bottomless pit.

   Definitely true (4)/ mostly true (3)/ mostly false (2)/ definitely false (1)

9. I am always hungry so it is hard for me to stop eating before I finish the food on my plate.

   Definitely true (4)/ mostly true (3)/ mostly false (2)/ definitely false (1)

10. When I feel lonely, I console myself by eating.

    Definitely true (4)/ mostly true (3)/ mostly false (2)/ definitely false (1)

11. I consciously hold back at meals in order not to weight gain.

    Definitely true (4)/ mostly true (3)/ mostly false (2)/ definitely false (1)

12. I do not eat some foods because they make me fat.

    Definitely true (4)/ mostly true (3)/ mostly false (2)/ definitely false (1)

13. I am always hungry enough to eat at any time.

    Definitely true (4)/ mostly true (3)/ mostly false (2)/ definitely false (1)

14. How often do you feel hungry?

    Only at meal times (1)/ sometimes between meals (2)/ often between meals (3)/ almost always (4)

15. How frequently do you avoid "stocking up" on tempting foods?

    Almost never (1)/ seldom (2)/ usually (3)/ almost always (4)

16. How likely are you to consciously eat less than you want?

    Unlikely (1)/ slightly likely (2)/ moderately likely (3)/ very likely (4)

17. Do you go on eating binges though you are not hungry?

    Never (1)/ rarely (2)/ sometimes (3)/ at least once a week (4)

18. On a scale of 1 to 8, where 1 means no restraint in eating (eating whatever you want, whenever you want it) and 8 means total restraint (constantly limiting food intake and never "giving in"), what number would you give yourself?

    _____

Appendix D: DASS-21

Please read each statement and circle a number 0, 1, 2 or 3 which indicates how much the statement applied to you *over the past week.* There are no right or wrong answers. Do not spend too much time on any statement.

The rating scale is as follows:

0 Did not apply to me at all
1 Applied to me to some degree, or some of the time
2 Applied to me to a considerable degree, or a good part of time
3 Applied to me very much, or most of the time

1	I found it hard to wind down	0	1	2	3
2	I was aware of dryness of my mouth	0	1	2	3
3	I couldn't seem to experience any positive feeling at all	0	1	2	3
4	I experienced breathing difficulty (e.g., excessively rapid breathing, breathlessness in the absence of physical exertion)	0	1	2	3
5	I found it difficult to work up the initiative to do things	0	1	2	3
6	I tended to over-react to situations	0	1	2	3
7	I experienced trembling (e.g., in the hands)	0	1	2	3
8	I felt that I was using a lot of nervous energy	0	1	2	3
9	I was worried about situations in which I might panic and make a fool of myself	0	1	2	3
10	I felt that I had nothing to look forward to	0	1	2	3
11	I found myself getting agitated	0	1	2	3
12	I found it difficult to relax	0	1	2	3
13	I felt down-hearted and blue	0	1	2	3
14	I was intolerant of anything that kept me from getting on with what I was doing	0	1	2	3
15	I felt I was close to panic	0	1	2	3
16	I was unable to become enthusiastic about anything	0	1	2	3
17	I felt I wasn't worth much as a person	0	1	2	3
18	I felt that I was rather touchy	0	1	2	3
19	I was aware of the action of my heart in the absence of physical exertion (e.g., sense of heart rate increase, heart missing a beat)	0	1	2	3

20	I felt scared without any good reason	0	1	2	3
21	I felt that life was meaningless	0	1	2	3

Appendix E: TSF

Please rate each statement by putting a circle around the number that best describes how much you agree with the statement, or how much it is true of you, in general. Even though some of your responses may seem irrational to you, we want to know what you think on an emotional level. Please answer every item without spending too much time on any particular item.

	Not at all	**Slightly**	**Moder-ately**	**Very much**	**Totally / Always**
1. I feel fatter after thinking about eating 'fattening'/'forbidden' foods (e.g., chocolate).	0	1	2	3	4
2. If I think about gaining weight, I want to check that my clothes aren't fitting more tightly.	0	1	2	3	4
3. Thinking about gaining weight is almost as immoral to me as actually gaining weight.	0	1	2	3	4
4. Just picturing myself gaining weight can really make me gain weight.	0	1	2	3	4
5. I feel huge if I just imagine not exercising for a month.	0	1	2	3	4
6. Just thinking about "pigging-out" makes me want to weigh myself.	0	1	2	3	4
7. Thinking about breaking my diet makes me want to check in the mirror that I don't look any fatter.	0	1	2	3	4
8. Just thinking about not exercising can change the way I really look.	0	1	2	3	4
9. I feel fatter if I just think about "pigging-out".	0	1	2	3	4
10. Just thinking about not exercising for a month makes me want to cut down on what I eat.	0	1	2	3	4
11. If I think about breaking my diet, it is almost as unacceptable as really breaking my diet	0	1	2	3	4
12. My shape can actually change, just by me planning to eat fattening food.	0	1	2	3	4
13. I feel fatter just by thinking about gaining weight.	0	1	2	3	4
14. Picturing myself eating 'fattening'/'forbidden' foods (e.g., chocolate) makes me want to check my body to make sure I haven't gained any weight.	0	1	2	3	4
15. How often do you have thoughts about the effects of eating fattening/'forbidden' foods?	0	1	2	3	4
16. Generally, to what extent do thoughts about 'forbidden' foods affect you, or interfere with your	0	1	2	3	4

daily life?					
17. When you have thoughts about 'forbidden' foods, to what extent is it *important* for you to get them out of your mind?	0	1	2	3	4
18. When you have thoughts about 'forbidden' foods, to what extent is it *difficult* to get them out of your mind?	0	1	2	3	4

Appendix F: CDS

Instructions: We would like to find out about the different types of thinking you use. In this questionnaire, you will read about 10 types of thinking. You will be given a description of each thinking type. You will also read two case examples that help explain the thinking type. There will always be two case examples: one dealing with social relationships (such as friends, partners and family) and one that deals with personal achievements (such as passing a test or failing a task at work). These case examples are used to help you understand how each type of thinking might look in a real life scenario.

Your task is to try and understand the thinking type that is described. Then, you are asked to estimate how often you use that type of thinking. You will be asked to think about how often you use that type of thinking in the two domains previously described (social and achievement scenarios). Please take time to think about your answers.

1. MINDREADING

People will sometimes assume that others are thinking negatively about them. This might occur even though the other person has not said anything negative. This is sometimes called mindreading. To illustrate this, please read the following passages:

A. Sonya is having coffee with her boyfriend Jim. Jim is quiet, and Sonya asks if anything is wrong. Jim replies that he is 'Okay.' Sonya does not believe Jim. She starts to think that he is unhappy with her.

B. Bob has been working on a project for weeks. He finally gives the final product to his boss, and is curious about his boss' opinion of his work. After a few days pass, Bob starts to worry that his boss thinks he is incompetent.

Please estimate how often you engage in Mindreading when in social situations (like when you're with friends, partners or family).

1	2	3	4	5	6	7
Never			Sometimes			All The Time

Please estimate how often you engage in Mindreading when in achievement situations (such as school or work).

1	2	3	4	5	6	7
Never			Sometimes			All The Time

2. CATASTROPHIZING

People can make negative predictions about the future. When there isn't much evidence for these predictions, it is called Catastrophizing. To illustrate, please read the following passage:

A. John is in his first year of university. He just received a 70 on his Biology exam. He immediately starts to worry that he will end up with a low grade in the course, and that he'll have a tough time getting into medical school.

B. Tina's boyfriend just gave her some feedback about their relationship. He told her that he would like to spend a little more time with his friends. Based on his feedback, Tina starts to predict that they will start to become distant, and eventually break-up.
Please estimate how often you engage in Catastrophizing when in social situations (like when you're with friends, partners or family).

1	2	3	4	5	6	7
Never			Sometimes			All The Time

Please estimate how often you engage in Catastrophizing when in achievement situations (such as school or work).

1	2	3	4	5	6	7
Never			Sometimes			All The Time

3. ALL-OR-NOTHING THINKING
When people make evaluations, they can view things as being "either-or." For example, a concert can be considered to be either good or bad. On the other hand, people can also see shades of gray when making evaluations. For example, a concert can have some negative aspects, but be considered fairly good overall. When a person considers something as being either good or bad, we call that all-or-nothing thinking. To illustrate this point further, please read the following passages:

A. Brian gets a B+ on an exam. He is disappointed because it was not an A. He tends to view success on exams as follows: 'I either do great, or my performance is a failure.'

B. Erin is the type of person who either likes a person, or dislikes them. You're either in her "good book" or you're not.

Please estimate how often you use All-or-Nothing Thinking when in social situations (like when you're with friends, partners or family).

1	2	3	4	5	6	7
Never			Sometimes			All The Time

Please estimate how often you use All-or-Nothing Thinking when in achievement situations (such as school or work).

1	2	3	4	5	6	7
Never			Sometimes			All The Time

4. EMOTIONAL REASONING
People can believe something to be true because it "feels" that way. To illustrate, please read the following passages:

A. Kim's friends told her that she could not come to the concert with them because they were

unable to get enough tickets for everyone. Kim knows they probably didn't exclude her on purpose, but she feels rejected. Therefore, part of her believes she was rejected.

B. Ted's boss told him that his performance at the company has been good. Yet, Ted wonders if he could have done better. In fact, he feels like a failure. Consequently, he starts to believe he is a failure.

Please estimate how often you engage in Emotional Reasoning when in social situations (like when you're with friends, partners or family).

1	2	3	4	5	6	7
Never			Sometimes			All The Time

Please estimate how often you engage in Emotional Reasoning when in achievement situations (such as school or work).

1	2	3	4	5	6	7
Never			Sometimes			All The Time

5. LABELING

People can label themselves as being a certain kind of person. If this occurs after something bad happens, it is called labeling. To illustrate, please read the following passages:

A. While at a social event, John asks a woman if she would like to dance. She turns him down. As a result, John considers himself to be a loser.

B. During class, Allison's teacher asks if anyone knows the answer to a question. Allison raises her hand and gives an answer. Her teacher says 'Unfortunately, that is incorrect. Does anyone else know the answer?' Allison tells herself that she is a moron.

Please estimate how often you engage in Labeling when in social situations (like when you're with friends, partners or family).

1	2	3	4	5	6	7
Never			Sometimes			All The Time

Please estimate how often you engage in Labeling when in achievement situations (such as school or work).

1	2	3	4	5	6	7
Never			Sometimes			All The Time

6. MENTAL FILTER

People sometimes have a filter for information. When there is positive and negative information, they only focus on the negative information. This is called Mental Filtering. To illustrate, please read the following passages:

A. Lauren overhears her new boyfriend, Tom, telling his friends about her. He says 'Yeah, things are going great so far. She's really smart and fun to be with, and we have a lot in common. She can be a bit demanding at times, but that's OK.' Although Tom had mostly positive things to say, Lauren dwelled on the one negative comment, and felt bad.

B. Ed is a high school student. He is reading comments from his teacher regarding his recent essay. His teacher wrote 'Ed, you have an excellent way of expressing ideas. I really enjoy the way you write. However, you should try and make better transitions from one idea to an- other.' Despite the fact that Ed clearly performed well, he could only think about the one piece of criticism, and felt poorly about himself.

Please estimate how often you engage in Mental Filtering when in social situations (like when you're with friends, partners or family).

1	2	3	4	5	6	7
Never			Sometimes			All The Time

Please estimate how often you engage in Mental Filtering when in achievement situations (such as school or work).

1	2	3	4	5	6	7
Never			Sometimes			All The Time

7. OVERGENERALIZATION

When a negative event occurs, people might assume more bad things are going to happen. They see the negative event as the start of a pattern. To illustrate, please read the following passages:

A. Janet's boyfriend just broke up with her. She thinks to herself: 'I am never going to get into a stable relationship.'

B. William recently failed his math exam. He thinks to himself: 'I'll probably fail the exams in my other courses as well.'

Please estimate how often you engage in Overgeneralization when in social situations (like when you're with friends, partners or family).

1	2	3	4	5	6	7
Never			Sometimes			All The Time

Please estimate how often you engage in Overgeneralization when in achievement situations (such as school or work).

1	2	3	4	5	6	7
Never			Sometimes			All The Time

8. PERSONALIZATION

People can believe they are responsible for negative things, even though they're not. In other

words, they take a negative event, and assume they are the cause of it. This is called Personalization. To illustrate, please read the following passages:

A. Sally's company did not get an important contract. Although many people worked hard on this project, she assumes that it is her fault.

B. Chris's best friend has been in a bad mood lately, and it has been hard to get in contact with him. Chris assumes that he must have personally done something wrong to make his friend act this way.

Please estimate how often you engage in Personalization when in social situations (like when you're with friends, partners or family).

1	2	3	4	5	6	7
Never			Sometimes			All The Time

Please estimate how often you engage in Personalization when in achievement situations (such as school or work).

1	2	3	4	5	6	7
Never			Sometimes			All The Time

9. SHOULD STATEMENTS

People sometimes think that things should or must be a certain way. To illustrate, please read the following passages:

A. "Billy is upset with getting an 85 on his exam because he thinks he should get at least a 90. He often has these thoughts for many things (e.g., he feels he should never drop a pass when playing football; his room should be organized a certain way)."

B. "Anne believes that she must be funny and interesting when socializing." Please estimate how often you tend to make Should Statements when in social situations (like when you're with friends, partners or family).

1	2	3	4	5	6	7
Never			Sometimes			All The Time

Please estimate how often you tend to make Should Statements when in achievement situations (such as school or work).

1	2	3	4	5	6	7
Never			Sometimes			All The Time

10. MINIMIZING OR DISQUALIFYING THE POSITIVE

People can sometimes ignore the positive things that happen to them. This is called Minimizing or Disqualifying the Positive. To illustrate, please read the following passages:

A. Brenda works as a real estate agent. Her boss recently told her that she did a wonderful job on

a recent sale. In her head, she dismisses her achievement because she probably ‘just got lucky.’

B. Cory is getting ready for a big first date. His friends tell him he looks good. He dismisses their complement because he thinks they’re just trying to be nice.

Please estimate how often you tend to Minimize or Disqualify the Positive when in social situations (like when you’re with friends, partners or family).

1	2	3	4	5	6	7
Never			Sometimes			All The Time

Please estimate how often you tend to Minimize or Disqualify the Positive when in achievement situations (such as school or work).

1	2	3	4	5	6	7
Never			Sometimes			All The Time

Appendix G: 6-Day Food Diary

The following forms will ask you to record your food and liquid consumption and answer questions about your eating experiences over the next 6 days.

Please complete the following for each eating episode each day:

1. **RECORD** the time you began eating, including AM or PM
2. **RATE** your level of negative affect before you begin eating on a scale from 1 (low negative affect) to 7 (high negative affect)

   Negative affect refers to a variety of negative moods including, but not limited to, sadness, anger, anxiety, guilt etc.
3. **RATE** your level of stress before you begin eating on a scale from 1 (not at all stressed) to 7 (very stressed)
4. **RECORD** a description of the food and/or beverage you consumed as well as the quantity of food you consumed
5. **INDICATE** whether the eating episode was a meal (e.g., breakfast, lunch, dinner) or a snack
6. **INDICATE** whether you consider the eating episode to be a binge or not
7. **INDICATE** whether what you ate was **an unusually large amount of food**

   An unusually large amount of food is what most people would feel is more than a large meal
8. **RATE** the degree of loss of control you experienced while eating from 1 (no loss of control) to 7 (extreme loss of control)

   A sense of having lost control while eating might be experienced as feeling driven or compelled to eat; not being able to stop eating once you have started; not being able to keep yourself from eating large amounts, or certain kinds of food in the first place; or giving up on even trying to control your eating because you know that, no matter what, you are going to overeat.

Sample Food Diary

TIME	NEGATIVE AFFECT	STRESS	FOOD AND DRINK	MEAL OR SNACK	BINGE	UNUSUALLY LARGE	LOSS OF CONTROL
8:30 AM	2	2	1 cup of coffee with milk and sugar, 1 cup of low fat yogurt with a quarter cup of granola, 1 banana	Meal (Breakfast)	No	No	1
10:50 AM	2	4	1 ounce cheddar cheese, 15 crackers, 1 500 mL bottle of water	Snack	No	No	1
1:00 PM	1	2	Large Greek salad with 1 grilled chicken breast, 1 small bowl of tomato soup	Meal (Lunch)	No	No	2
3:15 PM	3	3	1 medium size apple, 3 chocolate chip cookies	Snack	No	No	3
6:30 PM	6	5	1 6-inch Subway sandwich tuna on whole wheat with lettuce, tomato, onions and mayonnaise, 1 500 ml bottle of coke, 1 small bag of Doritos chips.	Meal (Dinner)	Yes	Yes	6

Date: _____

TIME	NEGATIVE AFFECT	STRESS	FOOD AND DRINK	MEAL OR SNACK	BINGE	UNUSUALLY LARGE	LOSS OF CONTROL

Appendix H: Demographic Questionnaire

Age:

Enter # of years
_____ years

Ethnicity

Please check off the ethnocultural groups to which you most strongly identify:

- □ Aboriginal Canadian
- □ African Canadian
- □ Caribbean Canadian
- □ East/Southeast Asian Canadian
- □ European Canadian
- □ Latin/Central/South American Canadian
- □ Oceanic Canadian
- □ Pacific Islander Canadian
- □ South Asian Canadian
- □ West Asian/Arab/Maghrebi Canadian
- □ Other

Education

Please indicate the highest level of education you have obtained:

- □ Graduated high school
- □ Completed some college/university
- □ Bachelor's degree
- □ Master's degree
- □ Doctorate
- □ Professional degree

Relationship Status

Are you currently:

- □ Single
- □ Dating but neither married nor common-law
- □ Married
- □ Common-Law
- □ Divorced
- □ Widowed

Appendix I: Exit Questionnaire

1. How often did you rate your negative affect and stress **right before** eating?

1	2	3	4
Every time	Most of the time	Some of the time	None of the time

2. How often did you record your food consumption and ratings of loss of control **directly after** eating?

1	2	3	4
Every time	Most of the time	Some of the time	None of the time

3. How often did you complete the entire food record at the end of the day (as opposed to throughout the day)?

6 days	5 days	4 days	3 days	2 days	1 day	No days

4. How often did you report a smaller amount of food than you actually ate, or omit recording a food item that you consumed?

1	2	3	4
Every time	Most of the time	Some of the time	None of the time

References

Abraham, S. F., & Beumont, P. J. (1982). How patients describe bulimia or binge eating. *Psychological Medicine, 12,* 625–635. doi:10.1017/S0033291700055732.

Aiken, L. S., & West, S. G. (1991). *Multiple regression: Testing and interpreting interactions.* Newbury, CA: Sage Publications, Inc.

American Psychiatric Association (1980) *Diagnostic and statistical manual of mental disorders* (3rd ed.). Washington, DC: Author.

American Psychiatric Association. (1987). *Diagnostic and statistical manual of mental disorders* (3rd ed., rev.). Washington, DC: Author.

American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders (*4th ed*).* Washington, DC: Author.

American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th ed., text rev.). Washington, DC: Author.

American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Arlington, VA: American Psychiatric Publishing.

Angle, S., Engblom, J., Eriksson, T., Kautiainen, S., Saha, M-T., Lindfors, P., … Rimpela, A. (2009). Three factor eating questionnaire-R18 as a measure of cognitive restraint, uncontrolled eating and emotional eating in a sample of young Finnish females. *International Journal of Behavioral Nutrition and Physical Activity*, 6, 41. doi:10.1186/1479-5868-6-41.

Ansell, E. B., Grilo, C. G., & White, M. A. (2012). Examining the interpersonal model of binge eating and loss of control over eating in women. *International Journal of Eating Disorders, 45,* 43-50. doi:10.1002/eat.20897.

Antony, M. M., Bieling, P. J., Cox, B. J., Enns, M. W. & Swinson, R. P. (1998). Psychometric properties of the 42-item and 21-item versions of the depression anxiety stress scales (DASS) in clinical groups and a community sample. *Psychological Assessment*, *10*, 176-181. doi:10.1037/1040-3590.10.2.176.

Apple, R.G., & Agras, W.S. (2008). *Overcoming your eating disorder: A cognitive-behavioral approach for bulimia nervosa and binge-eating disorder* (2nd ed.). New York, NY: Oxford University Press.

Arnow, B., Kenardy, J., & Agras, W. S. (1995). The emotional eating ecale: The development of a measure to assess coping with negative affect by eating. *International Journal of Eating Disorders, 18,* 79–90. doi:10.1002/1098-108X(199507)18:13.0.CO;2-V.

Arnow, B., Kenardy, J., & Agras, W. S. (1992). Binge eating among the obese: A descriptive study. *Journal of Behavioral Medicine, 15,* 155–170. doi:10.1007/BF00848323

Baron, R. M., & Kenny, D. A. (1986). The moderator-mediator variable distinction in social psychological research: Conceptual, strategic and statistical considerations. *Journal of Personality and Social Psychology, 51*, 1173-1182. doi:10.1037/0022-3514.51.6.1173

Beiter, R., Nash, R., McCrady, M., Rhoades, D., Linscomb, M., Clarahan, M., & Sammut, S. (2015). The prevalence and correlates of depression, anxiety, and stress in a sample of college students. *Journal of affective disorders, 173,* 90–96. doi:10.1016/j.jad.2014.10.054.

Berg, K. C., Peterson, C. B., Frazier, P., & Crow, S. J. (2011). Psychometric evaluation of the eating disorder examination and eating disorder examination-questionnaire: A systematic review of the literature. *International Journal of Eating Disorders, 45*, 428–438. doi:10.1002/eat.20931.

Brantley, P. J., Waggoner, C. D., Jones, G. N., & Rappaport, N. B. (1987). A daily stress inventory: Development, reliability, and validity. *Journal of Behavioural Medicine, 10,* 61-74. doi:10.1007/BF00845128.

Brownstone, L. M., Bardone-Cone, A. M., Fitzsimmons-Craft, E. E., Printz, K. S., Le Grange, D., Mitchell, J. E., … Joiner, T. E. (2013). Subjective and objective binge eating in relation to eating disorder symptomatology, negative affect, and personality dimensions. *International Journal of Eating Disorders, 46,* 66-76. doi:10.1002/eat.22066.

Bulik, C. M., Sullivan, P. F., & Kendler, K .S. (2002). Medical and psychiatric morbidity in obese women with and without binge eating. *International Journal of Eating Disorders. 32,* 72–78. doi:10.1002/eat.10072.

Burd, C., Mitchell, J. E., Crosby, R. D., Engel, S. G., Wonderlich, S. A., Lystad, C., … Crow, S. (2009). An assessment of daily food intake in participants with anorexia nervosa in the natural environment. *International Journal of Eating Disorders, 42,* 71–374. doi: 10.1002/eat.20628.

Castellini, G., Mannucci, E., Lo Sauro, C., Benni, L., Lazzaretti, L., Ravaldi, C., … Ricca, V. (2011). Different moderators of cognitive-behavioral therapy on subjective and objective binge eating in bulimia nervosa and binge eating disorder: A three-year follow-up study. *Psychotherapy and Psychosomatics, 81,* 11-20. doi:10.1159/000329358

Chua, J. L., Touyz, S., & Hill, A. J. (2004). Negative mood-induced overeating in obese binge eaters: An experimental study. *International Journal of Obesity, 28,* 606–610. doi: 10.1038/sj.ijo.0802595.

Coelho, J. S., Baeyens, C., Purdon, C., Shafran, R., Roulin, J., & Bouvard, M. (2013). Assessment and thought-shape fusion: Initial validation of a short version of the trait

thought-shape fusion scale. *International Journal of Eating Disorders, 46,* 77-85. doi: 10.1002/eat.22038.

Cohen, D. (1977). *Statistical Power for the Behavioral Sciences*. New York, NY: Academic Press.

Cools, J., Schotte, D. E., & McNally, R. J. (1992). Emotional arousal and overeating in restrained eaters. *Journal of Abnormal Psychology, 101,* 348–351. doi:10.1037/0021-843X.101.2.348,

Cooper, M. J. (1997). Cognitive theory in anorexia nervosa and bulimia nervosa: A review. *Clinical Psychology Review, 25,* 511-31. doi:10.1017/S1352465800018348.

Cooper, Z, & Fairburn, C. (2010). Cognitive behavior therapy for bulimia nervosa. In C. Grilo & J. Mitchell (Eds.), *The treatment of eating disorders*. New York, NY: Guilford.

Covin, R., Dozois, D. J. A., Ogniewicz, A., & Seeds, P. M. (2011). Measuring cognitive errors: Initial development of the cognitive distortions scale (CDS). *International Journal of Cognitive Therapy, 4,* 297-322. doi: 10.1521/ijct.2011.4.3.297.

Crow, S. J., Agras, W. S., Halmi, K., Mitchell, J. E., & Kraemer, H. C. (2002). Full syndromal versus subthreshold anorexia nervosa, bulimia nervosa, and binge eating disorder: A multicenter study. *International Journal of Eating Disorders, 32,* 309-318. doi: 10.1002/eat.10088

Crowther, J. H., Scaftner, J., Bonifazi, D. Z., & Shepherd, K. L. (2001). The role of daily hassles in binge eating. *International Journal of Eating Disorder, 29,* 449-454. doi: 10.1002/eat.1041.

de Lauzon, B., Romon, M., Deschamps, V., Lafay, L., Borys, J. M., Karlsson, J., … Fleurbaix Laventie Ville Sante Study Group. (2004). The three-factor eating questionnaire-R18 is

able to distinguish among different eating patterns in a general population. *Journal of Nutrition, 134,* 2372-2389. doi:10.1093/jpepsy/jsn002.

DeFife, J. (2009). Effect Size Calculator. Available from http://web.cs.dal.ca/~anwar/ds/Excel4.xlsx.

Devlin, M. J., Goldfein, J. A., & Dobrow, I. (2003). What is this thing called BED? Current status of binge eating disorder nosology. *International Journal of Eating Disorders, 34,* 2–18. doi:10.1002/eat.10201.

Dozois, D. J., Ahnberg, J. L., & Dobson, K. S. (1998). Psychometric Evaluation of the Beck Depression Inventory-II. *Psychological Assessment, 10*, 83-89. doi:10.1590/1516-4446-2012-1048.

Elran-Barak, R., Sztainer, M., Goldschmidt, A. B., Crow, S. J., Peterson, C. B., Hill, L. L., … Le Grange, D. (2015). Dietary restriction behaviors and binge eating in anorexia nervosa, bulimia nervosa and binge eating disorder: Transdiagnostic examination of the restraint model. *Eating Behaviors, 18,* 192-196. doi:10.1016/j.eatbeh.2015.05.012.

Gleaves, D. H., Williamson, D. A., & Barker. S. E. (1993). Additive effects of mood and eating forbidden foods upon the perceptions of overeating and binging in bulimia nervosa. *Addictive Behaviors, 18,* 299-309. doi:10.1016/0306-4603(93)90031-4.

Goldfein, J. A., Devlin, M. J., & Kamenetz, C. (2005). Eating disorder examination questionnaire with and without instructions to assess binge eating in patients with binge eating disorder. *International Journal of Eating Disorders, 37,* 107-111. doi:10.1002/eat.20075.

Heatherton, T. F., & Baumeister, R. F. (1991). Binge eating as escape from self-awareness. *Psychological Bulletin, 110,* 86–108. doi: 10.1037/0033-2909.110.1.86.

Heatherton, T. F., Striepe, M., & Wittenberg, L. (1998). Emotional distress and uncontrolled eating: The role of self. *Personality and Social Psychology Bulletin, 24,* 301–313. doi: 10.1177/ 0146167298243007.

Henry, J. D., & Crawford, J. R. (2005). The 21-item version of the Depression Anxiety Stress Scales (DASS–21): Normative data and psychometric evaluation in a large non-clinical sample. *British Journal of Clinical Psychology*, *44*, 227–239.

Fairburn, C. G. (1997). *Interpersonal psychotherapy for bulimia nervosa.* In D. M. Garner & P. E. Garfinkel (Eds.), Handbook of treatment for eating disorders (2 ed., pp. 278–294). New York, NY: Guilford.

Fairburn, C. G., & Beglin, S. J. (1994). Assessment of eating disorders: Interview or self-report questionnaire? *International Journal of Eating Disorders, 16*, 363–370. doi: 10.1002/1098-108X.

Fairburn, C. G., & Beglin, S. J. (2008). Eating disorder examination questionnaire (EDE-Q 6.0). In C. G. Fairburn (Ed.). *Cognitive behavior therapy and eating disorders* (pp. 309–313). New York: Guilford Press.

Fairburn, C. G., & Cooper, Z. (1993). The eating disorder examination. In C. G. Fairburn, & G. T. Wilson (Eds.). *Binge eating: Nature assessment and treatment* (12th ed.) (pp. 317–360). New York, NY: Guilford Press.

Fairburn, C. G., Cooper, Z., & O'Connor, M. (2008). Eating disorder examination (Edition 16.0D). In Fairburn C.G. *Cognitive behavior therapy and eating disorders.* New York, NY: Guilford Press.

Fairburn, C. G., Cooper, Z., & Shafran, R. (2003). Cognitive Behaviour therapy for eating disorder: A "transdiagnostic" theory and treatment. *Behaviour Research and Therapy, 41,* 509-528. doi:10.1016/S0005-7967(02)00088-8.

Fairburn, C. G., Cooper, Z., Bohn, K., O'Connor, M. E., Doll, H. A., & Palmer, R. L. (2007). The severity and status of eating disorder NOS: Implications for DSM-V. *Behaviour Research and Therapy, 45,* 1705–1715. doi: 10.1016/j.brat.2007.01.010.

Garson, G. D. (Ed.). (2013). *Hierarchical linear modeling: Guide and applications.* Thousand Oaks, CA: SAGE Publications Inc.

Grilo, C., Schiffman, S., & Carter-Campbell, J. T. (1994). Binge eating antecedents in normal-weight nonpurging females: Is there consistency? *International Journal of Eating Disorders, 16,* 239-249. doi: 10.1002/1098-108X.

Hadigan, C. M., Kissileff, H. R., & Walsh, B. T. (1989). Patterns of food selection during meals in women with bulimia. *American Journal of Clinical Nutrition, 50,* 759–766. doi:10.1016/j.appet.2014.12.098.

Haedt-Matt, A. A., & Keel, P. K. (2011). Revisiting the affect regulation model of binge eating: A meta-analysis of studies using ecological momentary assessment. *Psychological Bulletin, 137,* 660-681. doi: 10.1037/a0023660.

Hawkins, R. C., & Clement, P. F. (1984). Binge eating: Measurement problems and a conceptual model. In R. C. Hawkins, W. J. Fremouw & P. F. Clement (Eds.), *The binge purge syndrome: Diagnosis, treatment, and research* (pp. 229-251). New York, NY: Springer

Hayes, A. F. (2009). Beyond Baron and Kenny: Statistical mediation analysis in the new millennium. *Communication Monographs, 76,* 408-420. doi: 10.1080/03637750903310360.

Hildebrandt, T., & Latner, J. (2006). Effect of self-monitoring on binge eating: Treatment response or 'binge drift'? *European Eating Disorders Review, 14,* 17-22. doi:10.1002/erv.667.

IBM Corp. Released 2011. IBM SPSS Statistics for Windows, Version 20.0. Armonk, NY: IBM Corp.

Jenkins, P. E., Conley, C. S., Hoste, R. R., Meyer, C., & Blissett, J. M. (2012). Perception of control during episodes of eating: Relationships with quality of life and eating psychopathology. *International Journal of Eating Disorders, 45,* 115–119. doi: 10.1002/eat.20913.

Johnson, C., & Larson, R. (1982). Bulimia: An analysis of moods and behavior. *Psychosomatic Medicine, 44,* 341–351. doi: 10.1097/00006842-198209000-00003.

Karlsson, J., Persson, L-O., Sjöström, L., & Sullivan, M. (2000). Psychometric properties and factor structure of the three-factor eating questionnaire (TFEQ) in obese men and women. Results from the Swedish Obese Subjects (SOS) study. *International Journal of Obesity and Related Metabolic Disorders, 24,* 1715-1725. doi:10.1038/sj.ijo.0801442.

Kaye, W. H., Weltzin, T. E., McKee, M., McConaha, C., Hansen, D., & Hsu, L. K. G. (1992). Laboratory assessment of feeding behavior in bulimia nervosa and healthy women: Methods for developing a human-feeding laboratory. *American Journal of Clinical Nutrition, 55,* 372–380.

Keel, P. K., Crow, S., Davis, T. L., & Mitchell, J. E. (2002). Assessment of eating disorders: comparison of interview and questionnaire data from a long-term follow-up study of bulimia nervosa. *Journal of Psychosomatic Research, 53,* 1043-1047. doi:10.1016/S0022-3999(02)00491-9.

Keel, P. K., Haedt, A., & Edler, A. (2005). Purging disorder: An ominous variant of bulimia nervosa? *International Journal of Eating Disorders, 38,* 191-199. doi:10.1002/eat.20179.

Keel, P. K., Mayer, S. A., & Harnden-Fischer, J. H. (2001). Importance of size in defining binge eating episodes in bulimia nervosa. *International Journal of Eating Disorders, 29,* 294-301. doi:10.1002/eat.1021.

Keel, P. K., Wolfe, B. E., Gravener, J. A., & Jimerson, D. C. (2008). Co-morbidity and disorder-related distress and impairment in purging disorder. *Psychological Medicine, 38*, 1435-1442. doi: 10.1017/S0033291707001390.

Kelly-Weeder, S., Jennings, K. M., & Wolfe, B. E. (2012). Gender differences in binge eating and behavioral correlates among college students. *Eating and Weight Disorders, 17,* 200-2002. doi:10.1007/BF03325348.

Kerzhnerman, I., & Lowe, M. R. (2002). Correlates of subjective and objective binge eating in binge-purge syndromes. *International Journal of Eating Disorders, 31,* 220-228. doi:10.1002/eat.10026.

Landis, J. R., & Koch, G. G. (1977). The measurement of observer agreement for categorical data. *Biometrics*, *33*, 159-174. doi: 10.1177/096228029200100204.

Latner, J., Hildebrandt, T., Rosewall, J. K., Chisholm, A. M., & Hayashi, K. (2007). Loss of control reflects eating disturbances and general psychopathology. *Behavior Research and Therapy, 45,* 2203–2211. doi:10.1016/j.brat.2006.12.002.

Lewinsohn, P. M., Seeley, J. R., Moerk, K. C., & Striegel-Moore, R. H. (2002). Gender differences in eating disorder symptoms in young adults. *International Journal of Eating Disorders, 32,* 426-440. doi:10.1002/eat.10103.

Lovibond, S. H. & Lovibond, P. F. (1995). *Manual for the Depression Anxiety Stress Scales*. *(2nd. Ed.)* Sydney: Psychology Foundation.

Luce, K. H., & Crowther, J. H. (1999). The reliability of the eating disorder examination-self-report questionnaire version (EDE-Q). *International Journal of Eating Disorders, 25,* 349–351. doi:10.1002/(SICI)1098-108X(199904)25:3<349::AID-EAT15>3.0.CO;2-M.

Luce, K. H., Crowther, J. H., & Pole, M. (2008). Eating disorder examination questionnaire (EDE-Q): Norms for undergraduate women. *International Journal of Eating Disorders, 41,* 273-276. doi: 10.1002/erv.2324.

Mahmoud, J. S. R., Staten, R., Hall, L. A., & Lennie, T. A. (2012). The Relationship among young adult college students' depression, anxiety, stress, demographics, life satisfaction, and coping styles. *Issues in Mental Health Nursing*, 33, 149–156. doi: 10.3109/01612840.2011.632708.

Marlatt, G. A., & George, W. H. (1984). Relapse prevention: Introduction and overview of the model. *Addiction*, 79, 261–273. doi: 10.1111/j.1360-0443.1984.tb00274.x.

Mitchell, J., Crow, S., Peterson, C.B., Wonderlich, S., & Crosby, R.D. (1998). Feeding laboratory studies in patients with eating disorders: A review. *International Journal of Eating Disorders, 24,* 115–124. doi:10.1002/(SICI)1098-108X(199809)24:2<115::AID-EAT1>3.0.CO;2-H.

Mitchell, J. E., Karr, T. M., Peat, C., Wonderlich, S., Crosby, R. D., Engel, S., & Simonich, H. (2012). A fine-grained analysis of eating behavior in women with bulimia nervosa. *International Journal of Eating Disorders, 45,* 400-406. doi:10.1002/eat.20961.

MacDonald, D. E., McFarlane, T. L., & Olmstead, M. P. (2014). "Diagnostic shift" from eating disorder not otherwise specified to bulimia nervosa using DSM-5 criteria: A clinical

comparison with DSM-IV bulimia. *Eating Behaviors, 15,* 60-62. doi: 10.1016/j.eatbeh.2013.10.018.

Mochado, P. P., Goncalver, S., & Hoek, H. W. (2013). DSM-5 reduces the proportion of EDNOS cases: Evidence from community sample. *International Journal of Eating Disorders, 46,* 60-65. doi:10.1002/eat.22040.

Mond, J. M. (2013). Classification of bulimic-type eating disorders: From DSM-IV to DSM-5. *Journal of Eating Disorders, 1,* 33. doi:10.1186/2050-2974-1-33.

Mond, J. M., & Hay, P. J. (2010). Use of extreme weight control behaviors in the absence of binge eating with and without subjective bulimic episodes: A community-based study. *International Journal of Eating Disorders, 43,* 35–41. doi:10.1002/eat.20667.

Mond, J. M., Hay, P. J., Rodgers, B., & Owen, C. (2007). Self-report versus interview assessment of purging in a community sample of women. *European Eating Disorders Review, 15,* 403-409. doi:10.1002/erv.792.

Mond, J., Hay, P., Rodgers, B., Owen, C., & Beumont, P. J. V. (2004). Validity of the eating disorder examination questionnaire (EDE-Q) in screening for eating disorders in community samples. *Behaviour Research and Therapy, 42,* 551–567. doi:10.1016/S0005-7967(03)00161-X.

Mond, J. M., Latner, J. D., Hay, P. H., Own, C., & Rogers, B. (2010). Objective and subjective bulimic episodes in the classification of bulimic-type eating disorders: Another nail in the coffin of a problematic distinction. *Behavior Research and Therapy, 48,* 661-669. doi:10.1016/j.brat.2010.03.020.

Murphy, R., Straebler, S., Cooper, Z., & Fairburn, C. G. (2010). Cognitive behavioral therapy for eating disorders. *Psychiatric Clinics of North America, 33,* 611-627. doi:10.1016/j.psc.2010.04.004.

Myers, R. H. (1990). *Classical and modern regression application* (2nd ed.). CA: Duxbury press. CA.

Niego, S. H., Pratt, E. M., & Agras, W. S. (1997). Subjective or objective binge: Is the distinction valid? *International Journal of Eating Disorders, 22,* 291-298. doi:10.1002/(SICI)1098-108X(199711)22:3<291::AID-EAT8>3.0.CO;2-I.

Palmer, R. (2004). Bulimia nervosa: 25 years on. *The British Journal of Psychiatry, 185,* 447-448. doi:10.1192/bjp.185.6.447.

Polivy, J., & Herman, C. P. (1985). Dieting and binging: A causal analysis. *American Psychologist, 40,* 193–201. doi:10.1037/0003-066X.40.2.193.

Polivy, J., & Herman, C.P. (1993). Etiology of binge eating: Psychological mechanisms. In C. Fairburn (Ed.), *Etiology of binge eating: Psychological mechanisms* (pp. 173-205). London: Guilford Press.

Preacher, K. J., & Hayes, A. F. (2004). SPSS and SAS procedures for estimating indirect effects in simple mediation models. *Behavior Research Methods, Instruments, & Computers*, *36*, 717-731. doi:10.3758/BF03206553.

Preacher, K. J., & Hayes, A. F. (2008). Asymptotic and resampling strategies for assessing and comparing indirect effects in multiple mediator models. *Behavior Research Methods, 40,* 879-891. doi: 10.3758/BRM.40.3.879.

Reas, D. L., Gilo, C. M., & Masheb, R. M. (2006). Reliability of the eating disorder examination-questionnaire in patients with binge eating disorder. *Behavior Research and Therapy, 44,* 43-51. doi:10.1016/j.brat.2005.01.004.

Rockert, W., Kaplan, A. S., & Olmsted, M. P. (2007). Eating disorder not otherwise specified: The view from a tertiary care treatment center. *International Journal of Eating Disorders, 40,* 99–103. doi:10.1002/eat.20482.

Rosen, J. C., Leitenber, H., Fisher, C., & Khazam, C. (1986). Binge-eating episodes in bulimia nervosa: The amount and type of food consumed. *International Journal of Eating Disorders, 5,* 255-267. doi:10.1002/1098-108X(198602)5:2<255::AID-EAT2260050206>3.0.CO;2-D.

Rossiter, E. M., & Agras, W. S. (1990). An empirical test of the DSM-III definition of binge. *International Journal of Eating Disorders, 9,* 513-518. doi:10.1002/1098-108X(199009)9:5<513::AID-EAT2260090506>3.0.CO;2-T.

Rossiter, E.M., Agras, W.S., Telch, C.F., & Bruce, B. (1992). The eating patterns of non-purging bulimic subjects. *International Journal of Eating Disorders, 11,* 111-120. doi:10.1002/1098-108X(199203)11:2<111::AID-EAT2260110203>3.0.CO;2-J.

Russell, G. (1979). Bulimia nervosa: An ominous variant of anorexia nervosa. *Psychological Medicine*, 9, 429-448. doi:10.1080/21662630.2013.742319.

Rydin-Gray, S. (2007). Binge eating antecedents among female college students: An ecological momentary assessment study. *Dissertation Abstracts International: Section B: The Sciences and Engineering, 67,* 6076.

Safer, D. L., Agras, W. S., Lowe, M. R., & Bryson, S. (2004). Comparing two measures of eating restraint in bulimic women treated with cognitive-behavioral therapy. *International Journal of Eating Disorders, 36,* 83-88. doi:10.1002/eat.20008.

Shafran, R., Teachman, B. A., Kerry, S., & Rachman, S. (1999). A cognitive distortion associated with eating disorders: Thought-shape fusion. *British Journal of Clinical Psychology, 38,* 167–179. doi:10.1348/014466599162728.

Sinclair, S. J., Siefert, C. J., Slavin-Mulford, J. M., Stein, M. B., Renna, M., & Blais, M. A. (2012). Psychometric evaluation and normative data for the depression, anxiety, and stress scales-21 (DASS-21) in a nonclinical sample of U.S. adults. *Evaluation & the Health Professions, 35,* 259-279. doi:10.1177/0163278711424282.

Smith, D. E., Marcus, M. D., Kaye, W. (1992). Cognitive-behavioral treatment of obese binge eaters. *International Journal of Eating Disorders, 12,* 257–262. doi: 10.1037/a0025049.

Spoor, S. T. P., Stice, E., Burton, E., & Bohon, C. (2007). Relations of bulimic symptom frequency and intensity to psychosocial impairment and health care utilization: Results from a community-recruited sample. *International Journal of Eating Disorders, 40,* 505-514. doi:10.1002/eat.20410.

Stein, R. I., Kenardy, J., Wiseman, C. V., Dounchis, J. Z., Arnow, B. A., & Wilfley, D. E. (2007). What's driving the binge in binge eating disorder?: A prospective examination of precursors and consequences. *International Journal of Eating Disorders, 40,* 195-203. doi:10.1002/eat.20352.

Stice, E., Fisher, M., & Lowe, M. R. (2004). Are dietary restraint scales valid measures of acute dietary restriction? Unobtrusive observational data suggest not. *Psychological Assessment, 16,* 51-59. doi:10.1037/1040-3590.16.1.51.

Stickney, M. I., & Miltenberger, R. G. (1999). Evaluating direct and indirect measures for the functional assessment of binge eating. *International Journal of Eating Disorders, 26,* 195–204. doi:10.1002/(SICI)1098-108X(199909)26:2<195::AID-EAT9>3.0.CO;2-2.

Striegel-Moore, R. H., Wilfley, D. E., Pike, K. M., Dohm, F. A., & Fairburn, C. G. (2000). Recurrent binge eating in black American women. *Archives of Family Medicine, 9,* 83–87. doi:10.1016/S1471-0153(04)00050-9.

Stunkard, A. J., & Messick, S. (1985). The three-factor eating questionnaire to measure dietary restraint, disinhibition and hunger. *Journal of Psychosomatic Research, 29*, 71-83. doi: 10.1016/0022-3999(85)90010-8.

Sullivan, G. M., & Feinn, R. (2012). Using effect size—or why the *p* value is not enough. *The Journal of Graduate Medical Education, 4,* 279-282. doi: 10.4300/JGME-D-12-00156.1

Tuschen-Caffier, B., & Vogele, C. (1999). Psychological and physiological reactivity to stress: An experimental study on bulimic patients, restrained eaters and controls. *Psychotherapy and Psychosomatics, 68,* 333–340. doi: 10.1159/000012352.

Van der Merwe, M. T. (2007). Psychological correlates of obesity in women. *International Journal of Obesity, 31,* S14-S18. doi:10.1038/sj.ijo.0803731.

Vannucci, A., Theim, K. R., Kass, A. E., Trockel, M., Genkin, B., Rizk, M., … Taylow, C. B. (2013). What constitutes clinically significant binge eating? *International Journal of Eating Disorders, 46,* 226-232. doi: 10.1002/eat.22115.

Walsh, B.T. & Garner, D.M. (1997). Diagnostic issues. In D.M. Garner & P.E. Garfinkel (Eds.), *Handbook of Treatment for Eating Disorders* (pp. 25-33). New York, NY: The Guilford Press.

Watson, H. J., Fursland, A., Bulik, C. M., & Nathan, P. (2013). Subjective binge eating with compensatory behaviors: A variant presentation of bulimia nervosa. *International Journal of Eating Disorders, 46,* 119-126. doi: 10.1002/eat.22052.

West, S. G., Finch, J. F., & Curran, P. J. (1995). Structural equation models with nonnormal variables: Problems and remedies. In R. H. Hoyle (Ed.), *Structural equation modeling: Concepts, issues, and applications* (pp. 56 – 75). Thousand Oaks, CA: Sage.

Wilfley, D.E., Welch, R.R., Stein, R.I., Spurrell, E.B., Cohen, L.R., & Saelens, B.E., … Matt, G. E. (2002). Randomized comparison of group cognitive–behavioral therapy and group interpersonal psychotherapy for the treatment of overweight individuals with binge eating disorder. *Archive of General Psychiatry, 8,* 713–721. doi:10.1001/archpsyc.59.8.713.

Wilfley, D. E., Wilson, G. T., & Agras, W. S. (2003). The clinical significance of binge eating disorder. *International Journal of Eating Disorders, 34*, 96–106. doi: 10.1002/eat.10209.

Wolfe, B. E., Wood Baker, C., Smith, A. T., & Kelly-Weeder, S. (2009). Validity and utility of the current definition of binge eating. *International Journal of Eating Disorders, 42,* 674-686. doi:10.1002/eat.20728.

Wonderlich, S. A., Gordon, K. H., Mitchell, J. E., Crosby, R.D., & Engel, S. G. (2009). Validity and clinical utility of binge eating disorder. *International Journal of Eating Disorders*, 42, 687–705. doi:10.1002/eat.20719.

Yurica, C. (2002). *Inventory of cognitive distortions: Validation of a psychometric instrument for the measurement of cognitive distortions.* Unpublished doctoral dissertation. Philadelphia College of Osteopathic Medicine.

Yurica, C., & DiTomasso, R. A. (2001). *Inventory of Cognitive Distortions.* (Available from Robert DiTomasso, Ph.D., ABPP, 4170 City Avenue, Philadelphia, PA 19131).