Published in final edited form as:
Psychol Bull. 2011 July ; 137(4): 660–681. doi:10.1037/a0023660.

Revisiting the Affect Regulation Model of Binge Eating: A Meta-Analysis of Studies using Ecological Momentary Assessment

Alissa A. Haedt-Matt and
Department of Psychology, The University of Iowa

Pamela K. Keel
Department of Psychology, Florida State University

Abstract

The affect regulation model of binge eating, which posits that patients binge eat to reduce negative affect (NA), has received support from cross-sectional and laboratory-based studies. Ecological momentary assessment (EMA) involves momentary ratings and repeated assessments over time and is ideally suited to identify temporal antecedents and consequences of binge eating. This meta-analytic review includes EMA studies of affect and binge eating. Electronic database and manual searches produced 36 EMA studies with N = 968 participants (89% Caucasian women). Meta-analyses examined changes in affect before and after binge eating using within-subjects standardized mean gain effect sizes (ES). Results supported greater NA preceding binge eating relative to average affect (ES = .63) and affect before regular eating (ES = .68). However, NA increased further following binge episodes (ES = .50). Preliminary findings suggested that NA decreased following purging in Bulimia Nervosa (ES = −.46). Moderators included diagnosis (with significantly greater elevations of NA prior to bingeing in Binge Eating Disorder compared to Bulimia Nervosa) and binge definition (with significantly smaller elevations of NA before binge versus regular eating episodes for the DSM definition compared to lay definitions of binge eating). Overall, results fail to support the affect regulation model of binge eating and challenge reductions in NA as a maintenance factor for binge eating. However, limitations of this literature include unidimensional analyses of NA and inadequate examination of affect during binge eating as binge eating may regulate only specific facets of affect or may reduce NA only during the episode.

Keywords

binge eating; ecological momentary assessment; negative affect; bulimia nervosa

Binge eating, defined as the consumption of unusually large amounts of food coupled with a sense of loss of control over eating, is an essential feature of Bulimia Nervosa (BN) and Binge Eating Disorder (BED) and is included in a subtype of Anorexia Nervosa (AN) (American Psychiatric Association, 2000). Thus, binge eating cuts across all eating disorder syndromes currently recognized by the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-IV-TR). Further, binge eating is associated with significant psychological

Correspondence concerning this article should be addressed to Alissa A. Haedt-Matt, E11 Seashore Hall, Iowa City, IA 52242. alissa.haedt.matt@gmail.com.

comorbidity (Bulik, Sullivan, & Kendler, 2002; Striegel-Moore, Wilfley, Pike, Dohm, & Fairburn, 2000; Telch & Agras, 1994; Yanovski, Nelson, Dubbert, & Spitzer, 1993) and may increase risk for weight gain, obesity, and related medical consequences, such as heart disease, high blood pressure, and type 2 diabetes (Bulik et al., 2002; Hasler et al., 2004; Telch, Agras, & Rossiter, 1988; Yanovski et al., 1993). The clinical significance of binge eating has led to both theoretical work and empirical research to understand psychological factors that maintain this behavior. Cognitive-behavioral models typically focus on the influence of immediate environmental, cognitive, and emotional antecedents that trigger binge episodes and the consequences that follow these episodes. Among various models, affect regulation has gained considerable influence for understanding the function of binge eating (Aldao, Nolen-Hoeksema, & Schweizer, 2010; Stice, 2001; Wedig & Nock, 2010), and this model has contributed to a focus on both affective antecedents and consequences in the cognitive-behavioral treatment (CBT) regarded as the first choice treatment for BN (Fairburn, 2008; National Institute for Clinical Excellence, 2004). Thus, the affect regulation model of binge eating is the focus of this review.

Affect Regulation Model of Binge Eating

Dysregulated affect is implicated in most psychological disorders included in the DSM-IV-TR (American Psychiatric Association, 2000), and a substantial body of literature has evaluated the function of maladaptive behaviors in regulating affect. Although regulation can include the increase, maintenance, or decrease of positive or negative emotions, affect regulation models of psychopathology typically propose that maladaptive behaviors function to decrease negative emotions (see Gross, 2007 for a comprehensive review). Affect regulation models have been supported for a variety of clinical presentations, including self-injury (Klonsky, 2007), alcohol use (Sher & Grekin, 2007), and avoidance in anxiety and depression (Campbell-Sills & Barlow, 2007). Similarly, one of the most commonly cited explanations for binge eating emphasizes the role of regulating emotional distress or negative affect (Polivy & Herman, 1993).

The affect regulation model proposes that increases in negative emotions trigger binge episodes and that binge eating functions to alleviate negative affect by using food for comfort and distraction (Hawkins & Clement, 1984). Binge eating in response to negative emotions becomes a conditioned response that is maintained through negative reinforcement. Two basic hypotheses have been tested in relation to the affect regulation model: 1) increases in negative affect represent a proximal antecedent to binge eating, and 2) binge eating is associated with an immediate decrease in negative affect.

Several studies have provided support for the first hypothesis. Between 69% and 100% of participants with BN and BED retrospectively reported negative mood as a trigger of binge eating when asked open-ended questions regarding why they engaged in the behavior (Abraham & Beumont, 1982; Arnow, Kenardy, & Agras, 1992; Bruce & Agras, 1992; Lynch, Everingham, Dubitzky, Hartman, & Kasser, 2000; Mitchell, Hatsukami, Eckert, & Pyle, 1985), and when asked to complete questionnaires of variables that may precipitate binge eating (Davis & Jamieson, 2005; Hsu, 1990; Kjelsås, Børsting, & Gudde, 2004; Mitchell et al., 1999; Pyle, Mitchell, & Eckert, 1981; Tachi, Murakami, Murotsu, & Washizuka, 2001; Vanderlinden et al., 2004). In addition, experimental studies have tested this hypothesis utilizing negative mood inductions. In three separate studies of BED (Agras & Telch, 1998; Chua, Touyz, & Hill, 2004; Telch & Agras, 1996), participants were randomly assigned to a negative or neutral mood induction procedure followed by a measure of food consumption. Participants in the negative mood condition consumed significantly more food during the subsequent taste test (Chua et al., 2004) and experienced more binge episodes (40%) compared to those in the neutral condition (17%) (Agras & Telch, 1998;

Telch & Agras, 1996). Thus, increases in negative mood caused increases in the occurrence of binge eating. Because there were no baseline differences in negative mood between those who did and did not binge eat, negative affect appeared to serve as a proximal trigger of binge eating (Agras & Telch, 1998).

Mixed empirical support has emerged for the second hypothesis, that binge eating reduces negative affect. Some retrospective studies indicated that 50% – 66% of BN participants reported a decrease in negative affect following binge eating (Abraham & Beumont, 1982; Hawkins & Clement, 1984; Hsu, 1990) while others found that 85% – 100% reported an increase in negative mood states following binge eating (Arnow et al., 1992; Mitchell et al., 1985; Mitchell et al., 1999; Pyle et al., 1981; Tachi et al., 2001). In one experimental study (Agras & Telch, 1998), BED participants in the negative mood induction condition reported a significant decrease in negative affect following food consumption. However, negative affect decreased for both participants who did and did *not* binge eat, with no association between binge eating and mood changes within the negative mood induction condition. Thus, binge eating did not appear to "cause" the reduction in negative affect. Instead, decreases in negative mood may reflect a consequence of eating in general or the passage of time. Finally, two studies of BN examined mood ratings during a binge-purge episode following admission to an inpatient unit and found that anxiety decreased following binge eating; however, depression increased (Hetherington, Altemus, Nelson, Bernat, & Gold, 1994; Kaye, Gwirtsman, George, Weiss, & Jimerson, 1986).

Although the affect regulation model has been examined in relation to binge eating in both BN and BED, there is limited research examining whether the affective antecedents and consequences of binge eating differ between BN and BED. These syndromes differ considerably in their behavioral responses to binge eating. Specifically, individuals with BN engage in inappropriate compensatory behaviors (e.g., purging through self-induced vomiting) following binge episodes to reduce the influence of binge eating on weight. Individuals with BED do not engage in inappropriate compensatory behaviors. In addition to differences in the behavioral consequences of binge eating, there may be differences in the emotional consequences of binge eating. For example, although both BN and BED were associated with negative affective consequences of binge eating, women with BED have been found to report less post-binge anxiety compared to women with BN in retrospective research (Mitchell et al., 1999). Thus, support for the affect regulation model of binge eating may differ between BN and BED.

Given that BN is associated with a combination of disordered eating behaviors (i.e., binge eating and purging), it is possible that changes in affect depend on the behavior examined. Binge eating may begin as an attempt to reduce negative affect, but excessive food intake increases concerns about weight gain and anxiety in BN. Increased negative affect following binge eating in BN may then be reduced by purging. Thus, a modification of the affect regulation model for BN proposes that purging, rather than binge eating, becomes the central means for regulating affect (Rosen & Leitenberg, 1982). Supporting this modification of the affect regulation model, BN participants have retrospectively reported decreases in negative affect following purging (Cooper et al., 1988; Rosen & Leitenberg, 1982).

Related theories of affect and binge eating

In addition to the affect regulation model of binge eating, other theoretical models have posited increases in negative affect as a trigger of binge episodes. Restraint theory (Herman & Polivy, 1980) proposes that cognitive control plays a more influential role than physiological hunger and satiation in regulating food intake among those who chronically diet (Ruderman, 1986). The experience of negative affect disrupts cognitive control in restrained eaters, reducing their ability and/or desire to maintain dietary control (Herman &

Polivy, 1984). Thus, restraint theory proposes that increased negative emotions serve as an affective disinhibitor to cognitive controls over eating, resulting in counter-regulation in the form of binge eating. However, this model does not suggest that binge episodes are maintained through reductions in negative affect.

A related model is escape theory (Heatherton & Baumeister, 1991). Escape theory proposes that binge episodes reduce negative affect by narrowing cognitive attention from higher-level abstract thinking (particularly with regard to personal failures to meet high standards) to the more immediate environment (Baumeister, 1990). Cognitive narrowing precludes meaningful thought and, thus, provides escape from aversive self-awareness (Heatherton & Baumeister, 1991). Similar to affect regulation and restraint, the escape model posits that increases in negative affect would precede binge eating episodes. In contrast to the restraint model of binge eating, escape theory proposes that reductions of negative affect occur *during* binge eating as a consequence of lower self-awareness. In contrast to the affect regulation model, escape theory proposes that emotional distress increases upon completion of a binge episode when self-awareness returns.

Finally, expectancy theory proposes that binge eating is maintained through an individual's beliefs about the effects of binge eating, which develop as a product of learning history (Hohlstein, Smith, & Atlas, 1998), similar to expectancy theories for alcohol use (Goldman, Brown, Christiansen, & Smith, 1991). Expectancy theory has been supported by studies that examined individual differences in cognitive expectancies and their association with binge eating. Bulimic participants reported significantly greater expectancies that eating reduces negative affect compared to psychiatric and normal controls, and the belief that eating reduces negative affect distinguished participants with bulimic symptoms (i.e., binge eating) from those with other disordered eating symptoms (Hohlstein et al., 1998; Simmons, Smith, & Hill, 2002). In addition, eating expectancies (i.e., beliefs that eating will reduce negative mood or will be rewarding) have been linked to the later development of bulimic symptoms as well as the maintenance of bulimic syndromes in prospective, longitudinal investigations (Bohon, Stice, & Burton, 2009; Hayaki, 2009; Smith, Simmons, Flory, Annus, & Hill, 2007). In contrast to the affect regulation model, the cognitive expectancies model posits that *expected* consequences will be more important than *actual* consequences of binge eating in maintaining this behavior. Taken together, restraint, escape, and expectancy theories all predict that increases in negative affect will lead to binge eating, but do not propose that binge eating episodes are maintained by post-binge reductions in negative affect.

Summary

The affect regulation model of binge eating provides clear, testable hypotheses regarding the trajectory of affect before and after binge eating. In addition, this approach to understanding binge episodes lends itself to intervention using CBT, which has demonstrated efficacy in the treatment of BN and BED (Wilson, Grilo, & Vitousek, 2007). Thus, retrospective studies, laboratory studies, and the effectiveness of CBT in ameliorating binge eating support the validity of the affect regulation model for binge eating. However, these sources of support are not without limitations, and these limitations call into question the adequacy with which the affect regulation model of binge eating has been empirically evaluated.

Limitations of Retrospective and Experimental Research

Retrospective research is necessary to provide initial clues regarding potential antecedents and consequences of behavior. However, memory limitations and cognitive biases are two major concerns in retrospective designs. Retrospective reports often inquire about events, thoughts, behaviors, or mood anywhere from the past few days to the past few years. Thus,

retrospective reports may be inaccurate due to an individual's inability to remember information over extended periods of time (Shiffman, Stone, & Hufford, 2008).

In addition to forgetting information due to the length of recall period, there is evidence of several memory biases (see Shiffman & Stone, 1998 for a full review). For example, recall is frequently influenced by participants' current mood; negative events are more easily recalled during negative moods (Teasdale & Fogarty, 1979). In addition, more recent or more salient events are often over-emphasized in retrospective recall of behavior or mood over a longer period of time (Redelmeier & Kahneman, 1996). A final cognitive bias worth noting involves reconstruction of past events. Individuals may provide explanations for behavior that make sense given what they know or believe to be true rather than based on their actual experiences (Ross, 1989). These cognitive biases may be especially problematic for the investigation of transient changes in affect in eating disorders. Specifically, within a research or treatment context where binge episodes are expected to regulate affect, patients may be more likely to "recall" that this was true.

Laboratory research mitigates most of these concerns if participants are assessed in real-time during an experiment. In addition, experimental research allows for causal attributions. However, there are concerns about the ecological validity of research conducted in an experimental setting. Laboratory environments are often very different from participants' natural environments. Thus, research findings may not generalize to or be representative of what happens outside the laboratory setting. This is particularly problematic for research on eating disorders when setting variables, such as the time of day or whether participants are alone or with other people, may influence whether or not individuals engage in binge eating. In response to these concerns, researchers have employed a research design called ecological momentary assessment.

Ecological Momentary Assessment

Ecological momentary assessment (EMA; Stone & Shiffman, 1994) examines the daily experiences, behavior, and psychological states of individuals in their natural environment. This method is very similar to experience sampling (Larson & Csikszentmihalyi, 1983) and the daily diary method (Bolger, Davis, & Rafaeli, 2003). In this review, we use the term "EMA" to describe all of these methods. EMA can be implemented in a variety of ways, but all EMA studies have certain features in common. First, assessment takes place in participants' natural environments as they go about their daily lives. In addition, participants complete ratings regarding their current state (e.g., current mood, current behavior) rather than reporting on mood or behaviors that occurred several days or weeks ago. Finally, EMA involves repeated assessments over time. The first two features limit concerns associated with ecological validity and retrospective recall, and the third feature allows examination of variability over time and temporal ordering of the variables in question. Thus, this methodology is ideally suited to test the affect regulation model of binge eating.

EMA methods

The kinds of questions that can be answered using EMA depend on the assessment protocol used to sample participants' daily experiences. Wheeler and Reis (1991) described three categories of EMA protocols: *interval-contingent, signal-contingent*, and *event-contingent* recordings. Interval-contingent methods require participants to complete self-report measures after a specified period of time, typically at the end of each day (e.g., daily diary methods). While end of day ratings may substantially decrease participant burden (compared to multiple ratings per day), lengthy intervals are still subject to the retrospective recall biases that EMA was designed to overcome (e.g., Hedges, Jandorf, & Stone, 1985). More frequent intervals can reduce the level of bias but may become predictable so that

participants change their behavior in anticipation of making ratings, which threatens the ecological validity of resulting data (Smyth et al., 2001).

Signal-contingent methods require participants to complete self-report measures in response to randomly timed signals usually through a watch timer, pager, or palmtop computer. This approach has the advantage of unpredictability as well as gaining a representative sampling of participants' experiences throughout the day (Wheeler & Reis, 1991). Both interval- and signal-contingent methods can be used to address questions regarding daily fluctuations in factors such as mood (Wheeler & Reis, 1991). In addition, these methods are desirable for the assessment of antecedents because ratings made in response to time intervals or random signals are not tied to the behavior itself. Thus, cognitive biases to reconstruct past events are not associated with these ratings because individuals respond before the behavior has occurred (Shiffman et al., 2008). However, these methods are limited in eating disorders research because of their restricted ability to detect infrequent behaviors, such as binge eating or purging. Even participants who meet full DSM-IV criteria for BN or BED are required to binge, on average, a minimum of only twice per week. Thus, EMA that relies solely on interval- or signal-contingent methods may miss important consequences of disordered eating behaviors if those behaviors occur in between rating cycles.

Event-contingent methods require participants to complete self-report measures in response to a particular event or behavior. The advantage of this approach is that it is tied to events, which greatly reduces the likelihood of missing a behavior of interest (Wheeler & Reis, 1991). In addition, this approach is valuable for assessing the immediate consequences of behavior. However, event-contingent methods are not well-suited for identifying antecedents of behaviors that are not planned by the participant. Given that binge eating is associated with a loss of control over eating, participants may not be aware that they are going to engage in the behavior until it has already begun. Because each method has advantages and disadvantages, experts recommend a combination of time- and event-based approaches in eating disorders research (see Smyth et al., 2001). However, this solution can result in increased participant burden and decreased protocol compliance (Wheeler & Reis, 1991).

The Present Meta-Analysis

Importantly, there are a growing number of EMA studies on antecedents and consequences of binge eating; however, a comprehensive review of this literature has not been published. In addition, while negative affect is consistently reported as an antecedent of binge eating across studies, some studies have reported improvements in negative mood following a binge episode whereas others have reported exacerbations of negative mood (see Wolfe, Baker, Smith, & Kelly-Weeder, 2009 for a review). These conflicting results have made it difficult to draw conclusions regarding changes in negative affect as a consequence of binge eating. A meta-analysis can overcome this limitation by aggregating results across studies to determine an overall association between binge eating and negative affect. Further, although CBT is considered the treatment of choice for BN and BED, only 30% – 50% of individuals who complete treatment achieve remission from binge eating (Wilson et al., 2007). A review of studies examining the affective antecedents and consequences of binge eating is needed to improve our understanding of the psychological processes maintaining this pernicious symptom in order to enhance evidence-based treatment for eating disorders. Thus, our review seems both timely and clinically important.

This meta-analysis reviewed EMA studies of affect and binge eating. Hypotheses were based on predictions made by the affect regulation model of binge eating. Specifically, we hypothesized that 1) negative affect would be greater prior to binge eating compared to other times during the day, representing a proximal antecedent to binge eating, and 2) negative

affect would be lower after binge eating compared to before binge eating. Given that binge eating episodes in BN are often followed by purging, which may impact the affective response to binge eating, we also examined proximal changes in affect related to purging in BN and hypothesized that purging would be associated with a decrease in negative affect.

Potential Moderators

In addition to providing an index of the overall association between binge eating and affect, the meta-analyses allowed for a systematic examination of variables that differed across studies and may moderate the strength of associations between binge eating and affect. Thus, the present study sought to identify such moderators. Several potential moderators were identified *a priori*, including sample and methodological characteristics.

Sample characteristics

The moderating influence of diagnosis was examined because binge eating has been associated with distinct cognitive, behavioral, and emotional consequences in BN compared to BED in studies utilizing retrospective reports (Mitchell et al., 1999). Treatment-seeking was included as a moderator because previous research has suggested that treatment-seeking is associated with greater eating pathology (Keel et al., 2002), and because greater eating pathology has been associated with higher trait negative affectivity (Cassin & von Ranson, 2005). Thus, treatment-seeking samples with high trait negative affectivity may experience high levels of negative affect at all times regardless of whether or not they are binge eating. This could produce a ceiling effect which would reduce distinctions between negative affect pre- and post-binge eating with other time frames.

Methodological characteristics

Methodological features that would impact the reliability of assessments were examined as moderators because reduced reliability may result in underestimated effect sizes due to insensitivity to change over time (Hunter & Schmidt, 2004). Use of single- versus multi-item measures for affect assessment was considered a potential moderator because single-item assessments are less reliable than multiple-item measures (Wanous & Reichers, 1996). Use of structured interviews to diagnose participants was included because of assertions that structured interviews are more valid and reliable (Wilson, 1993) and thus may be associated with larger effect sizes. Whether or not binge eating was explicitly defined for participants using the DSM definition was included due to significant differences between lay definitions and DSM definitions of binge eating (Beglin & Fairburn, 1992; Telch, Pratt, & Niego, 1998). Lay definitions have tended to emphasize loss of control over eating and eating in response to emotions rather than ingestion of objectively large amounts of food coupled with a loss of control. As such, lay definitions may be associated with larger effect sizes when examining associations between negative affect and binge eating. Length of EMA was included because assessments of multiple instances of binge eating over an extended period of time are less likely to be influenced by chance events and thus result in more reliable effect sizes. Type of EMA method was included because of distinct advantages and disadvantages associated with each approach described above. For example, interval-contingent methods in which participants record information for the previous day rely on greater retrospective reporting than signal-contingent methods. Thus, use of interval-contingent ratings may be associated with larger effect sizes due to the potential for retrospective recall bias. Finally, frequency of daily assessments was included because greater frequency may be better able to capture temporary changes in negative affect that precede and follow binge episodes and thus may be associated with more reliable estimates and larger effect sizes. Alternatively, greater frequency of assessment increases participant burden and may decrease protocol compliance. If participants are more likely to skip

assessments when negative affect is high (i.e., there is an association between protocol compliance and negative affect that is more evident when participant burden is high), greater frequency of assessments could lead to smaller effect sizes.1 Given these possibilities and that these are the first moderator analyses of associations between affect and binge eating, all analyses used two-tailed significance testing.

Method

Article Search

Several methods were used to identify research articles for this review. First, we conducted electronic searches of published articles and dissertation abstracts using PsycInfo and PubMed databases with the following search terms: *ecological momentary assessment*, *experience sampling*, *event sampling*, *daily diary*, *eating disorder* (with explode option), *anorexia nervosa* (with explode option), *bulimia* (with explode option), *binge eating* (with explode option), and *purging* (with explode option) for articles published as of June, 2009. Second, we conducted manual searches of the reference sections of all articles obtained through the electronic search, including review articles, to identify additional articles that were not produced by electronic search. Both authors performed electronic and manual searches to increase the likelihood that all relevant research articles were obtained. Finally, unpublished and in press articles were requested from corresponding authors of previously identified articles and from emailing the listserv of the Academy for Eating Disorders (www.aedweb.org).

A total of 82 studies were initially identified through this process and examined for inclusion in the review. Inclusion criteria included: 1) use of EMA methods (i.e., use of momentary ratings and inclusion of multiple daily assessments to allow within-day analyses and temporal ordering of affect and binge eating), 2) an assessment of binge eating, and 3) an assessment of negative affect. Both authors independently evaluated each article for inclusion in the meta-analyses according to these criteria and demonstrated high agreement (kappa = 0.91) about which studies to include. Discrepancies were resolved by consensus. Based on this process, 36 studies were retained for inclusion in the meta-analyses. Excluded studies included three theoretical or review papers (McManus & Waller, 1995; Norton, Wonderlich, Myers, Mitchell, & Crosby, 2003; Smyth et al., 2001) and five studies using retrospective designs (Arnow et al., 1992; Cooper et al., 1988; Kjelsås et al., 2004; Lynch et al., 2000; Steinberg, Tobin, & Johnson, 1990). In addition, nine studies were excluded because they did not examine binge eating, which is defined by the consumption of an unusually large amount of food and a sense of loss of control over eating (American Psychiatric Association, 2000). Excluded studies assessed loss of control eating without evaluating amount of food consumed (Hilbert, Rief, Tuschen-Caffier, de Zwaan, & Czaja, 2009), night eating (Boseck et al., 2007; Greeno, Wing, & Marcus, 1995; Lundgren, Allison, O'Reardon, & Stunkard, 2008), and general food consumption without evaluating a loss of control over eating (Chua et al., 2004; O'Connor, Jones, Conner, McMillan, & Ferguson, 2008; Patel & Schlundt, 2001; Timmerman & Gregg, 2003; Wild et al., 2007). Finally, 29 studies did not assess affect as a within-day antecedent or consequence of binge eating (e.g., Engelberg, Gauvin, & Steiger, 2005; Lingswiler, Crowther, & Stephens, 1987; Waters, Hill, & Waller, 2001; Wolff, Crosby, Roberts, & Wittrock, 2000).

[1]We had initially included duration of illness and compliance as *a priori* moderators because the initiation of a behavior may not contribute to its maintenance over an extended period of time and higher proportions of missing data may reduce the representativeness of results based on recorded data. However, there were substantial missing data for both variables. In addition, there was great variability in how compliance was reported across studies related to differences in methodology and authors' definitions. Thus, duration of illness and compliance were not included in the final analyses.

Meta-Analyses

Meta-analyses were conducted separately for affective antecedents and affective consequences of binge eating. The available literature on binge eating antecedents was analyzed according to two main approaches: 1) comparisons of pre-binge eating affect to average affect and 2) comparisons of pre-binge eating affect to pre-regular eating affect. Comparisons of pre-binge eating affect to average affect reflected whether affect before a binge episode differed from individuals' general affect when they are not bingeing. Of note, this comparison cannot disentangle the affective antecedents of binge eating from affective antecedents of eating in general. Eating disordered individuals have reported elevated levels of general eating concerns even for episodes that were not characterized by a loss of control or consumption of a large amount of food (Cooper, Cooper, & Fairburn, 1989; Wilfley, Schwartz, Spurrell, & Fairburn, 2000). Thus, we also compared affect before *binge* eating to affect before *regular* eating episodes to examine the specificity of negative affect as an antecedent of binge eating. Studies that reported antecedents for regular eating typically used an event-contingent design in which participants were instructed to complete assessments prior to any eating episode (including meals, snacks, and binge eating) and then identified whether or not the eating constituted a binge episode. Affective consequences of binge eating were assessed by comparing affect after a binge episode to affect before the episode. Studies that reported consequences of binge eating typically asked participants to make ratings of affect following either a binge or any food consumption, indicating whether or not consumption constituted a binge.

Calculation of individual study effect sizes—Standardized mean gains were used as the index of effect size (ES) because ES in our analyses compared two time points within a single sample. Thus, these within-subjects ES should not be compared with between-subjects mean difference ES. ES were calculated according to the following formula (Lipsey & Wilson, 2001):

$$ES=\frac{X_{Time\ 2}-X_{Time\ 1}}{s_p}$$

Standardized mean gains for antecedent comparisons were calculated as mean affect prior to binge eating ($X_{Time\ 2}$) minus mean affect for average ratings or minus mean affect prior to regular eating episodes ($X_{Time\ 1}$) divided by the pooled standard deviation (s_p; Dunlap, Cortina, Vaslow, & Burke, 1996). Thus, positive ES in antecedent analyses of negative affect indicated that negative affect was higher prior to binge eating compared to average affect or affect prior to regular eating episodes. For affective consequences of binge eating, standardized mean gains were calculated as mean affect after binge eating minus mean affect prior to binge eating. Thus, negative ES for the analysis of negative affect as a consequence indicated that negative affect was lower following binge episodes compared to before the binge episode, consistent with a model in which binge eating regulates affect. Conversely, positive ES for consequence analyses indicated that negative affect was higher following binge episodes.

When means and standard deviations were not reported, standardized mean gains were calculated from paired *t*-test statistics, correlation values (*r*), and, in one case (Engelberg, Steiger, Gauvin, & Wonderlich, 2007), odds ratios. If insufficient data were provided to calculate ES from published data (initially $n = 16$ studies), corresponding authors were contacted a minimum of two times to obtain additional information. Of those contacted, 4 were able to provide additional data, 7 no longer had access to the data, and 5 did not respond. Notably, authors who did not have access to additional data had published their

studies at least eight years ago (mean length since publication = 15 years), and authors who did not respond had published their studies at least 12 years ago (mean length since publication = 16 years). When data could not be obtained to include study results in the meta-analyses (n = 12 studies), study findings were described qualitatively for comparison with meta-analytic results to evaluate for possible bias.

Standard meta-analyses require all studies for each analysis to be independent (i.e., only one ES per comparison per study). When multiple facets of negative affect were reported in a single study, a composite ES was obtained using meta-analytic techniques described by Rosenthal and Rubin (1986) for combining non-independent ES. For example, ES for depression, anxiety, and hostility in Powell and Thelen (1996) were combined to create a composite ES for negative affect.

Calculation of mean effect sizes—Individual study ES were weighted by their inverse variance according to the following formula for repeated measures (Lipsey & Wilson, 2001):

$$\text{inverse variance weight} = \frac{2n}{4(1 - r) + ES^2}$$

This formula requires the correlation, r, between affect measured at the two time points used in the calculation of ES, which was not reported in any study. An estimate of r was therefore obtained by averaging available data, including published test-retest reliabilities of the Positive and Negative Affect Schedule using momentary rating instructions (Watson, Clark, & Tellegen, 1988), autocorrelations between ratings two hours apart (Smyth et al., 2007), and the correlation between consecutive ratings (Deaver, Miltenberger, Smyth, Meidinger, & Crosby, 2003). Thus, inverse variance weights were calculated using an estimated r = .65 for analyses of negative affect. Importantly, results from the meta-analyses were essentially unchanged when assuming r = 0, suggesting that findings were not unduly influenced by this assumption.

A random-effects model was used to compute mean ES for each comparison. A random-effects model was more appropriate than a fixed-effects model because we expected true population ES to vary across studies due to differences in sample and methodology commonly associated with “real-world” data (Field, 2003). Thus, this model accounted for within-study error and variation in true ES across studies whereas a fixed-effect model would have used within-study error alone. A consequence of including between-study error in a random-effects model was larger standard errors of the summary ES, which made this a more conservative approach to testing the significance of computed mean ES. In addition, a random-effects model has the advantage of allowing inferences to extend beyond the studies included in this review. In our analyses, a statistically significant mean ES indicated that there was a greater than chance difference between assessment time points.

Analysis of heterogeneity and potential moderators—Heterogeneity was assessed using tau-squared (τ^2) as a measure of the between-study variance component. Tau-squared was evaluated according to standards for small (τ^2 = v/3), medium (τ^2 = 2v/3), and large (τ^2 = v) degrees of heterogeneity, where v represented the proportion of common variance (Hedges & Pigott, 2001). ES were analyzed for statistically significant heterogeneity using the Q statistic and a chi-square test (Hedges & Olkin, 1985). Moderator analyses were conducted to assess the source of variation when k > 3 studies. As chi-square tests for heterogeneity have low power, moderator analyses were not conditional on a significant chi-square (Hunter & Schmidt, 2004).

Sample characteristics that were examined as moderators included whether participants were treatment-seeking (yes, no, or a combination in the sample) and sample diagnosis (AN, BN, BED, or "unspecified binge eating"). When participants' use of compensatory methods was not explicitly stated, the sample was coded as "unspecified binge eaters." Methodological characteristics coded as moderators included method of affect assessment (single vs. multiple item measure), whether or not structured clinical interviews were used for diagnostic assessment (yes, no), whether or not the DSM definition of binge eating was provided to participants (yes, no), length of EMA (in days), type of EMA methods used (interval-, signal-, or event-contingent, each coded as "yes" or "no"), and frequency of daily assessments (number of signal- and interval ratings per day). Moderator data were extracted from each article and coded independently by both authors. Inter-rater reliability was excellent, ranging from kappa = 0.82 to 1.00 for categorical moderators and intra-class correlations ranging from .99 to 1.00 for continuous moderators. When authors disagreed on moderator coding, discrepancies were resolved by consensus.

Moderators were first examined in univariate analyses using an analog to the Analysis of Variance (for categorical variables) or regression (for continuous variables). To avoid potential confounding between variables and reduce Type I error, moderators for each comparison were then evaluated in a single multiple regression according to a variable reduction strategy suggested by Hosmer and Lemeshow (1999). All moderators that demonstrated a univariate association of $p < .20$ were included in an initial multiple regression model. This threshold was used because a more traditional threshold of $p < .05$ has been shown to exclude potentially important variables in the initial stage of variable reduction (Hosmer & Lemeshow, 1999). Each moderator was sequentially removed from this initial model to examine the percentage change in regression coefficients of the remaining variables. If removal of the moderator resulted in < 20% change in the regression coefficients of all remaining variables (indicating no significant interactions), this moderator was dropped from the multivariate regression model. This process continued until no more moderators could be dropped from the model. The statistical significance of each coefficient in the final multivariate regression model was used to determine whether each moderator variable accounted for heterogeneity in overall mean ES.

Moderator analyses were conducted using fixed-effects models. Interpretation of moderator variables as fixed effects is appropriate when all relevant levels of the moderator variable are included (Overton, 1998). With few exceptions, all relevant levels of the proposed moderator variables were included in the meta-analyses (e.g., use of interval-contingent methods as "yes" or "no"). The two exceptions to this were length of assessment and frequency of assessments. However, both variables demonstrated adequate range across studies and thus inability to generalize beyond this range does not significantly detract from conclusions that may be drawn from the analyses. In addition, fixed-effects models have more statistical power. Although mixed-effects analyses are generally preferred over fixed-effects analyses due to concerns about overly narrow confidence intervals and inflated Type I error rates associated with fixed-effects, Monte Carlo simulations have found that a random-effects model is biased when based on few studies (i.e., $k = 5–10$; Field, 2001; Hafdahl & Williams, 2009). Thus, a fixed-effects approach was appropriate given the relatively small k when studies were broken down into subgroups for moderator analyses. Further, as described above, we used a moderator variable reduction strategy to control for inflated Type I error. All statistical analyses were conducted using macros developed for SPSS (Lipsey & Wilson, 2001).

Results

Description of Studies

Methodological variables of the 36 reviewed EMA studies, including information regarding sample size (*N*), compliance rates, and all putative moderators are summarized in Table 1. Of the studies reviewed, 21 studies assessed participants with BN, 13 assessed participants with BED, and three assessed unspecified binge eaters. Binge eating can occur within the binge-purge subtype of AN and studies of AN were sought; however, only one EMA study that assessed binge eating and negative affect in participants with AN was found (Engel et al., 2005). Although this study was retained for analyses of associations between affect and binge eating, the diagnostic category of AN and this study were dropped from the univariate and multivariate moderator analyses.

Sample size ranged from $n = 8$ (Schlundt, Johnson, & Jarrell, 1985) to $n = 131$ (Smyth et al., 2007), and studies had a mean (*SD*) sample size of $n = 28.7$ (21.8) participants. Combining across all studies, a total of 968 independent participants were included in this review.2 All participants in these studies were female and, when participant race was reported (Agras & Telch, 1998; Dopp, 1995; Engel et al., 2005; le Grange, Gorin, Catley, & Stone, 2001; le Grange, Gorin, Dymek, & Stone, 2002; Rydin-Gray, 2007; Smyth et al., 2007; Stein et al., 2007; Telch & Agras, 1996), the majority (mean = 89.2%) were Caucasian. EMA studies ranged in duration from one eating episode (Agras & Telch, 1998; Telch & Agras, 1996) to five weeks (Redlin, Miltenberger, Crosby, Wolff, & Stickney, 2002), with a mean length of EMA of one and a half weeks ($M = 10.2$, $SD = 6.6$ days). The average study that utilized signal- or interval-based methods assessed participants' experiences 7.8 ($SD = 7.2$) times per day.

Assessment Protocols and Compliance

All three assessment protocols, interval-contingent, signal-contingent, and event-contingent, have been used in studies of binge eating (see Table 1). The majority of studies ($k = 28$, 78%) included event-contingent recording that asked participants to make ratings before and/or after any eating episode (including binge eating). Thus, as a group, studies were well-suited to examine affective differences in antecedents and consequences of binge eating compared to normal meals and snacks. In addition, several studies combined event and time-based methods (interval or signal; $k = 17$, 47%), which allowed comparisons of participant's average psychological state and their state immediately preceding or following binge eating or purging behavior. For signal-contingent methods, compliance rates ranged from 76% (Wegner et al., 2002) to 92% (Engel et al., 2005), and participants responded to an average of 84.1% of random signals. For interval-contingent methods, participants completed an average of 91.5% of interval ratings.

Antecedents of Binge Eating

Pre-binge versus average negative affect—Table 2 includes individual study ES (within-subjects standardized mean gains) for negative affect ($k = 17$) as well as an overall ES for comparisons of pre-binge eating affect to average affect. As predicted, negative affect was greater prior to binge eating compared to average ratings, suggesting that individuals experienced greater negative affect before binge eating compared to general levels of negative affect. The mean weighted ES was significantly greater than zero (ES = .63, 95%

[2]The total number of independent participants ($N = 968$) is different from the total number of participants in Table 1 ($N = 1005$) due to sample overlap between Davis et al. (1985) and Davis et al. (1988) and between Engelberg et al. (2007) and Steiger et al. (2005). Because these studies reported different analyses of the same sample, participant overlap did not contribute to non-independence of ES in the meta-analyses.

CI = .45 to .82; $p < .001$). Heterogeneity analyses suggested a large degree of variability among ES (τ^2 =.106) that was statistically significant ($Q(16) = 69.62$, $p < .001$).

Univariate moderator analyses are presented in Table 3. Diagnosis, use of interval-, signal-, and event-contingent methods, treatment-seeking, and length of EMA contributed to ES heterogeneity at $p < .20$ and were subsequently included in a multivariate regression model. Method of affect assessment, definition of binge eating, use of structured diagnostic assessments, and assessment frequency did not contribute to ES heterogeneity in univariate analyses and were not investigated further.

Each moderator variable included in the initial multiple regression model was sequentially eliminated. Of the six initial moderator variables, length of EMA and use of event-contingent methods could be removed from the model without > 20% change in the regression coefficients of any other variable. The final multivariate regression model is presented in Table 4. Diagnosis accounted for a significant amount of variability (B = .26, $p < .05$). Although negative affect was significantly higher prior to binge eating compared to average for both BN and BED, the magnitude of this ES was significantly smaller in BN participants compared to BED participants. Interval-contingent methods also contributed to ES variability (B = −.40, $p < .05$). Use of interval-contingent ratings was associated with smaller ES compared to studies that did not use interval-contingent responding. Treatment-seeking status and use of signal-contingent methods did not demonstrate significant associations with ES in the multivariate regression model. The significant residual found for this model indicated that there remained unexplained between-studies variance after accounting for the contribution of identified moderators.

Pre-binge versus pre-regular eating negative affect—Individual study ES ($k = 14$) as well as an overall ES for comparisons of pre-binge eating negative affect to pre-regular eating negative affect are listed in Table 2. Negative affect was higher prior to binge eating compared to pre-regular eating episodes, suggesting that individuals experienced greater negative affect before binge episodes than before they consumed regular meals or snacks. The average ES was significant (ES = .68, 95% CI = .40 to .95, $p < .001$). Heterogeneity analyses suggested a large degree of variability among ES ($\tau^2 = .238$) that was statistically significant ($Q(13) = 93.25$, $p < .001$).

Univariate moderator analyses are presented in Table 5. Use of event-contingent methods could not be examined as a moderator because event-contingent responding to eating episodes was used in all but one study. Diagnosis, definition of binge eating, use of signal-contingent methods, length of EMA, and frequency of assessments all demonstrated univariate associations of $p < .20$ and were included in multivariate analyses. Method of affect assessment, use of interval-contingent methods, treatment-seeking, and use of structured interviews in the diagnostic assessment did not contribute to ES heterogeneity in univariate analyses and were not examined further.

Following sequential elimination of each moderator variable included in the initial multiple regression model, diagnosis, definition of binge eating, use of signal-contingent methods, length of EMA, and frequency of assessments all remained significant predictors of ES heterogeneity (see Table 6). Mirroring results for pre-binge versus average comparisons, ES were significantly smaller for participants with BN compared to participants with BED (B = .32, $p < .01$). Thus, mood was significantly more negative prior to binge eating versus regular eating episodes in BED compared to BN. Smaller ES were observed when binge eating was defined for participants compared to when it was not defined (B = −.82, $p < .001$). Use of signal-contingent methods was associated with significantly larger ES than studies that did not use signal-contingent methods (B = 1.22, $p < .001$). Finally, longer

duration of EMA (B = −.07, $p < .01$) and greater frequency of assessments (B = −.02, $p < .05$) were associated with smaller ES. The significant residual for the model indicated significant unexplained between-studies variance after accounting for the contribution of identified moderators.

Consequences of Binge Eating

Post-binge versus pre-binge negative affect—Individual study ES ($k = 14$) and the overall ES for changes in negative affect as a consequence of binge eating are listed in Table 7. Notably, all ES were positive. Negative affect increased significantly from pre- to post-binge, indicating that individuals experienced greater levels of negative affect after binge eating than they experienced prior to their binge episode. The mean ES of increased negative affect following binge eating was significant (ES = .50, 95% CI: .35 to .64, $p < .001$). There was a large degree of heterogeneity in ES ($\tau^2 = .040$), which was statistically significant ($Q(13) = 30.86$, $p < .01$).

Univariate moderator analyses are presented in Table 8. Definition of binge eating, use of interval-, signal-, and event-contingent methods, and frequency of assessments contributed to ES heterogeneity at $p < .20$ and were included in the multivariate analysis. Diagnosis, method of affect assessments, treatment-seeking, use of structured interviews in diagnostic assessment, and length of EMA were not associated with ES heterogeneity and were dropped from further analyses.

None of the moderators that demonstrated univariate associations at $p < .20$ could be removed from the multivariate regression model (see Table 9). However, only assessment frequency remained a significant predictor of ES heterogeneity (B = .02, $p < .01$). Greater frequency of assessment was associated with larger ES. As with other models, the significant residual indicated that there remained unexplained between-studies variance after accounting for the contribution of identified moderators.3

Purging Comparisons

The hypothesis that purging serves to reduce negative affect could be examined in three studies not summarized in tables of study ES (Alpers & Tuschen-Caffier, 2001; Corstorphine, Waller, Ohanian, & Baker, 2006; Powell & Thelen, 1996).4 Negative affect was significantly lower after purging compared to before purging, and the average ES differed significantly from zero (ES = −.46, 95% CI: −.74 to −.18, $p < .01$). There was no statistically significant heterogeneity in these ES ($Q(2) = 3.59$, $p = .17$; $\tau^2 = .027$). In addition, negative affect levels following purging did not differ from pre-binge affect levels; the average ES for comparisons of pre-binge and post-purge negative affect was not statistically significant from zero (ES = .10, 95% CI: −.09 to .30, $p = .29$), and there was no significant heterogeneity in ES ($Q(2) = 1.02$, $p = .60$; $\tau^2 = .000$). Moderator analyses were not conducted because there were only $k = 3$ studies available. Preliminary analyses suggest

[3]Although results indicated that negative affect did not decrease following binge episodes as predicted by the affect regulation model, regulation may occur through an increase in positive affect. Specifically, experience of reward following ingestion of highly palatable food and central signaling of dopamine pathways (Small et al., 2003) may serve to positively reinforce binge episodes through increases in positive affect. Individual study ES ($k = 7$) and the mean weighted ES for comparisons of pre-binge eating and post-binge eating positive affect were calculated as described for negative affect. The mean weighted ES was significant (ES = −.52, 95% CI = −.81 to −.23). A negative mean ES indicated that there was lower positive affect after binge eating compared to before binge eating, suggesting that individuals experienced a significant decrease in positive emotions following a binge.

[4]One study found that negative affect was greater post-purging compared to postbingeing (Lingswiler, Crowther, & Stephens, 1989b). However, post-binge ratings included only those binge episodes that were not followed immediately with purging. Thus, the comparison was between negative affect after binge episodes that were not associated with later purging and negative affect after purged binge episodes. This study was not included in the meta-analysis because it did not document increasing negative affect over the course of a binge episode followed by purging, but may have reflected the tendency for purged binges to be associated with greater negative affect compared to non-purged binges.

that purging may reduce negative affect that follows binge episodes. However, negative affect returns to pre-binge levels and thus the binge-purge cycle does not appear to successfully regulate negative affect.

Publication Bias

Although we sought both published and unpublished manuscripts for this review, studies with non-significant findings may have been excluded from the meta-analyses due to the file-drawer phenomenon (Rosenthal, 1979). Thus, we conducted an analysis of publication bias to assess the potential impact of missing studies on meta-analytic results. Rosenthal's fail-safe *N* was calculated to determine the number of studies averaging a null effect that would be needed to elevate the significance of overall ES above a two-tailed $p = .05$. Rosenthal's fail-safe *N* was 748 for comparisons of pre-binge and average negative affect levels, 480 for comparisons of pre-binge negative affect and pre-regular eating negative affect, and 390 for comparisons of pre-binge and post-binge negative affect. Even the lowest fail-safe *N* of 390 indicates that there would need to be 28 missing studies of null effect for every study included in the meta-analysis for overall ES to become non-significant. Thus, the significance of meta-analytic findings for binge eating and negative affect was fairly robust and unlikely to have been influenced by publication biases. In contrast, Rosenthal's fail-safe *N* was only 11 studies for comparisons of post-binge and post-purge negative affect and two for comparisons of pre-binge and post-purge negative affect, which likely reflected that these meta-analyses were based on a very small number of studies ($k = 3$). Thus, findings for the affective consequences of purging should be considered preliminary as this may remain an important factor in reinforcing binge-purge cycles in women with BN.[5]

Discussion

Increases in negative affect were supported as antecedents of binge eating in BN and BED, as evidenced by elevated negative affect prior to binge eating compared to average levels of negative affect and compared to negative affect prior to regular eating episodes. Both effect sizes for negative affect were larger in magnitude for BED samples compared to BN samples. Results further indicated that negative affect *increases* after binge eating episodes in both BN and BED. This finding failed to support a key prediction of the affect regulation model, which proposes that binge eating is maintained through negative reinforcement by reducing negative affect (Hawkins & Clement, 1984). Further, this pattern of results contradicted retrospective participant reports that binge eating reduces negative emotions (Abraham & Beumont, 1982; Hawkins & Clement, 1984; Hsu, 1990; Stickney, Miltenberger, & Wolff, 1999).

Results from most EMA studies that could not be included in the quantitative analyses supported negative affect as an antecedent of binge eating (Johnson, Schlundt, Barclay, Carr-Nangle, & Engler, 1995; Schlundt et al., 1985; Stickney et al., 1999). However, two studies failed to find significant increases in negative affect prior to binge eating. A study of 37 BED women found no differences among negative affect ratings that occurred nine hours, five hours, and one hour before binge eating (Sanftner & Crowther, 1998). In addition, a study of 20 adolescents with BN found that morning and afternoon negative affect was not a significant predictor of evening bulimic symptoms (Dopp, 1995). Notably, both studies assessed negative affect over a substantial amount of time prior to binge eating and may have been unable to adequately capture more immediate antecedents of binge eating as assessed in studies included in the meta-analyses. Meta-analytic findings for post-

[5]Rosenthal's fail-safe *N* was 85 for comparisons of pre-binge and post-binge positive affect. Although smaller than comparisons of negative affect, overall conclusions regarding positive affect and binge eating were unlikely to be attributed to sampling bias.

binge increases in negative affect were generally consistent with results from EMA studies that could not be included in analyses due to the absence of data required to calculate effect sizes (Steiger, Gauvin, Jabalpurwala, Séguin, & Stotland, 1999; Steiger et al., 2005; Stickney et al., 1999). However, one study (Elmore & de Castro, 1990) found that changes in negative affect depended on the dimension of affect assessed. Although depression increased after binge eating (consistent with findings from our meta-analysis), anxiety decreased after binge eating, suggesting an equivocal effect of binge eating on negative affect in this study.

Although effect sizes for antecedent negative affect were larger for BED compared to BN, this does not necessarily mean that binge episodes in BED are associated with greater antecedent negative mood compared to BN. One possibility is that BN participants experience greater absolute negative mood in general and prior to all eating episodes compared to BED participants. Consistent with this hypothesis, BN participants have reported greater negative mood prior to binge episodes compared to BED participants (Hilbert & Tuschen-Caffier, 2007; Lingswiler, Crowther, & Stephens, 1989a), and greater overall negative affect compared to BED participants in EMA (Hilbert & Tuschen-Caffier, 2007) and cross-sectional studies (Striegel-Moore et al., 2001). If individuals with BN experience chronically high negative affect, then a smaller increase in negative affect may be required to trigger binge eating, or there may be a ceiling effect for examining changes in negative affect. Additional research is needed to determine if differences in negative affect between BN and BED may help explain why individuals with BN use compensatory behaviors whereas individuals with BED do not.

One possible explanation for the apparent contradiction between EMA results and predictions from the affect regulation model is that binge eating may result in an immediate but time-limited reduction in negative affect that is quickly replaced with an increase in negative affect as the potential consequences of binge eating become more salient. However, most studies included in the meta-analysis evaluated the consequences of binge eating through immediate post-binge reports. For example, using this approach, Wegner et al. (2002) found that immediate post-binge mood was more negative than immediate pre-binge mood. An alternative explanation is that binge eating is associated with immediate increases in negative affect, but is negatively reinforced by delayed reductions in negative mood. Supporting this assertion, Smyth et al. (2007) found that binge eating was associated with decreasing negative affect and anger over time. However, Wegner et al. (2002) found no differences in mood rated 30–60 minutes prior to binge eating and 30–60 minutes post binge eating. Comparisons of immediate post-binge mood and delayed mood have indicated that delayed negative affect was the same (Corstorphine et al., 2006; Lingswiler, Crowther, & Stephens, 1989b) or even worse (Sherwood, Crowther, Wills, & Ben-Porath, 2000) one hour after the binge episode compared to immediately post-binge. Thus, binge episodes do not seem to lead to delayed decreases in negative affect. In addition, negative reinforcement is most powerful for immediate rather than delayed consequences. Thus, it seems unlikely that binge eating would be maintained by eventually leading to decreases in negative affect.

Another possible explanation is that binge eating is maintained through changes in affect *during* binge eating episodes that are not maintained following a binge. Such effects would not be captured in our analyses of affect before and after binge eating. A handful of EMA studies have asked participants to make momentary ratings of affect before, *during*, and after binge eating. Of these, only one study found that negative affect decreased significantly from pre-binge to during the binge episode (Deaver et al., 2003). Two studies found no differences in negative affect from before to during binge episodes in participants with BN (Hilbert & Tuschen-Caffier, 2007; Powell & Thelen, 1996) and BED (Hilbert & Tuschen-Caffier, 2007). Finally, affect worsened during the binge episode compared to before binge

eating in two studies (Johnson & Larson, 1982; Stickney & Miltenberger, 1999). Thus, preliminary examinations indicated no clear support for reductions in negative affect during binge episodes. Of note, inconsistent results may be due to the nature of EMA methodology. A central premise of Heatherton and Baumeister's (1991) escape model is that binge eating is reinforced through reduced aversive self-awareness. Asking individuals to monitor their mood during binge eating could interfere with the posited influence of binge eating on self-awareness as self-monitoring requires participants to be self-aware. Thus, EMA is not ideal for testing hypotheses about changes in affect due to changes in self-awareness during binge eating. Interestingly, Stickney and Miltenberger (1999) assessed mood prior to and during binge eating using two methods: a retrospective questionnaire and a momentary monitoring form. Comparison of these two methods indicated that participants endorsed an increase in negative affect during binge eating when assessed using the momentary monitoring form but reported a decrease in negative affect during binge eating on the retrospective questionnaire. Given that both assessments were made for the same time period (and thus the *same* episodes of binge eating), retrospective recall bias may explain reported reductions in negative affect during binge episodes that did not actually occur. These results highlight the importance of EMA methods for providing a rigorous test of the affect regulation model of binge eating.

Some have argued for a "trade off" theory of affect regulation where a binge functions to trade one type of negative affect (e.g., anger reported prior to binge eating) for a less aversive type of negative affect (e.g., guilt reported following binge eating; Kenardy, Arnow, & Agras, 1996). Thus, binge eating may be reinforced by decreasing the aversiveness of the affect experienced rather than by producing a net decrease in overall negative affect. This effect would be missed by this meta-analysis and the majority of EMA studies which relied on global measures of mood (Alpers & Tuschen-Caffier, 2001; Davis, Freeman, & Solyom, 1985; Deaver et al., 2003; Engel et al., 2005; Hilbert & Tuschen-Caffier, 2007; Sherwood et al., 2000; Smyth et al., 2007; Stein et al., 2007). Partially supporting this possibility, four EMA studies included in the current review found that anxiety decreased from pre- to post-binge eating (Elmore & de Castro, 1990; Hetherington et al., 1994; Kaye et al., 1986; Redlin et al., 2002) while depression (Elmore & de Castro, 1990; Hetherington et al., 1994; Kaye et al., 1986) and guilt (Redlin et al., 2002) increased, and another study found that anger and irritability decreased while sadness and shame increased post-binge eating (Johnson & Larson, 1982). However, other studies found consistent post-binge increases in anxiety, depression, and hostility (Powell & Thelen, 1996); anxiety and shame/guilt (Corstorphine et al., 2006); or anger, guilt, and depression (Wegner et al., 2002), suggesting that a trade-off in facets of negative affect have not been reliably observed. Unfortunately, there were not enough studies that examined different facets of negative affect to examine this question using meta-analysis. Limited and contradictory research suggests that it would be useful to analyze specific facets of negative affect separately in future research.

Finally, although binge eating does not appear to be reinforced by post-binge reductions in negative affect, it is possible that episodes are positively reinforced by increases in positive affect related to the hedonic aspects of eating highly palatable foods (e.g., Small, Jones-Gotman, & Dagher, 2003). This has not been a central tenet of the affect regulation model for binge eating and was explored in only seven EMA studies. Results of these studies suggested that positive affect decreased after binge eating episodes (see Footnote 3). However, this result does not rule out the possibility that positive affect increases *during* a binge episode. Given the small number of studies, the role of positive affect in triggering and maintaining binge episodes represents an underdeveloped aspect of the literature.

Although most research attention has been given to testing the affect regulation model of binge eating, some researchers have proposed that purging rather than binge eating regulates affect (Rosen & Leitenberg, 1982) and that binge eating may be used simply to precipitate purging in BN (Leitenberg, Gross, Peterson, & Rosen, 1984). Partially supporting this modification of the affect regulation model, negative affect was significantly lower after purging compared to after binge eating in this meta-analysis, suggesting that purging may be negatively reinforced by a reduction in negative affect that follows binge eating episodes in women with BN. Importantly, affect levels following purging did not differ significantly from pre-binge affect levels. Thus, as a set of behaviors, the binge-purge episode does not appear to effectively regulate negative affect in BN.

Methodological Moderators

Several methodological variables emerged as significant moderators of effect sizes observed in the meta-analyses. Studies differed in whether or not they defined binge eating for participants, and this accounted for a significant proportion of the variability in effect sizes comparing affect before binge eating versus regular eating episodes. Studies that provided the DSM definition of a binge (i.e., an objectively large amount of food coupled with a loss of control over eating) were associated with smaller effect sizes compared to studies that relied on participant definitions. Previous research supports important differences between the DSM definition of a binge and lay definitions. Specifically, lay definitions rely more on the experience of loss of control rather than the amount of food consumed and often include episodes of emotional eating (i.e., eating to relieve negative affect and the presence of dysphoric mood while eating; Beglin & Fairburn, 1992; Telch et al., 1998). In the absence of an objective definition, participants may have been more likely to classify an emotional eating episode as a binge (Agras & Telch, 1998; Telch & Agras, 1996). This would have ensured larger effect sizes associated with subjectively reported binge episodes compared to DSM defined binge episodes, which may serve as a confound for understanding the association between negative affect and binge eating in studies using lay definitions. Importantly, effect sizes differed significantly from zero even when studies provided DSM definitions of a binge episode, and the valence of effect sizes was stable across all methodological variations. Thus, overall conclusions regarding the association between binge eating and antecedent negative affect appear to be representative of binge episodes as defined in the DSM.

Use of interval-contingent methods was associated with smaller effect sizes for comparisons of pre-binge and average levels of negative affect. When participants are assessed at the same time each day, as in interval-contingent methods, there may be predictable changes in behavior and mood (e.g., not engaging in binge behavior in anticipation of making a rating; Smyth et al., 2001). Thus, interval-contingent ratings may not be a representative sampling of average levels of negative affect, resulting in smaller differences between average ratings and pre-binge ratings. In contrast, signal-contingent methods were associated with larger effect sizes for comparisons of pre-binge and pre-regular eating negative affect. As signals are unpredictable, these ratings may be more sensitive to changes and more reliable leading to smaller estimates of error and larger differences between time points.

Frequency of non-event assessments (interval- and signal-contingent rating frequency) was also supported as a significant moderator of study effect sizes. Greater frequency was associated with smaller effect sizes for comparisons of pre-binge and pre-regular eating negative affect, but was associated with larger effect sizes for comparisons of pre-binge and post-binge negative affect. Mathematically, any factor that lowered pre-binge negative affect would contribute to both a smaller effect size when comparing pre-binge to pre-regular eating negative affect *and* a larger effect size when comparing pre-binge to post-binge negative affect. One possible explanation for how more frequent assessments contributed to

lower pre-binge negative affect is if increased participant burden led to decreased compliance, with participants more likely to skip non-event assessments when negative affect was particularly high.

Finally, length of assessment was a significant moderator of comparisons of pre-binge versus pre-regular eating negative affect. Contrary to our hypothesis, shorter duration of assessment was associated with larger effect sizes. One possible explanation for this is that several studies included participants who were in treatment during the course of EMA assessment. Thus, it is possible that longer durations of assessment captured decreased binge eating frequency and alternative responses to negative affect as a consequence of treatment. Of note, treatment-seeking was not a significant moderator in multivariate analyses, and we had an inadequate number of studies to evaluate whether there was an interaction between treatment-seeking and length of assessment in moderation of effect sizes. Thus, we cannot directly evaluate this possible explanation.

Methodological Considerations and Limitations of EMA

EMA is ideally suited to assess events that precede and follow a behavior within an individual's natural environment; however, a key concern for any study utilizing EMA is inability to draw causal inferences from a longitudinal design. Establishing that negative affect was higher before binge eating compared to other times during the day does not tell us that increases in negative affect *caused* a person to binge eat as this is essentially a correlational design over time. This concern may be particularly problematic for behaviors that require planning. For example, up to 75% of individuals acknowledge that they sometimes plan to binge eat, evidenced by purchasing or cooking special foods in advance (Abraham & Beumont, 1982). Potentially, individuals who know they will binge experience elevations in negative affect in anticipation of the binge rather than an increase in negative affect causing the binge. Similarly, an association between binge eating followed by increased negative affect cannot be used to establish that a binge episode *caused* an individual to feel worse. There may be another variable that contributes to both binge eating and negative affect, such as interpersonal conflict (the third-variable problem). Importantly, experimental studies are not subject to these limitations, and studies that have experimentally manipulated mood have found that increased negative affect caused increased caloric intake (Chua et al., 2004) and binge eating (Agras & Telch, 1998; Telch & Agras, 1996) in laboratory settings. Thus, EMA studies extend these findings by supporting the ecological validity of laboratory-based models of negative affect causing binge eating. To our knowledge, no studies have experimentally manipulated occurrence of binge eating to examine the impact of binge eating on mood.

In addition to not being able to infer causation from EMA, it is possible that the use of self-monitoring in EMA could cause changes in the behaviors being measured, or reactivity (Shiffman et al., 2008). Reactivity may be both behavioral, influencing the frequency of binge eating, and psychological, affecting scores on measures of affect. Thus, the failure of EMA studies to support the affect regulation model of binge eating could reflect the influence of EMA methods on the variables under study. Specifically, EMA may reduce the effectiveness of binge eating in reducing negative affect by making people more aware that they are engaging in an undesirable behavior. This may be particularly true for protocols that involve a short duration of assessment as reactivity is likely to decline as people become accustomed to self-monitoring (Moos, 2008). However, if this were true, we would have expected duration of assessment to be a significant moderator, and this was not observed for results supporting increased negative affect after binge eating. In addition, the direction of effect sizes for duration of assessment on pre-binge versus pre-regular eating (i.e., effect sizes decreased with duration of assessment) is in the opposite direction of that predicted by reactivity.

While reactivity has been recognized as a challenge in self-monitoring (Stone & Shiffman, 1994), to our knowledge only one study has attempted to examine the extent to which disordered eating behaviors are reactive to EMA. Stein and Corte (2003) asked participants with AN or BN to monitor their disordered eating behaviors over a four-week period. To assess reactivity, behavioral frequencies were compared for various time frames. The authors posited that if reactivity were present, it would decline over the period of observation and reported behavioral frequencies would increase. There were no differences in behavioral frequencies comparing the first and last halves of data collection or comparing the first, second, and last thirds of the assessment period. Results may indicate that there is no evidence of behavioral reactivity to self-monitoring of disordered eating behaviors, similar to other areas of EMA research (Shiffman et al., 2008). However, because the length of time during which reactivity might be observed is unknown, results may indicate that reactivity was present throughout the entire assessment period. Thus, while research on reactivity to EMA procedures is inconclusive, there is no strong support for the hypothesis that reactivity occurs or can account for evidence that negative affect increases after binge eating episodes.

Another concern associated with EMA is decreased compliance with study protocols when participant burden is high (Wheeler & Reis, 1991). When reported, compliance rates were approximately 80% or higher across EMA studies of binge eating, supporting good compliance in this methodology (Sonnenschein et al., 2007). Moreover, compliance rates for studies included in this review were comparable to those reported in EMA studies in other psychiatric samples (Hufford & Shields, 2002). However, many studies (k = 21; 58%) did not report compliance, raising the possibility that missing data may have skewed findings and interpretations.

Finally, overall mean effect sizes relied on within-person changes in affect and behavior and should not be compared with between-subjects mean difference effect sizes. Thus, current results focus on predicting when binge episodes will occur or recur among individuals who binge eat and do not capture who is vulnerable to developing binge eating problems. Trait negative affectivity as a between-subjects factor is supported as a risk factor for the development of eating pathology in prospective, longitudinal studies (Stice, 2002). Thus, while current results did not support post-binge reductions of negative affect in the maintenance of binge eating, they did not refute the relevance of trait negative affectivity as an individual difference that impacts whether or not an individual will develop binge eating.

Clinical Implications

A majority of patients who binge believe that binge eating will reduce their negative emotions (Abraham & Beumont, 1982; Hawkins & Clement, 1984; Hsu, 1990; Stickney et al., 1999), and this belief is associated with the development (Hayaki, 2009) and maintenance (Bohon et al., 2009) of bulimic symptoms. Thus, current results may be used to change how information regarding the function of binge eating is conveyed in treatment. Individuals with eating disorders may benefit from psychoeducation regarding predictable increases in negative affect as a consequence of binge eating. Given that the most likely outcomes are either no changes in negative affect or a worsening of negative affect, patients should be informed that binge eating is not an effective means of affect regulation in order to modify their beliefs about the effects of binge eating on mood.

Results further suggest that specific therapeutic techniques may be beneficial. Individuals who binge may benefit from treatments that emphasize distress tolerance and acceptance, such as Dialectical Behavior Therapy (Linehan, Cochran, & Kehrer, 2001) or Acceptance and Commitment Therapy (Hayes, Strosahl, & Wilson, 1999). In addition, exposure and response prevention techniques may be useful. Exposure to pre-binge levels of negative

affect and prevention of subsequent binge eating may lessen urges to binge in response to negative affect as individuals learn that negative affect decreases over time (Jansen, Van Den Hout, De Loof, Zandbergen, & Griez, 1989; Jansen, Broekmate, & Heymans, 1992).

Future Research

This review highlights the utility of EMA in examining complex temporal relationships among antecedents and consequences of binge eating. Although results failed to support the affect regulation model of binge eating, current findings may be consistent with alternative theoretical models that do not propose that binge eating leads to decreased negative affect. Additional research is needed to test which theory best fits the data. Several aspects of restraint theory have not been tested by EMA studies. Specifically, restraint theory proposes that loss of cognitive control can occur in the context of affective, cognitive, or pharmacological disinhibitors (Ruderman, 1986). Cognitive and pharmacological disinhibitors could be tested as proximal triggers of binge eating by including questions regarding violation of dietary rules (posited cognitive disinhibitor) and alcohol or drug consumption (posited pharmacological disinhibitors) in EMA protocols.

Studies also are needed to evaluate the validity of escape theory. According to this model, individuals are motivated to binge eat to escape from aversive self-awareness generated by perceived discrepancies between high personal ideals and subjective evaluations of the self (Duval & Wicklund, 1972). EMA studies could assess temporal changes in discrepancies between the ideal and perceived self prior to binge episodes. However, this does not address concerns that self-report EMA monitoring during binge episodes would interfere with the process under study. As an alternative to self-report, psychophysiological ambulatory monitoring, such as heart rate and vagal activity, has been used to examine affect regulation in borderline personality disorder (e.g., Ebner-Priemer et al., 2007) and could be used as an indirect measure of changes in affect during binge episodes. As more sophisticated methodologies for evaluating self-awareness are developed, these could be incorporated into EMA designs to test escape theory.

Finally, research on expectancy theory has focused exclusively on between-person differences and has not adequately examined within-person changes in cognitive expectancies as a proximal antecedent of binge eating. In addition, studies have not examined the extent to which eating expectancies may moderate associations between negative affect and the occurrence of binge eating in mixed samples. Given that participants reported increased negative affect after binge eating in EMA studies, it would be interesting to assess whether this is accompanied by temporary changes in expectancies. Moreover, EMA studies could assess whether changes in negative reinforcement expectancies predict when binge episodes occur.

In addition to testing alternative theoretical models, future EMA research needs to expand beyond evaluations of binge eating to examine antecedents and consequences of purging in the absence of binge eating, observed in a syndrome known as Purging Disorder (PD; Keel, Haedt, & Edler, 2005; Keel & Striegel-Moore, 2009). PD is currently a form of Eating Disorder Not Otherwise Specified (American Psychiatric Association, 2000) that has been recommended for inclusion as a provisional syndrome within Eating Disorders Not Elsewhere Classified in the DSM-5 (http://www.dsm5.org/ProposedRevisions/Pages/proposedrevision.aspx?rid=26). Our results suggested that the binge-purge episode was not effective in regulating affect. However, the affective consequences of purging may differ between BN and PD. At best, it appears that women with BN return to pre-binge levels of elevated negative affect. In contrast, women with PD may achieve a net decrease in negative affect given the absence of binge eating episodes and our preliminary evidence that purging does decrease negative affect. Such

results may explain why studies have found lower levels of depression in PD compared to BN (Keel, Wolfe, Gravener, & Jimerson, 2008; Keel et al., 2005; Wade, 2007). As noted above, although studies of binge eating and negative affect in AN were sought, only one was found (Engel et al., 2005). Additional studies are needed that speak to the antecedents or consequences of disordered eating behaviors that may be more central to this eating disorder, such as increased physical activity (Pieters et al., 2006; Vansteelandt, Rijmen, Pieters, Probst, & Vanderlinden, 2007).

Finally, all EMA studies conducted thus far have been in adult women who were primarily Caucasian. Caucasian women have reported greater body dissatisfaction and more disordered eating compared to African American women (Grabe & Hyde, 2006; Wildes, Emery, & Simons, 2001); however, more recent research has suggested that eating disorder prevalence in women does not differ across ethnic or racial groups (Marques et al., in press). However, the influence of any ethnic or racial differences on the function of binge eating remains unknown. Thus, future studies should examine temporal relations between putative antecedents and consequences of disordered eating among more ethnically diverse samples and in men and children to examine generalizability of these findings. Future EMA research has the unique potential to improve our understanding of the mechanisms maintaining disordered eating behaviors and to contribute to the development of more effective treatments – an important goal as the most efficacious intervention succeeds in only a minority of patients (Wilson et al., 2007).

Conclusion

Results challenge the widely accepted affect regulation model of binge eating. Although negative affect increases prior to binge eating, it continues to increase following binge eating, failing to support a key prediction of the affect regulation model. Results may be consistent with alternative models positing that negative affect triggers binge eating; however, these alternative models require further exploration using EMA. With modification and extension of previous studies, future EMA research has the potential to further test and refine theoretical models of bulimic symptoms and enhance evidence-based treatments for eating disorders.

Acknowledgments

This research was supported by the National Institute of Mental Health through a National Research Service Award (F31 MH085456, PI: Alissa Haedt-Matt). Portions of this work were presented at the 14th Annual Meeting of the Eating Disorders Research Society in Montreal, Canada and at the 16th Annual Meeting of the Eating Disorders Research Society in Cambridge, MA. We wish to acknowledge the following individuals for their helpful feedback: Lee Anna Clark, Kristian Markon, Mike O'Hara, and David Watson.

References

References marked with an asterisk indicate studies included in the meta-analyses.

Abraham SF, Beumont PJ. How patients describe bulimia or binge eating. Psychological Medicine. 1982; 12:625–635. [PubMed: 6957905]

Agras WS, Telch CF. The effects of caloric deprivation and negative affect on binge eating in obese binge-eating disordered women. Behavior Therapy. 1998; 29:491–503.

Aldao A, Nolen-Hoeksema S, Schweizer S. Emotion-regulation strategies across psychopathology: A meta-analytic review. Clinical Psychology Review. 2010; 30:217–237. [PubMed: 20015584]

Alpers GW, Tuschen-Caffier B. Negative feelings and the desire to eat in bulimia nervosa. Eating Behaviors. 2001; 2:339–352. [PubMed: 15001027]

American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders. Washington, D.C.: American Psychiatric Association; 2000. (Fourth Edition - Text Revision [DSM-IV-TR])

Arnow B, Kenardy J, Agras WS. Binge eating among the obese: A descriptive study. Journal of Behavioral Medicine. 1992; 15:155–170. [PubMed: 1583679]

Baumeister, RF. Self-inference processes: The Ontario symposium. Vol. Vol. 6. Hillsdale, NJ, England: Lawrence Erlbaum Associates, Inc.; 1990. Anxiety and deconstruction: On escaping the self; p. 259-291.

Beglin SJ, Fairburn CG. What is meant by the term "binge"? The American Journal of Psychiatry. 1992; 149:123–124. [PubMed: 1728161]

Bohon C, Stice E, Burton E. Maintenance factors for persistence of bulimic pathology: A prospective natural history study. International Journal of Eating Disorders. 2009; 42:173–178. [PubMed: 18951457]

Bolger N, Davis A, Rafaeli E. Diary methods: Capturing life as it is lived. Annual Review of Psychology. 2003; 54:579–616.

Boseck JJ, Engel SG, Allison KC, Crosby RD, Mitchell JE, de Zwaan M. The application of ecological momentary assessment to the study of the night eating. International Journal of Eating Disorders. 2007; 40:271–276. [PubMed: 17177212]

Bruce B, Agras WS. Binge eating in females: A population-based investigation. International Journal of Eating Disorders. 1992; 12:365–373.

Bulik CM, Sullivan PF, Kendler KS. Medical and psychiatric morbidity in obese women with and without binge eating. International Journal of Eating Disorders. 2002; 32:72–78. [PubMed: 12183948]

Campbell-Sills, L.; Barlow, DH. Handbook of emotion regulation. New York, NY, US: Guilford Press; 2007. Incorporating emotion regulation into conceptualizations and treatments of anxiety and mood disorders; p. 542-559.

Cassin SE, von Ranson KM. Personality and eating disorders: A decade in review. Clinical Psychology Review. 2005; 25:895–916. [PubMed: 16099563]

Chua JL, Touyz S, Hill AJ. Negative mood-induced overeating in obese binge eaters: An experimental study. International Journal of Obesity. 2004; 28:606–610. [PubMed: 14968127]

Cooper JL, Morrison TL, Bigman OL, Abramowitz SI, Levin S, Krener P. Mood changes and affective disorder in the bulimic binge-purge cycle. International Journal of Eating Disorders. 1988; 7:469–474.

Cooper PJ, Bowskill R. Dysphoric mood and overeating. British Journal of Clinical Psychology. 1986; 25:155–156. [PubMed: 3460666]

Cooper Z, Cooper PJ, Fairburn CG. The validity of the eating disorder examination and its subscales. British Journal of Psychiatry. 1989; 154:807–812. [PubMed: 2597887]

Corstorphine E, Waller G, Ohanian V, Baker M. Changes in internal states across the binge-vomit cycle in bulimia nervosa. Journal of Nervous and Mental Disease. 2006; 194:446–449. [PubMed: 16772863]

Davis R, Freeman RJ, Garner DM. A naturalistic investigation of eating behavior in bulimia nervosa. Journal of Consulting and Clinical Psychology. 1988; 56:273–279. [PubMed: 3372835]

Davis R, Freeman RJ, Solyom L. Mood and food: An analysis of bulimic episodes. Journal of Psychiatric Research. 1985; 19:331–335. [PubMed: 3862832]

Davis R, Jamieson J. Assessing the functional nature of binge eating in the eating disorders. Eating Behaviors. 2005; 6:345–354. [PubMed: 16257808]

Deaver CM, Miltenberger RG, Smyth J, Meidinger A, Crosby R. An evaluation of affect and binge eating. Behavior Modification. 2003; 27:578–599. [PubMed: 12971129]

Dopp DM. An analysis of the relationship between daily hassles, negative emotions, and bulimic symptoms. Dissertation Abstracts International: Section B: The Sciences and Engineering. 1995; 56:2321.

Dunlap WP, Cortina JM, Vaslow JB, Burke MJ. Meta-analysis of experiments with matched groups or repeated measures designs. Psychological Methods. 1996; 1:170–177.

Duval, S.; Wicklund, RA. A theory of objective self awareness. Oxford, England: Academic Press; 1972.

Ebner-Priemer UW, Welch SS, Grossman P, Reisch R, Linehan MM, Bohus M. Psychophysiological ambulatory assessment of affective dysregulation in borderline personality disorder. Psychiatry Research. 2007; 150:265–275. [PubMed: 17321599]

Elmore DK, de Castro JM. Self-rated moods and hunger in relation to spontaneous eating behavior in bulimics, recovered bulimics, and normals. International Journal of Eating Disorders. 1990; 9:179–190.

Engel SG, Wonderlich SA, Crosby RD, Wright TL, Mitchell JE, Crow SJ, Venegoni EE. A study of patients with anorexia nervosa using ecologic momentary assessment. International Journal of Eating Disorders. 2005; 38:335–339. [PubMed: 16261606]

Engelberg MJ, Gauvin L, Steiger H. A naturalistic evaluation of the relation between dietary restraint, the urge to binge, and actual binge eating: A clarification. International Journal of Eating Disorders. 2005; 38:355–360. [PubMed: 16231355]

Engelberg MJ, Steiger H, Gauvin L, Wonderlich SA. Binge antecedents in bulimic syndromes: An examination of dissociation and negative affect. International Journal of Eating Disorders. 2007; 40:531–536. [PubMed: 17573684]

Fairburn, CG. Cognitive behavior therapy and eating disorders. New York: Guilford Press; 2008.

Field AP. Meta-analysis of correlation coefficients: A Monte Carlo comparison of fixed- and random-effects methods. Psychological Methods. 2001; 6:161–180. [PubMed: 11411440]

Field AP. The problems in using fixed-effects models of meta-analysis on real-world data. Understanding Statistics. 2003; 2:105–124.

Gleaves DH, Williamson DA, Barker SE. Additive effects of mood and eating forbidden foods upon the perceptions of overeating and binging in bulimia nervosa. Addictive Behaviors. 1993; 18:299–309. [PubMed: 8342442]

Goldman MS, Brown SA, Christiansen BA, Smith GT. Alcoholism and memory: Broadening the scope of alcohol-expectancy research. Psychological Bulletin. 1991; 110:137–146. [PubMed: 1891515]

Grabe S, Hyde JS. Ethnicity and body dissatisfaction among women in the United States: A meta-analysis. Psychological Bulletin. 2006; 132:622–640. [PubMed: 16822170]

Greeno CG, Wing RR, Marcus MD. Nocturnal eating in binge eating disorder and matched-weight controls. International Journal of Eating Disorders. 1995; 18:343–349. [PubMed: 8580920]

Greeno CG, Wing RR, Shiffman S. Binge antecedents in obese women with and without binge eating disorder. Journal of Consulting and Clinical Psychology. 2000; 68:95–102. [PubMed: 10710844]

Gross, JJ. Handbook of emotion regulation. New York, NY, US: Guilford Press; 2007.

Hafdahl AR, Williams MA. Meta-analysis of correlations revisited: Attempted replication and extension of Field's (2001) simulation studies. Psychological Methods. 2009; 14:24–42. [PubMed: 19271846]

Hasler G, Pine DS, Gamma A, Milos G, Ajdacic V, Eich D, Angst J. The associations between psychopathology and being overweight: A 20-year prospective study. Psychological Medicine. 2004; 34:1047–1057. [PubMed: 15554575]

Hawkins, RC.; Clement, PF. Binge eating: Measurement problems and a conceptual model. In: Hawkins, RC.; Fremouw, WJ.; Clement, PF., editors. The binge purge syndrome: Diagnosis, treatment, and research. New York: Springer; 1984. p. 229-251.

Hayaki J. Negative reinforcement eating expectancies, emotion dysregulation, and symptoms of bulimia nervosa. International Journal of Eating Disorders. 2009; 42:552–556. [PubMed: 19172595]

Hayes, SC.; Strosahl, KD.; Wilson, KG. Acceptance and commitment therapy: An experiential approach to behavior change. New York, NY, US: Guilford Press; 1999.

Heatherton TF, Baumeister RF. Binge eating as escape from self-awareness. Psychological Bulletin. 1991; 110:86–108. [PubMed: 1891520]

Hedges, LV.; Olkin, I. Statistical methods for meta-analysis. Orlando, FL: Academic Press; 1985.

Hedges LV, Pigott TD. The power of statistical tests in meta-analysis. Psychological Methods. 2001; 6:203–217. [PubMed: 11570228]

Hedges SM, Jandorf L, Stone AA. Meaning of daily mood assessments. Journal of Personality and Social Psychology. 1985; 48:428–434.

Herman, CP.; Polivy, J. Restrained eating. In: Stunkard, JA., editor. Obesity. Philadelphia, PA: W.B. Saunders Company; 1980. p. 208-225.

Herman, CP.; Polivy, J. A boundary model for the regulation of eating. In: Stunkard, AB.; Stellar, E., editors. Eating and its disorders. New York: Raven Press; 1984. p. 141-156.

Hetherington MM, Altemus M, Nelson ML, Bernat AS, Gold PW. Eating behavior in bulimia nervosa: Multiple meal analyses. The American Journal of Clinical Nutrition. 1994; 60:864–873. [PubMed: 7985626]

Hilbert A, Rief W, Tuschen-Caffier B, de Zwaan M, Czaja J. Loss of control eating and psychological maintenance in children: An ecological momentary assessment study. Behaviour Research and Therapy. 2009; 47:26–33. [PubMed: 19010458]

Hilbert A, Tuschen-Caffier B. Maintenance of binge eating through negative mood: A naturalistic comparison of binge eating disorder and bulimia nervosa. International Journal of Eating Disorders. 2007; 40:521–530. [PubMed: 17573697]

Hohlstein LA, Smith GT, Atlas JG. An application of expectancy theory to eating disorders: Development and validation of measures of eating and dieting expectancies. Psychological Assessment. 1998; 10:49–58.

Hosmer, DW.; Lemeshow, S. Applied survival analysis: Regression modeling of time to event data. New York, NY: John Wiley & Sons Inc.; 1999.

Hsu LG. Experiential aspects of bulimia nervosa: Implications for cognitive behavioral therapy. Behavior Modification. 1990; 14:50–65. [PubMed: 2294901]

Hufford MR, Shields AL. Electronic diaries: Applications and what works in the field. Applied Clinical Trials. 2002; 11:38–43.

Hunter, JE.; Schmidt, FL. Methods of meta-analysis: Correcting error and bias in research findings. Thousand Oaks, CA, US: Sage Publications, Inc.; 2004.

Jansen A, Van Den Hout MA, De Loof C, Zandbergen J, Griez E. A case of bulimia successfully treated by cue exposure. Journal of Behavior Therapy and Experimental Psychiatry. 1989; 20:327–332. [PubMed: 2576871]

Jansen A, Broekmate J, Heymans M. Cue-exposure vs self-control in the treatment of binge eating: A pilot study. Behaviour Research and Therapy. 1992; 30:235–241. [PubMed: 1586360]

Johnson C, Larson R. Bulimia: An analysis of moods and behavior. Psychosomatic Medicine. 1982; 44:341–351. [PubMed: 6959174]

Johnson WG, Schlundt DG, Barclay DR, Carr-Nangle RE, Engler LB. A naturalistic functional analysis of binge eating. Behavior Therapy. 1995; 26:101–118.

Kaye WH, Gwirtsman HE, George DT, Weiss SR, Jimerson DC. Relationship of mood alterations to bingeing behaviour in bulimia. British Journal of Psychiatry. 1986; 149:479–485. [PubMed: 3814933]

Keel PK, Wolfe BE, Gravener JA, Jimerson DC. Co-morbidity and disorder-related distress and impairment in purging disorder. Psychological Medicine. 2008; 38:1435–1442. [PubMed: 17825120]

Keel PK, Dorer DJ, Eddy KT, Delinsky SS, Franko DL, Blais MA, Herzog DB. Predictors of treatment utilization among women with anorexia and bulimia nervosa. The American Journal of Psychiatry. 2002; 159:140–142. [PubMed: 11772705]

Keel PK, Haedt A, Edler C. Purging disorder: An ominous variant of bulimia nervosa? International Journal of Eating Disorders. 2005; 38:191–199. [PubMed: 16211629]

Keel PK, Striegel-Moore R. The validity and clinical utility of purging disorder. International Journal of Eating Disorders. 2009; 42:706–719. [PubMed: 19642215]

Kenardy J, Arnow B, Agras WS. The aversiveness of specific emotional states associated with binge-eating in obese subjects. Australian and New Zealand Journal of Psychiatry. 1996; 30:839–844. [PubMed: 9034475]

Kjelsås E, Børsting I, Gudde CB. Antecedents and consequences of binge eating episodes in women with an eating disorder. Eating and Weight Disorders. 2004; 9:7–15. [PubMed: 15185828]

Klonsky ED. The functions of deliberate self-injury: A review of the evidence. Clinical Psychology Review. 2007; 27:226–239. [PubMed: 17014942]

Larson, R.; Csikszentmihalyi, M. The experience sampling method. In: Reis, HT., editor. Naturalistic Approaches to Studying Social Interaction. Washington: Jossey-Bass; 1983. p. 41-56.

le Grange D, Gorin A, Catley D, Stone AA. Does momentary assessment detect binge eating in overweight women that is denied at interview? European Eating Disorders Review. 2001; 9:309–324.

le Grange D, Gorin A, Dymek M, Stone A. Does ecological momentary assessment improve cognitive behavioural therapy for binge eating disorder? A pilot study. European Eating Disorders Review. 2002; 10:316–328.

Leitenberg H, Gross J, Peterson J, Rosen JC. Analysis of an anxiety model and the process of change during exposure plus response prevention treatment of bulimia nervosa. Behavior Therapy. 1984; 15:3–20.

Linehan, MM.; Cochran, BN.; Kehrer, CA. Clinical handbook of psychological disorders: A step-by-step treatment manual. 3rd ed.. New York, NY, US: Guilford Press; 2001. Dialectical behavior therapy for borderline personality disorder; p. 470-522.

Lingswiler VM, Crowther JH, Stephens MA. Emotional reactivity and eating in binge eating and obesity. Journal of Behavioral Medicine. 1987; 10:287–299. [PubMed: 3612785]

Lingswiler VM, Crowther JH, Stephens MA. Affective and cognitive antecedents to eating episodes in bulimia and binge eating. International Journal of Eating Disorders. 1989a; 8:533–539.

Lingswiler VM, Crowther JH, Stephens MP. Emotional and somatic consequences of binge episodes. Addictive Behaviors. 1989b; 14:503–511. [PubMed: 2589128]

Lipsey, MW.; Wilson, DB. Practical meta-analysis. Thousand Oaks, CA: US: Sage Publications, Inc.; 2001.

Lundgren JD, Allison KC, O'Reardon JP, Stunkard AJ. A descriptive study of non-obese persons with night eating syndrome and a weight-matched comparison group. Eating Behaviors. 2008; 9:343–351. [PubMed: 18549994]

Lynch WC, Everingham A, Dubitzky J, Hartman M, Kasser T. Does binge eating play a role in the self-regulation of moods? Integrative Physiological & Behavioral Science. 2000; 35:298–313. [PubMed: 11330493]

Marques L, Alegria M, Becker AE, Chen CN, Fang A, Chosak A, Diniz JB. Comparative prevalence, correlates of impairment, and service utilization for eating disorders across US ethnic groups: Implications for reducing ethnic disparities in health care access for eating disorders. International Journal of Eating Disorders. (in press). Advance online publication.

McManus F, Waller G. A functional analysis of binge-eating. Clinical Psychology Review. 1995; 15:845–863.

Mitchell JE, Hatsukami D, Eckert ED, Pyle RL. Characteristics of 275 patients with bulimia. The American Journal of Psychiatry. 1985; 142:482–485. [PubMed: 3856401]

Mitchell JE, Mussell MP, Peterson CB, Crow S, Wonderlich SA, Crosby RD, Weller C. Hedonics of binge eating in women with bulimia nervosa and binge eating disorder. International Journal of Eating Disorders. 1999; 26:165–170. [PubMed: 10422605]

Moos RH. Context and mechanisms of reactivity to assessment and treatment: Comment. Addiction. 2008; 103:249–250. [PubMed: 18199303]

National Institute for Clinical Excellence [NICE]. NICE Clinical Guideline No. 9. London: NICE; 2004. Eating disorders: Core interventions in the treatment and management of anorexia nervosa, bulimia nervosa and related eating disorders. Retrieved from: http://guidance.nice.org.uk/CG9

Norton M, Wonderlich SA, Myers T, Mitchell JE, Crosby RD. The use of palmtop computers in the treatment of bulimia nervosa. European Eating Disorders Review. 2003; 11:231–242.

O'Connor DB, Jones F, Conner M, McMillan B, Ferguson E. Effects of daily hassles and eating style on eating behavior. Health Psychology. 2008; 27:S20–S31. [PubMed: 18248102]

Overton RC. A comparison of fixed-effects and mixed (random-effects) models for meta-analysis tests of moderator variable effects. Psychological Methods. 1998; 3:354–379.

Patel KA, Schlundt DG. Impact of moods and social context on eating behavior. Appetite. 2001; 36:111–118. [PubMed: 11237346]

Pieters G, Vansteelandt K, Claes L, Probst M, Van Mechelen I, Vandereycken W. The usefulness of experience sampling in understanding the urge to move in anorexia nervosa. Acta Neuropsychiatrica. 2006; 18:30–37.

Polivy, J.; Herman, CP. Binge eating: Nature, assessment, and treatment. New York, NY: US: Guilford Press; 1993. Etiology of binge eating: Psychological mechanisms; p. 173-205.

Powell AL, Thelen MH. Emotions and cognitions associated with bingeing and weight control behavior in bulimia. Journal of Psychosomatic Research. 1996; 40:317–328. [PubMed: 8861128]

Pyle RL, Mitchell JE, Eckert ED. Bulimia: A report of 34 cases. Journal of Clinical Psychiatry. 1981; 42:60–64. [PubMed: 6936397]

Rebert WM, Stanton AL, Schwarz RM. Influence of personality attributes and daily moods on bulimic eating patterns. Addictive Behaviors. 1991; 16:497–505. [PubMed: 1801573]

Redelmeier DA, Kahneman D. Patients' memories of painful medical treatments: Real-time and retrospective evaluations of two minimally invasive procedures. Pain. 1996; 66:3–8. [PubMed: 8857625]

Redlin JA, Miltenberger RG, Crosby RD, Wolff GE, Stickney MI. Functional assessment of binge eating in a clinical sample of obese binge eaters. Eating and Weight Disorders. 2002; 7:106–115. [PubMed: 17644864]

Rosen JC, Leitenberg H. Bulimia nervosa: Treatment with exposure and response prevention. Behavior Therapy. 1982; 13:117–124.

Rosenthal R. The file drawer problem and tolerance for null results. Psychological Bulletin. 1979; 86:638–641.

Rosenthal R, Rubin DB. Meta-analytic procedures for combining studies with multiple effect sizes. Psychological Bulletin. 1986; 99:400–406.

Ross M. Relation of implicit theories to the 4construction of personal histories. Psychological Review. 1989; 96:341–357.

Ruderman AJ. Dietary restraint: A theoretical and empirical review. Psychological Bulletin. 1986; 99:247–262. [PubMed: 3515384]

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

Sanftner JL, Crowther JH. Variability in self-esteem, moods, shame, and guilt in women who binge. International Journal of Eating Disorders. 1998; 23:391–397.

Schlundt DG, Johnson WG, Jarrell MP. A naturalistic functional analysis of eating behavior in bulimia and obesity. Advances in Behaviour Research & Therapy. 1985; 7:149–162.

Sher, KJ.; Grekin, ER. Alcohol and affect regulation. In: Gross, J., editor. Handbook of emotion regulation. New York, NY: US: Guilford Press; 2007. p. 560-580.

Sherwood NE, Crowther JH, Wills L, Ben-Porath Y. The perceived function of eating for bulimic, subclinical bulimic, and non-eating disordered women. Behavior Therapy. 2000; 31:777–793.

Shiffman, SM.; Stone, AA. Ecological momentary assessment: A new tool for behavioral medicine research. In: Krantz, DS.; Baum, A., editors. Technology and methods in behavioral medicine. Mahwah, NJ: Lawrence Erlbaum Associates Publishers; 1998. p. 117-131.

Shiffman S, Stone AA, Hufford MR. Ecological momentary assessment. Annual Review of Clinical Psychology. 2008; 4:1–32.

Simmons JR, Smith GT, Hill KK. Validation of eating and dieting expectancy measures in two adolescent samples. International Journal of Eating Disorders. 2002; 31:461–473. [PubMed: 11948651]

Small DM, Jones-Gotman M, Dagher A. Feeding-induced dopamine release in dorsal striatum correlates with meal pleasantness ratings in healthy human volunteers. NeuroImage. 2003; 19:1709–1715. [PubMed: 12948725]

Smith GT, Simmons JR, Flory K, Annus AM, Hill KK. Thinness and eating expectancies predict subsequent binge-eating and purging behavior among adolescent girls. Journal of Abnormal Psychology. 2007; 116:188–197. [PubMed: 17324029]

Smyth JM, Wonderlich SA, Heron KE, Sliwinski MJ, Crosby RD, Mitchell JE, Engel SG. Daily and momentary mood and stress are associated with binge eating and vomiting in bulimia nervosa patients in the natural environment. Journal of Consulting and Clinical Psychology. 2007; 75:629–638. [PubMed: 17663616]

Smyth J, Wonderlich S, Crosby R, Miltenberger R, Mitchell J, Rorty M. The use of ecological momentary assessment approaches in eating disorder research. International Journal of Eating Disorders. 2001; 30:83–95. [PubMed: 11439412]

Sonnenschein M, Mommersteeg PMC, Houtveen JH, Sorbi MJ, Schaufeli WB, van Doornen LJP. Exhaustion and endocrine functioning in clinical burnout: An in-depth study using the experience sampling method. Biological Psychology. 2007; 75:176–184. [PubMed: 17379387]

Steiger H, Gauvin L, Engelberg MJ, Kin NMKN, Israel M, Wonderlich SA, Richardson J. Mood- and restraint-based antecedents to binge episodes in bulimia nervosa: Possible influences of the serotonin system. Psychological Medicine: A Journal of Research in Psychiatry and the Allied Sciences. 2005; 35:1553–1562.

Steiger H, Gauvin L, Jabalpurwala S, Séguin JR, Stotland S. Hypersensitivity to social interactions in bulimic syndromes: Relationship to binge eating. Journal of Consulting and Clinical Psychology. 1999; 67:765–775. [PubMed: 10535243]

Stein KF, Corte CM. Ecologic momentary assessment of eating-disordered behaviors. International Journal of Eating Disorders. 2003; 34:349–360. [PubMed: 12949927]

Stein RI, Kenardy J, Wiseman CV, Dounchis JZ, Arnow BA, Wilfley DE. What's driving the binge in binge eating disorder?: A prospective examination of precursors and consequences. International Journal of Eating Disorders. 2007; 40:195–203. [PubMed: 17103418]

Steinberg S, Tobin DL, Johnson C. The role of bulimic behaviors in affect regulation: Different functions for different patient subgroups? International Journal of Eating Disorders. 1990; 9:51–55.

Stice E. A prospective test of the dual-pathway model of bulimic pathology: Mediating effects of dieting and negative affect. Journal of Abnormal Psychology. 2001; 110:124–135. [PubMed: 11261386]

Stice E. Risk and maintenance factors for eating pathology: A meta-analytic review. Psychological Bulletin. 2002; 128:825–848. [PubMed: 12206196]

Stickney MI, Miltenberger RG. Evaluating direct and indirect measures for the functional assessment of binge eating. International Journal of Eating Disorders. 1999; 26:195–204. [PubMed: 10422609]

Stickney MI, Miltenberger RG, Wolff G. A descriptive analysis of factors contributing to binge eating. Journal of Behavior Therapy and Experimental Psychiatry. 1999; 30:177–189. [PubMed: 10619542]

Stone AA, Shiffman S. Ecological momentary assessment (EMA) in behavioral medicine. Annals of Behavioral Medicine. 1994; 16:199–202.

Striegel-Moore R, Cachelin FM, Dohm F, Pike KM, Wilfley DE, Fairburn CG. Comparison of binge eating disorder and bulimia nervosa in a community sample. International Journal of Eating Disorders. 2001; 29:157–165. [PubMed: 11429978]

Striegel-Moore RH, Wilfley DE, Pike KM, Dohm FA, Fairburn CG. Recurrent binge eating in black American women. Archives of Family Medicine. 2000; 9:83–87. [PubMed: 10664648]

Tachi T, Murakami K, Murotsu K, Washizuka T. Affective states associated with bingeing and purging behaviours in Japanese patients with bulimia nervosa. British Journal of Medical Psychology. 2001; 74:487–496. [PubMed: 11780796]

Teasdale JD, Fogarty SJ. Differential effects of induced mood on retrieval of pleasant and unpleasant events from episodic memory. Journal of Abnormal Psychology. 1979; 88:248–257. [PubMed: 500952]

Telch CF, Agras WS. Obesity, binge eating and psychopathology: Are they related? International Journal of Eating Disorders. 1994; 15:53–61. [PubMed: 8124327]

Telch CF, Agras WS. The effects of short-term food deprivation on caloric intake in eating-disordered subjects. Appetite. 1996; 26:221–234. [PubMed: 8800479]

Telch CF, Agras WS, Rossiter EM. Binge eating increases with increasing adiposity. International Journal of Eating Disorders. 1988; 7:115–119.

Telch CF, Pratt EM, Niego SH. Obese women with binge eating disorder define the term binge. International Journal of Eating Disorders. 1998; 24:313–317. [PubMed: 9741042]

Timmerman GM, Gregg EK. Dieting, perceived deprivation, and preoccupation with food. Western Journal of Nursing Research. 2003; 25:405–418. [PubMed: 12790056]

Vanderlinden J, Grave RD, Fernandez F, Vandereycken W, Pieters G, Noorduin C. Which factors do provoke binge eating? An exploratory study in eating disorder patients. Eating and Weight Disorders. 2004; 9:300–305. [PubMed: 15844404]

Vansteelandt K, Rijmen F, Pieters G, Probst M, Vanderlinden J. Drive for thinness, affect regulation and physical activity in eating disorders: A daily life study. Behaviour Research and Therapy. 2007; 45:1717–1734. [PubMed: 17276387]

Wade TD. A retrospective comparison of purging type disorders: Eating disorder not otherwise specified and bulimia nervosa. International Journal of Eating Disorders. 2007; 40:1–6. [PubMed: 16958118]

Wanous JP, Reichers AE. Estimating the reliability of a single-item measure. Psychological Reports. 1996; 78:631–634.

Waters A, Hill A, Waller G. Internal and external antecedents of binge eating episodes in a group of women with bulimia nervosa. International Journal of Eating Disorders. 2001; 29:17–22. [PubMed: 11135328]

Watson D, Clark LA, Tellegen A. Development and validation of brief measures of positive and negative affect: The PANAS scales. Journal of Personality and Social Psychology. 1988; 54:1063–1070. [PubMed: 3397865]

Wedig MM, Nock MK. The functional assessment of maladaptive behaviors: A preliminary evaluation of binge eating and purging among women. Psychiatry Research. 2010; 178:518–524. [PubMed: 20537726]

Wegner KE, Smyth JM, Crosby RD, Wittrock D, Wonderlich SA, Mitchell JE. An evaluation of the relationship between mood and binge eating in the natural environment using ecological momentary assessment. International Journal of Eating Disorders. 2002; 32:352–361. [PubMed: 12210650]

Wheeler L, Reis HT. Self-recording of everyday life events: Origins, types, and uses. Journal of Personality. 1991; 59:339–354.

Wild B, Eichler M, Feiler S, Friederich H, Hartmann M, Herzog W, Zipfel S. Dynamic analysis of electronic diary data of obese patients with and without binge eating disorder. Psychotherapy and Psychosomatics. 2007; 76:250–252. [PubMed: 17570965]

Wildes JE, Emery RE, Simons AD. The roles of ethnicity and culture in the development of eating disturbance and body dissatisfaction: A meta-analytic review. Clinical Psychology Review. 2001; 21:521–551. [PubMed: 11413866]

Wilfley DE, Schwartz MB, Spurrell EB, Fairburn CG. Using the eating disorder examination to identify the specific psychopathology of binge eating disorder. International Journal of Eating Disorders. 2000; 27:259–269. [PubMed: 10694711]

Wilson, GT. Assessment of binge eating. In: Fairbum, CG.; Wilson, GT., editors. Binge eating: Nature, assessment and treatment. New York: Guilford Press; 1993. p. 227-249.

Wilson GT, Grilo CM, Vitousek KM. Psychological treatment of eating disorders. American Psychologist. 2007; 62:199–216. [PubMed: 17469898]

Wolfe BE, Baker CW, Smith AT, Kelly-Weeder S. Validity and utility of the current definition of binge eating. International Journal of Eating Disorders. 2009; 42:674–686. [PubMed: 19610126]

Wolff GE, Crosby RD, Roberts JA, Wittrock DA. Differences in daily stress, mood, coping, and eating behavior in binge eating and nonbinge eating college women. Addictive Behaviors. 2000; 25:205–216. [PubMed: 10795945]

Yanovski SZ, Nelson JE, Dubbert BK, Spitzer RL. Association of binge eating disorder and psychiatric comorbidity in obese subjects. The American Journal of Psychiatry. 1993; 150:1472–1479. [PubMed: 8379549]

Table 1

Methodological Features of Ecological Momentary Assessment Studies

Study	N	Mean Age	Diagnosis (Criteria)	Structured Diagnostic Interview	Treatment-Seeking	Affect Assessment	Binge Defined	Length of Assessment	Method of Assessment (frequency)	Compliance
Agras & Telch (1998)	60	42.7	BED (DSM-IV)	Yes	Not reported	Multiple items	No	1 eating episode	Event (eating episode)	Not reported
Alpers & Tuschen-Caffier (2001)	40	24.8	BN (DSM-III-R)	Yes	Combination	Multiple items for antecedents; single item for consequences	No	2 days	Interval (hourly) Event (all eating episodes)	Not reported
Cooper & Bowskill (1986)	12	18.3	BN (unspecified)	No	Yes	Multiple items	No	7 days	Interval (hourly)	Not reported
Corstorphine et al. (2006)	23	29.9	BN (DSM-IV)	Yes	Yes	Multiple items	No	7 days	Event (binge eating)	Not reported
Davis et al. (1985)	16	23.6	BN (DSM-III)	No	Yes	Single item	No	6 days	Interval (hourly)	Not reported
Davis et al. (1988)	20	23.7	BN (DSM-III)	No	Yes	Single item	No	6 days	Interval (hourly)	Not reported
Deaver et al. (2003)	22	19.8	Self-identified binge eaters (unspecified)	No	No	Single item	No	Range: 4–7 days	Event (all eating episodes)	97.4% completed; self-reported timeliness: 85–90%
Dopp (1995)	20	16.8	BN (DSM-III-R)	No	Yes	Multiple items	No	7 days	Signal (8)	78% completed
Elmore & de Castro (1990)	19	22	BN (DSM-III-R)	No	No	Multiple items	No	7 days	Event (all eating episodes)	Not reported
Engel et al. (2005)	10	27.6	AN (DSM-IV)	Yes	Not reported	Multiple items	No	14 days	Interval (end of day) Signal (6) Event (all eating episodes)	Interval: 85.7% completed Signal: 92% completed
Engelberg et al. (2007)	33	23.7	BN (DSM-IV)	Yes	Yes	Multiple items	Yes	Mean: 16.18 days	Signal (3) Event (all eating episodes)	Not reported
Gleaves et al. (1993)	20	Not reported	BN (DSM-III-R)	Yes	Yes	Single item	No	14 days	Event (all eating episodes)	Not reported
Greeno et al. (2000)	41	39	BED (unspecified)	Yes	Yes	Multiple items	No	6 days	Signal (6) Event (all eating episodes)	76% completed on time; 89% participants completed all days

Study	N	Mean Age	Diagnosis (Criteria)	Structured Diagnostic Interview	Treatment-Seeking	Affect Assessment	Binge Defined	Length of Assessment	Method of Assessment (frequency)	Compliance
Hetherington et al. (1994)	10	24.2	BN (DSM-III-R)	No	Yes	Single item	No	7 days	Event (all eating episodes)	Not reported
Hilbert & Tuschen-Caffier (2007)	20 20	24.47 36.65	BN (DSM-IV) BED (DSM-IV)	Yes	No	Single item	Yes	2 days	Signal (32) Event (all eating episodes)	BN: 77.6% completed BED: 92.3% completed
Johnson & Larson (1982)	15	23	BN (DSM-III)	No	Yes	Multiple items	No	7 days	Signal (7)	91.4% completed
Johnson et al. (1995)	25	39.9	BED (unspecified)	No	Combination	Single item	Yes	21 days	Event (all eating episodes)	Not reported
Kaye et al. (1986)	12	24	BN (DSM-III)	No	Not reported	Multiple items	No	1 day	Interval (hourly after binge eating) Event (all eating episodes)	Not reported
le Grange et al. (2001)	18	45.57	BED (DSM-IV)	Yes	No	Single item	Yes	14 days	Signal (5–6) Event (all eating episodes)	81.8% returned monitoring forms
le Grange et al. (2002)	41	44.2	BED (DSM-IV)	Yes	Yes	Not reported	Yes	14 days	Interval (6) Event (all eating episodes)	Not reported
Lingswiler et al. (1989a,b)	19 15	18.5 18.4	BN (DSM-III) BED (unspecified)	No	No	Single item for antecedents; multiple items for consequences	Yes	7 days	Event (all eating episodes)	Not reported
Powell & Thelen (1996)	22	20.25	BN (DSM-III-R)	Yes	No	Multiple items	No	6 days	Interval (every 2 hours) Event (binge eating)	Interval: 90% completed Event: 86% completed
Rebert et al. (1991)	13 13	19.5	BN (DSM-III-R) BED (unspecified)	No	No	Multiple items	No	20 days	Interval (2)	97.4% completed
Redlin et al. (2002)	10	44.7	BED (DSM-IV)	No	Yes	Multiple items	Yes	Range: 7–35 days	Interval (end of day) Event (binge eating)	Not reported
Rydin-Gray (2007)	38	18.18	BED (DSM-IV)	No	No	Multiple items	Yes	14 days	Signal (1) Event (all eating episodes)	Signal: 88% completed

Study	N	Mean Age	Diagnosis (Criteria)	Structured Diagnostic Interview	Treatment-Seeking	Affect Assessment	Binge Defined	Length of Assessment	Method of Assessment (frequency)	Compliance
Sanftner & Crowther (1998)	37	19.4	BED (unspecified)	No	No	Multiple items	No	7 days	Interval (every 4 hours) Event (all eating)	Not reported
Schlundt et al. (1985)	8	26.5	BN (unspecified)	No	Yes	Single item	No	14 days	Event (all eating)	81.9% days completed
Sherwood et al. (2000)	20	19.1	BN (DSM-III-R)	Yes	No	Multiple items	No	7 days	Event (all eating)	Not reported
Smyth et al. (2007)	131	25.3	BN (DSM-IV)	Yes	Combination	Multiple items	Yes	14 days	Signal (6) Interval (end of day) Event (binge eating)	Signal: 86% completed
Steiger et al. (2005)	21	24.1	BN (DSM-IV)	Yes	Yes	Multiple items	Yes	Mean: 15.2 days	Signal (3) Event (all eating)	Not reported
Steiger et al. (1999)	55	27.46	BN (DSM-IV)	Yes	Yes	Multiple items	Yes	Mean: 18.58 days	Interval (end of day) Event (social interactions)	Not reported
Stein et al. (2007)	33	45.2	BED (DSM-IV)	Yes	Yes	Multiple items	No	7 days	Interval (6)	92.9% completed
Stickney et al. (1999)	16	19.61	Binge eaters (unspecified)	No	No	Multiple items	No	28 days	Interval (end of day) Event (binge eating)	Not reported
Telch & Agras (1996)	30	43.8	BED (DSM-IV)	Yes	No	Multiple items	No	1 eating episode	Event (eating episode)	Not reported
Wegner et al. (2002)	27	19.4	Binge eaters (unspecified)	No	No	Multiple items	No	14 days	Signal (7) Event (binge eating)	76% signals completed

Note. AN = Anorexia Nervosa; BED = Binge Eating Disorder; BN = Bulimia Nervosa; DSM = Diagnostic and Statistical Manual of Mental Disorders.

Table 2

Effect Sizes for Studies Comparing Negative Affect Pre-Binge to Average Affect and Pre-Regular Eating Affect Ratings

	Pre-Binge vs. Average	Pre-Binge vs. Pre-Regular Eating
Study	**Effect size (SE)**	**Effect size (SE)**
Alpers & Tuschen-Caffier (2001)	.35 (.16)*	
Cooper & Bowskill (1986)	1.87 (.45)***	
Davis et al. (1985)	.74 (.25)**	
Davis et al. (1988)		.73 (.22)***
Deaver et al. (2003)		1.43 (.28)***
Elmore & de Castro (1990)		.19 (.19)
Engel et al. (2005)	.28 (.32)	
Engelberg et al. (2007)		.13 (.15)
Gleaves et al. (1993)		.24 (.19)
Greeno et al. (2000)	1.71 (.23)***	2.68 (.33)***
Hilbert & Tuschen-Caffier (2007)		
BED	.68 (.22)**	.65 (.21)**
BN	.45 (.20)*	.74 (.22)***
Johnson & Larson (1982)	.27 (.17)	
le Grange et al. (2001)	.62 (.20)**	.60 (.20)**
Lingswiler et al. (1989a)		
BED		−.17 (.19)
BN		.57 (.24)*
Powell & Thelen (1996)	.46 (.19)*	.49 (.19)*
Rebert et al. (1991)		
BED	.08 (.23)	
BN	.61 (.26)*	
Rydin-Gray (2007)	.98 (.18)***	1.21 (.19)***
Sherwood et al. (2000)		.54 (.13)***
Smyth et al. (2007)	.41 (.08)***	
Steiger et al. (1999)	.24 (.12)*	
Steiger et al. (2005)	1.42 (.28)***	
Stein et al. (2007)	.61 (.16)***	
	k = 17	*k* = 14
Weighted mean	.63 (.09)***	.68 (.14)***
95% CI	.45, .82	.40, .95

Note. All effect sizes are within-subjects standardized mean gains.

* $p < .05$.

** $p < .01$.

*** $p < .001$

Table 3

Univariate Moderator Analyses for Pre-Binge Versus Average Negative Affect Ratings

Moderator	Moderator level	*k*	Effect size	SE	*p*	Between Groups Q	*p*
Diagnosis	BN	10	.43[a]	.05	<.001	12.55	<.001
	BED	6	.77[b]	.08	<.001		
Affect Assessment	Single item	4	.61	.11	<.001	.83	.362
	Multiple items	13	.50	.05	<.001		
Binge Definition	Not provided	10	.56	.07	<.001	.65	.421
	Provided	7	.49	.05	<.001		
Interval Contingency	No	7	.78[a]	.08	<.001	15.87	<.001
	Yes	10	.41[b]	.05	<.001		
Signal Contingency	No	8	.42	.07	<.001	3.59	.058
	Yes	9	.58	.05	<.001		
Event Contingency[1]	No	6	.50[a]	.09	<.001	19.68	<.001
	Yes – all eating episodes	7	.86[b]	.08	<.001		
	Yes – binge episodes only	3	.40[a]	.07	<.001		
Treatment-Seeking	No	7	.58	.08	<.001	5.34	.069
	Yes	7	.60	.07	<.001		
	Combination	2	.39	.07	<.001		
Diagnostic Assessment	Not Structured Interview	6	.60	.09	<.001	.98	.322
	Structured Interview	11	.50	.05	<.001		
			B	**SE**	***p***	**Model R^2**	
Length			−.01	.01	.058	.05	
Frequency			.00	.01	.730	.00	

Note. Effect size superscripts that differ represent significant differences at $p < .05$ between levels of a moderator. The first set of p-values indicates whether the effect size differs significantly from zero within moderator level. The second set of p-values indicates whether effect sizes differ significantly across moderator levels.

[1] Event-contingent responding to social interactions was not included in moderator analyses because $k = 1$.

Table 4

Multivariate Regression Model for Moderators of Pre-Binge Versus Average Negative Affect Ratings

Moderator	B	SE	Beta	*p*
Diagnosis	.26	.11	.34	.021
Interval Contingent	−.40	.18	−.53	.022
Signal Contingent	−.08	.14	−.11	.561
Treatment-Seeking	.12	.09	.27	.196
	Q	***p***	**R^2**	
Model	21.21	<.001	.31	
Residual	47.85	<.001		

Table 5

Univariate Moderator Analyses for Pre-Binge Versus Pre-Regular Eating Negative Affect Ratings

Moderator	Moderator level	*k*	Effect size	SE	*p*	Between Groups Q	*p*
Diagnosis[1]	BN	8	.42[a]	.06	<.001	8.20	.004
	BED	5	.75[b]	.10	<.001		
Affect Assessment	Single item	8	.52	.08	<.001	.31	.577
	Multiple items	6	.58	.07	<.001		
Binge Definition	Not provided	7	.63	.07	<.001	2.06	.151
	Provided	7	.48	.07	<.001		
Interval Contingency	No	12	.55	.06	<.001	.08	.780
	Yes	2	.59	.14	<.001		
Signal Contingency	No	8	.44[a]	.07	<.001	6.93	.009
	Yes	6	.72[b]	.08	<.001		
Treatment-Seeking	No	10	.57	.06	<.001	.28	.594
	Yes	4	.51	.10	<.001		
Diagnostic Assessment	Not Structured Interview	6	.58	.09	<.001	.17	.683
	Structured Interview	8	.54	.06	<.001		
			B	**SE**	***p***	**Model R^2**	
Length			−.02	.01	.043	.04	
Frequency			.01	.01	.103	.03	

Note. Effect size superscripts that differ represent significant differences at $p < .05$ between levels of a moderator. The first set of p-values indicates whether the effect size differs significantly from zero within moderator level. The second set of p-values indicates whether effect sizes differ significantly across moderator levels.

[1] Diagnosis of binge eater was not included in moderator analyses because $k = 1$.

Table 6

Multivariate Regression Model for Moderators of Pre-Binge Versus Pre-Regular Eating Negative Affect Ratings

Moderator	B	SE	Beta	*p*
Diagnosis	.32	.11	.34	.005
Binge Definition	−.82	.15	−.81	<.001
Signal Contingency	1.22	.23	1.20	<.001
Length	−.07	.02	−.64	.002
Frequency	−.02	.01	−.48	.026
	Q	***p***	**R^2**	
Model	60.59	<.001	.65	
Residual	32.66	<.001		

Table 7

Effect Sizes for Studies Comparing Negative Affect Post-Binge to Pre-Binge Ratings

Study	Effect size (SE)
Alpers & Tuschen-Caffier (2001)	.51 (.16)**
Corstorphine et al. (2006)	.51 (.19)**
Davis et al. (1985)	.84 (.26)**
Deaver et al. (2003)	.04 (.18)
Engel et al. (2005)	.56 (.35)
Hilbert & Tuschen-Caffier (2007)	
BED	.58 (.21)**
BN	.92 (.24)***
Johnson & Larson (1982)	.15 (.17)
Powell & Thelen (1996)	.52 (.19)**
Redlin et al. (2002)	.20 (.27)
Sherwood et al. (2000)	.23 (.12)*
Smyth et al. (2007)	.56 (.08)***
Stein et al. (2007)	.58 (.16)***
Wegner et al. (2002)	1.11 (.22)***
	k = 14
Weighted mean	.50 (.07)***
95% CI	.35, .64

Note. All effect sizes are within-subjects standardized mean gains.

* $p < .05$.

** $p < .01$.

*** $p < .001$.

Table 8

Univariate Moderator Analyses for Post-Binge Versus Pre-Binge Eating Negative Affect Ratings

Moderator	Moderator level	*k*	Effect size	SE	*p*	Between Groups Q	*p*
Diagnosis	BN	8	.48	.05	<.001	.08	.959
	BED	3	.51	.12	<.001		
	Binge eater	2	.46	.14	<.001		
Affect Assessment	Single item	5	.50	.09	<.001	.07	.791
	Multiple items	9	.48	.05	<.001		
Binge Definition	Not provided	10	.42	.06	<.001	2.72	.099
	Provided	4	.57	.07	<.001		
Interval Contingency	No	7	.39	.07	<.001	3.21	.073
	Yes	7	.55	.06	<.001		
Signal Contingency	No	8	.39[a]	.06	<.001	4.41	.036
	Yes	6	.58[b]	.06	<.001		
Event Contingency	No	3	.45	.11	<.001	5.09	.078
	Yes – all eating episodes	5	.34	.08	<.001		
	Yes – binge episodes only	6	.57	.06	<.001		
Treatment-Seeking	No	6	.44	.07	<.001	1.55	.462
	Yes	5	.44	.09	<.001		
	Combination	2	.55	.07	<.001		
Diagnostic Assessment	Not Structured Interview	5	.39	.09	<.001	1.42	.234
	Structured Interview	9	.51	.05	<.001		
			B	**SE**	***p***	**Model R^2**	
Length			.01	.01	.428	.02	
Frequency			.02	.01	.004	.27	

Note. Effect size superscripts that differ represent significant differences at $p < .05$ between levels of a moderator. The first set of p-values indicates whether the effect size differs significantly from zero within moderator level. The second set of p-values indicates whether effect sizes differ significantly across moderator levels.

Table 9

Multivariate Regression Model for Moderators of Post-Binge Versus Pre-Binge Negative Affect Ratings

Moderator	B	SE	Beta	*p*
Binge Definition	−.22	.16	−.43	.170
Interval Contingency	.15	.10	.31	.135
Signal Contingency	.19	.13	.38	.154
Event Contingency	.10	.07	.32	.144
Frequency	.02	.01	.53	.008
	Q	***p***	**R^2**	
Model	14.81	.011	.48	
Residual	16.04	.042		