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Hunger and Binge Eating: A Meta-analysis of Studies using Ecological Momentary Assessment

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Abstract

Objective—Binge eating has been associated with increased hunger, suggesting a role for impaired appetite regulation. Ecological momentary assessment (EMA) is ideally suited to examine whether hunger is a precipitant of binge eating but results from such studies have not been systematically reviewed. This study provides a meta-analysis of EMA studies that have examined hunger as an antecedent of binge eating.

Method—Electronic database and manual searches produced seven EMA studies with N=180 participants. Meta-analyses were conducted to compare: 1) pre-binge eating hunger to average ratings of hunger, and 2) pre-binge eating hunger to hunger before regular eating.

Results—Across studies, hunger was significantly greater prior to binge eating compared to average hunger ratings, but was significantly lower prior to binge eating compared to before other eating episodes.

Discussion—Excessive hunger does not appear to be a precipitant of binge eating because higher levels of hunger are observed before regular eating episodes. However, lower hunger prior to food consumption may contribute to the experience of a particular eating episode as a binge.

Keywords

binge eating; ecological momentary assessment; hunger

Binge eating represents a central behavioral feature of both Bulimia Nervosa (BN) and Binge Eating Disorder (BED) and is associated with distress and risk of future weight gain and obesity.^{1–4} Thus, it is important to identify factors that contribute to binge eating in order to prevent and treat this pernicious symptom. Previous research indicates that individuals with BN and those with BED report greater levels of hunger compared to controls on the Three Factor Eating or Dutch Eating Behavior Questionnaires,^{5–8} suggesting that greater physiological drive to eat may contribute to risk of developing disorders characterized by binge eating. In addition, individuals who binge commonly endorse hunger as a precipitant to their binge eating in retrospective reports.^{9–14} Thus, high levels of hunger may be implicated as an antecedent trigger of binge eating. However, results based on traditional research methods are associated with methodological limitations that constrain conclusions about the role of hunger in binge eating. Cross-sectional studies cannot assess temporal sequences of changes in hunger and binge eating necessary to establish hunger as an antecedent of binge eating. In addition, the accuracy of retrospective recall is

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questionable due to memory limitations¹⁵ and cognitive biases.¹⁶ To address these limitations, there are a growing number of studies examining hunger and binge eating using ecological momentary assessment.

Ecological momentary assessment (EMA)¹⁷ takes place in participants' naturalistic environments, supporting the ecological validity of this approach. In addition, participants make ratings of current psychological states and behavior, reducing concerns associated with retrospective recall. Finally, EMA involves multiple assessments over time, which allows for the temporal ordering of changes in hunger and binge eating behavior. Thus, EMA is an ideal method for testing hypotheses regarding transient changes in hunger and subsequent binge eating, and several studies have utilized this approach. However, conclusions drawn from these individual studies are somewhat limited by small samples within studies and correspondingly low statistical power which constrains ability to identify potential moderators of associations between hunger and binge eating. Moreover, no systematic review has been conducted of EMA studies examining associations between hunger and binge eating. Thus, the purpose of this review was to examine hunger as an antecedent to binge eating using a meta-analysis of EMA studies.

A meta-analysis has the advantage of aggregating results across studies to increase statistical power. In addition, a meta-analysis allows for the examination of moderating variables, particularly those that vary across but not within studies. For example, studies have varied in sample characteristics (e.g., diagnosis) and methodology (e.g., duration of EMA), and these factors were systematically examined for their potential influence on the magnitude of associations between hunger and binge eating. Based on previous research, we hypothesized that hunger would be elevated prior to binge eating. Support for this hypothesis would provide further evidence for theoretical models that incorporate high levels of hunger as a trigger for binge eating and potentially explain the efficacy of treatment components for binge eating such as the prescription of regular eating and the use of selective serotonin reuptake inhibitors (SSRIs) as these influence appetite.

METHOD

Article Search

Relevant research articles were obtained by searching PsycNet and PubMed electronic databases for published articles and dissertation abstracts with the following keywords: *ecological momentary assessment, experience sampling, event sampling, daily diary, eating disorder, anorexia, bulimia, and binge eating*. Additional articles were identified through manual searches of the reference sections of all articles obtained through the electronic search and e-mail solicitation on the Academy for Eating Disorders listserv. This search was conducted independently by both authors in June of 2009 and produced 99 studies that were initially examined for inclusion in the review. Inclusion criteria required that studies: 1) use EMA methods (i.e., a naturalistic design and momentary ratings), 2) include an assessment of binge eating, 3) include an assessment of hunger prior to binge eating, and 4) include a comparison assessment of hunger rated at a time other than binge eating. Application of these criteria identified ten studies that were eligible for inclusion in this review.

Effect Sizes

Effect sizes were calculated according to two approaches. First, ratings of pre-binge eating hunger were compared to average ratings of hunger. These studies typically used combined event-contingent and time-based (signal- or interval-contingent) approaches. Event-contingent methods require participants to make ratings in response to a specific event or behavior.¹⁸ Participants in these studies were asked to make ratings of hunger before eating

and to indicate which eating episodes were binges. Time-based approaches require participants to make ratings after a specified period of time (interval-contingent) or at random times during the day in response to signals (signal-contingent) in order to get a representative sample of their experiences throughout the day.¹⁸ Average ratings of hunger were calculated from ratings made during these time-based assessments and were not linked to any type of eating episode. Thus, comparisons of pre-binge eating hunger and average hunger indicate whether hunger prior to binge eating is different from participants' usual state. However, hunger may be elevated prior to any food consumption. Thus, a second analysis compared pre-binge eating hunger to pre-regular eating hunger to address the specificity of hunger as a trigger for *binge* eating. These studies typically used event-contingent designs that asked participants to make hunger ratings prior to any eating episode and to identify which eating episodes were binges.

Standardized mean differences (Cohen's d) were used as the index of effect size. When standardized mean differences were not reported, Cohen's d was calculated from published means and standard deviations or from the paired t -test statistic. When means and standard deviations were reported, Cohen's d was computed as mean level of hunger prior to binge eating minus mean level of hunger for average ratings or minus mean level of hunger prior to regular eating episodes divided by the paired standard deviation of the difference scores (pooled standard deviation was used if the paired standard deviation was unavailable). Positive effect sizes indicate that hunger was higher prior to binge eating compared to average hunger or hunger prior to regular-eating episodes. Conversely, negative effect sizes indicate that hunger was lower prior to binge eating relative to the comparison assessment.

Of the ten studies identified for inclusion in the review, it was not possible to calculate an effect size from published data for four studies.^{19–22} Authors of these studies were contacted to determine if data were available to calculate needed effect sizes for inclusion in analyses. Of those contacted, one was able to provide additional data.²¹ Thus, seven studies were included in meta-analyses.

Meta-Analyses

A random-effects model was used to determine overall effect sizes for each comparison because we expected variability in the true population effect sizes across studies due to sample and methodological differences associated with “real-world” data.²³ Overall effect sizes were evaluated according to criteria for small ($d = .20$), medium ($d = .50$), and large ($d = .80$) effects.²⁴ In addition, the Q statistic and a chi-square test were used to test for statistically significant heterogeneity in effect sizes.²⁵ When significant heterogeneity was found, moderator analyses were used to evaluate the source of variation. We used mixed-effects models in which individual effect sizes represent random effects and moderator variables represent fixed effects for moderator analyses. Inclusion of moderator variables as fixed effects is appropriate when all relevant levels of the moderator variable are included.²⁶ Continuous moderators (e.g., duration of EMA) were examined with mixed effects regression using the method of moments. We fit a mixed-effects model using an analogue to the Analysis of Variance (ANOVA) for categorical moderators (e.g., diagnosis of BN vs. BED). We used a regression model Q statistic (for continuous moderators) or between-subjects Q statistic (for categorical moderators) to determine whether each moderator accounted for a significant proportion of the variability in overall mean effect sizes. Analyses were conducted using Comprehensive Meta-Analysis Version 2.0 software.²⁷

Moderators

Several sample and methodological characteristics were specified *a priori* as potential moderators, including participants' mean age, sample diagnosis (BN or BED), whether or

not diagnostic assessment was made using structured clinical interviews, whether or not an objective definition of binge eating was provided to participants (i.e., large amount of food coupled with a loss of control), duration of EMA (number of days during which participants completed momentary assessments), type of EMA methods used (interval-, signal-, and/or event-contingent, each coded as “yes” or “no”), and frequency of non-event assessments (number of assessments per day). It was hypothesized that these factors may affect the strength of association between hunger and binge eating. For example, diagnosis was considered because binge eating has been associated with distinct patterns of cognitions and behaviors in BN compared to BED²⁸ and because binge eating differs in behavioral consequences between these two syndromes. In addition, diagnosis was supported as a significant moderator of associations between negative affect and binge eating in a previous meta-analysis.²⁹

RESULTS

Sample characteristics and methodological features for each study are presented in Table 1. There were a total of $N = 180$ participants across all studies included in this review, with individual studies ranging in size from $n = 15$ ³⁰ to $n = 41$.³¹ Of these studies, four assessed women with BN and four assessed women with BED (one study included both diagnostic groups²¹). Mean age of participants ranged from 18.4 to 45.6 years, with an average (SD) age of 29.43 (11.79) years.

Duration of EMA ranged from six days^{31,32} to 14 days,³³ with an average (SD) length of 7.71 (2.81) days. Only half of the studies reported participant compliance with EMA procedures, and definitions of compliance varied across studies. Despite this variability, compliance rates were comparable to EMA studies in other psychiatric samples,³⁴ suggesting reasonably good compliance in EMA studies of hunger and binge eating.

Pre-Binge versus Average Hunger

Table 2 presents individual study effect sizes and the overall effect size for comparisons of pre-binge eating hunger to average ratings of hunger. Hunger was rated significantly greater prior to binge eating compared to average levels of hunger. The mean weighted effect size was very large and significant ($d = 2.13$, 95% CI = .38 to 3.87; $p < .05$). There was statistically significant heterogeneity in these effect sizes ($Q(4) = 138.26$, $p < .001$).

Moderator analyses indicate several factors that contributed to effect size variability (see Table 3). Sample diagnosis accounted for a significant proportion of this variability; differences between pre-binge and average hunger ratings were smaller in magnitude for BN compared to BED participants ($Q(1) = 4.06$, $p < .05$). This difference was reflected in a small effect size for BN that is not significantly different from zero versus a large effect size for BED that is significantly greater than zero. In addition, larger effect sizes were observed when a structured diagnostic interview was used in the diagnostic assessment ($Q(1) = 4.06$, $p < .05$). The absence of a structured diagnostic interview was associated with a small effect size that was not significantly greater than zero.

Methods of assessment were also supported as significant moderators. Use of signal-contingent methods was associated with larger effect sizes ($Q(1) = 6.84$, $p < .01$), whereas use of interval-contingent ratings was associated with smaller effect sizes ($Q(1) = 6.84$, $p < .01$). Further, larger effect sizes were observed for event-related contingencies that involved responding to all eating episodes compared to studies that did not use event-contingent responding ($Q(1) = 11.59$, $p < .001$), although both effect sizes were significantly greater than zero. Mean age of participants, duration and frequency of assessment, and definition of binge eating did not contribute to effect size heterogeneity.

Notably, the effect size of $d = 10.64$ for Greeno et al.³¹ is substantially higher than any other effect size, reflecting an outlier in this analysis. Thus we conducted analyses both with and without this study. When Greeno et al.³¹ was excluded, hunger remained significantly greater prior to binge eating compared to average hunger ratings, and the mean effect size was small and significant ($d = .34$, 95% CI = .05 to .63, $p < .05$). However, there was no longer significant variability ($Q(3) = 2.07$, $p = .558$). Thus, moderator analyses were not repeated.

Pre-Binge versus Pre-Regular Eating Hunger

Individual study effect sizes and the overall effect size for comparisons of pre-binge eating hunger to pre-regular eating hunger are presented in Table 2. In contrast to comparisons with average ratings of hunger, hunger was rated significantly *lower* prior to binge eating compared to before regular eating episodes. Thus, individuals reported being less hungry before binge eating than before consuming a regular meal. The mean effect size was medium and significant ($d = -.68$, 95% CI = $-.93$ to $-.42$, $p < .001$). There was no statistically significant heterogeneity in effect sizes ($Q(5) = 5.78$, $p = .33$). Thus, moderator analyses were not conducted for pre-binge hunger versus pre-regular eating hunger comparisons.

Publication Bias

Due to the small number of studies, 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 produce nonsignificant ($p > .05$) overall effect sizes. Rosenthal's fail-safe $N = 67$ for comparisons of pre-binge and average hunger and $N = 44$ for comparisons of pre-binge and pre-regular eating hunger. Thus, results appear to be fairly robust against the influence of unpublished studies.

DISCUSSION

Meta-analytic results indicate that hunger is greater prior to a binge episode compared to average hunger ratings, supporting retrospective reports that individuals binge when they are hungry.^{9–14} However, results further indicate that individuals are significantly *less* hungry prior to binge eating compared to before other eating episodes. Thus, excessive hunger does not appear to be a precipitant of binge eating given that higher levels of hunger are observed before regular eating episodes. Overall, this pattern of findings fails to explain the efficacy of establishing a regular eating pattern in cognitive behavioral therapy³⁵ and treatment with SSRIs, suggesting that these interventions may have beneficial effects beyond reducing states of hunger.

Although excessive hunger does not appear to explain the consumption of an unusually large amount of food during a binge episode, lower hunger prior to food consumption may contribute to the perception of a particular eating episode as being excessive and out of control. Indeed, one of the associated characteristics of binge eating in BED is "eating when not physically hungry".³⁶ Thus, individuals may experience a greater loss of control when eating for reasons other than physiological drives. Current findings suggest that other factors, such as cognitive or affective factors, may take precedence over physiological cues in triggering a binge episode. For example, restraint theories posit that cognitive control over food (e.g., eating in response to dietary rules) replaces eating in response to physiological cues, such as hunger and fullness.³⁷ Thus, cognitive control interferes with an individual's ability to eat in response to these physiological cues, setting the stage for consumption of large amounts of food that is not related to hunger.

Further, a recent meta-analysis of EMA studies of binge eating and negative affect suggests that negative affect is higher prior to a binge episode compared to on average and compared to before regular eating episodes.²⁹ Thus, dysregulated affect may be a more proximal trigger of binge eating than dysregulated hunger. Given that hunger is greater prior to binge eating compared to on average, one possibility is that individuals who experience increases in negative affect in the context of some hunger are more likely to turn to food in an attempt to reduce negative affect versus engaging in alternative affect regulation behaviors. Future EMA research is needed to further examine whether hunger moderates associations between changes in negative affect and binge eating.

Preliminary moderator analyses indicate that effect sizes for comparisons of pre-binge and average hunger were significantly larger for BED compared to BN participants, which may make it possible for women with BED to enjoy the hedonic aspects of food as indicated in previous work comparing BN and BED.²⁸ Thus, although the current DSM defines a binge episode the same way for both disorders, this suggests that there may be experiential differences between BN and BED that are consistent with additional research supporting greater elevations of negative affect prior to bingeing in BED compared to BN.²⁹ In addition, effect sizes were larger for studies using event-contingent methods. EMA studies that relies solely on interval- or signal-contingent methods have a restricted ability to detect infrequent behaviors, such as binge eating,³⁸ which may result in less reliable estimates of pre-binge hunger. Finally, use of signal-contingent methods was associated with larger effect sizes, whereas use of interval-contingent ratings was associated with smaller effect sizes. Intervals may become predictable for participants, such that they alter their behavior (e.g., not eating when hungry in anticipation of make a rating).³⁸ Importantly, when the very large effect size from Greeno et al.³¹ was excluded from analyses, we no longer found significant variability among effect sizes which is a prerequisite for conducting moderator analyses. Thus, results from all moderator analyses in this review are preliminary and in need of replication following publication of additional EMA studies to clarify effects.

Although a meta-analysis of EMA studies is ideally suited to test hypotheses regarding changes in hunger and binge eating, this review is not without limitations. First, meta-analyses were restricted to a relatively small number of studies. However, publication bias analyses suggest that overall results are fairly robust. Thus, this limitation may be more likely to have impacted moderation analyses. Second, reactivity is often a concern with EMA studies. Previous research has shown that binge behaviors show minimal reactivity to self-monitoring procedures in EMA.³⁹ However, reactivity may still be a concern for other psychological variables, such as hunger, and this question has yet to be addressed empirically. Finally, the use of self-reported binge eating is a limitation. Studies differed in how they defined binge eating for participants, so some of the binge episodes in this analysis may be better conceptualized as subjective binge episodes or objective overeating. However, we would have expected definition of binge eating to be supported as a significant moderator of effect sizes, and this was not the case.

In summary, results challenge models that emphasize excessive hunger in triggering binge eating episodes. Hunger prior to binge eating is greater than average hunger, but lower than hunger before regular eating episodes. Future EMA research is needed to determine if lower hunger prior to food consumption contributes to the experience of a loss of control.

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Table 1

Sample Characteristics and Methodological Features

Study	N	Mean Age	Diagnosis (Criteria)	Structured Diagnostic Interview	Binge Defined	Length of Assessment	Method of Assessment (frequency)	Compliance
Davis et al. ³²	20	23.7	BN (DSM-III)	No	No	6 days	Interval (hourly)	Not reported
Elmore & de Castro ⁴⁰	19	22	BN (DSM-III-R)	No	No	7 days	Event (all eating episodes)	Not reported
Greeno et al. ³¹	41	39	BED (unspecified)	Yes	No	6 days	Signal (6) Event (all eating episodes)	76% completed on time 89% participants completed all days
Johnson & Larson ³⁰	15	23	BN (DSM-III)	No	No	7 days	Signal (7)	91.4% completed
le Grange et al. ³³	18	45.6	BED (DSM-IV)	Yes	Yes	14 days	Signal (5-6) Event (all eating episodes)	81.8% returned monitoring forms
Lingswiler et al. ²¹	19 15	18.5 18.4	BN (DSM-III) 18.4BED (unspecified)	No	Yes	7 days	Event (all eating episodes)	Not reported
Stein et al. ⁴¹	33	45.2	BED (DSM-IV)	Yes	No	7 days	Interval (6)	92.9% completed

Table 2

Effect Sizes for Studies Comparing Pre-Binge Ratings of Hunger to Average Hunger and Pre-Regular Eating Hunger Ratings

Study	Pre-Binge vs. Average <i>d</i> (SE)	Pre-Binge vs. Pre-Regular Eating <i>d</i> (SE)
Davis et al. ³²	0.00 (.32)	−1.43 (.35) ***
Elmore & de Castro ⁴⁰		−.54 (.25) *
Greeno et al. ³¹	10.64 (.87) ***	−.53 (.23) *
Johnson & Larson ³⁰	.57 (.29) *	
le Grange et al. ³³	.23 (.33)	−.63 (.34)
Lingswiler et al. ²¹		
BED		−.78 (.38) *
BN		−.45 (.33)
Stein et al. ⁴¹	.44 (.26)	
Weighted mean	2.13 (.89) *	−.68 (.13) ***

*
 $p < .05$

 $p < .001$

Table 3

Moderator Analyses for Comparisons of Pre-Binge to Average Ratings of Hunger

Variable	Between Groups Q	k	d (SE)
Mean Age	.45	5	
Sample Diagnosis	4.06*		
BN		2	.31 (.21)
BED		3	.90 (.20)***
Binge Definition	1.53		
Not provided		3	.79 (.19)***
Provided		2	.42 (.22)
Length of Assessment	1.54	5	
Interval Contingency	6.84**		
No		3	1.03 (.21)***
Yes		2	.27 (.20)
Signal Contingency	6.84**		
No		2	.27 (.20)
Yes		3	1.03 (.21)***
Frequency	1.22	5	
Event Contingency	11.59***		
No		3	.37 (.16)*
Yes – all eating episodes		2	1.57 (.31)***
Treatment Seeking Sample	1.75		
No		1	.23 (.33)
Yes		4	.72 (.16)***
Diagnostic Assessment	4.06*		
Not structured interview		2	.31 (.21)
Structured interview		3	.90 (.20)***

*
 $p < .05$ **
 $p < .01$ ***
 $p < .001$