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Do Emotional Eating Urges Regulate Affect? Concurrent and Prospective Associations and Implications for Risk Models of Binge Eating

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Abstract

Objective—Emotional eating (EE) reflects an urge to eat in response to emotional rather than physical cues and is a risk factor for the development of binge eating. EE has been conceptualized as an attempt to regulate negative affect (NA), a posited maintenance factor for binge eating. However, no study has examined whether EE urges regulate affect. Further, no studies have examined longitudinal associations between EE urges and positive affect (PA).

Method—We examined within-subject longitudinal associations between affect and EE urges in a community-based sample of female twins (mean age=17.8 years). Participants (*N*=239) completed ratings of affect and EE urges for 45 consecutive days.

Results—Greater NA was concurrently associated with greater EE urges. Additionally, greater EE urges predicted *worse* NA for both concurrent and prospective (next-day) analyses. Finally, lower PA was associated with greater EE urges in concurrent analyses, but there were no prospective associations between changes in PA and EE urges.

Discussion—EE urges do not appear to effectively regulate affect. EE urges in a communitybased sample appears to have the same functional relationship with affect as binge eating in clinical samples, further supporting EE as a useful dimensional construct for examining processes related to binge eating.

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(2,3). Theoretically, EE promotes the consumption of excessive quantities of food and development of binge eating because eating is reinforced by a reduction in negative affect (NA) instead of being regulated by physiological cues of hunger and satiation. Supporting this model, several studies have found significant associations between EE and binge eating in clinical (4,5) and non-clinical samples (6–8), and EE was a prospective predictor of binge-eating onset among adolescent girls (6). However, no studies have established whether EE is maintained by reductions in NA. Thus, a key assumption regarding how EE contributes to the development of binge eating remains untested.

Longitudinal data are needed to determine the emotional precipitants and consequences of EE. In addition, although affect regulation models typically propose that maladaptive behaviors function to decrease NA, regulation may include increases in positive affect (PA) (9) as dopamine release following food consumption has been associated with subjective pleasure (10). Thus, EE may regulate affect through increases in PA. The current study examined concurrent and prospective associations between affect (positive and negative) and EE urges using a longitudinal, within-subjects design in a large, community-based sample. Such samples are ideal for informing risk models as they can examine longitudinal associations before clinical problems become established and thus can disambiguate consequences of clinical problems from their possible antecedents. Given meta-analytic findings for binge eating (11) as well as theoretical models regarding the role of NA in precipitating EE, we hypothesized that greater NA would predict increases in EE urges. A key question of this study, however, is whether EE urges will successfully regulate affect or whether EE urges would be followed by further deterioration of mood. If EE urges are associated with subsequent increases in NA, this would further challenge affect regulation models for dysregulated eating.

METHODS

Participants

Participants were 239 female twins, ages 16–25 years, (*M*(*SD*)=17.79(1.65)), drawn from the *Twin Study of Hormones and Behavior across the Menstrual Cycle* (12) within the Michigan State University Twin Registry (MSUTR (13,14)). Participant-reported ethnicity was 83.2% Caucasian, non-Hispanic; 12.6% African American; and 4.2% Hispanic. Participants had a mean (SD) body mass index of 23.98(5.66) kg/m².

Procedure and measures

Participants completed daily self-report measures after 5:00p.m. for 45 consecutive days using either a secure on-line system or pre-printed scantrons; 91% of eligible participants completed the study. This research study was reviewed and approved by an institutional review board; all participants completed informed consent prior to participation.

Emotional eating urges—The Dutch Eating Behavior Questionnaire-Emotional Eating subscale (DEBQ-EE (2)) measures desire to eat in response to affective cues (e.g., sadness, anger). The instructions were modified with permission to ask participants how well each item was "true in relation to you TODAY" to capture EE urges at the daily level. Internal consistencies using the unmodified and modified instructions are excellent (α =.93 and α =. 98, respectively (15)). This scale has demonstrated discriminant (16) and criterion validity (17), and has demonstrated significant associations with continuous measures of binge eating (15), supporting the relevance of the DEBQ-EE for assessing and understanding pathological eating behaviors. The DEBQ-EE has demonstrated unique associations with ovarian hormones that were not mediated by NA (15), supporting that the DEBQ-EE is not simply an alternative measure of NA. Internal consistency in the current study was α =.90.

Positive and negative affect—The Positive and Negative Affect Schedule (PANAS (18)) assesses 10 positive (e.g., excited) and 10 negative (e.g., sadness) emotions at the daily level. Good internal consistency, convergent validity, and discriminant validity have been demonstrated for the PANAS (18). In the current study, average internal consistency was a=.85 (NA) and a=.88 (PA).

Statistical analyses

Raw scores for EE urges, NA, and PA were log-transformed to correct for positive skew and multiplied by 10 to prevent boundary constraints. Predictor variables were person-centered on mean values. Multilevel models (MLM) with full maximum-likelihood estimation were used to examine how within-person changes in EE urges were associated with within-person changes in affect. MLM permitted inclusion of participants with missing data (6.82% of observations). A three-level model with random intercepts and slopes was used to control for the non-independence of repeated measures (level 1) within individuals (level 2) and the non-independence of individuals within families (level 3). Separate models were fit to examine the main effects of affect on EE urges and EE urges on affect. Concurrent (sameday) analyses examined associations between affect and EE urges, but are subject to bidirectional interpretations. Thus, time-lagged analyses also examined prospective associations between affect and next-day EE urges and between EE urges and next-day affect. To account for within-variable stability over time in longitudinal data, each dependent variable was entered as a covariate in prospective analyses (e.g., EE urges was entered as a covariate in analyses of affect and next-day EE urges to control for associations due to stability of EE urges across days).

RESULTS

Descriptive statistics were: mean (SD) EE urges=0.35(0.50), range=0-3.69; mean (SD) NA=15.11(5.50), range=10-47; mean (SD) PA=23.62(7.94), range=10-50. Results from concurrent models predicting EE urges and affect measured on the same day are presented in Table 1. Greater NA was concurrently associated with greater EE urges, and lower PA was concurrently associated with greater EE urges. Additionally, greater EE urges were a significant predictor of greater same-day NA and lower same-day PA. Results from prospective models predicting EE urges while controlling for within-variable stability of EE

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urges indicated that neither NA nor PA significantly predicted next-day EE urges (Table 2). However, greater EE urges were associated with greater next-day NA while controlling for within-variable stability of NA. EE urges did not predict PA on the following day.

DISCUSSION

NA was positively associated with EE urges, and greater EE urges predicted worse NA in both concurrent and prospective analyses. However, greater NA did not predict changes in next-day EE urges. Thus, NA may serve as a proximal trigger for EE urges (reflected in concurrent analyses) rather than a distal trigger (reflected in prospective analyses). The desire to eat in response to NA appears to maintain or increase NA over time, consistent with a meta-analysis of longitudinal studies demonstrating that NA is an *immediate* trigger of binge eating and that NA increases further following binge eating (11).

Theoretically, EE represents an attempt to regulate NA; however, current results suggest that this does not work. Instead, there appear to be reciprocal relations in which EE urges contribute to worsening affect, which then contributes to EE urges, resulting in a downward spiral that potentially explains prospective associations between EE and development of binge-eating episodes (6). Our results do not support that EE urges regulate affect through increasing positive feelings (10) because lower PA was concurrently associated with greater EE urges, and there were no significant prospective associations between PA and EE urges.

Taken together, EE urges in a community-based sample may have the same functional relationship with affect as binge eating in clinical samples (11). This is consistent with research demonstrating that EE urges in non-clinical samples has the same functional relationship with ovarian hormones across the menstrual cycle (12,15) as binge eating in clinical samples (19). Current findings and studies of dysregulated eating in children (20) suggest that EE urges may be useful in identifying early disordered eating patterns and etiological processes of binge eating. Research on EE may inform our understanding of the development and course of binge eating, consistent with the Research Domain Criteria initiative (21,22).

Strengths of this study included use of a longitudinal, within-subject design in a large community-based sample with a high retention rate. Study measures demonstrated strong psychometric properties, and study duration minimized concerns regarding the impact of reactivity on daily assessments and random error. This study filled a critical gap in the existing literature by examining both NA and PA, and by investigating both the influence of affect on EE urges *and* the influence of EE urges on affect. Finally, participants completed ratings in their own homes, which increases ecological validity (23).

The present study was limited by the use of once daily assessments of EE urges and affect. Patients often describe significant day-to-day fluctuations in binge eating (19), suggesting that "day" is a clinically meaningful unit of analysis. Nonetheless, it is possible that EE urges may improve affect during or immediately after eating, which would not be captured in the current study. Future research incorporating ecological momentary assessment is needed to examine immediate, momentary relations between affect and EE. A second

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limitation includes our assessment of the desire or urge to eat in response to negative emotions rather than actual eating behavior. However, previous research supports the reliability and validity of our measure of EE urges as tapping behavioral changes that have been directly linked to food intake in both human and animal studies (15), and another study supports the criterion validity of EE urges for assessing dysregulated eating in this sample (17). Finally, longitudinal studies cannot permit causal interpretations. Experimental designs are needed to determine whether food intake triggered by experimental manipulations in affect causes improvement or deterioration in mood.

In summary, EE urges appear to lead to worsening affect on subsequent days. These predictable increases in NA following EE urges may be incorporated into psychoeducational components of treatment to counter patients' belief that eating relieves negative emotions (1,24). Treatments that emphasize exposure to and tolerance of NA, such as exposure plus response prevention (25) or Dialectical Behavior Therapy (26), may help break the link between NA and eating. These approaches also may be integrated into prevention programs to reduce EE given that EE has been demonstrated to be a risk factor for binge eating (6).

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Table 1

Concurrent Models

	Emotional Eating Urges		
Fixed Effects	Estimate (SE)	<i>t</i> (<i>df</i>)	р
Intercept	1.07 (.07)	14.94 (238.00)	<.001
Negative Affect	.17 (.01)	22.68 (9828.04)	<.001
Positive Affect	03 (.01)	-4.82 (9828.03)	<.001

	Negative Affect		
Fixed Effects	Estimate (SE)	t (df)	р
Intercept	11.57 (.07)	176.89 (237.96)	<.001
Emotional Eating Urges	.30 (.01)	23.01 (9841.04)	<.001

	Positive Affect		
Fixed Effects	Estimate (SE)	<i>t</i> (<i>df</i>)	р
Intercept	13.46 (.08)	173.10 (237.87)	<.001
Emotional Eating Urges	09 (.01)	-6.08 (9840.97)	<.001

Table 2

Prospective Models

	Next-Day Emotional Eating Urges		
Fixed Effects	Estimate (SE)	<i>t</i> (<i>df</i>)	p
Intercept	1.09 (.07)	15.46 (238.00)	<.001
Emotional Eating Urges	15 (.01)	-14.90 (8029.44)	<.001
Negative Affect	<.01 (.01)	.60 (6991.69)	.55
Positive Affect	.01 (.01)	1.52 (7509.58)	.13

	Next-Day Negative Affect		
Fixed Effects	Estimate (SE)	<i>t</i> (<i>df</i>)	р
Intercept	11.57 (.07)	176.68 (237.75)	<.001
Negative Affect	22 (.01)	-21.38 (7644.96)	<.001
Emotional Eating Urges	.04 (.01)	2.47 (8803.91)	.005

	Next-Day Positive Affect		
Fixed Effects	Estimate (SE)	t (df)	р
Intercept	13.45 (.08)	171.39 (237.78)	<.001
Positive Affect	.36 (.01)	36.74 (3918.82)	<.001
Emotional Eating Urges	.02 (.01)	1.14 (3419.05)	.25