Journal of Psychopathology and Clinical Science

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Online First Publication, April 27, 2023. https://dx.doi.org/10.1037/abn0000830

CITATION

Leenaerts, N., Vaessen, T., Sunaert, S., Ceccarini, J., & Vrieze, E. (2023, April 27). How Negative Affect Does and Does Not Lead to Binge Eating—The Importance of Craving and Negative Urgency in Bulimia Nervosa. *Journal of Psychopathology and Clinical Science*. Advance online publication. https://dx.doi.org/10.1037/abn0000830

AMERICAN PSYCHOLOGICAL ASSOCIATION

Journal of Psychopathology and Clinical Science

© 2023 American Psychological Association
ISSN: 2769-7541

https://doi.org/10.1037/abn0000830

How Negative Affect Does and Does Not Lead to Binge Eating—The Importance of Craving and Negative Urgency in Bulimia Nervosa

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Studies suggest that negative affect (NA) can trigger binge eating (BE) in patients with bulimia nervosa (BN). Important factors in this relation between NA and BE could be craving (an intense desire for a BE episode) and negative urgency (the tendency to act rashly when NA is high). Therefore, this study wants to firstly explore the relations between NA, craving, rash action, and BE in daily life and secondly whether craving and rash action mediate the relationship between NA and BE. A sample of 70 female patients with BN and 76 female healthy controls (HC) took part in an experience sampling study where they reported on momentary NA, craving, rash action, and eating behaviors in daily life in a burst-measurement design over a period of 12 months. Assessments occurred eight times a day on Thursdays, Fridays, and Saturdays in seven bursts of 3 weeks, all separated by 5-week periods of no assessment. First, NA predicted subsequent rash action in the whole sample but this was more pronounced in patients with BN. Second, NA predicted subsequent craving in patients with BN, but not in HC. Third, rash action and craving predicted subsequent BE in patients with BN. Fourth, NA had competing effects on eating in patients with BN, predicting subsequent BE through rash action and craving, but also predicting subsequent not eating. These results suggest that NA can lead to BE in daily life through rash action and craving, but that NA can also lead to dietary restriction.

General Scientific Summary

This study shows that, on some occasions, negative emotions make patients with bulimia nervosa (BN) act rashly and experience a greater desire to binge eat and this could lead to a binge eating episode. However, on other occasions, negative emotions could make patients with BN not eat at all.

Keywords: ecological momentary assessment, negative affect, negative urgency, craving, binge eating

Supplemental materials: https://doi.org/10.1037/abn0000830.supp

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The present study's design and analyses were not preregistered. However, consistent with the Transparency and Openness Promotion guidelines, the data and scripts that support the findings of this study are available at the Research Data Repository of the KU Leuven at https://rdr.kuleuven.be/dataset.xhtml?persistentId=doi:10.48804/QQNNHO. The results of the current study were previously presented at the conference of the Eating Disorders Research Society in 2022.

No other disclosures were reported.

The authors assert that all procedures contributing to this work comply with the ethical standards of the relevant national and institutional committees on human experimentation and with the Helsinki Declaration of 1975, as revised in 2008.

A C1 grant (ECA-D4671-C14/18/096) of the Special Research Fund KU Leuven to Vrieze and Ceccarini served as a PhD Scholarship for Nicolas Leenaerts. Jenny Ceccarini and Thomas Vaessen were supported by a postdoc grant from FWO (12R1619N and 1243620N). No other grant of any kind was received.

Nicolas Leenaerts had full access to all the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis. Concept and design: Nicolas Leenaerts, Elske Vrieze, and Jenny Ceccarini. Acquisition, analysis, or interpretation of data: Nicolas Leenaerts, Thomas Vaessen, and Elske Vrieze. Drafting of the manuscript: Nicolas Leenaerts. Critical revision of the manuscript for important intellectual content: Nicolas Leenaerts, Thomas Vaessen, Stefan Sunaert, Jenny Ceccarini, and Elske Vrieze. Obtained funding: Elske Vrieze and Jenny Ceccarini. Supervision: Elske Vrieze.

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Bulimia nervosa (BN) is a psychiatric disorder that is characterized by recurrent binge eating (BE) episodes, compensatory behaviors, and an excessive influence of body shape and weight on self-evaluation (American Psychiatric Association [APA], 2013). BE is defined as eating an amount of food that is definitively larger than what most people would eat under similar circumstances, combined with a feeling of loss of control (APA, 2013). Despite the availability of treatments for BN, such as cognitive behavioral therapy and interpersonal therapy, up to 60% of patients may not be able to stop BE after undergoing therapy (Hagan & Walsh, 2021; Linardon & Wade, 2018). More effective therapies are therefore needed, but a better understanding of the causes of BE is required in order to develop them.

Most recent theoretical models hypothesize that BE can be triggered by negative affect (NA), which is often defined as a feeling of "subjective distress and unpleasurable engagement" and subsumes several negative emotions such as sadness, anxiety, anger, loneliness, or guilt (Burton & Abbott, 2017; Watson et al., 1988). These models assume that patients binge eat to cope with NA, though different models propose different mechanisms. The escape theory suggests that BE provides an escape from NA by shifting the patient's focus to simpler actions and sensations (Heatherton & Baumeister, 1991). In the emotion regulation theory, BE is not thought to provide an escape from NA, but rather reduce it (Lacey et al., 1986). However, the trade-off theory posits that BE does not alleviate NA, but rather exchanges more aversive emotions (e.g., anger) for less aversive and therefore more tolerable emotions (e.g., guilt; Kenardy et al., 1996).

Some studies have investigated this hypothesis in a laboratory setting and report that inducing NA can indeed cause patients to have a BE episode (Agras & Telch, 1998; Cardi et al., 2015). However, the controlled nature of these studies raises the question of whether these results also apply to daily life. To explore this question, other studies have used the experience sampling method (ESM), also known as an ecological momentary assessment, where a participant's emotions, behavior, and context are repeatedly assessed in daily life (Shiffman et al., 2008). These studies do find that NA is higher before a BE episode than before a regular eating episode and that NA increases in the hours before a patient has a BE episode (Haedt-Matt & Keel, 2011; Mikhail, 2021). These studies also show that stressors involving interpersonal relations or negative self-evaluation are an important cause of the NA experienced before a BE episode and that some negative emotions (e.g., anger and guilt) are more closely linked to BE than others (Berg et al., 2013; Goldschmidt et al., 2014; Reichenberger et al., 2021). However, through which mechanisms NA then leads to BE is less clear.

Two factors that could be important in this relationship are craving and negative urgency. First, craving is often defined as "an intense and conscious desire for a specific substance," with some authors adding "while attempting to abstain" to the definition (van Lier et al., 2018). This conceptualizes craving as a construct with both a motivational component (i.e., the desire for a substance) and an inhibitory component (i.e., the attempt to abstain; van Lier et al., 2018). When it comes to food, craving is typically directed at particular kinds of food and can only be satisfied by the consumption of these items (Meule, 2020). Furthermore, patients can experience a distinct craving for a BE episode and plan these episodes well in advance (Ferriday & Brunstrom, 2011; Gluck et al., 2004; Manasse et al., 2020). Second, negative urgency is often described as a tendency to act rashly when NA is high (Sharma et al., 2014). It is one of several distinct personality traits that can give rise to impulsive-like behavior (Strickland & Johnson, 2021; Whiteside & Lynam, 2001). For example, a meta-analysis on self-report measures finds that three distinct traits can lead to impulsive-like behavior: positive emotionality (i.e., positive urgency, sensation seeking), disinhibition, and negative emotionality (i.e., negative urgency; Sharma et al., 2014).

Both craving and negative urgency are thought to be inherently associated with NA and BE.

When it comes to the craving, the addictive appetite model posits that NA is an important trigger for craving in patients and that higher levels of craving can lead to BE (van Lier et al., 2018). Indeed, after inducing NA in a laboratory, studies find that NA is positively related to craving for a BE episode in individuals who binge eat, but not in healthy controls (HC; Gluck et al., 2004). Also, studies using ESM report that average craving levels in daily life are associated with BE symptoms (Smith, Mason, Schaefer, et al., 2021).

When it comes to negative urgency, the acquired preparedness model posits that individuals high in negative urgency may have different learning experiences that involve BE and NA (Combs et al., 2010). Namely, the general tendency to act rashly when experiencing NA could also make them more likely to binge eat when experiencing NA, which could cause them to acquire the expectancy that BE alleviates NA. The risk and maintenance model for BN then proposes that subsequent elevations of NA could activate these expectancies, while also decreasing self-control due to the higher levels of negative urgency, making patients more likely to engage in BE (Pearson et al., 2015). Studies using self-report measures do find that patients with BN report higher levels of negative urgency and that this is predictive of increases in BE over time (Anestis et al., 2007; Claes et al., 2015). ESM studies also show that patients who display more negative urgency need less of an increase in NA to trigger BE (Fischer et al., 2018; Smith, Mason, Reilly, et al., 2021; Smith, Mason, Schaefer, et al., 2021).

However, these studies have their limitations. First, they typically look at craving and negative urgency on a trait level and do not investigate the momentary changes in the underlying emotions and behaviors. Recent studies have shown that it is possible to deconstruct negative urgency in daily life and directly investigate the relation between NA and rash action, but this has not yet been done in the context of eating disorders (Sperry et al., 2018, 2021). Second, they often fail to explore the relation between NA, craving, rash action, and BE in its entirety. Most studies either investigate how craving or rash actions are related to NA or how they are associated with BE. Furthermore, they usually focus on the role of either craving or rash action and do not study them together. However, studies on alcohol or substance use suggest that craving and rash action are not independent of each other. This is because they consistently report that individuals who display higher levels of negative urgency also experience more cravings when NA is high (Chester et al., 2016; Li et al., 2021).

Because of these limitations, the precise role of craving and rash action in the relationship between NA and BE remains unclear. This study explores their roles with ESM and repeatedly assesses NA, craving, rash action, and eating behaviors in patients with BN and HC in daily life. This makes it possible to investigate whether emotional and behavioral changes within a person at a previous assessment ($t-1$) predict emotions and behaviors at the current assessment

(t_0). This study then firstly explores the direct relations between individual emotions and behaviors, and secondly investigates whether craving and rash action mediate the relation between NA and BE. More specifically, this study explores the following hypotheses:

1. Within-person NA predicts subsequent craving for a BE episode in patients with BN, but not in HC.
2. Within-person NA predicts subsequent rash action in patients with BN and HC, but more so in patients with BN. In other words, patients with BN display higher levels of negative urgency in daily life than HC.
3. Within-person craving for a BE episode and within-person rash action predict subsequent BE in patients with BN.
4. Within-person NA predicts subsequent BE in patients with BN and this is mediated by within-person rash action and within-person craving.

These hypothesized relations can also be seen in Figure 1.

Method

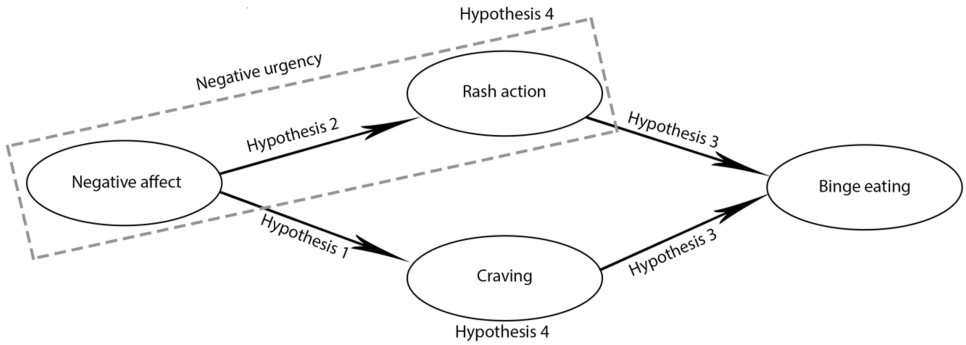
Study Sample

The participants were drawn from an ongoing ESM study. The study set out to include 70 HC and 70 patients with BN, which was based on recommendations for multilevel designs and previous dropout rates (Burke et al., 2017; Maas & Hox, 2005). Participant inclusion ran from September 2019 to February 2022. The participants were recruited in Flanders, Belgium, through residential and ambulatory care centers, patient groups, universities, social media, and by handing out flyers on the street. The inclusion criteria were the following: (a) female; (b) understand Dutch; (c) age $\geq$ 18 years; and (d) body mass index (BMI) $\geq$ 18.5 kg/m^2. Additional inclusion criteria for patients were: (e) meet the criteria for BN of the Diagnostic and Statistical Manual of Mental Disorders (5th ed.; APA, 2013); (f) illness duration $\leq$ 5 years. This maximum illness duration was set as the role of negative urgency is thought to be the largest in the first years after the onset of BN (Pearson et al., 2015). Participants were excluded for the following reasons: (a) major medical pathology; (b) chronic use of sedatives; (c) pregnancy; (d) presence of psychiatric pathology for HC or major psychiatric pathology (i.e., schizophrenia, autism spectrum disorder, bipolar disorder, or substance use disorder other than alcohol use disorder) for patients with BN. All participants gave their written consent, and the study was approved by the ethical committee of the UZ/KU Leuven.

Study Design

After an initial screening via telephone or email, potential participants attended an in-person assessment. Here, a resident of psychiatry confirmed an individual's eligibility to participate based on the in- and exclusion criteria. Afterward, the participants had their weight and height measured with a calibrated scale and stadiometer and completed clinical interviews and questionnaires. All participants underwent a briefing on the ESM questions and practiced the use of the mobile application. Then, participants entered the ESM protocol on the first Thursday after the in-person assessment. A visual representation of the protocol can be seen in Figure 2. It consisted of a repeated measurement design where seven bursts of data collection were spread out over a 12-month period. The bursts had a duration of 3 weeks and were separated by intervals of 5 weeks. During the bursts, data were only collected on Thursday, Friday, and Saturday to limit the protocol's impact on the participants. These specific days were selected to consecutively gather data on both week and weekend days. This resulted in 9 days of data collection per burst and 63 days in total. On a given day of data collection, participants received eight signals which were sent on a signal-contingent (i.e., semirandom) basis. This meant that

Figure 1
Study Hypotheses



Note. This study firstly explores the direct relations between negative affect (NA), craving, rash action and binge eating (BE), and secondly investigates whether craving and rash action mediate the relation between NA and BE. More specifically, this study explores the following hypotheses: (1) within-person NA predicts subsequent craving for a BE episode in patients with bulimia nervosa (BN), but not in healthy controls (HC). (2) within-person NA predicts subsequent rash action in patients with BN and HC, but more so in patients with BN. In other words, both patients with BN and HC display negative urgency in daily life, but this is more pronounced in patients with BN (3) within-person craving for a BE episode and within-person rash action predict subsequent BE in patients with BN. (4) within-person NA predicts subsequent BE in patients with BN and this is mediated by within-person rash action and within-person craving.

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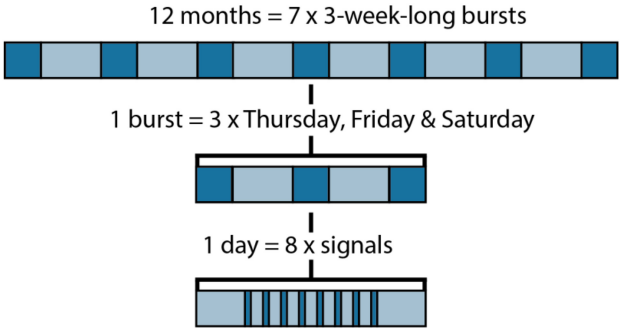
Figure 2

Experience Sampling Method Protocol



Note. The protocol consisted of seven bursts of data collection which were spread out over a 12-month period. The bursts had a duration of 3 weeks and were separated by intervals of 5 weeks. During the bursts, data were only collected on Thursday, Friday, and Saturday. On a given day of data collection, participants received eight signals which were sent on a signal-contingent (i.e., semirandom) basis. See the online article for the color version of this figure.

there were 72 signals scheduled per burst and 504 signals per participant. The number of days and signals per burst was similar to those in most cross-sectional ESM studies in patients with BN (Mikhail, 2021). The ESM data were initially collected with the app MobileQ (Meers et al., 2020). When the development of the app was discontinued in October 2020, data collection continued using m-Path (Mestdagh et al., 2022). More information about the apps can be found in eMethod 1 and eTable 1 in the online supplemental materials.

Measures

Baseline Measures

The Structured Clinical Interview for DSM-5 was used to confirm the diagnosis of BN and to screen for other psychiatric disorders (APA, 2017). Eating disorder severity was assessed using the Eating Disorder Examination Questionnaire (EDE-Q; Fairburn & Beglin, 1994). The EDE-Q had a good internal consistency with a Cronbach's alpha of .95.

ESM Measures

Negative Affect. Participants were asked to rate how much they agreed with feeling six emotions at the moment (afraid, lonely, insecure, sad, distressed, guilty) on a 7-point Likert scale (1: *totally disagree*; 7: *totally agree*). These scores were then averaged to get one score for NA at each assessment. The questions were based on previous ESM research investigating the role of NA in psychiatric disorders (Collip et al., 2011; Lataster et al., 2013; Rintala et al., 2020).

Rash Action. Participants needed to answer how much they agreed to have displayed five behaviors since the last prompt (doing something risky, without thinking, they will regret, that will get them into trouble, wish they had not done) on a 7-point Likert scale (1: *totally disagree*; 7: *totally agree*). The answers were then averaged to get one score for rash action. The questions were validated in previous ESM studies investigating rash action and were based on the UPPS-P Impulsive Behavior Scale (Sperry et al., 2018). Furthermore, they were used to deconstruct negative urgency in daily life and directly investigate the relation between NA and rash action (Sperry et al., 2021).

Craving. Participants were asked to rate their desire for a BE episode at the moment on a 5-point Likert scale (1: *none*; 5: *overwhelming*). This was based on previous ESM studies investigating cravings in eating disorders (Wonderlich et al., 2017).

Eating Behaviors. Participants needed to indicate if they had eaten since the last prompt. If so, they had to identify the eating event as undereating, normal eating, or overeating. Then, participants were asked if they experienced a loss of control over their eating behavior. The participants were trained to interpret undereating and overeating as eating an amount of food that is definitely smaller or larger than what most people would eat under similar circumstances. Based on previous studies, BE was defined as an episode of overeating with loss of control (Ambwani et al., 2015). More information on the ESM questions and the internal consistency of the ESM scales is found in eMethod 2, eMethod 3, and eTable 2 in the online supplemental materials.

Statistical Analysis

Sample Characteristics

If normally distributed, continuous variables were described by the mean and standard deviation. Otherwise, they were described by the median, first quartile, and third quartile. Count data were described by frequency and proportion. The 95% confidence intervals of the continuous, binomial, and multinomial variables were calculated with the CI, MedianCI, BinomCI, and MultinomCI functions in R, Version 4.1.1.

Data Characteristics

This study used the data collected up to July 2022. To measure the participants' compliance with the ESM protocol, per burst we calculated the percentage of answered signals out of the total number of signals received per burst by each participant who had not dropped out of the study. The compliance during the first burst was compared between the participant groups with a Wilcoxon rank-sum test. The compliance over the entire ESM protocol was evaluated with a linear mixed-effects model. This model included compliance as the outcome while the burst number and participant group were added as main and interaction effects. This made it possible to evaluate if there was a change in compliance over the bursts and if there was a difference between the groups. The model included random intercepts for the participants. The analyses were performed with PROC NPAR1WAY and PROC MIXED in SAS, Version 9.4.

Hypothesis Testing

For the first three hypotheses, three separate mixed models were fitted to the data to investigate the relations between NA, rash action, craving, and BE. These included an outcome at the current assessment (t_0) and a predictor at a previous assessment within the same day (t_{-1}), which was split into within- and between-person effects through person-mean centering. This made it possible to explore whether within-person deviations from the mean at t_{-1}

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predicted the outcome at t_0. All models included random intercepts for the participants as well as age, BMI, and the number of days since the participant started with the protocol as covariates. To test Hypothesis 1, a generalized linear mixed model was fitted to the data of the HC and patients with BN with maximum likelihood estimation. The model included craving for a BE episode as an outcome and NA as a predictor. Additionally, to compare patients with BN and HC, group was added as a main and interaction effect with the within-person predictor at t_{-1}. Due to the ordinal nature of the outcome, a multinomial distribution with a cumulative logit link was used. For Hypothesis 2, a linear mixed model was fit to the data of the HC and patients with BN with restricted maximum likelihood estimation. This model included rash action as an outcome and NA as a predictor. Group was also added as a main and interaction effect with the within-person predictor at t_{-1}. As mentioned previously, the relation between NA and rash action in this model can be seen as a measure of negative urgency (Sperry et al., 2021). To test Hypothesis 3, a generalized linear mixed model was fitted to the data of the patients with BN with maximum likelihood estimation. The model included BE as an outcome and NA, rash action, and craving as predictors. Due to the binary nature of the outcome, a binomial distribution with a logit link was used. The models were fit using PROC MIXED and PROC GLIMMIX in SAS, Version 9.4. The continuous variables in these models were standardized so that estimates can be interpreted as effect sizes. To deal with autocorrelation, an AR(1) covariance structure of the errors was assumed for the models of Hypotheses 2 and 3 (Allison, 2015). This was not possible for Hypothesis 1 as this was not implemented in SAS for ordinal outcomes. To test the robustness of the results, sensitivity analyses were performed that added compliance, treatment, app type, comorbidities, or medication use. *P* values below .05 were considered significant. All models were valid under a missing at-random assumption as they were fitted with different types of maximum likelihood estimation. The formulas for the models can be found in eMethod 4 in the online supplemental materials.

When it comes to Hypothesis 4, a 1-1-1 multilevel structural equation model (MSEM) was fitted to the data of the patients with BN. As in previous studies, a Bayesian estimation technique was used with noninformative priors, which can more easily estimate parameters and can result in more accurate estimates than frequentist approaches (Depaoli & Clifton, 2015; Smith, Mason, Reilly, et al., 2021). The MSEM jointly modeled the effect of NA on BE on a within-person and between-person level, though the hypothesis was tested on the within-person level. Here, the MSEM investigated whether NA at a previous assessment (t_{-1}) led to BE at the next assessment (t_{+1}) (i.e., total effect), whether this was mediated by rash action or craving at the current assessment (t_0; i.e., indirect effect) and whether NA at t_{-1} still led to BE at t_{+1} when controlled for rash action and craving at t_0 (i.e., direct effect). To deal with autocorrelation, a lagged outcome variable was included as a covariate in each of the separate regressions (Andersen & Mayerl, 2022). More information on how autocorrelation was handled in this study can be found in eMethod 5 in the online supplemental materials. A path diagram of the MSEM can be seen in Figure 3. The model was fit using the TWOLEVEL RANDOM procedure in Mplus Version 8.7. As a Bayesian approach was used, results were considered significant when the Bayesian credibility intervals did not include 0. The model was valid under a missing at random assumption as it was fitted with a Bayesian estimation approach. The data and scripts that support the findings of this study are available at the Research Data Repository of the KU Leuven at https://rdr.kuleuven.be/dataset.xhtml?persistentId=doi:10.48804/QQNNHO.

Results

Sample Characteristics

There were 146 study participants at the time of the analyses. This included 70 (47.9%) patients with BN and 76 (52.0%) HC. Their characteristics can be found in Table 1. More information can be found in eResults 1 in the online supplemental materials. There were no between-group differences when it comes to age, education, and ethnicity. However, the BMI of the patients with BN ($M = 25.1$; $SD = 5.30$, CI = 23.9, 26.4) was higher than that of the HC ($M = 22.3$; $SD = 2.22$; CI = 21.8, 22.8).

Data Characteristics

Data collection was completed for 115 (78.8%) participants with another 31 (21.2%) still needing to finish two bursts on average. A total of 28 (19.2%) participants (18 (25.7%) BN; 10 (13.2%) HC) dropped out of the study before the follow-up ended. The median compliance per participant during the first burst was 90.3% for the HC and 83.3% for the patients with BN. This is similar to the compliance rates of previous cross-sectional ESM studies in patients with an eating disorder (Fischer et al., 2018; Schaefer et al., 2020). The compliance during the first burst did not differ significantly between patients and controls ($z = -1.56$, $p = .119$). Compliance decreased over the course of the study in patients with BN ($\beta = -0.062$; $SE = 0.005$; CI = −0.072, −0.052, $p < .001$) and HC ($\beta = -0.034$; $SE = 0.005$; CI = −0.043, −0.025, $p < .001$), but this was more pronounced in patients with BN ($\beta = -0.028$; $SE = 0.007$; CI = −0.041, −0.015, $p \leq .001$). In total, the HC answered 23,168 (73.0%) of their scheduled beeps, while the patients with BN answered 16,222 (58.5%). Though no ESM studies of a similar length were performed in patients with an eating disorder, the overall compliance of this study fell in the range of the lengthier ESM studies on substance use (Jones et al., 2019). More information on the dropout and compliance rates as well as the average number of data points per burst can be found in eResults 1, eResults 2, and eTable 3 in the online supplemental materials.

Hypothesis Testing

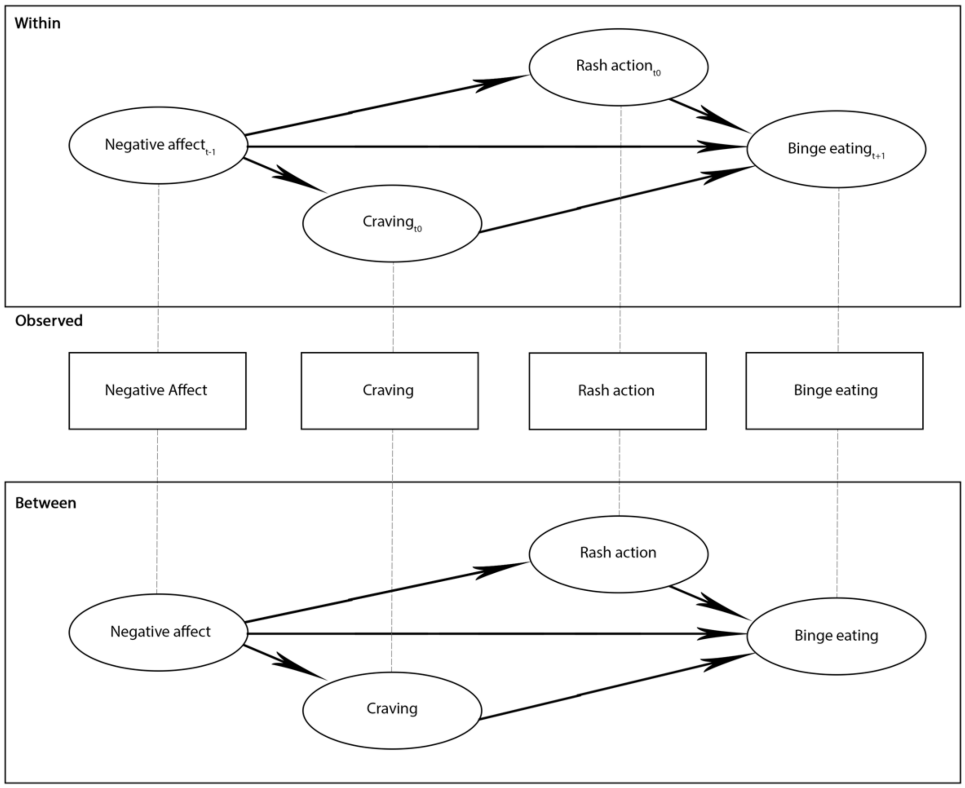
The results for Hypotheses 1–3 can be found in Table 2 and Figure 4. The inclusion of compliance, medication use, therapy, app type, or presence of comorbidities did not change the significance of these results. The results for Hypothesis 4 can be seen in Table 2 and Figure 5. The full results of all the statistical models can be seen in eTable 4 in the online supplemental materials.

Hypothesis 1 (Within-person NA predicts subsequent craving for a BE episode in patients with BN, but not in HC)

Higher levels of within-person NA at a previous assessment (t_{-1}) were associated with higher levels of craving at the current assessment (t_0) in patients with BN ($\beta = 0.128$; $SE = 0.019$; CI = 0.091,

Figure 3

Path Diagram of the 1-1-1 Multilevel Structural Equation Model to Explore Hypothesis 4 (Within-Person Negative Affect Predicts Binge Eating in Patients With Bulimia Nervosa and This Is Mediated by Rash Action and Craving)



0.165; $p < .001$), but not in HC ($\beta = 0.027$; $SE = 0.030$; CI = −0.033, 0.86; $p = .380$).

Hypothesis 2 (Within-person NA predicts subsequent rash action in patients with BN and HC, but more so in patients with BN)

Higher levels of within-person NA at t_{-1} were associated with higher levels of rash action at t_0 in HC ($\beta = 0.036$; $SE = 0.006$; CI = 0.025, 0.048, $p < .001$) and patients with BN ($\beta = 0.073$; $SE = 0.006$; CI = 0.062, 0.085, $p < .001$). Furthermore, there was a significant interaction effect where this relationship was more pronounced in patients with BN than in HC ($\beta = 0.036$; $SE = 0.006$; CI = 0.019, 0.52, $p = .001$). These results indicate that both patients with BN and HC display negative urgency in daily life, but that patients with BN display higher levels of negative urgency than HC.

Hypothesis 3 (Within-person craving and within-person rash action predict subsequent BE in patients with BN)

A higher probability of reporting a BE episode at t_0 was associated with higher levels of within-person rash action at t_{-1} ($\beta = 0.105$; $SE = 0.037$; CI = 0.033, 0.177; $p = .004$) and craving at t_{-1} ($\beta = 0.400$; $SE = 0.027$; CI = 0.349, 0.452; $p < .001$), but not with within-person NA at t_{-1} ($\beta = -0.026$; $SE = 0.033$; CI = −0.090, 0.083; $p = .431$).

Hypothesis 4 (Within-person NA predicts subsequent BE in patients with BN and this is mediated by within-person rash action and within-person craving)

At the within-person level, there was an indirect effect of NA at t_{-1} on BE at t_{+1} through rash action at t_0 ($\beta = 0.003$; $SD_{\text{posterior}} = 0.002$; CI = 0.001, 0.007) and craving at t_0 ($\beta = 0.006$; $SD_{\text{posterior}} = 0.002$; CI = 0.003, 0.011). There was no significant direct ($\beta = -0.013$; $SD_{\text{posterior}} = 0.022$; CI = −0.055, 0.032) or total effect ($\beta = -0.003$; $SD_{\text{posterior}} = 0.22$; CI = −0.046, 0.046) of NA at t_{-1} on BE at t_{+1}. The absence of a total effect of NA on BE does not invalidate the presence of indirect effects (Zhao et al., 2010). However, it could indicate that there are competing effects through which NA also lowers the probability of a BE episode.

Post Hoc Analyses

To explore possible competing effects, three post hoc analyses were performed. Their results can be seen in Table 2. Post hoc analysis 1 investigated whether NA can also predict not eating. This was done with a mixed-effects model that included eating (1: have been eating since the last beep; 0: having not eaten) as the outcome and the lagged within-person effect of NA as a fixed effect. In this model, higher levels of within-person NA at t_{-1} were associated with a lower probability of eating at t_0 in HC ($\beta = -0.035$; $SE = 0.016$; CI = −0.673, −0.003; $p = .032$) and patients with BN ($\beta = -0.043$; $SE = 0.018$;

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Table 1
Sample Characteristics

Variable	Healthy controls ($n = 76$)		Patients with BN ($n = 70$)	
	M (*SD*), *Mdn* (Q1–Q3) or No (%)	95% CI	*M* (*SD*), *Mdn* (Q1–Q3) or No (%)	95% CI
Age	21.7 (3.05)	[21.0, 22.4]	22.0 (3.87)	[21.1, 23.0]
BMI	22.3 (2.22)	[21.8, 22.8]	25.1 (5.30)	[23.9, 26.4]
Education (years)	15.0 (1.63)	[14.6, 15.3]	14.5 (2.14)	[14.0, 15.0]
Therapy (general)				
Past	23 (30%)	[20%, 41%]	50 (71%)	[61%, 82%]
Present	3 (4%)	[0%, 8%[	16 (23%)	[13%, 33%]
Therapy (ED)				
Past	0 (0%)	[0%, 0%]	26 (37%)	[26%, 49%]
Present	0 (0%)	[0%, 0%]	14 (20%)	[10%, 30%]
Ethnicity				
Caucasian	74 (97%)	[95%,100%]	63 (90%)	[84%, 96%]
Asian	1 (1%)	[0%, 4%]	3 (4%)	[0%, 11%]
Middle-Eastern	0 (0%)	[0%, 0%]	4 (6%)	[0%, 12%]
Mixed	1 (1%)	[0%, 4%]	0 (0%)	[0%, 0%]
Illness duration (years)	0 (0)	[0, 0]	2.55 (1.52)	[2.18, 2.91]
EDE-Q				
Restraint	0.54 (0.86)	[0.35, 0.74]	2.84 (1.50)	[2.48, 3.20]
Shape concern	1.10 (1.09)	[0.85, 1.34]	4.23 (1.43)	[3.89, 4.57]
Weight concern	0.87 (1.00)	[0.64, 1.10]	4.07 (1.60)	[3.69, 4.46]
Eating concern	0.25 (0.36)	[0.17, 0.33]	2.87 (1.45)	[2.52, 3.21]
Total	0.74 (0.77)	[0.57, 0.92]	3.60 (1.26)	[3.30, 3.90]
Eating disorder symptoms (days/4 weeks)				
Binge eating	0 (0)	[0, 0]	8.13 (6.79)	[6.51, 9.75]
Fasting	0 (0)	[0, 0]	7.81 (8.10)	[5.88, 9.74]
Vomiting	0 (0)	[0, 0]	2.11 (5.51)	[0.80, 3.43]
Laxative use	0 (0)	[0, 0]	0.31 (1.95)	[0, 0.78]
Diuretic use	0 (0)	[0, 0]	0.77 (4.45)	[0, 1.83]
Compensatory exercise	0 (0)	[0, 0]	6.53 (7.21)	[4.81, 8.25]
Psychoactive medication use	0 (0%)	[0%, 0%]	11 (16%)	[7%, 24%]
Comorbidities[a]				
MDD	0 (0%)	[0%, 0%]	9 (17%)	[6%, 32%]
AUD	0 (0%)	[0%, 0%]	19 (36%)	[25%, 51%]
PD	0 (0%)	[0%, 0%]	5 (9%)	[0%, 24%]
AP	0 (0%)	[0%, 0%]	4 (8%)	[0%, 22%]
SAD	0 (0%)	[0%, 0%]	6 (11%)	[0%, 26%]
PTSD	0 (0%)	[0%, 0%]	10 (19%)	[8%, 33%]
ESM measures				
NA	2.07 (1.02)	[2.06, 2.09]	3.13 (1.37)	[3.11, 3.15]
Rash action	1.56 (0.82)	[1.55, 1.57]	2.09 (1.07)	[2.07, 2.10]
Craving	1.10 (0.35)	[1.10, 1.11]	1.70 (1.04)	[1.68, 1.71]
BE[b]	0 (0–1)	[0, 0]	22 (7–41)	[14, 30]

Note. AP = agoraphobia; AUD = alcohol use disorder; BE = binge eating; BMI = body mass index; BN = bulimia nervosa; CI = confidence interval; ED = eating disorder; EDE-Q = Eating Disorder Examination Questionnaire; ESM = experience sampling method; Q1 = 25% quartile; Q3 = 75% quartile; MDD = major depressive disorder; n = number; NA = negative affect; PD = panic disorder; PTSD = posttraumatic stress disorder; SAD = social anxiety disorder. [a]Number and percentage of participants with certain comorbidity. [b]Binge eating episodes per participant.

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CI = −0.078, −0.008; $p = .015$). Post hoc analyses 2 and 3 wanted to explore whether NA does predict BE if its effect on eating is taken into consideration. In both analyses, a mixed model was constructed with BE as the outcome and the lagged within-person effect of NA as a predictor of interest. However, post hoc analysis 2 used all of the data of the patients with BN whereas post hoc analysis 3 only used the data when patients with BN indicated to have eaten. Here, higher levels of within-person NA at t_{-1} were not related to a higher probability of BE at t_0 in post hoc analysis 2 ($\beta = 0.055$; $SE = 0.031$; CI = −0.006, 0.115; $p = .079$), but there was a relation in post hoc analysis 3 ($\beta = 0.104$; $SE = 0.035$; CI = 0.035, 0.173; $p = .003$). This suggests that NA does predict BE when its competing effect on eating is taken into account.

Discussion

This study is the first to explore the mediating role of craving and rash action in the relationship between NA and BE in daily life. First, its results show that NA predicts craving and rash action in patients with BN, more so than in HC. Second, they suggest that NA can lead to BE in patients with BN through craving and rash action but that NA can also lead to not eating.

Table 2
Model Results

Hypothesis	Outcome	Variable	β	OR	*SE*/*SD*p	95% CI	*p*
1[a]	Craving t_0	Within-person NA t_{-1} (*HC*)	0.027	1.03	0.030	[−0.033, 0.086]	.380
		Within-person NA t_{-1} (*BN*)	0.128	1.14	0.019	[0.091, 0.165]	<.001
		Within-person NA t_{-1} (*BN vs. HC*)	0.102	1.11	0.036	[0.032 0.171]	.004
		Between-person NA	0.492	1.64	0.145	[0.206, 0.779]	.001
2[a]	Rash action t_0	Within-person NA t_{-1} (*HC*)	0.036		0.006	[0.025, 0.048]	<.001
		Within-person NA t_{-1} (*BN*)	0.073		0.006	[0.062, 0.085]	<.001
		Within-person NA t_{-1} (*BN vs. HC*)	0.036		0.006	[0.019, 0.052]	<.001
		Between-person NA	0.429		0.059	[0.312, 0.546]	<.001
3[b]	Binge eating t_0	Within-person NA t_{-1}	−0.026	0.97	0.033	[−0.090, 0.083]	.431
		Between-person NA	−0.142	0.87	0.130	[−0.404, 0.119]	.279
		Within-person rash action t_{-1}	0.105	1.11	0.037	[0.033, 0.177]	.004
		Between-person rash action	0.458	1.58	0.168	[0.123, 0.794]	.008
		Within-person craving t_{-1}	0.400	1.49	0.027	[0.349, 0.452]	<.001
		Between-person craving	0.504	1.65	0.100	[0.304, 0.703	<.001
4[b]	Binge eating t_{+1}	Direct effect (*within-person NA* t_{-1})	−0.013		0.022	[−0.055, 0.032]	
		Indirect effect (*within-person NA* t_{-1} → *within-person rash action* t_0)	0.003		0.002	[0.001, 0.007]	
		Indirect effect (*within-person NA* t_{-1} → *within-person craving* t_0)	0.006		0.002	[0.003, 0.011]	
		Total effect (*within-person NA* t_{-1})	−0.003		0.022	[−0.046, 0.046]	
Post hoc 1[a]	Eating t_0	Within-person NA t$_{-1}$ (*HC*)	−0.035	0.97	0.016	[−0.067, −0.003]	.032
		Within-person NA t_{-1} (*BN*)	−0.043	0.96	0.018	[−0.078, −0.008]	.015
		Between-person NA	0.053	1.06	0.039	[−0.025, 0.132]	.178
Post hoc 2[b]	Binge eating t_0	Within-person NA t_{-1}	0.055	1.06	0.031	[−0.006, 0.115]	.079
		Between-person NA	0.294	1.34	0.127	[0.040, 0.548]	.024
Post hoc 3[c]	Binge eating t_0	Within-person NA t_{-1}	0.104	1.11	0.035	[0.035, 0.173]	.003
		Between-person NA	0.294	1.34	0.137	[0.020, 0.568]	.036

Note. β = standardized estimate; BN = bulimia nervosa; CI = confidence interval/credibility interval; HC = healthy controls; NA = negative affect; *SD*p = standard deviation of the posterior; t_{-1} = previous assessment; t_0 = current assessment; t_{+1} = next assessment. [a]Based on the entire dataset. [b]Based on the data of the patients with BN. [c]Based on the data of the patients with BN when they indicated to have eaten.

Previous ESM studies report that NA is higher before a BE episode than before a regular eating episode and that NA increases in the hours before someone binge eats (Haedt-Matt & Keel, 2011; Mikhail, 2021). Based on these results, it is unexpected that the present study has not found that within-person NA at the previous assessment ($t-1$) predicts BE at the current assessment (t_0). However, we could identify nine other ESM studies that have performed the same analysis, and of these studies, only two have found a significant relationship (Ambwani et al., 2015; Fitzsimmons-Craft et al., 2016; Heron et al., 2014; Moskovich et al., 2019; Pearson et al., 2018; Smith et al., 2018, 2019; Smith, Mason, Juarascio, et al., 2020; Smith, Mason, Schaefer, et al., 2020). The findings of the current study suggest that this could be due to NA having competing effects on BE, meaning that NA could also lead to dietary restraint. Though most ESM research has focused on how NA leads to BE, this notion that NA can also result in dietary restraint has already been suggested by models such as the integrated cognitive model for eating disorders (Burton & Abbott, 2017).

This is supported by a previous ESM study which reports that NA has a positive indirect effect on BE through rumination, but after controlling for this effect, is associated with a lower probability of BE (Smith, Mason, Reilly, et al., 2021). Another study also finds that the induction of NA in individuals who binge eat can lead to both overeating and undereating (Russell et al., 2017). Furthermore, one study reports that patients with BN are less likely to choose high-fat food items, even when NA is high (Gianini et al., 2019). Likewise, studies in the general population show that NA is associated with both eating more and eating less (Torres & Nowson, 2007). Nevertheless, the number of studies investigating how NA leads to dietary restriction in patients with BN is limited. Therefore, to have a more complete understanding of how NA is related to disordered eating behaviors, future ESM studies should not only investigate how NA leads to BE, but also how it leads to dietary restriction.

On the one hand, NA could result in dietary restriction depending on its source and underlying emotions. Namely, some studies on healthy volunteers have shown that stressors causing strong physical responses (e.g., something threatening or frightening) are related to eating less (O'Connor et al., 2008). Furthermore, delivering a strong acute stressor reduces food intake in rats while repeatedly administering a mild stressor increases the intake of energy-dense food (Torres & Nowson, 2007). Also, studies on individuals who binge eat find that negative emotions such as feeling nervous or afraid are less related to BE than others (Schaefer et al., 2020). Future studies should therefore explore which stressors (interpersonal vs. physical, once vs. repeated, or mild vs. severe) and which emotions (e.g., down vs. nervous) lead to BE or dietary restriction in daily life.

On the other hand, NA could lead to BE through craving and a general tendency to act rashly, as proposed by the addictive appetite, acquired preparedness, and risk and maintenance models (Combs et al., 2010; Pearson et al., 2015; Treasure et al., 2018). In support of these models, this study is the first to show that patients with BN display a stronger relationship between NA and craving as well as between NA and rash action in daily life. It is also the first to show that NA has an indirect effect on BE through craving and rash actions in daily life. However, these findings leave several questions unanswered.

First, although our findings indicate that craving and rash action are both independent mediators of the relationship between NA and BE,

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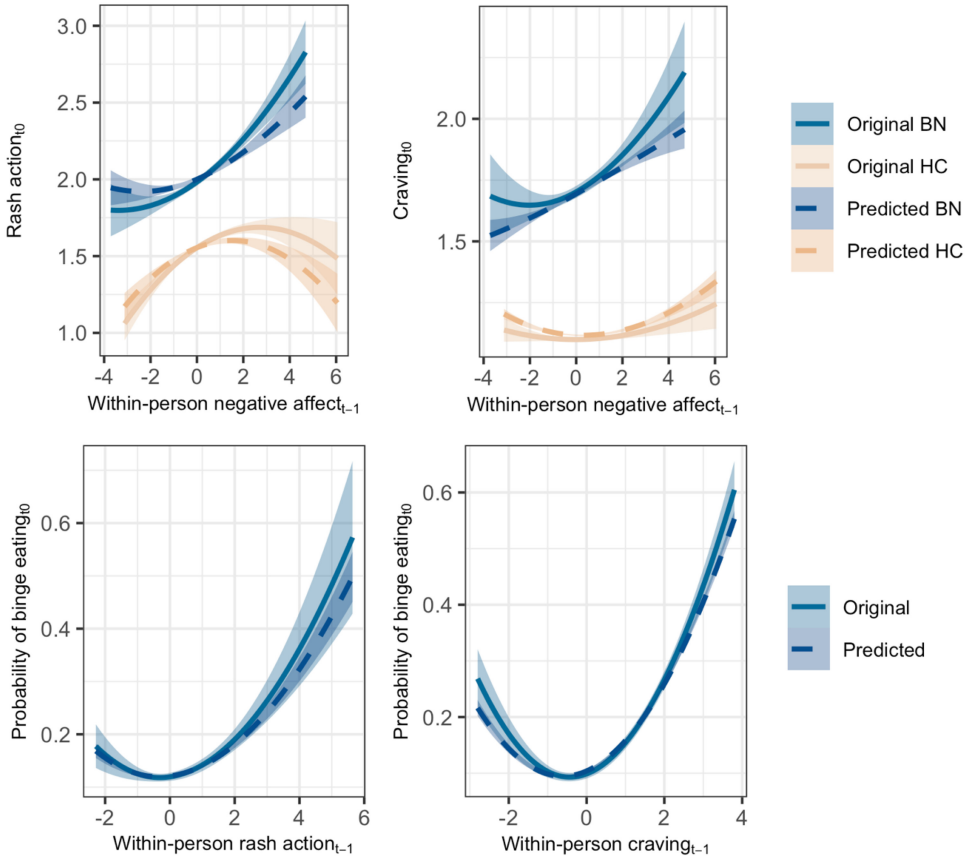
Figure 4

Smoothed Loess Curves for the Original Data as Well as the Data Predicted by the Models for Hypotheses 1, 2, and 3

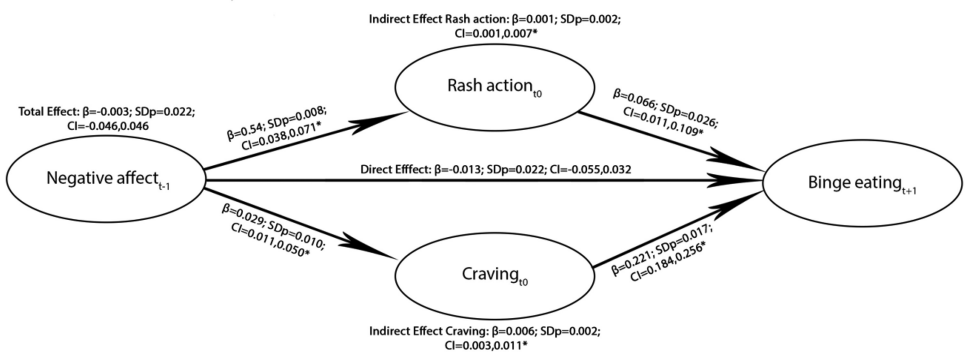


Note. BN = bulimia nervosa; HC = healthy controls; t_{-1} = previous assessment; t_0 = current assessment. See the online article for the color version of this figure.

it could be possible that their roles also interact. Namely, previous research on alcohol and substance use reports that individuals who score high on negative urgency also display more cravings when NA is elevated (Chester et al., 2016; Li et al., 2021). However, studies have also shown that not all BE episodes are the result of rash action; some are planned well in advance (Manasse et al.,

Figure 5

Within-Person Effects of the 1-1-1 Multilevel Mediation Model



Note. β = estimate; CI = 95% Bayesian credibility interval; *SD*p = standard deviation of the posterior. *Significant result.

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2020) Furthermore, patients may consume a substance without experiencing any craving at all (van Lier et al., 2018). Future studies should therefore explore the interdependence of craving and rash action in relation to BE.

Second, the results of this study raise the question how NA leads to craving and rash action in patients with BN. For craving, it has been suggested that the repeated use of BE episodes to manage NA could reinforce positive expectancies about food (e.g., that eating relieves NA; Schaefer et al., 2021). Subsequently, when patients experience NA, it could activate these positive expectancies and therefore increase the desire to binge eat (May et al., 2012). Indeed, ESM studies have found that stronger decreases in NA after a BE episode predict higher levels of eating expectancies and that NA is more strongly related to BE when eating expectancies are high (Schaefer et al., 2021; Smith, Mason, Juarascio, et al., 2020). However, whether this relation between eating expectancies and BE is mediated by craving has not yet been explored. For rash action, studies have shown that individuals with an eating disorder have a lower distress tolerance, meaning that NA is less bearable for them, which could urge them to act rashly to relieve NA (Corstorphine et al., 2007). Though studies using questionnaires do suggest that distress tolerance could mediate the relationship between NA and negative urgency on a trait level, whether it also mediates the relation between momentary changes in NA and rash action remains unclear (Barrios et al., 2022).

The results of this study could have important clinical implications. Namely, if it would be possible to prevent NA from leading to higher levels of rash action and craving, then it might be possible to prevent a BE episode from happening. Current treatments for BN typically do not target this relationship, but a few interventions focusing on food-related rash action and craving have been developed (I˙nce et al., 2021; Rebello & Greenway, 2016). The IMPULS and ImpulsE trials have focused on reducing food-related response inhibition and report reductions in BE that lasted longer than in the treatment as usual group (Preuss et al., 2017; Schag et al., 2019). A virtual reality treatment for reducing food craving reports higher abstinence rates than additional cognitive behavioral therapy in patients who did not respond to an initial program (Ferrer-García et al., 2017). These results are encouraging, but other interventions focusing on rash action or craving have shown no effect on BE frequency (I˙nce et al., 2021). These mixed findings could be the result of insufficient knowledge of how NA leads to rash action and craving and how craving and rash action interact. Future studies could significantly expand our understanding by exploring these relations and whether they can be changed by interventions.

This study has several limitations. First, the sample of patients with BN mostly consists of female participants with a short illness duration who compensate through fasting and excessive exercise. The limited compensation through purging could be due to most participants having a short illness duration and being nontreatment-seeking. This limits the generalizability of the results to all patients with BN. Future studies should therefore investigate the relationship between NA, craving, and rash action in other samples of patients with BN. Second, the use of some ESM measures could impact the measurement of behaviors and cognitions in this study. For example, this study has chosen to define craving as a desire for a BE episode, but craving can also be conceptualized as a desire for food in general (Smith, Mason, Schaefer, et al., 2021). The definition of the current study has several implications as the HC group does not engage in BE. Namely, it is a reason why we have hypothesized that HC do not display a relation between NA and craving (Gluck et al., 2004). Future studies should compare the relationship between NA and a more general concept of food cravings in patients with BN and HC. Also, this study does not assess negative emotions such as anger, which have been linked to BE (Reichenberger et al., 2021). Additionally, this study does not include questions about dietary restriction, so it investigates the relationship between NA and not eating. This is not ideal, as undereating can also be a sign of dietary restriction, and there may be other reasons why patients do not eat. For example, an individual could be ill and therefore experience more NA and be less inclined to eat, but the NA might not be the reason why they are not eating. Third, the decreasing compliance over the duration of the study could impact the results due to the missing data. However, the techniques used in this study are valid under a missing at random assumption and a sensitivity analysis finds no impact of compliance on the results. Fourth, limiting the ESM measurements to Thursday, Friday, and Saturday could have influenced the results if participants would experience a different relation between NA, craving, rash action, and BE on the other days of the week. This study also has several strengths. This ESM dataset is the largest in patients with BN when it comes to the number of signals and the second largest when it comes to the number of patients (Mikhail, 2021). Furthermore, it is the first to show that NA predicts rash action and craving in patients with BN in daily life and that this relationship is stronger than in HC. It is also the first to show that rash action and craving mediate the relationship between NA and BE in daily life. Importantly, they highlight that NA can lead to both BE and not eating in patients with BN. This could be an important reason why previous studies often do not find a relation between NA at the previous assessment (t_{-1}) and BE at the current assessment (t_0).

Conclusion

This study is the first to show that NA predicts subsequent craving and rash action in patients with BN, more so than in HC. It is also the first to show that NA predicts subsequent BE through craving and rash action, but that NA can also predict subsequent not eating. Future studies should explore how NA leads to rash action and craving, how craving and rash action interact and investigate when NA leads to BE and when NA leads to dietary restriction.

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Received September 3, 2022
Revision received January 24, 2023
Accepted February 1, 2023 ■

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