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# Predictors of neurofeedback treatment outcome in binge-eating disorder: An exploratory study

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## Abstract

**Objective:** Knowledge on predictors for treatment response to psychotherapy in binge-eating disorder (BED) is mixed and not yet available for increasingly popular neurofeedback (NF) treatment targeting self-regulation of aberrant brain activity. This study examined eating disorder- and psychopathology-related predictors for NF treatment success in BED.

**Method:** Patients with BED ($N = 78$) were randomized to 12 sessions of real-time functional near-infrared spectroscopy (rtfNIRS)-NF, targeting individual prefrontal cortex signal up-regulation, electroencephalography (EEG)-NF, targeting down-regulation of fronto-central beta activity, or waitlist (WL). The few studies assessing predictors for clinical outcomes after NF and evidenced predictors for psychotherapy guided the selection of baseline eating disorder-related predictors, including objective binge-eating (OBE) frequency, eating disorder psychopathology (EDP), food cravings, and body mass index (BMI), and general psychopathology-related predictors, including depressive and anxiety symptoms, impulsivity, emotion dysregulation, and self-efficacy. These questionnaire-based or objectively assessed (BMI) predictors were regressed on outcomes OBE frequency and EDP as key features of BED at post-treatment (t1) and 6-month follow-up (t2) in preregistered generalized mixed models (https://osf.io/4aktp).

**Results:** Higher EDP, food cravings, and BMI predicted worse outcomes across all groups at t1 and t2. General psychopathology-related predictors did not predict outcomes at t1 and t2. Explorative analyses indicated that lower OBE frequency and higher self-efficacy predicted lower OBE frequency, and lower EDP predicted lower EDP after the waiting period in WL.

**Discussion:** Consistent with findings for psychotherapy, higher eating disorder-related predictors were associated with higher EDP and OBE frequency. The specificity of psychopathological predictors for NF treatment success warrants further examination.

**Public Significance:** This exploratory study firstly assessed eating disorder- and psychopathology-related predictors for neurofeedback treatment outcome in binge-eating disorder and overweight. Findings showed an association between

higher eating disorder symptoms and worse neurofeedback outcomes, indicating special needs to be considered in neurofeedback treatment for patients with a higher binge-eating disorder symptom burden. In general, outcomes and assignment to neurofeedback treatment may be improved upon consideration of baseline psychological variables.

**KEYWORDS**
binge-eating disorder, binge-eating frequency, body mass index, eating disorder psychopathology, electroencephalography, food craving, functional near-infrared spectroscopy, neurofeedback, predictor, self-efficacy

## 1 | INTRODUCTION

Binge-eating disorder (BED), characterized by recurrent binge-eating episodes without inappropriate weight control behavior (American Psychiatric Association, 2013), is the most prevalent eating disorder, with European lifetime prevalence estimates between 1% and 4% (Keski-Rahkonen & Mustelin, 2016). BED is associated with obesity, physical and mental disorder comorbidity as well as eating disorder and general psychopathology (Udo & Grilo, 2019; Wilfley et al., 2016). Extant cross-sectional and longitudinal self-report and behavioral studies linked BED to emotion dysregulation (Mikhail et al., 2020; Prefit et al., 2019) and trait (Gerlach et al., 2015) and food-specific impulsivity (Kittel et al., 2015, 2017; Svaldi et al., 2014) relative to weight-matched and normal-weight controls (Lavagnino et al., 2016; Leehr et al., 2018; Svaldi et al., 2014). Accordingly, alterations in the prefrontal cortex (PFC), a brain area subserving cognitive control and affective functioning essential for behavioral and cognitive aspects of dietary self-regulation, were described in BED and obesity relative to groups with normal weight (Giel et al., 2022; Lowe et al., 2019), in particular toward food stimuli (Rösch et al., 2021; Veit et al., 2021). Mostly based on functional magnet resonance imaging (fMRI), previous research indicated the diminished recruitment of the dorsolateral prefrontal cortex (DLPFC) and the inferior frontal gyrus (IFG) as key hubs of the prefrontal control network in BED and obesity compared with normal weight (Giel et al., 2022; Lowe et al., 2019). These studies were recently complemented by electroencephalography (EEG)-based reports of elevated fronto-central beta activity in the resting state and in response to food cues in BED and obesity versus obesity only and versus normal weight (Blume et al., 2019).

These findings have stimulated the development of neurofeedback (NF), a novel treatment approach uniquely seeking to train patients to gain control over brain pathways underlying dysfunctional eating behaviors (Bartholdy et al., 2013; Forcano et al., 2018) in the hopes that the alteration of brain activity produces clinical symptom relief. Borrowing elements from cognitive and neuromodulatory treatments, NF possibly fills the apparent treatment gaps left by the most common psychological treatments for EDs (Bartholdy et al., 2013). Specifically, NF permits a detailed user-specific analysis to empower cortical activation necessary for food-related self-regulation and/or to target brain signals which are (re-) activated during the occurrence of specific symptoms (Arns et al., 2017), such as the putatively deactivated PFC signals during binge-eating episodes (Berner et al., 2022). The resulting changes may cause structural changes in the brain, with accompanying symptom improvements (Krell et al., 2019). NF as a treatment adjunct could further enhance learning experiences (e.g., functional regulation strategies) acquired during traditional treatments (MacDuffie et al., 2018; Unterrainer et al., 2014) and benefit patients through the observation of neuronal changes during psychological treatments (Adcock et al., 2005). Pioneering NF studies indeed showed reductions in binge-eating or overeating episodes, food cravings, anxiety symptoms, body mass index (BMI), or hunger ratings after food-specific NF in BED (Blume et al., 2022; Hilbert et al., 2023), individuals with overweight and obesity (Kohl et al., 2019), and restrained eaters (J. Schmidt & Martin, 2015, 2016, 2020). Most of these latter studies used EEG as a well-established NF technique, which, inter alia, aimed to down-regulate high beta activity linked to increased food cue awareness.

NF studies recently tried to overcome the apparent drawbacks of EEG-NF (e.g., its limited spatial resolution) through real-time functional near-infrared spectroscopy (rtfNIRS) as a more patient-friendly compromise regarding temporal and spatial resolution that uniquely targets the up-regulation of PFC signals associated with cognitive control over foods (Soekadar et al., 2021; Val-Laillet et al., 2015). In this context, the present study is a secondary analysis of a randomized-controlled trial in BED that documented decreases in binge-eating episodes and improvements in secondary symptoms and executive functions after rtfNIRS-NF and EEG-NF at post-treatment and 6-month-follow-up (Hilbert et al., 2023). Both NF paradigms outperformed waitlist (WL) in reducing food cravings ($\beta = -0.42$), anxiety symptoms ($\beta = 0.47$), and BMI ($\beta = 0.12$), but co-occurring highly favorable changes in WL deserve mention. Of note is that only one third of patients with BED abstained from binge eating after food-specific EEG-NF (Blume et al., 2022) and rtfNIRS-NF (Hilbert et al., 2023) at post-treatment, but further improvement to ≥50% was found at follow-up, indicating an apparent variability in NF treatment response. Regarding comparative assessment, rtfNIRS-NF versus EEG-NF tended to improve eating disorder psychopathology (EDP), and EEG-NF versus rtfNIRS-NF tended to ameliorate general mental health.

Regarding predictors, the only study assessing predictors for NF outcomes in eating disturbances found a non-significant trend for lower somatic self-efficacy to predict more post-treatment overeating tendencies after 10 EEG-NF sessions in female restrained eaters (J. Schmidt & Martin, 2020). Likewise, a systematic review (Linden, 2014) suggested a predictive role of higher self-efficacy for NF success in depression. EEG-NF studies in adults and children with attention-deficit/hyperactivity disorder (ADHD), paralleling BED in impulsive behavior (Nickel et al., 2019), identified a lower baseline symptom severity as predictor for beneficial treatment outcomes, including higher remission rates (Goth, 2006; Krepel et al., 2020). Moreover, more anxiety and depressive symptoms, and higher susceptibility to anger and lower attention abilities as proxies for emotion regulation and impulsivity, were linked to worse NF brain-based learning outcomes across diverse populations and paradigms in a systematic literature review (Kadosh & Staunton, 2019). Age, sex, and education did not predict favorable symptom outcomes according to another systematic review including EEG- and fMRI-NF studies in mental disorders (e.g., ADHD) or epilepsy (Weber et al., 2020).

In contrast to the scarce evidence on predictors for NF outcomes in general and specifically for NF in the context of eating disorders, ample studies examined predictors for psychological treatments, but remained largely inconclusive. A higher symptom severity was most consistently linked to impaired success of psychological treatments for BED in diverse formats (Haynos et al., 2021; Lydecker, Ivezaj, & Grilo, 2020; Vall & Wade, 2015). Better emotion regulation, higher self-efficacy, and a higher age were anecdotally associated with favorable treatment outcomes for BED after diverse psychological treatments (Anderson et al., 2020; Grilo et al., 2021; Thompson-Brenner et al., 2013; Vall & Wade, 2015). In contrast, findings were controversial regarding the predictive role of comorbid psychopathology and depression (Castellini et al., 2011; Dingemans et al., 2020; Lydecker & Grilo, 2021; Serra et al., 2020; Vall & Wade, 2015; Wilson et al., 2010), impulsivity (Boswell et al., 2021; Castellini et al., 2011; Manasse et al., 2016), BMI (Dingemans et al., 2020; Grilo et al., 2012, 2021; Thompson-Brenner et al., 2013), education (Dingemans et al., 2020; Grilo et al., 2012, 2021; Thompson-Brenner et al., 2013; Wilson et al., 2010), and sex (Dingemans et al., 2020; Grilo et al., 2012, 2021) for outcomes following diverse psychological treatments of BED.

In summary, there is an important knowledge gap regarding psychological pretreatment predictors for favorable treatment response in BED, in particular for NF studies. This pre-registered exploratory study (Rösch et al., 2022) sought to examine the relationship between baseline psychological variables and NF treatment success based on a randomized-controlled NF trial for BED (Hilbert et al., 2023), offering 12 sessions of rtNIRS-NF, EEG-NF, or delayed rtfNIRS-NF following WL or to patients with BED. Eating disorder-related predictors (i.e., objective binge-eating [OBE] frequency, EDP, food cravings, and BMI), general psychopathology-related predictors (i.e., depressive and anxiety symptoms, impulsivity, emotion dysregulation and self-efficacy), were considered for NF treatment success, defined by reductions in OBE frequency and EDP assessed at post-treatment (t1; 8 weeks after randomization) and 6-month follow-up (t2; Table 1). Age and sex were control variables across predictors against possible effects on treatment outcomes (Grilo et al., 2021; Lydecker, Gueorguieva, et al., 2020). Differences in predictors for success of rtfNIRS-NF versus EEG-NF were considered through interactions between all predictors and the NF group.

## 2 | METHOD

### 2.1 | Design and patients

$N = 78$ patients with BED provided written informed consent to participate in the study "Near Infrared Spectroscopy Neurofeedback for Binge-Eating Disorder and Obesity" (Hilbert et al., 2023), approved by the Ethical Committee of the University of Leipzig (476/17-ek). A diagnosis of BED or BED of low frequency and/or limited duration (i.e., meeting all criteria for BED except that the binge eating occurs, on average, less than once a week and/or for less than 3 months) according to the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (American Psychiatric Association, 2013) was required for inclusion. Furthermore, inclusion criteria encompassed 25 kg/m$^2$ ≤ BMI < 45 kg/m$^2$ (to prevent variability caused by heterogeneous weight status) and completion of ≥ 6 NF sessions, building on the well-established dose–response effect in psychotherapy for mental disorders (Howard et al., 1986; Steenbarger, 1994). A minimum number of sessions was chosen considering that higher intense treatments are likely suited to initiate treatment response in patients with BED (Chyurlia et al., 2019; Iacovino et al., 2012). Exclusion of $n = 10$ patients who did not finalize ≥ 6 NF sessions (rtfNIRS-NF, $n = 6$, [$n = 3$ programming problems, $n = 2$ COVID-19, $n = 1$ other reasons]; EEG-NF, $n = 4$ [$n = 2$ COVID-19, $n = 2$ other reasons]) yielded an analysis set of $N = 43$ patients with BED. Exclusion criteria included serious somatic (e.g., stroke) and/or mental (e.g., substance use disorder) disorders, prior or planned bariatric surgery, ongoing eating behavior-related psychotherapy, pregnancy or lactating, impediments in hearing, vision, or language possibly affecting testing, and medication with an effect on weight or executive functioning (e.g., antipsychotics), unless stable. Recruitment encompassed an in-house research database, information events for a behavioral weight loss treatment program at the Obesity Outpatient Unit at Leipzig University Medical Center, and the population (i.e., Internet advertisements).

### 2.2 | Procedure

At t0, patients provided information regarding age and sex, were interviewed by trained master- or doctoral-level research assistants with the Eating Disorder Examination (EDE; Fairburn et al., 2008; Hilbert & Tuschen-Caffier, 2016b) to derive BED diagnosis, and underwent an objective measurement of weight and height to derive BMI (kg/m$^2$). They filled in validated questionnaires on eating disorder- and psychopathology-related variables. After ascertaining eligibility,

**TABLE 1** Overview of variables.

| Construct | Measure | Computation | Assessment | Cronbach's $\alpha$ at t0[a] | McDonald's $\omega$ at t0[a] |
|---|---|---|---|---|---|
| *Eating disorder-related variables* | | | | | |
| Binge-eating frequency | EDE | Cumulative number of objective binge-eating episodes over the past 28 days | t0, t1, t2 | | |
| Eating disorder psychopathology | EDE-Q | Mean global score (range 0–6*) | t0, t1, t2 | 0.88 | 0.88 |
| Food cravings | FCQ-T-r | Sum score (range 15–75*) | t0 | 0.90 | 0.91 |
| Body mass index | BMI | Calculated from objectively measured height and weight (kg/m$^2$) | t0 | | |
| *Psychopathology-related predictors* | | | | | |
| Depressive symptoms | PHQ-D | Sum score (range 0–27*) | t0 | 0.77 | 0.78 |
| Anxiety symptoms | GAD-7 | Sum score (range 0–21*) | t0 | 0.86 | 0.85 |
| Impulsivity | BIS/BAS | Mean scores for the BIS and the BAS (range 1–4*) | t0 | BIS: 0.76 | BIS: 0.78 |
| | | | | BAS: 0.71 | BAS: 0.73 |
| Difficulties in emotion regulation | DERS | Global sum score (range 36–180*) | t0 | 0.94 | 0.94 |
| Self-efficacy | GSES | Sum score (range 10* – 40) | t0 | 0.89 | 0.90 |
| *Demographic control variables* | | | | | |
| Age | Questionnaire | Years | t0 | | |
| Sex | Questionnaire | Male, female | t0 | | |

*Note*: Less favorable scores are asterisked.
Abbreviations: BIS/BAS, Behavioral Inhibition/Behavioral Approach System Scales; DERS, Difficulties in Emotion regulation Scale; EDE, Eating Disorder Examination; EDE-Q, Eating Disorder Examination-Questionnaire; FCQ-T-r, Food Cravings Questionnaire – Trait reduced; GAD-7, Generalized Anxiety Disorder 7-Item Scale; GSES, General Self-Efficacy Scale; PHQ-D, Patient Health Questionnaire-Depression Scale; t0, baseline; t1, 8 weeks after randomization; t2, 6-month follow up after treatment.
[a]Based on the sample of $N = 41$ patients randomized to rtfNIRS-NF or EEG-NF who provided valid data at t0.

patients with BED were randomized to 12 sessions of rtfNIRS-NF or EEG-NF or WL, in which patients underwent rtfNIRS-NF after an 8-week waiting period. Additional assessments were at t1 and t2.

## 2.3 | Treatment

Food-specific NF treatment was described previously (Hilbert et al., 2023). In brief, rtfNIRS- and EEG-NF comprised 12 individual sessions provided over 8 weeks. Feedback stimuli included 12 personally appetitive food pictures based on a rating task at t0. Trained PhD or master's-level clinicians receiving regular supervision by AH implemented the rtfNIRS- and EEG-NF sessions. EEG-NF was derived from four electrodes (Cz, Fz, Fc1, Fc2) and targeted BED-specific increased awareness and attentional bias toward food-specific stimuli through the down-regulation of high beta activity in fronto-central areas during regulation versus preceding baseline trials. Patients were instructed to decrease a bar displayed on the screen, showing their neural activity, below a line, corresponding to their baseline, after they were presented personally appetizing food pictures. rtfNIRS-NF aimed to increase patients' ability to exert voluntary control over food stimuli through the up-regulation of neural activity in an individually selected region of interest in the PFC compared with passive watching of food pictures (Supporting Information Methods in Data S1). Patients in rtfNIRS-NF were instructed to minimize the picture size of the personally appetitive food picture on the screen, corresponding to their neural activity, with decreasing picture sizes reflecting increasing activity.

## 2.4 | Measures

Table 1 provides an overview of measures.

### 2.4.1 | Outcomes

The German version of the Eating Disorder Examination (Fairburn et al., 2008; Hilbert & Tuschen-Caffier, 2016b), conducted by trained interviewers blind to randomization at t0, t1, and t2, was used to assess OBE frequency, defined as the cumulative number of OBEs over the past 28 days.

EDP at t0, t1, and t2, was assessed through the German version of the EDE-Questionnaire (EDE-Q; Fairburn & Beglin, 2008; Hilbert &

Tuschen-Caffier, 2016a). The frequency with which a statement was endorsed in the past 28 days was assessed through 22 of the 28 items, rated on a 7-point Likert scale ranging from 0 = *never* or *not at all* to 6 = *always* or *to an extreme degree*. Higher global mean scores of these 22 items indicated higher EDP.

### 2.4.2 | Predictors

Predictors at t0, measured via patient-report questionnaires, included food cravings (Food Cravings Questionnaire-Trait Reduced [FCQ-T-r]; Meule et al., 2014), current symptoms of depression (9-item Patient Health Questionnaire [PHQ-D]; Gräfe et al., 2004; Spitzer et al., 1999) and anxiety (7-item Generalized Anxiety Disorder scale [GAD-7]; Löwe et al., 2008; Spitzer et al., 2006), impulsivity (German Version of the 24-item Behavioral Inhibition System/Behavioral Activation System [BIS/BAS] scales (Carver & White, 1994; Müller et al., 2013; Strobel et al., 2006), emotion dysregulation (Difficulties in Emotion Regulation Questionnaire [DERS]; Gratz & Roemer, 2004), and self-efficacy (Generalized Self-Efficacy Scale [GSES]; Schwarzer & Jerusalem, 1995).

## 2.5 | Statistical analysis

All analyses were pre-registered (Rösch et al., 2022; Supporting Information Materials for deviations in Data S1) and performed using R version 4.0.2 (R Core Team, 2020) with a two-tailed $\alpha < .05$. Multiplicity adjustment was not applicable in this exploratory study (Bender & Lange, 2001; Rubin, 2017). Continuous predictor variables were mean-centered, the dichotomous predictor sex was dummy-coded −0.5 and +0.5 based on recommendations before creating interaction terms (Kraemer et al., 2002). Univariate outliers were defined as observations ±3 *SD* of the mean and were winsorized to the respective upper or lower boundary of 3 *SD* ($n = 4$ values: $n = 1$ for OBE frequency at t0 and t1, respectively; and $n = 2$ for OBE frequency at t2).

Baseline predictors for OBE frequency at t1 and t2 were determined using generalized linear models, modeled as negative binomial with a log link. Baseline predictors for EDP at t1 and t2 were assessed via linear mixed models, assuming a Gaussian response and an identity function. Both model sets included a Predictor × Group interaction term, a Predictor × Group Mean interaction term to examine differential predictors between rtfNIRS-NF and EEG-NF and differential predictors depending on differing NF groups' baseline levels, and covariates age, sex, and the baseline value of the respective outcome. Additional analyses included patients in WL while accounting for differences in baseline diagnosis, and determined general predictors for symptom change over time in WL (Supporting Information Materials in Data S1).

All models were simplified via backward stepwise elimination (Table S3), retaining a random intercept for patient. Results apply to t1 and t2. Across models, collinearity between predictors was calculated by the variance inflation factors via the vif function of the car package (Fox & Weisberg, 2019). For negative binomial models (OBE frequency), incidence rate ratios (IRRs), and $\chi^2$ values for main effects of ANOVA type III were reported based on the ANOVA function of the car package (Fox & Weisberg, 2019). For linear mixed models (EDP), *F*-values, degrees of freedom and mean squared errors were calculated using the Satterthwaite method via the anova function of the lmerTest package (Kuznetsova et al., 2017). If analyses indicated significant interactions, post hoc analyses were conducted, estimating marginal means, and comparing simple slopes of covariates between groups using the emtrends function of the emmeans package (Lenth, 2022).

As measures of effect size, model-explained variance was reported, specifically, marginal $R^2_m$, explaining the variance accounted for by fixed effects in the model (small, $R^2_m \geq .02$, medium, $R^2_m \geq .13$, large, $R^2_m \geq .26$ [Cohen, 1988; Sotirchos et al., 2019; S. Nakagawa, personal communication, April 1, 2022]), and conditional $R^2_c$, explaining the joint variance of fixed and random effects (Johnson, 2014; Nakagawa & Schielzeth, 2013). In addition, semi-partial *r* (Jaeger, 2017) was reported and interpreted relying on common benchmarks of 0.10, 0.30, and 0.50, representing small, medium, and large effects (Cohen, 1988).

# 3 | RESULTS

## 3.1 | Descriptives

At t0, the analysis set ($N = 43$, rtfNIRS-NF $n = 22$, EEG-NF $n = 21$) was predominantly female ($n = 36$, 84%), had a mean age of 47.56 years ($SD = 13.31$), and mostly had obesity ($n = 37$, 86%). The rtfNIRS-NF and EEG-NF groups did not differ significantly on sociodemographic and clinical baseline characteristics (Table 2).

## 3.2 | Predictors of OBE frequency: Eating disorder-related predictors

Food cravings were a small-sized, $\chi^2(1) = 11.02$, IRR = 1.08, 95% CI [1.02, 1.09], $rs = 0.15$, $p = .001$, and BMI was a less-than-small-sized positive predictor for OBE frequency at t1 and t2, $\chi^2(1) = 5.47$, IRR = 1.05, 95% CI [1.01, 1.15], $rs = 0.09$, $p = .015$. No further predictors or interactions were found ($R^2_m = 0.292$, $R^2_c = 0.506$; large effect).

## 3.3 | Predictors of OBE frequency: Psychopathology-related predictors

The covariates higher OBE frequency, $\chi^2(1) = 5.90$, IRR = 1.17, 95% CI [1.03, 1.33], $rs = .069$, $p = .015$, and lower age, $\chi^2(1) = 5.55$, IRR = 0.97, 95% CI [0.95, 0.99], $rs = .08$, $p = .003$, were less-than-small-sized predictors for higher OBE frequency at t1 and t2

**TABLE 2** Descriptive characteristics of study patients.

| Study characteristics | Total ($n = 43$) | rtfNIRS-NF ($n = 22$) | EEG-NF ($n = 21$) | Test statistics | $p$ value | Post hoc tests | Effect size |
|---|---|---|---|---|---|---|---|
| *Sociodemographics* | | | | | | | |
| Age, *M* (*SD*) | 47.56 (13.31) | 49.01 (12.52) | 46.04 (14.23) | $F(1,41) = 0.53$ | .471 | | $\eta^2 = 0.01$ |
| Female sex, % (*n*) | 84% (36) | 81% (18) | 86% (18) | $\chi^2(1) = 0$ | 1.0 | | $V = 0.05$ |
| Education, % (*n*) | | | | | | | |
| ≥12 years | 53% (23) | 55% (12) | 52% (11) | $\chi^2(1) = 0$ | 1.0 | | $V = 0.02$ |
| *Clinical characteristics at baseline* | | | | | | | |
| BMI (kg/m$^2$), *M* (*SD*) | 36.82 (4.98) | 36.32 (5.08) | 36.89 (5.32) | $F(2,58) = 0.21$ | .812 | | $\eta^2 = 0.01$ |
| Weight status, % (*n*) | | | | $\chi^2(1) = 0.00$ | 1.00 | | $V = 0.01$ |
| Overweight (BMI 25 to <30 kg/m$^2$) | 14% (6) | 14% (3) | 14% (3) | | | | |
| Obesity (BMI ≥30 kg/m$^2$) | 86% (37) | 86% (19) | 86% (18) | | | | |
| *Eating disorder diagnosis (DSM-5)* | | | | | | | |
| BED, % (*n*) | 79% (34) | 81% (17) | 77% (17) | $\chi^2(1) = 0$ | 1.0 | | $V = 0.05$ |
| BED of low frequency and/or limited duration, % (*n*) | 21% (9) | 23% (5) | 19% (4) | $\chi^2(1) = 0$ | 1.0 | | $V = 0.06$ |
| *Eating disorder-related predictors* | | | | | | | |
| EDE binge-eating frequency past 28 days, *M* (*SD*) | 2.92 (2.76) | 3.35 (3.57) | 2.47 (1.50) | $F(1,41) = 1.09$ | .303 | | $\eta^2 = 0.03$ |
| Eating disorder psychopathology (EDE-Q global), *M* (*SD*) | 2.77 (0.94) | 2.63 (0.77) | 2.92 (1.09) | $F(1,39) = 0.97$ | .332 | | $\eta^2 = 0.02$ |
| Food cravings (FCQ-T-r), *M* (*SD*) | 57.88 (11.64) | 57.20 (11.43) | 58.55 (12.11) | $F(1,38) = 0.13$ | .719 | | $\eta^2 = 0.00$ |
| *Psychopathology-related predictors* | | | | | | | |
| Depressive symptoms (PHQ-D), *M* (*SD*) | 9.23 (4.07) | 9.30 (3.22) | 9.15 (4.89) | $F(1,39) = 0.01$ | .909 | | $\eta^2 = 0.00$ |
| Anxiety symptoms (GAD-7 sum score), *M* (*SD*) | 6.41 (4.14) | 5.76 (3.46) | 7.10 (4.73) | $F(1,39) = 1.07$ | .306 | | $\eta^2 = 0.03$ |
| Impulsivity (BIS mean score), *M* (*SD*) | 3.06 (0.51) | 2.96 (0.56) | 3.16 (0.44) | $F(1,39) = 1.69$ | .201 | | $\eta^2 = 0.04$ |
| Impulsivity (BAS mean score), *M* (*SD*) | 2.91 (0.34) | 2.91 (0.30) | 2.97 (0.30) | $F(1,37) = 0.34$ | .564 | | $\eta^2 = 0.01$ |
| Self-efficacy (GSES sum score), *M* (*SD*) | 27.07 (4.87) | 28.00 (5.04) | 26.10 (4.61) | $F(1,39) = 1.58$ | .216 | | $\eta^2 = 0.04$ |
| Emotion regulation (DERS global sum score), *M* (*SD*) | 87.49 (22.56) | 85.71 (21.25) | 89.35 (24.27) | $F(1,39) = 0.26$ | .612 | | $\eta^2 = 0.01$ |
| *Treatment completion* | | | | | | | |
| Attended 12 sessions, % (*n*) | 74% (32) | 81% (18) | 67% (14) | $\chi^2(1) = 0.62$ | .430 | | $V = 0.17$ |

*Note*: Effect sizes for continuous variables were reported as $\eta^2$ and interpreted as small (0.01), medium (0.06), and large (0.14). Effect sizes for categorical variables were reported as Cramer's *V* and interpreted as small (0.1), medium (0.3), and large (0.5).

Abbreviations: BIS/BAS, Behavioral Inhibition/Behavioral Approach System Scales; BMI, body mass index; DERS, Difficulties in Emotion Regulation Scale (36–180* less favorable scores are asterisked); DSM-5, Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (American Psychiatric Association, 2013); EDE, Eating Disorder Examination; EDE-Q, Eating Disorder Examination-Questionnaire (0–6*); EEG-NF, electroencephalography-neurofeedback; FCQ-T-r, Food Cravings Questionnaire-Trait reduced (0–90*); GAD-7, Generalized Anxiety Disorder 7-Item Scale (0–21*); GSES, General Self-Efficacy Scale (10*–40); PHQ-D, Patient Health Questionnaire-Depression Scale (0–27*); rtfNIRS-NF, real-time functional-near infrared spectroscopy neurofeedback.

($R^2_m = 0.197$, $R^2_c = 0.509$; large effect). Self-efficacy at t0, $\chi^2(1) = 0.84$, IRR = 0.97, 95% CI [0.90, 1.04], $rs = .01$, $p = .359$, did not predict OBE frequency at t1 and t2.

### 3.4 | Predictors of EDP: Eating disorder-related predictors

Higher EDP ($R^2_m = 0.519$, $R^2_c = 0.743$; large effect) was a large-sized, $F(1, 60.91) = 74.16$, $rs = .47$, $p < .001$, and female sex was a less-than-small-sized predictor, $F(1, 57.67) = 4.46$, $rs = .05$, $p = .038$, for higher EDP at t1 and t2.

### 3.5 | Predictors of EDP: Psychopathology-Related Predictors

Control variables EDP, $F(1,12.27) = 48.81$, $r_s = .50$, $p < .001$ and female sex, $F(1,1.12) = 4.45$, $r_s = .09$, $p = .026$, emerged as large-sized or less-than-small-sized, respectively, predictors for higher EDP at t1 and t2 ($R^2_m = 0.555$, $R^2_c = 0.728$; large effect). Impulsivity/behavioral approach did not significantly predict higher EDP at t1 and t2, $F(1,0.02) = 0.05$, $r_s = .41$, $p = .412$.

The interaction between anxiety symptoms and treatment group, $F(1,1.05) = 4.19$, $p = .050$, was not significant, indicating that anxiety symptoms score did not predict EDP at t1 and t2 in EEG-NF, $EMM = 0.00$, 95% CI [−0.06, 0.05], $p = .935$, but significantly predicted lower EDP in rtfNIRS-NF, $EMM = -0.09$, 95% CI [−0.17, −0.02], $p = .013$. The difference between groups was not significant, difference = −0.09, 95% CI [−0.18, 0.00], $p = .050$.

## 4 | DISCUSSION

This study investigated eating disorder- and general psychopathology-related predictors of rtfNIRS- or EEG-NF outcome for BED at post-treatment (t1) and at 6-month follow-up (t2). Eating disorder-related characteristics of lower food cravings, a higher BMI, and higher EDP were most consistent baseline predictors of favorable treatment responses, that is, lower OBE frequency and lower EDP. Findings for psychopathology-related predictors were inconclusive, while anxiety symptoms were descriptively associated with lower EDP after rtfNIRS-NF at t1 and t2.

Overall, the findings highlighted the relevance of BED- versus general psychopathology-related symptoms for NF treatment success, consistent with predictors for diverse psychological treatments in BED (Forrest et al., 2021) and meta-analytical predictors for diverse treatment options across EDs (Vall & Wade, 2015). Differential predictors emerged for OBE frequency and EDP, composed of restraint, eating, weight, and shape concern, indicating that the improvement of core symptoms versus EDP was associated with different baseline eating disorder- and general psychopathology-related characteristics of patients with BED. Previous studies demonstrated a faster behavioral remission (abstinence from binge eating) than psychological recovery of cognitive symptoms (EDE-Q mean global score within the healthy range) in BED after group (Dingemans et al., 2020) or individual cognitive–behavioral therapy (Hilbert et al., 2012). In the present study, higher baseline EDP predicted higher EDP up to 6 months after NF, in accordance with previous notions that higher EDP predicted higher EDP and poor treatment outcome in adult and adolescent BED after cognitive–behavioral therapy (Romero et al., 2019; R. Schmidt & Hilbert, 2022). In this context, the inclusion of EDP in addition to the behavioral remission (i.e., abstinence from binge eating) has been proposed in a potential comprehensive definition of remission from BED (Bardone-Cone et al., 2018). However, the present findings were qualified by the repeated measurement of OBE and EDP causing regression to the mean that likely contributed to the observed associations.

Considering eating disorder related-predictors, building on the addiction model (Franken, 2003), the association between higher food cravings at baseline and higher OBE frequency after NF may have mirrored difficulties to resist tempting foods arising from an automatic contingency between appetitive foods and patients' attention. The modulation of PFC responses toward appetitive food stimuli during NF possibly pronounced this attentional bias and promoted appetitive behaviors (Kessler et al., 2016; Stojek et al., 2018; Stott et al., 2021), while hampering concurrent abilities to draw attention to competing physiological cues (e.g., satiety; Hargrave et al., 2016; Sample et al., 2015). The positive association between baseline BMI and OBE frequency after NF in the present study is consistent with the previously reported cross-sectional link between more OBEs and higher BMI in BED (Dakanalis et al., 2017). Besides higher OBE, higher EDP and more food cravings, a higher BMI possibly reflected a greater chronicity and/or a higher symptom severity and was linked to an increased risk for sequelae like metabolic syndrome (Dakanalis et al., 2017). Thus, patients with BED who show relatively high baseline levels of OBE and/or EDP and/or food cravings and/or a higher BMI represent a group with special needs to be considered in clinical decision making. Furthermore, our findings implied younger patients as a vulnerable group, possibly due to an earlier disorder onset and greater symptom severity and accompanying psychopathology for younger patients with various EDs (Fernández-Aranda et al., 2021; Romero et al., 2019).

The interaction between anxiety symptoms and treatment group did not reach significance, but anxiety symptoms were surprisingly linked to lower EDP in rtfNIRS-NF in follow-up analyses. This vague finding resembled the inconclusive evidence on the predictive role of anxiety symptoms for outcomes of diverse BED treatments (Forrest et al., 2021; Lydecker & Grilo, 2021). Specifically, the presence of comorbid anxiety disorders was linked to higher EDP after psychological and pharmacological treatment for BED (Lydecker & Grilo, 2021). The relatively low and—in contrast to EEG-NF less variable—baseline anxiety levels in rtfNIRS-NF (Table 2) could thus indicate that only clinically significant anxiety symptoms impact symptom deterioration after treatment. Alternatively, improvements in anxiety symptoms throughout psychological treatments predicted favorable responses in bulimia nervosa (Matherne et al., 2022), suggesting the need for

mechanistic research to disentangle anxiety as a baseline predictor versus mediator for favorable treatment response. In general, the unexpected responses in the rtfNIRS-NF group underline the need to examine the validity of the present findings in future NF research.

The lacking link between baseline impulsivity and OBE frequency or EDP after NF treatment at first contradicted the documented food-related impulsivity in BED compared with groups with normal weight or overweight (Kittel et al., 2015, 2017; Svaldi et al., 2014), although a recent study similarly found no link between general impulsivity and ED outcomes or BMI after psychological and pharmacological treatment in BED (Boswell et al., 2021). A previous study only linked behaviorally assessed, but not self-reported, food-specific inhibitory control to the outcomes of a group psychological treatment in BED (Manasse et al., 2016). Future evidence that scrutinizes the impact of different dimensions of impulsivity, ideally with self-report and behavioral measures, on NF outcomes in BED remains consequently imperative.

Overall, the present findings indicated that a careful assessment of baseline patient factors may inform individualized treatment with the highest expectable success, in line with a recently proposed integrative clinical decision-making models for BED (Chyurlia et al., 2019). Possibly different predictors for EEG- versus rtfNIRS-NF (Supporting Information Results in Data S1) may be explained through differential mechanisms depending on the processes targeted by the respective modality. EEG-NF targeted high beta down-regulation presumably linked to decreased appetitive food-evoked responses (Blume et al., 2019), whereas rtfNIRS-NF aimed to modulate food cue reactivity through the up-regulation of individual PFC control regions. While patients with higher binge-eating frequency possibly benefit more from rtfNIRS-NF due to the intended enhanced cognitive control toward foods, a decrease in food-cue attentional bias and distress intended through high-beta EEG-NF may possibly enhance treatment success of patients with higher comorbid psychopathology. Future investigations of both imaging modalities applied simultaneously may shed light on the validity of each imaging modality and a potential added values of bimodal NF (Perronnet et al., 2017; Zich et al., 2015).

Regarding clinical translation, the evidenced pervasive impact of baseline psychological variables, in particular eating disorder-related variables, for worse NF treatment outcomes, is important for clinicians to consider in treatment decisions and planning. If future research supports the premise that patients with BED with higher OBE frequency and/or EDP show less symptom alleviation after NF interventions, tailored interventions warrant consistent clinical implementation.

Our study's strengths included the careful consideration of numerous psychological variables, taking into account eating disorder- and general psychopathology symptoms, all assessed via validated questionnaires or a validated clinical interview conducted by blind assessors, and standardized treatment protocols for rtfNIRS- and EEG-NF. However, some limitations must be acknowledged. The unexpectedly high spontaneous remission (Hilbert et al., 2023) and subsequent exclusion of the WL group from the analyses may have diminished the power of the present findings. The current sample was composed of predominantly female, white, treatment-seeking patients with BED, which limits generalizability of findings. Twelve individually appetitive food stimuli enhanced the salience of the presented food cues, and cognitive-behavioral therapy-based homework assigned after each NF session aimed to promote the transfer of NF skills to everyday live. However, the indirect assessment of this intended generalization through OBE frequency after the NF intervention prevents conclusions about food cues not targeted through the intervention.

## 5 | CONCLUSIONS

This study uniquely assessed eating disorder- and general psychopathology-related predictors for NF treatment response in BED, complementing previous research on treatment predictors for psychotherapy. We confirmed the key importance of eating disorder-related predictors for NF treatment response, indicating specific treatment needs for heavily burdened patients with BED. These findings paved the way for optimized treatment assignment based on underlying psychopathology in the future, while investigations of an incremental value of NF in addition to other interventions such as psychotherapy are warranted. Further mechanistic research for possibly divergent psychopathology-related predictors for EEG- versus rtfNIRS-NF depending on the targeted brain region and targeted processes is outstanding. Future investigations of brain-based predictors for NF treatment outcomes may assist NF protocol optimization by possibly enabling NF tailored to individual neurophysiological profiles.

### AUTHOR CONTRIBUTIONS

**Sarah Alica Rösch:** Data curation; formal analysis; investigation; visualization; writing – original draft; writing – review and editing. **Ricarda Schmidt:** Conceptualization; investigation; methodology; software; supervision; validation; writing – review and editing. **Anja Hilbert:** Conceptualization; funding acquisition; methodology; project administration; resources; software; supervision; validation; writing – review and editing.

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## CONFLICT OF INTEREST STATEMENT

The authors declare no conflicts of interest.

## OPEN RESEARCH BADGES

This article has earned a Preregistered Research Designs badge for having a preregistered research design, available at (https://osf.io/4aktp/).

## DATA AVAILABILITY STATEMENT

The data that support the findings of this study are available from the corresponding author upon reasonable request.

## ETHICS STATEMENT

The study was approved by the Ethics Committee of the University of Leipzig (476/17-ek). Informed Consent Statement: Informed consent was obtained from all subjects involved in the study.

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## REFERENCES

Adcock, R., Lutomski, K., Mcleod, S., Soneji, D., & Gabrieli, J. (2005). Real-time fMRI during the psychotherapy session: Toward a methodology to augment therapeutic benefit, exemplary data [Abstract presentation]. Human Brain Mapping Conference.

American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). APA.

Anderson, L. M., Smith, K. M., Schaefer, L. M., Crosby, R. D., Cao, L., Engel, S. G., Crow, S. J., Wonderlich, S. A., & Peterson, C. B. (2020). Predictors and moderators of treatment outcome in a randomized clinical trial for binge-eating disorder. *Journal of Consulting and Clinical Psychology*, *88*(7), 631–642. https://doi.org/10.1037/ccp0000503

Arns, M., Batail, J. M., Bioulac, S., Congedo, M., Daudet, C., Drapier, D., Fovet, T., Jardri, R., Le-Van-Quyen, M., Lotte, F., Mehler, D., Micoulaud-Franchi, J. A., Purper-Ouakil, D., & Vialatte, F. (2017). Neurofeedback: One of today's techniques in psychiatry? *Encephale*, *43*(2), 135–145. https://doi.org/10.1016/j.encep.2016.11.003

Bardone-Cone, A. M., Hunt, R. A., & Watson, H. J. (2018). An overview of conceptualizations of eating disorder recovery, recent findings, and future directions. *Current Psychiatry Reports*, *20*(9), 79. https://doi.org/10.1007/s11920-018-0932-9

Bartholdy, S., Musiat, P., Campbell, I. C., & Schmidt, U. (2013). The potential of neurofeedback in the treatment of eating disorders: A review of the literature. *European Eating Disorders Review*, *21*(6), 456–463. https://doi.org/10.1002/erv.2250

Bender, R., & Lange, S. (2001). Adjusting for multiple testing – When and how? *Journal of Clinical Epidemiology*, *54*, 343–349. https://doi.org/10.1177/13623613211007319

Berner, L. A., Winter, S. R., Ayaz, H., Shewokis, P. A., Izzetoglu, M., Marsh, R., Nasser, J. A., Matteucci, A. J., & Lowe, M. R. (2022). Altered prefrontal activation during the inhibition of eating responses in women with bulimia nervosa. *Psychological Medicine*, *53*, 3580–3590. https://doi.org/10.1017/S0033291722000198

Blume, M., Schmidt, R., & Hilbert, A. (2019). Abnormalities in the EEG power spectrum in bulimia nervosa, binge-eating disorder, and obesity: A systematic review. *European Eating Disorders Review*, *27*(2), 124–136. https://doi.org/10.1002/erv.2654

Blume, M., Schmidt, R., Schmidt, J., Martin, A., & Hilbert, A. (2022). EEG neurofeedback in the treatment of adults with binge-eating disorder: A randomized controlled pilot study. *Neurotherapeutics*, *19*, 352–365. https://doi.org/10.1007/s13311-021-01149-9

Boswell, R. G., Gueorguieva, R., & Grilo, C. M. (2021). Change in impulsivity is prospectively associated with treatment outcomes for binge-eating disorder. *Psychological Medicine*, *1-9*, 2789–2797. https://doi.org/10.1017/S003329172100475X

Carver, C., & White, T. (1994). Behavioral inhibition, behavioral activation, and affective responses to impending reward and punishment. *Journal of Personality and Social Psychology*, *67*(2), 319–333. https://doi.org/10.1037/0022-3514.67.2.319

Castellini, G., Mannucci, E., Lo Sauro, C., Benni, L., Lazzeretti, L., Ravaldi, C., Rotella, C. M., Faravelli, C., & Ricca, V. (2011). Different moderators of cognitive-behavioral therapy on subjective and objective binge eating in bulimia nervosa and binge eating disorder: A three-year follow-up study. *Psychotherapy and Psychosomatics*, *81*(1), 11–20. https://doi.org/10.1159/000329358

Chyurlia, L., Tasca, G. A., & Bissada, H. (2019). An integrative approach to clinical decision-making for treating patients with binge-eating disorder. *Frontiers in Psychology*, *10*, 2573. https://doi.org/10.3389/fpsyg.2019.02573

Cohen, J. (1988). *Statistical power analysis for the behavioral sciences* (2nd ed.). Erlbaum.

Dakanalis, A., Riva, G., Serino, S., Colmegna, F., & Clerici, M. (2017). Classifying adults with binge eating disorder based on severity levels. *European Eating Disorders Review*, *25*(4), 268–274. https://doi.org/10.1002/erv.2518

Dingemans, A. E., van Son, G. E., Vanhaelen, C. B., & van Furth, E. F. (2020). Depressive symptoms rather than executive functioning predict group cognitive behavioural therapy outcome in binge eating disorder. *European Eating Disorders Review*, *28*, 620–632. https://doi.org/10.1002/erv.2768

Fairburn, C. G., & Beglin, S. (2008). Eating disorder examination-questionnaire (EDE-Q 6.0). In C. G. Fairburn (Ed.), *Cognitive behavior therapy and eating disorders* (pp. 309–314). Guilford Press.

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

Fernández-Aranda, F., Treasure, J., Paslakis, G., Agüera, Z., Giménez, M., Granero, R., Sánchez, I., Serrano-Troncoso, E., Gorwood, P., Herpertz-Dahlmann, B., Bonin, E. M., Monteleone, P., & Jiménez-Murcia, S. (2021). The impact of duration of illness on treatment nonresponse and drop-out: Exploring the relevance of enduring eating disorder concept. *European Eating Disorders Review*, *29*(3), 499–513. https://doi.org/10.1002/erv.2822

Forcano, L., Mata, F., de la Torre, R., & Verdejo-Garcia, A. (2018). Cognitive and neuromodulation strategies for unhealthy eating and obesity: Systematic review and discussion of neurocognitive mechanisms. *Neuroscience and Biobehavioral Reviews*, *87*, 161–191. https://doi.org/10.1016/j.neubiorev.2018.02.003

Forrest, L. N., Ivezaj, V., & Grilo, C. M. (2021). Machine learning v. traditional regression models predicting treatment outcomes for binge-eating disorder from a randomized controlled trial. *Psychological Medicine*, *53*, 2777–2788. https://doi.org/10.1017/S0033291721004748

Fox, J., & Weisberg, S. (2019). *An R companion to applied regression* (3rd ed.). Sage Publications.

Franken, I. H. A. (2003). Drug craving and addiction: Integrating psychological and neuropsychopharmacological approaches. *Progress in Neuro-Psychopharmacology and Biological Psychiatry*, *27*(4), 563–579. https://doi.org/10.1016/S0278-5846(03)00081-2

Gerlach, G., Herpertz, S., & Loeber, S. (2015). Personality traits and obesity: A systematic review. *Obesity Reviews*, *16*(1), 32–63. https://doi.org/10.1111/obr.12235

Giel, K. E., Bulik, C. M., Fernandez-Aranda, F., Hay, P., Keski-Rahkonen, A., Schag, K., Schmidt, U., & Zipfel, S. (2022). Binge eating disorder. *Nature Reviews. Disease Primers*, *8*(1), 16. https://doi.org/10.1038/s41572-022-00344-y

Goth, G. (2006). Neurofeedbacktherapie bei Kindern mit Aufmerksamkeitsdefizit-/Hyperaktivitätsstörung: Prädiktoren für den Erwerb kortikaler Selbstkontrolle und die klinische Verbesserung [Doctoral dissertation; University of Tübingen]. http://deposit.ddb.de/cgi-bin/dokserv?idn=981019889&dok_var=d1&dok_ext=pdf&filename=981019889.pdf

Gräfe, K., Zipfel, S., Herzog, W., & Löwe, B. (2004). Screening psychischer Störungen mit dem "Gesundheitsfragebogen für Patienten (PHQ-D)": Ergebnisse der deutschen Validierungsstudie [screening for psychiatric disorders with the patient health questionnaire (PHQ). Results from the German validation study]. *Diagnostica*, *50*(4), 171–181. https://doi.org/10.1026/0012-1924.50.4.171

Gratz, K. L., & Roemer, L. (2004). Multidimensional assessment of emotion regulation and dysregulation. *Journal of Psychopathology and Behavioral Assessment*, *26*(1), 41–54. https://doi.org/10.1023/B:JOBA.0000007455.08539.94

Grilo, C. M., Masheb, R. M., & Crosby, R. D. (2012). Predictors and moderators of response to cognitive behavioral therapy and medication for the treatment of binge eating disorder. *Journal of Consulting and Clinical Psychology*, *80*(5), 897–906. https://doi.org/10.1037/a0027001

Grilo, C. M., Thompson-Brenner, H., Shingleton, R. M., Thompson, D. R., & Franko, D. L. (2021). Clinical moderators and predictors of cognitive-behavioral therapy by guided-self-help versus therapist-led for binge-eating disorder: Analysis of aggregated clinical trials. *International Journal of Eating Disorders*, *54*(10), 1875–1880. https://doi.org/10.1002/eat.23601

Hargrave, S. L., Jones, S., & Davidson, T. L. (2016). The outward spiral: A vicious cycle model of obesity and cognitive dysfunction. *Current Opinion in Behavioral Sciences*, *9*, 40–46. https://doi.org/10.1016/j.cobeha.2015.12.001

Haynos, A. F., Wang, S. B., Lipson, S., Peterson, C. B., Mitchell, J. E., Halmi, K. A., Agras, W. S., & Crow, S. J. (2021). Machine learning enhances prediction of illness course: A longitudinal study in eating disorders. *Psychological Medicine*, *51*(8), 1392–1402. https://doi.org/10.1017/S0033291720000227

Hilbert, A., Bishop, M. E., Stein, R. I., Tanofsky-Kraff, M., Swenson, A. K., Welch, R. R., & Wilfley, D. E. (2012). Long-term efficacy of psychological treatments for binge eating disorder. *British Journal of Psychiatry*, *200*(3), 232–237. https://doi.org/10.1192/bjp.bp.110.089664

Hilbert, A., Rösch, S., Petroff, D., Prettin, C., Lührs, M., Ehlis, A.-C., & Schmidt, R. (2023). Near-infrared spectroscopy neurofeedback for binge-eating disorder: An exploratory randomized comparison with electroencephalography neurofeedback. *Psychological Medicine*. In press.

Hilbert, A., & Tuschen-Caffier, B. (2016a). *Eating disorder examination-questionnaire: Deutschprachige Übersetzung [Eating disorder examination-questionnaire: German translation]*. dgvt-Verlag. https://doi.org/10.1026/0012-1924.53.3.144

Hilbert, A., & Tuschen-Caffier, B. (2016b). *Eating disorder examination: Deutschsprachige Übersetzung [Eating disorder examination: German translation]*. dgvt-Verlag. https://doi.org/10.1007/978-981-287-104-6_101

Howard, K. I., Kopta, S. M., Krause, M. S., & Orlinsky, D. E. (1986). The dose-effect relationship in psychotherapy. *American Psychologist*, *41*(2), 159–164. https://doi.org/10.1037/0003-066X.41.2.159

Iacovino, J. M., Drive, B., Louis, S., Gredysa, D. M., & Wilfley, D. E. (2012). Psychological treatments for binge eating disorder. *Current Psychiatry Reports*, *14*(4), 432–446. https://doi.org/10.1007/s11920-012-0277-8

Jaeger, B. C. (2017). R2glmm: Computes r squared for mixed (multilevel models; Version R package version 0.1.2). https://cran.r-project.org/package=%0Ar2glmm

Johnson, P. C. D. (2014). Extension of Nakagawa & Schielzeth's R2GLMM to random slopes models. *Methods in Ecology and Evolution*, *5*(9), 944–946. https://doi.org/10.1111/2041-210X.12225

Kadosh, K. C., & Staunton, G. (2019). A systematic review of the psychological factors that influence neurofeedback learning outcomes. *NeuroImage*, *185*, 545–555. https://doi.org/10.1016/j.neuroimage.2018.10.021

Keski-Rahkonen, A., & Mustelin, L. (2016). Epidemiology of eating disorders in Europe: Prevalence, incidence, comorbidity, course, consequences, and risk factors. *Current Opinion in Psychiatry*, *29*, 340–345.

Kessler, R. M., Hutson, P. H., Herman, B. K., & Potenza, M. N. (2016). The neurobiological basis of binge-eating disorder. *Neuroscience and Biobehavioral Reviews*, *63*, 223–238. https://doi.org/10.1016/j.neubiorev.2016.01.013

Kittel, R., Brauhardt, A., & Hilbert, A. (2015). Cognitive and emotional functioning in binge-eating disorder: A systematic review. *International Journal of Eating Disorders*, *48*(6), 535–554. https://doi.org/10.1002/eat.22419

Kittel, R., Schmidt, R., & Hilbert, A. (2017). Executive functions in adolescents with binge-eating disorder and obesity. *International Journal of Eating Disorders*, *50*(8), 933–941. https://doi.org/10.1002/eat.22714

Kohl, S. H., Veit, R., Spetter, M. S., Günther, A., Rina, A., Lührs, M., Birbaumer, N., Preissl, H., & Hallschmid, M. (2019). Real-time fMRI neurofeedback training to improve eating behavior by self-regulation of the dorsolateral prefrontal cortex: A randomized controlled trial in overweight and obese subjects. *NeuroImage*, *191*, 596–609. https://doi.org/10.1016/j.neuroimage.2019.02.033

Kraemer, H. C., Wilson, G. T., Fairburn, C. G., & Agras, W. S. (2002). Mediators and moderators of treatment effects in randomized clinical trials. *Archives of General Psychiatry*, *59*(10), 877–883. https://doi.org/10.1001/archpsyc.59.10.877

Krell, J., Todd, A., & Dolecki, P. K. (2019). Bridging the gap between theory and practice in neurofeedback training for attention. *Mind, Brain, and Education*, *13*(4), 246–260. https://doi.org/10.1111/mbe.12220

Krepel, N., Egtberts, T., Sack, A. T., Heinrich, H., Ryan, M., & Arns, M. (2020). A multicenter effectiveness trial of QEEG-informed neurofeedback in ADHD: Replication and treatment prediction. *NeuroImage: Clinical*, *28*, 102399. https://doi.org/10.1016/j.nicl.2020.102399

Kuznetsova, A., Brockhoff, P. B., & Christensen, R. H. B. (2017). lmerTest package: Tests in linear mixed effects models. *Journal of Statistical Software*, *82*(13), 1–26. https://doi.org/10.18637/jss.v082.i13

Lavagnino, L., Arnone, D., Cao, B., Soares, J. C., & Selvaraj, S. (2016). Inhibitory control in obesity and binge eating disorder: A systematic review and meta-analysis of neurocognitive and neuroimaging studies. *Neuroscience and Biobehavioral Reviews*, *68*, 714–726. https://doi.org/10.1016/j.neubiorev.2016.06.041

Leehr, E. J., Schag, K., Dresler, T., Grosse-Wentrup, M., Hautzinger, M., Fallgatter, A. J., Zipfel, S., Giel, K. E., & Ehlis, A. C. (2018). Food specific inhibitory control under negative mood in binge-eating disorder: Evidence from a multimethod approach. *International Journal of Eating Disorders*, *51*(2), 112–123. https://doi.org/10.1002/eat.22818

Lenth, R. V. (2022). emmeans: Estimated marginal means, aka least-squares means (R package version 1.7.5). https://cran.r-project.org/package=emmeans

Linden, D. E. J. (2014). Neurofeedback and networks of depression. *Dialogues in Clinical Neuroscience*, *16*(1), 103–112. https://doi.org/10.31887/dcns.2014.16.1/dlinden

Löwe, B., Decker, O., Müller, S., Brähler, E., Schellberg, D., Herzog, W., & Herzberg, P. Y. (2008). Validation and standardization of the generalized anxiety disorder screener (GAD-7) in the general population. *Medical Care*, *46*(3), 266–274.

Lowe, C. J., Reichelt, A. C., & Hall, P. A. (2019). The prefrontal cortex and obesity: A health neuroscience perspective. *Trends in Cognitive Sciences*, *23*(4), 349–361. https://doi.org/10.1016/j.tics.2019.01.005

Lydecker, J. A., & Grilo, C. M. (2021). Psychiatric comorbidity as predictor and moderator of binge-eating disorder treatment outcomes: An analysis of aggregated randomized controlled trials. *Psychological Medicine*, *1-9*, 1–9. https://doi.org/10.1017/S0033291721001045

Lydecker, J. A., Gueorguieva, R., Masheb, R., White, M. A., & Grilo, C. M. (2020). Examining sex as a predictor and moderator of treatment outcomes for binge-eating disorder: Analysis of aggregated randomized controlled trials. *International Journal of Eating Disorders*, *53*(1), 20–30. https://doi.org/10.1002/eat.23167

Lydecker, J. A., Ivezaj, V., & Grilo, C. M. (2020). Testing the validity and clinical utility of the severity specifiers for binge-eating disorder for predicting treatment outcomes. *Journal of Consulting and Clinical Psychology*, *88*(2), 172–178. https://doi.org/10.1037/ccp0000464

MacDuffie, K. E., MacInnes, J., Dickerson, K. C., Eddington, K. M., Strauman, T. J., & Adcock, R. A. (2018). Single session real-time fMRI neurofeedback has a lasting impact on cognitive behavioral therapy strategies. *NeuroImage: Clinical*, *19*, 868–875. https://doi.org/10.1016/j.nicl.2018.06.009

Manasse, S. M., Espel, H. M., Schumacher, L. M., Kerrigan, S. G., Zhang, F., Forman, E. M., & Juarascio, A. S. (2016). Does impulsivity predict outcome in treatment for binge eating disorder? A multimodal investigation. *Appetite*, *105*, 172–179. https://doi.org/10.1016/j.physbeh.2017.03.040

Matherne, C. E., Watson, H., Fassnacht, D. B., Ali, K., Zerwas, S., Peat, C., Runfola, C., Levine, M. D., Marcus, M. D., Zimmer, B., Moessner, M., Crosby, R., & Bulik, C. M. (2022). An exploratory investigation of predictors of outcome in face-to-face and online cognitive-behavioural therapy for bulimia nervosa. *European eating disorders review: the journal of the Eating Disorders Association*, *30*(4), 373–387. https://doi.org/10.1002/erv.2898

Meule, A., Hermann, T., & Kübler, A. (2014). A short version of the food cravings questionnaire-trait: The FCQ-T-reduced. *Frontiers in Psychology*, *5*(190). https://doi.org/10.3389/fpsyg.2014.00190

Mikhail, M. E., Keel, P. K., Burt, S. A., Neale, M., Boker, S., & Klump, K. L. (2020). Low emotion differentiation: An affective correlate of binge eating? *International Journal of Eating Disorders*, *53*(3), 412–421. https://doi.org/10.1002/eat.23207

Müller, A., Smits, D., Claes, L., & De Zwaan, M. (2013). Faktorenstruktur der deutschsprachigen version der BIS/BAS-Skalen in einer Bevölkerungsstichprobe. *Fortschritte Der Neurologie Psychiatrie*, *81*(2), 75–80. https://doi.org/10.1055/s-0032-1330482

Nakagawa, S., & Schielzeth, H. (2013). A general and simple method for obtaining $R^2$ from generalized linear mixed-effects models. *Methods in Ecology and Evolution*, *4*, 133–142.

Nickel, K., Maier, S., Endres, D., Joos, A., Maier, V., Tebartz van Elst, L., & Zeeck, A. (2019). Systematic review: Overlap between eating, autism spectrum, and attention-deficit/hyperactivity disorder. *Frontiers in Psychiatry*, *10*, 708. https://doi.org/10.3389/fpsyt.2019.00708

Perronnet, L., Lécuyer, A., Mano, M., Bannier, E., Lotte, F., Clerc, M., & Barillot, C. (2017). Unimodal versus bimodal EEG-fMRI neurofeedback of a motor imagery task. *Frontiers in Human Neuroscience*, *11*, 193. https://doi.org/10.3389/fnhum.2017.00193

Prefit, A.-B., Cândea, D. M., & Szentagotai-Tătar, A. (2019). Emotion regulation across eating pathology: A meta-analysis. *Appetite*, *143*, 104438. https://doi.org/10.1016/j.appet.2019.104438

R Core Team. (2020). R: a language and environment for statistical computing. R Foundation for Statistical Computing. https://www.r-project.org

Romero, X., Agüera, Z., Granero, R., Sánchez, I., Riesco, N., Jiménez-Murcia, S., Gisbert-Rodriguez, M., Sánchez-González, J., Casalé, G., Baenas, I., Valenciano-Mendoza, E., Menchon, J. M., Gearhardt, A. N., Dieguez, C., & Fernández-Aranda, F. (2019). Is food addiction a predictor of treatment outcome among patients with eating disorder? *European Eating Disorders Review*, *27*(6), 700–711. https://doi.org/10.1002/erv.2705

Rösch, S. A., Schmidt, R., & Hilbert, A. (2022). Predictors of neurofeedback in BED.

Rösch, S. A., Schmidt, R., Lührs, M., Ehlis, A. C., Hesse, S., & Hilbert, A. (2021). Evidence of fNIRS-based prefrontal cortex hypoactivity in obesity and binge-eating disorder. *Brain Sciences*, *11*(1), 19. https://doi.org/10.3390/brainsci11010019

Rubin, M. (2017). Do p values lose their meaning in exploratory analyses? It depends how you define the familywise error rate. *Review of General Psychology*, *21*(3), 269–275. https://doi.org/10.1037/gpr0000123

Sample, C. H., Martin, A. A., Jones, S., Hargrave, S. L., & Davidson, T. L. (2015). Western-style diet impairs stimulus control by food deprivation state cues. Implications for obesogenic environments. *Appetite*, *93*, 13–23. https://doi.org/10.1016/j.appet.2015.05.018.Western-style

Schmidt, J., & Martin, A. (2015). Neurofeedback reduces overeating episodes in female restrained eaters: A randomized controlled pilot-study. *Applied Psychophysiology and Biofeedback*, *40*(4), 283–295. https://doi.org/10.1007/s10484-015-9297-6

Schmidt, J., & Martin, A. (2016). Neurofeedback against binge eating: A randomized controlled trial in a female subclinical threshold sample. *European Eating Disorders Review*, *24*(5), 406–416. https://doi.org/10.1002/erv.2453

Schmidt, J., & Martin, A. (2020). The influence of physiological and psychological learning mechanisms in neurofeedback vs. mental imagery against binge eating. *Applied Psychophysiology and Biofeedback*, *45*(4), 295–305. https://doi.org/10.1007/s10484-020-09486-9

Schmidt, R., & Hilbert, A. (2022). Predictors of symptom trajectories after cognitive-behavioral therapy in adolescents with an age-adapted diagnosis of binge-eating disorder. *Behavior Therapy*, *53*(1), 137–149. https://doi.org/10.1016/j.beth.2021.08.002

Schwarzer, R., & Jerusalem, M. (1995). Generalized self-efficacy scale. In J. Weinman, S. Wright, & M. Johnston (Eds.), *Measures in health psychology: A user's portfolio. Causal and control beliefs* (pp. 35–37). Nfer-Nelson.

Serra, R., Kiekens, G., Tarsitani, L., Vrieze, E., Bruffaerts, R., Loriedo, C., An, A., & Vanderlinden, J. (2020). The effect of trauma and dissociation on the outcome of cognitive behavioural therapy for binge eating disorder: A 6-month prospective study. *European Eating Disorders Review*, *28*(3), 309–317. https://doi.org/10.1002/erv.2722

Soekadar, S. R., Kohl, S. H., Mihara, M., & von Lühmann, A. (2021). Optical brain imaging and its application to neurofeedback. *NeuroImage: Clinical*, *2021*, 102577. https://doi.org/10.1016/j.nicl.2021.102577

Sotirchos, E., Fitzgerald, K., & Crainiceanu, C. (2019). Reporting of R2 statistics for mixed-effects regression models. *JAMA Neurology*, *76*(4), 507. https://doi.org/10.1001/jamaneurol.2018.4720

Spitzer, R. L., Kroenke, K., & Williams, J. B. (1999). Validation and utility of a self-report version of PRIME-MD: The PHQ primary care study. *JAMA*, *282*(18), 1737–1744.

Spitzer, R. L., Kroenke, K., Williams, J. B. W., & Löwe, B. (2006). A brief measure for assessing generalized anxiety disorder: The GAD-7. *Archives of Internal Medicine*, *166*(10), 1092–1097. https://doi.org/10.1001/archinte.166.10.1092

Steenbarger, B. N. (1994). Duration and outcome in psychotherapy: An integrative review. *Professional Psychology: Research and Practice*, *25*(2), 111–119. https://doi.org/10.1037/0735-7028.25.2.111

Stojek, M., Shank, L. M., Vannucci, A., Bongiorno, D. M., Nelson, E. E., Waters, A. J., Engel, S. G., Boutelle, K. N., Pine, D. S., Yanovski, J. A., & Tanofsky-Kraff, M. (2018). A systematic review of attentional biases in disorders involving binge eating. *Appetite*, *123*, 367–389. https://doi.org/10.1016/j.appet.2018.01.019

Stott, N., Fox, J. R. E., & Williams, M. O. (2021). Attentional bias in eating disorders: A meta-review. *International Journal of Eating Disorders*, *54*(8), 1377–1399. https://doi.org/10.1002/eat.23560

Strobel, A., Beauducel, A., Debener, S., & Brocke, B. (2006). A german version of Carver and White's BIS/BAS scales. *Zeitschrift Für Differentielle Und Diagnostische Psychologie*, *22*(3), 216–227. https://doi.org/10.1024//0170-1789.22.3.216

Svaldi, J., Naumann, E., Trentowska, M., & Schmitz, F. (2014). General and food-specific inhibitory deficits in binge eating disorder. *International Journal of Eating Disorders*, *47*(5), 534–542. https://doi.org/10.1002/eat.22260

Thompson-Brenner, H., Franko, D. L., Thompson, D. R., Grilo, C. M., Boisseau, C. L., Roehrig, J. P., Richards, L. K., Bryson, S. W., Bulik, C. M., Crow, S. J., Devlin, M. J., Gorin, A. A., Kristeller, J. L., Masheb, R., Mitchell, J. E., Peterson, C. B., Safer, D. L., Striegel, R. H., Wilfley, D. E., & Wilson, G. T. (2013). Race/ethnicity, education, and treatment parameters as moderators and predictors of outcome in binge eating disorder. *Journal of Consulting and Clinical Psychology*, *81*(4), 710–721. https://doi.org/10.1037/a0032946.Race/Ethnicity

Udo, T., & Grilo, C. M. (2019). Psychiatric and medical correlates of DSM-5 eating disorders in a nationally representative sample of adults in the United States. *International Journal of Eating Disorders*, *52*(1), 42–50. https://doi.org/10.1002/eat.23004

Unterrainer, H. F., Chen, M. J. L., & Gruzelier, J. H. (2014). EEG-neurofeedback and psychodynamic psychotherapy in a case of adolescent anhedonia with substance misuse: Mood/theta relations. *International Journal of Psychophysiology*, *93*(1), 84–95. https://doi.org/10.1016/j.ijpsycho.2013.03.011

Vall, E., & Wade, T. D. (2015). Predictors of treatment outcome in individuals with eating disorders : A systematic review and meta-analysis. *International Journal of Eating Disorders*, *48*(7), 946–971. https://doi.org/10.1002/eat.22411

Val-Laillet, D., Aarts, E., Weber, B., Ferrari, M., Quaresima, V., Stoeckel, L. E., Alonso-Alonso, M., Audette, M., Malbert, C. H., & Stice, E. (2015). Neuroimaging and neuromodulation approaches to study eating behavior and prevent and treat eating disorders and obesity. *Neuroimage: Clinical*, *8*, 1–31. https://doi.org/10.1016/j.nicl.2015.03.016

Veit, R., Schag, K., Schopf, E., Borutta, M., Kreutzer, J., Ehlis, C., Zipfel, S., Giel, K. E., & Preissl, H. (2021). Diminished prefrontal cortex activation in patients with binge eating disorder associates with trait impulsivity and improves after impulsivity-focused treatment based on a randomized controlled IMPULS trial. *Neuroimage: Clinical*, *2021*, 102679. https://doi.org/10.1016/j.nicl.2021.102679

Weber, L. A., Ethofer, T., & Ehlis, A. C. (2020). Predictors of neurofeedback training outcome: A systematic review. *NeuroImage: Clinical*, *27*, 102301. https://doi.org/10.1016/j.nicl.2020.102301

Wilfley, D. E., Citrome, L., & Herman, B. K. (2016). Characteristics of binge eating disorder in relation to diagnostic criteria. *Neuropsychiatric Disease and Treatment*, *12*, 2213–2223. https://doi.org/10.2147/NDT.S107777

Wilson, G. T., Wilfley, D. E., Agras, W. S., & Bryson, S. W. (2010). Psychological treatments of binge eating disorder. *Archives of General Psychiatry*, *67*(1), 94–101. https://doi.org/10.1001/archgenpsychiatry.2009.170

Zich, C., Debener, S., Kranczioch, C., Bleichner, M. G., Gutberlet, I., & De Vos, M. (2015). Real-time EEG feedback during simultaneous EEG-fMRI identifies the cortical signature of motor imagery. *NeuroImage*, *114*, 438–447. https://doi.org/10.1016/j.neuroimage.2015.04.020

## SUPPORTING INFORMATION

Additional supporting information can be found online in the Supporting Information section at the end of this article.

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