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Near-infrared spectroscopy and electroencephalography neurofeedback for binge-eating disorder: an exploratory randomized trial

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Abstract

Background. Binge-eating disorder (BED) co-occurs with neurobehavioral alterations in the processing of disorder-relevant content such as visual food stimuli. Whether neurofeedback (NF) directly targeting them is suited for treatment remains unclear. This study sought to determine feasibility and estimate effects of individualized, functional near-infrared spectroscopy-based real-time NF (rtfNIRS-NF) and high-beta electroencephalography-based NF (EEG-NF), assuming superiority over waitlist (WL).

Methods. Single-center, assessor-blinded feasibility study with randomization to rtfNIRS-NF, EEG-NF, or WL and assessments at baseline (*t*0), postassessment (*t*1), and 6-month follow-up (*t*2). NF comprised 12 60-min food-specific rtfNIRS-NF or EEG-NF sessions over 8 weeks. Primary outcome was the binge-eating frequency at *t*1 assessed interview-based. Secondary outcomes included feasibility, eating disorder symptoms, mental and physical health, weight management-related behavior, executive functions, and brain activity at *t*1 and *t*2.

Results. In 72 patients (intent-to-treat), the results showed feasibility of NF regarding recruitment, attrition, adherence, compliance, acceptance, and assessment completion. Binge eating improved at *t*1 by −8.0 episodes, without superiority of NF *v.* WL (−0.8 episodes, 95% CI −2.4 to 4.0), but with improved estimates in NF at *t*2 relative to *t*1. NF was better than WL for food craving, anxiety symptoms, and body mass index, but overall effects were mostly small. Brain activity changes were near zero.

Conclusions. The results show feasibility of food-specific rtfNIRS-NF and EEG-NF in BED, and no posttreatment differences *v.* WL, but possible continued improvement of binge eating. Confirmatory and mechanistic evidence is warranted in a double-blind randomized design with long-term follow-up, considering dose–response relationships and modes of delivery.

Introduction

Binge-eating disorder (BED), characterized by recurrent non-compensatory binge eating (American Psychiatric Association, 2013), is the most prevalent eating disorder (Galmiche, Déchelotte, Lambert, & Tavolacci, 2019), co-occurring with mental and physical health impairments including obesity (Udo & Grilo, 2018). Recent research demonstrated neurobehavioral alterations in BED especially in the processing of disorder-relevant content such as visual food stimuli (Kober & Boswell, 2018; Waltmann, Herzog, Horstmann, & Deserno, 2021). These included a pronounced fronto-central high beta activity (21–32 Hz) measured by electroencephalography (EEG; Blume, Schmidt, & Hilbert, 2019; Hiluy *et al.*, 2021), which is commonly related to tense arousal (Pino & Romano, 2022). Further, a hypoactivation in a prefrontal inhibitory control network was consistently found through functional magnetic resonance imaging (fMRI; Donnelly *et al.*, 2018; Mele, Alfano, Cotugno, & Longarzo, 2020; Saruco & Pleger, 2021; Steward, Menchon, Jiménez-Murcia, Soriano-Mas, & Fernandez-Aranda, 2017) and functional near-infrared spectroscopy (fNIRS; Rösch *et al.*, 2021; Veit *et al.*, 2021), a novel optical imaging method measuring the neocortical neuronal activity.

To advance treatment for BED (Hilbert et al., 2019; Linardon, Wade, de la Piedad Garcia, & Brennan, 2017), neuromodulation interventions are being developed (Dalton, Campbell, & Schmidt, 2017; Forcano, Mata, de la Torre, & Verdejo-Garcia, 2018; Imperatori, Mancini,

Della Marca, Valenti, & Farina, 2018; Val-Laillet et al., 2015), directly targeting food-related neurobehavioral alterations. Neurofeedback (NF) is a noninvasive neuromodulation approach in which participants learn to self-regulate their brain activity, using online feedback through a brain–computer interface (Arns et al., 2017; Enriquez-Geppert, Huster, & Herrmann, 2017; Paret et al., 2019; Sitaram et al., 2017; Thibault, Lifshitz, & Raz, 2016). Following proof-of-concept studies (Schmidt & Martin, 2015, 2016, 2020), the first pilot randomized-controlled trial (RCT) in BED ($N = 43$) showed that food-specific EEG-NF targeting the fronto-central high beta/theta activity improved binge-eating symptomatology compared to a waiting period, as did a nonspecific slow cortical potentials training (Blume, Schmidt, Schmidt, Martin, & Hilbert, 2022). The effects remained stable over 3 months and were associated with changes in fronto-central beta and theta power.

While EEG-NF has potential disseminability into clinical practice, spatial resolution is lower than with NF based on functional imaging that allows for training of circumscribed brain areas. Food-specific real-time (rt) fMRI-NF approaches have been developed in proof-of-concept studies, however, with only one approach targeting individually determined regions of interest (ROIs) for maximizing spatial specificity (Ihssen, Sokunbi, Lawrence, Lawrence, & Linden, 2017; Sokunbi, Linden, Habes, Johnston, & Ihssen, 2014). In contrast, rtfNIRS-NF has not been explored for BED, despite its increased dissemination potential. In overweight and obesity (Percik et al., 2019) and other psychological conditions (Hudak et al., 2017; Kimmig et al., 2019; Kohl et al., 2020; Marx et al., 2015) emerging evidence supports feasibility of rtfNIRS-NF over prefrontal areas.

This exploratory RCT was aimed at evaluating the feasibility and estimating effects of two disseminable food-specific NF approaches for BED *v.* waitlist (WL): individualized rtfNIRS-NF, adapted from rtfMRI-NF (Ihssen et al., 2017; Sokunbi et al., 2014), and high-beta EEG-NF (Blume et al., 2022). It was hypothesized that rtfNIRS-NF would be feasible and estimates for efficacy would be better than for WL.

Methods

Study design and procedure

The Near-Infrared Spectroscopy Neurofeedback for Binge-Eating Disorder (NIRSBED) study is a single-center, assessor-blinded feasibility study with randomization to rtfNIRS-NF, EEG-NF, or WL. Ethical approval was granted by the Ethical Committee of Leipzig University (476/17-ek). The study protocol was registered at the German Clinical Trials Register (DRKS00014752; Supplement 1). Patients' written informed consent was obtained prior to enrollment. Assessments took place at baseline ($t0$), 8 weeks following randomization [postassessment ($t1$), representing the end of rtfNIRS- or EEG-NF or the end of the waiting period], and at 6-month follow-up ($t2$).

Participants

Adults with BED were recruited at Leipzig University Medical Center through advertising and clinic referrals between 06/2018 and 03/2020 (eFigure 1, Supplement 2), offering 80€ for compensation. Adults ⩾18 years with a DSM-5 (1) diagnosis of BED or BED of low frequency and/or limited duration (eTable 1, Supplement 2) and a body mass index (BMI, kg/m^2) between 25.0 and 44.9 kg/m^2 were included (eMethods, Supplement 2 for full inclusion/exclusion criteria).

Treatment

Both NF treatments were manualized and designed procedurally similar (eMethods, Supplement 2). Patients received 12 individual 60-min food-specific NF treatment sessions over 8 weeks, for which personally appetizing food pictures (Blechert, Meule, Busch, & Ohla, 2014) were selected at baseline based on self-reported craving. Each NF session comprised regulation trials, during which real-time brain activity feedback was continuously provided upon/through presentation of the food pictures, and transfer trials, where feedback was only provided in the trials' final seconds (eFigure 2, Supplement 2).

rtfNRS-NF, designed based on rtfMRI-NF work (Ihssen et al., 2017; Sokunbi et al., 2014), provided feedback for an individually selected ROI in brain areas known to be involved in inhibition and self-control, including the dorsolateral prefrontal cortex (dlPFC; e.g. Lavagnino, Arnone, Cao, Soares, & Selvaraj, 2016; Negoro et al., 2010; Wu et al., 2023) and the inferior frontal gyrus (IFG; e.g. Aron, Robbins, & Poldrack, 2014; Hampshire, Chamberlain, Monti, Duncan, & Owen, 2010; eFigure 3, Supplement 2). Unilateral ROIs were determined through a functional localizer task (food-specific Go/NoGo task) performed at the outset of treatment. During the localizer task, it was possible to identify two adjacent channels with highest activation indicating that the covered brain area was involved in the neuronal processing of the food stimuli. During regulation and transfer trials, the respective ROI was trained in terms of an upregulation to further increase food-specific self-regulation. Therefore, patients were asked to minimize the size of the personally appetizing food pictures on the screen. The picture size in regulation trials served to visualize the neural activity level in the selected feedback channels. The pictures became smaller if the measured oxygenated hemoglobin signal in the individual ROI exceeded the value of the previous sampling point; otherwise, the pictures became larger, though they could not exceed their initial size. Mirror trials presenting static food pictures were performed after each regulation trial as perceptual control condition.

EEG-NF, building on a validated protocol for BED (Blume et al., 2022), targeted the reduction of high beta activity over fronto-central areas via four electrodes (eFigure 3, Supplement 2). After the presentation of a personally appetizing food picture, patients were shown their current beta and muscle activity via two continuously moving bars on the screen. Patients were instructed to decrease the neural activity bar below a predefined line, which represented patients' baseline high beta activity, while keeping muscle activity down.

In both NF treatments, patients were encouraged to develop own strategies for brain activity regulation, without giving example strategies. As usual (Gevensleben et al., 2009; Gevensleben, Moll, & Heinrich, 2010), NF was supplemented by homework assignments following cognitive-behavioral principles, fostering transfer of NF skills to daily life. Essentially, in a graded procedure, patients were instructed to look at individually selected food pictures or real food in daily life and, if they had an appetite, to use the mental strategies they had worked out in the neurofeedback sessions. Homework achievements and difficulties were discussed, and new homework activities were given at the end of each session, lasting about 5 min. Both NF treatments were provided by trained master- or doctoral level clinical psychologists under regular supervision of AH.

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Wait-list control condition

WL patients were guaranteed rtfNIRS-NF after an 8-week waiting period and instructed not to seek any other treatment for BED during the WL period.

Randomization and sample size estimation

After baseline assessment, patients were randomized at the Clinical Trial Centre (CTC) of Leipzig University, ensuring concealment of allocation. The computer-assisted randomization was based on a minimization algorithm with a stochastic component (Pocock & Simon, 1975) and was stratified by BMI (<35.0, ⩾35.0 kg/m^2), sex (female, male), and current participation in a behavioral weight-loss program (yes, no), with an allocation ratio of 1:1:1.

Based on available evidence on the reduction of binge eating in BED (Blume et al., 2022) or overeating in restrained eaters (Schmidt & Martin, 2015; 2016) through EEG-NF, an effect of $d = 0.55$ was assumed. Considering a drop-out rate of 20% (Blume et al., 2022; Schmidt & Martin, 2015; 2016), 26 patients per group were to be randomized to reach a power of 80% with $\alpha = 0.05$ using t test comparing the pooled intervention arms to the control arm. Given the pilot nature of the trial, the expected width of the 95% confidence interval (CI) was also taken into account in planning, which was found to be 0.88 s.d..

Measures

Measures of feasibility included patients' treatment compliance and treatment evaluation at the final NF session, and adherence to treatment sessions and assessment completion at $t1$ (eMethods, Supplement 2). As primary outcome for effect estimation, the number of objective binge-eating episodes (OBEs) over the past 28 days was determined at $t1$ using the Eating Disorder Examination (EDE; Fairburn, Cooper, & O'Connor, 2008; Hilbert & Tuschen-Caffier, 2016a). The EDE is a semi-structured interview that was conducted face-to-face by trained and regularly supervised research assistants (B.Sc. or M.Sc. Psychology level) blinded to randomization. For secondary outcomes, the number of OBEs at $t2$ and abstinence from binge eating and remission from BED at $t1$ and $t2$ were assessed using the EDE. All other secondary outcomes were evaluated at $t1$ and $t2$. Specifically, eating disorder symptoms were determined by the Eating Disorder Examination-Questionnaire (EDE-Q; Hilbert and Tuschen-Caffier, 2016b) and Food Cravings Questionnaire-Trait-reduced (FCQ-Tr; Meule, Hermann, and Kübler, 2014). For assessing weight management-related behaviors, the General Self-Efficacy Scale (GSES; Schwarzer and Jerusalem, 1995) and the Difficulties in Emotion Regulation Questionnaire (DERS; Gratz and Roemer, 2004) were used. Patients' mental health was evaluated by the Patient Health Questionnaire (PHQ-D; Spitzer, Kroenke, Williams, and Patient Health Questionnaire Primary Care Study Group, 1999), the Generalized Anxiety Disorder 7 (GAD-7; Löwe et al., 2008), and the Short Form Health Survey (SF-12; Ware, Kosinski, and Keller, 1996). Regarding physical health, patients' BMI was derived from objectively measured body weight and height using calibrated instruments, and the waist-to-hip ratio was derived from objectively measured circumferences. Executive functioning was assessed by neuropsychological tests on cognitive flexibility (Trail Making Test; Rodewald, Weisbrod, and Aschenbrenner, 2014), planning (Tower of London; Kaller, Unterrainer, Kaiser, Weisbrod, and Aschenbrenner, 2015), inhibition (Stop-Signal Task and Go/No-Go; Kaiser, Aschenbrenner, Pfüller, Roesch-Ely, and Weisbrod, 2015), and decision making (Cards and Lottery Task; Müller, Schiebener, Stöckigt, and Brand, 2017).

For rtfNIRS-NF, brain-based regulation success in the individual ROI was determined by the brain signal values for the NF (i.e. regulation, transfer) *v.* perceptual cue control condition (i.e. mirror; Santosa, Zhai, Fishburn, and Huppert, 2018). For EEG-NF, resting-state EEG signals for theta, alpha, and (high) beta activity were obtained at $t0$ and $t1$ in an eyes-open, eyes-closed, and food-specific condition. Adverse events were assessed in a standardized manner at each NF session in verbal and written format.

Data management and analysis

Data management was performed by the CTC and monitored for completeness, consistency, and plausibility. Postassessment data were released after study completion only, and interim analyses were not conducted. Data were analyzed from 08/2021–10/2022.

Statistical methods

For the primary endpoint, a closed-testing procedure was used to test for a difference between randomization arms at postassessment (eMethods, Supplement 2). First, a test for the pooled intervention arms *v.* the control arm was considered and, if significant at the 5% level, tests for the three pairwise comparisons were performed. If the first test was not significant, estimates and 95% CI for the pairwise contrasts were provided without a formal test. For all other endpoints, formal statistical testing was not performed because of the pilot nature of the trial. Instead, estimates with 95% CI were provided.

At postassessment, generalized linear models were used with baseline value and stratification variables as covariates and the arm as the independent variable. Multiple imputation with 50 sets was used for missing data. At follow-up, generalized linear mixed models were used for the two intervention arms with time treated categorically, the stratification variables and arm allocation as fixed effects and patient as a random term. Besides unstandardized B coefficients, standardized β coefficients were reported as effect size measure (small, $\beta \geqslant 0.2$, medium, $\beta \geqslant 0.5$, large, $\beta \geqslant 0.8$; Cohen, 1988). Analyses were performed using the software R, version 4.1.1.

Results

Participants

Of 316 volunteers screened for eligibility over the telephone, $N = 78$ patients met inclusion criteria, determined by in-person assessment (Fig. 1; for changes to recruitment because of the COVID-19 pandemic, see eMethods, Supplement 2). Patients were randomized to rtfNIRS-NF ($n = 28$; 3 exclusions due to fNIRS programming problems, resulting in $n = 25$), EEG-NF ($n = 25$), or WL ($n = 25$), with 72 patients retained for intent-to-treat analysis (Table 1). Across arms, treatment dropout, defined as attending ⩽5 NF sessions, encompassed 11% (5/47 rtfNIRS-NF: 3, EEG-NF: 2). Information on control variables can be found in eTable 3 (Supplement 2).

Feasibility

Feasibility was documented including timely recruitment, low attrition, and high assessment completion (Supplement 2).

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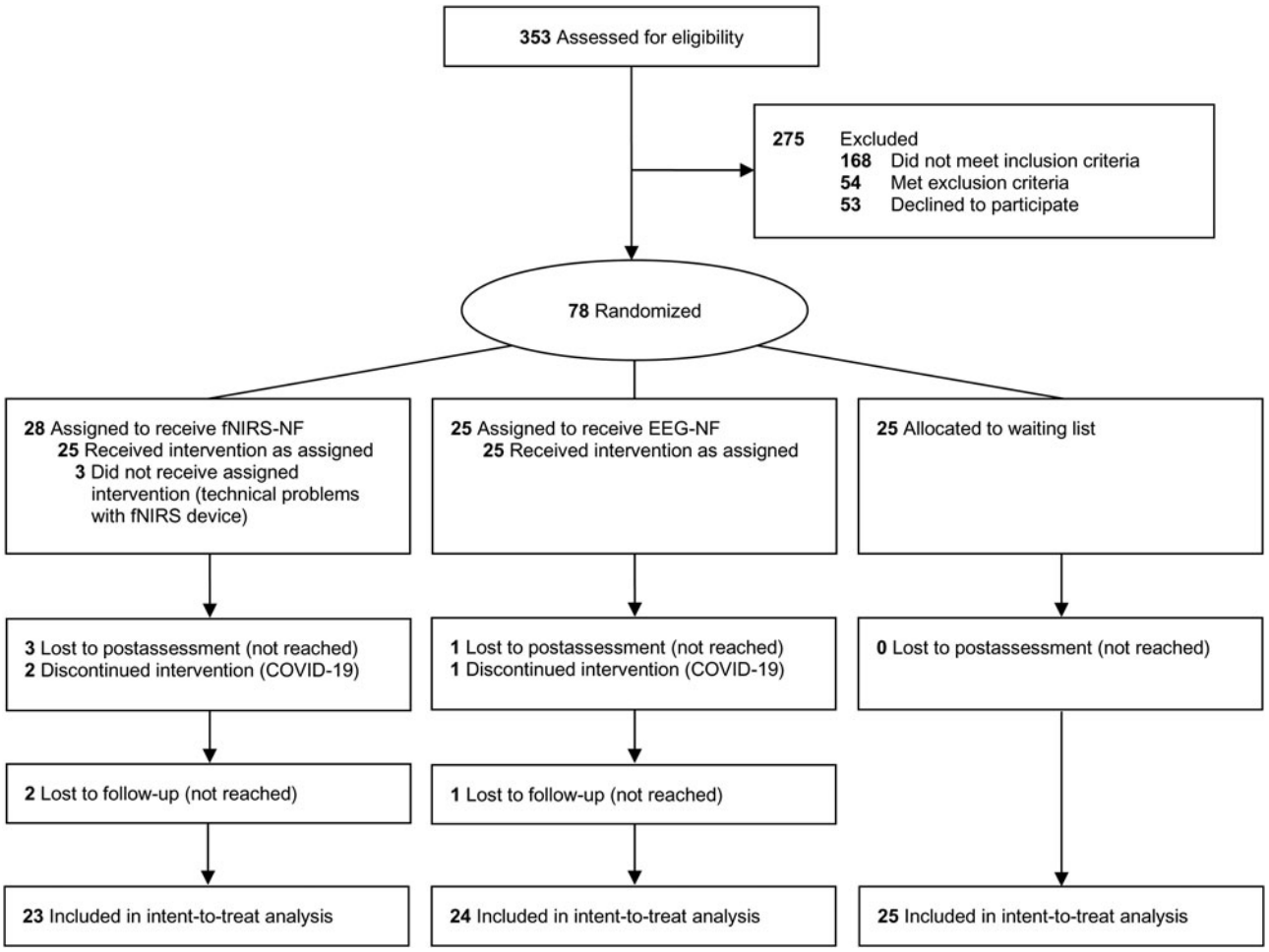

Figure 1. CONSORT flow diagram.

Adherence was high with patients attending 10.6 ± 2.9 (rtfNIRS-NF) and 9.5 ± 3.3 (EEG-NF) out of 12 sessions. Patients' session-wise treatment evaluation and therapist-rated patient compliance at posttreatment was moderate-to-high, with higher acceptance (i.e. greater treatment and strategy success for eating behaviors) and compliance in rtfNIRS-NF *v.* EEG-NF (eTables 5-6, Supplement 2).

Primary outcome

The number of OBEs decreased in rtfNIRS-NF, EEG-NF, and WL from 13.9, 12.1, and 12.6 to 5.3, 4.9 and 6.5, respectively (Table 2). The overall change from baseline was −8.0 episodes (95% CI −12.2 to −3.8). Compared to WL, the pooled NF arms had −0.8 fewer episodes (95% CI −4.0 to 2.4, $p = 0.61$), indicating less than a small effect (Table 3). Individually, rtfNIRS-NF had 1.0 (95% CI −2.7 to 4.7) and EEG-NF 0.6 (95% CI −3.2 to 4.4) fewer episodes than WL. Sensitivity analyses without imputation; with optimistic scenarios; and with pessimistic scenarios for missing data revealed similar results.

Secondary outcomes

Abstinence from binge eating was 23, 30, and 40% for rtfNIRS-NF, EEG-NF, and WL, respectively, and remission from BED was 41, 35, and 48 at *t*1. At *t*2, abstinence and remission rates increased to 55 and 60% in rtfNIRS-NF and 50 and 59% in EEG-NF.

Pooled intervention effects were found for greater reductions in food craving and anxiety (small effects), and BMI (very small effect) at *t*1 compared to WL (Table 3). Small effects in eating and weight concern, depressive symptoms, physical quality of life favored NF, while small effects in restraint and executive functioning favored WL (planning, decision making; Table 3, eTable 8, Supplement 2).

No differences between intervention arms were observed, with small effects indicating greater improvements in eating disorder-related aspects for rtfNIRS-NF and lower enhancements in general mental health (Table 4). Favorable time effects emerged for the majority of outcomes in both NF arms (Table 4, eResults; eTables 9-10, Supplement 2).

Safety

In the intervention arms, 74 adverse events were reported amongst 30 patients, with two events (moderate headaches: rtfNIRS-NF) judged to be attributable to NF (eResults; eTable 13, Supplement 2). There were two serious adverse events (acute hearing loss, salivary gland tumor: EEG-NF) unrelated to NF

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Table 1. Baseline sociodemographic characteristics and motivation

	rtfNIRS-NF ($n = 23$)	EEG-NF ($n = 24$)	Control ($n = 25$)	Total ($N = 72$)
	No. (%)	No. (%)	No. (%)	No. (%)
Sex, female	19 (83%)	19 (79%)	19 (76%)	57 (79%)
Age, mean (s.d.), years	48.6 (12.5)	47.0 (14.4)	45.2 (13.7)	46.9 (13.4)
Education				
⩾12 years	12 (52%)	13 (54%)	11 (44%)	36 (50%)
<12 years	11 (48%)	11 (46%)	14 (56%)	36 (50%)
Household income/month, €				
<1000€	2 (9%)	8 (33%)	3 (12%)	13 (18%)
1000–2000€	8 (35%)	7 (29%)	4 (16%)	19 (26%)
2000–4000€	9 (39%)	5 (21%)	8 (32%)	22 (31%)
⩾4000€	2 (9%)	3 (12%)	6 (24%)	11 (15%)
Not stated	2 (9%)	1 (4%)	4 (16%)	7 (10%)
Body weight, mean (s.d.), kg	102.9 (14.2)	106.9 (18.6)	107.3 (14.7)	105.8 (15.9)
Currently in weight loss program	2 (9%)	0 (0%)	3 (12%)	5 (7%)
Weight status				
Overweight (BMI 25–<30 kg/m^2)	3 (13%)	3 (12%)	2 (8%)	8 (11%)
Obesity class 1 (BMI 30–<35 kg/m^2)	6 (26%)	7 (29%)	7 (28%)	20 (28%)
Obesity class 2 (BMI 35–<40 kg/m^2)	6 (26%)	5 (21%)	7 (28%)	18 (25%)
Obesity class 3 (BMI ⩾40 kg/m^2)	8 (35%)	9 (38%)	9 (36%)	26 (36%)
Eating disorder diagnosis (DSM-5)[a]				
BED	18 (78%)	20 (83%)	19 (76%)	57 (79%)
BED of low frequency and/or limited duration	5 (22%)	4 (17%)	6 (24%)	15 (21%)
Mental comorbidity[b]				
Major depression (PHQ-D ⩾10)	11 (50%)	10 (43%)	8 (32%)	29 (41%)
Generalized anxiety disorder (GAD-7 ⩾10)	3 (14%)	6 (26%)	4 (16%)	13 (19%)
Therapy expectations (1–10)[c]				
Motivated to change eating behavior	8.6 (1.7)	8.9 (1.3)	8.1 (1.5)	8.5 (1.5)
Motivated to maintain change long term	8.8 (1.3)	8.6 (1.7)	8.2 (1.5)	8.5 (1.5)
Confidence to maintain change long term	6.6 (2.0)	6.3 (2.5)	6.5 (2.0)	6.5 (2.2)

BED, binge-eating disorder; BMI, body mass index (kg/m^2), DSM, Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition; EEG-NF, electroencephalography-neurofeedback; GAD-7, Generalized Anxiety Disorder 7-Item Scale (0–21* less favorable scores are asterisked); PHQ-D, Patient Health Questionnaire-Depression Scale (0–27*); rtfNIRS-NF, real-time functional near-infrared spectroscopy-neurofeedback.
[a]Eating disorder diagnosis was determined through the Eating Disorder Examination.
[b]Data missing from two patients.
[c]Data missing from one patient.

Brain activity

For rtfNIRS-NF, changes in hemoglobin signals during regulation or transfer *v.* mirror trials pointed in the expected direction, but were very close to zero (eTable 11, Supplement 2). No systematic within or between effects for mean picture size during regulation trials were found. The mean picture size showed a very small-sized association with a reduction in OBEs at *t*1, $\beta = -0.19$ (95% CI −0.67 to 0.28).

Against expectations, there were less than small-sized changes in EEG (high) beta activity at *t*2 *v.* *t*1 across conditions (food presentation, eyes-open, eyes-closed; eResults and eTable 12, Supplement 2). No within- or between-session effects for mean beta activity were detected. The mean beta activity showed a very small-sized association with a reduction in OBEs at *t*1, $\beta = 0.04$ (95% CI −0.37 to 0.44).

Discussion

This exploratory, assessor-blind RCT comparing food-specific individualized rtfNIRS-NF and food-specific high-beta EEG-NF to WL in adults with BED showed feasibility regarding recruitment, attrition, adherence, assessment completion, acceptance, and compliance. Regarding the primary outcome, both NF arms revealed a reduction in binge eating by 8 episodes at

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Table 2. Raw data for the primary and secondary outcomes

	rtfNIRS-NF			EEG-NF			Control	
	Pre-treatment	Post-assessment	6-month follow-up	Pre-treatment	Post-assessment	6-month follow-up	Pre-treatment	Post-assessment
	Mean (s.d.)	Mean (s.d.)	Mean (s.d.)	Mean (s.d.)	Mean (s.d.)	Mean (s.d.)	Mean (s.d.)	Mean (s.d.)
Eating disorder symptoms								
Binge-eating episodes (EDE)[a]	13.9 (12.5)	5.3 (6.5)	4.3 (6.9)	12.1 (8.5)	4.9 (6.6)	4.1 (6.7)	12.6 (12.1)	6.5 (8.7)
Abstinence from binge eating (EDE), No. (%)	1/23 (4%)	5/22 (23%)	11/20 (55%)	0/24 (0%)	7/23 (30%)	11/22 (50%)	1/25 (4%)	10/25 (40%)
Remission from BED (EDE), No. (%)	0/23 (0%)	9/22 (41%)	12/20 (60%)	0/24 (0%)	8/23 (35%)	13/22 (59%)	0/25 (0%)	12/25 (48%)
Eating disorder psychopathology (EDE-Q)								
Restraint	1.66 (1.21)	2.18 (1.07)	1.64 (0.85)	1.92 (1.38)	2.03 (1.23)	2.26 (1.79)	1.70 (1.32)	1.57 (1.48)
Eating concern	2.27 (1.03)	1.51 (1.10)	1.46 (0.98)	2.15 (1.66)	1.80 (1.06)	1.43 (1.30)	1.99 (1.50)	1.97 (1.57)
Weight concern	3.23 (0.84)	2.86 (1.01)	2.78 (1.02)	3.73 (1.26)	3.22 (1.16)	3.18 (1.44)	3.39 (1.23)	3.31 (1.43)
Shape concern	3.65 (1.05)	3.30 (1.13)	3.25 (1.34)	4.29 (1.19)	3.73 (1.31)	3.39 (1.58)	3.83 (1.29)	3.66 (1.59)
Global score	2.70 (0.82)	2.46 (0.88)	2.28 (0.91)	3.02 (1.07)	2.69 (0.89)	2.57 (1.20)	2.73 (1.11)	2.63 (1.26)
Food craving (FCQ-T-r)	57.5 (11.2)	46.4 (12.1)	44.2 (14.7)	60.5 (12.8)	52.4 (13.7)	49.9 (13.4)	53.4 (13.9)	52.7 (16.2)
Weight management-related behaviors								
Self-efficacy (GSES)	28.1 (4.9)	27.6 (4.1)	26.9 (5.7)	26.4 (5.3)	25.8 (5.2)	26.6 (5.4)	27.4 (6.4)	27.1 (5.2)
Emotion regulation (DERS)	86.0 (20.8)	84.2 (21.4)	85.9 (21.9)	87.9 (23.8)	86.2 (24.3)	84.9 (18.0)	92.3 (30.8)	89.8 (29.1)
Mental health								
Depressive symptoms (PHQ-D)	9.4 (3.2)	8.5 (3.3)	9.2 (4.5)	9.0 (4.8)	8.2 (4.1)	8.0 (3.4)	8.2 (4.4)	9.0 (5.4)
Anxiety symptoms (GAD-7)	6.0 (3.5)	4.9 (3.3)	6.3 (4.1)	6.9 (4.8)	5.6 (4.4)	6.0 (3.6)	5.5 (4.1)	7.0 (5.3)
Quality of life (SF-12)								
Mental	42.1 (9.6)	44.5 (8.9)	42.2 (11.5)	42.4 (11.9)	44.8 (11.5)	44.3 (9.0)	43.3 (11.7)	44.1 (12.4)
Physical	42.0 (8.8)	43.5 (8.7)	40.5 (12.0)	43.7 (10.6)	44.1 (10.7)	43.1 (10.4)	46.2 (8.1)	43.1 (11.8)
Physical health								
Body mass index (kg/m^2)	36.6 (5.2)	36.1 (5.4)	36.5 (5.0)	36.8 (5.2)	36.3 (5.4)	36.4 (5.9)	36.8 (4.6)	37.2 (4.7)
Waist-to-hip ratio	0.89 (0.07)	0.88 (0.08)	0.90 (0.07)	0.88 (0.10)	0.88 (0.07)	0.86 (0.10)	0.89 (0.08)	0.88 (0.07)

DERS, Difficulties in Emotion Regulation Scale (36–180* less favorable scores are asterisked); 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; SF-12, 12-Item Short Form Health Survey (0*–100).

[a]Number of objective binge-eating episodes over the past 28 days.

Table 3. Secondary outcomes in intent-to-treat analyses at postassessment

	Intervention (pooled) *v.* control		rtfNIRS-NF *v.* control		EEG-NF *v.* control	
	B (95% CI)	β (95% CI)	B (95% CI)	β (95% CI)	B (95% CI)	β (95% CI)
Eating disorder symptoms						
Binge-eating episodes (EDE)[a]	−0.8 (−4.0 to 2.4)	−0.11 (−0.53 to 0.32)	−1.0 (−4.7 to 2.7)	−0.13 (−0.63 to 0.36)	−0.6 (−4.4 to 3.2)	−0.08 (−0.58 to 0.42)
Abstinence from binge eating (EDE)	0.57 (0.17 to 1.85)	–	0.39 (0.08 to 1.65)	–	0.75 (0.19 to 2.88)	–
Remission from BED (EDE)	0.52 (0.18 to 1.46)	–	0.64 (0.19 to 2.09)	–	0.43 (0.12 to 1.41)	–
Eating disorder psychopathology (EDE-Q)						
Restraint	0.46 (−0.11 to 1.04)	0.36 (−0.09 to 0.80)	0.64 (−0.03 to 1.30)	0.50 (−0.02 to 1.01)	0.28 (−0.40 to 0.95)	0.21 (−0.31 to 0.74)
Eating concern	−0.34 (−0.78 to 0.11)	−0.26 (−0.62 to 0.09)	−0.52 (−1.04 to 0.00)	−0.41 (−0.82 to 0.00)	−0.15 (−0.68 to 0.38)	−0.12 (−0.53 to 0.30)
Weight concern	−0.24 (−0.66 to 0.17)	−0.20 (−0.55 to 0.14)	−0.25 (−0.73 to 0.23)	−0.21 (−0.61 to 0.19)	−0.24 (−0.73 to 0.25)	−0.20 (−0.61 to 0.21)
Shape concern	−0.17 (−0.57 to 0.23)	−0.13 (−0.43 to 0.17)	−0.13 (−0.59 to 0.33)	−0.10 (−0.44 to 0.25)	−0.21 (−0.69 to 0.27)	−0.16 (−0.52 to 0.20)
Global score	−0.09 (−0.40 to 0.22)	−0.09 (−0.39 to 0.22)	−0.08 (−0.44 to 0.28)	−0.08 (−0.43 to 0.28)	−0.10 (−0.47 to 0.27)	−0.10 (−0.47 to 0.27)
Food craving (FCQ-T-r)	−6.0 (−11.6 to −0.4)	−0.42 (−0.82 to −0.02)	−7.5 (−13.9 to −1.1)	−0.53 (−0.98 to −0.07)	−4.3 (−11.0 to 2.3)	−0.31 (−0.77 to 0.16)
Weight management-related behaviors						
Self-efficacy (GSES)	0.20 (−1.14 to 1.55)	0.04 (−0.24 to 0.32)	−0.15 (−1.70 to 1.41)	−0.03 (−0.36 to 0.29)	0.56 (−1.01 to 2.14)	0.12 (−0.21 to 0.45)
Emotion regulation (DERS)	−1.46 (−8.11 to 5.18)	−0.06 (−0.33 to 0.21)	−0.53 (−8.25 to 7.19)	−0.02 (−0.33 to 0.29)	−2.41 (−10.26 to 5.43)	−0.10 (−0.41 to 0.22)
Mental health						
Depressive Symptoms (PHQ-D)	−1.26 (−2.84 to 0.33)	−0.29 (−0.66 to 0.08)	−1.26 (−3.11 to 0.59)	−0.29 (−0.73 to 0.14)	−1.26 (−3.13 to 0.61)	−0.29 (−0.73 to 0.14)
Anxiety symptoms (GAD-7)	−2.09 (−3.80 to −0.38)	−0.47 (−0.86 to −0.08)	−2.09 (−4.07 to −0.11)	−0.47 (−0.92 to −0.02)	−2.09 (−4.11 to −0.07)	−0.47 (−0.93 to −0.01)
Quality of life (SF-12)						
Mental	−1.4 (−6.1 to 3.3)	−0.13 (−0.56 to −0.31)	−1.0 (−6.4 to 4.4)	−0.09 (−0.60 to 0.41)	−1.7 (−7.3 to 3.8)	−0.16 (−0.68 to 0.36)
Physical	−2.9 (−7.0 to 1.2)	−0.28 (−0.69 to 0.12)	−3.4 (−8.2 to 1.4)	−0.33 (−0.80 to 0.14)	−2.4 (−7.3 to 2.4)	−0.24 (−0.71 to 0.24)
Physical health						
Body mass index (kg/m^2)	−0.62 (−1.06 to −0.17)	−0.12 (−0.21 to −0.03)	−0.63 (−1.15 to −0.11)	−0.12 (−0.23 to −0.02)	−0.61 (−1.13 to −0.09)	−0.12 (−0.22 to −0.02)
Waist-to-hip ratio	0.003 (−0.028 to 0.033)	0.04 (−0.39 to 0.47)	0.003 (−0.032 to 0.037)	0.04 (−0.45 to 0.52)	0.003 (−0.034 to 0.040)	0.04 (−0.48 to 0.57)

DERS, Difficulties in Emotion Regulation Scale; EDE, Eating Disorder Examination; EDE-Q, Eating Disorder Examination-Questionnaire; EEG-NF, electroencephalography-NF; 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; rtfNIRS-NF, real-time functional near-infrared spectroscopy-neurofeedback; SF-12, 12-Item Short Form Health Survey.
For continuous measures, a negative value indicates that A is clinically better than B for A *v.* B, e.g., the minus sign for the first column of the row 'Binge-eating episodes' indicates that the interventions are superior to the control arm. For the odds ratios (abstinence and remission), a value smaller than 1 indicates that the B is clinically better than A, i.e., odds for abstinence are higher in the control group than the intervention groups.
[a]Number of objective binge-eating episodes over the past 28 days.

Table 4. Secondary outcomes in rtfNIRS-NF and EEG-NF in intent-to-treat analyses at postassessment and 6-month follow-up

	Posttreatment *v.* pretreatment		Follow-up *v.* pretreatment		EEG-NF *v.* rtfNIRS-NF	
	B (95% CI)	β (95% CI)	B (95% CI)	β (95% CI)	B (95% CI)	β (95% CI)
Eating disorder symptoms						
Binge-eating episodes (EDE)[a]	−7.7 (−10.5 to −5.0)	−0.85 (−1.15 to −0.55)	−8.8 (−11.6 to −6.0)	−0.97 (−1.27 to −0.66)	−1.1 (−5.0 to 2.9)	−0.12 (−0.54 to 0.31)
Abstinence from binge eating (EDE)[b]	–	–	–	–	1.1 (0.3 to 3.6)	–
Remission from BED (EDE)[b]	–	–	–	–	0.8 (0.2 to 3.9)	–
Eating disorder psychopathology (EDE-Q)						
Restraint	0.32 (−0.12 to 0.75)	0.25 (−0.09 to 0.58)	0.14 (−0.31 to 0.58)	0.11 (−0.24 to 0.45)	0.09 (−0.51 to 0.69)	0.07 (−0.39 to 0.53)
Eating concern	−0.55 (−0.87 to −0.23)	−0.44 (−0.70 to −0.18)	−0.73 (−1.06 to −0.40)	−0.59 (−0.85 to −0.32)	0.11 (−0.57 to 0.78)	0.09 (−0.45 to 0.62)
Weight concern	−0.44 (−0.72 to −0.17)	−0.38 (−0.62 to −0.14)	−0.50 (−0.78 to −0.21)	−0.43 (−0.67 to −0.19)	0.47 (−0.15 to 1.08)	0.40 (−0.12 to 0.92)
Shape concern	−0.43 (−0.72 to −0.14)	−0.33 (−0.55 to −0.11)	−0.62 (−0.91 to −0.32)	−0.48 (−0.70 to −0.25)	0.42 (−0.29 to 1.13)	0.32 (−0.21 to 0.86)
Global score	−0.27 (−0.50 to −0.03)	−0.27 (−0.51 to −0.03)	−0.42 (−0.66 to −0.18)	−0.43 (−0.67 to −0.18)	0.27 (−0.27 to 0.81)	0.28 (−0.27 to 0.82)
Food craving (FCQ-T-r)[c]	−9 (−13 to −5)	−0.67 (−0.94 to −0.39)	−12 (−16 to −8)	−0.85 (−1.14 to −0.57)	5 (−2 to 11)	0.32 (−0.14 to 0.79)
Weight management-related behaviors						
Self-efficacy (GSES)	−0.2 (−1.2 to 0.9)	−0.03 (−0.24 to 0.18)	0.1 (−1.0 to 1.2)	0.02 (−0.20 to 0.23)	−1.0 (−4.0 to 1.9)	−0.20 (−0.78 to 0.37)
Emotion regulation (DERS)	−4.4 (−8.5 to −0.2)	−0.20 (−0.39 to −0.01)	−4.2 (−8.5 to 0.0)	−0.20 (−0.39 to −0.00)	−0.8 (−13.8 to 12.2)	−0.04 (−0.63 to 0.56)
Mental health						
Depressive symptoms (PHQ-D)	−1.0 (−2.1 to 0.2)	−0.25 (−0.53 to 0.04)	−0.8 (−2.0 to 0.3)	−0.21 (−0.51 to 0.08)	−1.1 (−3.2 to 1.0)	−0.28 (−0.80 to 0.25)
Anxiety symptoms (GAD-7)	−1.2 (−2.3 to −0.1)	−0.30 (−0.57 to −0.04)	−0.5 (−1.6 to 0.6)	−0.13 (−0.41 to 0.15)	0.0 (−2.1 to 2.2)	0.01 (−0.52 to 0.55)
Quality of life (SF-12)						
Mental	−0.7 (−3.1 to 1.7)	−0.07 (−0.31 to 0.17)	0.7 (−1.8 to 3.2)	0.07 (−0.17 to 0.31)	−1.9 (−7.5 to 3.6)	−0.19 (−0.73 to 0.35)
Physical	−2.4 (−5.4 to 0.6)	−0.23 (−0.52 to 0.05)	−1.2 (−4.2 to 1.9)	−0.11 (−0.41 to 0.18)	−1.6 (−7.1 to 3.9)	−0.15 (−0.67 to 0.37)
Physical health						
Body mass index (kg/m^2)	−0.29 (−0.73 to 0.16)	−0.05 (−0.14 to 0.03)	−0.64 (−1.10 to −0.18)	−0.12 (−0.21 to −0.03)	0.03 (−3.27 to 3.32)	0.01 (−0.61 to 0.62)
Waist-to-hip ratio	0.003 (−0.014 to 0.020)	0.03 (−0.17 to 0.24)	−0.007 (−0.025 to 0.011)	−0.08 (−0.29 to 0.13)	−0.007 (−0.044 to 0.031)	−0.08 (−0.52 to 0.36)

DERS, Difficulties in Emotion Regulation Scale; EDE, Eating Disorder Examination; EDE-Q, Eating Disorder Examination–Questionnaire; EEG-NF, electroencephalography-neurofeedback; 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; rtfNIRS-NF, real-time functional near-infrared spectroscopy-neurofeedback; SF-12, 12-Item Short Form Health Survey. For continuous measures, a negative value indicates that A is clinically better than B for A *v.* B, e.g., the minus sign for the first column of the row 'Binge-eating episodes' indicates that there were fewer episodes at posttreatment than at pretreatment. For the odds ratios (abstinence and remission), a value smaller than 1 indicates that the B is clinically better than A.

[a]Number of objective binge-eating episodes over the past 28 days.

[b]Since patients were not abstinent pretreatment and had a diagnosis, odds ratios cannot be provided for comparisons with pretreatment.

[c]There is evidence for interaction between arm and time meaning that the results presented here without interaction are taken from models inappropriate to the data. Models with interaction are presented in eTable 10.

postassessment, which was however comparable to that in the WL arm. Abstinence from binge eating was lower (rtfNIRS-NF: 23%, EEG-NF: 30%) relative to WL (40%), but amounted to 55 and 50% at 6 months following rtfNIRS- and EEG-NF, similar to abstinence achieved by psychotherapy, the most well-established treatment approach for BED (Hilbert et al., 2019, 2020; Monteleone et al., 2022) and markedly higher than abstinence found in a no treatment control condition over a broadly comparable timeframe (10.3%; Schag et al., 2019). In contrast, abstinence following WL was unusually high when compared to meta-analytic results, and a 6-month follow-up was not assessed. The magnitude of effects on binge eating was comparable to a previous high-beta/theta EEG-NF pilot study for BED, with a similar increase in abstinence at 3-month follow-up in one NF arm (Blume et al., 2022), but was lower for overeating tendencies in preclinical pilot studies of female restrained eaters (Schmidt & Martin, 2015, 2016), suggesting more readily achievable changes. Overall, the results suggest support for a delayed NF effect on binge eating, accompanied by increased effect sizes in the improvement of further eating disorder symptoms at 6-month follow-up (Table 4). Delayed consolidation effects have been found following NF of other mental disorders, possibly associated with NF-induced neuroplasticity following neural self-regulation (Rubia, Westwood, Aggensteiner, & Brandeis, 2021). A double-blind randomized design with long-term follow-up across NF and WL conditions is needed to evaluate a possible delayed improvement of binge eating controlling for time and assessment effects.

Regarding secondary outcomes, both NF arms improved food craving, anxiety, and BMI at postassessment with largest differences in effects relative to WL, and the majority of further psychological outcomes showed small-sized advantages (e.g. eating concern, weight concern, depressive symptoms, and physical quality of life), with maintenance of changes or – regarding eating disorder symptoms – further enlargement of effects sizes over 6 months following NF. These results are consistent with NF findings in eating disturbances in general (Imperatori et al., 2018), but effect sizes fell below those following more intense psychotherapy for BED (Hilbert et al., 2019, 2020). Increased restraint following NF *v.* WL reflects a stronger patient focus on the cognitive control of food intake with – for BED – both functional and dysfunctional facets operationalized in the EDE-Q restraint scale (Fairburn & Beglin, 2008). Likewise, dietary self-efficacy was increased after food-specific high-beta NF in restrained eaters (Schmidt & Martin, 2015, 2016). Small-sized advantages in improving planning and decision-making in WL *v.* NF are consistent with overall unexpectedly favorable changes in the WL arm.

There was little indication for differences between NF modalities: Descriptively, rtfNIRS-NF reached higher acceptance, especially greater patient treatment evaluation regarding eating behavior, and therapist-rated patient compliance as well as session attendance, whereas EEG-NF had small advantages in patient treatment evaluation regarding relaxation. Due to the availability of different NF device systems, the food-related feedback visualization and/or the more novel setup of rtfNIRS-NF may have been more convincing and appealing to the patients than the bar graph visualization and separate food stimuli presentation in EEG-NF. Most importantly, patients in EEG-NF exhibited reduced decision making at posttreatment and 6-month follow-up, whereas those in rtfNIRS-NF showed improved decision making with small-to-large effect sizes, possibly related to rtfNIRS-NF targeting the recruitment of prefrontal control networks when exposing patients to individually appetitive food stimuli. Small-sized advantages in some eating disorder symptoms (i.e. shape and weight concern, food craving) in rtfNIRS-*v.* EEG-NF, along with the tendency for improved general psychopathological aspects of self-efficacy and depression in EEG-NF – in conjunction with the differences in acceptance –, suggest a more eating disorder-focused *v.* more general psychopathology-focused therapeutic effect, which may be related to differences in targets and set-up of the NF modalities.

Little evidence was found supporting brain-based mechanisms of NF: Across rtfNIRS-NF sessions, brain activity changes in oxygenated and deoxygenated hemoglobin in the individual prefrontal ROI were mostly in the expected direction during NF *v.* perceptual cue control, but effect sizes were very close to zero, possibly reflecting heterogeneity related to individual ROIs. Likewise, the mean picture size showed substantial variations, and was at best weakly associated with posttreatment binge-eating reduction. Across and within EEG-NF sessions, (high) beta band activity over fronto-central regions during regulation was reduced with very small effect size, and mean beta activity displayed a very small association with posttreatment binge-eating reduction. That the pre- to posttreatment EEG did not reveal a consistent decrease in the relative (high) beta band power upon food presentation or control conditions contrasts changes documented for high-beta/theta EEG-NF in BED (Blume et al., 2022). Downregulation of high beta only is limited by the natural zero, possibly impeding learning and/or motivation in this study. In addition, in order to create procedurally similar NF protocols, the number and duration of EEG-NF regulation trials had been reduced and transfer trials had been newly inserted in this study, potentially limiting the occurrence of brain activity changes. Also, brain activity data were available from only 12/25 patients due to restrictions related to on-site testing during the COVID-19 pandemic (n = 5), unplanned treatment termination before the 12th session, where post-EEG recordings were typically scheduled (n = 5), and invalid EEG recordings (n = 3). While inconclusive results regarding brain regulation success are a general limitation to NF treatment research (Rubia et al., 2021), further evidence is warranted on NF mechanisms in BED, combining EEG and fNIRS or fMRI data to increase robustness of results for delineating within-session as well as pre- to posttreatment and follow-up change (Kohl et al., 2020; Soekadar, Kohl, Mihara, & von Lühmann, 2021). Safety was documented for both food-specific NF modalities with few adverse events, mostly headaches possibly related to rtfNIRS-NF, and only two serious adverse events unrelated to EEG-NF. Further investigations should specify potential negative or positive effects from regional NF in neighboring, connected, or contralateral brain regions, for example, using fMRI (Rubia et al., 2019).

This exploratory assessor-blind RCT of two new standardized NF protocols provided feasibility data and effect estimates with a low selection, information, and confounding bias, as documented by minimal inclusion and exclusion criteria; standardized, internationally well-established assessments, including validated face-to-face EDE (Fairburn et al., 2008; Hilbert & Tuschen-Caffier, 2016a; Hilbert & Tuschen-Caffier, 2016b) by trained, supervised assessors; statistical confounder control; low study dropout; and reporting along established guidelines (Ros et al., 2020; Schulz, Altman, & Moher, 2010). Although the EDE is commonly used in intervention research in BED, its assessment is based on retrospective recall and thus, recall biases cannot be excluded. Given the exploratory nature of this study with a small sample size, we refrained from significance testing except

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for the primary endpoint and reported effect estimates with 95% confidence intervals (Eldridge et al., 2016). Therefore, the results are preliminary and generalizability is limited. The sample size calculation assumed an effect size of 0.55 based on earlier research and the assumptions regarding variance led to the expectation of a 95% CI with a width of 0.88 s.d.. The latter was borne out with an observed 95% CI of 0.85 s.d.. However, the effect size of 0.55 was just beyond the edge of the 95% CI, which was 0.53, and was thus probably overoptimistic. This and the other small effect sizes observed in this single-blind exploratory RCT contrast with the moderate to large effect sizes of previous unblinded pilot RCTs of high-beta EEG-NF in restrained eaters (Schmidt & Martin, 2015; 2016), suggesting that a larger sample size of patients with BED is needed for a confirmatory blinded RCT of NF with similar potency.

To conclude, the results showed feasibility of both food-specific NF modalities in adult BED, and no posttreatment differences *v.* WL, but a possible continued improvement of binge eating beyond posttreatment. Being in an early stage of intervention design, rtfNIRS-NF appeared to have promise for the treatment of BED regarding its ease of use, individualization, acceptance, and eating disorder-focused effects, whereas high-beta EEG-NF yielded less consistent training effects than previous high-beta/theta EEG-NF (Blume et al., 2022). Larger-scale, double-blind research with credible sham control conditions is warranted to explore efficacy of NF in BED and determine short- and long-term mechanisms of action including brain activation and psychosocial or placebo effects (Schönenberg, Weingärtner, Weimer, & Scheeff, 2021; Thibault & Raz, 2017; Thibault, Veissière, Olson, & Raz, 2018). Dose-finding work will be essential to determine optimal treatment protocols (Kohl et al., 2020). Clinically, the overall small-sized improvements suggest an adjunctive rather than monotherapeutic use of food-specific NF for the majority of patients, for example, during nutritional management and/or food cue exposure in cognitive-behavioral therapy of BED (de Zwaan et al., 2017), which should be clarified in predictor analyses (Barth et al., 2022; Weber, Ethofer, & Ehlis, 2020).

Supplementary material. The supplementary material for this article can be found at https://doi.org/10.1017/S0033291723002350.

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Ethical standards. 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.

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