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Heart Rate Variability Biofeedback Reduces Food Cravings in High Food Cravers

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Abstract Heart rate variability (HRV) biofeedback has been reported to increase HRV while decreasing symptoms in patients with mental disorders. In addition, associations between low HRV and lowered self-regulation were found in non-clinical samples, e.g., in individuals with strong chocolate cravings or unsuccessful dieting. The current study aimed at decreasing food cravings with HRV-biofeedback in individuals frequently experiencing such cravings. Participants (N = 56) with strong or low food cravings associated with a lack of control over eating were selected from the local community. Half of the participants with strong cravings (craving-biofeedback; n = 14) performed 12 sessions of HRV-biofeedback while the other half (craving-control; n = 14) and a group with low cravings (non-craving-control; n = 28) received no intervention. Subjective food cravings related to a lack of control over eating decreased from pre- to post-measurement in the craving-biofeedback group, but remained constant in the control groups. Moreover, only the cravingbiofeedback group showed a decrease in eating and weight concerns. Although HRV-biofeedback was successful in reducing food cravings, this change was not accompanied by an increase in HRV. Instead, HRV decreased in the

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craving-control group. This study provides preliminary evidence that HRV-biofeedback could be beneficial for attenuating dysfunctional eating behavior although specific mechanisms remain to be elucidated.

Keywords Food cravings · Eating behavior · Cardiac autonomic regulation · Heart rate variability · Biofeedback

Introduction

Heart rate variability (HRV) refers to the variation of heart beat intervals and is influenced by sympathetic and parasympathetic input to the sino-atrial node of the heart. Increased parasympathetically (or vagally) mediated modulations increase HRV while increased sympathetic activation (or sympathovagal imbalance) decreases HRV (Task Force of The European Society of Cardiology and The North American Society of Pacing and Electrophysiology 1996). HRV is associated with overall health and several physical conditions (Britton and Hemingway 2004; Thayer et al. 2010). For instance, frequent exercise positively influences HRV while unhealthy behaviors like smoking or alcohol consumption decrease HRV (Britton and Hemingway 2004; Thayer et al. 2010). Beyond indexing physical health, HRV has also been suggested as an endophenotype of self- and emotion-regulation (Appelhans and Luecken 2006; Thayer and Lane 2009). Accordingly, attenuated vagal-cardiac control has been associated with several mental disorders such as depression and anxiety (see Appelhans and Luecken 2006 for a review), posttraumatic stress disorder (PTSD; Blechert et al. 2007) or alcohol abuse (Thayer et al. 2006). In a sample of alcohol-dependent patients, reduced HRV was particularly pronounced in patients reporting strong substance cravings (Ingjaldsson et al. 2003).

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In relation to eating behavior, body weight is inversely related to HRV such that underweight patients (e.g., those with anorexia nervosa) have high HRV, while obesity is accompanied by low HRV (Karason et al. 1999; Latchman et al. 2011; Mazurak et al. 2011). Accordingly, weight loss is associated with an increase in HRV (Karason et al. 1999). Few studies have examined HRV as a marker of eating-related self-regulation that is independent of current body mass. Vögele et al. (2009) investigated a sample of patients with bulimia nervosa and classified those as individuals with current dietary restriction or without dietary restriction according to their biochemical profile. Only fasting women presented with increased vagal-cardiac control as compared to healthy controls; BMI, however was equal in both groups (Coles et al. 2005; Vögele et al. 2009). The authors speculated that current fasting status with accompanying parasympathetic dominance could be an index of successful eating-related self-regulation. Another study compared HRV between obese patients with binge eating disorder (BED) and those without BED (Friederich et al. 2006). Although there was no baseline difference between groups, an augmented reduction of vagal-cardiac control was observed in obese patients with BED when mentally challenged which was also correlated to binge eating frequency (Friederich et al. 2006).

Recent studies investigated HRV in non-clinical, normal-weight samples, particularly its relationship to food cravings. Food cravings refer to an urgent desire, longing, or yearning for a particular kind of food which most often involves chocolate (Weingarten and Elston 1990, 1991). Rodríguez-Ruiz et al. (2009) found an association between low HRV and eating disorder symptoms in trait chocolate cravers. In a subsequent study, low HRV was associated with increased eve-blink startle magnitude in women with bulimic symptoms and frequent food cravings, which is suggestive of reduced emotion regulation abilities and inhibitory control (Rodríguez-Ruiz et al. 2012). Accordingly, frequent experiences of food cravings are strongly related to reduced eating-related self-regulation such as unsuccessful dieting or binge eating (Meule et al. 2011, 2012a). HRV was also positively correlated with dieting success (Meule et al. 2012c, in revision). Taken together, these results suggest a positive association between HRV and successful self-regulation of eating behavior.

One approach to train vagal-cardiac control is HRVbiofeedback (Lehrer et al. 2000). Here, participants receive feedback of their respiratory sinus arrhythmia (RSA) amplitude. The aim is to increase RSA amplitude by breathing in resonance frequency (Lehrer et al. 2000). Individual resonance frequency depends on blood volume (Vaschillo et al. 2006), but usually is approximately .1 Hz or 6 breaths per minute (Vaschillo et al. 2002, 2004). Breathing in resonance frequency has been found to cause resonance in the cardiovascular system, thereby increasing HRV and baroreflex gain (Lehrer et al. 2003, 2006).

HRV-biofeedback has been demonstrated to successfully reduce symptoms in patients with physical conditions or mental disorders (Wheat and Larkin 2010). For instance, symptom reductions could be observed after HRVbiofeedback in patients with depression (Karavidas et al. 2007; Siepmann et al. 2008) or PTSD (Tan et al. 2011; Zucker et al. 2009). Notably, in the study by Zucker et al. (2009), there was a trend toward a decrease of drug cravings in the HRV-biofeedback group, although this was not specifically targeted. This finding may be particularly relevant for the application of HRV-biofeedback in relation to eating behavior as considerable evidence indicates common mechanisms underlying the experience of craving across addictions, e.g., food or drugs (Kühn and Gallinat 2011; Pelchat et al. 2004).

Based on these findings, the current study investigated if HRV-biofeedback is also useful to alter deregulated eating behavior. For this purpose, we trained with HRV-biofeedback a group with individuals that reported to frequently experience food cravings with concurrent lack of control over eating behavior. We compared this group to control groups with either high or low cravings, who did not receive an intervention. We expected an increase of vagal-cardiac control accompanied by a decrease of food cravings and increase of experienced control over eating behavior after the biofeedback intervention. Furthermore, we also explored whether HRV biofeedback had an influence on emotion regulation strategies and locus of control, as a positive effect has been reported by other biofeedbackassisted relaxation techniques (e.g., Sharp et al. 1997). All investigated parameters were expected to remain unchanged in the two control groups.

Methods

Participants

An online screening was conducted to recruit high and low food cravers. A link of the screening homepage was distributed via students' mailing lists of the University of Würzburg and an advertisement on a local website for inhabitants of Würzburg, Germany. The screening homepage included the subscale *lack of control over eating* of the *Food Cravings Questionnaire*—*Trait* (FCQ-T; see below). This subscale represents a major feature of food cravings and was chosen to keep the screening succinct. As an incentive for participation, 3×10 ,—Euro were raffled off among participants who completed the entire set of questions (N = 603).

Participants who indicated that they were interested in participating in a further study and whose questionnaire scores were in the upper and lower third of the distribution were contacted by e-mail. Inclusion criteria were normal- or over-weight (BMI = $18.50-29.99 \text{ kg/m}^2$, cf. World Health Organization 2000) and an age between 18 and 40 years. Of all individuals who were contacted, n = 56 (high cravers: n = 28, four males: low cravers: n = 28, five males) met these criteria and agreed to take part in the study. Participants had a mean age of M = 24.12 years (SD = 3.79) and a mean BMI of M = 22.65 kg/m^2 (SD = 3.19). None of the participants reported diagnoses of mental disorders. The majority of participants were students (n = 40). All participants were tested twice with an interval of 4 weeks between pre- and post-measurement. Half of the high cravers (n = 14, one male) were pseudo-randomly¹ assigned to the biofeedback group which performed HRV-biofeedback between the two measurements. Participants in the biofeedback group received 30 Euro for participation, and participants in the control groups received 10 Euro.

Questionnaires

Food Cravings Questionnaire—Trait

Habitual food cravings were assessed with the FCQ-T (Cepeda-Benito et al. 2000; Meule et al. 2012a). This 39-item instrument asks participants to indicate on a 6-point scale how frequently they experience food cravings (ranging from *never* to *always*). The FCQ-T consists of nine subscales measuring food cravings in relation to (1) intentions to consume food, (2) anticipation of positive reinforcement, (3) relief from negative states, (4) lack of control over eating, (5) preoccupation with food, (6) hunger, (7) emotions, (8) cues that trigger cravings, and (9) guilt. Subscales are highly inter-correlated and internal consistency of the total FCQ-T is $\alpha > .90$ (Cepeda-Benito et al. 2000; Meule et al. 2012a) and was $\alpha = .97$ in the current sample.

Eating Disorder Examination Questionnaire

Eating disorder symptomatology was assessed with the questionnaire version of the Eating Disorder Examination (EDE-Q; Fairburn and Beglin 1994; Hilbert and Tuschen-Caffier 2006). This 28-item instrument asks about eating disorder symptomatology during the past 28 days. Of these, 22 items assess *restraint*, *eating concerns*, *weight concerns*, and *shape concerns* on a 7-point scale (ranging from *never* to *every day*). Subscales have an internal consistency of $\alpha = .85-.93$ (Hilbert et al. 2007) and was $\alpha = .82-.93$ in the current sample. The remaining 6 questions assess overeating, binge frequency, days with binges, self-induced vomiting, use of laxatives, and compulsive exercising.

Yale Food Addiction Scale

Food addiction symptoms were assessed with the Yale Food Addiction Scale (YFAS; Gearhardt et al. 2009; Meule et al. in press-a). This 25-item instrument contains different scoring options (dichotomous and frequency scoring) to indicate experience of addictive eating behavior. A food addiction symptom count can be calculated which ranges between zero and seven symptoms, according to the diagnostic criteria for substance dependence (Gearhardt et al. 2009). Internal consistency of the YFAS is $\alpha > .80$ (Gearhardt et al. 2009; Meule et al. in press-a) and was $\alpha = .81$ in the current sample.

Perceived Self-Regulatory Success in Dieting

Dieting success was assessed with the Perceived Self-Regulatory Success in Dieting Scale (PSRS; Fishbach et al. 2003; Meule et al. 2012b). This three-item scale asks participants to rate on a 7-point scale how successful they are in watching their weight or losing extra weight and how difficult it is for them to stay in shape. Internal consistency of the PSRS is $\alpha > .70$ (Meule et al. 2012b) and was $\alpha = .72$ in the current sample.

Emotion Regulation Questionnaire

Emotion regulation strategies were assessed with the Emotion Regulation Questionnaire (ERQ; Abler and Kessler 2009; Gross and John 2003). This 10-item questionnaire assesses the use of *cognitive reappraisal* and *suppression* with a 7-point scale. Internal consistencies of the subscales are $\alpha > .70$ (Abler and Kessler 2009; Gross and John 2003) and were $\alpha = .78$ (suppression) and $\alpha = .77$ (reappraisal) in the current sample.

Locus of Control

Locus of control was assessed with the IPC-scales (Krampen 1981; Levenson 1973). This 24-item questionnaire consists of a subscale for *internal locus of control* (I) and two subscales for external locus of control

¹ Initially, participants who were identified as high cravers were randomly assigned to either the biofeedback or the control group. However, when participants assigned to the biofeedback group were contacted and told that the study would require several lab visits for 4 weeks (further details were not mentioned), n = 3 participants indicated that they could not participate in the study because of time constraints. Those participants were then assigned to the control group.

(P: *powerful others*, C: *chance orientations*). Subjects indicate on a 6-point scale the extent to which they believe to have control over their own life, they think they are dependent on powerful others and their perceptions of chance control. Internal consistencies of the subscales are $\alpha > .90$ (Krampen 1981) and ranged between $\alpha = .67$ and .69 in the current sample.

Participant Characteristics

Subjects were asked to report their age, gender, smoking status (smoker vs. non-smoker), and hours that elapsed since their last meal. They also indicated their level of physical activity ("How often do you work out?") on an 8-point scale ranging from *never* to *everyday*.

Heart Rate Recording

Heart rate was monitored with the Polar watch RS800CX (Polar Electro Oy, Kempele, Finland), which has a sampling rate of 1000 Hz. After attaching the chest strap, participants were seated in a quiet room. Subsequently, the experimenter instructed participants to close their eyes and relax and left the room for 10 min.

HRV-Biofeedback

HRV-biofeedback was applied using the Stress Pilot version 1.3.03 (Biocomfort Diagnostics GmbH & Co.KG, Wendingen, Germany). This device measures blood volume in the earlobe and calculates heart rate and HRV-indices. Participants are instructed to breath in accordance with a pacing bar that corresponds to the resonance frequency, thereby maximizing RSA. Feedback of RSA is provided in multiple ways, e.g., by a butterfly flying high and calm when RSA is maximal. In the first session, participants were informed about the feedback procedure according to the manual from Lehrer et al. (2000). The deep breathing test (Löllgen et al. 2009) was conducted to be able to set up individual levels of difficulty as recommended in the program's manual. In every subsequent session, level of difficulty was adjusted based on performance in the previous session. Twelve HRV-biofeedback sessions were conducted, each lasting 20 min. All training sessions took place in the Department of Psychology I (University of Würzburg, Germany) in a quiet room. Performance and possible problems (e.g., hyper- or hypoventilation) were discussed with an experimenter before and after each session.

Procedure

Participants were asked to refrain from eating, drinking caffeinated drinks, and smoking at least 1 h before the first

measurement. Individuals in the craving-biofeedback group were told about the health benefits of the HRVbiofeedback procedure, but not that the aim was the reduction of food cravings. After providing instructions and signing informed consent, a 10 min baseline heart rate recording was conducted. Then, participants performed a working memory task with pictures of food and neutral stimuli, which is reported elsewhere (Meule et al. in pressb). Finally, participants completed the questionnaires and height and weight were measured.

Half of the high cravers (n = 14) practiced HRV-biofeedback for 4 weeks, while the other half of high cravers (n = 14) and the non-craving control group (n = 28)received no intervention.

After 4 weeks, the very same routine was conducted as for the first measurement.

Data Analysis

R-R-recordings were analyzed with Kubios HRV 2.0 software (Tarvainen et al. 2009). Interbeat interval series were visually scanned by the experimenter and corrected for artifacts with the default settings of the program. Trend components were removed with the smoothness priors detrending method ($\lambda = 500$). Only the last 5 min of the 10 min heart rate recording were used for calculation of autonomic parameters to ensure that data reflected resting conditions. This time period is sufficient for calculating HRV-indices (Task Force of The European Society of Cardiology and The North American Society of Pacing and Electrophysiology 1996). Heart period (HP) was calculated as the interval [ms] between successive heart beats. Spectral power was obtained for high frequency (HF: .15-.4 Hz) and low frequency (LF: .04-.15 Hz) components by Fast Fourier Transformation. We used the HF-power to calculate with the following equation an HP-normalized index of RSA (Hayano index or RSAnorm), which has been shown to reflect vagal control independent of sympathetic influences, (cf. Blechert et al. 2007):

$$RSAnorm(\%) = 100 \times \frac{\sqrt{HF \text{ power}}}{\text{mean RR interval}}$$

RSAnorm was not normally distributed and logtransformed (ln) because of skewed distribution.

Univariate ANOVAs were calculated to compare groups with regards to age, physical activity and BMI, as measured in the pre-test. Differences in gender and smoking status (smoker vs. non-smoker) between groups were tested with χ^2 -tests. ANOVAs for repeated measures were calculated with group (craving-biofeedback vs. craving-control vs. non-craving-control) as between-subject factor, and time (pre- vs. post-measurement) as within-subject factor for each questionnaire and physiological parameter separately. Post hoc comparisons of significant main effects were performed with Scheffé-tests and interactions with *t*-tests. In case of the *lack of control* subscale of the FCQ-T, the within-factor included three levels, because participants already filled out this scale during the online screening.

We calculated effect sizes for all dependent variables for each group separately, the Standardized Effect Size (SES) and the Standardized Response Mean (SRM) with the following equations (cf. Hinz and Brähler 2011):

$$SES = \frac{M_1 - M_2}{SD_1}$$
$$SRM = \frac{M_1 - M_2}{SD_{(T_1 - T_2)}}$$

In case of the lack of control subscale of the FCQ-T, we calculated another effect size that takes into account a stable baseline phase before an intervention (Guyatt's Responsiveness Index, GRI, cf. Hinz and Brähler 2011):

$$\text{GRI} = \frac{\text{M}_1 - \text{M}_2}{\text{SD}_{(\text{T}_0 - \text{T}_1)}}$$

Effects sizes were evaluated as small (>.2), medium (>.5) or large (>.8) based on the criteria by Cohen (1988).

Results

Means, standard deviations, and interaction effects for each questionnaire and physiological parameters are reported in Table 1. Corresponding effect sizes are reported in Table 2.

Participant Characteristics

Groups did not differ in age $(F_{(2,53)} = .24, ns)$, BMI $(F_{(2,53)} = .16, ns)$, physical activity $(F_{(2,53)} = .20, ns)$, gender distribution $(\chi^2_{(2)} = 1.19, ns)$, or smoking status $(\chi^2_{(2)} = 1.70, ns)$.

Questionnaires

Food Cravings Questionnaire

There was a significant main effect for group ($F_{(2,53)} = 36.22$, p < .001), indicating higher FCQ-T total scores in the craving-biofeedback (Scheffé p < .001) and the craving-control group (Scheffé p < .001) compared to the non-craving control group while the two high craving groups did not differ. This group effect was also present in all FCQ-T subscales (all $F's_{(2,53)} > 9.50$, all p's < .001). There was further a main effect of time for FCQ-T-total score ($F_{(1,53)} = 12.58$, p < .01), and the subscales intentions to eat ($F_{(1,53)} = 24.13$, p < .001), positive reinforcement ($F_{(1,53)} = 4.44$, p < .05), feelings of hunger ($F_{(1,53)} = 8.96$, p < .01), negative affect

 $(F_{(1,53)} = 4.95, p < .05)$, and cue-dependent eating $(F_{(1,53)} =$ 4.68, p < .05), indicating decreases of food cravings. There were further significant interactions of group \times time for the FCO-T-total score, and the subscales lack of control, preoccupation with food, cue-dependent eating, and feelings of guilt (Table 1). For the craving-biofeedback group post hoc *t*-tests indicated reductions in FCQ-T total scores ($t_{(13)} = 2.81$, p < .05), and the subscales lack of control ($t_{(13)} = 2.67$, p < .05), preoccupation with food ($t_{(13)} = 2.90, p < .05$), and feelings of guilt ($t_{(13)} = 2.41$, p < .05). There was also a reduction of FCQ-T total scores ($t_{(27)} = 2.90, p < .01$) and cue-dependent eating ($t_{(27)} = 3.60, p < .01$) in the non-craving group. No changes occurred in the craving-control group. In case of the subscale lack of control, no changes were observed in any group between the online screening and the first measurement (Fig. 1). Inspection of Table 2 reveals that effect sizes were mostly medium-to-large in the craving-biofeedback group. Importantly, standardized effects sizes were consistently stronger in the craving-biofeedback group than in both control groups (Fig. 2).

Eating Disorder Examination Questionnaire

There were significant main effects for group on the scales restraint ($F_{(2,53)} = 7.71, p < .01$), eating concerns ($F_{(2,53)} =$ 11.59, p < .001), weight concerns ($F_{(2,53)} = 11.45$, p < .001) .001), and shape concerns $(F_{(2,53)} = 7.19, p < .01)$, indicating higher eating pathology in the craving-biofeedback and the craving-control group compared to the non-craving control group (all Scheffé p's < .05). There were also significant main effects for group for self-reported overeating $(F_{(2.53)} = 5.67, p < .01)$, binge frequency $(F_{(2.53)} = 5.54, p < .01)$ p < .01), and days with binges ($F_{(2.53)} = 5.40, p < .01$), indicating more frequent binge eating in both craving groups as compared to the non-craving control group (all Scheffé p's < .01). The craving groups did not differ from each other. Eating concerns $(F_{(1,53)} = 8.72, p < .01)$, shape concerns $(F_{(1,53)} = 9.60, p < .01)$, overeating $(F_{(1,53)} =$ 9.16, p < .01), binge frequency ($F_{(1.53)} = 7.44, p < .01$), and days with binges ($F_{(1,53)} = 4.12$, p < .05) decreased with time. There were further significant group \times time interactions for eating and weight concerns (Table 1). Post hoc t tests indicated a reduction of eating $(t_{(13)} = 2.59)$, p < .05) and weight concerns ($t_{(13)} = 3.58, p < .01$) only in the craving-biofeedback group, but not in the craving-control or the non-craving control group. Effect sizes ranged between small and large (Table 2).

Yale Food Addiction Scale

There was a significant main effect for group ($F_{(2,53)} = 17.84$, p < .001), indicating more food addiction symptoms in the craving-control group than in the craving-

	Online Screening	ning		Pre-measurement			Post-measurement			Interaction effect
	CB M (SD)	CC M (SD)	NCC M (SD)	CB M (SD)	CC M (SD)	NCC M (SD)	CB M (SD)	CC M (SD)	NCC M (SD)	(Group \times time)
Food cravings questionnaire—trai	'ire—trait									
Intentions to eat	I	I	I	10.50 (1.74)	11.50 (2.79)	7.57 (2.44)	9.14 (2.25)	10.71 (2.53)	6.39 (1.66)	$F_{(2,53)} = .48, ns$
Positive reinforcement	I	I	I	16.29 (3.25)	16.29 (3.02)	12.07 (4.44)	14.93 (2.53)	16.29 (5.00)	10.82 (3.58)	$F_{(2,53)} = 1.02, ns$
Negative reinforcement	I	I	I	8.50 (2.21)	8.50 (2.88)	6.18 (2.09)	7.43 (2.03)	8.50 (3.21)	5.50 (2.05)	$F_{(2,53)} = .83, ns$
Lack of control	21.86 (2.28)	23.64 (4.14)	10.25 (2.59)	20.79 (4.42)	23.86 (6.16)	10.75 (3.61)	18.29 (3.27)	23.21 (5.61)	10.54 (3.55)	$F_{(4,106)} = 2.94, p < .05$
Preoccupation with food	I	I	I	19.64 (3.95)	19.29 (7.48)	11.21 (4.63)	15.93 (4.41)	20.71 (6.83)	10.68 (4.70)	$F_{(2,53)} = 8.33, p < .01$
Feelings of hunger	I	I	I	14.86 (1.96)	14.79 (3.33)	10.89 (2.86)	13.36 (2.44)	13.86 (4.56)	10.57 (2.90)	$F_{(2,53)} = 1.42, ns$
Negative affect	I	I	I	13.71 (3.67)	13.43 (4.15)	7.43 (2.95)	11.79 (3.83)	12.93 (4.81)	6.89 (2.57)	$F_{(2,53)} = 1.04, ns$
Cue-dependent eating	I	I	I	16.86 (2.98)	15.57 (4.20)	12.29 (3.83)	15.07 (2.59)	16.29 (3.56)	10.82 (3.38)	$F_{(2,53)} = 3.62, p < .05$
Feelings of guilt	I	I	I	9.43 (3.88)	9.07 (4.46)	4.71 (1.90)	7.64 (2.41)	9.50 (4.22)	4.79 (2.13)	$F_{(2,53)} = 4.96, p < .05$
Total	I	I	I	130.57 (18.00)	132.29 (28.46)	83.11 (19.23)	113.57 (15.30)	132.00 (34.89)	77.00 (18.52)	$F_{(2,53)} = 4.20, p < .05$
Eating disorder examination questionnaire	ion questionnair	e.								
Restraint	I	I	I	1.14 (1.30)	2.00 (1.35)	.57 (.77)	.99 (1.30)	1.56 (1.25)	.49 (.80)	$F_{(2,53)} = .81, ns$
Eating concern	I	I	I	1.03 (1.14)	1.23 (1.00)	.12 (.16)	.53 (.92)	(1.02) (99)	.12 (.18)	$F_{(2,53)} = 3.38, p < .05$
Weight concern	I	Ι	Ι	2.10 (1.66)	2.23 (1.67)	.66 (.79)	1.39 (1.23)	2.41 (1.70)	.61 (.61)	$F_{(2,53)} = 4.32, p < .05$
Shape concern	I	Ι	I	2.36 (1.83)	2.58 (1.76)	1.17 (1.00)	1.72 (1.44)	2.55 (1.58)	.94 (.78)	$F_{(2,53)} = 2.84, ns$
Overeating	I	Ι	I	4.00 (4.51)	8.64 (12.75)	1.11 (1.50)	2.79 (2.36)	6.14 (9.92)	.86 (1.43)	$F_{(2,53)} = 2.49, ns$
Binge frequency	I	I	I	1.86 (3.35)	7.50 (13.04)	.21 (.57)	1.14 (2.45)	5.21 (10.00)	(00.) 00.	$F_{(2,53)} = 2.60, ns$
Days with binges	I	I	I	2.00 (2.72)	6.71 (11.84)	.18 (.48)	1.14 (2.45)	5.07 (10.05)	.07 (.26)	$F_{(2,53)} = 1.22, ns$
Perceived self-regulatory	I	I	I	10.14 (3.61)	10.71 (3.56)	13.96 (4.08)	11.00 (2.86)	9.64 (3.52)	13.79 (3.56)	$F_{(2,53)} = 1.21, ns$
Vale food addiction scale	1	I	1	2 14 (1 03)	2 03 (1 54)	1 11 7 50)	1 70 /1 10)	7 03 (1 73)	1 14 (53)	- 85 -
Tate jood daatchon state Loons of control	I	I	I	(00.1) +1.2	(+(-1)) ((-7)	(00.) 11.1	(61.1) 61.1	((1)) (6.7	(((()) +1))	$\Gamma(2,53) = .00, 10$
LOCUS OJ CONTROL Laternal lague of control				35 ED 13 EE	(LC 3/ LU 3C	107 17 17 EC	16L 17 76 56		17 30 17 24	E = -21
	I	I	I	(0.0) 00.00	(15.5) 10.05	01.14 (4.30)	(61.4) 00.00	(24.4) (0.10	(+0.4) 60.10	$\Gamma_{(2,53)} = .51, m$
	I	I	I	24.50 (4.49)	(c+.c) 67.c2	(1.1.24) (2.24)	(22.5) 11.52	(24·C) 02·C2	(16°C) 80°12	$\Gamma_{(2,53)} = .54, ns$
Chance orientations	I	I	I	23.50 (3.63)	23.93 (5.14)	23.50 (5.76)	23.79 (3.40)	25.00 (5.55)	22.36 (6.23)	$F_{(2,53)} = 2.36, ns$
Emotion regulation questionnaire	iomaire									
Suppression	I	I	I	12.29 (4.50)	12.36 (4.07)	13.54 (6.31)	13.07 (5.69)	13.07 (4.51)	13.07 (5.47)	$F_{(2,53)} = 1.17, ns$
Reappraisal	I	I	I	26.14 (4.83)	25.29 (6.40)	27.03 (6.99)	27.93 (5.05)	24.14 (5.17)	27.96 (5.47)	$F_{(2,53)} = .97, ns$
Vagal-cardiac control										
Heart period (ms)	I	Ι	I	805.81 (116.20)	755.04 (105.09)	747.92 (122.34)	782.87 (175.09)	743.24 (145.00)	781.70 (124.86)	$F_{(2,53)} = 1.84, ns$
HF power (ms ²)	I	Ι	I	1268.57 (1575.20)	676.50 (783.31)	670.79 (1077.76)	911.64 (961.49)	467.43 (710.47)	999.82 (1375.41)	$F_{(2,53)} = 2.27, ns$
LF power (ms ²)	I	I	I	1262.93 (1232.18)	841.64 (749.40)	1310.04 (1858.09)	1828.50 (3384.30)	596.29 (474.19)	1075.68 (1186.40)	$F_{(2,53)} = 1.14, ns$
ln(RSAnorm)	I	I	I	1.23 (.44)	.94 (.57)	.87 (.49)	1.14 (.36)	.68 (.54)	.62)	$F_{C2} = 3.27, \ p < .05$

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Table 2 Effect sizes for all variables

	Craving-biofeedback		k	Craving-control			Non-craving-control		
	SES	SRM	GRI	SES	SRM	GRI	SES	SRM	GRI
Food cravings questionnaire—trait									
Intentions to eat	.78	.85	_	.28	.45	_	.48	.78	_
Positive reinforcement	.42	.51	_	.00	.00	_	.28	.43	_
Negative reinforcement	.48	.49	_	.00	.00	_	.33	.47	_
Lack of control	.57	.71	.62	.11	.17	.15	.06	.09	.06
Preoccupation with food	.94	.77	_	19	46	_	.11	.20	_
Feelings of hunger	.77	.66	_	.28	.41	_	.11	.15	_
Negative affect	.52	.42	_	.12	.18	_	.18	.23	_
Cue-dependent eating	.60	.47	_	17	27	_	.38	.68	_
Feelings of guilt	.46	.64	_	10	18	_	04	06	_
Total	.95	.75	_	.01	.02	_	.32	.55	_
Eating disorder examination questionnaire									
Restraint	.12	.13	_	.33	.42	_	.10	.14	_
Eating concern	.44	.69	_	.24	.26	_	.00	.00	_
Weight concern	.43	.95	_	11	14	_	.07	.10	_
Shape concern	.35	.84	_	.02	.05	_	.23	.42	_
Overeating	.27	.44	_	.20	.47	_	.17	.22	_
Binge frequency	.22	.52	_	.18	.42	_	.37	.37	_
Days with binges	.32	.67	_	.14	.28	_	.23	.19	_
Perceived self-regulatory success in dieting	24	29	_	.30	.27	_	.04	.06	_
Yale food addiction scale	.34	.35	_	.00	.00	_	06	04	
Locus of control									
Internal locus of control	.04	.06	_	17	23	_	06	06	_
Powerful others	.18	.22	_	11	14	_	.02	.02	_
Chance orientations	08	10	_	21	50	_	.20	.30	_
Emotion regulation questionnaire									
Suppression	17	24	_	17	26	_	.08	.16	_
Reappraisal	37	54	_	.18	.27	_	13	13	_
Vagal-cardiac control									
Heart period (ms)	.20	.20	_	.11	.12	_	28	36	_
HF power (ms^2)	.23	.23	_	.27	.53	_	31	31	_
LF power (ms^2)	46	21	_	.33	.41	_	.13	.16	_
ln(RSAnorm)	.19	.17	_	.46	.72	_	22	23	_

SES standardized effect size, SRM standardized response mean, GRI Guyatt's responsiveness index

biofeedback group (Scheffé p < .05) and the non-craving control group (Scheffé p < .001). The craving-biofeedback group also had more food addiction symptoms than the non-craving control group (Scheffé p < .05). There was no main effect for time ($F_{(1,53)} = .64$, *ns*) or any interactions (Table 1).

Perceived Self-Regulatory Success in Dieting

There was a significant main effect for group $(F_{(2,53)} = 8.26, p < .01)$, indicating higher dieting success in the noncraving control group than in the craving-biofeedback group (Scheffé p < .05) and the craving-control group (Scheffé p < .01) while the high craving groups did not differ. There was no main effect for time ($F_{(1,53)} = .08, ns$) nor any interaction (Table 1).

Emotion Regulation Questionnaire

For both scales, there were no main effects for group (*Suppression*: $F_{(2,53)} = .09$, *ns*; *Reappraisal*: $F_{(2,53)} = 1.44$, *ns*) or time (*Suppression*: $F_{(1,53)} = .68$, *ns*; *Reappraisal*: $F_{(1,53)} = .41$, *ns*), or any interaction (Table 1).

Locus of Control

For none of the three scales, any main effects for group (*I*: $F_{(2,53)} = .89, ns; P: F_{(2,53)} = 1.42, ns; C: F_{(2,53)} = .44, ns$) or time (*I*: $F_{(1,53)} = .44, ns; P: F_{(1,53)} = .03, ns; C: F_{(1,53)} = .02, ns$) or interactions emerged (Table 1).

Vagal-Cardiac Control

Heart Period

There were no main effects for group $(F_{(2,53)} = .51, ns)$ or time $(F_{(1,53)} = .00, ns)$ and no interaction (Table 1).

HF Power

There were no main effects for group $(F_{(2,53)} = .91, ns)$ or time $(F_{(1,53)} = .27, ns)$ and no interaction (Table 1).

LF Power

There were no main effects for group $(F_{(2,53)} = 1.11, ns)$ or time $(F_{(1,53)} = .01, ns)$ and no interaction (Table 1).

ln(RSAnorm)

There were no main effects for group ($F_{(2,53)} = 2.39$, ns) or time ($F_{(1,53)} = 1.58$, ns), but a significant interaction (Table 1). Post hoc *t*-tests indicated that vagal-cardiac control did not change in the craving-biofeedback group ($t_{(13)} = .65$, ns) and the non-craving control group ($t_{(27)} = -1.20$, ns), but decreased from pre- to post-measurement in the craving-control group ($t_{(13)} = 2.69$, p < .05). Using BMI, age or hours since the last meal as covariates did not affect this result.

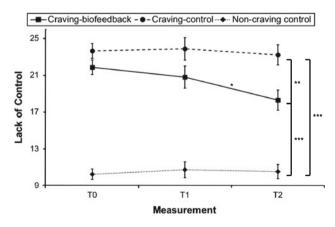


Fig. 1 Means of the food cravings questionnaire—subscale lack of control during online screening (T0), before (T1) and after intervention (T2). Error bars indicate the standard error of the mean. Asterisks indicate p values <.05*, <.01**, and <.001***

As high and low cravers did not differ in vagal-cardiac control, we investigated at pre-measurement the association between indexes of disordered eating behaviors and vagal-cardiac control. Here, we found that binge eating was negatively correlated with vagal-cardiac control in high cravers (*overeating*: r = -.59, *binge frequency*: r = -.59, *days with binges*: r = -.57, all p's < .01), but not in low cravers (all p's > .05).

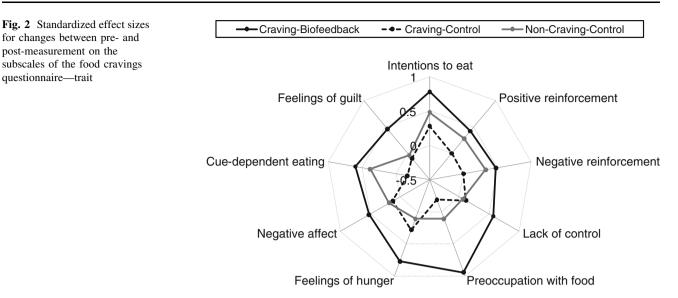
Discussion

In the current study, subjective food cravings and eatingand weight-related concerns were reduced in high food cravers after HRV-biofeedback training. Particularly, food cravings related to a lack of control, preoccupation with food, and feelings of guilt were significantly decreased after the intervention in the biofeedback-group only. Although changes were not significant for some aspects of food craving, analyses of effect sizes showed medium-tolarge reductions for all food craving subscales. Moreover, there was also a decrease in FCQ-T total scores, suggesting an overall effect on food cravings. Unexpectedly, reductions of food cravings elicited by external cues could also be found in the non-craving control group. However, effect sizes for FCQ-T subscales were consistently stronger in the craving-biofeedback group.

The biofeedback intervention may have altered cognitions and attitudes toward eating and weight, but did not influence behavioral aspects. Although we found reduced eating and weight concerns in high cravers, there were no significant changes in restraint, dieting success, or food addiction symptoms. In the craving-biofeedback group reduction of cravings were particularly pronounced with regards to thoughts and preoccupation with food and feelings of guilt from cravings or for giving into them. A lack of behavioral changes might be due to the short time period (1 month) between measurements.

Contrary to our hypotheses, vagal-cardiac control did not increase in the craving-biofeedback group; instead it *decreased* in the craving-control group. It is unclear why vagal-cardiac control would have decreased in the cravingcontrol group. One could argue that HRV-biofeedback might have protected the craving-biofeedback group against this decline. However, this hypothesis remains speculative and future studies are needed addressing the long-term development of HRV in high cravers and which psychological or physical conditions are associated with possible changes in HRV.

As reductions in craving and eating and weight concerns were not associated with an increase in HRV, other mechanisms must be responsible for the observed changes. A first possibility may be that the HRV-biofeedback had a



general effect on wellbeing (e.g., relaxation) rather than the presumed specific effect on vagal-cardiac control, resulting in a more relaxed attitude to eating and weight concerns. A second explanation, which does not preclude the first, is an increased sense of mastery or perceived self-efficacy as a result of carrying out the biofeedback training. Such cognitive changes have been observed in other treatment groups (e.g., EMG-biofeedback in tension-headache patients; Holroyd et al. 1984; Lacroix et al. 1986) and may pose a mechanism underlying the effects of biofeedback, which is independent from direct physiological changes in the target variable. In line with this, most studies using HRV-biofeedback find changes in psychological variables without changes in resting HRV (Wheat and Larkin 2010). The HRV-biofeedback procedure may have immediate effects and people may strategically use the breathing technique to control symptoms. Hence, the lack of effect of HRV-biofeedback on resting HRV may be explained by the fact that resting vagal function is not affected when an individual is not experiencing craving, but that HRV will be higher during the experience of craving when they breathe in the low frequency range.

Our study has several limitations. Firstly, we did not investigate a sample of patients but recruited a non-clinical sample. This may have rendered it difficult to detect effects, especially in light of the small sample size. Our sample consisted of young and healthy individuals and, therefore, a further increase in HRV might have been difficult to achieve because of ceiling effects. Moreover, while most studies instruct participants to further practice the technique at home, we decided to restrict training to lab visits to standardize the amount of sessions for each participant. However, additional home practice and, therefore, more frequent sessions might be necessary to produce physiological changes. Secondly, our control groups did not receive a placebo or alternative treatment. The factors leading to the observed psychological changes can only be elucidated with appropriate control groups. Thirdly, with the current design demand or placebo effects cannot be ruled out. The questions asked in the online screening may have focused study participants' attention on eating, craving and food. Nevertheless, participants were not told that the aim of the study was the reduction of food cravings or alteration of eating behavior. Finally, we only assessed subjective indices of eating behavior. While we found changes in various eating-related attitudes and cognitions, further studies may investigate if HRV-biofeedback has an effect on actual, and objectively measured eating behavior.

In conclusion, the current findings demonstrate that HRV-biofeedback attenuates subjective food cravings and other eating- and weight-related concerns in a non-clinical sample. More frequent and longer HRV-biofeedback may be necessary to implement those cognitive aspects into actual eating behavior. Moreover, rather than solely practicing HRV-biofeedback, it might be more effective in producing behavioral changes when it is applied in conjunction with a cognitive-behaviorally oriented intervention. Given that cravings related to emotional eating were not influenced by the biofeedback intervention, effects of HRV-biofeedback might also be further enhanced by targeting emotional reactions to food-cues, e.g., with food exposure and response prevention. Notably, high food cravers did not have lower HRV compared to low food cravers prior to the intervention, but a subsequent analysis revealed that binge eating behaviors were negatively correlated with HRV in high food cravers. Thus, the current results lend further support to the notion that frequent and intense experiences of craving are not related to autonomic dysregulation per se, but only in combination with eating disorder symptoms (Rodríguez-Ruiz et al. 2009). Further **Acknowledgments** Funding for this study was provided by a grant of the research training group 1253/2 which is supported by the German Research Foundation (DFG) by federal and Länder funds. DFG had no role in the study design, collection, analysis or interpretation of the data, writing the manuscript, or the decision to submit the paper for publication.

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