Neurofeedback as a psychophysiological treatment for disinhibited eating –

An analysis of efficacy and mechanisms

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Eine Analyse der Wirksamkeit und Mechanismen

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Somewhere ages and ages hence:
Two roads diverged in a wood, and I—
I took the one less traveled by,
And that has made all the difference.

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Abbreviations*

µV	micro Volt
ADHD	attention deficit hyperactivity disorder
APA	American Psychiatric Association
BED	binge eating disorder
BMI	Body Mass Index
BN	bulimia nervosa
BP	blood pressure
Corr.	Musculus corrucator supercillii
DBP	diastolic blood pressure
DE	disinhibited eating
DSM-5	Diagnostic and Statistical Manual of Mental Disorders, 5th edition
ECG	electrocardiogram
EDA	electrodermal activity
EEG	electroencephalogram / electroencephalographic
EI-theory	Elaborated intrusion theory of desire
EMG	electromyogram / electromyographic
ERP	event-related potential
FEV	Fragebogen zum Essverhalten
fMRI	functional magnetic resonance imaging
FPA	finger pulse amplitude
HCs	healthy controls
HF	high frequency activity
HR	heart rate
HRV	heart rate variability
Hz	hertz
IBI	inter-beat-interval
LF	low frequency activity
LF/HF	low frequency / high frequency index (sympathovagal balance)
LPP	late positive potential
M.	Musculus
NFB	neurofeedback
NW	normal weight
OCD	obsessive-compulsive disorder
QEEG	quantitative electroencephalogram
RCT	randomized controlled trial
REs	restrained eaters
resp.	respiration amplitude
RS	Restraint Scale
rt-fMRI	real-time functional magnetic resonance imaging
SBP	systolic blood pressure
SC	skin conductance
SCL	skin conductance level
SDNN	standard deviation of normal to normal R-R intervals
SPW	slow positive wave
TFEQ	Three-Factor Eating Questionnaire
temp.	skin temperature
VLF	very low frequency activity
WHO	World Health Organization
Zygo.	Musculus zygomaticus major

*Abbreviations in the empirical articles may deviate and are introduced in the respective papers.

Abstract

Disinhibited eating (DE) behaviors – such as overeating or binge eating – are common dysfunctional behaviors, especially in female populations. These behaviors can contribute to developments of overweight or obesity, and associated negative health consequences, like non-communicable diseases or eating disorders. Still, treatments for DE show moderate efficacy and may benefit from improvements. Recently, experts called for more research on brain-directed treatments to target DE and related eating disorders. Neurofeedback (NFB), using electroencephalographic (EEG) activity, is a well-established and safe neuromodulatory approach, based on an acquisition and online feedback of EEG-signals. It enables patients to regulate brain activity associated with dysfunctional psychological states or behaviors. However, EEG NFB had – to date – not been evaluated for the treatment of DE.

The present doctoral thesis reports on the development and evaluation of an EEG NFB protocol for the treatment of DE. The protocol is based on a synthesis of current theories on the etiology of DE, empirical findings regarding associated psychophysiological activity, and adaptable treatment components. It consists of ten sessions and incorporates self-regulation of tense physiological arousal, marked by EEG high beta activity, after exposure with personalized food cues.

Two randomized controlled trials and an experimental EEG study aimed at the evaluation of the general and specific treatment efficacy, as well as treatment mechanisms of the NFB. The studies were conducted in subclinical samples of female restrained eaters with DE.

Study 1 (initial sample: $n = 34$) compared a NFB group to a waitlist group and yielded evidence for the general efficacy of the NFB in reducing DE episodes. Results remained stable to a three-month follow-up. Further, the new NFB was well-accepted by the participants.

In Study 2 (initial sample: $n = 75$), NFB and an additional intervention with a highly comparable treatment (mental imagery) were compared to a waitlist condition. NFB, but not the alternative intervention, resulted in significant post-treatment reductions of DE episodes compared to the waitlist. Results remained stable to a three-month follow-up and indicated specific efficacy of the NFB approach.

Study 3 ($n = 36$) aimed at an evaluation of relevant treatment mechanisms in NFB. The experimental study compared the presence and influence of physiological learning (regulation of EEG high beta activity) and psychological learning (enhanced somatic self-efficacy) in the NFB and control intervention group. Analyses showed that physiological learning was only present in the NFB group and showed stronger relations to treatment outcomes than somatic self-efficacy. According to these results, physiological learning constitutes a relevant treatment mechanism in the developed NFB protocol.

In summary, the developed NFB could be evaluated as an efficacious and physiologically-based treatment approach for the treatment of DE. It may be a beneficial adjunct for treatments of eating disorders (e.g., binge eating disorder). Based on the results of this dissertation project, replications in clinical groups are now warranted. Additional implications for research and practice are discussed.

Zusammenfassung

Heißhungeranfälle, als Variante enthemmten Essverhaltens, sind insbesondere unter Frauen weit verbreitet. Sie können die Entstehung von Übergewicht und assoziierten negativen Gesundheitsfolgen begünstigen. Aktuelle Behandlungen für enthemmtes Essen mit Heißhungeranfällen sind langfristig jedoch nur moderat wirksam. Zur Behandlungsoptimierung sollten Verbesserungsmöglichkeiten daher systematisch untersucht werden. Experten stellen hierbei vor allem die Erforschung neurophysiologisch basierter Behandlungsmethoden in den Fokus.

Eine sichere und etablierte Variante in diesem Feld ist das Neurofeedback (NFB) basierend auf dem Elektroenzephalogramm (EEG). Im EEG-NFB wird die elektrische Hirnaktivität in Echtzeit erfasst und an Patienten zurückgemeldet. Hierdurch sollen Patienten lernen ihre Hirnaktivität bewusst zu regulieren und damit assoziierte dysfunktionale Prozesse im Erleben und Verhalten zu beeinflussen. Für die Behandlung von enthemmtem Essverhalten wurde das EEG-NFB allerdings bislang nicht evaluiert.

Die vorliegende Dissertation beschreibt die Entwicklung und Evaluation eines neuen NFB-Protokolls zur Behandlung von Heißhungeranfällen. Dieses basiert auf einer Synthese einflussreicher Theorien des Essverhaltens, verhaltenstherapeutischer Behandlungselemente und empirischer psychophysiologischer Befunde. Das resultierende Protokoll besteht aus zehn Sitzungen in denen NFB zur Regulation der High-Beta-Aktivität im EEG - einem Korrelat physiologischer Erregungszustände - bei Konfrontation mit Heißhunger-auslösenden Nahrungsmittelreizen eingesetzt wird.

Zwei randomisiert-kontrollierte Studien und eine experimentelle EEG-Studie dienten der Evaluation des NFB-Protokolls in Hinblick auf seine generelle und spezifische Wirksamkeit sowie zugrundeliegende Wirkmechanismen. Die Studien wurden in subklinischen Stichproben weiblicher Probanden mit gezügeltem Essverhalten und Heißhungeranfällen durchgeführt.

Studie 1 (Ausgangsstichprobe: n = 34) diente dem Vergleich einer NFB-Gruppe mit einer Wartekontrollgruppe und zeigte erste Evidenz für eine generelle Wirksamkeit von NFB in der Reduktion von Heißhungeranfällen. Die NFB-Behandlung wurde von den Teilnehmerinnen gut akzeptiert und die Effekte blieben in einer Drei-Monats-Katamnese stabil.

In Studie 2 (Ausgangsstichprobe: n = 75) wurden NFB und eine hochvergleichbare Alternativbehandlung (Mental Imagery) mit einer Wartekontrollgruppe vergleichen. In der NFB-Gruppe, jedoch nicht in der Alternativbehandlung, zeigte sich zum Behandlungsabschluss eine signifikante Reduktion der Heißhungeranfälle im Vergleich zur Wartekontrollgruppe. Die Ergebnisse blieben wiederum zur Drei-Monats-Katamnese stabil und weisen auf eine spezifische Wirksamkeit des NFB hin.

Studie 3 (n = 36) diente der Evaluation relevanter Wirkmechanismen des NFB. In dieser experimentellen Studie wurden das Vorliegen und der Einfluss physiologischer Lerneffekte (Regulation der High-Beta-Aktivität im EEG) und psychologischer Lerneffekte (Stärkung der körperbezogenen

Selbstwirksamkeit) in NFB und der alternativen Kontrollbehandlung untersucht. Die Analysen zeigen, dass physiologische Lerneffekte spezifisch in der NFB-Gruppe auftraten und hier eine stärkere Prädiktionskraft für die Behandlungseffekte vorwiesen als psychologische Lerneffekte. Entsprechend kann physiologisches Lernen als relevanter Wirkmechanismus des NFB-Protokolls angesehen werden.

Zusammenfassend konnte das entwickelte NFB-Protokoll als wirksamer neurophysiologisch-fundierter Behandlungsansatz evaluiert werden. Entsprechend könnte NFB eine vielversprechende Methode in der Behandlung enthemmten Essverhaltens mit Heißhungeranfällen (z.B. bei der Binge-Eating-Störung) darstellen. Die Ergebnisse dieser Dissertation bilden eine solide Basis für zukünftige Evaluationen in klinischen Gruppen. Weitere Implikationen für Forschung und Praxis werden diskutiert.

1. Introduction

Eating is a natural behavior and a vital part of everyone's life, providing us with energy and nutrients to master the requirements of our daily routines. Yet, apart from its biological function, eating has also become a source of hedonic experiences in present societies. Many people report that tasty food contributes to their perceived quality of life. However, sometimes we tend to overindulge in these tasty foods and eat more than needed: Palatable dinner buffets or hearty holiday meals are common triggers that tempt people into overeating. Further, numerous individuals tend to grab a chocolate bar to get some "extra energy" in stressful situations, or try to numb emotional states with a bowl of ice cream.

Occasional incidences of these *disinhibited eating* (DE) behaviors may not pose a problem and can easily be compensated for. Still, if these behaviors occur more frequently or even habitually, several negative consequences will arise. Increased caloric intake facilitates weight gain and developments of overweight or obesity. Worldwide, prevalence rates of overweight are increasing (Finucane et al., 2011). In Germany, 67.1 % of the male and 53.0 % of the female population are currently overweight (Mensink et al., 2013). The World Health Organization (WHO) has labeled this development as an *obesity pandemic* (James, 2008). The biggest pitfalls of this development are presumably the associated negative health consequences, like increased odds for the development of several diseases and general mortality due to overweight and obesity (Guh et al., 2009).

Attempts to consciously restrict food intake, like dieting, are common means to try to prevent weight gain (Andreyeva, Long, Henderson, & Grode, 2010). Still, diets often fail and despite of an increased knowledge on healthy nutrition, people often fall back into habitual patterns of dysfunctional eating behavior (Mann et al., 2007). Hereby, episodes of DE are common catalysts for a breakdown of dieting intentions (Papies, Stroebe, & Aarts, 2008; Polivy & Herman, 1985). On the one hand, these failures may lead to even more weight gain in the long run (Hays & Roberts, 2008; Siahpush et al., 2015). On the other hand, psychological consequences, like repeated loss-of-control experiences, perceived distress, body dissatisfaction, and impaired self-efficacy, can contribute to the development of clinical eating disorders, such as binge eating disorder or bulimia nervosa (Colles, Dixon, & O'Brian, 2008; Stice, 2002).

Researchers in the field of eating behavior and eating disorders proposed several psychological models to explain the etiology of DE. The acknowledgement of psychological factors that contribute to these behaviors has in return fostered psychological treatments for DE.

Still, especially with regard to the treatment of clinical eating disorders, current remission rates leave room for improvements. Several researchers pointed out that new and advanced techniques should be considered and evaluated (Brownley, Berkman, Sedway, Lohr, & Bulik, 2007; Wilson, Grilo, & Vitousek, 2007).

Recent neuroscientific research provided new insights into the mechanisms accountable for DE and its antecedents. The discovery of neurophysiological similarities between food and drug consumption support the assumption that processes in the brain, likely, contribute to the disinhibition of eating behavior (Avena, Bocarsly, Hoebel, & Gold, 2011; Sinha & Jastreboff, 2013). Therefore, calls to consider treatment techniques that directly target neurophysiological dysregulations in eating, have become louder: So-called *brain-directed treatments* would possibly solve problems on a fundamental, neuronal basis, and thus deliver means to increase the efficacy of interventions for dysfunctional eating behaviors and eating disorders (Iacovino, Gredysa, Altman, & Wilfley, 2012; Schmidt & Campbell, 2013).

The present doctoral thesis reports on the development and evaluation of such a brain-directed treatment, an electroencephalographic (EEG) *neurofeedback* (NFB) protocol to target DE. EEG NFB has been successfully applied to treat several psychological and neurological disorders, for example attention deficit hyperactivity disorder (ADHD; Arns, de Ridder, Strehl, Breteler, & Coenen, 2009) and epilepsy (Tan et al., 2009). Still, up to now, EEG NFB had not been applied in the field of dysfunctional eating behavior or eating disorders (Bartholdy, Musiat, Campbell, & Schmidt, 2013; Korn & Niepoth, 2009).

Following an initial conceptualization of DE behaviors (cf. 2.1), the present thesis outlines theoretical considerations for the development of the NFB protocol. After a presentation and synthesis of etiologic factors in DE (cf. 2.2), associated psychophysiological correlates (cf. 2.3), and recent developments in therapeutic approaches to target DE (cf. 2.4), the development and rationale of the NFB protocol will be described (cf. 2.5). As an empirical core of this dissertation project, two randomized controlled trials (RCTs) aimed at evaluating the general (cf. 3.1 & 4.) and specific efficacy (cf. 3.2 & 5.) of this NFB protocol in the treatment of subclinical DE. Further, an experimental EEG-study (cf. 3.3 & 6.) was integrated in the second RCT, to analyze physiological and psychological treatment mechanisms. Results of these three studies will be discussed in the light of current research with a look on practical implications (cf. 7.1 & 7.2). Strengths and limitations of the conducted studies (cf. 7.3), as well as future research options (cf. 7.4) will be outlined.

2. Theoretical background

2.1 Disinhibited eating

The majority of individuals is aware of the relationship between heightened caloric intake and weight gain that contributes to the development of overweight and obesity (Swinburn et al., 2009; Westerterp, 2010). Many people therefore try to exert at least some control over their food intake (Andreyeva et al., 2010; Montani, Schulz, & Dulloo, 2015). For example, Fayet, Petocz, and Samman (2012) found that among female students, 43 % reported dieting and another 32 % exerted practices to avoid weight gain. In general populations, increasing trends for dieting behaviors have been found, with up to 57 % of women and 40 % of men reporting weight loss dieting practices (Montani et al., 2015).

The intention to control the caloric intake may manifest in various forms, for example, in an *inhibition* regarding the consumption of unnecessary high amounts of food or an avoidance of unhealthy calorie-dense food (Polivy & Herman, 1985; Mooney & Wallbourn, 2001). However, exerting continuous control over behavior may be subject to perturbation by several internal and external influences (Ruderman, 1986; Tice, Bratslavsky, & Baumeister, 2001). With regard to eating behavior, a breakdown of inhibitory efforts may subsequently lead to *disinhibition* of eating as a form of undesired consumption (Ruderman, 1986; Stunkard & Messick, 1985).

This chapter will introduce current concepts and taxonomies of DE and the selected definition of DE for this dissertation project. Afterwards, epidemiology and associated health-related consequences will be depicted briefly.

2.1.1 Concepts and taxonomies of disinhibited eating

According to a general definition, DE occurs “when an individual is unable to control intake and overeats in response to internal (e.g., emotional stressors) or external (e.g., presence of palatable foods) cues despite his or her intentions not to do so” (Goldstein et al., 2014, p. 164). DE may manifest in a range of different eating behaviors that are characterized by lack of control, for example, eating without hunger, overeating, and binge eating episodes (Warren, 2008; Zocca et al., 2011). Thus, several dysfunctional eating behaviors that share lack of control characteristics can be subsumed under the umbrella term of DE. These behaviors can either be facilitated by individual predispositions, external or internal cues and stressors, or cognitive and affective states (Vanucci et al., 2013; Zocca et al., 2011).

With respect to distinct DE behaviors, researchers in the field of eating behavior and eating disorders further refer to its different manifestations by means of divergent conceptualizations and taxonomies. The most common conceptualizations either base on distinct antecedents of DE behaviors, their diagnostic characteristics, or the dimensional degree of severity (Davis, 2013; Goldstein et al, 2014; Mailloux, Bergeron, Meilleur, D'Antono, & Dubé, 2014).

In the antecedent-driven conceptualizations, the most popular taxonomy separates DE behaviors caused by the disinhibition of dietary restraint, emotional eating, and external eating (Van Strien, Frijters, Bergers, & Defares, 1986). The respective antecedents will be presented in more detail later on (cf. 2.2.1 - 2.2.4).

Regarding *disinhibition of dietary restraint*, researchers assume that individuals who exert long-term effortful control over their food intake (*restrained eaters*) would be especially vulnerable to exhibit DE (Herman & Mack, 1975; Herman & Polivy, 1975). Once self-imposed diet boundaries are violated, a breakdown of cognitive control- or restraint-processes is seen as the proximate antecedent of DE (Herman & Polivy, 1984; Polivy & Herman, 1985). Still, this lack of control may again be triggered by the two other antecedents that receive attention in antecedent-driven conceptualizations.

Emotional eating subsumes DE behaviors that are fostered by aversive emotional or stress-related states, for example anxiety, anger, or sadness (Groesz et al., 2012; Macht, 2008; Van Strien et al., 1986). Here, eating behavior is instrumental and serves as an emotion regulation or coping strategy to improve an individuals' affect (Evers, Stok, & de Ridder, 2010; Gianini, White, & Masheb, 2013; Macht, Haupt, & Ellgring, 2005). Given the close relationship of emotional states and stress, emotional eating can also be referred to as (or includes) *stress-induced eating* (Greeno & Wing, 1994).

The term *external eating* refers to DE behaviors that are triggered by the presence of external cues associated with eating or food (e.g., the sight or smell of food, but also associated locations or times of day). Assumptions on this externality of eating propose that some individuals would be especially sensitive and reactive to external cues. They would thus tend to initiate DE in response to food *cue exposure* (Rodin & Slochower, 1976; Van Strien et al., 1986). This could then increase the likelihood of overeating when food is readily available and individuals are confronted with respective cues (Jansen, 1998; Swinburn et al., 2011).

Researchers in the field of clinical eating disorders often prefer a DE conceptualization based on the diagnostic criteria for binge eating as stated by the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-5; American Psychiatric Association [APA], 2013). Here,

two essential components mark a binge eating episode: (1) the consumption of a large amount of food in a certain time period, (2) accompanied by a sense of loss of control over eating behavior (APA, 2013). A popular diagnostic conceptualization of DE is based on the presence or absence of these two criteria (Grilo, Masheb, & Wilson, 2001; Mailloux et al., 2014; see Figure 1).

Diagnostic Criteria		**1) Objectively large amount of food**	
		no	*yes*
2) Loss of Control	*no*	no Overeating	Objective Overeating
	yes	Loss-of-Control Eating / Subjective Binge Eating	Objective Binge Eating

Figure 1. Diagnostic conceptualization of disinhibited eating behaviors (adapted from Mailloux et al., 2014).

DE can be classified as *objective overeating*, whenever the amount of food an individual consumes exceeds the amounts that other individuals would usually consume in the same time and under comparable circumstances (Cooper & Fairburn, 2003; Striegel-Moore et al., 2009). Thus, the quantity of food is the central criterion (e.g., a meal of 3,000 kcal in one occasion), while individuals with objective overeating are not supposed to experience any loss of control (Latner, Hildebrandt, Rosewall, Chrisholm, & Hayashi, 2007; Mailloux et al., 2014).

Contrary, *loss-of-control eating* (also termed: *subjective binge eating*) refers to situations, in which the individual does not consume an objectively large amount (e.g., one chocolate bar) but experiences loss of control over his or her eating behavior (Mailloux et al., 2014). Thus, the associated psychological experience is central to this type of DE. Loss-of-control eating is frequently followed by severe distress and has been identified as very critical regarding the development of eating pathology (Latner, Vallance, & Buckett, 2008; Pollert et al., 2013). Some researchers even propose that it should be considered as the key feature of clinically relevant binge eating (Colles et al., 2008; Wolfe, Baker, Smith, & Kelly-Weeder, 2009).

Lastly, following the categorization of binge eating according to the DSM-5 (APA, 2013), *objective binge eating* is assumed when both diagnostic criteria are fulfilled: consumption of a large quantity of food, as well as the experienced loss of control. This type of DE is also named *objective bulimic episode* (Grilo et al., 2001; Mond, Latner, Hay, Owen, & Rodgers, 2010) and

is the stereotypical behavior necessary to be considered in the diagnosis of eating disorders, like bulimia nervosa (BN) and binge eating disorder (BED). Still, for a full eating disorder diagnosis, frequency and duration of this behavior are additionally assessed (≥ 1 episode per week over the past 3 months) and further criteria have to be fulfilled (APA, 2013).

In contrast to antecedent-driven and diagnostic conceptualizations of DE, dimensional approaches view different manifestations of DE as occurring on a continuum of severity and compulsivity (Davis, 2013). Here, BED and *food addiction* (cf. 2.1.3) mark one end of the dimensional continuum, while occasional binge episodes, emotional eating, and objective overeating represent less pathological and more common behaviors (Davis, 2013; Vainik, Neseliler, Konstabel, Fellows, & Dagher, 2015). Vainik and colleagues (2015) proposed that different DE behaviors, like external eating, emotional eating, or binge eating, have considerable overlaps and are altogether influenced by a common latent factor of *uncontrolled eating.* Their hypothesis was backed up in two studies using structural equation modeling in analyzing different measures to assess DE behaviors.

The notion of a common underlying factor may also reflect the conception of the lay-population more accurately. In the general public, individuals who experience DE do seldom rely on the technical taxonomies used in eating behavior research. In turn, prevalence rates may be affected by diffuse inherent conceptions in community samples (Beglin & Fairburn, 1992; Coker, von Lojewski, Luscombe, & Abraham, 2015; Striegel-Moore & Franko, 2003).

The evaluation studies in this dissertation project aimed at addressing effects of NFB on DE behaviors in a German subclinical community sample. To prevent distorted self-reports of DE due to misconceptions, participants reported the frequency of these behaviors based on a clear definition. Hereby, the target behavior should constitute a subclinical equivalent to subjective binge eating, allowing for different, more distal triggers of DE.

The selected definition is based on the German term *Heißhungeranfall,* which is equally used among laymen as well as clinicians. This term also serves as a German description for clinical binge eating (Becker & Zipfel, 2010, Waschburger & Kröller, 2005). To further clarify the term, the applied definition first based on *food craving* (*Heißhunger*) as the most proximate motivational antecedent of DE (cf. 2.2.5), the specific desire for rather high caloric foods, the lack of control to withstand food craving urges and subsequent actual consumption:

> Als *Heißhunger* definieren wir das intensive und drängende Verlangen und die Lust, bestimmte Nahrungsmittel zu sich zu nehmen. Meist handelt es sich dabei um landläufig eher als ungesund betrachtete Nahrungsmittel mit einem hohen Kaloriengehalt. Häufig wird

Heißhunger von intensiven, wiederkehrenden Gedanken an diese Nahrungsmittel begleitet. Das Verlangen dient nicht allein oder vorwiegend der Befriedigung körperlicher Hungergefühle, sondern entsteht eher aus Appetit auf bestimmte Nahrungsmittel. Ein *Heißhungeranfall* bedeutet, diesem drängenden Verlangen nachzugeben und die entsprechenden Nahrungsmittel tatsächlich zu sich zu nehmen.

Despite of this distinct and important definition for the conducted studies, the theoretical introduction of this thesis will predominantly apply the umbrella term DE (behaviors) to introduce epidemiological, etiological, and therapeutic factors. This procedure will allow for an undistorted presentation and integration of important theories and findings that may originally have based on diverging taxonomies of DE.

2.1.2 Epidemiology

The aforementioned differing conceptualizations of DE behaviors affect epidemiological data. These data further vary dependent on the type (e.g, categorical vs. dimensional assessment) and strictness (e.g., broad vs. narrow definitions of the behavior) of diagnostic assessments (Beglin & Fairburn, 1992; Coker et al., 2015). To provide an overview on the epidemiology of DE, major observations will be pointed out briefly and some exemplary prevalence rates of distinct DE behaviors are provided in Table 1. This overview will focus on DE in the subclinical range. For current epidemiological data on clinical eating disorders associated with binge eating, the reader may refer to Grucza, Przybek, and Clininger (2007), as well as Kessler et al. (2013).

In general, DE is highly prevalent in various samples, especially among women. In healthy community samples, prevalence rates for any DE behavior in women range from 3 % (Preti et al., 2009) to 56 % (Katzman et al., 1994). In male samples, prevalence rates are lower, with ranges between 1.2 % (Preti et al., 2009) and 28 % (Lewinsohn, Seeley, Moerk, & Striegel-Moore, 2002). Thus, comparable with prevalence rates in clinical eating disorders (e.g., Preti et al., 2009), significant gender differences are frequently reported for the prevalence of DE.

The majority of epidemiological studies observed higher prevalence of subjective binge eating, objective binge eating, stress-induced eating, and emotional eating in female compared to male samples (De França, Gigante, & Olinto, 2014; Jääskeläinen et al. 2014; Keel, Baxter, Heatherton, & Joiner Jr., 2007; Loth, Wall, Larson, & Neumark-Sztainer, 2015; Striegel-Moore et al., 2009). Solely, objective overeating without loss of control is more frequently observed among men than women (Lewinsohn et al., 2002; Striegel-Moore et al., 2009).

Table 1

Exemplary prevalence rates for disinhibited eating behaviors in different populations.

Source	Disinhibited Eating Behavior	Population	Prevalence
Wallis & Hetherington (2009)	Stress-induced eating	Undergraduate female students	Eating more: 43 % No change: 9 % Eating less: 48 %
Jääskeläinen et al. (2014)	Stress-induced eating	Adolescents	Boys: 15 % Girls: 43 %
Lewinsohn et al. (2002)	Overeating	Depressive patients	Women: 14 % Men: 28 %
Striegel-Moore et al. (2009)	Overeating	Health plan members	Men: 26.0 % Women: 18.0 %
	Loss-of-control eating		Men: 20.0 % Women: 29.6 %
	Binge eating (min. 1 / per week)		Men: 8.0 % Women: 10.0 %
Schlüter et al. (2015)	Loss-of-control eating	Adolescents	Recurrent: 9.5 % Non-recurrent: 13.8 %
Hudson et al. (2007)	Any binge eating	College sample	4.5 %
French et al. (1999)	Any binge eating	Female community sample	Normal weight women: 9 % Overweight women: 21 %
Preti et al. (2009)	Any binge eating	Representative European sample (6 countries), adults	Life time prevalence: Women: 3.0 % Men: 1.2 %
Coker et al. (2015)	Objective binge eating	Obese women awaiting bariatric surgery	55 %
Hilbert, de Zwaan, & Braehler (2012)	Objective binge eating (any episode within the last 28 days)	Representative German sample	Women: 4.2 % Men: 4.2 %

Further, DE shows increased prevalence rates in overweight and obese samples over the lifespan (de Zwaan, 2001; Dingemans & Furth, 2012; Shunk & Birch, 2004) with a positive linear relationship of DE behaviors and Body Mass Index (BMI) in cross-sectional-studies (Coker et al., 2015; Loth et al., 2015) and the highest prevalence rates in obese individuals (de

Zwaan, 2001). Still, it has to be pointed out that the causality may be rather seen the other way round, with severe and frequent DE leading to overweight and obesity due to increased energy intake (cf. 2.1.3).

Prevalence rates show positive correlations with psychopathology (Isnard et al., 2003), which of course relates to the phenomenological nature of clinical eating disorder diagnoses (Hudson, Hiripi, Pope, & Kessler, 2007). Still, DE is also frequently observed as a comorbidity in patients with affective and anxiety disorders, substance-use disorders, impulse-control disorders, or personality disorders (Hudson et al., 2007; Wilfley et al., 2000). Here, common psychological correlates of psychopathology (e.g., perceived distress, negative affect, and depression), or dispositional factors (e.g., impulsivity and enhanced cue reactivity) may be shared causes (cf. 2.2.2 & 2.2.4).

2.1.3 Health-related consequences

DE is associated with a variety of negative health outcomes, with physiological causes on the one hand and psychological causes on the other hand. Most obviously, recurring DE usually leads to overriding energy intake and increases an individual's likelihood to develop overweight or become obese. Correspondingly, several studies report positive correlations between BMI and DE (Hays et al., 2002; Hays & Roberts, 2008; Savage et al., 2009). Further, DE behaviors are seen as etiological factors in the development of eating disorders (Stice, 2002) and other psychological pathology (e.g., depression, Luppino et al., 2010) up to *food addiction* (Davis et al. 2011).

Physical health

Several studies provide evidence for the causal role of DE in the development of overweight and obesity and its negative influence on attempted weight regulation (Bryant, King, & Blundell, 2008; Chaput et al., 2009; Niemeier, Phelan, Fava, & Wing, 2007).

Overweight and obesity clearly exert negative influences on further health developments, as they increase the likelihood of high blood pressure (hypertension), heart diseases, and several forms of cancer (Guh et al., 2009), musculoskeletal and pain-related problems (Després, 2001; Hu, 2003), and the onset of diabetes mellitus type II (Després, 2001; Kahn, Hull, & Utzschneider, 2006). Further, respiratory problems, like sleep apnea, may arise, as well as liver and gall bladder diseases (Kopelman, 2007).

Altogether, overweight and obesity thus contribute to increased morbidity and even mortality rates, due to their central role in the development of non-communicable diseases

(Finkelstein, Brown, Wrage, Allaire, & Hoerger, 2010; Mokdad, Marks, Stroup, & Gerberding, 2004). Therefore, the WHO stated that a combat against the worldwide obesity epidemic is an international health goal with high priority (James, 2008).

Mental health

Beneath the bodily impairments, the psychological consequences of DE mark another important pillar with regard to negative health-related consequences. DE itself was found to impair well-being in women (Provencher et al., 2007). Especially its loss of control-component has been linked to the development of depressive symptoms (White, Kalarchian, Masheb, Marcus, & Grilo, 2009).

As a possible consequence of DE, overweight and obesity predict depression or depressive symptoms and impaired health-related quality-of-life (Jia & Lubetkin, 2005; Luppino et al., 2010). Further psychological distress may result from weight-based stigmatization (Ashmore, Friedman, Reichmann, & Musante, 2008). These psychological distress components again increase the probability of future DE behaviors due to negative affect and perceived stress. This may lead to a vicious circle regarding the occurrence and maintenance of DE behaviors (Ashmore et al., 2008).

As a result, the risk for the development of clinical eating disorders, as a prominent negative consequence of DE, is increased (Stice, 2002). Binge eating with repeated experiences of loss of control over eating behavior, can contribute to impaired self-esteem, body dissatisfaction, and general eating pathology (Colles et al., 2008; Provencher et al., 2007). Further, compensatory dieting behaviors, like severe calorie restriction or purging, may develop to counteract binge eating episodes. All of the aforementioned consequences are known risk factors for the development of clinical eating disorders (Haines & Neumark-Sztainer, 2006; Keel & Heatherton, 2010; Stice, 2002). BED and BN are nearby clinical diagnoses associated with regular occurrence of objective binge eating episodes, either with or without compensatory behaviors such as purging, fasting, or excessive sport (APA, 2013).

Even when diagnostic criteria for a specific eating disorder are not fulfilled, eating pathology associated with DE may lead to diagnoses of *other specified eating disorders* (formerly named *eating disorder not otherwise specified*), which constitute the vast majority of all eating disorder diagnoses (Allen, Byrne, Oddy, & Crosby, 2013; Machado, Machado, Goncalves & Hoek, 2007).

Food Addiction

Another important consequence can be seen in *food addiction*, a psychophysiological impairment that has recently gathered more attention among researchers and clinicians. During the last decade, several studies from neurophysiological research identified common physiological mechanisms associated with intake of psychoactive substances and foods (Avena et al., 2011; Davis & Carter, 2009; Gearhardt, Yokum et al. 2011; Sinha & Jastreboff, 2013).

The frequent consumption of energy-dense *comfort foods* in DE may foster brain developments comparable to addiction (Dallman, Pecoraro, & la Fleur, 2005). As a result, individuals experience symptoms in relation to eating, which would also characterize addictions: tolerance and increasing consumption; withdrawal symptoms; irresistible craving urges; continuous behavior despite of intentions to stop; etc. (Davis et al., 2011; Gearhardt, Corbin, & Brownell, 2009; Meule & Gearhardt, 2014; Pelchat, 2009). Food addiction then describes compulsive urges that arise from dependence on certain foods and manifest in DE behaviors, especially in objective binge eating (Davis, 2013), but also in the subclinical range of DE (Filbey, Myers, & DeWitt, 2012).

Altogether, the high prevalence rates and the potential negative health consequences of DE point out the need for effective treatments of this behavioral dysregulation.

2.2 Etiology of disinhibited eating

To understand the phenomenology of DE and derive options for interventions, it is crucial to scrutinize the most common antecedents of these behaviors. That is, the various factors that contribute to the etiology of single DE episodes, as well as maintenance factors for their recurrence.

Of course, biological and metabolic factors have been extensively discussed regarding their importance in food intake. Known mechanisms include the physiological regulation of hunger and appetite in the gastrointestinal system (e.g., Cummings & Overduin, 2007; Strader & Woods, 2005), influences of hormones and neurotransmitters (e.g., Morton, Cummings, Baskin, Barsh & Schwartz, 2006; Woods, Seeley, Porte, & Schwartz, 1998), and genetic influences (Rankinen & Bouchard, 2006).

However, during the last decades, eating-related psychological processes have received more attention and are today postulated to play a central role in the regulation of eating behavior (e.g., Berthoud, 2011; Egger & Swinburn, 1997; Haedt-Matt & Keel, 2011a). Several theories and empirical investigations in the field of obesity, eating disorders, and eating behavior have

contributed to the understanding of the multi-factorial psychological mechanisms that foster DE. The following chapter will introduce and focus on theories and studies that contributed to the development of the NFB protocol evaluated in this dissertation project.

The presentation of theories and findings on factors that etiologically contribute to DE will be organized as follows: (1) environmental factors, (2) individual factors, (3) cognitive factors, (4) affective factors, and (5) motivational factors. Afterwards, these different components will be integrated to a *synthesis model of antecedents in DE.* This heuristic model serves as a foundation and starting point for the identification of relevant psychophysiological activity that can be subject to regulation in a NFB treatment.

2.2.1 Environmental factors

One classic view on factors that influence DE comprises the influence of environmental or external cues. In 1968, Schachter proposed his *Externality Hypothesis of obesity*, stating that eating behavior in overweight and obese individuals is steered by external food-related cues rather than physiological hunger. Thus, heightened externality and impaired interoceptive abilities would mark the obese phenotype (Schachter, 1968).

Shortly afterwards, the view on externality – as a special and exclusive feature of obese individuals – has been challenged. Research findings indicated that heightened reactivity for external food cues is present throughout all weight classes (Herman, Olmsted, & Polivy, 1983; Meyers & Stunkard, 1980) and that dysregulated eating behavior is far more complex than plainly attributable to externality (Milich, 1975; Pliner, 1973; Rodin, 1981). However, various researchers have repeatedly found associations between responsiveness to external cues, dysregulated eating behaviors, and weight gain (e.g., R. G. Boswell & Kober, 2016; Rodin & Slochower, 1976; Van Strien, Herman, & Verheiden, 2012).

This becomes especially problematic in the light of an *obesogenic environment* (French, Story, & Jeffrey, 2001; Swinburn, Egger, & Raza, 1999). Egger and Swinburn (1997) highlighted the contribution of omnipresent environmental food cues and facilitated access to unhealthy foods in their ecological approach to the obesity pandemic. While this ecological approach predominantly appeals to health policy makers, psychological researchers have also repeatedly identified *food cue exposure* as a risk factor for DE (e.g., Fedoroff, Polivy, & Herman, 1997; Ferriday & Brunstrom, 2008; Stroebe, Papies, & Aarts, 2008). The omnipresence of food cues has been debated as crucial in boycotting attempts of dieting or

healthy eating, as food-rich environments tend to activate goals of hedonic consumption (Swinburn, Sachs, & Ravussin, 2009).

Aside from food cues, other body-related cues might indirectly contribute to the development of DE and bulimic symptoms, like repeated exposures with a skinny beauty ideal and the associated societal pressure to be thin (Stice, 2002). These unrealistic beauty ideals would foster bulimic symptomatology via pathways including *thin-ideal-internalization* and body dissatisfaction, resulting in dietary restraint and negative affect (Hawkins, Richards, Granles, & Stein, 2004; Stice, 2002; Stice & Shaw, 1994). The latter two factors will receive detailed attention in chapters 2.2.2 and 2.2.4.

In addition, various external stressors may result in the individual's perception of stress (or strain). Stressors like low socioeconomic status (McLaren, 2007), financial insecurity (Olson, Bove, & Miller, 2007), sleep restrictions (Spaeth, Dinges, & Goel, 2013; St-Onge et al., 2012), and urbanization (Van Son, Van Hoeken, Bartelds, Van Furth, & Hoek, 2006) are all assumed to contribute to sustained eating pathology. Psychosocial stressors, like teasing and weight-based stigmatization, may further aggravate the individual's stress experience (Almeida, Savoy, & Boxer, 2011; Friedman, Reichmann, Costanzo, & Musante, 2002).

2.2.2 Individual factors

Many environmental factors interact with individual dispositions. Three important trait-like factors will be summarized in this chapter: Cue reactivity, reward sensitivity, and dietary restraint. Schachter's *Externality Hypothesis* contributed to the research on two closely related dispositional factors: *external eating* as a personality trait (Schachter & Rodin, 1974; Van Strien et al., 1986) and the concept of food *cue reactivity* (Jansen, 1998; Overduin & Jansen, 1996). Both concepts are closely related and this chapter will therefore focus on the latter one.

The *cue reactivity* concept per se is derived from research on drug-use and addiction (Tiffany, 1990). It is based on the central assumption that physiological effects of drugs can be associated with external or internal cues via classical conditioning (Drummond, 2000; Rohsenow, Childress, Monti, Niaura, & Abrams, 1991). Jansen (1998) transferred the cue reactivity model to eating behavior, attempting to explain the occurrence of binge eating in terms of conditioning processes. She concluded that "cues which reliably signal food intake . . . may start to act as conditioned stimuli which trigger cue reactivity or conditioned responses. It is assumed that learned cue reactivity, e.g. the autonomic or biochemical responding and craving, increases the probability of (excessive) food intake." (Jansen, 1998, p. 269).

Jansen and her research group later included the known physiological phenomena of conditioned anticipatory responses to food-cues (*cephalic phase responses*) into the cue reactivity model. The researchers highlighted, how these physiological and psychological processes are directly linked to food craving and food intake, rather than solely constituting metabolic reactions preparing the digestion of food (Nederkoorn, Smulders, & Jansen, 2000).

Cue reactivity is also marked by an attentional and motivational bias, and therefore an enhanced salience of food-related cues (Castellanos et al., 2009; Giel et al., 2011). This in turn leads to more frequent confrontations with these cues, increases consumption, and hereby again strengthens conditioned responses. This chain of events may then subsequently result in a vicious circle, by maintaining and strengthening individual food cue reactivity.

The fact that individual food cue reactivity is related to craving, DE, and weight gain has been consistently reported in the literature on eating behavior (for a recent meta-analysis, see R. G. Boswell & Kober, 2016). Further, heightened cue reactivity reliably manifests in subgroups with stronger eating pathology, such as overweight and obese individuals, restrained eaters, or individuals with eating disorders (Brunstrom, Yates, & Witcomb, 2004; Ferriday & Brunstrom, 2011; Nijs & Franken, 2012; Tetley, Brunstrom & Griffiths, 2009). Thus, cue reactivity can be seen as an important dispositional, yet learned, risk factor for the occurrence of DE.

Another dispositional risk factor is *reward sensitivity*, a trait that roots in an over-activation of dopaminergic brain structures. It is characterized by dispositional increases in the tendencies and motivations to approach pleasurable stimuli and situations (Davis, Strachan, & Berkson, 2004). This trait is principally congruent with Gray's (1987) *Behavioral Approach System*, the derived *Reinforcement Sensitivity Theory* and the concept of *Impulsivity* (Nederkoorn, Smulders, Havermans, Roefs, & Jansen, 2006). All of these models indicate that some individuals tend to show disinhibition and approach tendencies whenever reward is obtainable, regardless of possible negative outcomes (Berridge, 2009; Mitchell & Nelson-Gray, 2006). Thus, individuals with high reward sensitivity are less likely to show inhibitory control and do automatically respond to pleasurable cues, disregarding possible adverse consequences.

This trait has severe implications for eating behavior. Eating per se is a pleasurable experience for the majority of individuals, and reward sensitivity fosters a motivational bias towards this behavior (Davis et al., 2004). The resulting impaired response inhibition has been stated as a crucial factor in disinhibition, binge eating, and unhealthy eating behaviors in general (Jasinska et al., 2012; Loeber et al., 2012; Rosval et al., 2006). A combination of high reward

sensitivity, with the aforementioned obesogenic environment (Guerrieri, Nederkoorn, & Jansen, 2008) and/or the trait of restrained eating (Jansen et al., 2009), increases the likelihood for DE to occur.

The third dispositional factor is continuous dietary restraint, most prominently known as *restrained eating* (Herman & Mack, 1975; Herman & Polivy, 1975). *Restraint Theory* focuses on individuals, so-called *restrained eaters* (REs), who intend to restrict their caloric intake with the goal of weight loss or weight maintenance (Herman & Mack, 1975). REs often use methods like dieting, meal skipping, calorie counting, or abstinence from certain – mainly high caloric – foods to reach their goal (Westenhöfer, 1992).

Most importantly, REs (in the notion of Herman and colleagues) usually do *not* attain their goal of weight loss or maintenance. Instead, they can be described as unsuccessful dieters who tend to show weight gain over time (Israeli & Stewart, 2001; Tuschl, Laessle, Platte & Pirke, 1990). Once REs exceed their preset dieting threshold (e.g., a certain amount of calories, or consumption of "forbidden" high caloric foods), they tend to consume even more food than usual and often experience binge eating episodes (Heatherton, Polivy, & Herman, 1991; Lowe, 1993; Ruderman, 1986). The occurrence of DE thus contradicts their original intentions of dieting, marking an *ironic process* that elicits distress and general dissatisfaction (Boon, Stroebe, Schut, & IJntema, 2002; Griffiths et al. 2000).

Exposure to thin beauty ideals (cf. 2.2.1) has been considered an important factor that fosters restrained eating as a means to attain a goal of weight control (Stice, 2001). Further, REs are more vulnerable to experience other cognitive, affective, and motivational factors contributing to DE (cf. 2.2.3 - 2.2.5). Hence, they constitute one of the most representative subclinical populations for studies on mechanisms that contribute to the development of eating disorders (e.g., Field et al., 2005; Harrison & Cantor, 1997).

2.2.3 Cognitive factors

A wide range of cognitive factors facilitates DE, like general cognitive load that undermines capacities for self-control, intrusive thoughts about food, goal conflicts, perceived ego-threat, and rumination. All of these processes are related to simultaneous affective responses that can initiate motivations to eat. The focus will therefore first lie on some influential cognitive theories that have informed research on DE, before affective and motivational factors are addressed separately.

One important finding from studies on REs was the observation that cognitive load, for example, task demands or cognitive stressors, generally tends to undermine intentions of restrained eating (Byrd-Bredbenner, Quick, Koenings, Martin-Biggers, & Kattelmann, 2016; Ward & Mann, 2000; Wallis & Hetherington, 2004). Researchers proposed that these cognitive demands compete for resources, which are usually allocated to cognitive restraint in REs (e.g., planning of meals, calorie counting, etc.). Thus, tasks that are more urgent in a specific situation suppress original long-term intentions (weight control) and cognitive resources are allocated to ad-hoc situational demands.

In more general research on self-control processes, this effect has been coined as *ego depletion* (Baumeister, Bratslavsky, Muraven, & Tice, 2000) with the metaphor of self-control resembling a muscle. It was assumed that self-control capacities can be exhausted, leaving no more strength for willpower (Muraven & Baumeister, 2000). Thus, self-control is seen as a limited resource in general and the strength-model has received empirical support (for a meta-analysis, see Hagger, Wood, Stiff, & Chatzisarantis, 2010). While ego depletion refers to overall self-control processes in relation to performance, it can also explain the strong tendency towards disinhibition in REs (Hofmann, Rauch, & Gawronski, 2007; Kahan, Polivy, & Herman, 2003).

Addressing another related cognitive process, the *Elaborated Intrusion Theory of Desire* (EI-theory; Kavanagh, Andrade, & May, 2005) states, that food cravings and subsequent disinhibition with resultant binge eating can be initiated and aggravated by (apparently) spontaneous thoughts about food (May, Andrade, Kavanagh, & Hetherington, 2012). These thoughts could however be generated by bottom-up processes, for example, even in semi-conscious confrontations with food-related cues. Following EI-theory, spontaneous thoughts about foods are then intensively and vividly elaborated. This leads to high working memory capacity utilization by imagery processes and results in appetitive motivations, being an intense desire to consume a specific food (i.e., *food craving*). Further, it is assumed that this cognitive process is accompanied by emotional correlates (negative affect and stressful experiences). In line with theories on cognitive load, the high utilization of cognitive resources (i.e., elaboration and imagery processes) contributes to subsequent disinhibition (Kavanagh et al., 2005; Tiggemann & Kemps, 2005).

Goal conflicts are likely to arise, especially, in individuals who intend to watch or reduce their weight. In the *Goal Conflict Model of Eating Behavior*, Stroebe and colleagues (2008) pointed out how the competing goals of long term weight control (restrained eating) and hot

(i.e., extremely attractive and salient) short term temptations of hedonic eating compete, lead to ambivalence, and influence myopic behavioral responses. In dieters, these responses often cause dietary failure, because the short-term goal of eating enjoyment inhibits the long-term goal of weight control (Stroebe, Mensink, Aarts, Schut, & Kruglanski, 2008). Goal conflicts may result in rumination, negative affect, and stressful arousal (Mansell, 2005; Thomsen, Tønnesvang, Schnieber, & Olesen, 2011), all three in themselves important factors in the etiology of DE.

Another classical theory views binge eating behavior as a means to avoid ruminative and distressing thoughts, for example after ego-threatening situations. In 1991, Heatherton and Baumeister proposed the *Escape Theory* to explain disinhibition. The researchers suggested that high self-awareness due to perfectionism or perceived demands, is an aversive and ego-threatening state for many individuals. This would in turn lead to an intense desire to escape from ego-threatening thoughts (Heatherton & Baumeister, 1991). Binge eating would then serve as an instrument to attain relief from these aversive states, as concentration is focused on more basic, hedonic processes that require awareness. The assumptions of this theory have been strongly influential and are still in line with many theories on the development of binge eating and bulimic symptomatology (e.g., Sherry & Hall, 2009; Stice, 2002; Waters, Hill, & Waller, 2001). However, empirical support is mixed (e.g., Blackburn, Johnston, Blampied, Popp, & Kallen, 2006; Stein et al., 2007; Wallis & Hetherington, 2004). Some recent studies conclude that the aversive and stressful emotional state and subsequent emotion regulation attempts might be the most crucial factor contributing to DE (for a review, see Leehr et al., 2015).

This latter point is taken into account by the *emotional cascades* model postulated by Selby, Anestis, and Joiner (2008). The authors drew from research on emotion regulation (Gross, 1998) to explain how dysfunctional emotion regulation strategies, like rumination and catastrophizing, potentiate the effect of small emotional triggers by eliciting overwhelming emotional cascades. This process is perceived as aversive and leads to aroused states of tension that the individual tries to regulate. Ongoing rumination is notably identified as a predictor of resulting dysfunctional behaviors, whereof the authors explicitly mention binge eating as a possible outcome (Selby et al., 2008).

There is a broad evidence for the relationship between rumination, increased cognitive and physiological arousal, and negative affect (Brosschot, Gerin, & Thayer, 2006; Thomsen, 2006). The emotional cascades model emphasizes the close connection between rumination and states of aversive tense arousal that the individual seeks to calm by means of dysfunctional behaviors,

such as binge eating. The theory further supports the general notion of a close and bidirectional entanglement of cognitive and affective processes (e.g., Lazarus, 1991). In line with earlier theories, DE may hence be seen as a result of entangled dysfunctional cognitive and affective processes marked by arousal – an important connection that will be further explored in the following chapter.

2.2.4 Affective factors

Theories on the influence of affective states on eating behavior have a long tradition. In 1957, Harold and Helen Kaplan published their *Psychosomatic Concept of Obesity*, proposing that obese individuals use overeating as a strategy to cope with negative emotions, for example, to reduce anxiety. This early theory inspired a huge number of offspring models and affective antecedents have continuously been taken into account in various emerging theories on DE.

Even in the first studies on restrained eating, Herman and Polivy (1975) found anxiety to disinhibit dietary restraint and increase food intake in REs. In the *Dual Pathway Model of Bulimia*, Stice (Stice, 1994; 2001) included negative affect as one of two essential pathways (beneath restrained eating) to explain the development of bulimic symptoms through multiple influence factors, such as social pressure to be thin and body mass.

Waters and colleagues (2001) provided an even more central role for negative affect. They stated that the influence of negative cognitions and resulting negative affect, together with reduced inhibitory processing and emerging food cravings causally lead to binge eating. The previously introduced emotional cascades model (Selby et al., 2008) highlighted the role of emotion and dysfunctional attempts of emotion regulation in the etiology of DE. Finally the concept of emotional eating (e.g., Van Strien et al., 1986) proposed that attempted affect-regulation by means of food intake and dysfunctional coping or emotion regulation strategies are a core antecedent of DE and should be assessed with a primary focus (e.g., Arnow, Kenardy, & Agras, 1995; Gianini et al., 2013; McCarthy, 1990; Telch, 1997).

In the less clinically oriented *Five-way Model*, Macht (2008) highlighted the bidirectional relationship between emotions and eating. He pointed out that emotions are in several ways related to eating and vice versa, while mechanisms may be differentially dominant in individuals (e.g., REs and emotional eaters) and dependent on the distinct emotion categories and precursors. Macht explained, that (1) emotional properties of foods influence the food choices of an individual, (2) extremely intense emotions (e.g., panic), reduce rather than increase eating, but still (3) moderate to intense negative emotional states reduce the cognitive regulation of eating, (4) negative emotions lead to eating as an emotion regulation strategy and

finally, (5) some individuals also tend to eat in congruence with their emotions, marking hedonic eating in pleasurable situations (Macht, 2008, p. 5). However, it has to be annotated that emotional eating does most frequently occur in circumstances of negative affect, which are marked by diffuse emotional states (Van Strien et al., 1986), long term chronic stressors, or cognitively aroused states (Adam & Epel, 2007, Groesz et al., 2012).

Another affective factor that is often used synonymously with emotional states is stress (Lazarus, 2000). While the concepts are distinct, emotional states as well as perceived stress are usually dependent on a certain degree of arousal (Boucsein, 2012; Russell, 2003), and thus accompany each other. Negative emotional states may be perceived as stressful and stressful situations may be accompanied by negative emotional responses, like anxiety or anger.

Stress influences eating behavior via various biological and psychological pathways, for example in a complex interplay of neurophysiological (activation of the hypothalamus-pituitary-adrenal axis) and hormonal responses (release of cortisol and its influence on insulin and other hunger-regulative hormones) (Adam & Epel, 2007; Dallmann, 2010). It may also impair volitional processes in a physiological way, as there has been some evidence for dysfunctional neuronal alterations in prefrontal cortical areas due to chronic stress (Arnsten, 2009; Radley et al., 2004). Peters and colleagues (2004; 2011) proposed the *Selfish-Brain-Theory*, stating that stress may be a central mechanism of the brain to demand energy in the form of glucose, by eliciting food cravings and initiate eating. In line with theories of McEwen (2000; 2007), he thus underlined the central position of the brain within the stress-network and also delivered a theory to explain the increased need for energy dense food in stressful times reported by individuals (Adam & Epel, 2007, Peters et al., 2011), which contributed to the understanding of DE.

Beneath theoretical considerations, there is broad empirical evidence for the causal role of affective factors, like emotions, mood, and stress, in DE. These factors seem to exert even stronger influence in some subpopulations, such as REs, overweight populations, and individuals diagnosed with eating disorders. Studies that highlight the crucial influence of affective factors have accumulated within the last years (Ball & Lee, 2000; Stickney, Miltenberger, & Wolff, 1999), with stable associations found in cross-sectional studies (e.g., Vanderlinden, Dalle, Grave, Vandereycken, & Noorduin, 2001; Vanderlinden et al., 2004), experimental and quasi-experimental studies (e.g., Chua, Touyz, & Hill, 2004; Habhab, Sheldon, & Loeb, 2009; Royal & Kurtz, 2010), longitudinal prospective studies (e.g., Greeno,

Wing, & Shiffman, 2000; Wardle, Steptoe, Oliver, & Lipsey, 2000), as well as in studies using ecological momentary assessment (for a meta-analysis, see: Haedt-Matt et al., 2011b).

Affective states have generally long been known to influence or even fuel motivation and subsequent behavior (Cardinal, Parkinson, Hall, & Everitt, 2002; M. G. Seo, Feldman-Barrett, & Bartunek, 2004). Therefore, it is not surprising that the presented affective antecedents are closely related to not only DE, but also to proximate motivational antecedents of DE.

2.2.5 Motivational factors

With regard to the motivational factors that influence DE, the most proximate and dominant factor is *food craving*. Food craving is defined as "an intense desire to eat a specific food" (Pelchat, 2002, p. 347) that may induce food-seeking behaviors and subsequent consumption (Cepeda-Benito & Gleaves, 2001; Lobera, 2012). The term *craving* is also the common description for a motivational state that initiates drug-seeking behaviors with a corresponding definition in addiction (Verheul, van den Brink, & Geerlings, 1999). This underlines the close connection of food and substance craving, which has also been established in terms of shared neural circuitry and neurotransmitter pathways (e.g., Styn, Bovbjerg, Lipsky, & Erblich, 2013; Volkow, Wang, Fowler, Tomasi, & Baker, 2012) and the concept of food addiction (cf. 2.1.3).

The phenomenology of food craving is marked by a higher prevalence in women compared to men (Lafay et al., 2001; Pelchat, 2002), and a negative correlation with age (Pelchat, 1997). Food craving is frequently described as being associated with ruminative and intrusive thoughts and imagery about the craved food (Curtis & Davis, 2014; Harvey, Kemps, & Tiggemann, 2005; Kavanagh et al., 2005), accompanied by states of tense arousal (Waters et al., 2001).

Furthermore, it has been stated that food craving has a close connection to dysphoric mood (Hill, Weaver, & Blundell, 1991), especially in women (Dubé, LeBel, & Lu, 2005; Lafay et al., 2001). While some researchers found that food cravings do not necessarily depend on physiological deprivation (e.g., Hill et al., 1991; Lafay et al., 2001), others reported that dietary restraint – especially among REs – increases the likelihood of food cravings to occur (e.g., Pelchat, 1997; Polivy, Coleman, & Herman, 2005).

Food craving elicits irresistible urges, disinhibition, and loss of control experiences (Hill, 2007; Pelchat, 2009). Thus, it often results in DE and other bulimic symptoms (Chao, Grilo, & Sinha, 2016; Gendall, Joyce, Sullivan, & Bulik, 1998; Mussell et al., 1996), as well as increased caloric intake and weight gain (R. G. Boswell & Kober, 2016). Food craving further reliably discriminates between individuals with successful versus unsuccessful dieting experiences

(Meule, Lutz, Vögele, & Kübler, 2012a), marking the importance of this motivational factor in the etiology of DE.

2.2.6 Synthesis of antecedents

The previous chapters provided an overview on different environmental, individual, cognitive, affective, and motivational factors that have been considered in psychological theories to explain DE. The proximate antecedents of DE seemingly share one common ground, which was essential for considerations on the NFB protocol developed for the present dissertation project: the relevant cognitive, affective, and motivational factors are closely linked to stressful states of tense arousal.

For example, rumination, goal conflict, and intrusive thoughts about food have been described as prominent antecedents of DE that are accompanied by stressful states of tense arousal (e.g., Curtis, & Davis, 2014; Mansell, 2005; Selby et al., 2008). Affective factors, such as negative emotions and mood, or stress, have been proclaimed as the most prominent and best explored antecedents of DE that are naturally marked by tense arousal (e.g., Adam & Epel, 2007; Gibson, 2006). The same pattern accounts for food craving as the proximate motivational antecedent of DE (Chao et al., 2016; Curtis & Davis, 2014; Waters et al., 2001).

Further, it was pointed out how environmental aspects, (e.g., repeated cue exposure and other external stressors), as well as individual dispositions (e.g., restrained eating, reward sensitivity, and cue reactivity), contribute to the more proximate antecedents and may amplify the stressful experience. Disinhibition and subsequent DE behaviors may then occur as a means to reduce the tense state of arousal (Heatherton & Baumeister, 1991; Macht, 2008; Selby et al., 2008), while self-control and restraint are undermined (Herman & Mack, 1975; Tice et al., 2001). Repeated occurrences of DE may then directly or indirectly (mediated by weight gain or lowered self-efficacy and self-esteem) elicit distress and negative affective states (e.g., shame, guilt, fear of weight gain), more rumination and perceived conflict, and possibly even more dietary restriction. When DE behaviors occur frequently, learned associations and physiological developments may be influenced, leading to more cue reactivity and reward sensitivity. Together, antecedents and outcomes form a vicious circle that may in the long run be responsible for the establishment of several negative health-related consequences.

The review of previous influential theories shows that most etiological models consider the prominent influence of tense arousal in DE, either directly or indirectly. Table 2 summarizes the introduced theories, highlighting the key constructs related to tense arousal.

Table 2

Theories on the etiology of DE with key antecedents and their association with arousal.

Theory	Explained construct	Key antecedents
Externality Hypothesis of Obesity (Schachter, 1968)	Obesity	• Heightened externality • Impaired interoceptive abilities
Obesogenic environment (Egger & Swinburn, 1997)	Overeating and obesity	• Availability and omnipresence of food in the environment
Cue reactivity model of binge eating (Jansen, 1998)	Binge eating	• Conditioned salience of food cues • Individual cue reactivity*
Restraint theory (Herman & Mack, 1975)	Disinhibition (in REs)	• Dietary restraint • Disinhibitive factors (e.g., negative affect*, forced consumption, cognitive load*)
Dual pathway model of bulimia (Stice, 1994; 2001)	Bulimic symptomatology	• Dietary Restraint • Negative affect*
Model of the psychological processes involved in non-restrictive binge-eating (Waters et al. 2001)	Binge eating	• Negative cognitive states • Negative affective states* • Food Craving* • Reduction in inhibitory processing
Ego depletion (Baumeister et al., 2000)	Failure in self-control	• Limited resources for self-control • Cognitive load / arousal*
Elaborated Intrusion theory of desire (Kavanagh et al., 2005)	Food craving and subsequent consumption	• Spontaneous intrusive thoughts about food • Cognitive elaboration* / rumination*
Goal Conflict Model (Stroebe et al., 2008)	Disinhibition / Dietary failure	• Goal conflict* • Anticipated reward
Escape Theory (Heatherton & Baumeister, 1991)	Binge Eating	• Perceived ego-threat* • Intended distraction from self-awareness • Rumination*
Emotional cascades model (Selby et al. 2008)	Binge eating (and other dysfunctional behaviors)	• Negative affect* • Dysfunctional emotion regulation • Rumination*
Psychosomatic concept of obesity (Kaplan & Kaplan, 1957)	Obesity	• Emotional / physiological arousal* • Impaired interoceptive abilities
Five-Way-Model (Macht, 2008)	Emotional eating	• Associations between emotions and eating • Emotional arousal* • Anticipated reward • Trait eating behavior (e.g., dietary restraint)
Selfish Brain Theory (Peters et al., 2004; Peters, 2011)	Stress-induced eating, Obesity	• Priority of the cerebral metabolism • Stress* • Impaired *brain-pull*, exaggerated *body pull*

Note. Asterisks (*) mark key antecedents that are linked to states of tense arousal.

Based on the reviewed key factors in the etiology of DE, the relationship among antecedents is depicted in a heuristic integrative model, accounting for the multifaceted psychological influences in the etiology of DE (see Figure 2).

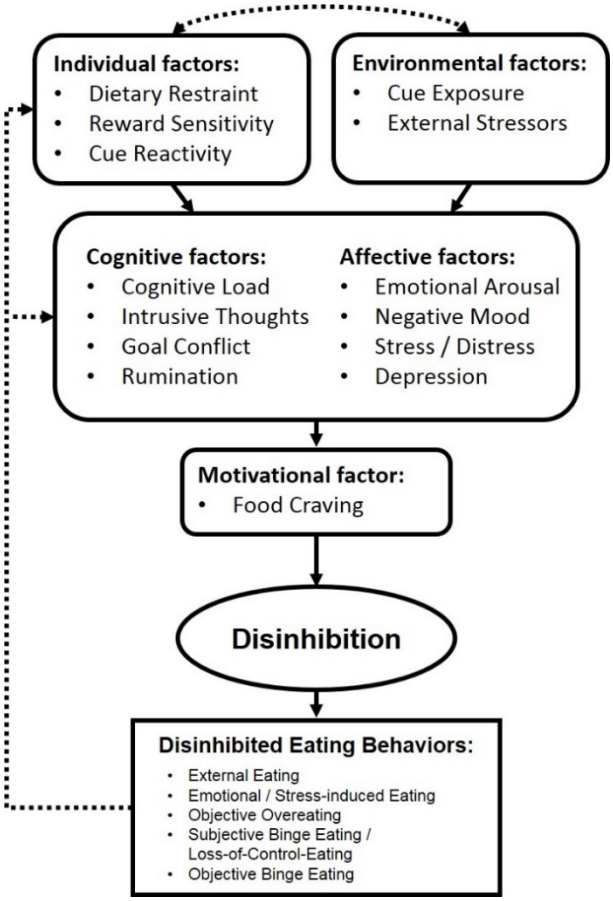

Figure 2. Synthesis-model of relevant antecedents of disinhibited eating behaviors.

Given the proposed common ground in tense arousal, this synthesis provides a relevant starting point to identify suitable treatment components and relevant psychophysiological target activity that can be addressed in a NFB protocol for DE. In the next chapter, psychophysiological factors associated with DE will be identified, with a focus on correlates of tense arousal in DE and its antecedents (cf. 2.3). Afterwards, a short overview on relevant treatment options based on some of the introduced antecedents will be provided (cf. 2.4). Both, physiological correlates of the antecedent cluster and probable efficacious treatment-setups, constitute the foundation for the development of the present NFB protocol.

2.3 Psychophysiological factors in disinhibited eating

Psychophysiology describes an area of research, which studies "relations between psychological manipulations and resulting physiological responses, measured in the living organism, to promote understanding of the relation between mental and bodily processes" (Andreassi, 2007, p.2). Within the field of psychophysiology, research on arousal (or activation) constitutes a major interest, because different physiological indicators obtained with unobtrusive measurements (e.g., electrodermal activity, electrocardiogram, and EEG) provide detailed insights into arousal processes (Andreassi, 2007). Given the central role of tense arousal in the etiology of DE, a view on psychophysiological correlates of DE and its antecedents can provide deeper insights for possible treatment options.

With regard to previous psychophysiological research on DE, two main paradigms can be outlined: One stream of studies compares baseline or state-specific (e.g., stress- and mood-related) differences between individuals without manifest DE and those who show DE (e.g., binge eating) or its antecedents (e.g., restrained eating). The other stream of research examines distinct responses during food cue exposure (i.e., cue reactivity), often in comparison to neutral or emotional cues, in subgroups with or without DE.

In line with the scope of this dissertation, the focus of this chapter will lie on arousal-related physiological responses in DE that inform classical biofeedback applications. These measures comprise peripheral physiology, like skin conductance, blood pressure, heart rate, and heart rate variability. Moreover, studies that apply spectral EEG research are naturally of major interest for the development of a NFB protocol and will therefore be highlighted. For an overview, the key findings of relevant psychophysiological studies on DE and related antecedents are systematically presented in Tables 3 to 5.

Other frequently used psychophysiological indicators include EEG event-related potentials (ERPs), visual processing (eye-tracking), and functional brain imaging. For details on results obtained with these measures, the interested reader is referred to some recent systematic reviews on attentional processing of food cues (Giel et al., 2011), ERPs in abnormal eating (Wolz, Fagundo, Treasure, & Fernández-Aranda., 2015), brain regions involved in food cue processing (Pursey et al., 2014), food related impulsivity (Schag, Schönleber, Teufel, Zipfel, & Giel, 2013), and compulsive seeking of food rewards (Berridge, 2009).

2.3.1 Peripheral psychophysiology in relation to disinhibited eating

Electrodermal activity (EDA) is a classical indicator of arousal due to the sympathetic innervation of the sweat glands (Boucsein, 2012). It has been applied in various studies on DE. With regard to induced states of stress, Tuschen-Caffier and Vögele (1999) found increases in skin conductance levels (SCL) in females (BN, REs, controls) viewing film clips as interpersonal stressors. REs showed larger SCL responses than BN patients and healthy controls. More often, EDA was investigated in food cue exposure paradigms. Here, several studies found increased SCLs and thus heightened arousal in various samples exposed to salient food cues (Nederkoorn, Smulders, Havermans, & Jansen, 2004; Nederkoorn, Smulders, & Jansen, 2000; Rodríguez, Fernàndez, Cepeda-Benito, & Vila, 2005; Vögele & Florin, 1997). These findings are in line with observations on electrodermal cue reactivity in drug addiction (B. L. Carter & Tiffany, 1999).

Blood pressure (BP) is another traditional arousal indicator used in psychophysiological research on DE. In line with findings on SCL, arousal due to interpersonal stressors also leads to increases in BP (Tuschen-Caffier & Vögele, 1999). During a stress task, Koo-Loeb and colleagues (2000) found increased BP in female subjects with high bulimic symptoms compared with subjects who reported low bulimic symptoms (Koo-Loeb, Costello, Light, & Girdler, 2000). Corresponding results were reported by Klatzkin, Gaffney, Cyrus, Bigus, and Brownley (2015). Klatzkin et al. (2015) further found higher stress-related BP in obese subjects with BED compared with obese non-BED subjects and normal-weight controls.

In food cue exposure, BP responses corresponded to results obtained for SCL with marked increases during exposure (Nederkoorn et al., 2000; Vögele & Florin, 1997). Further, Nederkoorn and colleagues (2000) found significant relations between increases in BP during food cue exposure and subjective food craving. They also observed that BP increases were positively correlated with restrained eating. Again, these results mirror those in substance-related cue reactivity research (B. L. Carter & Tiffany, 1999).

Electrocardiographic (ECG) measures are another means to assess arousal. ECG parameters include heart rate (HR) and heart rate variability (HRV), as well as phasic responses to stimuli, manifesting in deceleration (orienting responses) or acceleration (defense response) of the heart beats.

For HR, Tuschen-Caffier and Vögele (1999) reported lower baseline HR in REs compared with BN subjects and controls. In response to stress, stronger HR increases have been reported for patients with eating disorders (BN & BED) compared with healthy controls (Messerli-

Bürgy, Engesser, Lemmenmeier, Steptoe, & Laedrach-Hofmann, 2010). However, Ginty, Phillips, Higgy, Heaney, and Carroll (2012) found reduced stress-related HR activity in subjects with disordered eating compared with healthy controls. In response to food cues, HR usually is marked by an increase, indicating heightened arousal (Nederkoorn et al., 2000; Nederkoorn et al., 2004; Vögele & Florin, 1997). Svaldi, Tuschen-Caffier, Peyk, and Blechert (2010) further reported increased HR in response to high versus low caloric food.

Recently, HRV measures have gathered more attention in psychophysiological research on DE. HRV measures provide indicators of sympathetic (low frequency activity: LF; very low frequency activity: VLF) or parasympathetic (high frequency activity; HF) dominance in heart innervation, and markers of sympathovagal balance (LF/HF). These measures are indicative for stress-reactivity and flexible adaptation to environmental demands, with low HRV (sympathetic dominance) indicating worse adaptation.

In a study on obese subjects with BED and weight-matched controls, Friederich et al. (2006) found lower HRV-HF in BED subjects than in controls during a mental-stress task. Low HRV-HF during stress was further related to more frequent binge eating. Messerli-Bürgy et al. (2010) found less adaptive HRV in subjects with eating disorders compared to healthy controls. Further, Meule, Vögele, and Kübler (2012) found sympathetic dominance to be related to trait restrained eating. In another study, this research group reported a positive correlation between cardiac vagal control (parasympathetic dominance) and successful dieting (Meule, Lutz, Vögele, & Kübler, 2012b).

For food cue exposure, Nederkoorn, Smulders, and Jansen (2000) reported increased sympathetic arousal (HRV-LF) during exposure in healthy women. However, Udo et al. (2014) reported higher parasympathetic arousal (HRV-HF) in obese compared to non-obese subjects during food cue exposure and no differences in baseline HRV or during mood induction. Geisler, Kleinfeldt, and Kubiak (2016) reported increased HRV during food cue exposure in REs after a self-control demanding task, which was interpreted as an indication of effortful self-control processes after ego depletion. Thus, research on HRV yielded mixed results.

Another interesting insight on psychophysiological responses can be inferred from two electromyographic (EMG) valence indicators: Startle reflex magnitude and activity of the M. corrugator supercilii; both indicating negative valence. Despite of the usually assumed hedonic properties of food, several studies showed that implicit responses to food cues are ambivalent and may even be rather negatively than positively toned. These findings were especially pronounced in populations with eating disorders (Drobes et al., 2001; Mauler, Hamm, Weike,

& Tuschen-Caffier, 2006) and in response to high caloric foods (Rodríguez et al., 2005; Svaldi et al., 2010). The observed negative valence of food cues contributes to the assumption that tense arousal, as a correlate of DE antecedents, has a negative connotation.

Table 3 provides an overview on key findings of studies that applied peripheral psychophysiology in DE research.

2.3.2 Spectral EEG research on disinhibited eating

Spectral analyses of the spontaneous EEG, acquired from the human scalp, constitute a classical paradigm in psychophysiological arousal research: it is assumed that the spontaneous EEG reflects states of consciousness and different levels of cognitive arousal (Andreassi, 2007). A dominance of slower EEG frequencies is associated with states of sleep, drowsiness, or relaxation, whereas a dominance of faster EEG frequencies is indicative for alert and aroused cognitive states (e.g., attention, reasoning, concentration, or stress). Hence, EEG researchers traditionally postulate a positive relationship between EEG frequency rate and arousal (Andreassi, 2007; Demos, 2005).

As early as in 1979, Rau, Struve, and Green found phenomenological abnormalities in the spontaneous EEGs of patients with compulsive eating, based on case studies and visual inspection of the EEG signals. Alas, this research stream has not been perpetuated for several decades. Despite of the assumed association of the spectral EEG with arousal and the importance of arousal in DE, spectral EEG research on DE is very rare to date (Bartholdy et al., 2013; Wolz et al., 2015).

With regard to resting state phenomenology, two studies addressed frontal EEG asymmetry in the alpha band (8-12 Hz, on F3 and F4) associated with DE. Silva and colleagues (2002) found restrained eating in a female student sample to be associated with greater right frontal asymmetry (i.e., an avoidance tendency), whereas Ochner and colleagues (2009) observed a left frontal asymmetry (i.e., an approach tendency) in overweight and obese females. In addition, left frontal asymmetry was correlated with self-reported eating-related disinhibition, but not with binge eating symptoms in particular (Ochner, Green, van Steenburg, Kounious, & Lowe, 2009). Thus, there may be population-based differences with regard to frontal EEG asymmetry.

Three studies addressed EEG spectral activity in response to confrontations with food cues and found evidence for increases of high frequency EEG beta activity (14-30 Hz), an indicator of cognitive arousal (Demos, 2005), to be involved in DE. For obese women with binge eating

behaviors, Tammela and colleagues (2010) reported increased frontal EEG beta activity (14-20 Hz) during food cue exposure. EEG beta power was greater in women with BED, compared to women without BED. Increases in EEG beta activity correlated positively with self-reported, eating-related disinhibition tendencies and binge eating severity. The researchers did not observe any differences or effects for other EEG spectral ranges (Tammela et al., 2010). Similar results were obtained by Hume, Howells, Rauch, Kroff, and Lambert (2015), who found elevated EEG beta activity (15-30 Hz) on frontal and central electrode positions in overweight and obese – but otherwise healthy – women during a food-modified Stroop-test. In a recent study, the same research group (Hume, Howells, Karpul et al., 2015) found higher right frontal EEG beta activity in response to a food-modified Stroop-test in women who had previously been overweight but reduced their weight, compared with low weight controls. Results of the EEG studies on DE are summarized in Table 4.

Together, these studies provide first evidence for the relevance of fast EEG spectral activity during food cue exposure in populations with DE. However, given the small number of spectral EEG studies on DE, a broader view on studies which examined related concepts – such as addiction and aforementioned antecedents of DE – can assist in backing up this first evidence.

2.3.3 Spectral EEG research in areas related to disinhibited eating

A look on addiction research provides further striking evidence for the involvement of fast EEG beta activity in processes associated with dysfunctional consumption behaviors (for a review, see Parvaz, Alia-Klein, Woicik, Volkow, & Goldstein, 2011). For example, enhanced cue reactivity in response to drug cues was marked by general EEG desynchronization, an increase of EEG high beta activity and a decrease in EEG alpha activity in individuals with cocaine-dependence (Liu, Vaupel, Grant, & London, 1998). Similarly, Reid et al. (2003) showed increased EEG beta activity among participants with cocaine-dependence in response to drug videos, accompanied with a decrease in EEG delta activity. Knott and colleagues (Knott, Cosgrove et al., 2008; Knott, Nacchache et al., 2008) found elevated EEG beta activity among smokers when confronted with smoking-related cues and imagery, especially in female participants. Here, EEG beta activity was also distinctly associated with the urge to smoke. Littel, Franken, and Van Strien (2009) obtained similar findings, showing elevated EEG beta in response to cigarette cues in smokers, but not in ex-smokers.

The hyperarousal reflected in EEG beta activity (Saletu-Zyhlarz et al., 2004) is also characteristic for dysfunctional brain activity in patients with alcohol-dependence. It was therefore discussed as an electrophysiological vulnerability factor in the general development

of addictions (Begleiter & Porjesz, 1999; Rangswamy et al., 2002; Winterer et al, 1998). Further, EEG high beta activity in baseline was an accurate classifier to predict relapse among formerly abstinent patients with alcohol-dependence (Bauer, 2001). In conclusion, Parvaz and colleagues summarized that "drug-associated stimuli are related to significantly higher neural activations, suggesting an increase in incentive salience and arousal" (Parvaz et al., 2011, p. 613). The consistent pattern shows concordance with results from peripheral psychophysiology and neuroimaging in responses to food cues and associated with food craving. In addition, recent research suggests, that EEG high beta activity (20-35 Hz) in frontal to central scalp areas may be related to confrontation with stimuli that generally indicate reward (HajiHosseini & Holroyd, 2015). Given the rewarding properties of addictive substances and foods, these results point in a unifying direction to explain the phenomenon of increased EEG beta activity during food and drug exposure.

Besides the findings from addiction research, a wide range of psychological factors presented in the synthesis model of antecedents in DE is associated with EEG beta activity. Several researchers reported aroused states of general and emotional stress to be associated with shifts to faster EEG spectral activity in the beta ranges (e.g., Hall et al., 2000; Hayashi et al., 2009; Jena, 2015; S.-H. Seo & Lee, 2010). On a cognitive level, researchers have linked rumination, worry, and negative affect to EEG beta activity (Andersen, Moore, Venables, & Corr, 2009; W. R. Carter, Johnson, & Borkoves, 1986; Sokhadze, 2007), lending even more evidence to the importance of this physiological correlate in relation to DE. Detailed results from EEG spectral studies in addiction research and on DE-related antecedents are depicted in Table 5.

Interestingly, EEG beta activity as an indicator of hyperarousal also frequently occurs in relation to insomnia (e.g., Hall et al., 2000; Maes et al., 2014; Nofzinger, et al., 2000). Within the last decade, researchers suggested that certain subcortical structures in the hypothalamic areas (so-called *hypocretins*, with *orexins* as active neuropeptides) serve as a shared hub to elicit hyperarousal in sleep disturbance, dysfunctional eating behaviors, and substance-use disorders (Berridge, Ho, Richard, & DiFeliceantonio, 2010; Furlong, Vianna, Liu, & Carrive, 2009; Piccoli et al., 2012). These subcortical structures are also linked to the generation of fast spectral EEG activity. They might thus contribute to simultaneous activation of cortical areas involved in problematic consumption and fast EEG activity as an indicator of hyperarousal.

Altogether, psychophysiological research on DE highlights the relevance of tense arousal as a correlate of DE, especially in response to stressors and food cues. Arousal responses

manifest in peripheral physiological indicators, such as HR and SCL responses or BP, as well as in central nervous system indicators of EEG spectral activity. Despite of the small number in DE-related spectral EEG studies, findings from this field of research and related areas show that EEG beta activity seems to be a crucial marker associated with DE, other disinhibited consumption tendencies, and their respective antecedents (see Figure 3).

In NFB, it is essential to identify brain activity that is associated with the dysnfunctional target behaviors, their antecedents or relevant psychological states (Hammond, 2006; Vernon, 2005). These maladaptive patterns are then altered in the training process to attain desired changes. The vast majority of NFB applications targets specified spectral activity in the spontaneous EEG (Gruzelier, 2014; Ros, Baars, Lanius, & Vuilleumier, 2014). Previously introduced findings support the possible use of EEG beta activity as a correlate of tense arousal and target frequency range in a NFB protocol to treat DE. Especially during food cue-induced states of craving, an inhibition of EEG beta activity may constitute a promising approach to reduce arousal and in turn prevent motivational states of food craving and subsequent DE.

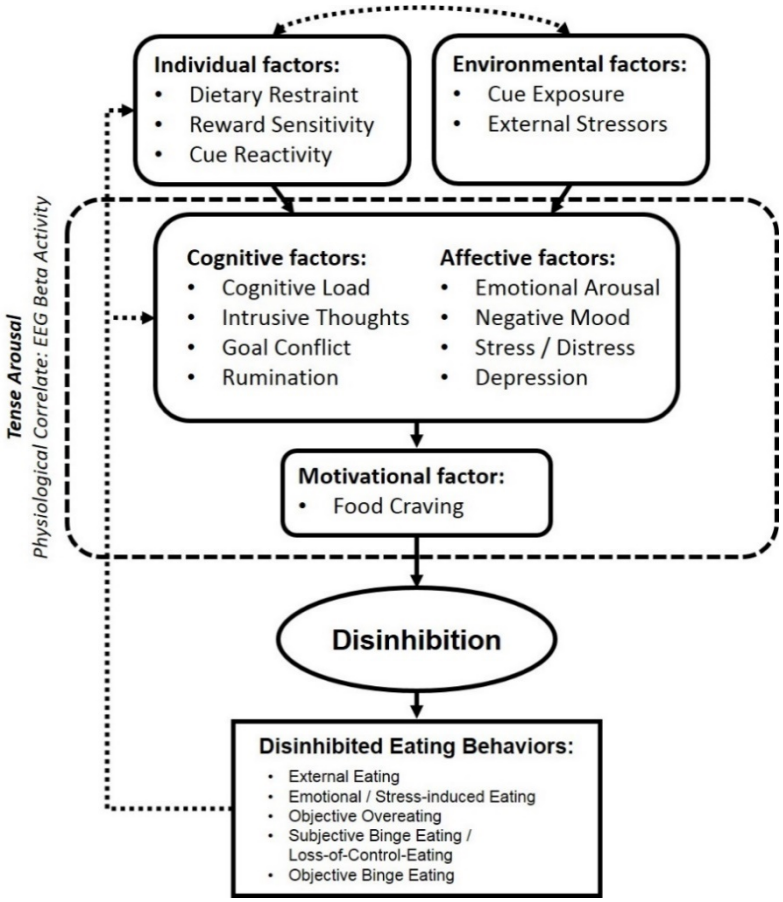

Figure 3. Synthesis-model of disinhibited eating behaviors incorporating relevant psychophysiological activity.

Table 3

Findings of studies examining peripheral psychophysiological responses related to disinhibited eating.

Study	Sample	Psychophysiology	Findings
Baseline & state-specific differences			
Tuschen-Caffier & Vögele (1999)	Female participants with BN (*n* = 27), female REs (FEV; *n* = 27) and female controls (*n* = 27)	Heart rate Blood pressure (SBP, DBP) Skin conductance	• Baseline HR: REs < BN & controls Interpersonal stressor (film): BP ↑, SCL ↑ • SCL responses: REs > BN & controls
Koo-Loeb, Costello, Light, & Girdler (2000)	Females with high (*n* = 26) or low (*n* = 27) bulimic symptomatology	Blood pressure Heart rate reactivity Urinary cortisol	• Physiological stress reactivity (Stress task): High BN symptoms > low BN symptoms (blood pressure ↑, Heart rate ↑, urinary cortisol ↑)
Friederich et al. (2006)	Obese women with BED (*n* = 38) and BMI-matched healthy controls (*n* = 34)	HRV HF (Parasympathetic cardiac regulation)	• Mental stress: HRV-HF in BED < HCs • HRV-HF ↓ → binge eating frequency ↑ & hunger (TFEQ) ↑
Messerli-Bürgy et al. (2010)	Patients with BN (*n* = 12) or BED (*n* = 13) and healthy controls (*n* = 13)	Heart rate HRV	• Stress reactivity (HR ↑): BN & BED > HC. • Less adaptive HRV in BN and BED • Limited HRV Recovery capacity in BN and BED
Vögele, Hilbert, & Tuschen-Caffier (2009)	Female patients with BN (*n* = 17) and female healthy controls (*n* =16)	Baseline HRV Mental stress HRV	• Baseline HRV: fasting BN < non-fasting BN & HC • Mental stress HRV n.s.
Meule, Vögele & Kübler (2012)	Female students (*n* = 47)	Baseline HRV	• Sympathetic dominance in restrained eaters: Restraint ↑ → Cardiac vagal control (LF/HF) ↓ (trend only)
Meule, Lutz, et al. (2012b)	Female students (*n* = 50)	Baseline HRV	• Self-reported dieting success ↑ → cardiac vagal control (LF/HF) ↑
Ginty, Phillips, Higgy, Heaney, & Carroll (2012)	Females with disordered eating behaviors (*n* = 12) and healthy controls (*n* = 12)	Blood pressure Heart rate Salivary cortisol	• Physiological stress reactivity (acute stress): Disordered eating < HC (HR ↓, stroke volume ↓, salivary cortisol ↓)

(cont.)

Table 3 (continued)

Findings of studies examining peripheral psychophysiological responses related to disinhibited eating.

Study	Sample	Psychophysiology	Findings
Klatzkin et al. (2015)	Female participants: obese BED (*n* = 9), obese non-BED (*n* = 15), normal weight controls (*n* = 15)	HRV: Baseline, Stress (Trier Social Stress Test) Blood Pressure (SBP, DBP)	• Stress: HR ↑, DBP ↑, SBP ↑ • Blood pressure: obese BED > obese non-BED & NW control (DBP, SBP) • BED: DBP ↑, SBP ↑ → subjective hunger ↑ Non-BED: Restraint ↑ → stress related HR ↑
Food cue exposure paradigms			
Overduin & Jansen (1996)	Healthy normal weight students (*n* = 21), fasting vs. non-fasting	Heart rate Skin conductance Salivation Respiration amplitude	• Arousal during food cue exposure: fasting < non-fasting (SC responses, HR [trend only])
Vögele & Florin (1997)	Women with (*n* = 30) and without (*n* = 30) regular binge eating	Heart rate Blood pressure (SBP, DBP) Skin conductance Respiratory activity	• Sympathetic arousal: binge eaters > non-binge eaters (SCL ↑, SBP ↑) • Heightened arousal during food cue exposure (both groups): HR ↑, BP ↑, SCL ↑, resp. ↑ • HR (during food cue exposure) ↑ → caloric intake in restrained binge eaters ↑
Overduin, Jansen, & Eilkes (1997)	Restrained / disinhibitive (*n* = 11) and unrestrained (*n* = 13) subjects	Heart rate Skin conductance Startle Reflex Facial EMG (Corr., Zygo.)	• Heightened arousal during food cue exposure (individually appealing foods, both groups): no detailed statistics provided • Startle reflex onset: food cues ≠ neutral cues, no detailed statistics provided • No group differences between restrained and unrestrained eaters except for EMG corr. (no detailed statistics provided)
Nederkoorn, Smulders, & Jansen (2000)	Healthy women, normal weight, non-dieting (*n* = 24)	Heart rate HRV (RSA, LF, VLF) Blood Pressure (SBP, DBP) Finger pulse volume Swallowing (EMG) Skin conductance / temperature	• Heightened arousal during food cue exposure: HR ↑, HRV-LF ↑, DBP ↑, SBP ↑, temp. ↑, SCL ↑, swallowing ↑, HRV-RSA ↓ • BP ↑ → food craving ↑ • Restraint (RS) ↑ → BP during food exposure ↑
Nederkoorn et al. (2004)	Women with eating disorders (*n* = 52) and non-dieting HCs (*n* = 20)	Heart rate Finger pulse amplitude Skin conductance	• Increased physiological arousal during food cue exposure: HR ↑, SCL ↑, FPA ↑ (both groups) • FPA ↑ during cue exposure → urges to eat ↓ & caloric intake ↓ (cont.)

Table 3 (continued)

Findings of studies examining peripheral psychophysiological responses related to disinhibited eating.

Study	Sample	Psychophysiology	Findings
Drobes et al. (2001)	Restrained eaters (*n* = 13), binge eaters (*n* = 19), deprivation subjects (*n* = 21), control subjects (*n* = 23)	Startle reflex Heart rate Skin conductance	• Startle reflex magnitude to food pictures: binge eating & deprivation > control & restrained eating • HR response to food cues: restrained & deprived eating > binge eating & control
Rodríguez et al. (2005)	Female students (*n* = 72), high or low food-cravers	Heart rate Skin conductance Startle reflex	• Cardiac defense response: chocolate cue < unpleasant cue • Startle reflex magnitude: High cravers: chocolate cue > pleasant cue • Skin conductance response: High cravers: chocolate > neutral Low cravers: chocolate < neutral
Mauler et al. (2006)	Women with BN (*n* = 32) and healthy controls (*n* = 32), deprived vs. non-deprived	Startle reflex Heart rate Skin conductance response Facial EMG (Corr.)	• Startle reflex magnitude: BN: food cues > neutral & pleasant cues Healthy controls: food cues = pleasant cues • EMG Corr.: BN: food cues > neutral & pleasant cues Healthy controls: food cues = pleasant cues • HR deceleration in response to food cues: deprived < non-deprived • SC responses in response to food cues: deprived < non-deprived
Svaldi et al. (2010)	Women with BED (*n* = 22) and overweight HCs (*n* = 22)	Electroencephalography Skin conductance Heart rate Facial EMG (Corr.) Finger pulse amplitude	• EEG ERPs: High caloric food BED > HC (LPP & SPW) • EMG Corr.: High caloric > low caloric food cues • PTT: High caloric food > low caloric food • IBI: high caloric pictures < low caloric pictures (trend only)
Udo et al. (2014)	Obese participants (*n* = 12) and normal weight (*n* = 14) participants	Heart rate variability (HF)	• HRV-HF during favorite food cue exposure: obese > non-obese • No differences in baseline or mood inductions
Geisler et al. (2016)	Healthy participants (*n* = 111)	Heart Rate Variability	• Ego depletion in REs → HRV ↑ (effortful self-regulation)

Note. BED = binge eating disorder; BMI = Body Mass Index; BN = bulimia nervosa; BP = Blood pressure; Corr. = Musculus corrugator supercilii; DBP = diastolic blood pressure; EEG = electroencephalogram; EMG = electromyogram; ERP = event-related potential; FEV = Fragebogen zum Essverhalten; FPA = finger pulse amplitude; HCs = healthy controls; HF = high frequency; HR = heart rate; HRV = heart rate variability; IBI = Inter-beat-interval; LF = low frequency; LPP = late positive potential; NW = normal weight; PTT = pulse transit time change; REs = restrained eaters; resp. = respiration amplitude; RS = Restraint Scale; RSA = respiratory sinus arrhythmia; SBP = systolic blood pressure; SC = skin conductance; SCL = skin conductance level; SPW = slow positive wave; temp. = skin temperature; TFEQ = Three-Factor Eating Questionnaire, Zygo. = Musculus zygomaticus major; ↑ = increase; ↓ = decrease.

Table 4

Findings of spectral EEG studies related to disinhibited eating.

Study	Sample	Psychophysiology	Findings
Eating related studies			
Silva et al. (2002)	Female students, REs (RS; *n* = 23) and unrestrained eaters (*n* = 32)	EEG Prefrontal alpha asymmetry (alpha activity [8-13 Hz] at F3 vs. F4), resting state	• Greater right-sided prefrontal asymmetry (avoidance tendency) in REs • Right-sided prefrontal asymmetry ↑ → Restraint score ↑
Ochner et al. (2009)	Overweight and obese adults (*n* =28)	EEG prefrontal asymmetry (alpha activity [8-13 Hz] at F3 vs. F4), resting state	• No association between Binge eating and prefrontal asymmetry • Positive association between left-sided prefrontal asymmetry (approach-tendency) and Disinhibition (TFEQ)
Tammela et al. (2010)	Obese women with BE (*n* = 12) and obese women without BE (*n* = 13)	Quantitative spectral EEG: Resting state, cue exposure Fronto-central derivations (F3-C3; F4-C4)	• Eyes closed: EEG beta (14-20 Hz) BE > non BE • Cue exposure: EEG beta (14-20 Hz) BE > non BE • No differences in alpha, delta or theta • BE subjects: Beta ↑ → BE symptoms ↑ & disinhibition ↑ • No hemispheric difference
Hume, Howells, Rauch et al. (2015)	Healthy women, normal weight (*n* = 41), overweight (*n* = 21), obese (*n* = 19)	EEG spectral activity Resting state, food cue exposure, office task (modified stroop tasks) (Fp1, Fp2, F3, F4, F7, F8, C3, C4, P3, & P4, linked to Cz)	• Relative beta power (15-30 Hz) food cue exposure: overweight > normal-weight (right frontal, left central) • Relative beta power (15-30 Hz) food cue exposure ↑ → percentage body fat ↑
Hume, Howells, Karpul et al. (2015)	Women: reduced weight (*n* = 14), low weight controls (*n* = 18), weight relapsed (*n* = 10), high weight controls (*n* = 9)	EEG spectral activity Stroop tasks: neutral vs. food	• Right frontal relative delta power (F4) Reduced weight < low weight controls • Right frontal relative beta power (F4) Reduced weight > low weight controls

Note. BE = binge eating; EEG = electroencephalogram; Hz = hertz; REs = restrained eaters; RS = Restraint Scale; TFEQ = Three-Factor Eating Questionnaire, ↑ = increase; ↓ = decrease.

Table 5

Findings of spectral EEG studies examining constructs and antecedents related to disinhibited eating.

Study	Sample	Psychophysiology	Findings
Addiction-related studies			
Liu et al. (1998)	Male cocaine abusers (*n* = 17) and control subjects (*n* = 5)	EEG spectral activity Baseline, during cocaine and neutral cues (C3, C4, P3, P4, Cz, and Pz)	• Cocaine users in cocaine cue exposure: EEG arousal (desynchronization) ↑ (diffuse localization) • Cocaine users: alpha activity (8-13 Hz) cocaine cues < neutral cues
Bauer (2001)	Patients from substance abuse treatment programs (*n* = 107) and healthy controls (*n* = 22)	Quantitative EEG resting state activity (eyes-closed)	• Fast EEG beta activity (19.5 – 39.8 Hz) predicted relapse at a 6-month follow-up
Reid et al. (2003)	Cocaine dependent participants (*n* = 24)	Quantitative EEG during Video cue exposure (eyes open) and scripted imagery (eyes closed)	• Video cue exposure: EEG beta ↑ (frontal, occipital), EEG delta ↓ (frontal) • Scripted imagery: EEG beta ↑ (occipital), EEG theta ↑ (frontal), EEG delta ↑ (frontal)
Saletu-Zyhlarz et al. (2004)	Detoxified former alcohol abusers (*n* = 22) and matched healthy controls (*n* = 22)	EEG spectral activity Resting state, vigilance	• Baseline EEG: EEG Beta power: Former alcohol abusers > HC EEG Alpha power: Former alcohol abusers < HC • 6-months follow-up EEG Beta power ↓, EEG delta power ↑ • Baseline EEG high beta power: Relapse > HC & abstinent patients Baseline EEG alpha power: Relapse < abstinent patients
Knott, Cosgrove et al. (2008)	Smokers (*n* = 20)	EEG spectral activity during scripted imagery	• EEG beta (13.75-29.75 Hz) urge scripts > non-urge scripts • EEG delta activity (0.75-3.75 Hz) urge < non-urge scripts (males only)
Knott, Naccache et al. (2008)	Smokers (*n* = 22)	Frontal EEG spectral activity cue exposure and mood induction	• High beta activity (20.75-31 Hz) during cigarette cue exposure ↑ (females only) • Left frontal alpha activity (7.75-13.75) during cigarette cue exposure ↑ (females only)
Littel, Franken, & Van Strien (2009)	Smokers (*n* = 22) and ex-smokers (*n* = 21)	EEG spectral activity during cue exposure (neutral vs. cigarette cue)	• Smokers: EEG beta activity (13.75-29.75 Hz) cigarette cue > neutral cue • EEG beta activity (cigarette – neutral cue) smokers > ex-smokers • No significant differences for EEG theta alpha or prefrontal asymmetry (cont,)

Table 5 (continued)

Findings of spectral EEG studies examining constructs and antecedents related to disinhibited eating.

Study	Sample	Psychophysiology	Findings
Antecedents of disinhibition			
Carter, Johnson, & Borkovec (1986) *worry*	Undergraduate students (*n* = 40)	EEG (F3, F4, P3, P4) in baseline, worry, cognitive task, relaxation	EEG low beta (13-23 Hz) and high beta (24-30 Hz): • Trait worriers > trait non-worriers • Experimental worry > cognitive task (bilateral)
Andersen et al. (2009) *rumination*	Healthy students (*n* = 63)	EEG in baseline (eyes open, eyes closed), nominal and personal rumination, count condition	• EEG beta (20-30 Hz) ↑ in nominal rumination > personal rumination & count condition (scalp wide)
Seo & Lee (2010) *stress*	Healthy participants (*n* = 33)	EEG (FC5, FC6, O1,O2, linked to Cz) HRV, salivary cortisol Resting state, positive and negative images, stress vs. no stress	EEG high beta activity is related to stress: • Relative EEG high beta power: stress > no stress • Relative EEG high beta power ↑ → HRV (SDNN) ↓ • Relative EEG high beta power ↑ → salivary cortisol ↑
Hayashi et al. (2009) *stress*	Healthy graduate students (*n* = 22)	EEG (F3, F4, T3, T4) in relaxed, pleasant, unpleasant mood conditions	• Relative EEG beta power (14-30 Hz) ↑ in unpleasant mood condition • Beta: Stress group > non stress group, Beta ↑ → negative affect ↑
Jena (2015) *stress*	Medical students: mild stress (*n* = 18), moderate stress (*n* = 20), high stress (*n* = 15)	EEG recordings during baseline and examination stress: peak frequency	EEG peak frequency is related to stress levels: • Baseline EEG peak frequency: High stress group (20.5 Hz) > low & moderate stress groups (9-10 Hz) • Examination stress EEG peak frequency (22-25 Hz) > baseline (9-10 Hz) in low and moderate stress groups
Hall et al. (2000) *stress-related intrusions*	Adults with primary insomnia (*n* = 14)	EEG (C3 or C4) during laboratory sleep studies	Significant relations of EEG parameters (sleep) with intrusion and stress • EEG beta power (17-32 Hz) ↑ → stress-related intrusion tendencies ↑ • EEG delta power (0.5-4 Hz) ↓ → subjective stress burden ↑ (cont.)

Table 5 (continued)

Findings of spectral EEG studies examining constructs and antecedents related to disinhibited eating.

Study	Sample	Psychophysiology	Findings
Valck, Cluydts & Pirrera (2004) *cognitive arousal*	Healthy adults (*n* = 12)	EEG recordings during sleep latency tests Heart rate, HRV	• EEG high beta power (20-35 Hz): induced cognitive arousal > neutral condition • EEG theta (4-8 Hz): induced cognitive arousal < neutral condition
Sokhadze (2007) *negative affect*	Healthy undergraduate students (*n* = 29)	EEG (F3, F4, T3, T4, O1, O2) in response to emotional cues and music	• Aversive Emotional cues: EEG slow alpha (8-9.99 Hz) ↓, EEG high beta (20-30 Hz) ↑ • Pleasant and sad music (music was associated with subjective and heart rate recovery from stress): EEG slow alpha (8-9.99 Hz) ↑ (right only), EEG high beta (20-30 Hz) ↓ (bilateral)
HajiHosseini & Holroyd (2015) *reward feedback*	Undergraduate students (*n* = 26)	EEG (F5, FZ, F6, C5, CZ, C6, P5, PZ, P6) during a T-maze task + reinforcement cues	• Reward signaling cues: Frontal EEG high beta activity (20-30 Hz) ↑

Note. EEG = electroencephalogram; HRV = heart rate variability; Hz = hertz; SDNN = standard deviation of normal to normal R-R intervals; ↑ = increase; ↓ = decrease.

2.4 Psychological treatments for disinhibited eating

Different treatment approaches have been evaluated for manifest eating disorders associated with binge eating. Existing treatment rationales range from cognitive behavioral interventions, over interpersonal therapy and dialectic behavioral therapy to behavioral weight loss treatments and medication (for reviews, see Hay, 2013; Iacovino et al., 2012; Reas & Grilo, 2008). Cognitive-behavioral interventions have been advocated as first-line treatment options for BED (for a meta-analysis, see: Vocks et al., 2010). However, researchers have recently pointed out that treatments for eating disorders need further improvements (Brownley et al. 2007; Wilson et al., 2007).

Therefore, treatment-enhancements by means of brain-directed treatments or treatment adjuncts continuously attract more notice (Schmidt & Campbell, 2013). As stated by Iacovino and colleagues, "psychological treatments for BED may produce even more robust outcomes when including . . . behavioral therapies that specifically target brain reward system abnormalities associated with reinforcement pathology" (2012, p. 442). Here, *neuromodulation* techniques are considered as useful adjuncts and have so far shown success in the regulation of diverse dysregulated eating behaviors (McClelland, Bozhilova, Campbell, & Schmidt, 2013; Val-Laillet et al., 2015). Although these studies focus on passive and external neuromodulation techniques (e.g., transcranial magnetic or direct current stimulation, or invasive deep brain and vagus nerve stimulation), neuromodulation can essentially be achieved by safer and less invasive techniques, like NFB (Brunoni et al., 2011; Thibault, Lifshitz, Birbaumer, & Raz, 2015).

However, given the lack of NFB studies in eating behavior research, some ideas and findings from other related and useful behavior modification approaches can inform the development of a NFB protocol that is suitable to treat DE behaviors. Therefore, this chapter will briefly introduce some psychological treatments that have implications for the development of the NFB protocol based on the synthesis model.

2.4.1 Cue exposure treatments

Cue exposure treatments are classical behavior therapeutic approaches that rely on principles of basic learning theory, especially the conditioned association of cues with dysfunctional behaviors (Conklin & Tiffany, 2002). These treatments base on the assumption that repeated exposure with a salient cue (e.g., a cigarette for a smoker) followed by response prevention, can lead to an extinction of conditioned associations between cue and behavior.

Subsequently, the undesired (consumption) behavior can be reduced (F. A. Carter & Bulik, 1994; Conklin & Tiffany, 2002, Wardle, 1990). Cue exposure is a successful application in the treatment of various addictions and eating disorders (Conklin & Tiffany, 2002; Loeber, Croissant, Heinz, Mann, & Flor, 2006; McIntosh, Carter, Bulik, Frampton, & Joyce, 2011).

In the 1980's, Schmidt and Marks (1988), as well as the research group around Anita Jansen (Jansen et al., 1989), started using cue exposure treatments to treat patients with BN. Jansen, Broekmate, and Heymans (1992) extended the therapeutic approach to the treatment of BED, with success rates being three times larger than in a self-control-based treatment without cue exposure. By now, cue exposure treatments have become a classical and successful behavior therapeutic technique in the treatment of eating disorders (e.g., Bulik, Sullivan, Carter, McIntosh, & Joyce, 1998; McIntosh et al., 2011, Toro et al., 2003) and are also advocated for treatments of subclinical DE behaviors (F. A. Carter & Jansen, 2012; Jansen, Schyns, Bongers, & van den Akker, 2016).

Given the calls to consider reinforcement pathologies in the brain-directed treatment of dysfunctional eating behaviors (Iacovino et al., 2012), cue exposure posits an important component that should be considered in NFB protocols for DE. Besides the basic extinction processes (Jansen et al., 2016), food cues can reliably serve as a means to induce craving in a treatment setting (R. G. Boswell & Kober, 2016) and thus model the self-control demands in the obesogenic environment (Swinburn et al., 2011). Therefore, cue exposure treatments exhibit high external validity. In result, cue exposure is probably especially efficacious in preventing relapse after the treatment periods (F. A. Carter & Jansen, 2012; Havermans, & Jansen, 2003).

2.4.2 Stress-reduction approaches

As affective factors and stress play a crucial role as antecedents of DE (cf. 2.2.4), it is not surprising that clinical researchers tested various stress-reduction approaches to regulate patterns in dysfunctional eating. Ong, Linden, and Young (2004) found that a range of interventions is regularly applied for stress-reduction purposes. Among them are palliative techniques like meditation, relaxation, and mindfulness-based practices, as well as imagery treatments or cognitive strategies for stress-reduction (e.g., emotion-regulation trainings, reappraisal trainings, and self-monitoring).

Stress-reduction approaches in fact dominate the area of treatments for subclinical eating dysregulations. Two systematic reviews found mindfulness-based treatments to be effective in the reduction of binge eating and other dysfunctional eating behaviors associated with

overweight and obesity, like emotional eating and external eating (Katterman, Kleinmann, Hood, Nackers, & Corsica, 2014; O'Reilly, Cook, Spruijt-Metz, & Black, 2014). Katzer and colleagues (2008) found a relaxation-based treatment to be superior to programs covering eating and activity advice with regard to an enhancement of self-efficacy and healthy eating. Interventions that increase emotion-regulation skills were successful to achieve and maintain binge eating abstinence in women with BED (Clyne, Latner, Gleaves, & Blampied, 2010). Some studies even found beneficial effects of stress-reduction methods on weight loss (Christaki et al., 2013; Daubenmier et al., 2011). However, there are also studies that did not find any beneficial effects of mindfulness-based stress reduction on eating behavior (Kearney et al., 2012).

Cognitive approaches have been successfully applied to reduce food craving: For example, trainings that focus on long term positive consequences of not giving in to food cravings (reappraisal trainings) successfully modulated food cravings and associated brain activity (Yokum & Stice, 2013). Further, mental imagery treatments (Pearson, Deeprose, Wallace-Hadrill, Heyes, & Holmes, 2013) have been applied frequently with respect to dysfunctional eating behaviors and were considered a successful adjunct in the treatment of eating disorders (Espen et al., 1998; Tatham, 2011). Some recent mental imagery techniques do theoretically base on EI-Theory and focus the elimination of intrusive food imagery in food cravings to replace them with alternative imagery based on visuospatial interference (Andrade, May, & Kavanagh, 2012; Steel, Kemps, & Tiggemann, 2006). The use of these mental imagery techniques led to a reduced intensity of food cravings in several intervention studies (Hamilton, Fawson, May, Andrade, & Kavanagh, 2013; Kemps & Tiggeman, 2007; Knäuper, Pillay, LaCaille, McCollam & Kelso, 2010).

In the light of these promising results, mindfulness, relaxation, or imagery instructions can constitute strategies that may be applied in NFB. Some researchers did previously successfully combine relaxation and mindfulness trainings with new technologies, such as virtual reality (Manzoni et al., 2008). By suggesting these strategies, or even a global relaxation instruction, participants receive several options to try out during the NFB treatment for DE. They will then be able to identify adequate strategies for the reduction of physiological arousal using the NFB equipment (Siniatchkin, Kropp, & Gerber, 2000).

2.4.3 Biofeedback treatments

Biofeedback is a therapeutic method based on basic learning principles and applied psychophysiology. It uses an online assessment of physiological activity regarding different parameters (e.g., EMG, SCL, BP, HRV, or EEG) to analyze and communicate relevant activity to a patient. Based on objective feedback on real-time dysfunctional physiological activity, the patient is then able to gather control over these bodily processes, influence them in a beneficial way and via individual control strategies (Schwartz & Schwartz, 2003; for further information on treatment mechanisms, see 2.5.1). Using Biofeedback, it may be possible to objectively alter physiological correlates of DE and its antecedents, especially with respect to the strong and dysfunctional arousal component manifesting in several antecedents (cf. 2.3).

Still, biofeedback research has to date hardly addressed dysfunctional eating behaviors (Korn & Niepoth, 2009). In an early study on two female pre-adolescent samples, obese girls and girls with anorexia nervosa, Pop-Jordanova (2000) added electrodermal biofeedback to a multicomponent-treatment (e.g., dietary changes, behavior therapy, relaxation) to mitigate eating disorder symptoms. Although the author reports that biofeedback was successful to reduce eating disorder symptoms, the results only show decreases in electrodermal responses. No changes in eating symptomatology were reported in either of the two samples. The biofeedback component was poorly described and constituted one therapeutic module among many others. Due to the design, lack of any control group, and omission of pre-post comparisons for eating-related variables, the contribution of biofeedback cannot be inferred from this study.

Over a decade later, Meule and colleagues (2012) applied a HRV biofeedback protocol in a female student sample of high food-cravers. The researchers delivered twelve HRV biofeedback sessions and compared the effects to two non-treatment control groups with either high or low food cravings. The authors reported preliminary evidence for the efficacy of this treatment to reduce food craving. At post-treatment, participants in HRV biofeedback reported significantly less trait food craving with a large within-group effect size. Further, HRV biofeedback exerted beneficial effects with regard to a reduction in weight- and shape-related concerns, but only with small effect sizes. There was no influence of HRV biofeedback on the assessed DE behaviors (overeating, subjective and objective binge eating) and no changes in HRV were observed. The authors subsumed that the training affected cognitive and attitudinal factors rather than actual behavior (Meule, Freund, Skirde, Vögele, & Kübler, 2012).

In another study, Teufel et al. (2013) examined the effects of an eight session electrodermal biofeedback to reduce stress-related eating behaviors in obese women. Subjects participated in either electrodermal biofeedback with a mere relaxation-setup or with a setup that incorporated food cue exposure. Both treatments had positive effects of eating-related self-efficacy regarding challenging food. Only the cue-exposure setup enhanced eating-related self-efficacy with respect to high fat foods. Changes in electrodermal activity arose in the cue-exposure condition only, while the relaxation-based intervention increased abilities to relax after the training. The researchers did not report any outcomes related to eating behavior. Apart from effect sizes, the study further omitted reports on the statistical significance of the observed treatment effects. Thus, it is difficult to judge the relevance of achieved outcomes.

In a very brief two-session NFB-study, using real-time functional magnetic resonance imaging (rt-fMRI), Frank et al. (2012) found enhanced physiological self-regulatory abilities in obese participants after rt-fMRI NFB for a regulation of insular brain activity. There were no significant changes in psychological variables (e.g., positive and negative affect). There was no assessment of eating behavior. The study only included two sessions and was conducted in a small sample with limited statistical power. Both aspects may have led to the small effects on psychological variables. Nevertheless, the researchers stated that "The ability to intervene directly on the brain by voluntarily regulation of eating-related regions could be used to establish a tool to increase the control of such brain regions and affect eating-related behavior." (Frank et al., 2012, p. 5).

With regard to other possible NFB-treatments in manifest eating disorders, Bartholdy and colleagues (2013) provided a thorough review on previous research attempts regarding NFB in eating behavior. They pointed out the lack in interventions studies, as well as in basic EEG-research, and made some suggestions to transfer protocols from more extensively studied disorders to eating behavior (e.g., NFB in ADHD and substance use disorders: for reviews on NFB in these disorders, see Arns et al., 2009; Sokhadze, Cannon, & Trudeau, 2008). In summary, the authors concluded that further research is necessary to analyze the potential of NFB in the treatment of eating disorders.

Given the aforementioned insights on the concept of DE, its etiology, psychophysiological correlates, and related intervention approaches, it becomes evident that there is a potential to develop a NFB protocol for the treatment of DE behaviors. Several researchers have pointed out that brain-directed neuromodulatory treatments may serve as promising adjuncts in the

treatment of eating disorders (Bartholdy et al., 2013; Iacovino et al., 2012; Schmidt & Campbell, 2013). With regard to NFB, Frank Duffy even stated that it "should play a major therapeutic role in many difficult areas . . . if any medication had demonstrated such a wide spectrum of efficacy it would be universally accepted and widely used" (Duffy, 2000, as cited by Hammond, 2006, p. 27).

Still, in intervention research for dysfunctional eating behaviors there are no studies using NFB, despite of its properties as a well-established, affordable, and safe technique for neuromodulatory brain-directed treatments that may enrich clinical practice in the treatment of DE (Myers & Young, 2012; Niv, 2013). Addressing this gap in the research on eating behavior and psychological interventions is therefore a main goal of this dissertation project.

2.5 Development of a neurofeedback intervention to reduce disinhibited eating

The following chapter will provide an overview on the theoretical and practical considerations within the development of the NFB protocol for DE. First, proposed NFB mechanisms and the general treatment rationale are introduced. Then, the distinct setup, selected NFB protocol, and target group will be explained. Finally, the resulting NFB protocol is presented in detail, followed by a description of the studies evaluating this protocol as a core of this dissertation (cf. 3.).

2.5.1 General treatment rationale

NFB is a treatment method which bases on the premise that dysfunctional psychological states or behaviors co-occur with certain physiological patterns in brain activity (Abarbanel, 1995; Demos, 2005). As in other biofeedback-treatments, physiological activity of a patient is assessed via psychophysiological measures on electrical, hemodynamic, or other quantifiable bodily responses. In NFB, these responses comprise brain activity.

Special software algorithms conduct an online-analysis of the acquired physiological activity according to predefined settings and provide the patient with real-time visual and/or auditory feedback (Hammond, 2006). Thus, the patient's brain activity is made visible to him or her so that it becomes possible to observe objective changes initiated by means of altered psychological processes, like relaxation or enhanced attention (Masterpasqua & Healey, 2003). Additionally, a feedback is provided to reward functional physiological responses and/or inhibit unwanted responses. The patient then learns to establish psychological states related to functional brain-activity, so that these states will occur more frequently.

The underlying treatment mechanisms can be understood in terms of basic learning theory (Sherlin et al., 2011): Activity in the desired ranges is rewarded by means of animations, success scores, or bar diagrams. This feedback serves as a positive reinforcement. In line with the assumptions of operant conditioning theory, individual strategies that induce the desired physiological states associated with positive feedback will be established through this reinforcement schedule (Siniatchkin et al., 2000; Strehl, 2014).

Further, classical conditioning processes may occur in NFB. Dating back as early as in the 1940s, Jasper and Shagass (1941) observed how the EEG alpha blockade, a pattern which usually occurs in response to opening one's eyes, can be conditioned to external stimuli. Repeated pairing of certain provoked states (such as craving) and improved brain activity patterns (e.g., decreased arousal) could therefore lead to classical conditioning and enduring physiological changes, even in the absence of conscious efforts to regulate brain activity (Sherlin et al., 2011). Birbaumer, Ruiz, and Sitaram (2013) proposed that self-regulatory competence with regard to brain activity would resemble motor learning and occurs in subcortical areas of the basal ganglia. Therefore, conscious strategies would not be necessary for successful brain activity regulation, as shaping processes occur implicitly and on an idiosyncratic level.

Based on these learning mechanisms, NFB is supposedly able to restore dysfunctional physiological states based on physiological learning (Sherlin et al., 2011). Associated psychological states and behaviors should subsequently be regulated (Vernon, 2005). This view on treatment mechanisms is derived from a standpoint regarding *neuroplasticity* (e.g., Lövdén, Bäckman, Lindenberger, Schaefer, & Schmiedek, 2010; Niv, 2013, Ros et al., 2014), meaning that neurophysiological learning can indeed occur through a strengthening of synaptic connections by repeated neuronal activity as enforced in NFB (Niv, 2013, p. 683). Learned self-regulation in a frequency-modulation NFB-approach can thereby not only alter psychological states; it can also reflect on other physiological reactions which are not distinctly targeted in NFB, for example event-related potentials in the EEG (Egner & Gruzelier, 2001) or subcortical metabolic processes related to self-control (Critchley, Melmed, Featherstone, Mathias, & Dolan, 2001; Ninaus et al., 2013).

Another important mechanism considered in the general biofeedback approach is the subjective experience of control over one's own physiological processes which is supposed to enhance self-efficacy (Holroyd et al., 1998; Rokicki et al., 1997; Wickramasekera, 1999). This

process is often described in terms of *agency*, giving the individual a sense of control over bodily processes that have formerly been perceived as uncontrollable (Brenninkmeijer, 2013; Glannon, 2014). In consequence, the subjective concept on the dysfunctional behavior may change. A patient might no longer perceive himself or herself as being subject to autonomous bodily reactions, but regains the power to volitionally influence bodily processes. With the help of NFB, a patient will learn how to translate intentions into manifest physiological changes; in result, a sense of autonomy and control can be restored (Glannon, 2014). The patient will further be able to overcome a dysfunctional dualistic view on bio-psychological mechanisms, for example that bodily responses (e.g., food craving and DE) are out of one's range of influence.

Biofeedback (and NFB) applications can further enhance an individual's interoceptive abilities due to the continuous confrontation with and concentration on bodily responses (Miller, 1978; Schwartz & Schwartz, 2003). Hence, a patient can become more sensitive to changes in bodily states and in consequence will be more likely to intervene at early antecedent stages that signal possible dangers of showing undesired behaviors (Bagdasaryan, & Le Van Quyen, 2013; Kotchoubey, Kübler, Strehl, Flor, & Birbaumer, 2002).

All of these mechanisms are relevant in the treatment of DE. With EEG beta activity, a supposed distinct electrophysiological correlate of DE is present that could be altered with NFB. With regard to physiological changes, it can be derived that operant and classical conditioning in a NFB protocol, aimed at a reduction of EEG beta activity, should reduce states of high arousal. As a consequence, perceived stress and tension should be diminished, so that the motivational food craving component receives "less fuel" to manifest in behavioral disinhibition and subsequent DE.

Operant conditioning will provide the individual with personally functional strategies to apply whenever food craving is likely to occur, for example in the presence of appealing food cues. Through the feedback provided by NFB, successful strategies for EEG beta regulation will be reinforced, making them more likely to occur. Giving the patient feedback on which strategy will individually work for him or her can subsequently provide directions for strategies that will be successful even without technical feedback. Thus, patients can use the NFB sessions to identify self-regulation strategies that are transferable to everyday situations, for example, in cases of everyday stress situations or tempting food cues.

Classical conditioning of brain activity through NFB may even result in a lower occurrence of any tension (and EEG beta activity) in the presence of appealing food cues, independent of

an individual's conscious attempts to alter his or her brain activity. Established connections between cues and physiological arousal, as well as arousal and disinhibition, can be subject to extinction processes (Jansen et al., 2016) when EEG beta is repeatedly down-regulated in NFB sessions.

Self-efficacy is generally known to be a very important factor in a wide range of health-related behaviors and successful behavior change (Bandura, 1977; O'Leary, 1985; Strecher, DeVellis, Becker, & Rosenstock, 1986). With regard to eating behavior it has been identified as a crucial predictor of long term success in reduction of or even abstinence from binge eating (Glasofer et al., 2013; Goodrick et al., 1999; Wolff & Clark, 2001) and subsequent weight management (Linde et al., 2004). By learning strategies for arousal-regulation using NFB, participants should further experience higher self-efficacy in regulating aversive affective states and thus be less dependent on eating as a means of emotion regulation or coping in stressful situations. Thus, enhancing self-efficacy is another beneficial mechanism that may contribute to success of NFB.

Finally, enhancing interoceptive abilities is important in the light of early theories on eating behavior. These theories postulated that impaired interoceptive abilities and misinterpretations of emotional arousal as states of hunger could lead to overeating (e.g., Kaplan & Kaplan, 1957). Further, REs supposedly unlearn the ability to detect physiological needs due to ongoing cognitive regulation of eating (Herman & Polivy, 1984). The focus on internal states and the associated *interoceptive exposure* (J. F. Boswell, Anderson, & Anderson, 2015) with regard to craving states in NFB may therefore contribute to increased awareness of factual physiological needs. This could help in reestablishing skills to detect bodily responses or physiological arousal, and differentiate them from a need to consume food.

In addition, Ninaus et al (2013) found that the mere intention to control one's brain activity in a sham-NFB condition, compared to passive watching of bar diagrams (both assessed with simultaneous fMRI measurement), activated several subcortical areas, for example, the bilateral insular cortex, anterior cingulate cortex, and dorsolateral prefrontal cortex. Regulation of these distinct areas has been identified as important to alter impaired interoceptive abilities, craving, approach motivation, and disinhibition (Hanlon et al., 2013; Shackman et al., 2011). Thus, interoceptive learning and its potential neuroplasticity-effects may constitute another important mechanism in NFB for DE.

Summarizing these mechanisms, it can be assumed that learned reduction of electrophysiological correlates of tense arousal (as elicited by food cues and food craving, stress or negative affect) by means of NFB should exert beneficial effects to restore self-regulatory capabilities in individuals with recurrent DE behaviors. The various physiological and psychological learning processes would contribute to reductions in experienced disinhibition and thus diminish the frequency of DE.

2.5.2 Neurofeedback setup

For the general setup of the NFB treatment, several peripheral as well as protocol-related issues were considered and pondered.

Given the prerequisite of a female target group (cf. 2.5.5), only female trainers provided the NFB. On the one hand, this choice should account for a better identification and a personal connection between participants and trainers. On the other hand, previous research has shown that gender differences among researchers and participants might influence outcomes in the light of *demand characteristics* within the sample (Nichols & Maner, 2008). Given the nature of self-reported outcomes, choosing only female trainers would account for undistorted outcomes because participants would less likely be subject to social desirability.

The number of treatment sessions was determined due to a literature research on comparable NFB and biofeedback protocols in non-clinical samples. Here, Vernon (2005) reported average treatment durations of ten sessions in his review on NFB for performance improvement. Further, the two most comparable biofeedback studies on eating behavior applied twelve (Meule, Freund et al., 2012) and eight (Teufel et al., 2013) biofeedback sessions. Given the target sample of the first evaluation studies (cf. 2.5.5), a ten session protocol was considered as adequate in accordance with previous research.

With closer regard to the protocol, feedback modalities were kept relatively simple, using bar diagrams for the targeted EEG high beta activity as well as for muscular artifacts on the client screen. Only a calm animation of a beach landscape at sunset accompanied the feedback. The animation was presented fluently in case of successful regulation and was interrupted when any bar exceeded the preset threshold. Additionally, a success count was displayed, adding one point for every second of successful regulation. This minimalistic feedback was previously advocated to guarantee concentration on the learning processes and avoid distractions in fancy feedback settings (Sherlin et al., 2011).

Muscular artifacts were detected with a software algorithm. It calculates excess power in higher frequency ranges because fast EEG beta activity may overlap with higher power EMG activity (Muthukumaraswamy, 2013). All trainers should therefore explain this relationship to participants and instruct them to sit calm, avoid unnecessary movements, and relax all facial muscles so that they would not mistake artifacts for EEG high beta activity. The use of a comfortable chair should further contribute to a calm seating position during sessions and thus prevent artifacts. Throughout the course of each session, trainers should additionally monitor the EEG raw signal. Minor and major artifacts would be logged in a paper-pencil protocol.

For multi-session protocols, NFB researchers suggest a task-difficulty adjustment to allow for behavioral shaping processes to occur (Enriquez-Geppert, Huster, & Herrmann, 2013; Sherlin et al., 2011). Therefore, the use of stepwise adjustments of success rates was applied to maintain a challenging nature of the NFB over the whole treatment.

Some critiques on NFB perceive this treatment as equipment-dependent, meaning that patients would become dependent on technical gadgets to successfully regulate behavior. To prevent this process, it is important to include transfer instructions into the protocol (Strehl, 2014). The focus of the first four sessions would be on the detection and establishment of strategies to regulate EEG activity. In session 5, participants would then receive a small trigger card. This card displays the client monitor under successful regulation circumstances. Using the trigger card, participants shall practice the individual strategies identified by NFB in different challenging situations of their everyday life. This should account for successful, equipment-independent transfer processes.

2.5.3 Selection of target brain activity

In chapter 2.3, tense physiological arousal indicated by fast EEG beta activity has been presented as a potential EEG spectral range that may be closely connected to craving and subsequent relapse (Parvaz et al., 2011), eating related disinhibition, and binge eating (Tammela et al., 2010). It is further associated with antecedents of DE, such as stress, ruminative thoughts, and negative affect. While the EEG beta spectrum is relatively broad, ranging from 13 to 30 Hz, the aforementioned states have predominantly been associated with faster frequency ranges above 16 Hz (Parvaz et al., 2011).

Further, traditional NFB research connects slower activity ranges of the beta spectrum to functional cognitive processes, like wakeful attention, concentration, and general cognitive performance (Egner & Gruzelier, 2001; Gruzelier, 2014; Thompson & Thompson, 2007).

Lower beta is therefore rewarded and up-regulated in NFB protocols that aim at influencing these mental states (e.g., Egner, Zech, & Gruzelier, 2004; Vernon et al., 2003).

On the contrary, especially the EEG high beta activity, ranging from approximately 23 to 28 Hz, has been linked to dysfunctional hyperarousal (Hammond, 2006; Rangaswamy et al., 2002), anxious rumination (Anderson et al., 2009; Thompson & Thompson, 2007), stress (S.-H. Seo & Lee, 2010), and compulsions (Pogarell et al., 2009). It further constitutes a spectral range that is frequently inhibited for control purposes in various NFB protocols for the treatment of ADHD (Butnik, 2005) and enhancement of performance (Ros et al., 2009; Gruzelier, 2014). Two recent studies explicitly targeted EEG high beta down-regulation in the treatment of migraines (Walker, 2011) and addiction (Keith, Rapgay, Theodore, Schwartz, & Ross, 2015). Thus, a down-regulation of EEG high beta activity (23-28 Hz) constitutes a reasonable and safe procedure for a NFB protocol to reduce hyperarousal in relation to DE.

As a recording and training site, the vertex position (Cz, according to the 10-20 system by Jasper, 1958) with a unipolar measurement was seen as appropriate for a novel protocol. This position was chosen due to recommendations by Demos (2005), as it is likely to assess activity derived from the cingulate cortex, which is seen as “an important aspect of dysregulation in substance abuse and . . . may be likely to accompany preparatory approach tendencies” (p. 74). The author further explicitly recommends the choice of a unipolar protocol on the vertex position to assess absolute power and not the relative power in comparison to a different electrode position in a bipolar setup. Another argument can be seen in the safety of this measurement setup, as a regulation of unilateral or frontal beta activity might influence EEG asymmetry (Demos, 2005; p. 120), which is linked to depression (Henriques & Davidson, 1991).

2.5.4 Inclusion of cue exposure

Given the aforementioned association of the targeted EEG high beta activity with antecedents of DE, it is crucial for the NFB protocol to first elicit the distinct state of physiological hyperarousal in the sessions. Participants would have to attend several scheduled sessions and one cannot expect that naturally occurring food cravings would be present at each training session. Thus, exposure with food cues was included as a core component of the treatment, as it has previously been found to provoke craving and associated EEG patterns in eating and other dysregulated consumption behaviors (R. G. Boswell & Kober, 2016; Parvaz et al. 2011; Sobik, Hutchison, & Craighead, 2005; Tammela et al., 2010).

Another argument for the cue exposure protocol lies in the previous positive effect of repeated cue exposure with response prevention in the treatment of binge eating (Jansen et al., 1992; McIntosh et al. 2011, Toro et al., 2003). Using this technique, individual food cue-reactivity, as a strong risk factor for binge eating (Nederkoorn & Jansen, 2002; Tetley et al. 2009), can be reduced. Further, self-control, in the light of challenging situations, can be enhanced (Marlatt, 1990; Nederkoorn et al. 2000). This would account for higher external validity and proper adjustment to environmental demands. In addition, previous intervention trials (Jansen, 1992; McIntosh et al., 2011; Teufel et al., 2013) have shown superiority of cue exposure setups in comparison to mere relaxation or self-control treatments. Because of the aforementioned assumptions and findings, cue exposure was incorporated in the present NFB protocol.

With regard to the sensory modality of the cue, visual food cues (in contrast to auditory or even olfactory food cues) have shown the strongest influence on participants' food craving in earlier research (e.g., R. G. Boswell & Kober, 2016; Tiggemann & Kemps, 2005). Furthermore, personalized food cues that depict individual binge foods are more salient and have the highest potential to induce craving and associated brain activity (Jastreboff et al., 2013; Loxton, Dawe, & Cahill, 2011). With regard to the number of exposures, Van Gucht et al. (2008) found ten consecutive exposures with chocolate cues to be more effective in reducing chocolate craving between sessions than only two exposures. Before the treatment, each participant would therefore be asked to provide a list of the ten most craved food items that would then be presented as photographic food cues during the sessions.

In addition to pictorial presentation, instructions are given to imagine the cued foods as vividly as possible, in line with comparable cue exposure protocols (B. L. Carter & Tiffany, 1999; Green, Rogers, & Elliman, 2000; Kemps & Tiggeman, 2007). In laboratory studies on cue-induced craving, cue exposure duration varied from 2 s (e.g., Nijs, Franken, & Muris, 2008) to 180 s (e.g., Tetley et al., 2009). For cue exposure within the NFB protocol, an intermediate exposure duration of 30 s was chosen to allow for vivid imagination processes of the cued foods while keeping session duration in an adequate time frame.

2.5.5 Target group of the intervention

Given the novelty of the NFB approach in the treatment of DE, the first evaluation studies should be conducted in an appropriate sample that would benefit from this kind of treatment, but is not affected in a clinically relevant way. For this purpose, the subpopulation of female REs without manifest eating disorders constitutes a suitable target group.

The aforementioned antecedents of DE have been extensively studied in REs and several theories on DE even base on this population (Heatherton & Baumeister, 1991; Ruderman, 1986; Stroebe et al., 2008). Perceived stress and negative affect have repeatedly been found to facilitate food craving, disinhibition, and binge eating in female REs (Greeno & Wing, 1994; Habhab et al., 2009; Wardle et al., 2000). Moreover, several researchers found enhanced cue reactivity in response to exposure with food cues in REs (e.g., Brunstrom et al., 2004; Papies, Stroebe, & Aarts, 2007). Furthermore, women generally show higher tendencies for eating pathology, restrained eating, and DE (cf. 2.1.2) and are therefore more suitable as a target group for first evaluation of this NFB protocol.

Finally, restrained eating has commonly been identified as a risk factor for eating disorders (Polivy & Herman, 1985; Stice, 2002). Here, unsuccessful REs – that is, those individuals who frequently experience disinhibition and therefore do not attain self-set weight goals – would be most likely to benefit from the intervention. The *Restraint Scale* (Herman & Polivy, 1980; German version: Dinkel et al., 2005) would therefore serve best to categorize potential participants as REs: Several studies found restrained eating, assessed with this instrument, to reflect unsuccessful rather than successful restraint (Heatherton, Herman, Polivy, King, & McGree. 1988; Ogden, 1993; Van Strien, 1999).

Thus, the target group of the intervention trials consisted of adult female REs who experience DE, characterizing a subclinical sample yet without clinically-relevant eating pathology or manifest eating disorders. In the light of possible ethical concerns and potential intervening factors, legal age (≥ 18 years) was required and further exclusion criteria applied. Details on operationalization of these criteria are included in the respective articles.

2.5.6 Final treatment protocol

The final NFB protocol consists in a ten-session individual treatment based on EEG high beta-reduction after repeated confrontation with individually appealing food cues. For each participant, the ten personal food items that are most likely to induce food craving and DE are assessed prior to treatment. Pictures of these food items are printed on presentation cards (DIN

A4) to be used for craving-induction within the sessions. In line with other studies on food craving, participants are instructed to abstain from eating for at least three hours prior to sessions, to assure an appealing character of the displayed food cues (Kemps & Tiggemann, 2007; Larsen, Hermans, & Engels, 2012).

NFB is conducted in comfortable therapy rooms by exclusively female trainers. After preparation of the scalp skin with a conductive peeling paste, unipolar EEG activity is derived from the vertex position (Cz), linked to the right earlobe. A ground electrode is placed on the left earlobe. EEG high beta activity, as well as muscular artifacts which might influence this EEG spectral range, are displayed on the patient screen, whereas raw EEG activity and possible artifacts are monitored by the trainer on a distinct trainer screen.

Every NFB session consist of an adaptation phase to allow for physiological arousal to settle down (180 s), followed by ten exposure phases with food cues (each 30 s) altering with self-regulation phases (each 180 s in Study 1, later adjusted to 120 s due to perceived drowsiness reported by some participants in the pilot-study). The final adjusted protocol after refinement contained an additional relaxation period at the end of the treatment (180 s). Here, EEG alpha activity, as a commonly used indicator of relaxation in NFB, is additionally displayed on the patient screen (see Figure 4). Altogether, session duration sums up to ~45-60 minutes.

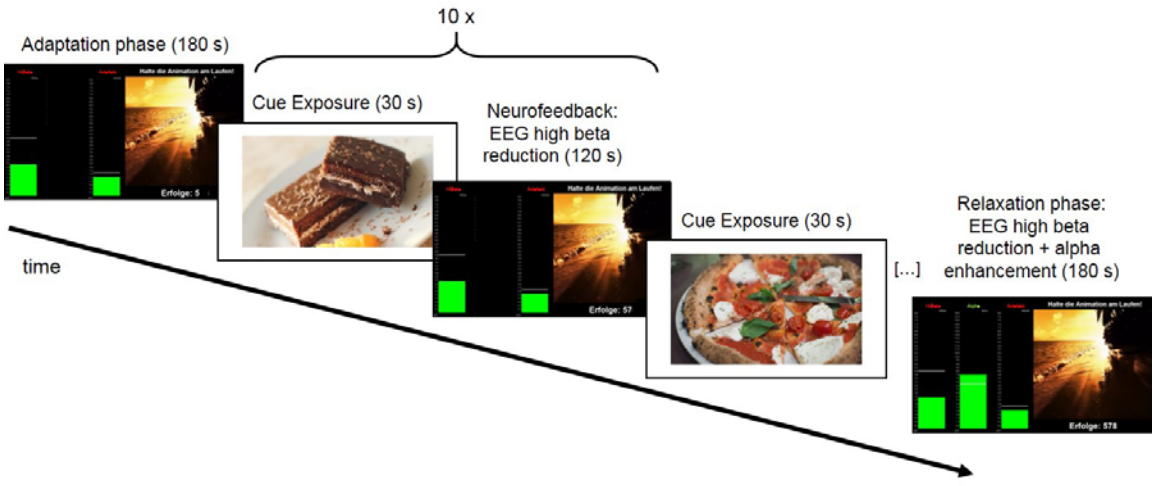

Figure 4. Schematic presentation of a single neurofeedback session.

For exposure phases, participants are instructed to focus on the presentation cards in front of them and to vividly imagine the cued food items, including their multisensory properties, such as taste and smell. For self-regulation phases, presentation cards are removed and trainers globally instruct participants to relax. Trainers further encourage the participants to try different relaxation strategies within the first sessions, to identify those that would result in the most effective EEG high beta reductions. Here, choice of strategies is open for the participants. However, open eyes are a prerequisite for these relaxation strategies, because closing one's eyes alters the topography and power of EEG activity (e.g., Barry, Clarke, Johnstone, Magee, & Rushby, 2007).

As a feedback modality, green bar diagrams display EEG high beta and artifacts on the participant's screen together with thresholds that should not be exceeded. In case those bars exceed the thresholds, the bars turn red. Thresholds are initially set to 4 µV for EEG high beta and 1-1.5 µV for artifacts, but are adjusted to the individual's baseline within the adaptation phase. In addition, a relaxing animation of a beach scenery at sunset is displayed continuously, which then stops whenever a bar exceeds the threshold. For each second of successful regulation in both bars, a success score is added to a score count on the participant screen.

Success rates are set to 85% within the first four sessions, and then gradually lowered in 5 %-steps. This would allow for shaping processes to occur and maintain a challenging nature of the protocol with progress in sessions by means of task-difficulty adjustment (sessions 1 to 4: 85%, sessions 5 & 6: 80%, sessions 7 & 8: 75 %, sessions 9 & 10: 70%). In the fifth session, participants receive a trigger card for transfer purposes and to practice the acquired strategies in everyday life.

All sessions are journalized by the trainers. Before the sessions, participants are encouraged to report on successes and difficulties to apply the strategies learned in NFB at home. After each session, participants are asked to briefly describe the experience of their progress and subjective observations in the respective session with regard to perceived cravings and states of arousal. In the last session, trainers and participants reflect on the treatment. Trainers answer remaining questions and participants evaluate the overall treatment experience. Until a follow-up at three months post-treatment, trainers would be available to answer questions by telephone or e-mail. The treatment is manualized to ensure standardized treatment delivery by different trainers.

3. Aims and research questions of the conducted studies

3.1 General efficacy of neurofeedback for disinhibited eating

After the theoretical and methodological development of a NFB protocol based on EEG high beta reduction after food cue exposure, **Study 1** examined the **general efficacy and acceptance of the developed NFB protocol** in the treatment of DE regarding subjective binge eating episodes (termed *overeating episodes* in Paper 1, as required during the journal's review process). It was a primary aim of the study to determine, if the NFB protocol would be suitable as a brain-directed intervention in the treatment of DE. Given the lack of NFB studies for the treatment of DE and the relatively thin empirical basis of EEG spectral research in DE (cf. 2.3.2), a piloting trial was crucial to validate the general possibilities to treat DE by means of NFB, and whether this approach would be feasible and well-accepted by participants.

This pilot-study was conducted in 2013 as an RCT in a target-group of female REs. A total of 34 female participants were assigned to either NFB or a waitlist group (n = 17 each) with the assessment of several outcome variables pre and post treatment. Besides an analysis of effects on the frequency of subjective binge eating episodes and associated distress as primary outcomes, possible changes in secondary outcomes related to DE were evaluated. Here, treatment effects on food craving, perceived stress, specific dietary self-efficacy (perceived dieting success), and general well-being were of interest. To determine whether the NFB would exert sustained beneficial effects after the treatment period, all NFB-participants attended a three-month follow-up assessment.

An additional aim of the pilot-study was to assess qualitative feedback and participants' overall treatment experience with regard to the novel intervention approach. This procedure served the purpose to determine, whether adjustment of the treatment setup and manual would be necessary for future studies. In summary, Study 1 addresses the following research questions:

- Does a ten-session NFB protocol, based on EEG high beta reduction after food cue exposure, reduce DE and associated distress?
- Does the NFB protocol exert beneficial effects on secondary outcomes associated with DE (food craving, perceived stress, dietary self-efficacy, and well-being)?
- Are the effects of the NFB stable up to the three-month follow-up?
- Is the NFB approach accepted by the participants with respect to the treatment of DE?

- Is there any need for improvement in the treatment setup or NFB protocol?

3.2 Specific efficacy of neurofeedback for disinhibited eating

After promising results of the pilot-study with regard to the general efficacy of NFB in the treatment of DE, it was an important second step to establish a follow-up study, which would replicate findings and provide insights into the specific efficacy of the brain-directed NFB approach in reducing DE. In line with this intention, **Study 2** aimed at assessing the **specific efficacy of the NFB protocol in reducing subjective binge eating episodes.**

Aside from the NFB technique, the developed treatment protocol consists of several components that have previously been shown to exert positive effects on DE, for example repeated food cue exposure (Jansen, 1992; McIntosh et al., 2011) and relaxation instructions (Katterman et al., 2014). Further, general attributes of the therapeutic process may have contributed to improvements (Campbell, Fitzpatrick, Haines, & Kinmonth, 2000; Thibault, Lifshitz, & Raz, 2016) as NFB was only compared to a waitlist group in the pilot-study.

A second RCT therefore aimed at providing more reliable insights on the specific efficacy of the NFB approach. This study included NFB, a waitlist and an additional, highly comparable alternative treatment control group in a larger, yet independent sample (n = 75; n = 25 per group). The alternative treatment consisted of a treatment with the exact same setup regarding session frequency and composition (cue exposure and subsequent self-regulation) except for the self-regulation task. Here, participants were instructed to apply relaxing mental imagery strategies (Kemps & Tiggemann, 2007; Knäuper et al., 2010). Given the similarities of the interventions, it is possible to control for the effects of cue exposure, relaxation, and unspecific treatment factors.

The frequency of subjective binge eating during the last week and associated distress were primary outcomes. Dietary self-efficacy, food craving, and perceived stress were secondary outcomes, supplemented by somatic self-efficacy (i.e., perceived abilities to relax). Both active treatment groups were compared to a waitlist to determine differential efficacy. Participants of the active groups further provided data on subjective treatment evaluations and pre-treatment expectations to determine possible differences in treatment credibility and satisfaction. NFB and mental imagery participants attended a three-month follow-up to determine intermediate stability of treatment effects.

With this study setup, the second trial addresses the following research questions:

- Can the positive effects of NFB in the pilot-study be replicated?
- Does the NFB treatment lead to higher reductions in the frequency of binge eating episodes and associated distress than a highly-comparable alternative treatment?
- Does NFB exert stronger effects on secondary outcomes (food craving, dietary and somatic self-efficacy, perceived stress) than a highly-comparable alternative treatment?
- Does the subjective treatment experience differ among the NFB and mental imagery treatments?
- Are results of the active treatments stable up to the three-month follow-up?

3.3 Treatment mechanisms in neurofeedback for disinhibited eating

In biofeedback research, it is a classical debate, which mechanisms contribute to treatment effects (e.g., Holroyd et al., 1984; Wickramasekera, 1999). Especially with regard to novel NFB protocols – such as the one developed for the present dissertation – the contribution of physiological learning versus psychological processes has to be determined to understand treatment mechanisms. Thus, **Study 3** served for an initial **analysis of physiological versus psychological learning mechanisms** in the NFB protocol for DE.

While the second trial indicated specific efficacy of the general NFB approach in the treatment of DE, it does not directly provide information on the role of EEG high beta reduction after food cue exposure. Thus, the influence of physiological learning mechanisms in the NFB group and self-regulatory abilities regarding physiological activity could not be inferred from the results of Study 2. Former research has shown that enhancements in self-efficacy contribute to the success of biofeedback (Holroyd et al., 1984; Wickramasekera, 1999), and that mere attempts to control brain activity can elicit subcortical changes (Ninaus et al., 2013). Analyses of progress in physiological learning and comparisons to psychological learning processes were therefore necessary to gather insights into the relevant treatment mechanisms involved in the NFB intervention.

To assess differential influences of physiological and psychological learning and provide insights on treatment mechanisms, an EEG-experimental study (Study 3) was incorporated in the second RCT. Changes in physiological self-regulatory abilities, as well as subjective somatic self-efficacy were assessed and their relationships with treatment success were analyzed.

The merged-groups sample (n = 54, including former waitlist participants) of the second trial served as an analysis sample for the EEG-experiment. Before and after the treatment period, participants in NFB and mental imagery conditions participated in an experiment confronting them with food cues (3 × 30 s) followed by self-regulation periods (3 × 120 s) in a fully standardized order. At pre-treatment, experimenters instructed the participants to apply individual relaxation strategies in self-regulation phases; at post-treatment, each woman applied the strategy learned in the respective intervention (NFB-strategies or mental imagery). During self-regulation, no feedback was provided.

EEG high beta activity at the vertex position (Cz) during self-regulation phases served as a target variable, corresponding to the training position in NFB. In addition, self-rated somatic self-efficacy, as well as treatment success (i.e., reduction in binge eating frequency), were assessed. Only artifact-free EEG data were analyzed in this experiment (n = 36, n = 18 each in NFB and mental imagery).

Based on these data, objective physiological learning was determined (pre- to post-treatment reductions in EEG high beta activity during self-regulation phases without feedback), as well as subjective changes in specific somatic self-efficacy. Learning effects regarding EEG regulation should be compared between the two active groups and between successful (≥ 50% reduction in binge eating) and unsuccessful participants (< 50% reduction in binge eating). Further, their respective contribution to post-treatment outcomes should be analyzed in regression analyses.

In summary, Study 3 addresses the following research questions:

- Does NFB, with an EEG high beta reduction protocol, result in physiological learning (in the absence of feedback)?
- Does physiological learning differ between successful and unsuccessful participants in NFB at post treatment?
- Does physiological learning show stronger relations to treatment outcomes than psychological learning (i.e., somatic self-efficacy)?

4. Study 1: General efficacy of neurofeedback for disinhibited eating

4.1 Citation and author contributions

Title: Neurofeedback reduces Overeating Episodes in female Restrained Eaters – A Randomized Controlled Pilot-Study

Authors: Jennifer Schmidt & Alexandra Martin

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Author contributions:

- The study was designed by Jennifer Schmidt with supervision of Alexandra Martin.
- The study was organized and conducted by Jennifer Schmidt.
- The manuscript was written by Jennifer Schmidt.
- Participants were recruited by Jennifer Schmidt.
- Data were analyzed and interpreted by Jennifer Schmidt with intellectual input and advice by Alexandra Martin.
- The revision of the manuscript was conducted by Jennifer Schmidt with intellectual input and advice by Alexandra Martin.

Further assistance:

- Rooms and equipment for conducting the treatments were provided by Prof. Dr. Ralf Stürmer (*psyrecon research & consulting GmbH*, Wuppertal).
- Rooms for information sessions were provided by Prof. Dr. Ralf Stürmer (*psyrecon research & consulting GmbH*, Wuppertal) and by the *Praxisklinik im Südpark* (Solingen).
- Neurofeedback supervision was provided by Prof. Dr. Ralf Stürmer and Gisela Ulmer.
- Participant recruitment was administratively supported by Karin Bohr, Kamila Lewicki, and Rahel Kuttner.
- Training sessions were partly conducted by Kamila Lewicki and Rahel Kuttner.
- Assessments and data entry were assisted by Kamila Lewicki and Rahel Kuttner.

4.2 Published manuscript

Neurofeedback Reduces Overeating Episodes in Female Restrained Eaters: A Randomized Controlled Pilot-Study

Jennifer Schmidt, Alexandra Martin

Abstract

Overeating episodes, despite of intentions to control weight, are a common problem among women. Recurring episodes of overeating and dietary failure have been reported to result in higher Body Mass Indexes and to induce severe distress even in non-clinical groups. Based on findings from physiological research on eating behavior and craving, as well as previous biofeedback studies, we derived a cue exposure based EEG neurofeedback protocol to target overeating episodes. The treatment was evaluated in a randomized controlled trial, comparing a neurofeedback group (NFG; $n = 14$) with a waiting list control group (WLG; $n = 13$) in a sub-clinical sample of female restrained eaters. At post-treatment, the number of weekly overeating episodes and subsequent distress were significantly reduced in the NFG compared to the WLG ($p < .01$; $r > .50$). In a 3 month follow-up, effects in the NFG remained stable. As secondary outcomes, perceived dieting success was enhanced after the treatment. At follow-up, additional beneficial effects on trait food craving were observed. Altogether, we found preliminary evidence for the cue exposure neurofeedback against overeating episodes in female restrained eaters, although specific effects and underlying mechanisms still have to be explored in future research.

Keywords: Neurofeedback, Overeating, Restrained eating, Food craving, Randomized controlled trial

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Introduction

In food rich environments of Western developed countries, temptation of palatable food is not easy to resist. Thus, it is not surprising, that overweight and obesity (Swinburn et al. 2011), as well as eating disorders related to dieting and overeating (Mitchison et al. 2012) have been on the rise throughout the last decades. One common important factor with regard to the development of these problems are episodes of overeating that often precede weight control failures, obesity, and binge-related eating disorders (Klesges et al. 1992; Polivy and Herman 1985). Further, overeating episodes induce distress and negative affect (Stein et al. 2007), which again facilitates future occurrence of overeating to regulate these aroused emotional states, resulting in a vicious circle (Cools et al. 1992; Gluck 2006; Hay and Williams 2013).

On a physiological basis, these antecedents can be found in states of tension that is stressful arousal (Freeman and Gil 2004; Jastreboff et al. 2013), especially after confrontation with food cues and in subsequent states of craving (Hill 2007; Pelchat 2002). With regard to the possible negative effects on bodily and mental health, even overeating episodes in sub-clinical groups should be addressed by psychological intervention research.

To date, several psychological interventions that target antecedents of unwanted consumption and overeating have been assessed. With respect to autonomic physiological arousal in food cravers, Meule et al. (2012a) reported preliminary evidence for the effectiveness of heart rate variability biofeedback in altering food craving. However, the researchers reported that the training affected cognitions and attitudes rather than behavior. Using a different physiological measure, Teufel et al. (2013) applied electrodermal biofeedback in obese women, either in a cue exposure setup or in a merely relaxation-based setup. Here, the cue exposure setup enhanced self-efficacy regarding food intake and had higher long term effects than the relaxation-based approach.

Reducing the stressful arousal at its origin in the brain (McEwen 2007; Saletu-Zyhlarz et al. 2004), neurofeedback targeting associated brain responses may constitute a promising approach for the treatment of overeating episodes. In a real time fMRI neurofeedback study among men, Frank et al. (2012) targeted upregulation of the anterior insular cortex as a brain region involved in the processing of food cues. Despite of promising results regarding self-regulation ability, no results of possible effects on eating behavior were provided. Further, more affordable and applicable neurofeedback techniques, such as electroencephalographic (EEG) neurofeedback, might be more appropriate for the treatment of widespread overeating episodes. Yet, in a recent

review on neurofeedback in disordered eating, Bartholdy et al. (2013) did not report any studies applying EEG neurofeedback against overeating or binge eating episodes.

With regard to possible spectral ranges that may constitute target frequencies in an EEG neurofeedback protocol, especially fast frequencies in the higher beta range (~ 18–30 Hz) have been shown to accompany ruminative states of stressful arousal (Andersen et al. 2009; Thompson and Thompson 2007; Seo and Lee 2010). Further, Tammela et al. (2010) reported that EEG activity in the beta range throughout food picture presentation is related to disinhibition in obese women with binge eating disorder.

Excess EEG high beta activity has also been shown with respect to drug-induced stressful states of craving (for a review see: Parvaz et al. 2011) which are described as comparable to states of food craving preceding overeating episodes (Sinha and Jastreboff 2013; Styn et al. 2013). Therefore, high beta frequencies are target to inhibition in the reduction of stressful arousal in several neurofeedback protocols (Egner and Gruzelier 2001, 2004; Paquette et al. 2009).

The aim of the present study was to develop and evaluate a neurofeedback protocol based on previous psychophysiological findings. To target a sample which is especially vulnerable to overeating episodes, we chose to address restrained eaters (REs). Peter Herman and colleagues (Herman and Mack 1975; Herman and Polivy 1975) defined restrained eating (RE) as the intention to cognitively restrict caloric intake with the purpose of losing or maintaining weight. Many REs are especially cue-reactive with regard to food cues (Brunstrom et al. 2004), prone to experience food craving, subsequent disinhibition, and finally overeating or binge eating episodes (Polivy and Herman 1985; Polivy et al. 2005; Ruderman 1986; Westenhöfer 1991).

With reference to their repeated failures in weight control (Heatherton et al. 1991) and resulting distress (Stein et al. 2007), we concluded that this population constitutes an especially suitable target group for this neurofeedback pilot study. Further, we decided to include females only, because dysfunctional eating behaviors, such as overeating episodes, are generally more prevalent among women compared to men (Provencher et al. 2003).

We hypothesized that a neurofeedback protocol, inhibiting EEG high beta activity after food cue exposure, is effective in reducing the occurrence of overeating episodes. Subsequently, distress associated with binge eating episodes should be alleviated. As secondary outcomes, effects on food craving, perceived dieting success, perceived stress, and well-being were scrutinized to gather insights into possible mechanisms of the training. We expected effects to

be stable at a 3 month follow-up. Additionally, we collected qualitative feedback to improve future applications of the intervention.

Due to the exploratory nature of this pilot study, we decided to conduct a randomized controlled trial using a waiting list control group. With regard to the novelty of this setup, we selected participants in the sub-clinical range of RE only, excluding pathological eating behavior related to overeating and dieting (e.g. manifest bulimia nervosa or binge eating disorder). In line with this decision, overeating episodes in our sample were not required to fulfill DSM-criteria for binge episodes (i.e. larger amount than other people would eat in a comparable time period and loss of control over eating). Instead we primarily aimed at a reduction of subjective overeating episodes as a result of craving, which include undesired ingestion of high calorie food.

Method

Study Design

In this randomized controlled trial, a treatment group (neurofeedback group (NFG)), receiving a neurofeedback-based intervention against overeating episodes was compared to a waiting list group (WLG) at a pre-treatment (T0) and post-treatment (T1) assessment. The occurrence of overeating episodes and distress due to overeating episodes were analyzed as primary outcome measures. Trait-food craving, perceived dieting success, perceived stress, and well-being were assessed as secondary outcome measures. The NFG was additionally invited to a follow-up session (T2) 3 months after the last training session. The research protocol was approved by the local ethics committee.

Sample

Inclusion criteria were self-reported occurrences of overeating episodes, female gender, legal age, and restraint scores ≥ 12 on the German adaptation of the Restraint Scale (Dinkel et al. 2005). Exclusion criteria were (1) diagnoses of eating disorders within the last 10 years, (2) Body Mass Index (BMI) < 20 kg/m², (3) alcohol abuse, (4) diabetes mellitus, (5) neurological disorders (e.g. epilepsy), (6) current medication with drugs eliciting weight fluctuations (e.g. cortisone, lithium), (7) current pregnancy, (8) a current weight reduction diet other than long term lifestyle diets (e.g. Weight Watchers, calorie counting, low carb nutrition). Since any unreported eating disorders should be excluded, the Eating Disorder Examination Questionnaire (EDE-Q; Fairburn and Beglin 1994; Hilbert and Tuschen-Caffier 2006) was

additionally used for screening. Participants with critical EDE-Q values were interviewed by a trained psychologist and excluded from participation in case of manifest eating disorder symptomatology.

We recruited participants using information leaflets at the University of Wuppertal and in medical offices in the area, e-mail newsletters and website announcements. Sixty-four persons responded to our recruiting attempts and were assessed for eligibility. Twenty-two respondents withdrew before the kick-off event. Three respondents did not meet inclusion criteria, one respondent had to be excluded due to former eating disorder diagnosis and four respondents were excluded due to a BMI < 20. This resulted in a sample of 34 participants, whereof 17 were assigned to the NFG and 17 were assigned to the WLG.

Table 1 Sample characteristics in demographics and screening instruments.

Variable		NFG		WLG		Test statistics
n		14		13		
		M (*SD*)		*M* (*SD*)		
Age		37.93 (11.18)		31.15 (9.56)		$t(25) = -1.69$, $p = .104$
Overall score Restraint Scale		19.14 (4.17)		18.30 (3.43)		$t(25) = -0.57$, $p = .576$
Body Mass Index		27.34 (5.48)		27.36 (4.99)		$t(25) = 1.72$, $p = .109$
		n	%	*n*	%	
Employment status (*n* / %)	Student	4	28.6	3	23.1	$\chi^2(5) = 3.18$, $p = .673$
	Apprentice	0	0.0	2	15.4	
	Employee	6	42.9	4	30.8	
	Clerk	1	7.1	2	15.4	
	Self-employed	2	14.3	1	7.7	
	Retired	1	7.1	1	7.7	
Smoking (*n* / %)	No	11	78.6	9	69.2	$\chi^2(2) = 0.50$, $p = .780$
	Yes	2	14.3	2	15.4	
	Occasionally	1	7.1	2	15.4	

In each of the groups, three participants dropped out. Data of $n = 1$ were excluded from analysis due to an excessive amount of missing data, resulting in samples of $n = 14$ for the NFG and $n = 13$ for the WLG post-treatment. At follow-up, $n = 2$ NFG participants did not respond

to our invitation, resulting in a sample of $n = 12$. Detailed sample characteristics are shown in Table 1. A participant flow diagram according to CONSORT-guidelines is presented in Fig. 1.

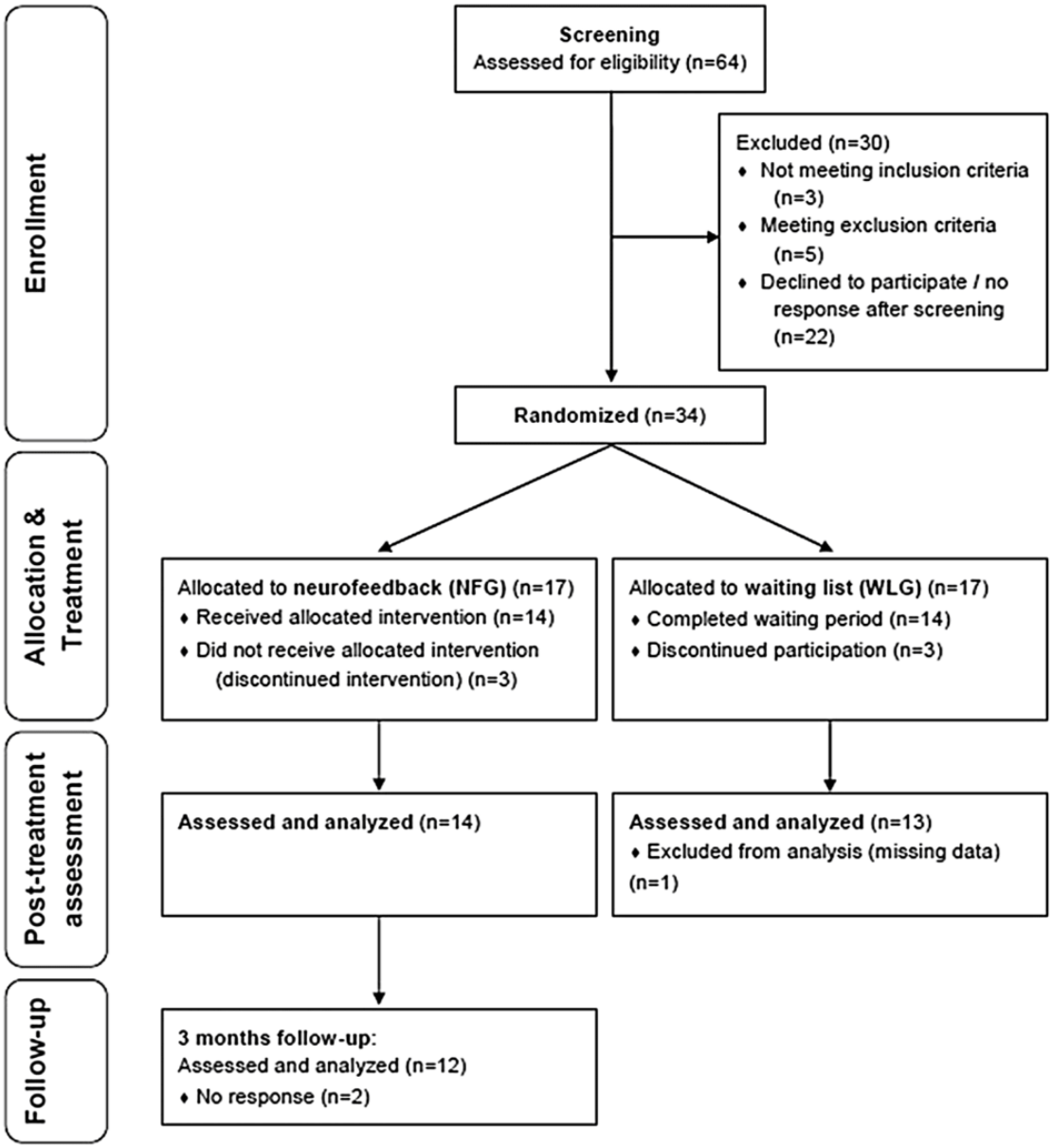

Fig. 1 Design and patient flow.

Procedure

Women interested in participation received an e-mail containing written information. Further, a web link to an online questionnaire was provided for assessment of inclusion and exclusion criteria. When the requirements were fulfilled, we invited participants to a kick-off

event, starting with general information on the study and the neurofeedback method. Participants then filled in informed consent and questionnaires (see "Assessment Instruments"'). Thereafter, a psychoeducative presentation on healthy nutrition and overeating episodes was held. At the end of the event, we assessed each participant's weight to determine weight status.

We used a stratified randomization approach, to ensure comparable weight distributions within the groups. Participants fulfilling inclusion criteria were first classified as normal weight (BMI < 25) or overweight (BMI ≥ 25). Participants in both subgroups were then randomly assigned to either the NFG or WLG. After randomization, the treatment started in the NFG, with a simultaneous 8 week waiting period in the WLG. Treatment was offered to the WLG participants after the waiting period (n = 9 accepted) but data were not included in our analyses. The study was conducted between February and November 2013 in rooms provided by psyrecon GmbH in Wuppertal, Germany.

Neurofeedback Training

Trainers. Two graduate psychology students, experienced in clinical practice, operated the neurofeedback sessions. They were extensively trained by an experienced neurofeedback trainer in terms of the neurofeedback equipment, electrode attachment and software use. All procedures and instructions were standardized based on a treatment manual that we developed for the present trial.

Neurofeedback protocol. For this pilot study, we chose a relatively low-threshold ten session treatment that would be suitable for a sub-clinical sample, based on session numbers in comparable bio- and neurofeedback studies (Meule et al. 2012a; Teufel et al. 2013; Vernon 2005). The neurofeedback was administered with two sessions during week 1–4 and one weekly session in weeks 5 and 6. Each session lasted approximately 45 min. Sessions started with an adaptation phase (180 s) to ensure participants had the opportunity to calm down and to allow for adjustment of high beta thresholds to the participants' individual baseline values (ranging from approx. 2–8 µV). This phase was followed by ten alternating phases of cue exposure (30 s) and subsequent relaxation (180 s).

Before the training period, we asked each participant to name ten specific food items that frequently elicit food craving and episodes of overeating. To ensure individual appeal, pictures of the respective items where either digitally provided or personally selected by the participants at the research facilities. Printed presentation cards of these pictures served as stimuli for the

cue exposure phases of the training. During cue exposure, stimuli were presented in random order on a presentation desk in front of the participants. Here, the trainers instructed participants to focus on the picture and imagine the food as vividly as possible (including smell, taste, and consistency). After 30 s the picture was removed for the following relaxation phase. Participants then had to focus on the screen displaying physiological reactions and to try keeping both bars below the thresholds. We asked participants to avoid eating for 3 h prior to each session, to ensure sufficient appeal of the presented stimuli.

In the first session, the therapists explained that high beta activity would decrease in a state of relaxation, and how artifacts would result from heavy movements or speaking. Participants were encouraged to try different techniques of relaxation with the only prerequisite of keeping their eyes open. Thus, each participant was able to develop an individual strategy to efficiently reduce EEG high beta activity according to the feedback presented on the client screen.

Probability of success in high beta regulation was lowered with progress of the training sessions. It was set to 85 % in sessions 1–4, 80 % in sessions 5 and 6, 75 % in sessions 7 and 8, and 70 % in sessions 9 and 10. This procedure was chosen to preserve challenging effects during the training. Throughout each session, the trainer adjusted the threshold for high beta activity whenever probability of success derived more than 5 % from the intended value for more than 1 min of the relaxation phases.

Apparatus. We performed the training using the Mindfield Mindmaster EEG and the corresponding software BioEra Clinical Basic 1.63. The software works in a split screen-mode. The client screen displays bar diagrams of selected EEG frequency ranges, while a trainer screen serves to adjust thresholds and monitor clients' mean power in different EEG spectra. Probability of successful high beta regulation according to the preset thresholds is displayed on the trainer screen.

For the sessions, participants were seated in a comfortable armchair at a distance of approximately 1 m to a 22" computer monitor displaying the client screen. After skin preparation with an EEG peeling paste on scalp and earlobes, electrodes were attached, with the target electrode on the vertex position (Cz; Jasper 1958), reference electrode on the left and ground electrode on the right earlobe. Impedance was kept below 5 kΩ. For the intended high beta reduction protocol, EEG high beta activity in the spectral range of 23–28 Hz was selected as feedback frequency. We selected this range of the relatively broad beta-spectrum to prevent down training of beneficial ranges such as sensorimotor rhythm (12–15 Hz) or lower and

intermediate beta (16–22 Hz) which are associated with common states of attention. Instead, we aimed at a specific reduction of cortical hyperarousal (Egner and Gruzelier 2004; Thompson and Thompson 2007). After online Fast Fourier Transformation of the raw EEG, power (in µV) of this frequency range was displayed as a bar diagram on the client screen.

For control reasons, power of muscular artifacts was shown in addition, as high beta activity may be influenced by muscular electrical activity, due to similar frequency ranges. This assured that participants would not misinterpret artifacts from movements or swallowing as signs of tension. Bar diagrams in a desired range were displayed in green color, and an animated video of a beach landscape at sunset was presented. When any of the bars excessed the preset threshold, the bar turned red and the animation stopped. The initial thresholds were set to 4 µV for high beta and to 1.5 µV for artifacts.

Assessment Instruments

Screening instruments. All screening instruments were applied prior to randomization, either in the online screening-questionnaire or at the kick-off event.

Restraint Scale. For assessment of RE, the Restraint Scale (RS; Dinkel et al. 2005; Herman and Polivy 1980) was applied. Answer options are provided on 4- or 5-point Likert-scales, ranging from 0 to 3 or 0 to 4 respectively. The sum score ranges from 0 to 34. Evidence for the construct and criterion validities of the German version were reported, and internal consistency showed to be good with $\alpha = .83$ (Dinkel et al. 2005). In the present study internal consistency was still satisfactory ($\alpha = .64$). Referring to Dinkel et al. (2005), we used a cut-off score ≥ 12 to classify respondents as REs. The RS was administered online.

Screening of inclusion and exclusion criteria. Inclusion and exclusion criteria were assessed by an online screening-questionnaire, consisting of seven items with a yes/no format, e.g. “Have you been diagnosed with an eating disorder within the last 10 years? *Yes / No*”. Concerning medication, participants could indicate a not sure option and type in names of their medicaments. We then checked for possible negative effects of the respective medication. BMI was calculated as weight in kg / (height in m)^2.

Eating Disorder Examination Questionnaire. The Eating Disorder Examination Questionnaire (EDE-Q; Fairburn and Beglin 1994; Hilbert and Tuschen-Caffier 2006) was used to screen for undetected, clinically relevant eating disorders. It consists of 28 items with a 7-point rating scale (0 = *attribute non-existent*; 6 = *attribute existent every day/in an extreme*

degree) referring to symptoms and eating behavior during the previous 28 days. Concurrent validity is reported as good and construct validity as acceptable (Mond et al. 2004). Internal consistency of the German version has been reported as excellent, $\alpha = .97$ (Hilbert and Tuschen-Caffier 2006), with $\alpha = .90$ in the present study. We pre-screened diagnostic criteria regarding eating disorders (e.g. vomiting behavior in bulimia nervosa) which were not fulfilled by any participant. An overall mean sum score ≥ 4 then served as critical value in line with reference scores for female populations (Mond et al. 2006).

Demographics. For demographics, we assessed age and employment status on a questionnaire. The questionnaire further contained questions about smoking (*yes / no / occasional*) and possible lifestyle diets (such as vegetarianism, Weight Watchers, calorie counting or low carb nutrition).

Outcome measures. Outcome measures were assessed at T0 and T1. In addition, the NFG filled in all outcome measures at follow-up (T2).

Primary outcome measures. A self-constructed questionnaire was used for the assessment of overeating episodes and caused distress. Following a definition (overeating episodes induced by craving urges, resulting in consumption of high calorie food without experiencing physiological hunger), subjects were asked to report the number of overeating episodes during the last 7 days and the average distress experienced due to overeating on a 6-point rating scale (0 = *not at all*, 1 = *light*, 2 = *rather light*, 3 = *rather strong*, 4 = *strong*, 5 = *very strong*) with an additional option (*not applicable*) in case of no episode (= 0).

Secondary outcome measures.

Food craving. The Food Cravings Questionnaire, trait form (FCQ-T; Cepeda-Benito et al. 2000; Meule et al. 2012b) was applied. The questionnaire contains 39 items on habits and behaviors related to food craving with a 6-point rating scale (1 = *never / not applicable*, 2 = *seldom*, 3 = *sometimes*, 4 = *often*, 5 = *almost always*, 6 = *always*). Convergent and divergent validity of the FCQ-T are given, and internal consistency of the German version has been reported as high ($\alpha = .96$; Meule et al. 2012b) with $\alpha = .96$ in the present study. For this study, the sum score was used to assess food craving.

Perceived dieting success. As a measure of self-regulatory competence we assessed perceived dieting success with the Perceived Self-Regulatory Success in Dieting Scale (PSRS; Fishbach et al. 2003; Meule et al. 2012c). The scale contains three items (one item reverse coded) with a 7-point rating scale (1 = *not successful/not difficult*; 7 = *very successful/very*

difficult) allowing for a total score in the range of 3–21. Validity of the measure has been demonstrated, for example by negative correlations with BMI, and internal consistency for the German version is satisfactory (α > .70; Meule et al. 2012c), with α = .70 in the present study.

Perceived stress. Perceived stress within the last month was assessed with the Perceived Stress Scale (PSS; Cohen et al. 1983; Cohen and Williamson 1988). The applied version consists in ten items (e.g. "How often have you felt nervous or stressed?") and a 5-point answer scale (0 = never; 4 = very frequently). Concurrent, convergent, and predictive validity have been reported for the PSS, and internal consistency was reported as good with α > .80 in different samples and cultural backgrounds (e.g. Mitchell et al. 2008; Reis et al. 2010). In the present study the internal consistency was high (α = .89).

Well-being. We applied the German version of the World Health Organization-five well-being index (WHO-5; Psychiatric Research Unit 1998). Its five items assess aspects of well-being within the last 2 weeks on 6-point rating scales (0 = *at no time*; 5 = *all of the time*), resulting in a sum score of 0 (very poor well-being) to 25 (excellent well-being). WHO-5 has demonstrated good psychometric properties, e.g. α = .82 in a sample of diabetics (De Wit et al. 2007), reaching α = .88 in the present study. External and internal validity of the WHO-5 have been shown in different populations (e.g. diabetics: De Wit et al. 2007; elderly: Heun et al. 2001).

Subjective outcomes. A questionnaire was developed to assess general acceptance of the treatment, perceived outcomes, and strategy applicability in daily routine. Further, satisfaction and intentions to recommend the treatment were assessed. The questionnaire consisted of an overall evaluation ("Altogether, how did you experience the neurofeedback training?") with a 5-point rating scale (1 = *very negative*; 5 = *very positive*); five items to assess the treatment effects (e.g. "The neurofeedback training influenced my eating behavior."); two items on perceived changes in behavior ("I perceived changes in behavior due to the neurofeedback training." / "Others perceived changes in my behavior due to the neurofeedback training."); five items on satisfaction and applicability of the training (e.g. "I am satisfied with the neurofeedback training." / "I would recommend the neurofeedback training to persons experiencing overeating episodes."). Except for the overall evaluation, all items included a 5-point rating scale, 1 = *not at all* to 5 = *very strong*.

Statistical Procedure

Statistical analyses were performed using Statistical Package for the Social Sciences (SPSS) 22.0 for Windows. Single points of missing data were replaced by the participant's mean value in the respective scale (or subscale, if applicable). For analyses of group differences in demographic data, Chi-square tests were performed for categorical variables and *t*-tests for continuous variables. For outcome measures, normality of data within the groups at each assessment session was tested by means of Shapiro–Wilk Test. Possible baseline differences in outcome variables were assessed by means of between groups *t*-tests. For analysis of intervention effects, mixed 2 (group) × 2 (time) ANOVAs were conducted. Post-hoc, Bonferroni corrected pairwise comparisons within and between groups were performed to scrutinize effects. With regard to follow-up analyses, repeated measures ANOVAs were performed for the NFG with Bonferroni-corrected post hoc tests analyzing pairwise differences between T0, T1, and T2.

Some data showed deviations from normality and skewness or curtosis. However, we will report ANOVA results, as ANOVA is robust to violations of normality assumptions when group sizes are comparable and nonparametric reanalyses delivered the same pattern of results. Effect sizes were calculated as r, using z-values to adjust for partly skewness of the data (Fritz et al. 2012). Between group effect sizes were calculated as $r = \left|\frac{z}{\sqrt{N}}\right|$ for intervention outcomes. Effect sizes for within subjects follow-up analyses, were calculated as $r = \left|\frac{z}{\sqrt{2n}}\right|$. Guidelines for *r* are that values > .50 account for large effects, values > .30 for a medium effect and values > .10 for a small effect respectively (Coolican, 2009, p. 395, as cited in Fritz et al. 2012). Effects were tested at a two-sided significance level of .05.

Results

Group Comparison of Baseline Scores

Demographic and screening data showed that mean age, employment status, smoking habits, RS-scores, and BMIs of the NFG and WLG did not differ significantly at baseline (all $p > .104$; see Table 1). At pre-treatment (T0), the NFG and WLG did not differ significantly in occurrence of overeating episodes [$t(25) = -0.33$, $p = .740$] or distress induced by overeating episodes [$t(25) = -1.87$, $p = .073$], nor in any secondary measures (all $p > .335$).

Treatment Outcomes

Descriptives and *F*-statistics of primary and secondary outcome measures are displayed in Table 2 with an additional visualization of primary outcome results in Fig. 2. Descriptives and *F*-statistics of the NFG follow-up analysis are shown in Table 3. An overview on descriptives of subjective outcomes in the treatment evaluation is presented in Table 4.

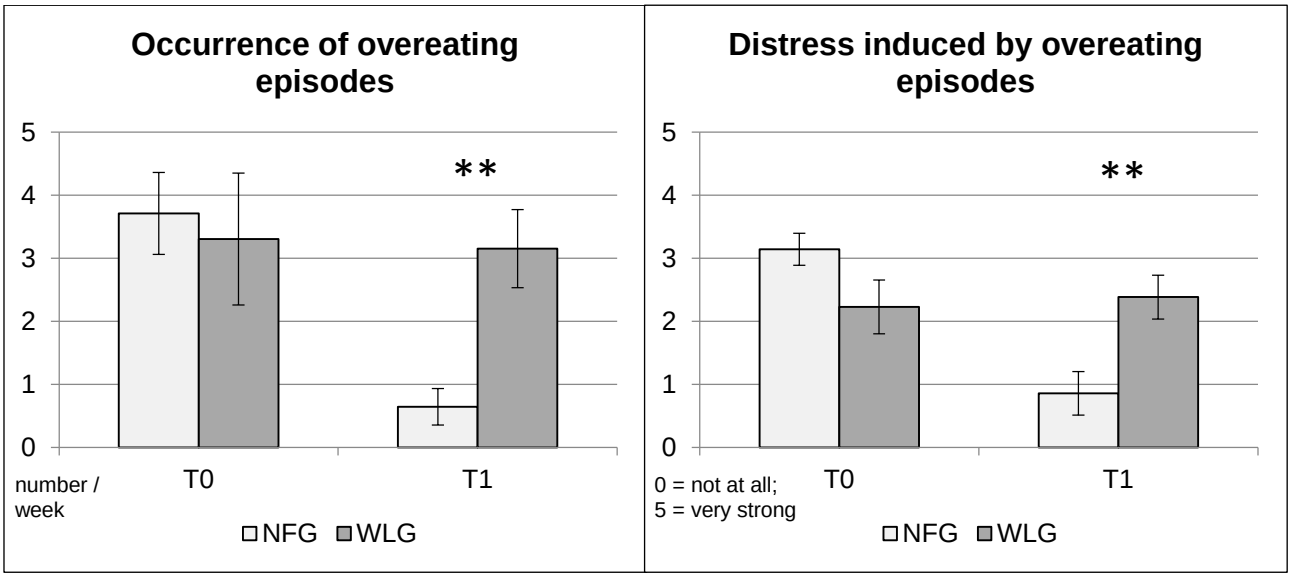

Fig. 2 Means of primary outcome measures. Note: error bars indicate standard errors; ** = significant with $p < .01$; NFG: neurofeedback group; WLG: waiting list group. T0: pre-treatment; T1: post-treatment.

Primary outcome measures. Mixed ANOVAs for the number of overeating episodes per week revealed a significant main effect of time ($p = .011$) and a significant group × time interaction ($p = .020$). Post-hoc analysis showed a significant reduction of overeating within the NFG ($p = .001$) but not within the WLG ($p = .857$). At post-treatment the NFG reported less overeating episodes than the WLG ($p = .001$) with a large between group effect size ($r = .64$).

A comparable pattern was identified for distress induced by overeating episodes. Here, a significant main effect of time ($p = .002$) as well as a significant time × group interaction ($p < .001$) were found. A significant reduction of distress induced by overeating episodes was observed within the NFG ($p < .001$) but not within the WLG ($p = .729$). Between groups comparison showed a significant difference post-treatment ($p = .005$) with a large effect size ($r = .54$).

Table 2 Primary and secondary outcome measures pre- and post-treatment.

Variable	Time	NFG	WLG	Test statistics
N		14	13	
Primary outcome measures		*M* (*SD*)	*M* (*SD*)	
Overeating episodes / week	T0 T1	3.71 (2.43) 0.64 (1.08)	3.31 (3.77) 3.15 (2.23)	**F_t (1, 25) = 7.60, p = .011** F_g (1, 25) = 1.80, p = .192 **F_{int} (1, 25) = 6.22, p = .020**
Distress due to overeating	T0 T1	3.14 (0.95) 0.86 (1.29)	2.23 (1.54) 2.38 (1.26)	**F_t (1, 25) = 12.25, p = .002** F_g (1, 25) = 0.64, p = .430 **F_{int} (1, 25) = 16.03, p < .001**
Secondary outcome measures				
Food Craving Questionnaire	T0 T1	135.64 (26.22) 122.14 (37.39)	135.46 (33.28) 123.54 (29.70)	F_t (1, 25) = 3.11, p = .090 F_g (1, 25) < 0.01, p = .952 F_{int} (1, 25) = 0.01, p = .914
Perceived Self-Regulatory success in Dieting Scale	T0 T1	7.36 (4.65) 10.14 (2.91)	8.92 (3.50) 9.31 (3.88)	**F_t (1, 25) = 5.40, p = .029** F_g (1, 25) = 0.08, p = .780 F_{int} (1, 25) = 3.10, p = .091
Perceived Stress Scale	T0 T1	20.57 (6.97) 17.71 (7.32)	18.85 (6.22) 17.08 (6.81)	F_t (1, 25) = 3.18, p = .087 F_g (1, 25) = 0.26, p = .612 F_{int} (1, 25) = 0.18, p = .679
WHO-5 Well-Being Index	T0 T1	10.29 (5.84) 12.29 (5.30)	11.38 (4.81) 13.69 (5.75)	**F_t (1, 25) = 4.70, p = .040** F_g (1, 25) = 0.46, p = .504 F_{int} (1, 25) = 0.02, p = .878

Significant effects are in bold print; F_t: main effect of time; F_g: main effect of group; F_{int}: time × group interaction effect; NFG: neurofeedback group; WLG: waiting list group. T0: pre-treatment; T1: post-treatment.

Secondary outcome measures. For perceived dieting success, a significant main effect of time (p = .029) with a trend towards significance (p = .091) in the group × time interaction was found. Descriptively, perceived dieting success was enhanced within the NFG (post hoc: p = .007) but not in the WLG. Still, group differences were not significant post-treatment and results have to be regarded critically due to a lack of significant interaction effect.

With regard to well-being there was a significant main effect of time only (p = .029) yielding descriptive, but non-significant improvements of well-being within both groups. For food craving and perceived stress no significant effects were observed in either analysis of the intervention effects.

NFG Follow-up. Short term stability of the neurofeedback effects was assessed at follow-up (T2). Changes in the primary outcome measures remained significant to follow-up (both main effects of time $p < .001$). From pre-treatment to follow-up, a significant and large reduction in overeating episodes was observable in post hoc comparisons ($p = .003$, $r = .59$). The same pattern was found for the reduction of distress induced by overeating ($p = .006$, $r = .56$).

For the secondary outcome measures, there still was an observable significant main effect of time for perceived dieting success ($p = .034$), although post hoc tests showed that significance vanished at follow-up (T0–T2: $p = .212$, $r = .32$). However, follow-up analysis now revealed a significant main effect of time for trait food craving ($p = .008$), caused by a significant reduction in food craving from pre-treatment to follow-up ($p = .008$) with a medium effect size ($r = .40$).

Table 3 Three month follow-up data for the neurofeedback group.

Variable	Time	NFG	Test statistics
n		12	
Primary outcome measures		*M (SD)*	
Overeating episodes / week	T0	4.17 (2.33)	**F_t (2, 22) = 19.72, p < .001**
	T1	0.67 (1.15)	
	T2	1.08 (1.24)	
Distress due to overeating	T0	3.08 (0.79)	**F_t (2, 22) = 17.46, p < .001**
	T1	0.83 (1.34)	
	T2	1.42 (1.31)	
Secondary outcome measures			
Food Craving Questionnaire	T0	135.67 (28.40)	**F_t (2, 22) = 6.05, p = .008**
	T1	118.33 (39.07)	
	T2	91.92 (36.34)	
Perceived Self-Regulatory success in Dieting Scale	T0	6.75 (4.27)	**F_t (2, 22) = 3.95, p = .034**
	T1	10.08 (2.50)	
	T2	10.08 (4.72)	
Perceived Stress Scale	T0	20.50 (7.13)	F_t (2, 22) = 1.46, p = .253
	T1	17.50 (7.35)	
	T2	18.08 (8.28)	
WHO-5 Well-Being Index	T0	11.08 (5.85)	F_t (2, 22) = 1.82, p = .186
	T1	13.08 (5.11)	
	T2	12.67 (6.26)	

Significant effects are in bold print; F_t : main effect of time; NFG: neurofeedback group. T0: pre-treatment; T1: post-treatment; T2: follow-up.

Subjective Outcomes

Overall acceptance of the neurofeedback was high. Altogether, 85.7 % of the participants rated the treatment experience as positive or very positive (positive: 71.4 %; very positive: 14.3 %). No single participant rated the treatment experience as negative or very negative. Satisfaction ratings were good, with 14.3 % rating satisfaction as very strong, 35.7 % as strong, whereas 35.7 % reported to be relatively satisfied.

Subjective feedback further indicated that 64.3 % of the participants would very strongly or strongly recommend the treatment to people experiencing overeating episodes. Additional results of subjective outcomes are displayed in Table 4. The only negative side effect stated by some participants was drowsiness during the sessions.

Table 4 Subjective outcomes of the neurofeedback training (n = 14).

Variable	*M*	*SD*
Overall evaluation	4.00	0.55
Specific evaluations		
Adequacy of the treatment	2.86	1.03
Satisfaction with the treatment	3.50	0.94
Profitability of the treatment	3.29	1.20
Applicability into daily routine	3.36	1.01
Recommendation	3.79	1.05
Subjectively perceived treatment effects on …		
Eating behavior	2.71	1.14
Occurrence of overeating episodes	3.14	1.10
Handling of overeating episodes	3.29	0.99
Perceived stress	2.64	1.22
Well-being	2.79	1.05
Changes in general behavior	3.00	1.11
Others' responses to changes in behavior	1.71	0.91

Range of answer scales: 1 = *very negative*; 5 = *very positive* for overall evaluation; 1 = *not at all;* 5 = *very strong* for all other items.

Discussion

Overeating episodes are a common problem within the population of REs. Stressful arousal, associated with craving and ruminative conflicts and its physiological correlates, might play a crucial role as antecedents of this eating behavior. For a randomized controlled pilot-study, we developed a ten session neurofeedback protocol, based on previous findings on EEG arousal, which combined cue exposure with subsequent down regulation of EEG high beta activity.

The present study demonstrated that this new training method accounted for significant improvement in overeating-related primary outcome measures. Overeating episodes were significantly reduced within the NFG only. At post-treatment, subjects in the NFG reported less frequent overeating episodes compared with a waiting list group. The same pattern was found for overeating induced distress.

These primary effects remained stable at a 3 month follow-up. Large effect sizes underline the relevance of the improvements induced by the training. Further, our outcome measures relate to actual (albeit retrospective) reports on eating behavior, rather than assessing attitudes towards food or latent constructs which are supposed to be related to eating behavior. Therefore, the present results provide high external validity.

Participants reported a positive evaluation of the treatment, with high acceptance, satisfaction, and recommendation rates, whereas the drop-out rates were relatively low. Thus, the neurofeedback protocol not only showed good efficacy but also provided a well-accepted approach for the treatment of overeating episodes in a sub-clinical sample of female REs. First evidence in this sample suggests that neurofeedback might help escape the vicious circle of stress and overeating by self-regulation of brainwave patterns, even in a low-threshold treatment consisting of ten sessions.

As secondary outcome, perceived dieting success was descriptively enhanced within the NFG, although group comparisons post treatment did not yield significant differences. The significant main effect on improvement of perceived dieting success was also observable in the follow-up sample. Still, pairwise comparisons between pre-treatment and follow-up did not yield statistical significance. In contrast, a significant reduction in food craving with a medium effect was observed within the NFG at follow-up.

This finding might be a result of the relatively small follow-up sample size, where punctual deviations might influence results to a strong degree. But it is also possible, that the treatment

first enhanced perceived dieting success by explicitly providing strategies against cue-induced food craving, while food craving in general was reduced in the long run by implicit transfer of these strategies. As a measure of self-regulatory competence, perceived dieting success is very specific and might not always be the predominant goal of women experiencing overeating episodes. Self-regulatory competence on more superordinate levels, for example general self-efficacy or self-regulatory competence regarding bodily responses, should therefore additionally be assessed in future studies on this neurofeedback protocol.

Other secondary outcome measures, such as perceived stress and general well-being were not significantly affected by the treatment, although they descriptively showed slight improvements within the NFG. Still, the latter outcome measures are relatively general and influenced by a wide range of factors besides overeating episodes, for example by general life circumstances, interpersonal relationships, or individual working load. Since we did not assess this range of possible influence factors, we cannot control for intervening effects.

Despite of high acceptance ratings, some women reported drowsiness throughout the sessions. In contrast to other neuro- or biofeedback protocols, the sessions included no breaks and relatively long and repetitive phases of relaxation, which might have been too monotonous or demanding for the participants. In future studies, relaxation periods should either be shortened or training sessions should be interrupted by short breaks.

To our knowledge, this is the first study evaluating a cue exposure neurofeedback paradigm to address overeating episodes in REs. Strengths of this study lie in the availability of follow-up data, showing stability of primary outcome effects and developments in secondary outcomes. In contrast to previous eating related biofeedback studies (Meule et al. 2012a; Teufel et al. 2013) the present sample included wide ranges in age, employment status, and normal weight as well as overweight participants. Although the results of this pilot study are promising, the study is subject to limitations.

First of all, due to technical limitations, we were not able to analyze EEG data in terms of psychophysiological learning. Therefore, it remains unclear whether participants have learned to regulate EEG activity, especially in the absence of the feedback signal. Besides the neurofeedback itself, the treatment contained multiple components, such as repeated cue exposure and some relaxation instructions. Both aspects have previously shown to exert beneficial effects in comparable intervention studies (Conklin and Tiffany 2002; Jansen et al. 1992; Manzoni et al. 2009).

To determine neurofeedback effects on spontaneous EEG and participants' control over EEG parameters, more sophisticated psychophysiological measurements and an experimental assessment of pre- and post-treatment EEG data in the absence of feedback should be included in following studies.

We did not compare the neurofeedback to an alternative treatment. Since other researchers have shown efficacy of different treatment methods on food craving or eating-related self-efficacy in different populations (e.g. electrodermal biofeedback: Teufel et al. 2013; or heart rate variability training: Meule et al. 2012a), effects might also be accountable to biofeedback in general or even to the mere intervention experience based on the experience of self-control or expectation.

In future studies on this protocol, it could help to assess participants' initial expectations towards the treatment, to address their impact on outcome and resulting placebo effects implicitly. For a more detailed assessment of specific neurofeedback effects, a consecutive study is planned, comparing the developed protocol with a highly comparable treatment, for example an imagery-based relaxation training combined with cue exposure.

Sample sizes in this study were relatively small, accounting for limited statistical power despite of significant and large effects. Future studies on this protocol should aim at recruiting a larger sample to account for sufficient statistical power. As women are more prone than men to dysfunctional eating and overeating episodes (Provencher et al. 2003), we tested the new method on an exclusively female sub-clinical sample. To assess general effectiveness in terms of overeating symptoms, the method should also be tested in a male sample reporting overeating episodes.

Further, the applicability and effectiveness in a clinical sample is yet an unexplored but interesting topic and a field of application for future trials. Finally, future studies on this topic should control for individual characteristics, influencing overeating and treatment effectiveness, such as impulsivity, perfectionism, body-dissatisfaction, affect (Stice 2002), or flexible and rigid eating behavior (Westenhöfer 1991).

The results of this pilot-study need to be confirmed and the distinct beneficial influences of the neurofeedback method yet are to be scrutinized. Still, our first results are promising. Neurofeedback with a high beta reduction protocol is an approach that should be considered in future intervention research against overeating episodes in REs, and might probably also be helpful in other populations prone to food craving, disinhibition and overeating.

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Compliance with ethical standards

Conflict-of-interest-statement The authors declare no conflict of interest.

References

Andersen, S. B., Moore, R. A., Venables, L., & Corr, P. J. (2009). Electrophysiological correlates of anxious rumination. *International Journal of Psychophysiology, 71*(2), 156-169.

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.

Brunstrom, J. M., Yates, H. M., & Witcomb, G. L. (2004). Dietary restraint and heightened reactivity to food. *Physiology & Behavior, 81*(1), 85-90.

Cepeda-Benito, A., Gleaves, D. H., Williams, T. L., & Erath, S. A. (2000). The development and validation of the state and trait Food-Cravings Questionnaires. *Behavior Therapy, 31*(1), 151-173.

Cohen, S., & Williamson, G. (1988). Perceived stress in a probability sample of the United States. In: S. Spacapan & S. Oskamp (Eds.). *The social psychology of health* (pp. 31-67). Newbury Park: Sage.

Cohen, S., Kamarck, T., & Mermelstein, R. (1983). A global measure of perceived stress. *Journal of Health and Social Behavior*, 385-396.

Conklin, C. A., & Tiffany, S. T. (2002). Applying extinction research and theory to cue-exposure addiction treatments. *Addiction, 97*(2), 155-167.

Coolican, H. (2009). *Research methods and statistics in psycho*logy. London, United Kingdom: Hodder.

Cools, J., Schotte, D. E., & McNally, R. J. (1992). Emotional arousal and overeating in restrained eaters. *Journal of Abnormal Psychology, 101*(2), 348-351.

De Wit, M., Pouwer, F., Gemke, R. J., Delemarre-van de Waal, H. A., & Snoek, F. J. (2007). Validation of the WHO-5 Well-Being Index in adolescents with type 1 diabetes. *Diabetes Care, 30*(8), 2003-2006.

Dinkel, A., Berth, H., Exner, C., Rief, W., & Balck, F. (2005). Deutsche Adaptation der Restraint Scale zur Erfassung gezügelten Essverhaltens. *Diagnostica, 51*(2), 67-74.

Egner, T., & Gruzelier, J. H. (2001). Learned self-regulation of EEG frequency components affects attention and event-related brain potentials in humans. *NeuroReport, 12*(18), 4155-4159.

Egner, T., & Gruzelier, J. H. (2004). EEG biofeedback of low beta band components: Frequency-specific effects on variables of attention and event-related brain potentials. *Clinical Neurophysiology, 115*(1), 131-139.

Fairburn, C. G., & Beglin, S. J. (1994). Assessment of eating disorders. Interview or self-report questionnaire? *International Journal of Eating Disorders, 16*(4), 363-370.

Fishbach, A., Friedman, R. S., & Kruglanski, A. W. (2003). Leading us not unto temptation. Momentary allurements elicit overriding goal activation. *Journal of Personality and Social Psychology, 84*(2), 296-309.

Frank, S., Lee, S., Preissl, H., Schultes, B., Birbaumer, N., & Veit, R. (2012). The obese brain athlete: Self-regulation of the anterior insula in adiposity. *PloS one, 7*(8), e42570.

Freeman, L. M. Y., & Gil, K. M. (2004). Daily stress, coping, and dietary restraint in binge eating. *International Journal of Eating Disorders, 36*(2), 204-212

Fritz, C. O., Morris, P. E., & Richler, J. J. (2012). Effect size estimates: Current use, calculations, and interpretation. *Journal of Experimental Psychology: General, 141*(1), 2-18.

Gluck, M. E. (2006). Stress response and binge eating disorder. *Appetite, 46*(1), 26-30.

Hay, P., & Williams, S. E. (2013). Exploring relationships over time between psychological distress, perceived stress, life events and immature defense style on disordered eating pathology. *BMC Psychology, 1*, 27, doi:10.1186/2050-7283-1-27.

Heatheron, T. F., Polivy, J., & Herman, C. P. (1991). Restraint, weight loss, and variability of body weight. *Journal of Abnormal Psychology, 100*(1), 78-83.

Herman, C. P., & Mack, D. (1975). Restrained and unrestrained eating. *Journal of Personality, 43*(4), 647-660.

Herman, C. P., & Polivy, J. (1975). Anxiety, restraint, and eating behavior. *Journal of Abnormal Psychology, 84*(6), 666-672.

Herman, C. P., & Polivy, J. (1980). Restrained Eating. In A. J. Stunkard (Ed.), *Obesity.* (pp. 208-225). Philadelphia, PA: Saunders.

Heun, R., Bonsignore, M., Barkow, K., & Jessen, F. (2001). Validity of the five-item WHO Well-Being Index (WHO-5) in an elderly population. *European Archives of Psychiatry and Clinical Neuroscience, 251*(2), 27-31.

Hilbert, A., & Tuschen-Caffier, B. (2006). *Eating Disorder Examination. Deutschsprachige Übersetzung*. Münster: Verlag für Psychotherapie, PAG Institut für Psychologie AG.

Hill, A. J. (2007). The psychology of food craving. *Proceedings of the Nutrition Society, 66*(02), 277–285.

Jansen, A., Broekmate, J., & Heymans, M. (1992). Cue-exposure vs self-control in the treatment of binge eating: a pilot study. *Behaviour Research and Therapy, 30*(3), 235-241.

Jasper, H. H. (1958). The ten twenty electrode system of the international federation. *Electroencephalography and Clinical Neurophysiology, 10*, 371-375.

Jastreboff, A. M., Sinha, R., Lacadie, C., Small, D. M., Sherwin, R. S., & Potenza, M. N. (2013). Neural correlates of stress- and food cue–induced food craving in obesity Association with insulin levels. *Diabetes Care, 36*(2), 394-402.

Klesges, R. C., Isbell, T. R., & Klesges, L. M. (1992). Relationship between dietary restraint, energy intake, physical activity, and body weight: A prospective analysis. *Journal of Abnormal Psychology, 101*(4), 668-674.

Manzoni, G. M., Pagnini, F., Gorini, A., Preziosa, A., Castelnuovo, G., Molinari, E., & Riva, G. (2009). Can relaxation training reduce emotional eating in women with obesity? An exploratory study with 3 months of follow-up. *Journal of the American Dietetic Association, 109*(8), 1427-1432.

McEwen, B. S. (2007). Physiology and neurobiology of stress and adaptation: Central role of the brain. *Physiological Reviews, 87*(3), 873-904.

Meule, A., Freund, R., Skirde, A. K., Vögele, C., & Kübler, A. (2012a). Heart rate variability biofeedback reduces food cravings in high food cravers. *Applied Psychophysiology and Biofeedback, 37*(4), 241-251.

Meule, A., Lutz, A., Vögele, C., & Kübler, A. (2012b). Food cravings discriminate differentially between successful and unsuccessful dieters and non-dieters. Validation of the Food Cravings Questionnaires in German. *Appetite, 58*(1), 88-97.

Meule, A., Papies, E. K., & Kübler, A. (2012c). Differentiating between successful and unsuccessful dieters. Validity and reliability of the Perceived Self-Regulatory Success in Dieting Scale. *Appetite, 58*(3), 822-826.

Mitchell, A. M., Crane, P. A., & Kim, Y. (2008). Perceived stress in survivors of suicide: Psychometric properties of the perceived stress scale. *Research in Nursing & Health, 31*(6), 576-585

Mitchison, D., Hay, P., Slewa-Younan, S., & Mond, J. (2012). Time trends in population prevalence of eating disorder behaviors and their relationship to quality of life. *PLoS ONE 7*(11): e48450. doi:10.1371/journal.pone.0048450

Mond, J. M., Hay, P. J., Rodgers, B., & Owen, C. (2006). Eating Disorder Examination Questionnaire: Norms for young adult women. *Behavior Research and Therapy, 44*(1), 53-62.

Mond, J. M., Hay, P. J., Rodgers, B., Owen, C., & Beumont, P. J. V. (2004). Validity of the Eating Disorder Examination Questionnaire (EDE-Q) in screening for eating disorders in community samples. *Behaviour Research and Therapy, 42*(5), 551-567.

Paquette, V., Beauregard, M., & Beaulieu-Prévost, D. (2009). Effect of a psychoneurotherapy on brain electromagnetic tomography in individuals with major depressive disorder. *Psychiatry Research: Neuroimaging, 174*(3), 231-239.

Parvaz, M. A., Alia-Klein, N., Woicik, P. A., Volkow, N. D., & Goldstein, R. Z. (2011). Neuroimaging for drug addiction and related behaviors. *Reviews in the Neurosciences, 22*(6), 609-624.

Pelchat, M. L. (2002). Of human bondage: Food craving, obsession, compulsion, and addiction. *Physiology & Behavior, 76*(3), 347-352.

Polivy, J., & Herman, C.P. (1985). Dieting and binging. A causal analysis. *American Psychologist, 40*(2), 193-201.

Polivy, J., Coleman, J., & Herman, C. P. (2005). The effect of deprivation on food cravings and eating behavior in restrained and unrestrained eaters. *International Journal of Eating Disorders, 38*(4), 301-309.

Provencher, V., Drapeau, V., Tremblay, A., Després, J. P., & Lemieux, S. (2003). Eating behaviors and indexes of body composition in men and women from the Quebec family study. *Obesity Research, 11*(6), 783-792.

Psychiatric Research Unit (1998). WHO (Fünf) – Fragebogen zum Wohlbefinden (Version 1998): Assessed: Sep 11th 2013 http://www.cure4you.dk/354/WHO-5_German.pdf

Reis, R. S., Hino, A. A. F., & Añez, C. R. R. (2010). Perceived Stress Scale reliability and validity study in Brazil. *Journal of Health Psychology*, *15*(1), 107-114.

Ruderman, A. J. (1986). Dietary restraint: A theoretical and empirical review. *Psychological Bulletin*, *99*(2), 247-261.

Saletu-Zyhlarz, G. M., Arnold, O., Anderer, P., Oberndorfer, S., Walter, H., Lesch, O. M., Böning, J., & Saletu, B. (2004). Differences in brain function between relapsing and abstaining alcohol-dependent patients evaluated by EEG mapping. *Alcohol & Alcoholism, 39*(3), 233-240.

Seo, S.-H., & Lee, J.-T. (2010). Stress and EEG. Convergence and Hybrid Information Technologies, M. Crisan (Ed.), *InTech*, Available from: http://www.intechopen.com/books/convergence-and-hybrid-information-technologies/stress-and-eeg

Sinha, R., & Jastreboff, A. M. (2013). Stress as a common risk factor for obesity and addiction. *Biological Psychiatry*, *73*(9), 827-835.

Stein, R. I., Kenardy, J., Wiseman, C. V., Dounchis, J. Z., Arnow, B. A., & Wilfley, D. E. (2007). What's driving the binge in binge eating disorder?: A prospective examination of precursors and consequences. *International Journal of Eating Disorders, 40*(3), 195-203.

Stice, E. (2002). Risk and maintenance factors for eating pathology: a meta-analytic review. *Psychological Bulletin*, *128*(5), 825-848.

Styn, M. A., Bovbjerg, D. H., Lipsky, S., & Erblich, J. (2013). Cue-induced cigarette and food craving: A common effect? *Addictive Behaviors, 38*(3), 1840-1843.

Swinburn, B. A., Sacks, G., Hall, K. D., McPherson, K., Finegood, D. T., Moodie, M. L., & Gortmaker, S. L. (2011). The global obesity pandemic: Shaped by global drivers and local environments. *The Lancet, 378*(9793), 804-814.

Tammela, L. I., Pääkkönen, A., Karhunen, L. J., Karhu, J., Uusitupa, M. I., & Kuikka, J. T. (2010). Brain electrical activity during food presentation in obese binge-eating women. *Clinical Physiology and Functional Imaging, 30*(2), 135-140.

Teufel, M., Stephan, K., Kowalski, A., Käsberger, S., Enck, P., Zipfel, S., & Giel, K. E. (2013). Impact of biofeedback on self-efficacy and stress reduction in obesity: A randomized controlled pilot study. *Applied Psychophysiology and Biofeedback, 38*(3), 177-184.

Thompson, M., & Thompson, L. (2007). Neurofeedback for stress management. In P. M. Lehrer, R. L. Woolfolk & W.E. Sime (eds.), *Principles and practice of stress management. 3rd Ed.* (pp. 249-287), New York: The Guilford Press.

Vernon, D. J. (2005). Can neurofeedback training enhance performance? An evaluation of the evidence with implications for future research. *Applied Psychophysiology and Biofeedback*, *30*(4), 347-364.

Westenhöfer, J. (1991). Dietary restraint and disinhibition: Is restraint a homogeneous construct? *Appetite, 16*(1), 45-55.

5. Study 2: Specific efficacy of neurofeedback for disinhibited eating

5.1 Citation and author contributions

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Authors: Jennifer Schmidt & Alexandra Martin

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For enhanced readability and congruency with the published article, tables and figures are included in the main text and the manuscript layout has been adjusted to the journal style (e.g., citation style, use of British English, style of legends).

Author contributions:

- The study was designed by Jennifer Schmidt with supervision of Alexandra Martin.
- The study was organized and conducted by Jennifer Schmidt.
- The manuscript was written by Jennifer Schmidt.
- Participants were recruited by Jennifer Schmidt.
- Data were analyzed and interpreted by Jennifer Schmidt with intellectual input and advice by Alexandra Martin.
- The revisions of the manuscript were conducted by Jennifer Schmidt with intellectual input and advice by Alexandra Martin.

Further assistance:

- Rooms and equipment for conducting the treatments and information sessions were provided by Prof. Dr. Ralf Stürmer (*psyrecon research & consulting GmbH*, Wuppertal).
- Neurofeedback supervision was provided by Prof. Dr. Ralf Stürmer and Gisela Ulmer.
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- Training sessions were partly conducted by Jenny Bullerjahn, Dilek Soysal, Ruth Schmitz, Corinna Vollmert, Nicole Bias, and Victoria Strothmann.
- Assessments and data entry were assisted by Jenny Bullerjahn, Dilek Soysal, Ruth Schmitz, Corinna Vollmert, Nicole Bias, Victoria Strothmann, and Katharina Behncke.

5.2 Published manuscript

Neurofeedback against Binge Eating: A Randomized Controlled Trial in a female subclinical threshold sample

Jennifer Schmidt, Alexandra Martin

Abstract

Brain-directed treatment techniques, such as neurofeedback, have recently been proposed as adjuncts in the treatment of eating disorders to improve therapeutic outcomes. In line with this recommendation, a cue exposure EEG-neurofeedback protocol was developed. The present study aimed at the evaluation of the specific efficacy of neurofeedback to reduce subjective binge eating in a female subthreshold sample. A total of 75 subjects were randomized to EEG-neurofeedback, mental imagery with a comparable treatment set-up or a waitlist group. At post-treatment, only EEG-neurofeedback led to a reduced frequency of binge eating (p = .015, g = 0.65). The effects remained stable to a 3-month follow-up. EEG-neurofeedback further showed particular beneficial effects on perceived stress and dietary self-efficacy. Differences in outcomes did not arise from divergent treatment expectations. Because EEG-neurofeedback showed a specific efficacy, it may be a promising brain-directed approach that should be tested as a treatment adjunct in clinical groups with binge eating.

Keywords binge eating, neurofeedback, overeating, food craving, randomized controlled trial

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Introduction

Binge eating, defined as eating a large amount of food in a short period, accompanied by a sense of loss of control over eating (American Psychiatric Association, 2013), is a central feature of eating disorders, such as binge eating disorder and bulimia nervosa. Binge eating episodes – with consumption of either subjectively or objectively large amounts of food – are highly prevalent among women with disordered eating (Hay et al., 2012), obese individuals (de Zwaan, 2001) and even in nonclinical community samples (French, Jeffery, Sherwood, & Neumark-Sztainer, 1999). Prevalence rates range between 9% (French et al., 1999) in female community samples and up to 55% in obese women (Coker, von Lojewski, Luscombe, & Abraham, 2015).

Binge eating is one causal factor of distress, weight and shape concerns and impaired mood or even depression (Hay et al., 2012; Isnard et al., 2003). It further contributes to weight gain and thus to the increasing prevalence of overweight and obesity (Fairburn, Cooper, Doll, Norman, & O'Connor, 2000), being one potential cause of further health-related consequences associated with the observed epidemic of overweight and obesity (James, 2008; Swinburn et al., 2011). Therefore, the reduction of binge eating behaviours is an important goal in the treatment of disordered eating.

Although several psychological treatments have been established to address binge eating and related eating disorders (e.g. Iacovino, Gredysa, Altman, & Wilfley, 2012), researchers highlighted that evidence for therapeutic success is moderate (Brownley, Berkman, Sedway, Lohr, & Bulik, 2007) and remission rates are not as high as desired (Wilson, Grilo, & Vitousek, 2007). In 2013, Schmidt and Campbell pointed out how brain-directed treatments may have the potential to enhance therapeutic effects in the treatment of eating disorders. Here, the use of neurotechnologies was identified as a promising adjunct to existing therapies or even as a single treatment option (Schmidt & Campbell, 2013). One established method among the brain-directed neurotechnological approaches is neurofeedback.

In the 1960s, neurofeedback emerged as a treatment to provide individuals with real-time feedback on brain activity associated with dysfunctional states of mind or behaviours (Thibault, Lifshitz, & Raz, 2016; Vernon et al., 2003). Based on this feedback, the patient shall identify individual strategies that may enable him or her to voluntarily regulate dysfunctional physiological states (Siniatchkin, Kropp, & Gerber, 2000; Vernon, 2005). These strategies are then established by learning processes based on operant and classical conditioning, with brain

activity in desired ranges being rewarded, for example by use of success scores or animations (Sherlin et al., 2011; Strehl, 2014).

The term neurofeedback now refers to various brain imaging-based treatment techniques following this paradigm, for example electroencephalography (EEG) or real-time functional magnetic resonance imaging, with EEG-neurofeedback being the most widely used technique (Thibault, Lifshitz, Birbaumer, & Raz, 2015). Despite some methodological controversies (see Thibault et al., 2015; Thibault et al., 2016), neurofeedback has received increasing attention among researchers and practitioners throughout the past decades.

The potential of neurofeedback in the treatment of eating disorders was reviewed by Bartholdy, Musiat, Campbell, and Schmidt (2013), concluding that EEG-neurofeedback may be of use in the treatment of some eating disorders. Ease of use, flexibility and low running costs of EEG-neurofeedback systems, in contrast to other neuroimaging methods, are valid arguments for EEG-neurofeedback (Niv, 2013). However, studies on EEG-neurofeedback in the treatment of eating disorders are very rare (Bartholdy et al., 2013).

For applications of this technique, it is essential to identify patterns in the spontaneous EEG that are associated with relevant behaviours or psychological processes (Abarbanel, 1995; Gruzelier, 2014). These patterns are then modified by means of neurofeedback first, in order to change the biological mechanism of dysfunctional psychological and behavioural processes (Vernon, 2005).

With regard to dysfunctional eating behaviours, such as binge eating, research has provided some evidence for potentially relevant EEG spectral activity. Binge eating was previously described as a compulsive, addiction-like behaviour (e.g. Davis, 2013; Smith & Robbins, 2013), sharing neural circuits with drug cravings and consumption (Filbey, Myers, & DeWitt, 2012; Sinha & Jastreboff, 2013). Waters, Hill, and Waller (2001) pointed out how cue-induced food craving and negative aroused affective states (e.g. stress) can promote binge eating. Thus, addiction-and stress-related EEG research may help to derive suitable neurofeedback protocols.

Both states, cue-induced craving and stressful arousal, showed to co-occur with EEG activity in the fast spectral ranges, that is higher EEG beta activity (~20–30 Hz) (Parvaz, Alia-Klein, Woicik, Volkow, & Goldstein, 2011; Seo & Lee, 2010; Thompson & Thompson, 2007). In an analysis of particular EEG correlates of dysfunctional eating behaviour, Tammela et al. (2010) found EEG beta activity to be associated with disinhibition in female binge eaters. Similarly, Hume, Howells, Rauch, Kroff, and Lambert (2015) showed excess EEG beta activity

after food cue exposure in overweight women. Thus, states of tense arousal marked by elevated EEG beta activity may contribute to binge eating behaviours.

With respect to EEG beta activity, the spectrum can be divided into lower-frequency ranges (13–20 Hz) and EEG high beta activity (>22–30 Hz). Lower ranges are usually upregulated in neurofeedback because of an association with attention and cognitive performance (e.g. Gruzelier, 2014). The EEG high beta activity is associated with the aforementioned states of tense arousal and is therefore downregulated in neurofeedback protocols (e.g. Egner & Gruzelier, 2001; Keith, Rapgay, Theodore, Schwartz, & Ross, 2015; Walker, 2011).

Against this background, a neurofeedback protocol based on reductions of EEG high beta activity after cue exposure with craved foods may constitute a promising approach to use this brain-directed method in the treatment of binge eating.

In a pilot study (n = 27), we examined the general efficacy of neurofeedback with an inhibition of EEG high beta activity in restrained eaters (Schmidt & Martin, 2015). We found evidence for neurofeedback to reduce overeating episodes compared with a waitlist group, showing stability to a 3-month follow-up. Overall the observed effects were very promising.

Therefore, the present study aimed to further improve the neurofeedback treatment and to evaluate its effects in a randomized controlled trial with a larger sample size. To determine the specific efficacy of the neurofeedback, an additional alternative treatment group was established in the study (cue exposure with mental imagery; Kemps & Tiggemann, 2007; Knäuper, Pillay, Lacaille, McCollam, & Kelso, 2011). Both groups were then contrasted with a waitlist control group to analyze differential efficacy.

The target was a reduction in subjective binge eating episodes, marked by loss of control and preceded by food craving urges. This behaviour occurs even in subclinical groups (French et al., 1999). Here, the loss-of-control experience is associated with impaired self-regulatory control (Manasse et al., 2014) and considered a central aspect in the development of eating pathology (Colles, Dixon, & O'Brien, 2008). The target group of the present study was composed of women who reported regular subjective binge eating episodes, recruited from a community sample of restrained eaters (Herman & Mack, 1975), a group that is considered especially prone to binge eating (Polivy & Herman, 1985; Stice, 2002).

Because of the objective regulation of physiological mechanisms that are associated with or even precede dysfunctional consumption behaviours, we hypothesized that neurofeedback – but not mental imagery – would significantly reduce subjective binge eating episodes compared

with the waitlist. Considering that disordered eating is associated with psychological distress (e.g. Hay et al., 2012), we hypothesized that distress due to binge eating would also be alleviated through neurofeedback.

We further investigated possible differential treatment expectations as well as effects on secondary outcomes that are prominent antecedents of binge eating (Freeman & Gil, 2004; Gluck, 2006; Waters et al., 2001): food craving and perceived stress. Beyond these, given the importance of self-efficacy in the regulation of eating behaviour in general (Glasofer et al., 2013) and binge eating in particular (Goodrick et al., 1999; Wolff & Clark, 2001), we examined treatment effects on domain-specific (dietary and somatic) self-efficacy.

Materials and methods

Study design

Two cue exposure treatments, with either neurofeedback or mental imagery after exposure with food cues, were compared with a waitlist, assessing data pre- and post-treatment and at a 3-month follow-up. The participants did not receive any allowance except for the treatment participation being free of charge. Informed consent was obtained from all participants. The ethics committee of the University of Wuppertal approved the study.

Sample

For inclusion, the participants were required to be female, report regular occurrence of subjective binge eating episodes and report restrained eating with values ≥12 on the Restraint Scale (RS; German version: Dinkel, Berth, Exner, Rief, & Balck, 2005). Exclusion criteria were: (i) former diagnoses of eating disorders; (ii) body mass index (BMI) < 20 kg/m^2; (iii) alcohol abuse; (iv) diabetes mellitus with insulin therapy; (v) neurological disorders (e.g. epilepsy); (vi) severe mental disorders; (vii) medication that potentially elicits weight fluctuations (e.g. thyroxine or cortisone); (viii) pregnancy; and (ix) a current weight loss diet other than long-term lifestyle diets (e.g. vegetarianism or low-carb nutrition).

Given that the intervention protocol still was at an evaluation stage, we decided to exclude subjects with clinical eating disorders at this research phase. To screen for potentially undiagnosed eating disorders, the Eating Disorder Examination Questionnaire (EDE-Q; Hilbert & Tuschen-Caffier, 2006) was applied. After diagnostic prescreening, a trained clinical

psychologist interviewed the participants with critical EDE-Q values (overall score > 4; Mond, Hay, Rodgers, & Owen, 2006) to exclude women with eating disorders from the study.

The required sample size was determined by using G*POWER 3 (Faul, Erdfelder, Lang, & Buchner, 2007) via a priori tests for ANCOVA with three groups. We referred to a large effect size, as observed in the pilot study (Schmidt & Martin, 2015), with $\alpha = .05$ and $1\text{-}\beta = .80$. Calculations resulted in a required sample size of $n = 64$. In anticipation of dropouts, a margin of 17% (according to dropout rates in the pilot study) was added, resulting in a recruitment target of $n = 75$.

Altogether, 123 respondents were assessed for eligibility. Seven persons did not meet inclusion criteria; 10 women met exclusion criteria. A total of 31 subjects dropped out before randomization (for reasons, see Figure 1). Eligible participants ($n = 75$) were randomly assigned to either neurofeedback, mental imagery or a waitlist. Randomized allocation to groups was performed by an external and blinded person, according to a computer-generated list following an equal distribution ($n = 25$ per group).

Throughout the intervention period, $n = 7$ women dropped out from neurofeedback, $n = 6$ from mental imagery and $n = 3$ from the waitlist. Two participants were excluded from analyses due to heavy outliers (see the section ‘Statistical analyses’), resulting in an analysis sample of $n = 18$ for neurofeedback and mental imagery respectively and $n = 21$ for the waitlist. Women in the waitlist were randomly assigned to treatments after the waiting period but were not included in analyses. For details on participant flow, see the Consolidated Standards of Reporting Trials (CONSORT) diagram (Figure 1).

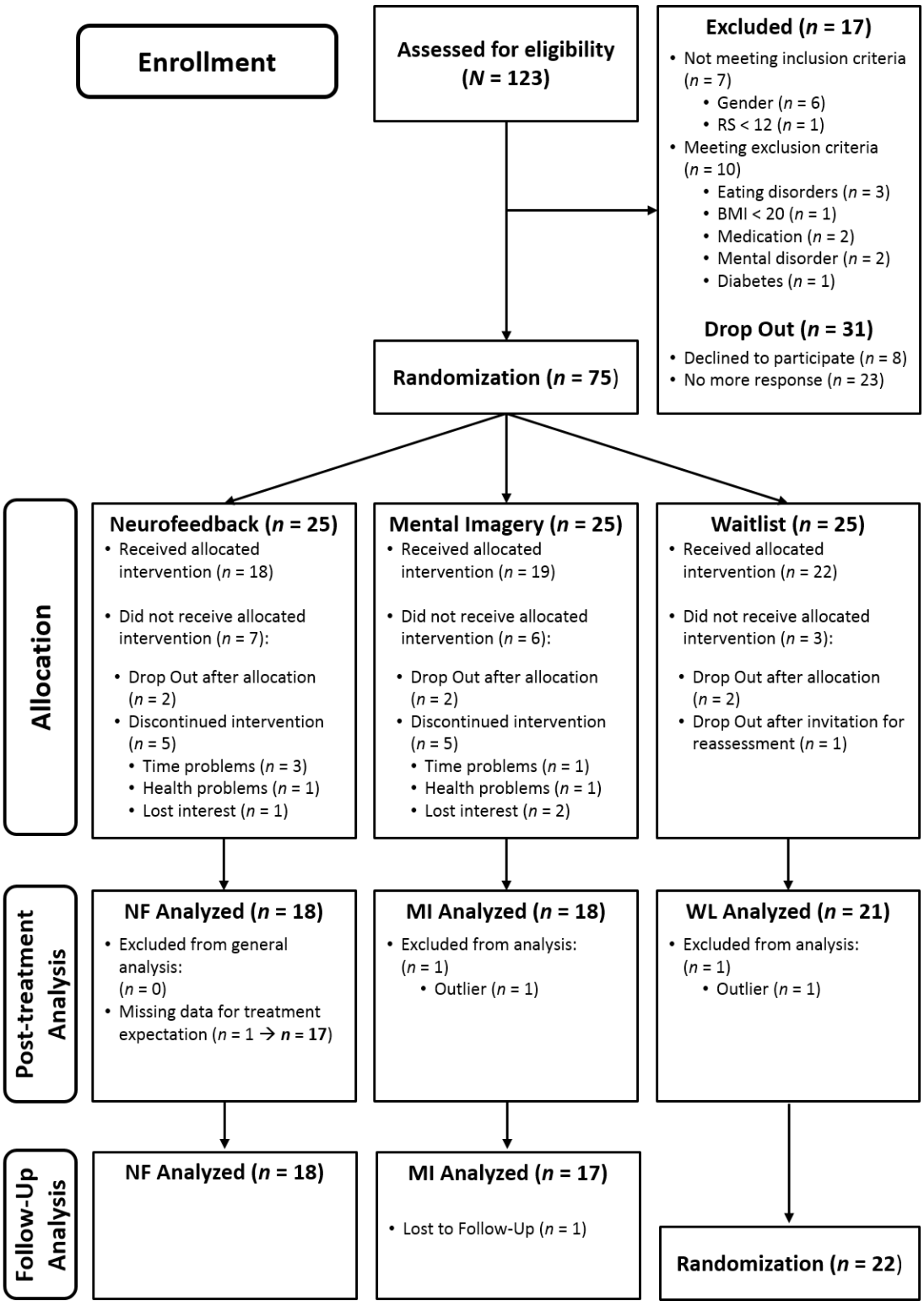

Figure 1. Patient flow according to CONSORT guidelines. RS, restraint score; BMI, body mass index; NF, neurofeedback; MI, mental imagery; WL, waitlist

Procedure

For recruitment, the study was announced in the local media, through the website and the newsletter of the university and with flyers in medical practices.

After an online screening for inclusion and exclusion criteria, eligible women received written information and were invited to an information session. In this session, the participants were pro-vided details on the study procedures, treatment methods and randomization. They filled in a questionnaire, containing baseline assessment of outcome measures, demographics and the EDE-Q. Finally, the session concluded with a brief psychoeducation regarding general guidelines for healthy nutrition according to the German Nutritional Society. Because the participants' awareness of the experimenters' hypotheses might influence study outcomes (Nichols & Maner, 2008), we took care not to reveal to the participants any hypotheses regarding the possible superiority of a certain treatment.

Recruitment and all sessions were conducted between March and October 2014. Training rooms and equipment were provided by psyrecon GmbH (Wuppertal, Germany). In pre- and post-treatment sessions, each participant attended a psychophysiological experiment with cue exposure while skin conductance, cardiovascular responses, and electrical brain activity were recorded (results will be reported elsewhere). Follow-up data were collected 3 months after the treatment, in a personal session or via online questionnaire.

Treatments

Trainers. A trained psychologist (JS) and six female research assistants, who received extensive training regarding set-up and application of neurofeedback and mental imagery, conducted the sessions. Instructions and session protocols were conveyed based on standardized treatment manuals adapted from an earlier study (neurofeedback: Schmidt & Martin, 2015) or developed for the present study with reference to previous research (mental imagery: Kemps & Tiggemann, 2007; Knäuper et al., 2011).

General treatment set-up. All participants attended 10 individual sessions with repeated food cue exposure and subsequent self-regulation tasks, with two sessions in weeks 1–4 and one session in weeks 5 and 6. All sessions were conducted in a calm room, where the participants were seated in a comfortable armchair at a 1-m distance from a 22-inch monitor that presented treatment animations.

For both treatments, each session consisted of an initial adaptation phase (180 seconds) and 10 cue exposure phases (30 seconds) alternating with 10 self-regulation phases incorporating the treatment tasks (neurofeedback or mental imagery, 120 seconds per phase) and concluded with a relaxation phase (180 seconds). For cue exposure, the participants were provided with pictures of 10 individually appealing foods that are regular subjects of binge eating episodes.

Stimuli were presented as presentation cards, which the trainer put in front of the participant in each cue exposure phase. The participants were instructed to imagine the foods as vividly as possible, including taste and smell. The presentation cards were then removed for self-regulation phases. To ensure sufficient appeal of the food cues, all women abstained from eating 3 hours prior to the sessions.

At the beginning of each session, the trainers encouraged the participants to report experiences, such as successes or difficulties to apply the methods at home. In the fifth session, small trigger cards were handed to the participants, displaying screenshots of the client monitors as viewed in the training sessions. These cards could be used for transfer purposes, serving as cues to apply the acquired self-regulation strategies whenever food craving or urges to binge would occur.

Neurofeedback. The present neurofeedback protocol is based on the instruction to reduce EEG high beta activity (23–28 Hz). Feedback on brain activity was presented as bar diagrams on a client screen that indicated the amount of EEG high beta activity as well as possible artefacts emerging from muscle tension, as EEG high beta activity is influenced by these artefacts because of an overlap in frequency ranges (Muthukumaraswamy, 2013). Both bars should be kept below preset thresholds. When the thresholds were exceeded, the bar diagrams turned red and a displayed animation of a beach landscape at sunset stopped.

The trainers informed the participants how movements or facial expressions induce muscular artefacts in the EEG and how to avoid those by choosing a comfortable seating position and relaxing facial muscles (Muthukumaraswamy, 2013). Higher power muscular activity (≥ 1 μV) was indicated by the artefact bar. The trainers additionally monitored EEG curves to inform the participants in case of minor muscle artefacts.

The participants were instructed to reduce EEG high beta activity by trying different self-regulation strategies. Except for the prerequisite of open eyes, the choice of strategies was individual and free, as advocated for neurofeedback treatments (Siniatchkin et al., 2000). The

participants reported the use of various self-regulation strategies, such as meditation techniques, thoughts of pleasant situations and use of mantras.

The EEG signal was acquired via a Mindfield (Berlin, Germany) MindMaster EEG system and corresponding software (BIOERA CLINICAL BASIC 1.63) from the vertex position (Cz). Neurofeedback preparation (scalp peeling and electrode placement) occurred according to standard procedures and in the exact same set-up as in the pilot study (detailed information see: Schmidt & Martin, 2015). The participants were instructed that the probabilities of success would be lowered throughout the training programme to maintain challenging training effects (sessions 1–4: 85%; sessions 5 and 6: 80%; sessions 7 and 8: 75%; sessions 9 and 10: 70%). Throughout the sessions, EEG activity was monitored and thresholds were adjusted by the trainers.

After 10 cue exposure phases and subsequent neurofeedback self-regulation, the relaxation period (180 seconds) completed the session. For this purpose, alpha activity (8–12 Hz), a spectral range frequently used in relaxation-based neurofeedback (Demos, 2005; Wang et al., 2013), was additionally displayed, with an instruction to upregulate alpha activity. The overall session duration was approximately 45 minutes.

Mental imagery. The general treatment set-up of the mental imagery training corresponded to the neurofeedback set-up except for the lack of neurofeedback equipment and the task in self-regulation phases: the participants in the mental imagery group (MIG) were instructed to imagine pleasant and vivid imagery in self-regulation phases. The images used would consume visuospatial capacities and should be associated with a state of relaxation (Kemps & Tiggemann, 2007).

In the first session, the trainers encouraged the participants to explore the suitability of different imagery contents in terms of vividness, difficulty of retrieval, dominance of the impression and relaxation potential. Imagery should not be linked to food, and the imagination task should be operated with eyes open. The participants were then instructed to use the most vivid and lively image with a high potential to relax in self-regulation phases.

To deliver visual support for relaxation, an animated beach landscape at sunset was presented throughout the session on the monitor in front of the client, by using MS PowerPoint 2007. Each session closed with 180 seconds of relaxation without imagery instructions. Mental imagery sessions lasted approximately 35 minutes.

Assessment instruments

Screening instruments. Screening included questionnaires for inclusion and exclusion criteria, as well as an assessment of eating disorders.

Restraint Scale. The RS (German version: Dinkel et al., 2005) consists of 10 items assessing dieting concerns and weight fluctuations. Answer options were provided on Likert scales. Good psychometric properties and validity have been reported (Dinkel et al., 2005).

Inclusion and exclusion criteria. The screening questionnaire contained items to assess inclusion and exclusion criteria, age, gender, weight and height (for BMI calculation). Furthermore, current dieting status, possible pregnancy and medication, as well as histories of eating disorders, alcohol abuse, neurological and mental disorders and diabetes, were assessed in a *yes/no* format with an additional box to specify the status in case of yes answers.

Eating Disorder Examination Questionnaire. The German EDE-Q (Hilbert & Tuschen-Caffier, 2006) assesses specific eating disorder symptoms, eating and shape-related thoughts and behaviours. For an overall score, 22 items (7-point rating scale: 0 = *attribute non-existent*; 6 = *attribute existent every day/in an extreme degree*) are summed up to mean scores. Good psychometric properties are documented; internal consistency is excellent with $\alpha = .97$ (Hilbert & Tuschen-Caffier, 2006).

Demographics. In the information session, age, employment status and facts on special diets (e.g. vegetarianism/veganism) were assessed via paper–pencil questionnaire.

Primary outcome measures. Subjective binge eating episodes and resulting distress were retrospectively determined on a self-report questionnaire (Schmidt & Martin, 2015). After an initial definition (subjective binge eating episodes defined as unwanted consumption of high-calorie food preceded by craving but not by physiological hunger), the number of such episodes during the last 7 days should be provided. For validation, the frequency of pretreatment binge eating episodes was correlated with respective EDE-Q values (items on ‘loss of control-eating’ and ‘objective binge eating’). A significant correlation was found with EDE-Q loss of control-eating, $r = .29$, $p = .030$, indicating the validity of the measure in assessing subjective binge eating (Palavras, Morgan, Borges, Claudino, & Hay, 2013). Distress experienced due to subjective binge eating was assessed on a 6-point rating scale (0 = *not at all*; 5 = *very strong*).

Secondary outcome measures. Measures on psychological constructs related to subjective binge eating episodes served as secondary outcomes: food craving, perceived stress and self-efficacy.

Food craving. We applied the German version of the Food Cravings Questionnaire-Trait in the reduced 15-item form (Meule, Hermann & Kübler, 2014a). It captures habits and behaviours related to food craving on 6-point answer scales (1 = *never/not applicable*; 6 = *always*). Internal consistency is reported to range between α = .90 and .93. Food craving is assessed as a trait, but the questionnaire is reportedly sensitive to episodic changes (Meule et al., 2014b). In the instruction, we adapted the period from in general to throughout the last week to allow for comparability with primary outcome measure.

Stress. The Perceived Stress Questionnaire (German version: Fliege, Rose, Arck, Levenstein, & Klapp, 2001) contains 20 items (e.g. 'You feel tense') with 4-point rating scales (1 = *almost never*; 4 = *most of the time*). In the present study, the instructions were altered to a time frame of the last week. Sum scores were used as indicators of overall stress. Resulting values are standardized between 0 and 1, with values closer to 1 indicating higher perceived stress. Good psychometric properties have been reported, with α = .85 (Fliege et al., 2001).

Self-efficacy. Two different aspects of domain specific self-efficacy were assessed in this study: dietary self-efficacy and somatic self-efficacy.

Dietary self-efficacy was measured by using the Perceived Self-Regulatory Success in Dieting Scale (Meule, Papies, & Kübler, 2012). It consists of three items with a 7-point rating scale (1 = *not successful/not difficult*; 7 = *very successful/very difficult*).

The scale has recently been discussed as a measure of dietary self-efficacy rather than actual regulatory success (Haynes, Kemps, Moffitt, & Mohr, 2014). For analyses, item scores are summed up. Higher values indicate higher dietary self-efficacy. Satisfactory internal consistency has been reported, α > .70 (Meule et al., 2012).

The scale on somatic self-efficacy consisted of five items, assessing the perceived ability to control bodily responses (e.g. 'I have a strong degree of control over my bodily reactions'; 'For me, it is easy to calm down, when I am upset') with 7-point rating scales (0 = *do not agree at all*; 6 = *fully agree*). Item scores are averaged, with higher values indicating higher somatic self-efficacy. In the present study, the internal consistency was acceptable, α = .70.

Treatment expectations and evaluation. To assess possible differences in the credibility of the applied treatments (e.g. due to the prominent use of technical equipment in neurofeedback), the participants in the active groups were asked to rate treatment expectations and overall treatment evaluation. At baseline, the participants reported expectations on a three-item scale ('I trust this treatment'; 'This treatment is a valuable experience'; 'This treatment is promising to target binge eating episodes') with a 5-point answer scale (1 = *not at all*; 5 = *absolutely*). Internal consistency was high, $\alpha = .84$. Post-treatment, the overall treatment evaluation was assessed with the item 'Altogether, how did you experience the training?' by using a 5-point rating scale, 1 = *very negative*, 5 = *very positive* (Schmidt & Martin, 2015).

Statistical analyses

Statistical analyses were performed with SPSS 22. Single points of missing data were replaced by using the individual's mean for the respective scale. Tests of normality were conducted per group and assessment, by using Shapiro–Wilk tests. Parametric tests were applied whenever acceptable. If the data deviated from normality, the results were re-examined by bootstrapping techniques ($n = 1000$, CI = 95%). To avoid distortions in the relatively small sample, a box plot outlier analysis was performed regarding differences in primary outcome measures. If pre–post differences in weekly binge eating episodes deviated more than three standard deviations from group means, outliers were excluded from analyses ($n = 2$).

For a check of successful randomization, group differences in demographics and baseline outcome measures were examined by using chi-squared tests and univariate ANOVAs. To allow for comparisons of both intervention groups to the waitlist, post-treatment data were examined by means of ANCOVAs with baseline data as covariates, in line with recommendations for pre–post designs (Senn, 2006).

Results for weekly binge eating episodes were additionally reassessed by intent-to-treat analyses using ANCOVA. Here, the last available values before dropout were put forward, resulting in a sample size of $n = 75$ ($n = 25$ per group). Post hoc *t*-tests with Bonferroni corrections were applied. Hedges' *g* was used as effect size because of the unequal group sizes, calculated for post-treatment means compared with the waitlist group (WLG). Conventions for *g* are equivalent with those for effect size *d*, with a small effect for $g \geq 0.20$, a medium effect for $g \geq 0.50$ and a large effect for $g \geq 0.80$ (Cohen, 1988).

For primary outcomes in active intervention groups, the stability of treatment effects to follow-up was analyzed by 3 (session) × 2 (group) mixed ANOVAs and Bonferroni-corrected

post hoc tests. Subjective treatment expectation and post-treatment evaluation were compared between active treatment groups via independent samples *t*-test. Values of $p < .05$ were regarded as significant throughout all analyses.

Results

Demographics and baseline values

The sample ($n = 57$) showed a mean age of 44.77 years ($SD = 15.15$) and a mean BMI of 28.77 kg/m² ($SD = 5.47$ kg/m²). Further sample characteristics and test statistics on group differences are displayed in Table 1. At baseline, the groups did not differ in any outcome variable, which indicates successful randomization (see Table 2).

Table 1 Demographic and screening data.

Variable	Neuro-feedback		Mental Imagery		Waitlist		Total		Test statistics
n	18		18		21		57		
	M (*SD*)		*M* (*SD*)		*M* (*SD*)		*M* (*SD*)		
Age	45.72 (13.91)		43.72 (13.64)		44.86 (17.84)		44.77 (15.15)		F (2, 54) = 0.08, p = .927
BMI	28.24 (4.64)		27.81 (5.09)		30.06 (6.38)		28.77 (5.47)		F (2, 54) = 0.94, p = .397
Restraint score (Restraint Scale)	19.89 (4.46)		20.22 (4.04)		18.67 (3.68)		19.54 (4.04)		F (2, 54) = 0.81, p = .450
Eating pathology (EDE-Q total)	2.23 (0.85)		2.50 (1.09)		2.48 (0.77)		2.41 (0.90)		F (2, 54) = 0.49, p = .615
	n	%	*n*	%	*n*	%	*n*	%	
Employment									χ^2 (12) = 10.89, p = .588
student	2	11.1	2	11.1	3	14.3	7	12.3	
apprentice	0	0	1	5.6	1	4.8	2	3.5	
employee	10	55.6	10	55.6	9	42.9	29	50.9	
clerk	1	5.6	1	5.6	1	4.8	3	5.3	
self-employed	2	11.1	4	22.2	3	14.3	9	15.8	
retired	1	5.6	0	0	4	19.0	5	8.8	
unemployed	2	11.1	0	0	0	0	2	3.5	
Special diet									χ^2 (2) = 2.09, p = .380
yes	5	27.8	9	50.0	7	33.3	21	36.8	
no	13	72.2	9	50.0	14	66.7	36	63.2	

Treatment effects

Descriptive data and detailed test statistics for all analyses of treatment effects are displayed in Table 2.

Primary outcomes. Data on changes in primary outcomes for the neurofeedback group (NFG), the MIG and the WLG are displayed in Figure 2.

For weekly binge eating episodes and resulting distress, significant group main effects were observed. At post-treatment, binge eating episodes were significantly lower in the NFG compared with the WLG (p = .015, medium effect) but not in the MIG compared with the WLG (p = .139, small effect). Additional bootstrapping analyses confirmed the significant difference between NFG and WLG (p = .008), again without a significant effect between the MIG and WLG (trend only: p = .055).

In both active groups, distress resulting from binge eating was significantly lower than in the WLG at post-treatment, NFG versus WLG: p < .001, large effect; MIG *versus* WLG: p = .010, large effect. The results were confirmed in bootstrapping analyses (NFG *versus* WLG: p = .001; MIG *versus* WLG: p = .011).

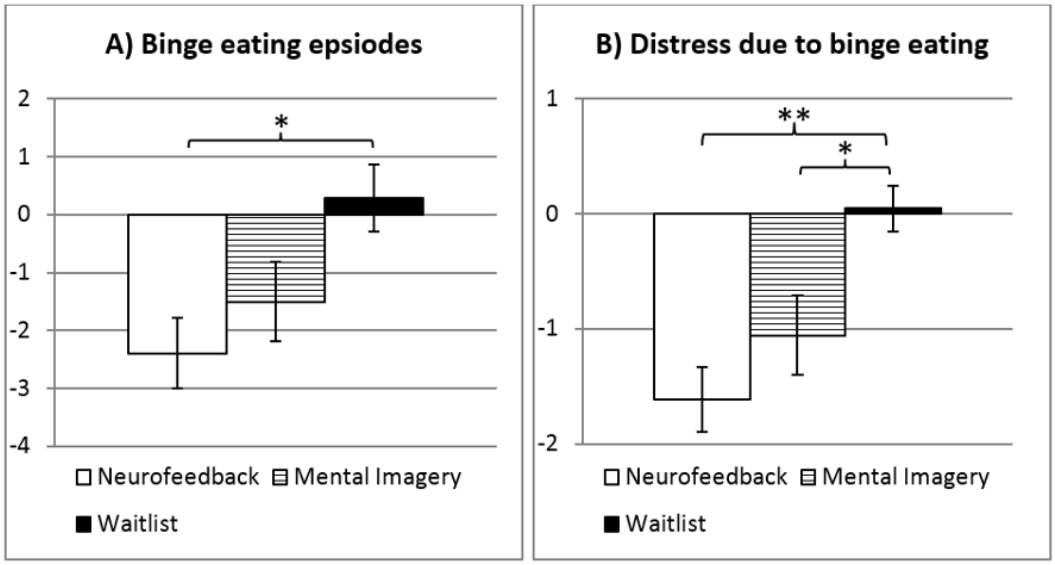

Figure 2. Changes in primary outcomes displaying (A) reduced frequency of binge eating episodes (reported per week) following neurofeedback and (B) reduced distress related to binge eating (original rating scale: 0 = *not at all*; 5 = *extremely*) in both treatment groups. Change scores are calculated as post-treatment means minus pre-treatment means. Error bars indicate standard errors. *p < .05; ** p < .01.

Table 2 Pre-post means and group differences in treatment outcomes

Outcome	Neurofeedback (n = 18)		Mental Imagery (n = 18)		Waitlist (n = 21)		Test statistics
	T0	T1	T0	T1	T0	T1	
Primary outcomes	*M* (*SD*)	*M* (*SD*)	*M* (*SD*)	*M* (*SD*)	*M* (*SD*)	*M* (*SD*)	
Binge eating episodes per week	5.00 (2.77)	2.61[a] (2.59)	4.56 (3.45)	3.06 (3.64)	4.23 (2.00)	4.52[a] (3.12)	T0: F (2, 54) = 0.37, p = .693 **T1: F (2, 53) = 4.57, p = .015** gs: NF = 0.65, MI = 0.43
Distress due to Binge eating	3.00 (0.59)	1.39[a] (1.09)	2.78 (1.06)	1.72[b] (1.49)	2.86 (0.85)	2.90[a,b] (1.09)	T0: F (2, 54) = 0.31, p = .734 **T1: F (2, 53) = 10.07, p < .001** gs: NF = 1.36, MI = 0.90
Secondary outcomes							
Food Craving	54.11 (12.25)	37.83[a] (11.00)	56.89 (9.83)	41.61[b] (13.36)	56.52 (15.50)	51.95[a,b] (14.26)	T0: F (2, 54) = 0.25, p = .780 **T1: F (2, 53) = 7.41, p = .001** gs: NF = 1.08, MI = 0.73
Perceived Stress	0.46 (0.21)	0.38 (0.17)	0.46 (0.18)	0.37 (0.21)	0.49 (0.20)	0.50 (0.21)	T0: F (2, 54) = 0.15, p = .860 **T1: F (2, 53) = 3.28, p = .045** gs: NF = 0.61, MI = 0.61
Dietary self-efficacy	8.33 (4.10)	11.61[a] (3.60)	8.89 (4.25)	10.22 (5.47)	7.95 (3.61)	8.10[a] (4.22)	T0: F (2, 54) = 0.27, p = .765 **T1: F (2, 53) = 3.25, p = .047** gs: NF = 0.87, MI = 0.43
Somatic self-efficacy	2.61 (0.84)	3.59[a] (0.81)	2.64 (1.07)	3.59[b] (0.92)	2.59 (1.02)	2.81[a,b] (1.24)	T0: F (2, 54) = 0.02, p = .985 **T1: F (2, 53) = 5.78, p = .005** gs: NF = 0.72, MI = 0.69

T0: Baseline values, T1: Postintervention values; NF, neurofeedback; MI, mental imagery; T0 statistics: univariate ANOVA results; T1 statistics: ANCOVA results with T0 values as covariates. Bold entries indicate statistical significance. Hedges' g values are calculated from post-treatment values compared with the waitlist group. Conventions for Hedges' g: $g \geq 0.20$ - small effect; $g \geq 0.50$ - medium effect; $g \geq 0.80$ - large effect. Superscripts a, b: Bonferroni-corrected post-hoc tests significant at $p < .05$.

In intent-to-treat analyses (n = 75), a comparable pattern of results was observed, although the group main effect was only marginally significant for binge eating episodes, $F(2, 72) = 2.88$, $p = .063$. Again, the NFG showed less frequent binge eating episodes post-treatment, while the MIG did not (NFG *versus* WLG: $p = .022$, $g = 0.67$; MIG *versus* WLG: $p = .136$, $g = 0.34$).

Treatment effects were stable at follow-up. Compared with baseline, both the frequency of binge eating episodes (follow-up values: NFG: $M = 3.0$, $SD = 3.0$; MIG: $M = 2.2$; $SD = 3.9$) and the resulting distress (follow-up values: NFG: $M = 1.8$, $SD = 1.2$; MIG: $M = 1.8$; $SD = 1.8$) were lowered in the two active groups. In ANOVA, we observed a significant main effect of

session for binge eating episodes, $F(2, 66) = 12.70$, $p < .001$, and significant baseline to follow-up post hoc tests in both active groups (ps < .018). The same pattern was observed for distress due to binge eating, with a significant main effect of session, $F(2, 66) = 19.67$, $p < .001$, and significant baseline to follow-up post hoc tests (ps < .017). Group main effects and interactions were not significant in any analysis.

Secondary outcomes.

Food craving. Food craving was reduced in both treatment groups. At post-treatment, food craving was significantly lower in the NFG compared with the WLG ($p = .002$, bootstrap: $p = .003$, large effect) and in the MIG compared with the WLG ($p = .015$, bootstrap: $p = .011$, medium effect).

Stress. Perceived stress improved in both treatment groups. A significant main effect of group was observed, yet pairwise post hoc comparisons were not significant (both ps > .05, medium effects). However, bootstrapping analyses yielded a significant post-treatment difference for the NFG compared with the WLG ($p = .037$) but not for the MIG compared with the WLG (trend only: $p = .052$).

Self-efficacy. For domain-specific dietary self-efficacy, we observed a significant group main effect at post-treatment. Post hoc tests showed that only the NFG showed significantly higher dietary self-efficacy than the WLG ($p = .041$, large effect), whereas MIG and WLG did not differ ($p = .632$, small effect).

Regarding domain-specific somatic self-efficacy, there was a significant group main effect, with both active groups showing significant post-treatment differences compared with the WLG (NFG *versus* WLG: $p = .014$, bootstrap: $p = .013$, medium effect; MIG *versus* WLG: $p = .018$, bootstrap: $p = .013$, medium effect).

Subjective treatment expectations and evaluation. At baseline, treatment expectations were equally high for both interventions, NFG: $M = 4.4$ ($SD = 0.6$); MIG: $M = 4.4$ ($SD = 0.6$), and did not differ, $t(33) = 0.07$, $p = .943$. Both treatments received positive evaluations, NFG: $M = 4.2$ ($SD = 0.7$); MIG: $M = 3.9$ ($SD = 0.8$). Evaluation ratings did not differ between groups, $t(34) = 1.34$, $p = .189$. No harms or unintended side effects were reported or observed.

Discussion

The present randomized controlled trial was designed to further evaluate the efficacy of neurofeedback in the treatment of dysfunctional eating behaviours. Specifically, we aimed at investigating the effects of neurofeedback on subjective binge eating in a subclinical threshold sample.

In a previous pilot study (Schmidt & Martin, 2015), a cue exposure neurofeedback based on a reduction of EEG high beta activity after food cue exposure was efficacious in reducing overeating episodes compared with a waitlist group. Therefore, in the present study we were now interested in detecting possible specific effects of this brain-directed treatment method on subjective binge eating by contrasting the EEG-neurofeedback and an alternative cue exposure treatment with mental imagery to a waitlist group.

Post-treatment, neurofeedback, but not mental imagery, accounted for a reduced number of weekly subjective binge eating episodes compared with a waitlist condition. This pattern of results was confirmed in an intent-to-treat analysis, supporting our hypotheses on the specific efficacy of neurofeedback. Distress caused by binge eating improved in both treatments. The beneficial effects on primary outcomes were stable to a 3-month follow-up.

With this promising replication and first evidence of possible specific efficacy of neurofeedback in the treatment of subjective binge eating, it can be inferred that EEG-neurofeedback might in fact have a potential as an adjunct treatment option in clinical groups with eating disorders marked by binge eating, for example binge eating disorder or bulimia nervosa (Bartholdy et al., 2013).

We also found beneficial effects on secondary outcomes related to binge eating. Food craving, as a prominent antecedent of binge eating (Waters et al., 2001), was reduced in both active groups, yet with a higher post-treatment effect size for neurofeedback. Furthermore, both treatments led to enhanced somatic self-efficacy (i.e. relaxation abilities), which may in itself serve the purpose of preventing binge eating episodes due to stressful arousal (Freeman & Gil, 2004; Manzoni et al., 2009).

Only neurofeedback led to reductions in perceived stress compared with waitlist controls. This may be caused by the specific alteration in EEG high beta activity, which has been observed to co-occur with states of stressful arousal (Seo & Lee, 2010; Thompson & Thompson, 2007).

Furthermore, only participants in the neurofeedback condition showed enhanced domain-specific dietary self-efficacy at post-treatment. The enhancement of dietary (eating- and weight-related) self-efficacy has been identified as an important psychological mechanism in the treatment of binge eating (Goodrick et al., 1999; Wolff & Clark, 2001). Dietary self-efficacy is further related to lower body weight and less dysfunctional eating behaviours (Meule et al., 2012).

The self-regulatory focus of the neurofeedback together with its objective feedback on self-regulatory abilities after food cue exposure might have been responsible for improvements in dietary self-efficacy. Further, enhanced dietary self-efficacy may contribute to self-regulation capacities in eating behaviour and thus to less binge eating. Still, these assumptions are preliminary.

In the EEG experiment conducted as part of this study (see the section ‘Procedure’), we observed the first evidence of specific improvements in physiological self-regulation due to neurofeedback (results will be reported elsewhere: Schmidt & Martin, 2016). For detailed analyses of treatment mechanisms and the relationship between decreased EEG high beta activity and binge eating, larger samples would be necessary.

Neurofeedback is supposed to address correlates of dysfunctional states and behaviours. Yet relevant brain activity might manifest in complex patterns of spectral activity rather than single frequency ranges. Furthermore, changes in EEG correlates still do not account for causality. Here, experimental studies could help to understand the relationship between EEG activity in states of tense arousal and binge eating.

By now we can assume that the use of neurofeedback lead to specific improvements. Yet we cannot rule out whether unspecific treatment factors, rather than physiological changes, contributed to these result (Thibault et al., 2016).

The present study holds the advantage of assessing self-reported eating behaviour as a primary outcome instead of traits associated with eating, thus accounting for a certain degree of external validity. Furthermore, the availability of intermediate follow-up data can be considered a strength. Still, it would have been desirable to use additional objective measures of eating behaviour, because subjective reports can be subject to attitudinal changes, retrospective bias or social desirability (Taren et al., 1999). Future studies should address this challenge, for example by using ecological momentary assessment.

The presence of an alternative treatment control group is an asset of this study. The reported treatment expectations at the beginning of the intervention and subjective evaluations at the end did not differ between the two active groups. These results indicate that the participants did not hold differing views about the treatments and were not aware of experimental hypotheses (Nichols & Maner, 2008). Still, individuals may be subject to neuroenchantment (Ali, Lifshitz, & Raz, 2014), meaning that techniques using brain imaging appear overly powerful. For our study set-up, we cannot entirely rule out such an influence on the NFG.

The latter point also refers to an important limitation of our study: to determine the physiological efficacy of neurofeedback, it would be desirable to establish control groups receiving sham neurofeedback (Thibault et al., 2016). In sham neurofeedback, the same equipment and set-up is used, while the presented feedback does not correspond to the brain activity in target but to irrelevant physiological activity. Sham neurofeedback would be highly comparable and is an appropriate method to demonstrate the specificity of neurofeedback (Thibault et al., 2016). However, sham neurofeedback does also bear the risk of discouragement of participants' self-regulatory expectations and behaviours. In this case, demotivation of the participants is likely and may affect results (Gruzelier, 2014) or even produce nocebo effects (Colloca & Miller, 2011). According to the current stage of research, we decided for mental imagery as control condition.

Further limitations apply to our study: firstly, some cases dropped out before completing the assessments, which attenuated statistical power. Although bootstrapping and intent-to-treat analyses led to similar results for primary outcomes, replications in larger samples would still be desirable.

Secondly, while the present study confirms the beneficial effects of the general neurofeedback treatment, determination of the optimal treatment set-up needs more research on essential treatment components (e.g. optimal cue exposure set-up, self-regulation instructions, extensions of the protocol) and on mechanisms of change, such as psychophysiological learning and habituation (Niv, 2013; Strehl, 2014).

Lastly, we applied the cue exposure neurofeedback as a single treatment method against subjective binge eating. We did not incorporate any treatment components that would target weight loss, general eating behaviour or other important psychological aspects of disordered eating, such as body image (Cargill, Clark, Pera, Niaura, & Abrams, 1999; Legenbauer, Schütt-Strömel, Hiller, & Vocks, 2011) or dietary attitudes and beliefs (Stice, 2002). Outcomes might

be further improved when neurofeedback is used as an adjunct technique in combination with psychological therapies (Iacovino et al., 2012), targeting biological as well as cognitive mechanisms simultaneously.

In sum, we found further evidence for beneficial effects of cue exposure EEG-neurofeedback for subjective binge eating in a female subthreshold sample. Integrating the brain-directed neurofeedback method as an adjunct treatment for clinical groups with binge eating episodes might assist to solve the problem of low remission rates (Brownley et al., 2007) and moderate treatment effectiveness (Wilson et al., 2007) and may thus improve therapeutic outcomes in the treatment of eating disorders.

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References

Abarbanel, A. (1995). Gates, states, rhythms, and resonance: The scientific basis of neurofeedback training. *Journal of Neurotherapy, 1*(2), 15-38. 10.1300/J184v01n02_02

Ali, S. S., Lifshitz, M., & Raz, A. (2014). Empirical neuroenchantment: from reading minds to thinking critically. Frontiers in Human Neuroscience, 8: 357. 10.3389/fnhum.2014.00357

American Psychiatric Association (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Washington, DC: Author.

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. 10.1002/erv.2250

Brownley, K. A., Berkman, N. D., Sedway, J. A., Lohr, K. N., & Bulik, C. M. (2007). Binge eating disorder treatment: A systematic review of randomized controlled trials. *International Journal of Eating Disorders, 40*(4), 337–348. 10.1002/eat.20370

Cargill, B. R., Clark, M. M., Pera, V., Niaura, R. S., & Abrams, D. B. (1999). Binge eating, body image, depression, and self-efficacy in an obese clinical population. *Obesity Research, 7*(4), 379-386. 10.1002/j.1550-8528.1999.tb00421.x

Cohen, J. (1988). *Statistical Power Analysis for the Behavioral Sciences*, 2nd ed. Hillsdale, New Jersey: Erlbaum.

Coker, E. L., von Lojewski, A., Luscombe, G. M., & Abraham, S. F. (2015). The difficulty in defining binge eating in obese women: How it affects prevalence levels in presurgical bariatric patients. *Eating Behaviors, 17*, 130-135. 10.1016/j.eatbeh.2015.01.014

Colles, S. L., Dixon, J. B., & O'Brien, P. E. (2008). Loss of Control is central to psychological disturbance associated with Binge Eating Disorder. *Obesity, 16*(3) 608–614. 10.1038/oby.2007.99

Colloca, L., & Miller, F. G. (2011). The nocebo effect and its relevance for clinical practice. *Psychosomatic Medicine, 73*(7), 598-603. 10.1097/PSY.0b013e3182294a50

Davis, C. (2013). Compulsive overeating as an addictive behavior: overlap between food addiction and binge eating disorder. *Current Obesity Reports, 2*(2), 171-178. 10.1007/s13679-013-0049-8

de Zwaan, M. (2001). Binge eating disorder and obesity. *International Journal of Obesity, 25*(Suppl. 1), S51-S55.

Demos, J. N. (2005). *Getting started with neurofeedback*. New York: Norton & Company.

Dinkel, A., Berth, H., Exner, C., Rief, W., & Balck, F. (2005). Deutsche Adaptation der Restraint Scale zur Erfassung gezügelten Essverhaltens. *Diagnostica, 51*(2), 67-74. 10.1026/0012-1924.51.2.67.

Egner, T., & Gruzelier, J. H. (2001). Learned self-regulation of EEG frequency components affects attention and event-related brain potentials in humans. *NeuroReport, 12*(18), 4155-4159. 10.1097/00001756-200112210-00058

Fairburn, C. G., Cooper, Z., Doll, H. A., Norman, P., & O'Connor, M. (2000). The natural course of bulimia nervosa and binge eating disorder in young women. *Archives of General Psychiatry, 57*(7), 659-665. 10-1001/pubs.Arch Gen Psychiatry-ISSN-0003-990x-57-7-yoa9404.

Faul, F., Erdfelder, E., Lang, A.-G., & Buchner, A. (2007). G*Power 3: A flexible statistical power analysis program for the social, behavioral, and biomedical sciences. *Behavior Research Methods, 39*(2), 175-191.

Filbey, F. M., Myers, U. S., & DeWitt, S. (2012). Reward circuit function in high BMI individuals with compulsive overeating: similarities with addiction. *NeuroImage, 63*(4), 1800-1806. 10.1016/j.neuroimage.2012.08.073

Fliege, H., Rose, M., Arck, P., Levenstein, S., & Klapp, B. F. (2001). Validierung des "Perceived Stress Questionnaire"(PSQ) an einer deutschen Stichprobe. *Diagnostica, 47*(3), 142-152. 10.1026//0012-1924.47.3.142.

Freeman, L. M. Y., & Gil, K. M. (2004). Daily stress, coping, and dietary restraint in binge eating. *International Journal of Eating Disorders, 36*(2), 204-212. 10.1002/eat.20012

French, S. A., Jeffery, R. W., Sherwood, N. E., & Neumark-Sztainer, D. (1999). Prevalence and correlates of binge eating in a nonclinical sample of women enrolled in a weight gain prevention program. *International Journal of Obesity, 23*(6), 576-585.

Glasofer, D. R., Haaga, D. A., Hannallah, L., Field, S. E., Kozlosky, M., Reynolds, J., Janovski, J. A., & Tanofsky-Kraff, M. (2013). Self-efficacy beliefs and eating behavior in adolescent girls at-risk for excess weight gain and binge eating disorder. *International Journal of Eating Disorders, 46*(7), 663-668. 10.1002/eat.22160

Gluck, M. E. (2006). Stress response and binge eating disorder. *Appetite, 46*(1), 26-30. 10.1016/j.appet.2005.05.004

Goodrick, G. K., Pendleton, V. R., Kimball, K. T., Carlos Poston, W. S., Reeves, R. S., & Foreyt, J. P. (1999). Binge eating severity, self-concept, dieting self-efficacy and social support during treatment of binge eating disorder. *International Journal of Eating Disorders, 26*(3), 295-300. 10.1002/(SICI)1098-108X(199911)26:3<295::AID-EAT7>3.0.CO;2-7

Gruzelier, J. H. (2014). EEG-neurofeedback for optimising performance. III: A review of methodological and theoretical considerations. *Neuroscience & Biobehavioral Reviews, 44*, 159-182. 10.1016/j.neubiorev.2014.03.015

Hay, P. J., Buettner, P., Mond, J., Paxton, S. J., Quirk, F., & Rodgers, B. (2012). A community-based study of enduring eating features in young women. *Nutrients, 4*(5), 413-424. 10.3390/nu4050413

Haynes, A., Kemps, E., Moffitt, R., & Mohr, P. (2014). Resisting temptation of unhealthy food: interaction between temptation-elicited goal activation and self-control. *Motivation and Emotion, 38*(4), 485-495. 10.1007/s11031-014-9393-6

Herman, C. P., & Mack, D. (1975). Restrained and unrestrained eating. *Journal of Personality, 43*(4), 647-660. 10.1111/j.1467-6494.1975.tb00727.x

Hilbert, A., & Tuschen-Caffier, B. (2006). *Eating Disorder Examination-Questionnaire. Deutschsprachige Übersetzung*. Münster: Verlag für Psychotherapie, PAG Institut für Psychologie AG.

Hume, D. J., Howells, F. M., Rauch, H. L., Kroff, J., & Lambert, E. V. (2015). Electrophysiological indices of visual food cue-reactivity. Differences in obese, overweight and normal weight women. *Appetite, 85*(1), 126-137. 10.1016/j.appet.2014.11.012

Iacovino, J. M., Gredysa, D. M., Altman, M., & Wilfley, D. E. (2012). Psychological treatments for binge eating disorder. *Current Psychiatry Reports, 14*(4), 432-446. 10.1007/s11920-012-0277-8

Isnard, P., Michel, G., Frelut, M. L., Vila, G., Falissard, B., Naja, W., Navarro, J., & Mouren-Simeoni, M. C. (2003). Binge eating and psychopathology in severely obese adolescents. *International Journal of Eating Disorders, 34*(2), 235-243. 10.1002/eat.10178

James, W. P. T. (2008). WHO recognition of the global obesity epidemic. *International Journal of Obesity, 32*(Suppl. 7), S120-S126. 10.1038/ijo.2008.247

Keith, J. R., Rapgay, L., Theodore, D., Schwartz, J. M., & Ross, J. L. (2015). An assessment of an automated EEG biofeedback system for attention deficits in a substance use disorders residential treatment setting. *Psychology of Addictive Behaviors, 29*(1), 17-25. 10.1037/adb0000016

Kemps, E., & Tiggemann, M. (2007). Modality-specific imagery reduces cravings for food: an application of the elaborated intrusion theory of desire to food craving. *Journal of Experimental Psychology: Applied, 13*(2), 95-104. 10.1037/1076-898X.13.2.95

Knäuper, B., Pillay, R., Lacaille, J., McCollam, A., & Kelso, E. (2011). Replacing craving imagery with alternative pleasant imagery reduces craving intensity. *Appetite, 57*(1), 173-178. 10.1016/j.appet.2011.04.021

Legenbauer, T., Schütt-Strömel, S., Hiller, W., & Vocks, S. (2011). Predictors of improved eating behaviour following body image therapy: A pilot study. *European Eating Disorders Review, 19*(2), 129-137. 10.1002/erv.1017

Manasse S. M., Juarascio A. S., Forman E. M., Berner L. A., Butryn M. L., & Ruocco A. C. (2014), Executive Functioning in Overweight Individuals with and without Loss-of-Control Eating. *European Eating Disorders Review, 22*(5), 373-377. 10.1002/erv.2304

Manzoni, G. M., Pagnini, F., Gorini, A., Preziosa, A., Castelnuovo, G., Molinari, E., & Riva, G. (2009). Can relaxation training reduce emotional eating in women with obesity? An exploratory study with 3 months of follow-up. *Journal of the American Dietetic Association, 109*(8), 1427-1432. 10.1016/j.jada.2009.05.004

Meule, A., Beck Teran, C., Berker, J., Gründel, T., Mayerhofer, M., & Platte, P. (2014b). On the differentiation between trait and state food craving: Half-year retest-reliability of the *Food Cravings Questionnaire-Trait-reduced* (FCQ-T-r) and the *Food Cravings Questionnaire-State* (FCQ-S). *Journal of Eating Disorders, 2*(25). 10.1186/s40337-014-0025-z

Meule, A., Hermann, T., & Kübler, A. (2014a). A short version of the *Food Cravings Questionnaire – Trait*: The FCQ-T-reduced. *Frontiers in Psychology, 5:*190, 10.3389/fpsyg.2014.00190

Meule, A., Papies, E. K., & Kübler, A. (2012). Differentiating between successful and unsuccessful dieters. Validity and reliability of the Perceived Self-Regulatory Success in Dieting Scale. *Appetite, 58*(3), 822-826. 10.1016/j.appet.2012.01.028

Mond, J. M., Hay, P. J., Rodgers, B., & Owen, C. (2006). Eating Disorder Examination Questionnaire: Norms for young adult women. *Behaviour Research and Therapy, 44*(1), 53-62. 10.1016/j.brat.2004.12.003

Muthukumaraswamy, S. D. (2013). High-frequency brain activity and muscle artifacts in MEG/EEG: a review and recommendations. *Frontiers in Human Neuroscience, 7:* 138. 10.3389/fnhum.2013.00138

Nichols, A. L., & Maner, J. K. (2008). The good-subject effect: Investigating participant demand characteristics. *The Journal of General Psychology, 135*(2), 151-166. 10.3200/GENP.135.2.151-166

Niv, S. (2013). Clinical efficacy and potential mechanisms of neurofeedback. *Personality and Individual Differences, 54*(6), 676-686. 10.1016/j.paid.2012.11.037

Palavras, M. A., Morgan, C. M., Borges, F. M. B., Claudino, A. M., & Hay, P. J. (2013). An investigation of objective and subjective types of binge eating episodes in a clinical sample of people with co-morbid obesity. *Journal of Eating Disorders, 1*(26). 10.1186/2050-2974-1-26

Parvaz, M. A., Alia-Klein, N., Woicik, P. A., Volkow, N. D., & Goldstein, R. Z. (2011). Neuroimaging for drug addiction and related behaviors. *Reviews in the Neurosciences, 22*(6), 609-624. 10.1515/RNS.2011.055

Polivy, J., & Herman, C. P. (1985). Dieting and bingeing: A causal analysis. *American Psychologist, 40*(2), 193-201. 10.1037/0003-066X.40.2.193

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. 10.1007/s10484-015-9297-6

Schmidt, J. & Martin, A. (2016). Physiological vs. Psychological Learning in Neurofeedback against Binge Eating Episodes. Manuscript submitted for publication.

Schmidt, U., & Campbell, I. C. (2013). Treatment of Eating Disorders can not Remain 'Brainless': The Case for Brain-Directed Treatments. *European Eating Disorders Review, 21*(6), 425-427. 10.1002/erv.2257

Senn, S. (2006). Change from baseline and analysis of covariance revisited. *Statistics in Medicine, 25*(24), 4334-4344. 10.1002/sim.2682

Seo, S.-H., & Lee, J.-T. (2010). Stress and EEG. Convergence and hybrid information technologies. In: M. Crisan (Ed.), *InTech*, 10.5772/9651 http://www.intechopen.com/books/convergence-and-hybrid-information-technologies/stress-and-eeg Retrieved online: 13th January 2013

Sherlin, L. H., Arns, M., Lubar, J., Heinrich, H., Kerson, C., Strehl, U., & Sterman, M. B. (2011). Neurofeedback and basic learning theory: implications for research and practice. *Journal of Neurotherapy, 15*(4), 292-304. 10.1080/10874208.2011.623089

Sinha, R., & Jastreboff, A. M. (2013). Stress as a common risk factor for obesity and addiction. *Biological Psychiatry, 73*(9), 827-835. 10.1016/j.biopsych.2013.01.032

Siniatchkin, M., Kropp, P., & Gerber, W. D. (2000). Neurofeedback—The significance of reinforcement and the search for an appropriate strategy for the success of self-regulation. *Applied Psychophysiology and Biofeedback, 25*(3), 167-175. 10.1023/A:1009502808906

Smith, D. G., & Robbins, T. W. (2013). The neurobiological underpinnings of obesity and binge eating: a rationale for adopting the food addiction model. *Biological Psychiatry, 73*(9), 804-810. 10.1016/j.biopsych.2012.08.026

Stice, E. (2002). Risk and maintenance factors for eating pathology: a meta-analytic review. *Psychological Bulletin, 128*(5), 825-848. 10.1037/0033-2909.128.5.825

Strehl, U. (2014). What learning theories can teach us in designing neurofeedback treatments. *Frontiers in Human Neuroscience, 8:*894. 10.3389/fnhum.2014.00894

Swinburn, B. A., Sacks, G., Hall, K. D., McPherson, K., Finegood, D. T., Moodie, M. L., & Gortmaker, S. L. (2011). The global obesity pandemic: shaped by global drivers and local environments. *The Lancet, 378*(9793), 804-814. 10.1016/S0140-6736(11)60813-1

Tammela, L. I., Pääkkönen, A., Karhunen, L. J., Karhu, J., Uusitupa, M. I., & Kuikka, J. T. (2010). Brain electrical activity during food presentation in obese binge-eating women. *Clinical Physiology and Functional Imaging, 30*(2), 135-140. 10.1111/j.1475-097X.2009.00916.x

Taren, D. L., Tobar, M., Hill, A., Howell, W., Shisslak, C., Bell, I., & Ritenbaugh, C. (1999). The association of energy intake bias with psychological scores of women. *European Journal of Clinical Nutrition, 53*(7), 570-578. 10.1038/sj.ejcn.1600791

Thibault, R. T., Lifshitz, M., Birbaumer, N., & Raz, A. (2015). Neurofeedback, Self-Regulation, and Brain Imaging: Clinical Science and Fad in the Service of Mental Disorders. *Psychotherapy and Psychosomatics, 84*(4), 193-207. 10.1159/000371714

Thibault, R. T., Lifshitz, M., & Raz, A. (2016). The self-regulating brain and neurofeedback: Experimental science and clinical promise. *Cortex, 74*(1), 247-261. 10.1016/j.cortex.2015.10.024

Thompson, M., & Thompson, L. (2007). Neurofeedback for stress management. In P. M. Lehrer, R. L. Woolfolk & W.E. Sime (eds.), *Principles and practice of stress management. 3rd Ed.* (pp. 249-287), New York: The Guilford Press.

Vernon, D. J. (2005). Can neurofeedback training enhance performance? An evaluation of the evidence with implications for future research. *Applied Psychophysiology and Biofeedback, 30*(4), 347-364. 10.1007/s10484-005-8421-4

Vernon, D. J., Egner, T., Cooper, N., Compton, T., Neilands, C., Sheri, A., & Gruzelier, J. (2003). The effect of training distinct neurofeedback protocols on aspects of cognitive performance. *International Journal of Psychophysiology, 47*(1), 75-85. 10.1016/S0167-8760(02)00091-0

Walker, J. E. (2011). QEEG-guided neurofeedback for recurrent migraine headaches. *Clinical EEG and Neuroscience, 42*(1), 59-61. 10.1177/155005941104200112

Wang, S., Zhao, Y., Chen, S., Lin, G., Sun, P., & Wang, T. (2013). EEG biofeedback improves attentional bias in high trait anxiety individuals. *BMC Neuroscience, 14:*115. 10.1186/1471-2202-14-115

Waters, A., Hill, A., & Waller, G. (2001). Bulimics' responses to food cravings: is binge-eating a product of hunger or emotional state?. *Behaviour Research and Therapy, 39*(8), 877-886. 10.1016/S0005-7967(00)00059-0

Wilson, G. T., Grilo, C. M., & Vitousek, K. M. (2007). Psychological treatment of eating disorders. *American Psychologist, 62*(3), 199-216. 10.1037/0003-066X.62.3.199

Wolff, G. E., & Clark, M. M. (2001). Changes in eating self-efficacy and body image following cognitive–behavioral group therapy for binge eating disorder: A clinical study. *Eating Behaviors, 2*(2), 97-104. 10.1016/S1471-0153(01)00021-6

6. Study 3: Treatment mechanisms in neurofeedback for disinhibited eating

6.1 Status and author contributions

Title: Physiological vs. Psychological Learning in Neurofeedback against Binge Eating.

Authors: Jennifer Schmidt & Alexandra Martin

A version of this manuscript has been submitted for publication in: *Applied Psychophysiology and Biofeedback.*

For improved readability and congruence with the two published papers, tables and figures are included in the main text and the manuscript layout corresponds to the journal style of *Applied Psychophysiology and Biofeedback.*

Author contributions:

- The study was designed by Jennifer Schmidt with supervision of Alexandra Martin.
- The study was organized and conducted by Jennifer Schmidt.
- The manuscript was written by Jennifer Schmidt.
- EEG data were parameterized by Jennifer Schmidt with technical support (see below).
- Data were analyzed by Jennifer Schmidt.
- Data were interpreted by Jennifer Schmidt with intellectual input and advice by Alexandra Martin.

Further assistance:

- Rooms and equipment for conducting the experimental sessions and treatments were provided by Prof. Dr. Ralf Stürmer (*psyrecon research & consulting GmbH*, Wuppertal).
- Experimental sessions were partly conducted by Jenny Bullerjahn, Dilek Soysal, Ruth Schmitz, and Corinna Vollmert.
- Assessments and data entry were assisted by Jenny Bullerjahn, Dilek Soysal, Ruth Schmitz, Corinna Vollmert, Nicole Bias, and Victoria Strothmann.
- Technical support and assistance in EEG data parameterization were provided by Prof. Dr. Bertrand Massot (*INSA Lyon*, France) and Jan Hetzel (*psyrecon research & consulting GmbH*, Wuppertal).

6.2 Submitted manuscript

Physiological vs. Psychological Learning in Neurofeedback against Binge Eating

Jennifer Schmidt, Alexandra Martin

Abstract

In biofeedback research, the debate on physiological versus psychological learning has a long tradition. Yet, it is still or even more relevant today, regarding recent developments of biofeedback for behavior modification. Analyzing the particular impact of these learning mechanisms may help improving the protocols and answer the question, whether feedback of physiological functions is necessary to modify a target behavior. We explored the presence and impact of physiological versus psychological learning in a recently developed EEG neurofeedback protocol for binge eating. The protocol targets a reduction of food-cue induced cortical arousal through regulation of EEG high beta activity. A randomized controlled trial compared the efficacy of neurofeedback (n = 18) and a mental imagery treatment without physiological feedback (n = 18) with EEG measurements in experimental sessions pre- and post-treatment. Physiological learning in terms of EEG high beta reduction was observed in neurofeedback only. Post treatment, neurofeedback participants with successfully reduced binge eating episodes (≥ 50% reduction) showed lower EEG high beta activity than unsuccessful participants (p = .02). These results were specific for neurofeedback. Further, lower EEG high beta activity at post-treatment predicted fewer binge eating episodes in neurofeedback only. No comparable predictive effect was found for psychological learning in terms of somatic self-efficacy. Altogether, the study provides evidence for the presence and importance of physiological learning as a change mechanism in neurofeedback against binge eating. Reducing cortical arousal may improve dysfunctional eating behavior and corresponding neurofeedback techniques should therefore be considered in future treatments.

Keywords: Neurofeedback, Binge eating, Overeating, Treatment mechanisms, Electroencephalography

Introduction

"The spirit is willing, but the flesh is weak." This proverb is frequently quoted at dinner parties or cafeteria buffets – that is, in a context of unwanted food consumption. Although we often try to resist temptations of palatable food, various factors, like stress, emotions or repeated food exposure, regularly boycott these intentions (Adam and Epel 2007; Haedt-Matt and Keel 2011; Swinburn et al. 2011). Under these circumstances, many people report food craving, a loss of control and binge eating as a consequence (Boswell and Kober 2016; Stroebe et al. 2008).

Repeated occurrences of binge eating can result in weight gain (Dulloo and Montani 2015; Ozier et al. 2008) and may cause body dissatisfaction, distress, or depressive symptoms (Presnell et al. 2004; Skinner et al. 2012). Providing individuals with enhanced capabilities to control overwhelming bodily urges that lead to dysfunctional eating behaviors is therefore an important objective in treatments for obesity, eating disorders, but also for general health behavior change.

Biofeedback (BF) treatments are traditional and well-approved means with the goal to strengthen control over somatic activity (Epstein and Blanchard 1977). Here, psychophysiological recordings are used to provide patients with external feedback on patterns in their physiological activity (Bagdasaryan and Le Van Quyen, 2013; Schwartz, 1976). Through the implemented feedback, it is possible to enable a person to control bodily responses by reinforcing individual strategies that result in desired physiological changes (Shapiro et al. 1964; Siniatchkin et al. 2000).

Given the association between loss of control in dysfunctional eating and the focus on control-processes in BF, it is not surprising that BF applications have recently emerged as a promising technique to treat dysfunctional eating behaviors (Meule et al. 2012; Schmidt and Martin 2015; Teufel et al. 2013). Yet, while the main share of these BF studies found beneficial effects on different eating-related outcomes, one prominent old debate is yet not satisfactorily resolved in these novel BF-applications: Does BF work through specific physiological learning, enabling people to regulate dysfunctional physiological activity? Or do psychological effects (e.g., increased self-efficacy and subjective self-control) primarily account for beneficial outcomes in BF?

While physiological learning constitutes the central assumption of treatment mechanisms in BF (e.g., Schwartz 1976, Shapiro et al. 1964), the aforementioned psychological changes have become strongly advocated treatment mechanisms in this approach (Holroyd et al. 1984; Wickramasekera, 1999). Today, the analysis and comparison of *physiological* versus *psychological* learning in BF is still crucial to provide answers to the aforementioned questions and identify key treatment mechanisms, especially in novel applications and protocols (Gruzelier 2014b; La Vaque et al. 2002, Schwartz and Andrasik 2003).

A detailed look at eating-related BF studies shows, that evidence for relevant physiological changes is mixed and dependent on the physiological target parameter. Using heart rate variability (HRV) BF, Meule et al. (2012) did not observe significant changes in HRV, despite of beneficial treatment effects on food craving. Teufel and colleagues (2013) found an increase in eating-related self-efficacy and a reduction of sympathetic activity using electrodermal BF combined with food cue exposure.

In real time fMRI-BF, Frank et al. (2012) observed changes in obese participants' physiological regulatory abilities, yet without beneficial effects on eating behavior. To our best knowledge, no analyses are currently available for electroencephalographic (EEG) BF (i.e., neurofeedback: NF) applied to change dysfunctional eating behaviors (see: Bartholdy et al. 2013).

In two randomized controlled trials, we evaluated a ten session NF treatment to reduce overeating and binge eating episodes (Schmidt and Martin, 2015; Schmidt and Martin, 2016). The rationale of this approach is based on down-regulation of dysfunctional EEG high beta activity associated with states of tense arousal and craving or disinhibition (Parvaz et al. 2011; Tammela et al. 2010; Thompson and Thompson 2007). Both studies showed efficacy of NF in reducing overeating and binge eating. However, change mechanisms in this protocol still have to be explored.

Previous research in other application fields provided general evidence for EEG changes achieved by NF (Gruzelier 2014a). While EEG high beta activity has frequently been added as a supplementary spectral range to control hyperarousal (e.g., Egner and Gruzelier, 2001; Keith et al. 2015; Rostami et al. 2012), it has seldom been the main target of regulation in NF protocols. The few studies using EEG high beta as a target range found positive results regarding the presence and influence of physiological learning in this spectral range (Paquette et al. 2009; Zotev et al. 2014). Still, none of these studies targeted eating behavior, which

impedes a transfer of assumed treatment mechanisms to the novel NF protocol for dysfunctional eating behaviors.

The primary aim of the present study was, to explore, whether the NF protocol for dysfunctional eating specifically enables participants to regulate EEG high beta activity, and how physiological learning relates to treatment success in comparison to psychological learning regarding somatic self-efficacy.

To scrutinize the presence and contribution of physiological vs. psychological learning in NF against binge eating, we included a psychophysiological experiment in our second randomized controlled trial (Schmidt and Martin 2016). We assessed EEG high beta activity pre and post treatment of NF and a comparable treatment without a feedback component (mental imagery: MI). In contrast to the NF training itself, the experimental setup did not incorporate feedback. This allowed for an assessment of physiological self-regulation in the absence of feedback, which provides insights on the participants' ability to transfer the learned self-regulation strategies to everyday contexts (Sherlin et al. 2011). The following research questions are addressed:

Does the NF treatment result in physiological learning?

H1: NF (but not MI) reduces EEG high beta activity during self-regulation after cue exposure at post-treatment (compared to pre-treatment).

Does physiological learning differ between successful (≥ 50% binge eating reduction) and unsuccessful (< 50% binge eating reduction) participants in NF at post-treatment?

H2: At post-treatment, successful participants in NF (but not in MI) show lower levels of EEG high beta activity during self-regulation phases after cue exposure than unsuccessful participants.

Does physiological learning show stronger relations to post-treatment outcomes than psychological learning in NF?

H3: Post-treatment EEG high beta activity predicts binge eating episodes more strongly than subjective somatic self-efficacy in NF (but opposite in MI).

Method

Study design

The study is based on a 2 (session) × 2 (group) mixed within-between-subjects design. Data were obtained in a randomized controlled trial to examine the specific efficacy of a NF training to reduce binge eating episodes in female restrained eaters compared to two control groups (Schmidt and Martin 2016).

Only participants who completed one of the two ten session cue exposure treatments–either NF or the MI treatment–were subject to analyses in this study. We assessed EEG and self-report on binge eating episodes, as well as somatic self-efficacy prior to the first treatment session (T0) and after the treatment (T1). Prior to treatment, participants were informed on the experimental procedures, including randomization, physiological measurement, data handling, and treatment protocols. Informed consent was obtained from all individual participants included in the study. The ethics committee at the University of Wuppertal approved the research protocol.

Sample

The sample consisted of adult female participants, screened as restrained eaters (values ≥ 12; German version of the *Restraint Scale*, Dinkel et al. 2005) who reported regular occurrences of binge eating episodes. Women priory diagnosed with (or positively screened for) clinical eating disorders, insulin-dependent diabetes mellitus, any neurological or severe mental disorders were excluded from the study. Further exclusion criteria encompassed regular use of medication associated with weight fluctuations, alcohol dependency, pregnancy, and adherence to a time limited weight-loss diet (e.g., formula diets). We recruited participants with media reports and flyers in medical practices. Eligibility for participation was assessed via online questionnaire during recruitment.

A total of 123 persons were screened for eligibility, whereof 48 were either not eligible to participate (n = 17) or did not respond our invitation to an information session on the study (n = 31). A blinded and uninvolved person then randomly assigned the remaining 75 subjects to either NF treatment, MI treatment, or a waitlist group (n = 25 each). Throughout the first study phase, 16 women discontinued the study. Waitlist participants were randomly assigned to one of the two active treatments after an eight week waiting period (n = 11 each).

In the second study phase, *n* = 7 women dropped out. The resulting merged groups sample (*n* = 26 in NF and MI resp.) served as the target sample for the present EEG study. Here, some participants (*n* = 8 in NF and MI respectively) had to be excluded from statistical analyses due to storage problems, bad signal quality, or heavy artifacts in EEG recording, resulting in a final sample of *n* = 18 for each group (for participant flow, see Fig. 1).

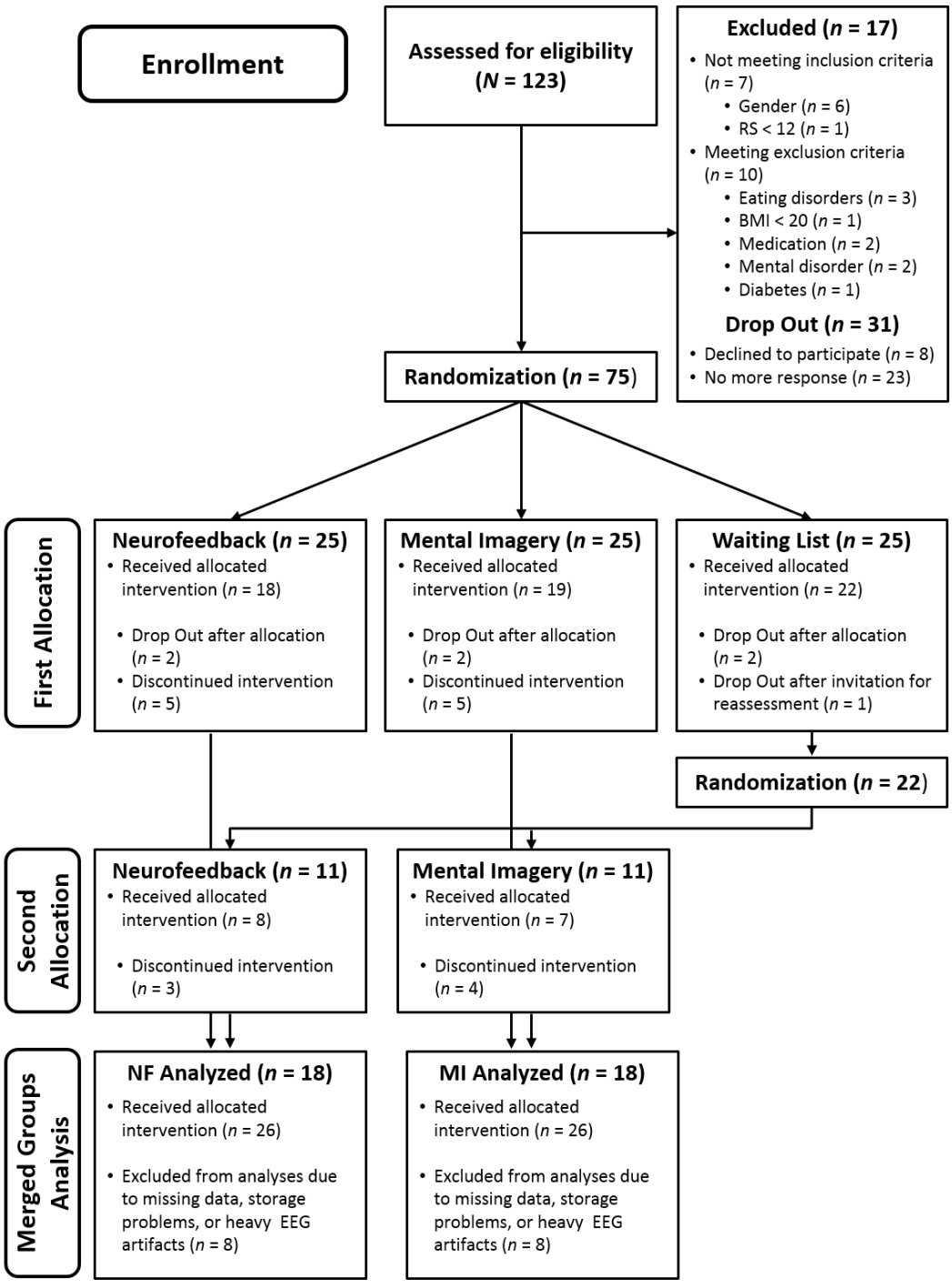

Fig. 1 Participant flow according to CONSORT guidelines

Procedure

We conducted experimental and treatment sessions between April and October 2014. Two calm and highly comparable training rooms, as well as the NF equipment and psychophysiological recording devices were provided by *psyrecon GmbH* (Wuppertal, Germany). According to the scope of this study, descriptions will focus on the experimental study and only give a brief overview on the treatments from T0 to T1. For a more detailed description of the treatment procedures, see Schmidt and Martin (2016).

Experimental sessions. At T0 and T1, all women attended the individual experimental sessions, including psychophysiological recording during food cue exposure and in subsequent self-regulation. They were asked not to eat for three hours prior to the sessions, to ensure appeal of the selected food cues. During the sessions, participants sat in a comfortable armchair in 1 m distance of a 22" flat screen. The screen displayed the experimental presentations by using standardized presentations, yet with personalized food cues in *MS PowerPoint*.

All women first filled in a questionnaire booklet containing the target instruments (see: *Assessment instruments*). Experimenters then attached EEG electrodes after corresponding preparation (see: *Physiological recording and analysis*). When signal quality was satisfactory, the presentation was started, displaying a standard instruction in black letters on a white background. Participants were informed about the procedure and duration of alternating cue exposure and self-regulation phases. They were instructed to avoid movements or speaking, and to keep their eyes open during the presentation.

For cue exposure phases (T0 & T1), participants had to imagine the displayed foods as vividly as possible, including smell, taste, and consistency, which has previously been evaluated as a successful strategy to induce craving (Sobik et al. 2005). During self-regulation phases at T0, participants should relax the way they would usually relax with open eyes; for self-regulation phases at T1, participants used the strategies learned in the treatments (NF or MI).

After the instruction, physiological recording started and was synchronized with the presentation. A baseline recording was performed for 120 s. Then, three alternating phases of cue exposure (30 s each) and self-regulation (120 s each) succeeded. In cue exposure phases, participants were confronted with three individually selected, appealing digital pictures of foods they regularly crave and binge on. These pictures were also used during the corresponding treatments. In self-regulation phases, an animated landscape of a beach at sunset was displayed,

which was also used in the treatment sessions. Altogether, the experimental sessions lasted 9.5 minutes and contained the same stimuli (pictures, animation) at T0 and T1.

Physiological recording and analysis. We obtained physiological data using the *Varioport Biosignal Recorder* (*Becker MediTec*) and the *Variograf* software. Besides EEG recording, galvanic skin response and heart rate were assessed for another research project. Results will be presented elsewhere. EEG was derived with an active and pre-amplified, unipolar 5-channel EEG device (Ag/AgCl electrodes) with reference and ground electrodes on the right and left mastoid. Recording sites were Cz, Fz, F3, F4, and Pz, according to the international 10-20 system (Jasper 1958). We used flexible EEG caps (*EasyCap*) to attach electrodes. Skin preparation was conducted with abrasive *One-Step EEG* peeling paste and 65% isopropyl alcohol to ensure satisfactory impedance levels. To retain skin contact and sufficient conductivity, we used *SuperVisc* (*EasyCap*) electrode paste for active EEG recordings.

After electrode attachment, the experimenter checked signal quality and adjusted electrodes whenever signals were not satisfying or impedance levels were too high. Additionally, for correction of ocular artifacts, we acquired a vertical electrooculogram (EOG) via a 2 mm Ag/AgCl electrode and conductivity enhancing electrode paste (*Electrode Cream, GE Medical Systems*) below the left eye. Analogue sampling rate was 1024 Hz. A 50 Hz notch-filter was included in the recording device. During experimental recordings, the experimenters monitored the signals and logged visible muscular artifacts or decreasing signal quality.

Analysis of EEG data was performed offline, using a *MatLab* based tool (programmed by Prof. Dr. Bertrand Massot, *INSA Lyon*), to perform Fast Fourier Transformation and obtain spectral power of the relevant EEG frequency ranges. The method implied bases on a shifting window over 10 s-segments without overlap throughout the course of the experimental session. It uses the Welch periodogram (Welch 1967) due to the advantage of being independent of predetermined window size. A rectangular window was applied to analyze spectral power in the whole range of EEG frequencies from 1 to 30 Hz. Correction of ocular artifacts was performed based on the EOG recordings, using principal component analysis (PCA) as the superior method for automatic corrections, avoiding spectral distortions (Wallstrom et al. 2004).

We exported calculated values (absolute Power, μV^2) as data sheets and screened them for artifacts logged during the sessions. This is especially important because the frequency range of interest, EEG high beta activity (23-28 Hz) may be influenced by muscular activity due to

overlapping frequency ranges (Muthukumaraswamy 2013). Values were then averaged for each electrode position and every separate 10 s-interval over the spectral ranges of interest (delta: 1-3 Hz; theta: 4-7 Hz; alpha: 8-12 Hz; sensorimotor rhythm [SMR]: 13-15 Hz; low beta: 16-22 Hz; high beta 23-28 Hz).

To calculate an EEG power indicator for statistical analyses of the current research questions, we determined mean values over all three 120 s self-regulation phases after cue exposure at T0 and T1. Whenever artifacts only affected single 10 s-intervals, values were replaced by mean values in the respective phase. Participants with more than five 10 s-intervals affected by artifacts (i.e., more than 15% of the recording) or those who showed decreasing signal quality during the sessions were excluded from analyses (n = 16).

Since absolute spectral power values in EEG recording can vary heavily between participants and between repeated measurements, relative spectral power was calculated dividing absolute power for each target frequency (e.g., high beta in μV^2) by the overall sum of absolute power of the spectral ranges (delta to high beta in μV^2). Decimal values (ranging from 0 to 1) were then transformed to percentages. Due to the potential effects of the NF training on baseline EEG activity from T0 to T1 (Gruzelier 2014b), we did not perform any baseline corrections to avoid neglecting those possible outcomes. Only values of the electrode site which was used as a training position in NF (Cz) will be reported in the present paper.

Treatments. Both treatments–NF and MI–consisted of ten sessions based on standardized treatment manuals. Each session began with a 180 s adaptation phase. Then participants in both groups were repeatedly exposed with individual pictures of foods which regularly induce craving and binge eating (ten exposures, 30 s each). Participants should imagine the foods as vividly as possible. Each exposure phase was followed by 120 s of the self-regulation task.

For the NF group, the self-regulation task was the down-regulation of EEG high beta activity (23-28 Hz), derived from a unipolar online EEG assessment (*Mindfield Mindmaster EEG*) at the vertex position (Cz), with reference and ground electrodes on the earlobes. Feedback on EEG high beta activity was displayed as bar diagrams to be kept below a threshold as well as through a beach landscape animation. Activity below thresholds was rewarded (green bar, fluent animation); Activity surpassing thresholds was inhibited (red bar, stopping animation). Participants could try different strategies for self-regulation and should pursue the most rewarded. Trainers adjusted thresholds according to predefined success rates, which were reduced over the training course (stepwise: 85% to 70%). After ten exposure phases, alpha

activity (8-12 Hz) was additionally displayed for 180 s to be up-regulated for relaxation purposes. Each session lasted approximately 45 min, including preparation.

For the MI group, participants were made familiar with the mental imagery approach (Kemps and Tiggemann 2007; Knäuper et al. 2011), which incorporates vivid imagination of pleasant, relaxing, and food-unrelated mental images. Through this procedure, a state of relaxation should be induced. Alternative imagery should replace craving related food imagery by claiming visuospatial working memory capacities. To find the most suitable mental image, all woman should try different image contents and observe which image would fulfil the prerequisite of being easy to retrieve, relaxing, and vivid. Participants then visualized this image in every self-regulation phase. In all sessions, a visual beach animation was fluently presented to assist relaxation. Each session lasted approximately 35 min.

Assessment Instruments

Screening instruments. For screening purposes regarding inclusion and exclusion criteria, we assessed age, gender, BMI, current dieting status, medication, histories of eating disorders, alcohol abuse, neurological and mental disorders, and diabetes online. Further, we used the *Restraint Scale* (RS; Dinkel et al. 2005) with a ten item cut-off sum score ≥ 12 to determine restrained eating, and the German *Eating Disorder Examination Questionnaire* (EDE-Q; Hilbert and Tuschen-Caffier 2006) with a 22 item cut-off mean score < 4 (Mond et al. 2012) to determine disordered eating. For both measures, good psychometric properties have been reported (Dinkel et al. 2005, Hilbert and Tuschen-Caffier 2006).

Binge eating episodes. We assessed the frequency of binge eating episodes with a questionnaire (Schmidt and Martin 2015), asking participants to retrospectively rate the number of binge eating episodes within the last seven days. The rating scale was preceded by a definition of binge eating episodes in the subclinical context of this study, defining them as being induced by food craving urges and resulting in undesired consumption of high calorie food without physiological hunger. The reported number of binge eating episodes was used as an indicator of binge eating frequency at T0 and T1. To separate successful and unsuccessful participants for subgroup analyses, a criterion of at least 50% symptom reduction in binge eating from T0 to T1 was regarded as clinically relevant success, in line with previous suggestions (e.g., Blanchard and Schwartz 1988).

Somatic self-efficacy. We assessed somatic self-efficacy with a five item questionnaire on the perceived ability to control bodily responses and to relax (e.g., “I am able to control my

bodily reactions"; "For me, it is easy to calm down when I am upset") with 7-point answer scales (0 = *do not agree at all*; 6 = *fully agree*). The mean score served as an indicator of somatic self-efficacy. Internal consistency of the questionnaire was acceptable, $\alpha = .70$.

Statistical analyses

Some data distributions violated normality assumptions. We predominantly used parametric methods, but backed up the results with non-parametric equivalents or bootstrapping techniques.

To analyze possible reductions in relative spectral EEG high beta activity throughout self-regulation phases at T0 and T1, we performed separate within-groups *t*-tests for the NF and MI group (H1) and backed them up with non-parametric Wilcoxon-tests. We used the same procedure for somatic self-efficacy to account for comparable effects. To address the question whether successful and unsuccessful participants differ in their amount of relative spectral EEG high beta post-treatment (H2), we used Mann-Whitney *U*-tests for either group.

We conducted hierarchic regression analyses for each group to determine, whether EEG high beta activity or somatic self-efficacy would predict binge eating episodes at post-treatment (H3). EEG high beta activity was first inserted as predictor for NF (model 1), followed by the addition of somatic self-efficacy (model 2). For MI, the procedure was vice versa. To back up regressions, we used a bootstrapping procedure ($n = 1000$). Significance levels were determined at $p < .05$, one-sided for H1 and H2, and two sided for H3.

In line with recommendations (Fritz et al. 2012), effect sizes for H1 and H2 were calculated as *r* based on *Z*-values due to partly skewed data. Effect sizes for H1 were calculated as $r = \left|\frac{z}{\sqrt{2n}}\right|$. Effect sizes for H2 were calculated as $r = \left|\frac{z}{\sqrt{N}}\right|$. For *r*, values ≥ .50 indicate large effects, values ≥ .30 indicate medium effects, and values ≥ .10 indicate small effects (Fritz et al. 2012).

Results

We did neither observe any significant differences between groups in demographic or screening variables (see Table 1), nor in any outcome variable at pre-treatment (all *p*s > .080), indicating comparable groups.

Table 1 Demographic and screening data of the analyzed sample

Variable	Neurofeedback	Mental Imagery	Total	Test statistics
n	18	18	36	
	M (*SD*)	*M* (*SD*)	*M* (*SD*)	
Age	47.94 (14.24)	39.22 (14.75)	43.58 (14.96)	$t(34) = 1.81$, $p = .080$
Body Mass Index	27.89 (4.93)	27.26 (4.86)	27.58 (4.84)	$t(34) = 0.39$, $p = .702$
Restraint score	19.39 (4.39)	19.28 (3.97)	19.33 (4.13)	$t(34) = 0.08$, $p = .937$
Eating pathology (EDE-Q total)	2.19 (0.91)	2.39 (1.09)	2.29 (0.99)	$t(34) = -0.58$, $p = .566$

EDE-Q = Eating Disorder Examination Questionnaire

Addressing the first research question, we found that NF participants showed significantly reduced EEG high beta activity during self-regulation phases after cue exposure at T1 compared to T0 (p = .031, medium effect). This effect was not observed for MI participants (p = .129, small effect). The same pattern resulted from non-parametric analyses. Descriptive data and test statistics are displayed in Table 2.

Table 2 Group EEG and self-report data pre and post treatment

	Pre-treatment		Post-treatment		Test statistics		
	M	(*SD*)	*M*	(*SD*)	*t*(17)	*p*	*r*
Neurofeedback (*n* = 18)							
Binge Eating Episodes	4.38	(2.77)	3.27	(3.34)	1.91	.037*	.33
EEG High Beta Activity %	4.68	(3.19)	3.19	(1.31)	2.00	.031*	.32
Somatic Self-Efficacy	2.93	(0.91)	3.79	(0.81)	- 4.81	< .001**	.58
Mental Imagery (*n* = 18)							
Binge Eating Episodes	4.50	(3.84)	2.83	(3.49)	2.21	.021*	.35
EEG High Beta Activity %	4.04	(2.57)	3.39	(2.06)	1.17	.129	.19
Somatic Self-Efficacy	2.71	(0.89)	3.41	(1.06)	- 3.36	.002**	.47

Note. Test statistics: within-groups *t*-tests; *p*-values: one-sided, * $p < .05$; ** $p < .01$.
Conventions for effect size *r*: $r \geq .10$ small effect; $r \geq .30$ medium effect; $r \geq .50$ large effect.

For the second research question, groups were divided into subgroups of patients with successful (NF: $n = 9$, MI: $n = 11$) and non-successful (NF: $n = 9$, MI: $n = 7$) treatment outcomes based on at least 50 % reductions in weekly binge eating. At T1, successful NF participants had significantly lower EEG high beta activity ($M = 2.67$ %, $SD = 1.18$ %) compared to unsuccessful NF participants ($M = 3.70$ %, $SD = 1.31$ %), $Z = -2.08$, $p = .020$, $r = .50$. This difference cannot be attributed to initial EEG high beta activity, as at T0, no difference was observed, $Z = -0.84$, $p = .218$, $r = .20$. Further, no effect was found comparing successful ($M = 3.59$ %, $SD = 2.44$ %) and unsuccessful ($M = 3.07$ %, $SD = 1.36$ %) participants in MI, T1: $Z = 0.23$, $p = .430$, $r = .05$; T0: $Z = 0.50$, $p = .330$, $r = .12$. Results are depicted in Fig. 2.

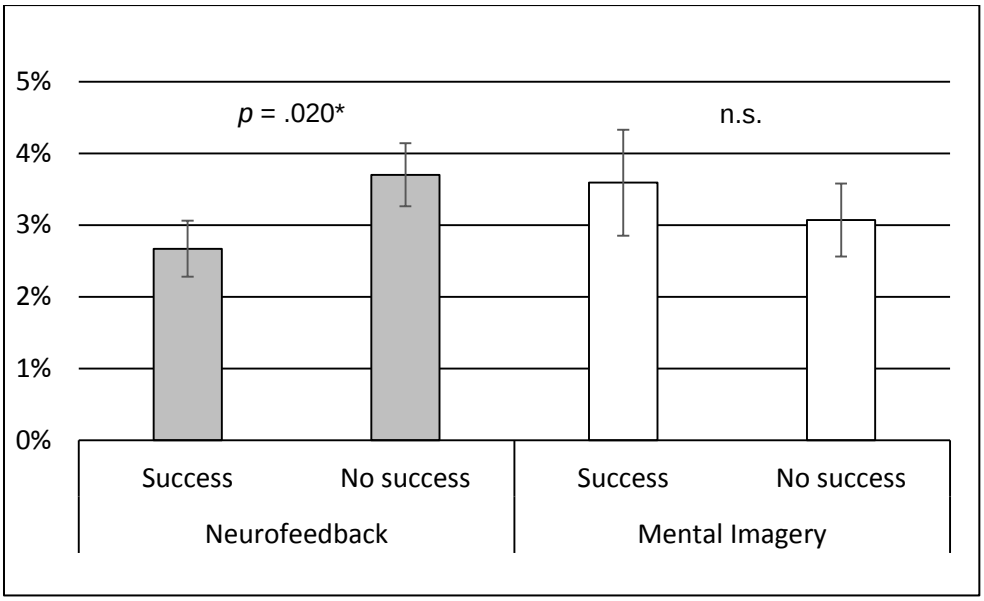

Fig. 2 Comparison of post-treatment EEG high beta activity (relative) in subjects with or without clinically relevant success (≥ 50% vs. < 50% symptom reduction).
Note: Test statistics: Mann-Whitney U-Test, error bars indicate standard errors, * $p < .05$.

Hierarchic regression analyses for the third research question showed that in NF, model 1, with EEG high beta activity as a predictor, explained 21 % of the variance in binge eating at post-treatment. When somatic self-efficacy was added as a predictor in model 2, the amount of variance explained increased to 34%. While EEG high beta activity remained significant as a predictor, somatic self-efficacy only showed a trend towards significance as a predictor, yet became significant in the bootstrapped model ($p = .046$). Statistical details for both models are shown in Table 3.

For MI, the regression model using somatic self-efficacy as a single predictor (model 1) explained 36% of the variance in post-treatment binge eating. However, in the bootstrapped model, only a trend towards significance was observed for somatic self-efficacy (p = .075). When EEG high beta activity was added as a predictor (model 2), it did not explain any additional variance in post-treatment binge eating. This was confirmed in the bootstrapped model (p = .877). Statistical details for both models are shown in Table 4.

Table 3 Hierarchic regression for the prediction of binge eating episodes after neurofeedback

Variable	*B*	β	*t*	*p*	B: CI 95%	ΔR^2
Model 1						
Constant	-0.82		-0.44	.699	[-4.79; 3.16]	
EEG high beta T1	1.29	.51	2.36	.032	[0.13; 2.44]	.26
R^2_{adj} = .21, F(1, 16) = 5.55, p = .032						
Model 2						
Constant	5.10		1.52	.149	[-2.05; 12.25]	
EEG high beta T1	1.41	.56	2.82	.013	[0.34; 2.48]	.26
Somatic self-efficacy T1	-1.67	-.41	2.46	.058	[-3.41; 0.07]	.16
R^2_{adj} = .34, F(2, 15) = 5.43, p = .017						

Y = Binge eating episodes post treatment, n = 18, T1 = post-treatment.

Table 4 Hierarchic regression for the prediction of binge eating episodes after mental imagery

Variable	*B*	β	*t*	*p*	B: CI 95%	ΔR^2
Model 1						
Constant	9.90		4.34	.001	[5.06; 14.74]	
Somatic self-efficacy T1	-2.07	-.63	-3.23	.005	[-3.43; -.0.71]	.40
R^2_{adj} = .36, F(1, 16) = 10.45, p = .005						
Model 2						
Constant	9.77		3.61	.003	[3.99; 15.54]	
Somatic self-efficacy T1	-2.07	-.63	-3.11	.007	[-3.48; -0.65]	.40
EEG high beta T1	0.04	.02	0.10	.920	[-0.69; 0.76]	.00
R^2_{adj} = .32, F(2, 15) = 4.91, p = .023						

Y = Binge eating episodes post treatment, n = 18, T1 = post-treatment.

Discussion

The present study aimed at investigating the presence and influence of physiological learning mechanisms (i.e., reductions in EEG high beta as a marker of cortical arousal) versus psychological learning mechanisms (i.e., enhancement of somatic self-efficacy and abilities to relax) in a NF treatment against binge eating. We aimed at exploring, how these processes relate to reductions in binge eating, and whether effects of physiological learning are specific for NF. Therefore we compared NF to an alternative MI treatment in a highly corresponding setup with imagery-related self-regulation after food cue exposure.

The results indicate the presence of physiological learning in NF: Participants in NF were able to reduce their cortical arousal–as measured by EEG high beta activity–from pre- to post-treatment. On the contrary, we did not observe EEG high beta reductions in MI, confirming H1. Hence, this physiological learning mechanism seems to be specific for NF. Enhancements in somatic self-efficacy and abilities to relax were observed in both treatments, indicating that the reduced EEG arousal in NF is not only caused by subjective relaxation (e.g., Kim et al. 2014).

We further obtained results that mark the relation between a reduction in cortical arousal and treatment success: Post-treatment EEG high beta activity differed among successful and unsuccessful participants, with lower EEG high beta activity in successful participants. This effect was found for NF but not for MI, confirming H2.

After the treatment, lower EEG high beta activity predicted the frequency of binge eating episodes. Again, this pattern was exclusively observed in NF. While post-treatment somatic self-efficacy did exert some influence in NF, it was only a significant predictor of post-treatment binge eating in MI, confirming H3.

In conclusion, both treatments seem to work on different pathways: Although perceived regulatory abilities play a certain role in NF – which is in line with previous findings in BF research (Holroyd et al. 1984; Wickramasekera 1999) – physiological learning still showed a greater influence in this treatment modality, while psychological learning accounted for improvements in MI. One important prerequisite of the NF and BF approach is the view that physiological activity associated with dysfunctional states or behaviors is altered to change the behavior or state itself as a consequence (Niv 2013; Schwartz 1976). Critiques have recently again challenged this view, pointing out that treatment effects in NF may be attributed to

unspecific treatment factors (Thibault et al. 2016) or *neuroenchantment* (i.e., enhanced credibility of studies or treatments that use brain imaging; Ali et al. 2014).

In fact, there is a lack of evidence on the role of physiological learning in many protocols (Gruzelier 2014b). According to the results of the present study, physiological learning seems to be a veritable mechanism in NF, linking reduced cortical arousal to less frequent binge eating (Tammela et al. 2010). This finding is also important because EEG high beta activity has seldom been the main target frequency range in NF protocols (Paquette et al. 2009; Zotev et al. 2014), but has instead mostly been used as a supplementary control range (Egner and Gruzelier 2001; Keith et al. 2015). Our findings indicate, that high beta is a trainable frequency range that can be targeted in NF when psychological correlates are indicative for this procedure.

Still, a discussion of limitations is warranted. Statistical power is reduced due to missing EEG data that had to be excluded because of artifacts. Although we tried to limit constraints exerted by sample size with appropriate statistical analyses, the study should be replicated with larger samples.

The present study allowed to assess changes in EEG activity across treatment sessions. However, we were not able to determine within-session-learning for NF because of technical constraints. Within-session learning is a parameter, which is frequently used in NF and BF studies (Gruzelier 2014b, Rokicki et al. 1997) and should therefore be measured in further evaluations of this protocol.

For the intervention trial that incorporated this experimental EEG study, we chose a probably efficacious MI treatment (Kemps and Tiggemann 2007; Knäuper et al. 2011) as a control condition due to ethical reasons (LaVaque and Rossiter, 2001). In contrast to the present design, comparisons with a sham feedback control condition (i.e., the exact same setup as in NF but with a feedback on unrelated or irrelevant physiological activity) would allow to assess influences of the particular EEG high beta reduction protocol even more specifically (Thibault et al. 2016). Thus, a sham-controlled study design would be desirable in future research on this protocol.

Apart from limitations, our study has the strengths of analyzing objective physiological regulatory abilities with sophisticated EEG equipment and proper artifact corrections (Muthukumaraswamy 2013; Wallstrom, et al. 2004), in a standardized experimental design using reliable methods.

The experimental setup of the study implies another strength: We measured the EEG in the absence of feedback. Through this setup we found, that the down-regulation of cortical arousal was no longer dependent on provided feedback. These findings indicate that NF participants should be able to control their cortical arousal in everyday situations that include tempting confrontations with food cues (e.g., at dinner parties or cafeteria buffets). Hence, our results show a transfer process that accounts for external validity and effectiveness of the NF (Sherlin et al. 2011).

Evidence on physiological learning was mixed in prior research on BF protocols aimed at eating behaviors and none of these studies applied EEG NF (Frank et al., 2012; Meule et al. 2012; Teufel et al. 2013). To our best knowledge, our study provides the first available insights into mechanisms in a NF protocol to reduce binge eating. Overall, the present results contribute to the body of physiological evidence that is heavily demanded by NF researchers (Bagdasaryan and Le Van Quyen, 2013; Gruzelier 2014b; Niv 2013; Strehl 2014).

Altogether, our results contribute to the notion that self-control abilities regarding the "flesh" (i.e., physiological changes) can help increase the "spirit's" ability to resist temptation, showing that NF indeed can provide specific physiological contributions to change dysfunctional eating.

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Ethical approval

All procedures performed in this study were in accordance with the ethical standards of the institutional research committee and with the 1964 Helsinki declaration and its later amendments.

Conflict-of-interest-statement

The authors declare that they have no conflict of interest.

References

Adam, T. C., & Epel, E. S. (2007). Stress, eating and the reward system. *Physiology & Behavior, 91*(4), 449-458.

Ali, S. S., Lifshitz, M., & Raz, A. (2014). Empirical neuroenchantment: From reading minds to thinking critically. *Frontiers in Human Neuroscience, 8*:357.

Bagdasaryan, J., & Le Van Quyen, M. (2013). Experiencing your brain: Neurofeedback as a new bridge between neuroscience and phenomenology. *Frontiers in Human Neuroscience, 7*:680.

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.

Blanchard, E. B., & Schwarz, S. P. (1988). Clinically significant changes in behavioral medicine. *Behavioral Assessment, 10*(2), 171–188.

Boswell, R. G., & Kober, H. (2016). Food cue reactivity and craving predict eating and weight gain: a meta-analytic review. *Obesity Reviews*, *17*(2), 159-177.

Dinkel, A., Berth, H., Exner, C., Rief, W., & Balck, F. (2005). Deutsche Adaptation der Restraint Scale zur Erfassung gezügelten Essverhaltens. *Diagnostica*, *51*(2), 67-74.

Dulloo, A. G., & Montani, J. P. (2015). Pathways from dieting to weight regain, to obesity and to the metabolic syndrome: an overview. *Obesity Reviews*, *16*(S1), 1-6.

Egner, T., & Gruzelier, J. H. (2001). Learned self-regulation of EEG frequency components affects attention and event-related brain potentials in humans. *NeuroReport, 12*(18), 4155-4159.

Epstein, L. H., & Blanchard, E. B. (1977). Biofeedback, self-control, and self-management. *Biofeedback and Self-regulation*, *2*(2), 201-211.

Frank, S., Lee, S., Preissl, H., Schultes, B., Birbaumer, N., & Veit, R. (2012). The obese brain athlete: Self-regulation of the anterior insula in adiposity. *PloS one*, *7*(8), e42570.

Fritz, C. O., Morris, P. E., & Richler, J. J. (2012). Effect size estimates: Current use, calculations, and interpretation. *Journal of Experimental Psychology: General, 141*(1), 2-18.

Gruzelier, J. H. (2014a). EEG-neurofeedback for optimising performance. I: A review of cognitive and affective outcome in healthy participants. *Neuroscience & Biobehavioral Reviews*, *44*, 124-141.

Gruzelier, J. H. (2014b). EEG-neurofeedback for optimising performance. III: A review of methodological and theoretical considerations. *Neuroscience & Biobehavioral Reviews*, *44*, 159-182.

Haedt-Matt, A. A., & Keel, P. K. (2011). Revisiting the affect regulation model of binge eating: A meta-analysis of studies using ecological momentary assessment. *Psychological Bulletin, 137*(4), 660-681.

Hilbert, A., & Tuschen-Caffier, B. (2006). *Eating Disorder Examination. Deutschsprachige Übersetzung*. Münster: Verlag für Psychotherapie, PAG Institut für Psychologie AG.

Holroyd, K. A., Penzien, D. B., Hursey, K. G., Tobin, D. L., Rogers, L., Holm, et al. (1984). Change mechanisms in EMG biofeedback training: Cognitive changes underlying improvements in tension headache. *Journal of Consulting and Clinical Psychology*, *52*(6), 1039-1053.

Jasper, H. H. (1958). The ten twenty electrode system of the international federation. *Electroencephalography and Clinical Neurophysiology*, *10*, 371-375.

Keith, J. R., Rapgay, L., Theodore, D., Schwartz, J. M., & Ross, J. L. (2015). An assessment of an automated EEG biofeedback system for attention deficits in a substance use disorders residential treatment setting. *Psychology of Addictive Behaviors*, *29*(1), 17-25.

Kemps, E., & Tiggemann, M. (2007). Modality-specific imagery reduces cravings for food: An application of the elaborated intrusion theory of desire to food craving. *Journal of Experimental Psychology: Applied, 13*(2), 95-104.

Kim, D. K., Rhee, J. H., & Kang, S. W. (2014). Reorganization of the brain and heart rhythm during autogenic meditation. *Frontiers in Integrative Neuroscience, 7*:109.

Knäuper, B., Pillay, R., Lacaille, J., McCollam, A., & Kelso, E. (2011). Replacing craving imagery with alternative pleasant imagery reduces craving intensity. *Appetite, 57*(1), 173-178.

La Vaque, T. J., & Rossiter, T. (2001). The ethical use of placebo controls in clinical research: the Declaration of Helsinki. *Applied Psychophysiology and Biofeedback, 26*(1), 23-37.

La Vaque, T. J., Hammond, D. C., Trudeau, D., Monastra, V., Perry, J., Lehrer, P. et al. (2002). Template for developing guidelines for the evaluation of the clinical efficacy of psychophysiological interventions. *Applied Psychophysiology and Biofeedback, 27*(4), 273-281.

Meule, A., Freund, R., Skirde, A. K., Vögele, C., & Kübler, A. (2012). Heart rate variability biofeedback reduces food cravings in high food cravers. *Applied Psychophysiology and Biofeedback, 37*(4), 241-251.

Mond, J. M., Hay, P. J., Rodgers, B., & Owen, C. (2006). Eating Disorder Examination Questionnaire: Norms for young adult women. *Behaviour Research and Therapy, 44*(1), 53-62.

Muthukumaraswamy, S. D. (2013). High-frequency brain activity and muscle artifacts in MEG/EEG: A review and recommendations. *Frontiers in Human Neuroscience, 7*:138.

Niv, S. (2013). Clinical efficacy and potential mechanisms of neurofeedback. *Personality and Individual Differences, 54*(6), 676-686.

Ozier, A. D., Kendrick, O. W., Leeper, J. D., Knol, L. L., Perko, M., & Burnham, J. (2008). Overweight and obesity are associated with emotion-and stress-related eating as measured by the eating and appraisal due to emotions and stress questionnaire. *Journal of the American Dietetic Association, 108*(1), 49-56.

Paquette, V., Beauregard, M., & Beaulieu-Prévost, D. (2009). Effect of a psychoneurotherapy on brain electromagnetic tomography in individuals with major depressive disorder. *Psychiatry Research: Neuroimaging, 174*(3), 231-239.

Parvaz, M. A., Alia-Klein, N., Woicik, P. A., Volkow, N. D., & Goldstein, R. Z. (2011). Neuroimaging for drug addiction and related behaviors. *Reviews in the Neurosciences, 22*(6), 609-624.

Presnell, K., Bearman, S. K., & Stice, E. (2004). Risk factors for body dissatisfaction in adolescent boys and girls: A prospective study. *International Journal of Eating Disorders, 36*(4), 389-401.

Rokicki, L. A., Holroyd, K. A., France, C. R., Lipchik, G. L., France, J. L., & Kvaal, S. A. (1997). Change mechanisms associated with combined relaxation/EMG biofeedback training for chronic tension headache. *Applied Psychophysiology and Biofeedback, 22*(1), 21-41.

Rostami, R., Sadeghi, H., Karami, K. A., Abadi, M. N., & Salamati, P. (2012). The effects of neurofeedback on the improvement of rifle shooters' performance. *Journal of Neurotherapy, 16*(4), 264-269.

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.

Schmidt, J., & Martin, A. (2016). Neurofeedback against binge eating – A randomized controlled trial a female subclinical threshold Sample. *European Eating Disorders Review, 24*(5), 406-416.

Schwartz, G. E. (1976). Self-regulation of response patterning. *Biofeedback and Self-Regulation, 1*(1), 7-30.

Schwartz, M. S., & Andrasik, F. (2003) Evaluation research in clinical biofeedback. In M. S. Schwartz & F. Andrasik (Eds.), *Biofeedback – A Practitioner's Guide*, 3rd ed. (pp. 867-880). New York, NY: The Guilford Press.

Shapiro, D., Crider, A. B., & Tursky, B. (1964). Differentiation of an autonomic response through operant reinforcement. *Psychonomic Science*, *1*(1-12), 147-148.

Sherlin, L. H., Arns, M., Lubar, J., Heinrich, H., Kerson, C., Strehl, U., & Sterman, M. B. (2011). Neurofeedback and basic learning theory: implications for research and practice. *Journal of Neurotherapy*, *15*(4), 292-304.

Siniatchkin, M., Kropp, P., & Gerber, W. D. (2000). Neurofeedback–The significance of reinforcement and the search for an appropriate strategy for the success of self-regulation. *Applied Psychophysiology and Biofeedback*, *25*(3), 167-175.

Skinner, H. H., Haines, J., Austin, S. B., & Field, A. E. (2012). A prospective study of overeating, binge eating, and depressive symptoms among adolescent and young adult women. *Journal of Adolescent Health*, *50*(5), 478-483.

Sobik, L., Hutchison, K., & Craighead, L. (2005). Cue-elicited craving for food: A fresh approach to the study of binge eating. *Appetite*, *44*(3), 253-261.

Strehl, U. (2014). What learning theories can teach us in designing neurofeedback treatments. *Frontiers in Human Neuroscience*, 8:894.

Stroebe, W., Mensink, W., Aarts, H., Schut, H., & Kruglanski, A. W. (2008). Why dieters fail: Testing the goal conflict model of eating. *Journal of Experimental Social Psychology*, *44*(1), 26-36.

Swinburn, B. A., Sacks, G., Hall, K. D., McPherson, K., Finegood, D. T., Moodie, M. L., & Gortmaker, S. L. (2011). The global obesity pandemic: Shaped by global drivers and local environments. *The Lancet*, *378*(9793), 804-814.

Tammela, L. I., Pääkkönen, A., Karhunen, L. J., Karhu, J., Uusitupa, M. I., & Kuikka, J. T. (2010). Brain electrical activity during food presentation in obese binge-eating women. *Clinical Physiology and Functional Imaging*, *30*(2), 135-140.

Teufel, M., Stephan, K., Kowalski, A., Käsberger, S., Enck, P., Zipfel, S., & Giel, K. E. (2013). Impact of biofeedback on self-efficacy and stress reduction in obesity: A randomized controlled pilot study. *Applied Psychophysiology and Biofeedback*, *38*(3), 177-184.

Thibault, R. T., Lifshitz, M., & Raz, A. (2016). The self-regulating brain and neurofeedback: Experimental science and clinical promise. *Cortex, 74*, 247-261.

Thompson, M., & Thompson, L. (2007). Neurofeedback for Stressmanagement. In P. M. Lehrer, R. L. Woolfolk, & W.E. Sime (eds.), *Principles and practice of stress management. 3rd Ed.* (pp. 249-287), New York, NY: The Guilford Press.

Wallstrom, G. L., Kass, R. E., Miller, A., Cohn, J. F., & Fox, N. A. (2004). Automatic correction of ocular artifacts in the EEG: A comparison of regression-based and component-based methods. *International Journal of Psychophysiology*, *53*(2), 105-119.

Welch, P. D. (1967). The use of fast Fourier transform for the estimation of power spectra: A method based on time averaging over short, modified periodograms. *IEEE Transactions on Audio and Electroacoustics*, *15*(2), 70-73.

Wickramasekera, I. (1999). How does biofeedback reduce clinical symptoms and do memories and beliefs have biological consequences? Toward a model of mind-body healing. *Applied Psychophysiology and Biofeedback*, *24*(2), 91-105.

Zotev, V., Phillips, R., Yuan, H., Misaki, M., & Bodurka, J. (2014). Self-regulation of human brain activity using simultaneous real-time fMRI and EEG neurofeedback. *NeuroImage*, *85*, 985-995.

7. General discussion

Disinhibited eating (DE) behaviors, such as binge eating episodes, have become a widespread problem within our society. They may lead to negative consequences for physical health (e.g., overweight, obesity, and increased morbidity) as well as mental health (e.g., depressive symptoms or clinical eating disorders). Still, to alleviate DE behaviors, persons concerned mainly rely upon dieting with limited success rates (Mann et al., 2007), rather than psychological interventions. Although cognitive behavioral therapy can be considered as an evidence-based treatment for clinical eating disorders that include DE (Vocks et al., 2010), remission rates are not as high as desired (Brownley et al., 2007; Wilson et al. 2007).

Given this current state in intervention research, leading researchers in the field of eating disorders have encouraged and advocated studies on brain-directed treatments and neuromodulation techniques for dysfunctional eating behaviors (Schmidt & Campbell, 2013, Val-Laillet et al., 2015). EEG NFB is probably the most prominent approach among the brain-directed treatments (Hammond, 2006; Thibault et al., 2015) and constitutes a very safe non-invasive intervention-method (Myers & Young, 2012; Ros et al., 2014). However, research on EEG NFB in the treatment of dysfunctional eating behaviors has not been existent at the starting-point of this dissertation project (Bartholdy et al., 2013). The present dissertation therefore aimed at filling this research gap.

Based on an integrative synthesis of theories of DE, electrophysiological correlates associated with common antecedents, and treatment protocols in related areas of research, a ten-session NFB protocol for a treatment of DE was developed, based on EEG high beta reduction after food cue exposure.

The NFB was subject to evaluation in two RCTs, in subclinical samples of female REs with episodes of DE, to analyze the general (Study 1) and specific efficacy (Study 2). Furthermore, treatment mechanisms were examined in an EEG experiment incorporated in the second intervention trial (Study 3), comparing physiological and psychological learning processes and their relation to treatment outcomes.

In this section, the key findings of the three studies will be discussed in relation to current research (cf. 7.1), followed by a look on practical implications (cf. 7.2). Limitations and strengths of the studies will then be pondered (cf. 7.3). Finally, an outlook on future research will be provided (cf. 7.4).

7.1 Summary of key findings and relation to current research

The key finding of Study 1 was that the new NFB protocol was in fact efficacious in reducing DE behaviors and associated distress. At post-treatment, participants who received NFB reported significantly less DE episodes and associated distress compared to the waitlist.

Up to the start of this dissertation project, there had not been any studies to examine NFB as a treatment for DE, although its potential had been outlined for the treatment of eating disorders (Bartholdy et al., 2013). Study 1 therefore extends on the state-of-the-art in intervention research. The obtained results showed large effect sizes for primary outcomes, which is in line with biofeedback studies in other modalities, such as HRV biofeedback (Meule, Freund et al., 2012) and electrodermal biofeedback (Teufel et al., 2013). Effects on actual eating behavior were found, as opposed to other biofeedback studies that mainly found changes in psychological correlates and antecedents of dysfunctional eating (Meule, Freund, et al., 2012; Teufel et al., 2013), or no changes in psychological and behavioral factors at all (Frank et al., 2012; Pop-Jordanova, 2000).

Another key finding was the stability of changes in primary outcomes. Here, results are in line with the study by Teufel et al. (2013) and extend the general knowledge on maintenance of effects in eating-related biofeedback treatments. In NFB research, transfer processes of learned strategies to everyday situations are important to avoid a dependence of beneficial effects on technical equipment (Siniatchkin et al., 2000; Strehl et al., 2014). The stability of the obtained changes suggests that respective transfer processes occurred following the NFB treatment.

Another key finding was the high acceptance of the NFB approach. More than 85% of the participants rated the treatment experience as positive. This finding is in line with previous reports on the acceptance of biofeedback interventions: Rief and Birbaumer (2006) reported that almost 90% of patients who received biofeedback interventions (n = 4188), rated this specific treatment as helpful (p. 6). Further, apart from slight drowsiness perceived by some participants, no negative side-effects were reported. This indicates that the new protocol is as safe as other NFB protocols (Hammond, 2006; Myers & Young, 2012), and probably safer than other neuromodulatory treatments, like transcranial magnetic stimulation (Brunoni et al., 2011).

The findings of Study 1 further indicate that the selected NFB setup and combination of treatment components (e.g., session number and frequency, incorporation of cue exposure) were appropriate, and thus validly derived in light of theoretical considerations and previous

intervention studies (e.g., Demos, 2005; Jansen et al., 2016; Vernon, 2005). In the iterative process of intervention development, this validation constitutes another important aspect (Campbell et al., 2000). Still, the study design in Study 1, which used a waitlist control group to examine treatment efficacy, did not allow for an inference of specific effects of the NFB. For example, repeated food cue exposure (Jansen et al., 1992; Jansen et al., 2016; McIntosh et al., 2011) or relaxation practices (Katterman et al., 2014; Manzoni et al., 2008), as well as unspecific treatment effects (Gruzelier, 2014; Kraemer, Wilson, Fairburn, & Agras, 2002; Thibault et al. 2016) may have contributed to the beneficial outcomes of this rather complex NFB protocol.

It was therefore a central aim of Study 2 to determine the specific contribution of the NFB technique in this new treatment protocol for DE. In a second RCT, a highly comparable treatment condition was established in addition to a waitlist control group. The alternative treatment setup corresponded to the NFB regarding all components except for the self-regulation strategies (mental imagery; Kemps & Tiggemann, 2006; Knäuper et al., 2010).

Study 2 again showed a reduction of DE behaviors. The frequency of weekly subjective binge eating and associated distress were significantly reduced by means of NFB, with stable results to a three-month follow-up, supporting the validity of results from the pilot study by a replication in another independent sample. A key finding of Study 2 was that NFB did in fact show specific efficacy. NFB, but not mental imagery, accounted for significant reductions in the frequency of subjective binge eating episodes compared to the waitlist at post-treatment. Both treatments reduced binge-related distress. The same pattern was observed in intent-to-treat analyses. Thus, on a behavioral level, NFB was superior to the alternative treatment. Given the comparable setup of both active treatment conditions, the differences in treatment efficacy cannot be attributed to cue exposure (Jansen et al., 2016), relaxation (Katterman et al., 2014) or unspecific treatment factors (Gruzelier, 2014; Kraemer et al., 2002; Thibault et al., 2016).

Further, the assessment of pre-treatment expectations and post-treatment evaluations in the two conditions rejects assumptions of differential credibility of the two treatment methods and resulting distortions in self-reported outcomes (Nichols & Maner, 2008). The latter point may be a nearby assumption due to possible *neuroenchantment* effects (i.e., beliefs that studies using an assessment of brain activity are especially scientific and therefore superior to other approaches; Ali, Lifshitz, & Raz, 2014). Thus, the results of Study 2 extend on previous empirical evidence for specific efficacy of NFB. Specific efficacy can not only be inferred for

classical application fields, like ADHD (Arns, Heinrich, & Strehl, 2014), but also in the treatment of dysfunctional eating as a novel application field.

Other key findings relate to secondary outcomes. Food craving, as assessed with an episodic measure (Meule, Hermann, & Kübler, 2014), was now significantly reduced in both treatment groups, while somatic relaxation-related self-efficacy was enhanced. Yet, only NFB had beneficial effects on dietary self-efficacy and on perceived stress, assessed with a more sensitive measure (Fliege, Rose, Arck, Levenstein, & Klapp, 2001). Altogether, mental imagery as a self-regulation instruction had some beneficial effects: In accordance with previous findings, mental imagery led to a reduction of food craving (Kemps & Tiggeman, 2007; Knäuper et al., 2010), but not of DE. In contrast to Knäuper and colleagues (2010), the present study found even larger effect sizes for the reduction of food craving in both active groups. This finding could have emerged from the large difference in intervention duration, with ten sessions over several weeks in the present studies versus four days in the study of Knäuper et al. (2010).

Still, for food craving, as well as for the other secondary outcomes, effects of NFB were consistently larger than for mental imagery, and a wider range of secondary outcomes improved. For example, only NFB influenced dietary self-efficacy. The construct of dietary self-efficacy relates to the experience of eating- and dieting-related control (Meule, Papies, & Kübler, 2012). As loss of control is a key feature in the phenomenology and etiology of DE (Latner et al., 2007), NFB with its focus on control-processes may have contributed to enhanced control perceptions (Niv, 2013; Hammond, 2006). In previous research, Ninaus and colleagues (2013) found that the mere intention to control one's brain activity can lead to an activation of subcortical structures involved in control processes, such as the insula, anterior cingulate cortex and prefrontal areas. With repeated practice, these control processes could likely result in changes regarding neuroplasticity and hence facilitate self-regulation (Lövdén et al., 2010; Ros et al., 2014).

Another cause of the NFB superiority may lie in its explicit target to regulate brain activity associated with tense arousal. As mentioned in the integrative synthesis model (cf. 2.2.6), states of tense arousal constitute important affective factors in the recurring onset of DE (Ball & Lee, 2000; Curtis & Davis, 2014; Selby et al., 2008; Waters et al., 2001). The distinct reduction of EEG high beta activity as a central physiological correlate of tense arousal (cf. 2.3) may have contributed to these effects. However, to determine the presence and specific contributions of

physiological learning processes in contrast to psychological changes beyond speculation, these treatment mechanisms had to be analyzed and compared.

Study 3 analyzed physiological and psychological treatment mechanisms and their contribution to outcomes of the NFB protocol for DE. This experimental EEG study assessed the ability to regulate EEG high beta activity after food cue exposure, prior to and after the treatment. These regulatory abilities were then compared to psychological learning regarding self-efficacy.

A key finding of Study 3 was the observed presence of physiological learning in NFB. After the treatment phase, NFB participants were able to reduce EEG high beta activity during self-regulation after food cue exposure in contrast to the pre-treatment assessment. These changes did not manifest in the mental imagery group, thus indicating specific physiological learning effects of NFB. These results extend on previous research in two ways:

On the one hand, they add to the general body of empirical evidence that NFB can actually account for physiological learning processes (Gruzelier, 2014). These processes might be related to neuroplasticity effects (Lövdén et al., 2010) that can establish long term changes in dysfunctional brain activity, even without feedback (Niv, 2013; Strehl, 2014). The results indicate that comparable changes may be present in NFB for the treatment of DE.

On the other hand, the findings add evidence for learning mechanisms to occur in EEG high beta activity. While several NFB protocols have included EEG high beta as a supplementary control range (e.g., Gruzelier, Foks, Steffert, Chen, & Ros 2014), it has seldom been a main target of physiological regulation (Paquette, Beauregard, & Beaulieu-Prévost, 2009; Walker, 2011; Zotev et al., 2014), and – to my best knowledge – no prior studies examined physiological learning in EEG high beta activity achieved by unipolar NFB on the electrode site Cz.

Paquette et al. (2009) found reductions in EEG high beta activity using a NFB assessment of activity on T3/4 and AF3/4 in depressive patients. Reductions in right frontal areas were related to treatment outcomes, that is, reduced depression. Zotev and colleagues (2014) found that participants were able to modulate frontal EEG high beta activity in a single session with four trials. Walker (2011) did not report any learning indices for changed EEG high beta activity in the treatment of migraines, although he used QEEG-guided NFB with EEG high beta down-regulation. Thus, the results of Study 3 extend on previous research by demonstrating physiological learning to down-regulate EEG high beta activity at Cz, a common and safe position to be used in standard NFB protocols (Demos, 2005).

Further, reductions in EEG high beta were related to treatment outcomes, but only for the NFB group. Post-treatment, successful NFB participants with at least 50% reduction of DE symptoms showed lower EEG high beta activity than unsuccessful participants. Again, this result was not observed in mental imagery participants, indicating a specific asset of NFB. Physiological activity was a more suitable predictor of post-treatment binge eating symptoms than subjective somatic self-efficacy, although the latter factor was also enhanced by means of NFB and showed some – yet no significant – relations to treatment outcomes. In mental imagery, somatic self-efficacy, but not EEG high beta, predicted post-treatment DE. These results inform NFB researchers, as more evidence is being provided for the importance of physiological learning as a treatment mechanism in NFB (Gruzelier, 2014; Sherlin et al., 2011). In addition, results underline the specific contribution of physiological learning in the developed NFB protocol for the treatment of DE.

Physiological learning is neglected in many NFB studies (Strehl, 2014). Opposing recent critique on the unspecific treatment effects in NFB interventions (Thibault et al., 2016), Study 3 found specific contributions of physiological learning to outperform the influence of psychological learning, which in turn is a highly-valued treatment mechanism in several biofeedback applications (Holroyd et al., 1984; Schwartz & Schwartz, 2003; Wickramasekera, 1999). Still, the relation of physiological learning and treatment outcomes might be higher in EEG NFB than in other biofeedback modalities, given previous null-findings in biofeedback studies that addressed eating behavior (Frank et al., 2012; Meule, Freund et al., 2012).

Together, the studies conducted as part of this doctoral thesis point into the direction of beneficial, specific, and physiologically-based effects of the developed NFB protocol. Thus, the research fills a previous gap in intervention studies on neuromodulatory, brain-directed treatments (Bartholdy et al., 2013; Schmidt & Campbell, 2013; Val-Laillet et al., 2015). It can be concluded that NFB in fact constitutes a promising approach in the treatment of dysregulated eating based on obtained empirical support for the previous theoretical and conceptual considerations. However, the protocol still constitutes a new treatment method in this field. Long-term effects have to be analyzed and its distinct value as an adjunct to existing first-line treatments, like cognitive behavioral treatments, has yet to be evaluated.

7.2 Practical implications

The positive evaluation of the new NFB protocol for DE behaviors has several practical implications. The following discussion will consider possible fields of application that may be

enriched by the use of NFB in a range of nearby, but also more distant, application fields. Of course, applications in these fields would require initial research.

The most proximate field of application is certainly the treatment of eating disorders that include binge eating behaviors – such as BED and BN – because the aforementioned calls for an evaluation and application of brain-directed treatments specifically arose from intervention research in clinical eating disorders (Iacovino et al., 2012; Schmidt & Campbell, 2013). Here, it has been pointed out that treatments still need improvement regarding outcomes and remission rates (Brownley et al., 2007; Wilson et al., 2007).

The developed NFB protocol was based on an integrative model that takes into account various etiological theories for the development of DE and clinical eating disorders (e.g., Heatherton & Baumeister, 1991; Selby et al., 2008; Stice, 2002; Waters et al., 2001). Regarding the target physiological activity for the NFB, previous results that were distinctly associated with DE predominantly stemmed from objective binge eaters (Tammela et al., 2010). Thus, one could expect the NFB to be effective in populations with clinical eating disorders. Here, NFB may serve as a promising treatment-adjunct, complementing cognitive behavioral therapy as a first-line treatment in BED (Vocks et al., 2010).

One interesting application can be seen in NFB as a treatment-module that could be specifically offered to slow-responders in other psychological treatments, as part of a stepped-care approach (Wilson, Vitousek, & Loeb, 2000). In intervention research on BN and BED, it is a consistent result that certain patients respond rapidly to psychological treatments, showing an early abstinence and improved long-term outcomes (e.g., Bulik, Sullivan, Carter, McIntosh, & Joyce, 1999; Fairburn, Agras, Walsh, Wilson, & Stice, 2004; Hilbert, Hildebrandt, Agras, Wilfley, & Wilson, 2015; Masheb & Grilo, 2007). Patients that do not show an early abstinence from binge eating might be offered NFB as an adjunct in a more intensive treatment. As some previous studies showed that the severe BED and BN symptomatology may be accompanied by specific neuronal deviations (e.g., Friederich, Wu, Simon, & Herzog, 2013; Schienle, Schäfer, Hermann, & Vaitl, 2009), NFB may be especially successful in subgroups with stronger eating pathology that would be less likely to rapidly respond to other treatments.

Another nearby field of application can of course be seen in the general treatment of overweight and obesity. Dieting with calorie restrictions and other behavioral weight-loss practices are still the most common means to target weight loss (Andreyeva et al, 2010; Montani et al., 2015), although long-term success of these practices is limited (Goodrick et al., 1998;

Mann et al., 2007). During the last decades, the importance of psychological and, especially, self-regulatory processes in dysfunctional eating behaviors has gathered more recognition (Berridge, 2009; Wing, Tate, Gorin, Raynor, & Fava, 2006). Here, emotional distress has been highlighted to play a crucial role in the onset of DE behaviors (Selby et al., 2008; Stice, 1994; Waters et al., 2001) and also in the development of overweight and obesity (Hemmingsson, 2014). As Hemmingsson concluded, "these inner disturbances eventually cause a psycho-emotional overload, triggering a cascade of weight gain-inducing effects . . . . Tackling this proposed cause of weight gain could potentially improve both treatment and prevention outcomes." (2014, p. 769). The distinct scope of the developed NFB protocol to target physiological correlates of tense arousal accompanying affective and motivational antecedents of DE may therefore inform the treatment – and maybe even the prevention – of overweight, and obesity. NFB could be used to support behavioral weight-loss attempts by a reduction of physiological arousal as a risk factor for DE and subsequent dietary failure. Furthermore, an enhancement of self-efficacy, as a commonly known protective factor with regard to dysfunctional eating behaviors (Glasofer et al., 2013; Linde, Rothman, Baldwin, & Jefferey, 2006) could improve results of behavioral weight-loss treatments. In consequence, the probability of associated negative health consequences might be reduced (Guh et al., 2009).

The clinical phenomenon of food addiction constitutes another possible application area for NFB treatments. Food addiction symptoms have frequently been associated with severe binge eating (Avena et al., 2011; Davis, 2013; Gearhardt, Grilo, DiLeone, Brownell, & Potenza, 2011) and weight-regulatory problems (Burmeister, Hinma, Koball, Hoffmann, & Carels, 2013). Several findings from research on shared neuronal circuits and processes between food addiction and other addictions suggest that dysfunctional, brain-regulatory processes in subcortical areas are involved in DE among individuals with food addiction symptoms, for example striatal, insular and prefrontal areas (Gearhardt, Yokum et al., 2011; Jastreboff et al, 2013; Volkow, Wang, & Baler, 2011). Ninaus et al. (2013) showed that attempts to regulate brain activity, even in sham-NFB, can beneficially influence the reestablishment of control-processes in subcortical areas. Thus, NFB may be beneficial to treat food addiction symptoms, given their neuronal underpinnings.

Another argument for possible application of NFB in food addiction has to be seen in the development process of the new NFB protocol: Because of the lack of spectral EEG studies to explain the phenomenology of DE behaviors (Bartholdy et al., 2013; Hume et al., 2015; Tammela et al., 2010), the selection of the targeted EEG high beta activity relied on several

studies from the field of addiction (Parvaz et al., 2011) and EEG correlates of antecedents that are commonly shared in food addiction and substance dependence, like craving and perceived stress (Sinha & Jastreboff; 2013; Styn et al., 2013). Thus, when physiological underpinnings of food and drug addiction are even more similar, the NFB protocol might possibly attain even more effective results.

Regarding these remarks on food addiction, it is of course a small step to consider other addictions, as a possible field of application. In contrast to intervention research in eating dysregulations, researchers in the field of substance-dependence by now have a longer tradition of using NFB (e.g., Dehghani-Arani, Rostami, & Nadali, 2013; Scott, Kaiser, Othmer, S., & Sideroff, 2005). Here, some protocols, such as the Peniston-protocol based on alpha/theta training (Peniston & Kulkolsky, 1990), and its Scott-Kaiser-modification with a training of the sensorimotor rhythm (Scott & Kaiser, 1998), are frequently and successfully applied to alter dysfunctional changes in the spontaneous EEG (for a review, see: Sokhadze et al., 2008).

However, in their review, Sokhadze et al. (2008) pointed out that these traditional protocols might not be suitable for all types of addictions, as EEG baseline changes are not constantly observed for patients with substance-dependence. Besides the positive results in the treatment of DE, the developed NFB protocol took into account several prerequisites that have been stated as important in the treatment of substance dependence: a decrease of the reward value of substance stimuli, alterations of learned, stereotypical seeking-behaviors (craving), and methods to improve inhibitory control-processes (Volkow, Fowler, & Wang, 2004).

Given its foundation in EEG high beta reduction after cue exposure, the developed protocol might posit another optional tool that could be considered in NFB for substance dependence, especially in the absence of EEG baseline alterations and for individuals who do not profit from the Peniston- or Scott-Kaiser-protocols.

7.3 Strengths and Limitations

The studies conducted as part of this dissertation project have some inherent strengths as well as limitations that will be reflected upon in this chapter.

Strengths

Most importantly, the studies integrated in this doctoral thesis constitute the first published studies on NFB for the treatment of DE. Following the call for more research on brain-directed treatments (Schmidt & Campbell, 2013), the studies provide the first empirical support for a

possible application of NFB in the treatment of DE (Bartholdy et al., 2013). The pioneering character of the conducted studies can therefore be considered as an asset of this research project.

Another important strength lies in the study design of the two intervention studies: Both trials were RCTs, the design, which is considered the gold standard in the evaluation of psychological interventions (Kraemer et al., 2002). The RCTs were planned in an iterative manner allowing the pilot-study to inform the second RCT in terms of possible necessary adjustments in the intervention setup, such as shortening the self-regulation phases to prevent drowsiness (Campbell et al., 2000).

In both studies, catamnestic data were available. The three-month follow-up assessment allowed for analyses of the intermediate stability of treatment outcomes. While even longer catamnestic intervals would be desirable and are, in fact, quite common in eating disorder intervention research (e.g., Keel & Heatherton, 2010; McIntosh et al., 2011), follow-up data are commonly not available for the majority of NFB studies (Gruzelier, 2014). Regarding previous meta-analyses on the duration to form new patterns of behavior (approx. 66 days: Lally, Van Jaarsveld, Potts, & Wardle, 2010), a three-month follow-up should still provide a good indicator for possible long-term effects.

The design of Study 2, with a highly comparable alternative treatment group, is a strong advantage. With a fully corresponding setup, despite the NFB in self-regulation phases, specific efficacy of the NFB approach could reliably be assessed. Both treatments followed a rationale of ten sessions with cue exposure, conducted in the same location and by the same experimenters, which guaranteed high treatment fidelity and a strong degree of standardization, as important factors in treatment evaluation studies (Bellg et al., 2004). With this design it was possible to distinguish separate contributions of the NFB method from those of repeated cue exposure, relaxation practice, or general unspecific treatment effects that might all very well influence treatment outcomes, but were controlled for in Study 2. Further, the assessment of pre-treatment expectations did not indicate differential credibility of the two active treatments, due to the assessment of brain activity in NFB (Ali et al., 2014) that could have contributed to biased self-reports in the NFB group.

While previous biofeedback studies were limited to relatively homogeneous samples, like obese female participants (Teufel et al., 2013) or female student populations (Meule, Freund et al., 2012), the samples in the present studies were recruited from the local community via

various recruitment attempts in different modes and media. This led to a representative sample of adult female participants of all ages (18 to 70 yrs) and weight classes (normal-weight, overweight, and obese). Still, randomization was successful in both RCTs, resulting in comparable groups. With the selection of REs, a suitable target group was selected that reflects a vulnerable subpopulation for DE behaviors (Polivy & Herman, 1985; Ruderman, 1986; Stroebe et al., 2008). Regarding their intermediate position on a dimension from nonclinical to clinical populations, studies on REs allow for careful result transfers to nonclinical as well as clinical populations.

The presence of objective EEG data, obtained in Study 3, is another strength of the research agenda for this project. The experimental assessment of psychophysiological data allowed for analyses of the presence and particular contributions of physiological and psychological learning mechanisms, which provide deeper insights into the relevant treatment mechanisms in the new protocol. They also update general NFB research with new data on EEG learning in a NFB protocol that targets reductions in EEG high beta activity (Gruzelier, 2014).

In Study 3, the setup aimed at measuring self-regulatory abilities for brain-activity in the absence of feedback. In NFB research, it is important to foster transfer processes so that learning not only takes place in the protected atmosphere of the therapeutic sessions, but also proliferates its effects into everyday setups (Sherlin et al., 2011; Strehl, 2014). Assessment of regulatory abilities in the absence of feedback, as conducted in the EEG experiment, serves as a proxy to determine out-of-the-lab performances with regard to transfer processes.

The use of personalized food cues during exposure phases of the developed NFB protocol accounted for individualized treatment sessions that would reliably elicit craving urges (Loxton et al., 2011). This provided an advantage over the use of standardized pictorial cues. Repeated exposure with food cues further prepared participants for challenging situations when stressors would easily undermine control over eating behavior. In an obesogenic environment (Swinburn et al., 2011), this setup is probably superior to NFB protocols that target baseline activity, because the cue exposure setup prepares participants for challenges that might arise outside of the safe, treatment environment. Therefore, external validity of this intervention is enhanced.

In contrast to other neuromodulatory techniques, like transcranial magnetic or direct current stimulation, the EEG NFB approach itself is a non-invasive, well-researched method that is quite unlikely to produce long-term, negative side-effects (Brunoni et al., 2011; Hammond, 2006; Ros et al., 2014). Further, EEG NFB equipment is more affordable than other

neuromodulatory techniques (e.g., rt-fMRI), has a higher usability and provides flexibility for practitioners (Thibault et al., 2015; Niv, 2013). Thus, it can indeed be practically transferred to therapeutic practices.

Finally, it was observed that an economic ten-session approach was sufficient to initiate clinically relevant changes with medium to large effects sizes, keeping the timely requirements of the treatment in practical limits (Gruzelier, 2014). Further, the developed protocol might not only serve the purpose to treat DE, but might also have a potential as an intervention add-on in other psychological conditions, as mentioned before (cf. 7.2).

Limitations

Of course, the studies presented in this doctoral thesis also have some limitations. One limitation has to be mentioned with regard to the primary outcome measures of the two RCTs (Studies 1 and 2). Treatment outcomes were assessed by means of self-report on the frequency of DE episodes in the previous week. The self-report measures might be subject to memory distortions, retrospective bias, and social-desirability (Taren et al., 1999). These measures are therefore less reliable than less deferred measures on food intake that possibly could have been acquired – for example, by means of ecological momentary assessment. Still, retrospective self-reports are a common means in the assessment of clinical and subclinical DE (e.g., Arnow, Kenardy, & Agras, 1995; Gormally, Black, Daston, & Rardin, 1982; Hilbert & Tuschen-Caffier, 2007) and the same measures were used at pre- and post-treatment in both studies. Thus, congruence with common techniques in eating behavior research, as well as consistency of assessment instruments is given, although more simultaneous outcome assessments would be desirable in future studies on this NFB protocol.

Despite of the promising results of NFB, it has to be mentioned, that not all individuals profited from the treatment. Especially in Study 2, complete abstinence from DE was scarce. Several factors might have contributed to this result. Younger samples may be more flexible with regard to learning processes, than older samples. Abstinence rates were higher in Study 1 (with a slightly younger sample) compared to Study 2, which hints at this possible connection. With regard to general NFB practices, it has been stated that almost 30% of the participants in NFB and other brain-computer-interface studies have difficulties to gain any control over their physiological signals – a phenomenon termed *brain-computer-interface-illiteracy* (Vidaurre & Blankertz, 2010). This problem might be one factor involved in success rates of NFB that should be accounted and controlled for in future studies.

Study 2 used a highly comparable, alternative treatment as an additional control group for comparisons of specific treatment efficacy. However, some researchers have recently ascribed general limitations to NFB studies that do not include sham-NFB as a control condition (Thibault et al., 2016). In sham-NFB, a popular placebo-condition in biofeedback research, participants would also use NFB equipment and try to control brain activity. Yet, they would only receive a false feedback, for example recordings from other participants, other physiological activity (e.g., electromyographic activity), or irrelevant brain activity, other than the targeted spectral range (Thibault et al., 2016).

Indeed, with the data obtained from the studies as part of this dissertation project, specific efficacy can be accounted to the NFB *method*, but not to the distinct NFB *protocol* using EEG high beta reduction. For the treatment of several psychological conditions, veritable NFB has not been found to be superior to sham-NFB (Thibault et al., 2016). Thus, the lack of a sham-NFB condition may be seen as a limitation in the assessment of specific efficacy, despite of the identified physiological learning effect in Study 3. However, it has to be mentioned that sham-feedback might raise ethical concerns due to the deception of participants (La Vaque & Rossiter, 2001). Sham-feedback can further contribute to nocebo-effects (Colloca & Miller, 2011) due to an impairment of patient motivation when no coherence among individual efforts and brain activity is observable in NFB (Gruzelier, 2014). The lack of credibility in this setup – especially when applied in a multi-session protocol – may hence obscure veritable treatment effects of NFB. These possible adverse side-effects should therefore be considered when establishing a sham-NFB control group.

The sample size is a constraint in all studies: Although significant effects were found and intent-to-treat analyses were included in Study 2, drop-outs did limit the range of possible analyses. It would have been highly desirable to test a range of possible treatment mediators and moderators (Grilo, Masheb, & Crosby, 2012; Kraemer et al., 2002). While some insights were gathered in Study 3, larger samples would have allowed for more sophisticated statistical analyses, using multiple-mediator models and path analyses.

Study 3 applied a basic rationale regarding the analysis of physiological learning, targeting the trained frequency range at the trained position of the NFB. Although this approach is adequate to answer the research questions of Study 3, some might see this “reductionist” approach as a limitation. In previous studies, researchers reported that NFB for a single frequency range can exert effects on other frequency ranges and that training effects may

manifest on topographic sites other than the one trained (Egner et al. 2004; Gruzelier, 2014). Multichannel-analyses, for example with quantitative EEG (QEEG) measures, may shed light on this open point in future studies on this protocol.

For the studies conducted on the NFB protocol, a very strict and rigid procedure was chosen with regard to the trained frequency and position. All individuals down-regulated EEG high beta activity at the electrode position Cz to enhance standardization and internal validity regarding the therapeutic procedure. Still, some NFB researchers may view this standardized procedure as a limitation: Especially researchers in clinical settings and NFB practitioners argue for NFB protocols that base on an initial assessment of individual EEG-dysregulations to select a corresponding, QEEG-guided treatment protocol (Gruzelier, 2014; Hammond, 2006). The differential contributions of standardized and individual protocols certainly comprise a topic that future NFB research should consider in the application field of DE.

Lastly, the lack of fundamental EEG studies in the field of DE might have affected the development of the NFB protocol. Despite the careful review of influential theories on antecedents of DE and a meticulous analysis of possible spectral EEG correlates associated with these antecedents, such as craving and disinhibition (Parvaz et al., 2011; Tammela et al., 2010), spectral EEG studies that distinctly address DE behaviors had been very scarce at the development phase of the NFB protocol. Despite of further evidence added by recent research (Hume, Howells et al., 2015), there is still a need for more basic EEG research with regard to subclinical DE behaviors to provide a stable empirical basis for the developed protocol and probably adjust its setup.

7.4 Perspectives for future research

The results of this dissertation project open several perspectives for fundamental research. The lack of studies on spectral EEG activity related to DE behaviors should be addressed in future studies. Here, it is crucial to provide an empirical basis on EEG phenotypes that are distinctly linked to DE behaviors. While Tammela and colleagues (2010) as well as Hume et al. (2015) found increased EEG beta activity in overweight and obese women when confronted with food cues, it is yet unclear whether these results are transferable to normal-weight individuals with DE behaviors. Especially with regard to the divergent types or taxonomies of DE behaviors (cf. 2.1.1), it would further be of interest, whether DE behaviors marked by different antecedents or diagnostic properties show different EEG correlates during

confrontation with food cues and in baseline, or whether the dimensional model could be supported by a linear relationship between symptom severity and EEG deviations.

Basic research should also be conducted with regard to the NFB protocol itself, aiming at systematic analyses of efficacious treatment components and mechanisms in NFB for DE. Considerations of the optimal treatment setup were hitherto based on the existing literature on NFB and biofeedback approaches and successful components in the treatment of DE behaviors and addictions (e.g., Conklin & Tiffany, 2002; Jansen et al., 1992; Teufel et al., 2013). However, it should be tested, whether the configuration of the NFB could be improved with regard to session setup, duration, frequency, and the mode of cue exposure and self-regulation.

For example, in cue exposure for anxiety disorders, habituation is an advocated mechanism for reductions of arousal (Craske et al., 2008). This would propose an advantage of self-regulation in the presence of cues, whereas food cues are removed for self-regulation phases in the current setup of the NFB protocol for DE. However, Jansen and colleagues (2016) recently argued for the extinction paradigms to be superior to habituation settings for food cue exposure treatments. Experimental studies that systematically vary these treatment components and assess the corresponding trajectories in EEG activity could inform and foster improvements of the protocol configuration and possibly lead to enhanced success rates. Some of these aspects are already planned to be examined in future studies on this NFB protocol.

With regard to intervention research, even more insightful results on the specific efficacy of the NFB protocol with EEG high beta reduction after food cue exposure could be obtained by comparisons with other established NFB and biofeedback protocols, as well as additional sham-control groups (Thibault et al., 2016). It would be of particular interest to compare the effects of the new NFB protocol to those of other established NFB protocols that have exerted beneficial effects in samples with substance dependence, such as the Peniston-protocol and the Scott-Kaiser-modification of this protocol (Sokhadze et al., 2008). Here, it would also be possible to obtain insights into possible differential effects of cue exposure NFB versus baseline NFB. Comparisons to other biofeedback modalities, such as HRV biofeedback (Meule, Freund et al., 2012) or electrodermal biofeedback (Teufel et al., 2013), would constitute another interesting attempt to determine whether neuromodulation (Val-Laillet et al., 2015) is superior to regulation of peripheral psychophysiological parameters.

Besides these fundamental research topics, a transfer of the protocol to other populations constitutes a nearby research scope. The majority of considerations on this topic has already been provided in chapter 7.2. Still, some aspects will be highlighted or summarized here.

In the initial introduction of DE behaviors, it has been shown that binge eating and loss-of-control eating, as well as eating disorders and eating pathology are generally more prevalent among women compared to men (Keel et al., 2007; Stiegel-Moore et al., 2009). Still, for objective binge eating episodes and BED in particular, the ratio of affected males and females does not deviate as much (Hilbert et al., 2012; Smink, van Hoeken, & Hoek, 2012). With regard to general overeating, overweight, and obesity, men show even higher prevalence rates than women (Lewinsohn et al., 2002; Mensink et al., 2013; Striegel-Moore et al., 2009). In the light of these findings, it would be a desirable next step to test the protocol in male samples.

Other closely connected populations that could benefit from an application of the developed NFB protocol are individuals with clinical eating disorders, such as BED and BN. In the light of a need for improvement of existing therapies (Brownley et al., 2007; Wilson et al., 2007) and with regard to the recent calls for brain-directed treatments (Schmidt & Campbell, 2013), the NFB protocol could at this stage be well tested in clinical samples. Given the two promising RCTs that did not point into the direction of negative side-effects, a treatment study in samples with eating disorders, or also in individuals with food addiction symptomatology, would be warranted at this stage.

Other possible fields of application encompass the treatment of individuals with impulse control disorders (e.g., trichotillomania or pathological skin picking), behavioral addictions (e.g., pathological buying, compulsive gambling, internet addiction), and obsessive-compulsive disorders (OCDs) and thus a variety of newly classified psychological disorders in the DSM-5 (APA, 2013). In the emotional cascades model, Selby et al. (2008) explicitly mentioned destructive behaviors aside from binge eating to serve as a means for emotion regulation, for example non-suicidal self-injury. Further, several researchers highlighted the shared etiology and common comorbidity of OCDs, impulse control disorders, and eating disorders (e.g., Altman & Shankman, 2009; Angst, et al. 2004; Fernández-Aranda et al., 2008; Schreiber, Odlaug, & Grant, 2011). In addition, Sherlin and Cogendo (2005) reported excess EEG beta activity in subjects with OCDs. The similarities in etiology, phenomenology, and neurophysiology among DE and these conditions indicate a possible transfer of the protocol.

However, basic research on the electrophysiology of these populations – especially in response to cue exposure – is a necessary prerequisite prior to treatment transfer.

7.5 Conclusion

The present doctoral thesis reported on the development and evaluation of a new NFB protocol for the treatment of DE. The rationale was derived from a synthesis of current theories that attempt to explain the etiology of DE behaviors and an analysis of corresponding dysfunctional psychophysiological activity. These analyses, together with considerations of former successful treatment components for DE behaviors and recommendations in NFB research, resulted in the development of a ten-session NFB, targeting EEG high beta reduction after food cue exposure. The protocol was evaluated in two RCTs and an experimental EEG study.

The studies yielded promising results regarding the general and specific efficacy of the NFB protocol with regard to a reduction of DE behaviors and improvements in some secondary outcomes. First analyses of treatment mechanisms in this NFB protocol support learning mechanisms that occur on a physiological basis. Results of the conducted studies have been critically discussed in the light of current research, their practical implications, and the strengths and limitations of the studies. On the one hand, the protocol will profit from extended basic research to approve or improve its configuration. On the other hand, several reasons suggest a possible transfer of the protocol to clinical samples and related psychological conditions.

Altogether, the developed NFB has been evaluated as a promising, well-accepted, and safe brain-directed treatment for subclinical DE that may inform or complement interventions in clinical populations. Thus, the application of NFB may possibly alleviate negative consequences of DE for mental and physical health and improve outcomes in the treatment of dysfunctional eating behaviors and eating disorders.

8. References

Abarbanel, A. (1995). Gates, states, rhythms, and resonance: The scientific basis of neurofeedback training. *Journal of Neurotherapy, 1*(2), 15-38.

Adam, T. C., & Epel, E. S. (2007). Stress, eating and the reward system. *Physiology & Behavior, 91*(4), 449-458.

Ali, S. S., Lifshitz, M., & Raz, A. (2014). Empirical neuroenchantment: From reading minds to thinking critically. *Frontiers in Human Neuroscience, 8*:357, doi:10.3389/fnhum.2014.00357.

Allen, K. L., Byrne, S. M., Oddy, W. H., & Crosby, R. D. (2013). DSM–IV–TR and DSM-5 eating disorders in adolescents: Prevalence, stability, and psychosocial correlates in a population-based sample of male and female adolescents. *Journal of Abnormal Psychology, 122*(3), 720-732.

Almeida, L., Savoy, S., & Boxer, P. (2011). The role of weight stigmatization in cumulative risk for binge eating. *Journal of Clinical Psychology, 67*(3), 278-292.

Altman, S. E., & Shankman, S. A. (2009). What is the association between obsessive–compulsive disorder and eating disorders?. *Clinical Psychology Review, 29*(7), 638-646.

American Psychiatric Association (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Washington, DC: Author.

Andersen, S. B., Moore, R. A., Venables, L., & Corr, P. J. (2009). Electrophysiological correlates of anxious rumination. *International Journal of Psychophysiology, 71*(2), 156-169.

Andrade, J., May, J., & Kavanagh, D. K. (2012). Sensory imagery in craving: From cognitive psychology to new treatments for addiction. *Journal of Experimental Psychopathology, 3*(2), 127-145.

Andreassi, J. L. (2007). *Psychophysiology: Human behavior and physiological response* (5th ed.). Mahwal, NJ: Lawrence Erlbaum Associates, Inc.

Andreyeva, T., Long, M. W., Henderson, K. E., & Grode, G. M. (2010). Trying to lose weight: Diet strategies among Americans with overweight or obesity in 1996 and 2003. *Journal of the American Dietetic Association, 110*(4), 535-542.

Angst, J., Gamma, A., Endrass, J., Goodwin, R., Ajdacic, V., Eich, D., & Rössler, W. (2004). Obsessive-compulsive severity spectrum in the community: Prevalence, comorbidity, and course. *European Archives of Psychiatry and Clinical Neuroscience, 254*(3), 156-164.

Arnow, B., Kenardy, J., & Agras, W. S. (1995). The Emotional Eating Scale: The development of a measure to assess coping with negative affect by eating. *International Journal of Eating Disorders, 18*(1), 79-90.

Arns, M., de Ridder, S., Strehl, U., Breteler, M., & Coenen, A. (2009). Efficacy of neurofeedback treatment in ADHD: The effects on inattention, impulsivity and hyperactivity: A meta-analysis. *Clinical EEG and Neuroscience, 40*(3), 180-189.

Arns, M., Heinrich, H., & Strehl, U. (2014). Evaluation of neurofeedback in ADHD: The long and winding road. *Biological Psychology, 95*, 108-115.

Arnsten, A. F. (2009). Stress signalling pathways that impair prefrontal cortex structure and function. *Nature Reviews Neuroscience, 10*(6), 410-422.

Ashmore, J. A., Friedman, K. E., Reichmann, S. K., & Musante, G. J. (2008). Weight-based stigmatization, psychological distress, & binge eating behavior among obese treatment-seeking adults. *Eating Behaviors, 9*(2), 203-209.

Avena, N. M., Bocarsly, M. E., Hoebel, B. G. & Gold, M. S (2011). Overlaps in the nosology of substance abuse and overeating: the translational implications of “food addiction”. *Current Drug Abuse Reviews, 4*(3), 133-139.

Bagdasaryan, J., & Le Van Quyen, M. (2013). Experiencing your brain: Neurofeedback as a new bridge between neuroscience and phenomenology. *Frontiers in Human Neuroscience*, *7*:680, doi: 10.3389/fnhum.2013.00680.

Ball, K., & Lee, C. (2000). Relationships between psychological stress, coping and disordered eating: A review. *Psychology & Health*, *14*(6), 1007-1035.

Bandura, A. (1977). Self-efficacy: Toward a unifying theory of behavioral change. *Psychological Review*, *84*(2), 191-215.

Barry, R. J., Clarke, A. R., Johnstone, S. J., Magee, C. A., & Rushby, J. A. (2007). EEG differences between eyes-closed and eyes-open resting conditions. *Clinical Neurophysiology*, *118*(12), 2765-2773.

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.

Bauer, L. O. (2001). Predicting relapse to alcohol and drug abuse via quantitative electroencephalography. *Neuropsychopharmacology*, *25*(3), 332-340.

Baumeister, R. F., Bratslavsky, E., Muraven, M., & Tice, D. M. (1998). Ego depletion: Is the active self a limited resource?. *Journal of Personality and Social Psychology*, *74*(5), 1252-1265.

Becker, S., & Zipfel, S. (2010). Binge Eating und Binge-Eating-Störung. In G. Reich, & M. Cierpka (Eds.), *Psychotherapie der Essstörungen* (pp. 62-71). Stuttgart, Germany: Thieme.

Beglin, S. J., & Fairburn, C. G. (1992). What is meant by the term "binge"?. *The American Journal of Psychiatry*, *149*(1), 123-124.

Bellg, A. J., Borrelli, B., Resnick, B., Hecht, J., Minicucci, D. S., Ory, M., ... & Czajkowski, S. (2004). Enhancing treatment fidelity in health behavior change studies: Best practices and recommendations from the NIH Behavior Change Consortium. *Health Psychology*, *23*(5), 443-451.

Berridge, K. C. (2009). 'Liking' and 'wanting' food rewards: Brain substrates and roles in eating disorders. *Physiology & Behavior*, *97*(5), 537-550.

Berridge, K. C., Ho, C. Y., Richard, J. M., & DiFeliceantonio, A. G. (2010). The tempted brain eats: Pleasure and desire circuits in obesity and eating disorders. *Brain Research*, *1350*, 43-64.

Berthoud, H. R. (2011). Metabolic and hedonic drives in the neural control of appetite: Who is the boss?. *Current Opinion in Neurobiology*, *21*(6), 888-896.

Birbaumer, N., Ruiz, S., & Sitaram, R. (2013). Learned regulation of brain metabolism. *Trends in Cognitive Sciences*, *17*(6), 295-302.

Blackburn, S., Johnston, L., Blampied, N., Popp, D., & Kallen, R. (2006). An application of escape theory to binge eating. *European Eating Disorders Review*, *14*(1), 23-31.

Boon, B., Stroebe, W., Schut, H., & IJntema, R. (2002). Ironic processes in the eating behaviour of restrained eaters. *British Journal of Health Psychology*, *7*(1), 1-10.

Boswell, J. F., Anderson, L. M., & Anderson, D. A. (2015). Integration of interoceptive exposure in eating disorder treatment. *Clinical Psychology: Science and Practice*, *22*(2), 194-210.

Boswell, R. G., & Kober, H. (2016). Food cue reactivity and craving predict eating and weight gain: A meta-analytic review. *Obesity Reviews*, *17*(2), 159-177.

Boucsein, W. (2012). *Electrodermal activity* (2nd ed.). New York, NY: Springer.

Brenninkmeijer, J. (2013). Neurofeedback as a dance of agency. *BioSocieties*, *8*(2), 144-163.

Brosschot, J. F., Gerin, W., & Thayer, J. F. (2006). The perseverative cognition hypothesis: A review of worry, prolonged stress-related physiological activation, and health. *Journal of Psychosomatic Research*, *60*(2), 113-124.

Brownley, K. A., Berkman, N. D., Sedway, J. A., Lohr, K. N., & Bulik, C. M. (2007). Binge eating disorder treatment: A systematic review of randomized controlled trials. *International Journal of Eating Disorders*, *40*(4), 337-348.

Brunoni, A. R., Amadera, J., Berbel, B., Volz, M. S., Rizzerio, B. G., & Fregni, F. (2011). A systematic review on reporting and assessment of adverse effects associated with transcranial direct current stimulation. *The International Journal of Neuropsychopharmacology*, *14*(8), 1133-1145.

Brunstrom, J. M., Yates, H. M., & Witcomb, G. L. (2004). Dietary restraint and heightened reactivity to food. *Physiology & Behavior*, *81*(1), 85-90.

Bryant, E. J., King, N. A., & Blundell, J. E. (2008). Disinhibition: Its effects on appetite and weight regulation. *Obesity Reviews*, *9*(5), 409-419.

Bulik, C. M., Sullivan, P. F., Carter, F. A., McIntosh, V. V., & Joyce, P. R. (1998). The role of exposure with response prevention in the cognitive-behavioural therapy for bulimia nervosa. *Psychological Medicine*, *28*(3), 611-623.

Bulik, C. M., Sullivan, P. F., Carter, F. A., McIntosh, V. V., & Joyce, P. R. (1999). Predictors of rapid and sustained response to cognitive-behavioral therapy for bulimia nervosa. *International Journal of Eating Disorders*, *26*(2), 137-144.

Burmeister, J. M., Hinman, N., Koball, A., Hoffmann, D. A., & Carels, R. A. (2013). Food addiction in adults seeking weight loss treatment. Implications for psychosocial health and weight loss. *Appetite*, *60*, 103-110.

Byrd-Bredbenner, C., Quick, V., Koenings, M., Martin-Biggers, J., & Kattelmann, K. K. (2016). Relationships of cognitive load on eating and weight-related behaviors of young adults. *Eating Behaviors*, *21*, 89-94.

Campbell, M., Fitzpatrick, R., Haines, A., & Kinmonth, A. L. (2000). Framework for design and evaluation of complex interventions to improve health. *British Medical Journal*, *321*(7262), 694-696.

Cardinal, R. N., Parkinson, J. A., Hall, J., & Everitt, B. J. (2002). Emotion and motivation: The role of the amygdala, ventral striatum, and prefrontal cortex. *Neuroscience & Biobehavioral Reviews*, *26*(3), 321-352.

Carter, B. L., & Tiffany, S. T. (1999). Meta-analysis of cue-reactivity in addiction research. *Addiction*, *94*(3), 327-340.

Carter, F. A., & Bulik, C. M. (1994). Exposure treatments for bulimia nervosa: Procedure, efficacy, and mechanisms. *Advances in Behaviour Research and Therapy*, *16*(2), 77-129.

Carter, F. A., & Jansen, A. (2012). Improving psychological treatment for obesity. Which eating behaviours should we target?. *Appetite*, *58*(3), 1063-1069.

Carter, W. R., Johnson, M. C., & Borkovec, T. D. (1986). Worry: An electrocortical analysis. *Advances in Behaviour Research and Therapy*, *8*(4), 193-204.

Castellanos, E. H., Charboneau, E., Dietrich, M. S., Park, S., Bradley, B. P., Mogg, K., & Cowan, R. L. (2009). Obese adults have visual attention bias for food cue images: Evidence for altered reward system function. *International Journal of Obesity*, *33*(9), 1063-1073.

Cepeda-Benito, A., & Gleaves, D.H. (2001). A critique of food cravings research: Theory, measurement, food intake. In M.M. Hetherington (Ed.), *Food cravings and addiction* (pp.1-12). Surrey, U.K.: Leatherhead.

Chao, A. M., Grilo, C. M., & Sinha, R. (2016). Food cravings, binge eating, and eating disorder psychopathology: Exploring the moderating roles of gender and race. *Eating Behaviors*, *21*, 41-47.

Chaput, J. P., Leblanc, C., Pérusse, L., Després, J. P., Bouchard, C., & Tremblay, A. (2009). Risk factors for adult overweight and obesity in the Quebec Family Study: Have we been barking up the wrong tree?. *Obesity*, *17*(10), 1964-1970.

Christaki, E., Kokkinos, A., Costarelli, V., Alexopoulos, E. C., Chrousos, G. P., & Darviri, C. (2013). Stress management can facilitate weight loss in Greek overweight and obese women: A pilot study. *Journal of Human Nutrition and Dietetics*, *26*(s1), 132-139.

Chua, J. L., Touyz, S., & Hill, A. J. (2004). Negative mood-induced overeating in obese binge eaters: An experimental study. *International Journal of Obesity, 28*(4), 606-610.

Clyne, C., Latner, J. D., Gleaves, D. H., & Blampied, N. M. (2010). Treatment of emotional dysregulation in full syndrome and subthreshold binge eating disorder. *Eating Disorders, 18*(5), 408-424

Coker, E. L., von Lojewski, A., Luscombe, G. M., & Abraham, S. F. (2015). The difficulty in defining binge eating in obese women: How it affects prevalence levels in presurgical bariatric patients. *Eating Behaviors, 17*, 130-135.

Colles, S. L., Dixon, J. B., & O'Brien, P. E. (2008). Loss of control is central to psychological disturbance associated with binge eating disorder. *Obesity, 16*(3), 608-614.

Colloca, L., & Miller, F. G. (2011). The nocebo effect and its relevance for clinical practice. *Psychosomatic Medicine, 73*(7), 598-603.

Conklin, C. A., & Tiffany, S. T. (2002). Applying extinction research and theory to cue-exposure addiction treatments. *Addiction, 97*(2), 155-167.

Cooper, Z., & Fairburn, C. G. (2003). Refining the definition of binge eating disorder and nonpurging bulimia nervosa. *International Journal of Eating Disorders, 34*(S1), S89-S95.

Craske, M. G., Kircanski, K., Zelikowsky, M., Mystkowski, J., Chowdhury, N., & Baker, A. (2008). Optimizing inhibitory learning during exposure therapy. *Behaviour Research and Therapy, 46*(1), 5-27.

Critchley, H. D., Melmed, R. N., Featherstone, E., Mathias, C. J., & Dolan, R. J. (2001). Brain activity during biofeedback relaxation. *Brain, 124*(5), 1003-1012.

Cummings, D. E., & Overduin, J. (2007). Gastrointestinal regulation of food intake. *The Journal of Clinical Investigation, 117*(1), 13-23.

Curtis, C., & Davis, C. (2014). A qualitative study of binge eating and obesity from an addiction perspective. *Eating Disorders, 22*(1), 19-32.

Dallman, M. F. (2010). Stress-induced obesity and the emotional nervous system. *Trends in Endocrinology & Metabolism, 21*(3), 159-165.

Dallman, M. F., Pecoraro, N. C., & la Fleur, S. E. (2005). Chronic stress and comfort foods: Self-medication and abdominal obesity. *Brain, Behavior, and Immunity, 19*(4), 275-280.

Daubenmier, J., Kristeller, J., Hecht, F. M., Maninger, N., Kuwata, M., Jhaveri, K., ... & Epel, E. (2011). Mindfulness intervention for stress eating to reduce cortisol and abdominal fat among overweight and obese women: An exploratory randomized controlled study. *Journal of Obesity, 2011*: 651936, doi:10.1155/2011/651936.

Davis, C. (2013). Compulsive overeating as an addictive behavior: Overlap between food addiction and binge eating disorder. *Current Obesity Reports, 2*(2), 171-178.

Davis, C., & Carter, J. C. (2009). Compulsive overeating as an addiction disorder. A review of theory and evidence. *Appetite, 53*(1), 1-8.

Davis, C., Curtis, C., Levitan, R. D., Carter, J. C., Kaplan, A. S., & Kennedy, J. L. (2011). Evidence that 'food addiction' is a valid phenotype of obesity. *Appetite, 57*(3), 711-717.

Davis, C., Strachan, S., & Berkson, M. (2004). Sensitivity to reward: Implications for overeating and overweight. *Appetite, 42*(2), 131-138.

De França, G. V. A., Gigante, D. P., & Olinto, M. T. A. (2014). Binge eating in adults: Prevalence and association with obesity, poor self-rated health status and body dissatisfaction. *Public Health Nutrition, 17*(04), 932-938.

De Zwaan, M. (2001). Binge eating disorder and obesity. *International Journal of Obesity & Related Metabolic Disorders, 25*(Suppl. 1), S51-S55.

Dehghani-Arani, F., Rostami, R., & Nadali, H. (2013). Neurofeedback training for opiate addiction: Improvement of mental health and craving. *Applied Psychophysiology and Biofeedback*, *38*(2), 133-141.

Demos, J. N. (2005). *Getting started with neurofeedback*. New York, NY: WW Norton & Company.

Després, J. P. (2001). Health consequences of visceral obesity. *Annals of Medicine*, *33*(8), 534-541.

Dingemans, A. E., & van Furth, E. F. (2012). Binge eating disorder psychopathology in normal weight and obese individuals. *International Journal of Eating Disorders*, *45*(1), 135-138.

Drobes, D. J., Miller, E. J., Hillman, C. H., Bradley, M. M., Cuthbert, B. N., & Lang, P. J. (2001). Food deprivation and emotional reactions to food cues: Implications for eating disorders. *Biological Psychology*, *57*(1), 153-177.

Drummond, D. C. (2000). What does cue-reactivity have to offer clinical research?. *Addiction*, *95*(8S2), 129-144.

Dubé, L., LeBel, J. L., & Lu, J. (2005). Affect asymmetry and comfort food consumption. *Physiology & Behavior*, *86*(4), 559-567.

Duffy, F. H. (2000). The state of EEG biofeedback therapy (EEG operant conditioning) in 2000: An editor's opinion. *Clinical Electroencephalography*, *31*(1), v-viii.

Egger, G., & Swinburn, B. (1997). An "ecological" approach to the obesity pandemic. *British Medical Journal*, *315*(7106), 477-480.

Egner, T., & Gruzelier, J. H. (2001). Learned self-regulation of EEG frequency components affects attention and event-related brain potentials in humans. *NeuroReport*, *12*(18), 4155-4159.

Egner, T., Zech, T. F., & Gruzelier, J. H. (2004). The effects of neurofeedback training on the spectral topography of the electroencephalogram. *Clinical Neurophysiology*, *115*(11), 2452-2460.

Enriquez-Geppert, S., Huster, R. J., & Herrmann, C. S. (2013). Boosting brain functions: Improving executive functions with behavioral training, neurostimulation, and neurofeedback. *International Journal of Psychophysiology*, *88*(1), 1-16.

Evers, C., Stok, F. M., & de Ridder, D. T. (2010). Feeding your feelings: Emotion regulation strategies and emotional eating. *Personality and Social Psychology Bulletin*, *36*(6), 792-804.

Fairburn, C. G., Agras, W. S., Walsh, B. T., Wilson, G. T., & Stice, E. (2004). Prediction of outcome in bulimia nervosa by early change in treatment. *American Journal of Psychiatry*, *161*(12), 2322-2324.

Fayet, F., Petocz, P., & Samman, S. (2012). Prevalence and correlates of dieting in college women: A cross sectional study. *International Journal of Women's Health* 2012:4, 405-411, doi: 10.2147/IJWH.S33920.

Fedoroff, I. D., Polivy, J., & Herman, C. P. (1997). The effect of pre-exposure to food cues on the eating behavior of restrained and unrestrained eaters. *Appetite*, *28*(1), 33-47.

Fernández-Aranda, F., Pinheiro, A. P., Thornton, L. M., Berrettini, W. H., Crow, S., Fichter, M. M., ... & Rotondo, A. (2008). Impulse control disorders in women with eating disorders. *Psychiatry Research*, *157*(1), 147-157.

Ferriday, D., & Brunstrom, J. M. (2008). How does food-cue exposure lead to larger meal sizes?. *British Journal of Nutrition*, *100*(6), 1325-1332.

Ferriday, D., & Brunstrom, J. M. (2011). 'I just can't help myself': Effects of food-cue exposure in overweight and lean individuals. *International Journal of Obesity*, *35*(1), 142-149.

Filbey, F. M., Myers, U. S., & DeWitt, S. (2012). Reward circuit function in high BMI individuals with compulsive overeating: Similarities with addiction. *NeuroImage*, *63*(4), 1800-1806.

Finkelstein, E. A., Brown, D. S., Wrage, L. A., Allaire, B. T., & Hoerger, T. J. (2010). Individual and aggregate years-of-life-lost associated with overweight and obesity. *Obesity*, *18*(2), 333-339.

Finucane, M. M., Stevens, G. A., Cowan, M. J., Danaei, G., Lin, J. K., Paciorek, C. J., ... & Farzadfar, F. (2011). National, regional, and global trends in body-mass index since 1980: Systematic analysis

of health examination surveys and epidemiological studies with 960 country-years and 9·1 million participants. *The Lancet*, *377*(9765), 557-567.

Fliege, H., Rose, M., Arck, P., Levenstein, S., & Klapp, B. F. (2001). Validierung des "Perceived Stress Questionnaire"(PSQ) an einer deutschen Stichprobe. *Diagnostica*, *47*(3), 142-152.

Frank, S., Lee, S., Preissl, H., Schultes, B., Birbaumer, N., & Veit, R. (2012). The obese brain athlete: Self-regulation of the anterior insula in adiposity. *PloS one*, *7*(8):e42570, doi: 10.1371/journal.pone.0042570.

French, S. A., Jeffery, R. W., Sherwood, N. E., & Neumark-Sztainer, D. (1999). Prevalence and correlates of binge eating in a nonclinical sample of women enrolled in a weight gain prevention program. *International Journal of Obesity*, *23*(6), 576-585.

French, S. A., Story, M., & Jeffery, R. W. (2001). Environmental influences on eating and physical activity. *Annual Review of Public Health*, *22*(1), 309-335.

Friederich, H. C., Schild, S., Schellberg, D., Quenter, A., Bode, C., Herzog, W., & Zipfel, S. (2006). Cardiac parasympathetic regulation in obese women with binge eating disorder. *International Journal of Obesity*, *30*(3), 534-542.

Friederich, H. C., Wu, M., Simon, J. J., & Herzog, W. (2013). Neurocircuit function in eating disorders. *International Journal of Eating Disorders*, *46*(5), 425-432.

Friedman, K. E., Reichmann, S. K., Costanzo, P. R., & Musante, G. J. (2002). Body image partially mediates the relationship between obesity and psychological distress. *Obesity Research*, *10*(1), 33-41.

Furlong, T. M., Vianna, D. M., Liu, L., & Carrive, P. (2009). Hypocretin / orexin contributes to the expression of some but not all forms of stress and arousal. *European Journal of Neuroscience*, *30*(8), 1603-1614.

Gearhardt, A. N., Corbin, W. R., & Brownell, K. D. (2009). Food addiction: An examination of the diagnostic criteria for dependence. *Journal of Addiction Medicine*, *3*(1), 1-7.

Gearhardt, A. N., Grilo, C. M., DiLeone, R. J., Brownell, K. D., & Potenza, M. N. (2011). Can food be addictive? Public health and policy implications. *Addiction*, *106*(7), 1208-1212.

Gearhardt, A. N., Yokum, S., Orr, P. T., Stice, E., Corbin, W. R., & Brownell, K. D. (2011). Neural correlates of food addiction. *Archives of General Psychiatry*, *68*(8), 808-816.

Geisler, F. C., Kleinfeldt, A., & Kubiak, T. (2016). Restrained eating predicts effortful self-control as indicated by heart rate variability during food exposure. *Appetite*, *96*, 502-508.

Gendall, K. A., Joyce, P. R., Sullivan, P. F., & Bulik, C. M. (1998). Food cravers: Characteristics of those who binge. *International Journal of Eating Disorders*, *23*(4), 353-360.

Gianini, L. M., White, M. A., & Masheb, R. M. (2013). Eating pathology, emotion regulation, and emotional overeating in obese adults with binge eating disorder. *Eating Behaviors*, *14*(3), 309-313.

Gibson, E. L. (2006). Emotional influences on food choice: Sensory, physiological and psychological pathways. *Physiology & Behavior*, *89*(1), 53-61.

Giel, K. E., Teufel, M., Friederich, H. C., Hautzinger, M., Enck, P., & Zipfel, S. (2011). Processing of pictorial food stimuli in patients with eating disorders—A systematic review. *International Journal of Eating Disorders*, *44*(2), 105-117.

Ginty, A. T., Phillips, A. C., Higgs, S., Heaney, J. L., & Carroll, D. (2012). Disordered eating behaviour is associated with blunted cortisol and cardiovascular reactions to acute psychological stress. *Psychoneuroendocrinology*, *37*(5), 715-724.

Glannon, W. (2014). Neuromodulation, agency and autonomy. *Brain Topography*, *27*(1), 46-54.

Glasofer, D. R., Haaga, D. A., Hannallah, L., Field, S. E., Kozlosky, M., Reynolds, J., ... & Tanofsky-Kraff, M. (2013). Self-efficacy beliefs and eating behavior in adolescent girls at-risk for excess weight gain and binge eating disorder. *International Journal of Eating Disorders*, *46*(7), 663-668.

Goldstein, S. P., Forman, E. M., Meiran, N., Herbert, J. D., Juarascio, A. S., & Butryn, M. L. (2014). The discrepancy between implicit and explicit attitudes in predicting disinhibited eating. *Eating Behaviors, 15*(1), 164-170.

Goodrick, G. K., Pendleton, V. R., Kimball, K. T., Poston II, W. S. C., Reeves, R. S., & Foreyt, J. P. (1999). Binge eating severity, self-concept, dieting self-efficacy and social support during treatment of binge eating disorder. *International Journal of Eating Disorders, 26*(3), 295-300.

Goodrick, G. K., Poston II, W. S. C., Kimball, K. T., Reeves, R. S., & Foreyt, J. P. (1998). Nondieting versus dieting treatment for overweight binge-eating women. *Journal of Consulting and Clinical Psychology, 66*(2), 363-368.

Gormally, J., Black, S., Daston, S., & Rardin, D. (1982). The assessment of binge eating severity among obese persons. *Addictive Behaviors, 7*(1), 47-55.

Gray, J. A. (1987). Perspectives on anxiety and impulsivity: A commentary. *Journal of Research in Personality, 21*(4), 493-509.

Green, M. W., Rogers, P. J., & Elliman, N. A. (2000). Dietary restraint and addictive behaviors: The generalizability of Tiffany's cue reactivity model. *International Journal of Eating Disorders, 27*, 419-427.

Greeno, C. G., & Wing, R. R. (1994). Stress-induced eating. *Psychological Bulletin, 115*(3), 444-464.

Greeno, C. G., Wing, R. R., & Shiffman, S. (2000). Binge antecedents in obese women with and without binge eating disorder. *Journal of Consulting and Clinical Psychology, 68*(1), 95-102.

Griffiths, R. A., Mallia-Blanco, R., Boesenberg, E., Ellis, C., Fischer, K., Taylor, M., & Wyndham, J. (2000). Restrained eating and sociocultural attitudes to appearance and general dissatisfaction. *European Eating Disorders Review, 8*(5), 394-402.

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.

Grilo, C. M., Masheb, R. M., & Wilson, G. T. (2001). A comparison of different methods for assessing the features of eating disorders in patients with binge eating disorder. *Journal of Consulting and Clinical Psychology, 69*(2), 317-322.

Groesz, L. M., McCoy, S., Carl, J., Saslow, L., Stewart, J., Adler, N., ... & Epel, E. (2012). What is eating you? Stress and the drive to eat. *Appetite, 58*(2), 717-721.

Gross, J. J. (1998). The emerging field of emotion regulation: An integrative review. *Review of General Psychology, 2*(3), 271-299.

Grucza, R. A., Przybeck, T. R., & Cloninger, C. R. (2007). Prevalence and correlates of binge eating disorder in a community sample. *Comprehensive Psychiatry, 48*(2), 124-131.

Gruzelier, J. H. (2014). EEG-neurofeedback for optimising performance III: A review of methodological and theoretical considerations. *Neuroscience & Biobehavioral Reviews, 44*, 159-182.

Gruzelier, J. H., Foks, M., Steffert, T., Chen, M. L., & Ros, T. (2014). Beneficial outcome from EEG-neurofeedback on creative music performance, attention and well-being in school children. *Biological Psychology, 95*, 86-95.

Guerrieri, R., Nederkoorn, C., & Jansen, A. (2008). The interaction between impulsivity and a varied food environment: Its influence on food intake and overweight. *International Journal of Obesity, 32*(4), 708-714.

Guh, D. P., Zhang, W., Bansback, N., Amarsi, Z., Birmingham, C. L., & Anis, A. H. (2009). The incidence of co-morbidities related to obesity and overweight: A systematic review and meta-analysis. *BMC Public Health, 9*:88, doi: 10.1186/1471-2458-9-88.

Habhab, S., Sheldon, J. P., & Loeb, R. C. (2009). The relationship between stress, dietary restraint, and food preferences in women. *Appetite, 52*(2), 437-444.

Haedt-Matt, A. A., & Keel, P. K. (2011a). Hunger and binge eating: A meta-analysis of studies using ecological momentary assessment. *International Journal of Eating Disorders*, *44*(7), 573-578.

Haedt-Matt, A. A., & Keel, P. K. (2011b). Revisiting the affect regulation model of binge eating: A meta-analysis of studies using ecological momentary assessment. *Psychological Bulletin*, *137*(4), 660–681.

Hagger, M. S., Wood, C., Stiff, C., & Chatzisarantis, N. L. (2010). Ego depletion and the strength model of self-control: A meta-analysis. *Psychological Bulletin*, *136*(4), 495-525.

Haines, J., & Neumark-Sztainer, D. (2006). Prevention of obesity and eating disorders: A consideration of shared risk factors. *Health Education Research*, *21*(6), 770-782.

HajiHosseini, A., & Holroyd, C. B. (2015). Reward feedback stimuli elicit high-beta EEG oscillations in human dorsolateral prefrontal cortex. *Scientific Reports*, *5*:13021, doi:10.1038/srep1302.

Hall, M., Buysse, D. J., Nowell, P. D., Nofzinger, E. A., Houck, P., Reynolds III, C. F., & Kupfer, D. J. (2000). Symptoms of stress and depression as correlates of sleep in primary insomnia. *Psychosomatic Medicine*, *62*(2), 227-230.

Hamilton, J., Fawson, S., May, J., Andrade, J., & Kavanagh, D. J. (2013). Brief guided imagery and body scanning interventions reduce food cravings. *Appetite*, *71*, 158-162.

Hammond, D. C. (2006). What is neurofeedback?. *Journal of Neurotherapy*, *10*(4), 25-36.

Hanlon, C. A., Hartwell, K. J., Canterberry, M., Li, X., Owens, M., LeMatty, T., ... & George, M. S. (2013). Reduction of cue-induced craving through realtime neurofeedback in nicotine users: The role of region of interest selection and multiple visits. *Psychiatry Research: Neuroimaging*, *213*(1), 79-81.

Harvey, K., Kemps, E., & Tiggemann, M. (2005). The nature of imagery processes underlying food cravings. *British Journal of Health Psychology*, *10*(1), 49-56.

Havermans, R. C., & Jansen, A. T. M. (2003). Increasing the efficacy of cue exposure treatment in preventing relapse of addictive behavior. *Addictive Behaviors*, *28*(5), 989-994.

Hawkins, N., Richards, P. S., Granley, H. M., & Stein, D. M. (2004). The impact of exposure to the thin-ideal media image on women. *Eating Disorders*, *12*(1), 35-50.

Hay, P. (2013). A systematic review of evidence for psychological treatments in eating disorders: 2005–2012. *International Journal of Eating Disorders*, *46*(5), 462-469.

Hayashi, T., Okamoto, E., Nishimura, H., Mizuno-Matsumoto, Y., Ishii, R., & Ukai, S. (2009). Beta activities in EEG associated with emotional stress. *International Journal of Intelligent Computing in Medical Sciences & Image Processing*, *3*(1), 57-68.

Hays, N. P., & Roberts, S. B. (2008). Aspects of eating behaviors "disinhibition" and "restraint" are related to weight gain and BMI in women. *Obesity*, *16*(1), 52-58.

Hays, N. P., Bathalon, G. P., McCrory, M. A., Roubenoff, R., Lipman, R., & Roberts, S. B. (2002). Eating behavior correlates of adult weight gain and obesity in healthy women aged 55–65 y. *The American Journal of Clinical Nutrition*, *75*(3), 476-483.

Heatherton, T. F., & Baumeister, R. F. (1991). Binge eating as escape from self-awareness. *Psychological Bulletin*, *110*(1), 86-108.

Heatherton, T. F., Herman, C. P., Polivy, J., King, G. A., & McGree, S. T. (1988). The (mis) measurement of restraint: An analysis of conceptual and psychometric issues. *Journal of Abnormal Psychology*, *97*(1), 19-28.

Hemmingsson, E. (2014). A new model of the role of psychological and emotional distress in promoting obesity: Conceptual review with implications for treatment and prevention. *Obesity Reviews*, *15*(9), 769-779.

Henriques, J. B., & Davidson, R. J. (1991). Left frontal hypoactivation in depression. *Journal of Abnormal Psychology*, *100*(4), 535-545.

Herman, C. P., & Mack, D. (1975). Restrained and unrestrained eating. *Journal of Personality*, *43*(4), 647-660.

Herman, C. P., & Polivy, J. (1975). Anxiety, restraint, and eating behavior. *Journal of Abnormal Psychology*, *84*(6), 666-672.

Herman, C. P., & Polivy, J. (1980). Restrained eating. In A. Stunkard (Ed.), *Obesity* (pp. 208-225). Philadelphia, PA: Saunders.

Herman, C. P., & Polivy, J. (1984). A boundary model for the regulation of eating. In A. J. Stunkard, & E. Stellar (Eds.), *Eating and its Disorders* (pp. 141-156). New York, NY: Raven Press.

Herman, C. P., Olmsted, M. P., & Polivy, J. (1983). Obesity, externality, and susceptibility to social influence: An integrated analysis. *Journal of Personality and Social Psychology*, *45*(4), 926-934.

Hilbert, A., De Zwaan, M., & Braehler, E. (2012). How frequent are eating disturbances in the population? Norms of the Eating Disorder Examination-Questionnaire. *PloS one*, *7*: e29125, doi: 10.1371/journal.pone.0029125.

Hilbert, A., Hildebrandt, T., Agras, W. S., Wilfley, D. E., & Wilson, G. T. (2015). Rapid response in psychological treatments for binge eating disorder. *Journal of Consulting and Clinical Psychology*, *83*(3), 649-654.

Hill, A. J. (2007). The psychology of food craving. *Proceedings of the Nutrition Society*, *66*(02), 277-285.

Hill, A. J., Weaver, C. F., & Blundell, J. E. (1991). Food craving, dietary restraint and mood. *Appetite*, *17*(3), 187-197.

Hofmann, W., Rauch, W., & Gawronski, B. (2007). And deplete us not into temptation: Automatic attitudes, dietary restraint, and self-regulatory resources as determinants of eating behavior. *Journal of Experimental Social Psychology*, *43*(3), 497-504.

Holroyd, K. A., Penzien, D. B., Hursey, K. G., Tobin, D. L., Rogers, L., Holm, J. E., ... & Chila, A. G. (1984). Change mechanisms in EMG biofeedback training: Cognitive changes underlying improvements in tension headache. *Journal of Consulting and Clinical Psychology*, *52*(6), 1039-1053.

Hu, F. B. (2003). Overweight and obesity in women: Health risks and consequences. *Journal of Women's Health*, *12*(2), 163-172.

Hudson, J. I., Hiripi, E., Pope, H. G., & Kessler, R. C. (2007). The prevalence and correlates of eating disorders in the National Comorbidity Survey Replication. *Biological Psychiatry*, *61*(3), 348-358.

Hume, D. J., Howells, F. M., Karpul, D., Rauch, H. L., Kroff, J., & Lambert, E. V. (2015). Cognitive control over visual food cue saliency is greater in reduced-overweight/obese but not in weight relapsed women: An EEG study. *Eating Behaviors*, *19*, 76-80.

Hume, D. J., Howells, F. M., Rauch, H. L., Kroff, J., & Lambert, E. V. (2015). Electrophysiological indices of visual food cue-reactivity. Differences in obese, overweight and normal weight women. *Appetite*, *85*, 126-137.

Iacovino, J. M., Gredysa, D. M., Altman, M., & Wilfley, D. E. (2012). Psychological treatments for binge eating disorder. *Current Psychiatry Reports*, *14*(4), 432-446.

Isnard, P., Michel, G., Frelut, M. L., Vila, G., Falissard, B., Naja, W., ... & Mouren-Simeoni, M. C. (2003). Binge eating and psychopathology in severely obese adolescents. *International Journal of Eating Disorders*, *34*(2), 235-243.

Jääskeläinen, A., Nevanperä, N., Remes, J., Rahkonen, F., Järvelin, M. R., & Laitinen, J. (2014). Stress-related eating, obesity and associated behavioural traits in adolescents: A prospective population-based cohort study. *BMC Public Health*, *14*:321, doi: 10.1186/1471-2458-14-321.

James, W. P. T. (2008). WHO recognition of the global obesity epidemic. *International Journal of Obesity*, *32*, S120-S126.

Jansen, A. (1998). A learning model of binge eating: Cue reactivity and cue exposure. *Behaviour Research and Therapy*, *36*(3), 257-272.

Jansen, A., Broekmate, J., & Heymans, M. (1992). Cue-exposure vs self-control in the treatment of binge eating: A pilot study. *Behaviour Research and Therapy*, *30*(3), 235-241.

Jansen, A., Nederkoorn, C., van Baak, L., Keirse, C., Guerrieri, R., & Havermans, R. (2009). High-restrained eaters only overeat when they are also impulsive. *Behaviour Research and Therapy*, *47*(2), 105-110.

Jansen, A., Schyns, G., Bongers, P., & van den Akker, K. (2016). From lab to clinic: Extinction of cued cravings to reduce overeating. *Physiology & Behavior*, *162*, 174-180.

Jansen, A., Van den Hout, M. A., De Loof, C., Zandbergen, J., & Griez, E. (1989). A case of bulimia successfully treated by cue exposure. *Journal of Behavior Therapy and Experimental Psychiatry*, *20*(4), 327-332.

Jasinska, A. J., Yasuda, M., Burant, C. F., Gregor, N., Khatri, S., Sweet, M., & Falk, E. B. (2012). Impulsivity and inhibitory control deficits are associated with unhealthy eating in young adults. *Appetite*, *59*(3), 738-747.

Jasper, H., & Shagass, C. (1941). Conditioning the occipital alpha rhythm in man. *Journal of Experimental Psychology*, *28*, 373-387.

Jastreboff, A. M., Sinha, R., Lacadie, C., Small, D. M., Sherwin, R. S., & Potenza, M. N. (2013). Neural correlates of stress- and food cue-induced food craving in obesity: Association with insulin levels. *Diabetes Care*, *36*(2), 394-402.

Jena, S. K. (2015). Examination stress and its effect on EEG. *International Journal of Medical Science and Public Health*, *11*(4), 1493-1497.

Jia, H., & Lubetkin, E. I. (2005). The impact of obesity on health-related quality-of-life in the general adult US population. *Journal of Public Health*, *27*(2), 156-164.

Kahan, D., Polivy, J., & Herman, C. P. (2003). Conformity and dietary disinhibition: A test of the ego-strength model of self-regulation. *International Journal of Eating Disorders*, *33*(2), 165-171.

Kahn, S. E., Hull, R. L., & Utzschneider, K. M. (2006). Mechanisms linking obesity to insulin resistance and type 2 diabetes. *Nature*, *444*(7121), 840-846.

Kaplan, H. I., & Kaplan, H. S. (1957). The psychosomatic concept of obesity. *Journal of Nervous and Mental Disease*, *125*, 181-201.

Katterman, S. N., Kleinman, B. M., Hood, M. M., Nackers, L. M., & Corsica, J. A. (2014). Mindfulness meditation as an intervention for binge eating, emotional eating, and weight loss: A systematic review. *Eating Behaviors*, *15*(2), 197-204.

Katzer, L., Bradshaw, A. J., Horwath, C. C., Gray, A. R., O'Brien, S., & Joyce, J. (2008). Evaluation of a "nondieting" stress reduction program for overweight women: A randomized trial. *American Journal of Health Promotion*, *22*(4), 264-274.

Katzman, M. A., Wolchik, S. A., & Braver, S. L. (1984). The prevalence of frequent binge eating and bulimia in a nonclinical college sample. *International Journal of Eating Disorders*, *3*(3), 53-62.

Kavanagh, D. J., Andrade, J., & May, J. (2005). Imaginary relish and exquisite torture: The elaborated intrusion theory of desire. *Psychological Review*, *112*(2), 446-467.

Kearney, D. J., Milton, M. L., Malte, C. A., McDermott, K. A., Martinez, M., & Simpson, T. L. (2012). Participation in mindfulness-based stress reduction is not associated with reductions in emotional eating or uncontrolled eating. *Nutrition Research*, *32*(6), 413-420.

Keel, P. K., & Heatherton, T. F. (2010). Weight suppression predicts maintenance and onset of bulimic syndromes at 10-year follow-up. *Journal of Abnormal Psychology*, *119*(2), 268-275.

Keel, P. K., Baxter, M. G., Heatherton, T. F., & Joiner Jr., T. E. (2007). A 20-year longitudinal study of body weight, dieting, and eating disorder symptoms. *Journal of Abnormal Psychology*, *116*(2), 422-432.

Keith, J. R., Rapgay, L., Theodore, D., Schwartz, J. M., & Ross, J. L. (2015). An assessment of an automated EEG biofeedback system for attention deficits in a substance use disorders residential treatment setting. *Psychology of Addictive Behaviors*, *29*(1), 17-25.

Kemps, E., & Tiggemann, M. (2007). Modality-specific imagery reduces cravings for food: An application of the elaborated intrusion theory of desire to food craving. *Journal of Experimental Psychology: Applied*, *13*(2), 95-104.

Kessler, R. C., Berglund, P. A., Chiu, W. T., Deitz, A. C., Hudson, J. I., Shahly, V., ... & Bruffaerts, R. (2013). The prevalence and correlates of binge eating disorder in the World Health Organization World Mental Health Surveys. *Biological Psychiatry*, *73*(9), 904-914.

Klatzkin, R. R., Gaffney, S., Cyrus, K., Bigus, E., & Brownley, K. A. (2015). Binge eating disorder and obesity: Preliminary evidence for distinct cardiovascular and psychological phenotypes. *Physiology & Behavior*, *142*, 20-27.

Knäuper, B., Pillay, R., Lacaille, J., McCollam, A., & Kelso, E. (2011). Replacing craving imagery with alternative pleasant imagery reduces craving intensity. *Appetite*, *57*(1), 173-178.

Knott, V. J., Naccache, L., Cyr, E., Fisher, D. J., McIntosh, J. F., Millar, A. M., & Villeneuve, C. M. (2008). Craving-induced EEG reactivity in smokers: Effects of mood induction, nicotine dependence and gender. *Neuropsychobiology*, *58*(3-4), 187-199.

Knott, V., Cosgrove, M., Villeneuve, C., Fisher, D., Millar, A., & McIntosh, J. (2008). EEG correlates of imagery-induced cigarette craving in male and female smokers. *Addictive behaviors*, *33*(4), 616-621.

Koo-Loeb, J. H., Costello, N., Light, K. C., & Girdler, S. S. (2000). Women with eating disorder tendencies display altered cardiovascular, neuroendocrine, and psychosocial profiles. *Psychosomatic Medicine*, *62*(4), 539-548.

Kopelman, P. (2007). Health risks associated with overweight and obesity. *Obesity Reviews*, *8*(S1), 13-17.

Korn, H.-J., & Niepoth, L. (2009). Essstörungen. In A. Martin & W. Rief (Eds.), *Wie wirksam ist Biofeedback? Eine therapeutische Methode* (pp. 89-90). Bern, Switzerland: Huber.

Kotchoubey, B., Kübler, A., Strehl, U., Flor, H., & Birbaumer, N. (2002). Can humans perceive their brain states?. *Consciousness and Cognition*, *11*(1), 98-113.

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.

Lafay, L., Thomas, F., Mennen, L., Charles, M. A., Eschwege, E., & Borys, J. M. (2001). Gender differences in the relation between food cravings and mood in an adult community: Results from the Fleurbaix Laventie Ville Sante study. *International Journal of Eating Disorders*, *29*(2), 195-204.

Lally, P., Van Jaarsveld, C. H., Potts, H. W., & Wardle, J. (2010). How are habits formed: Modelling habit formation in the real world. *European Journal of Social Psychology*, *40*(6), 998-1009.

Larsen, J. K., Hermans, R. C., & Engels, R. C. (2012). Food intake in response to food-cue exposure. Examining the influence of duration of the cue exposure and trait impulsivity. *Appetite*, *58*(3), 907-913.

Latner, J. D., Hildebrandt, T., Rosewall, J. K., Chisholm, A. M., & Hayashi, K. (2007). Loss of control over eating reflects eating disturbances and general psychopathology. *Behaviour Research and Therapy*, *45*(9), 2203-2211.

Latner, J. D., Vallance, J. K., & Buckett, G. (2008). Health-related quality of life in women with eating disorders: Association with subjective and objective binge eating. *Journal of Clinical Psychology in Medical Settings*, *15*(2), 148-153.

La Vaque, T. J., & Rossiter, T. (2001). The ethical use of placebo controls in clinical research: the Declaration of Helsinki. *Applied Psychophysiology and Biofeedback*, *26*(1), 23-37.

Lazarus, R. S. (1991). Cognition and motivation in emotion. *American Psychologist*, *46*(4), 352-367.

Lazarus, R. S. (2000). Toward better research on stress and coping. *American Psychologist, 55*(6), 665-673.

Leehr, E. J., Krohmer, K., Schag, K., Dresler, T., Zipfel, S., & Giel, K. E. (2015). Emotion regulation model in binge eating disorder and obesity–a systematic review. *Neuroscience & Biobehavioral Reviews, 49*, 125-134.

Lewinsohn, P. M., Seeley, J. R., Moerk, K. C., & Striegel-Moore, R. H. (2002). Gender differences in eating disorder symptoms in young adults. *International Journal of Eating Disorders, 32*(4), 426-440.

Linde, J. A., Jeffery, R. W., Levy, R. L., Sherwood, N. E., Utter, J., Pronk, N. P., & Boyle, R. G. (2004). Binge eating disorder, weight control self-efficacy, and depression in overweight men and women. *International Journal of Obesity, 28*(3), 418-425.

Linde, J. A., Rothman, A. J., Baldwin, A. S., & Jeffery, R. W. (2006). The impact of self-efficacy on behavior change and weight change among overweight participants in a weight loss trial. *Health Psychology, 25*(3), 282-291.

Littel, M., Franken, I. H., & Van Strien, J. W. (2009). Changes in the electroencephalographic spectrum in response to smoking cues in smokers and ex-smokers. *Neuropsychobiology, 59*(1), 43-50.

Liu, X., Vaupel, D. B., Grant, S., & London, E. D. (1998). Effect of cocaine-related environmental stimuli on the spontaneous electroencephalogram in polydrug abusers. *Neuropsychopharmacology, 19*(1), 10-17.

Lobera, I. J. (2012). Neurophysiological basis of food craving. In S. Magdeldin (Ed.), *State of the Art of therapeutic endocrinology* (pp.29-44). Retrieved from http://cdn.intechopen.com/pdfs-wm/39488.pdf

Loeber, S., Croissant, B., Heinz, A., Mann, K., & Flor, H. (2006). Cue exposure in the treatment of alcohol dependence: Effects on drinking outcome, craving and self-efficacy. *British Journal of Clinical Psychology, 45*(4), 515-529.

Loeber, S., Grosshans, M., Korucuoglu, O., Vollmert, C., Vollstädt-Klein, S., Schneider, S., . . . & Kiefer, F. (2012). Impairment of inhibitory control in response to food associated cues and attentional bias of obese participants and normal-weight controls. *International Journal of Obesity, 36*(10), 334-1339.

Loth, K., Wall, M., Larson, N., & Neumark-Sztainer, D. (2015). Disordered eating and psychological well-being in overweight and nonoverweight adolescents: Secular trends from 1999 to 2010. *International Journal of Eating Disorders, 48*(3), 323-327.

Lövdén, M., Bäckman, L., Lindenberger, U., Schaefer, S., & Schmiedek, F. (2010). A theoretical framework for the study of adult cognitive plasticity. *Psychological Bulletin, 136*(4), 659-676.

Loxton, N. J., Dawe, S., & Cahill, A. (2011). Does negative mood drive the urge to eat? The contribution of negative mood, exposure to food cues and eating style. *Appetite, 56*(2), 368-374.

Luppino, F. S., de Wit, L. M., Bouvy, P. F., Stijnen, T., Cuijpers, P., Penninx, B. W., & Zitman, F. G. (2010). Overweight, obesity, and depression: A systematic review and meta-analysis of longitudinal studies. *Archives of General Psychiatry, 67*(3), 220-229.

Machado, P. P., Machado, B. C., Gonçalves, S., & Hoek, H. W. (2007). The prevalence of eating disorders not otherwise specified. *International Journal of Eating Disorders, 40*(3), 212-217.

Macht, M. (2008). How emotions affect eating: A five-way model. *Appetite, 50*(1), 1-11.

Macht, M., Haupt, C., & Ellgring, H. (2005). The perceived function of eating is changed during examination stress: A field study. *Eating Behaviors, 6*(2), 109-112.

Maes, J., Verbraecken, J., Willemen, M., De Volder, I., van Gastel, A., Michiels, N., ... & Willemen, T. (2014). Sleep misperception, EEG characteristics and autonomic nervous system activity in primary insomnia: A retrospective study on polysomnographic data. *International Journal of Psychophysiology, 91*(3), 163-171.

Mailloux, G., Bergeron, S., Meilleur, D., D'Antono, B., & Dubé, I. (2014). Examining the associations between overeating, disinhibition, and hunger in a nonclinical sample of college women. *International Journal of Behavioral Medicine*, *21*(2), 375-384.

Mann, T., Tomiyama, A. J., Westling, E., Lew, A. M., Samuels, B., & Chatman, J. (2007). Medicare's search for effective obesity treatments: Diets are not the answer. *American Psychologist*, *62*(3), 220-233.

Mansell, W. (2005). Control theory and psychopathology: An integrative approach. *Psychology and Psychotherapy: Theory, Research and Practice*, *78*(2), 141-178.

Manzoni, G. M., Gorini, A., Preziosa, A., Pagnini, F., Castelnuovo, G., Molinari, E., & Riva, G. (2008). New technologies and relaxation: An explorative study on obese patients with emotional eating. *Journal of Cybertherapy and Rehabilitation*, *1*(2), 182-193.

Marlatt, G. A. (1990). Cue exposure and relapse prevention in the treatment of addictive behaviors. *Addictive Behaviors*, *15*(4), 395-399.

Masheb, R. M., & Grilo, C. M. (2007). Rapid response predicts treatment outcomes in binge eating disorder: Implications for stepped care. *Journal of Consulting and Clinical Psychology*, *75*(4), 639-644.

Masterpasqua, F., & Healey, K. N. (2003). Neurofeedback in psychological practice. *Professional Psychology: Research and Practice*, *34*(6), 652-656.

Mauler, B. I., Hamm, A. O., Weike, A. I., & Tuschen-Caffier, B. (2006). Affect regulation and food intake in bulimia nervosa: Emotional responding to food cues after deprivation and subsequent eating. *Journal of Abnormal Psychology*, *115*(3), 567-579.

May, J., Andrade, J., Kavanagh, D. J., & Hetherington, M. (2012). Elaborated Intrusion Theory: A cognitive-emotional theory of food craving. *Current Obesity Reports*, *1*(2), 114-121.

McCarthy, M. (1990). The thin ideal, depression, and eating disorders in women. *Behaviour Research and Therapy*, *28*(3), 205-218.

McClelland, J., Bozhilova, N., Campbell, I., & Schmidt, U. (2013). A systematic review of the effects of neuromodulation on eating and body weight: Evidence from human and animal studies. *European Eating Disorders Review*, *21*(6), 436-455.

McEwen, B. S. (2000). The neurobiology of stress: From serendipity to clinical relevance. *Brain Research*, *886*(1), 172-189.

McEwen, B. S. (2007). Physiology and neurobiology of stress and adaptation: Central role of the brain. *Physiological Reviews*, *87*(3), 873-904.

McIntosh, V. V. W., Carter, F. A., Bulik, C. M., Frampton, C. M. A., & Joyce, P. R. (2011). Five-year outcome of cognitive behavioral therapy and exposure with response prevention for bulimia nervosa. *Psychological Medicine*, *41*(5), 1061-1071.

McLaren, L. (2007). Socioeconomic status and obesity. *Epidemiologic Reviews*, *29*(1), 29-48.

Mensink, G. B., Schienkiewitz, A., Haftenberger, M., Lampert, T., Ziese, T., & Scheidt-Nave, C. (2013). Übergewicht und Adipositas in Deutschland. *Bundesgesundheitsblatt-Gesundheitsforschung-Gesundheitsschutz*, *56*(5-6), 786-794.

Messerli-Bürgy, N., Engesser, C., Lemmenmeier, E., Steptoe, A., & Laederach-Hofmann, K. (2010). Cardiovascular stress reactivity and recovery in bulimia nervosa and binge eating disorder. *International Journal of Psychophysiology*, *78*(2), 163-168.

Meule, A., & Gearhardt, A. N. (2014). Food addiction in the light of DSM-5. *Nutrients*, *6*(9), 3653-3671.

Meule, A., Freund, R., Skirde, A. K., Vögele, C., & Kübler, A. (2012). Heart rate variability biofeedback reduces food cravings in high food cravers. *Applied Psychophysiology and Biofeedback*, *37*(4), 241-251.

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, doi: 10.3389/fpsyg.2014.00190.

Meule, A., Lutz, A., Vögele, C., & Kübler, A. (2012a). Food cravings discriminate differentially between successful and unsuccessful dieters and non-dieters. Validation of the Food Cravings Questionnaires in German. *Appetite, 58*(1), 88-97.

Meule, A., Lutz, A., Vögele, C., & Kübler, A. (2012b). Self-reported dieting success is associated with cardiac autonomic regulation in current dieters. *Appetite, 59*(2), 494-498.

Meule, A., Papies, E. K., & Kübler, A. (2012). Differentiating between successful and unsuccessful dieters. Validity and reliability of the Perceived Self-Regulatory Success in Dieting Scale. *Appetite, 58*(3), 822-826.

Meule, A., Vögele, C., & Kübler, A. (2012). Restrained eating is related to accelerated reaction to high caloric foods and cardiac autonomic dysregulation. *Appetite, 58*(2), 638-644.

Meyers, A. W., & Stunkard, A. J. (1980). Food accessibility and food choice: A test of Schachter's externality hypothesis. *Archives of General Psychiatry, 37*(10), 1133-1135.

Milich, R. S. (1975). A critical analysis of Schachter's externality theory of obesity. *Journal of Abnormal Psychology, 84*(5), 586-588.

Miller, N. E. (1978). Biofeedback and visceral learning. *Annual Review of Psychology, 29*(1), 373-404.

Mitchell, J. T., & Nelson-Gray, R. O. (2006). Attention-deficit/hyperactivity disorder symptoms in adults: Relationship to Gray's behavioral approach system. *Personality and Individual Differences, 40*(4), 749-760.

Mokdad, A. H., Marks, J. S., Stroup, D. F., & Gerberding, J. L. (2004). Actual causes of death in the United States, 2000. *The Journal of the American Medicinal Association, 291*(10), 1238-1245.

Mond, J. M., Latner, J. D., Hay, P. H., Owen, C., & Rodgers, B. (2010). Objective and subjective bulimic episodes in the classification of bulimic-type eating disorders: Another nail in the coffin of a problematic distinction. *Behaviour Research and Therapy, 48*(7), 661-669.

Montani, J. P., Schutz, Y., & Dulloo, A. G. (2015). Dieting and weight cycling as risk factors for cardiometabolic diseases: Who is really at risk? *Obesity Reviews, 16*(S1), 7-18.

Mooney, K. M., & Walbourn, L. (2001). When college students reject food: Not just a matter of taste. *Appetite, 36*(1), 41-50.

Morton, G. J., Cummings, D. E., Baskin, D. G., Barsh, G. S., & Schwartz, M. W. (2006). Central nervous system control of food intake and body weight. *Nature, 443*(7109), 289-295.

Muraven, M., & Baumeister, R. F. (2000). Self-regulation and depletion of limited resources: Does self-control resemble a muscle?. *Psychological Bulletin, 126*(2), 247-259.

Mussell, M. P., Mitchell, J. E., de Zwaan, M., Crosby, R. D., Seim, H. C., & Crow, S. J. (1996). Clinical characteristics associated with binge eating in obese females: A descriptive study. *International Journal of Obesity and Related Metabolic Disorders, 20*(4), 324-331.

Muthukumaraswamy, S. D. (2013). High-frequency brain activity and muscle artifacts in MEG/EEG: A review and recommendations. *Frontiers in Human Neuroscience, 7*, doi: 10.3389/fnhum.2013.00138.

Myers, J. E., & Young, J. S. (2012). Brain wave biofeedback: Benefits of integrating neurofeedback in counseling. *Journal of Counseling & Development, 90*(1), 20-28.

Nederkoorn, C., Smulders, F. T., Havermans, R. C., & Jansen, A. (2004). Exposure to binge food in bulimia nervosa: Finger pulse amplitude as a potential measure of urge to eat and predictor of food intake. *Appetite, 42*(2), 125-130.

Nederkoorn, C., Smulders, F. T., Havermans, R. C., Roefs, A., & Jansen, A. (2006). Impulsivity in obese women. *Appetite, 47*(2), 253-256.

Nederkoorn, C., Smulders, F. T., & Jansen, A. (2000). Cephalic phase responses, craving and food intake in normal subjects. *Appetite, 35*(1), 45-55.

Nichols, A. L., & Maner, J. K. (2008). The good-subject effect: Investigating participant demand characteristics. *The Journal of General Psychology*, *135*(2), 151-166.

Niemeier, H. M., Phelan, S., Fava, J. L., & Wing, R. R. (2007). Internal disinhibition predicts weight regain following weight loss and weight loss maintenance. *Obesity*, *15*(10), 2485-2494.

Nijs, I. M., & Franken, I. H. (2012). Attentional processing of food cues in overweight and obese individuals. *Current Obesity Reports*, *1*(2), 106-113.

Nijs, I. M., Franken, I. H., & Muris, P. (2008). Food cue-elicited brain potentials in obese and healthy-weight individuals. *Eating behaviors*, *9*(4), 462-470.

Ninaus, M., Kober, S. E., Witte, M., Koschutnig, K., Stangl, M., Neuper, C., & Wood, G. (2013). Neural substrates of cognitive control under the belief of getting neurofeedback training. *Frontiers in Human Neuroscience*, *7*:914, doi: 10.3389/fnhum.2013.00914.

Niv, S. (2013). Clinical efficacy and potential mechanisms of neurofeedback. *Personality and Individual Differences*, *54*(6), 676-686.

Nofzinger, E. A., Price, J. C., Meltzer, C. C., Buysse, D. J., Villemagne, V. L., Miewald, J. M., ... & Kupfer, D. J. (2000). Towards a neurobiology of dysfunctional arousal in depression: The relationship between beta EEG power and regional cerebral glucose metabolism during NREM sleep. *Psychiatry Research: Neuroimaging*, *98*(2), 71-91.

Ochner, C. N., Green, D., van Steenburgh, J. J., Kounios, J., & Lowe, M. R. (2009). Asymmetric prefrontal cortex activation in relation to markers of overeating in obese humans. *Appetite*, *53*(1), 44-49.

Ogden, J. (1993). The measurement of restraint: Confounding success and failure?. *International Journal of Eating Disorders*, *13*(1), 69-76.

O'Leary, A. (1985). Self-efficacy and health. *Behaviour Research and Therapy*, *23*(4), 437-451.

Olson, C. M., Bove, C. F., & Miller, E. O. (2007). Growing up poor: Long-term implications for eating patterns and body weight. *Appetite*, *49*(1), 198-207.

Ong, L., Linden, W., & Young, S. (2004). Stress management: What is it?. *Journal of Psychosomatic Research*, *56*(1), 133-137.

O'Reilly, G. A., Cook, L., Spruijt-Metz, D., & Black, D. S. (2014). Mindfulness-based interventions for obesity-related eating behaviours: A literature review. *Obesity Reviews*, *15*(6), 453-461.

Overduin, J., & Jansen, A. (1996). Food cue reactivity in fasting and non-fasting subjects. *European Eating Disorders Review*, *4*(4), 249-259.

Overduin, J., Jansen, A., & Eilkes, H. (1997). Cue reactivity to food-and body-related stimuli in restrained and unrestrained eaters. *Addictive Behaviors*, *22*(3), 395-404.

Papies, E. K., Stroebe, W., & Aarts, H. (2007). Pleasure in the mind: Restrained eating and spontaneous hedonic thoughts about food. *Journal of Experimental Social Psychology*, *43*(5), 810-817.

Papies, E. K., Stroebe, W., & Aarts, H. (2008). Healthy cognition: Processes of self-regulatory success in restrained eating. *Personality and Social Psychology Bulletin*, *34*(9), 1290-1300.

Paquette, V., Beauregard, M., & Beaulieu-Prévost, D. (2009). Effect of a psychoneurotherapy on brain electromagnetic tomography in individuals with major depressive disorder. *Psychiatry Research: Neuroimaging*, *174*(3), 231-239.

Parvaz, M. A., Alia-Klein, N., Woicik, P. A., Volkow, N. D., & Goldstein, R. Z. (2011). Neuroimaging for drug addiction and related behaviors. *Reviews in the Neurosciences*, *22*(6), 609-624.

Pearson, D. G., Deeprose, C., Wallace-Hadrill, S. M., Heyes, S. B., & Holmes, E. A. (2013). Assessing mental imagery in clinical psychology: A review of imagery measures and a guiding framework. *Clinical Psychology Review*, *33*(1), 1-23.

Pelchat, M. L. (1997). Food cravings in young and elderly adults. *Appetite*, *28*(2), 103-113.

Pelchat, M. L. (2002). Of human bondage: Food craving, obsession, compulsion, and addiction. *Physiology & Behavior*, *76*(3), 347-352.

Pelchat, M. L. (2009). Food addiction in humans. *The Journal of Nutrition*, *139*(3), 620-622.

Peniston, E. G., & Kulkosky, P. J. (1990). Alcoholic personality and alpha-theta brainwave training. *Medical Psychotherapy*, *2*, 37-55.

Peters, A. (2011). The selfish brain: Competition for energy resources. *American Journal of Human Biology*, *23*(1), 29-34.

Peters, A., Schweiger, U., Pellerin, L., Hubold, C., Oltmanns, K. M., Conrad, M., ... & Fehm, H. L. (2004). The selfish brain: Competition for energy resources. *Neuroscience & Biobehavioral Reviews*, *28*(2), 143-180.

Piccoli, L., Di Bonaventura, M. V. M., Cifani, C., Costantini, V. J., Massagrande, M., Montanari, D., ... & Merlo-Pich, E. (2012). Role of orexin-1 receptor mechanisms on compulsive food consumption in a model of binge eating in female rats. *Neuropsychopharmacology*, *37*(9), 1999-2011.

Pliner, P. L. (1973). Effects of cue salience on the behavior of obese and normal subjects. *Journal of Abnormal Psychology*, *82*(2), 226-232.

Pogarell, O., Juckel, G., Mavrogiorgou, P., Mulert, C., Folkerts, M., Hauke, W., ... & Hegerl, U. (2006). Symptom-specific EEG power correlations in patients with obsessive–compulsive disorder. *International Journal of Psychophysiology*, *62*(1), 87-92.

Polivy, J., & Herman, C. P. (1985). Dieting and binging: A causal analysis. *American Psychologist*, *40*(2), 193-201.

Polivy, J., Coleman, J., & Herman, C. P. (2005). The effect of deprivation on food cravings and eating behavior in restrained and unrestrained eaters. *International Journal of Eating Disorders*, *38*(4), 301-309.

Pollert, G. A., Engel, S. G., Schreiber-Gregory, D. N., Crosby, R. D., Cao, L., Wonderlich, S. A., ... & Mitchell, J. E. (2013). The role of eating and emotion in binge eating disorder and loss of control eating. *International Journal of Eating Disorders*, *46*(3), 233-238.

Pop-Jordanova, N. (2000). Psychological characteristics and biofeedback mitigation in preadolescents with eating disorders. *Pediatrics International*, *42*(1), 76-81.

Preti, A., de Girolamo, G., Vilagut, G., Alonso, J., de Graaf, R., Bruffaerts, R., ... & ESEMeD-WMH Investigators. (2009). The epidemiology of eating disorders in six European countries: Results of the ESEMeD-WMH project. *Journal of Psychiatric Research*, *43*(14), 1125-1132.

Provencher, V., Bégin, C., Piché, M. È., Bergeron, J., Corneau, L., Weisnagel, S. J., ... & Lemieux, S. (2007). Disinhibition, as assessed by the Three-Factor Eating Questionnaire, is inversely related to psychological well-being in postmenopausal women. *International Journal of Obesity*, *31*(2), 315-320.

Pursey, K. M., Stanwell, P., Callister, R. J., Brain, K., Collins, C. E., & Burrows, T. L. (2014). Neural responses to visual food cues according to weight status: A systematic review of functional magnetic resonance imaging studies. *Frontiers in Nutrition*, *1*, doi: 10.3389/fnut.2014.00007

Radley, J. J., Sisti, H. M., Hao, J., Rocher, A., McCall, T., Hof, P. R., ... & Morrison, J. H. (2004). Chronic behavioral stress induces apical dendritic reorganization in pyramidal neurons of the medial prefrontal cortex. *Neuroscience*, *125*(1), 1-6.

Rangaswamy, M., Porjesz, B., Chorlian, D. B., Wang, K., Jones, K. A., Bauer, L. O., ... & Begleiter, H. (2002). Beta power in the EEG of alcoholics. *Biological Psychiatry*, *52*(8), 831-842.

Rankinen, T., & Bouchard, C. (2006). Genetics of food intake and eating behavior phenotypes in humans. *Annual Review of Nutrition*, *26*(1), 413-434.

Rau, J. H., Struve, F. A., & Green, R. S. (1979). Electroencephalographic correlates of compulsive eating. *Clinical EEG and Neuroscience*, *10*(4), 180-189.

Reas, D. L., & Grilo, C. M. (2008). Review and meta-analysis of pharmacotherapy for binge-eating disorder. *Obesity*, *16*(9), 2024-2038.

Reid, M. S., Prichep, L. S., Ciplet, D., O'Leary, S., Tom, M., Howard, B., ... & John, E. R. (2003). Quantitative electroencephalographic studies of cue-induced cocaine craving. *Clinical EEG and Neuroscience*, *34*(3), 110-123.

Rief, W., & Birbaumer, N. (2006). Grundsätzliches zum Biofeedback. In: W. Rief & N. Birbaumer (Eds.). *Biofeedback: Grundlagen, Indikationen, Kommunikation, praktisches Vorgehen in der Therapie* (2nd ed., pp. 1-7). Stuttgart, Germany: Schattauer.

Rodin, J. (1981). Current status of the internal–external hypothesis for obesity: What went wrong?. *American Psychologist*, *36*(4), 361-372.

Rodin, J., & Slochower, J. (1976). Externality in the nonobese: Effects of environmental responsiveness on weight. *Journal of Personality and Social Psychology*, *33*(3), 338-344.

Rodríguez, S., Fernández, M. C., Cepeda-Benito, A., & Vila, J. (2005). Subjective and physiological reactivity to chocolate images in high and low chocolate cravers. *Biological Psychology*, *70*(1), 9-18.

Rohsenow, D. J., Childress, A. R., Monti, P. M., Niaura, R. S., & Abrams, D. B. (1991). Cue reactivity in addictive behaviors: Theoretical and treatment implications. *International Journal of the Addictions*, *25*(sup7), 957-993.

Rokicki, L. A., Holroyd, K. A., France, C. R., Lipchik, G. L., France, J. L., & Kvaal, S. A. (1997). Change mechanisms associated with combined relaxation/EMG biofeedback training for chronic tension headache. *Applied Psychophysiology and Biofeedback*, *22*(1), 21-41.

Ros, T., J Baars, B., Lanius, R. A., & Vuilleumier, P. (2014). Tuning pathological brain oscillations with neurofeedback: A systems neuroscience framework. *Frontiers in Human Neuroscience*, *8:*1008, doi: 10.3389/fnhum.2014.01008.

Ros, T., Moseley, M. J., Bloom, P. A., Benjamin, L., Parkinson, L. A., & Gruzelier, J. H. (2009). Optimizing microsurgical skills with EEG neurofeedback. *BMC Neuroscience*, *10*:87, doi: 10.1186/1471-2202-10-87

Rosval, L., Steiger, H., Bruce, K., Israël, M., Richardson, J., & Aubut, M. (2006). Impulsivity in women with eating disorders: Problem of response inhibition, planning, or attention?. *International Journal of Eating Disorders*, *39*(7), 590-593.

Royal, J. D., & Kurtz, J. L. (2010). I ate what?! The effect of stress and dispositional eating style on food intake and behavioral awareness. *Personality and Individual Differences*, *49*(6), 565-569.

Ruderman, A. J. (1986). Dietary restraint: A theoretical and empirical review. *Psychological Bulletin*, *99*(2), 247-262.

Russell, J. A. (2003). Core affect and the psychological construction of emotion. *Psychological Review*, *110*(1), 145-172.

Saletu-Zyhlarz, G. M., Arnold, O., Anderer, P., Oberndorfer, S., Walter, H., Lesch, O. M., ... & Saletu, B. (2004). Differences in brain function between relapsing and abstaining alcohol-dependent patients, evaluated by EEG mapping. *Alcohol and Alcoholism*, *39*(3), 233-240.

Schachter, S. (1968). Obesity and eating. *Science*, *161(3843)*, 751-756.

Schag, K., Schönleber, J., Teufel, M., Zipfel, S., & Giel, K. E. (2013). Food-related impulsivity in obesity and binge eating disorder–a systematic review. *Obesity Reviews*, *14*(6), 477-495.

Schienle, A., Schäfer, A., Hermann, A., & Vaitl, D. (2009). Binge-eating disorder: Reward sensitivity and brain activation to images of food. *Biological Psychiatry*, *65*(8), 654-661.

Schlüter, N., Schmidt, R., Kittel, R., Tetzlaff, A., & Hilbert, A. (2015). Loss of control eating in adolescents from the community. *International Journal of Eating Disorders*, *49*(4), 413-420.

Schmidt, U., & Campbell, I. C. (2013). Treatment of eating disorders can not remain 'brainless': The case for brain-directed treatments. *European Eating Disorders Review*, *21*(6), 425-427.

Schmidt, U., & Marks, I. (1988). Cue exposure to food plus response prevention of binges for bulimia: A pilot study. *International Journal of Eating Disorders, 7*(5), 663-672.

Schreiber, L., Odlaug, B. L., & Grant, J. E. (2011). Impulse control disorders: Updated review of clinical characteristics and pharmacological management. *Frontiers in Psychiatry*, *2*(1), doi: 10.3389/fpsyt.2011.00001.

Schwartz, N. M., & Schwartz, M. S. (2003). Definitions of biofeedback and applied psychophysiology. In M. S. Schwartz & F. Andrasik (Eds.), *Biofeedback – A practitioner's guide* (3rd ed., pp. 27-39). New York, NY: The Guilford Press.

Scott, W. C., Kaiser, D., Othmer, S., & Sideroff, S. I. (2005). Effects of an EEG biofeedback protocol on a mixed substance abusing population. *The American Journal of Drug and Alcohol Abuse, 31*(3), 455-469.

Scott, W. C., & Kaiser, D. (1998). Augmenting chemical dependency treatment with neurofeedback training. *Journal of Neurotherapy, 3*(1), 66.

Selby, E. A., Anestis, M. D., & Joiner, T. E. (2008). Understanding the relationship between emotional and behavioral dysregulation: Emotional cascades. *Behaviour Research and Therapy*, *46*(5), 593-611.

Seo, M. G., Feldman-Barrett, L., & Bartunek, J. M. (2004). The role of affective experience in work motivation. *Academy of Management Review, 29*(3), 423-439.

Seo, S.-H., & Lee, J.-T. (2010). Stress and EEG, convergence and hybrid information technologies. In M. Crisan (Ed.), *Convergence and hybrid information technologies* (pp. 413-426). Retrieved from http://www.intechopen.com/books/convergence-and-hybrid-information-technologies/stress-and-eeg

Shackman, A. J., Salomons, T. V., Slagter, H. A., Fox, A. S., Winter, J. J., & Davidson, R. J. (2011). The integration of negative affect, pain and cognitive control in the cingulate cortex. *Nature Reviews Neuroscience*, *12*(3), 154-167.

Sherlin, L. H., Arns, M., Lubar, J., Heinrich, H., Kerson, C., Strehl, U., & Sterman, M. B. (2011). Neurofeedback and basic learning theory: Implications for research and practice. *Journal of Neurotherapy*, *15*(4), 292-304.

Sherlin, L. H., & Congedo, M. (2005). Obsessive-compulsive dimension localized using low-resolution brain electromagnetic tomography (LORETA). *Neuroscience Letters, 387*(2), 72-74.

Sherry, S. B., & Hall, P. A. (2009). The perfectionism model of binge eating: Tests of an integrative model. *Journal of Personality and Social Psychology, 96*(3), 690-709.

Shunk, J. A., & Birch, L. L. (2004). Girls at risk for overweight at age 5 are at risk for dietary restraint, disinhibited overeating, weight concerns, and greater weight gain from 5 to 9 years. *Journal of the American Dietetic Association, 104*(7), 1120-1126.

Siahpush, M., Tibbits, M., Shaikh, R. A., Singh, G. K., Kessler, A. S., & Huang, T. T. K. (2015). Dieting increases the likelihood of subsequent obesity and BMI gain: Results from a prospective study of an Australian national sample. *International Journal of Behavioral Medicine, 22*(5), 662-671.

Silva, J. R., Pizzagalli, D. A., Larson, C. L., Jackson, D. C., & Davidson, R. J. (2002). Frontal brain asymmetry in restrained eaters. *Journal of Abnormal Psychology*, *111*(4), 676-681.

Sinha, R., & Jastreboff, A. M. (2013). Stress as a common risk factor for obesity and addiction. *Biological Psychiatry, 73*(9), 827-835.

Siniatchkin, M., Kropp, P., & Gerber, W. D. (2000). Neurofeedback–the significance of reinforcement and the search for an appropriate strategy for the success of self-regulation. *Applied Psychophysiology and Biofeedback, 25*(3), 167-175.

Slochower, J. A. (1983). *Excessive Eating.* New York, NY: Human Sciences Press, Inc.

Smink, F. R., Van Hoeken, D., & Hoek, H. W. (2012). Epidemiology of eating disorders: Incidence, prevalence and mortality rates. *Current Psychiatry Reports*, *14*(4), 406-414.

Sokhadze, E. M. (2007). Effects of music on the recovery of autonomic and electrocortical activity after stress induced by aversive visual stimuli. *Applied Psychophysiology and Biofeedback*, *32*(1), 31-50.

Sokhadze, T. M., Cannon, R. L., & Trudeau, D. L. (2008). EEG biofeedback as a treatment for substance use disorders: Review, rating of efficacy and recommendations for further research. *Journal of Neurotherapy*, *12*(1), 5-43.

Spaeth, A. M., Dinges, D. F., & Goel, N. (2013). Effects of experimental sleep restriction on weight gain, caloric intake, and meal timing in healthy adults. *Sleep*, *36*(7), 981-990.

Steel, D., Kemps, E., & Tiggemann, M. (2006). Effects of hunger and visuo-spatial interference on imagery-induced food cravings. *Appetite*, *46*(1), 36-40.

Stein, R. I., Kenardy, J., Wiseman, C. V., Dounchis, J. Z., Arnow, B. A., & Wilfley, D. E. (2007). What's driving the binge in binge eating disorder?: A prospective examination of precursors and consequences. *International Journal of Eating Disorders*, *40*(3), 195-203.

Stice, E. (1994). Review of the evidence for a sociocultural model of bulimia nervosa and an exploration of the mechanisms of action. *Clinical Psychology Review*, *14*(7), 633-661.

Stice, E. (2001). A prospective test of the dual-pathway model of bulimic pathology: Mediating effects of dieting and negative affect. *Journal of Abnormal Psychology*, *110*(1), 124-135.

Stice, E. (2002). Risk and maintenance factors for eating pathology: A meta-analytic review. *Psychological Bulletin*, *128*(5), 825-848.

Stice, E., & Shaw, H. E. (1994). Adverse effects of the media portrayed thin-ideal on women and linkages to bulimic symptomatology. *Journal of Social and Clinical psychology*, *13*(3), 288-308.

Stickney, M. I., Miltenberger, R. G., & Wolff, G. (1999). A descriptive analysis of factors contributing to binge eating. *Journal of Behavior Therapy and Experimental Psychiatry*, *30*(3), 177-189.

St-Onge, M. P., McReynolds, A., Trivedi, Z. B., Roberts, A. L., Sy, M., & Hirsch, J. (2012). Sleep restriction leads to increased activation of brain regions sensitive to food stimuli. *The American Journal of Clinical Nutrition*, *95*(4), 818-824.

Strader, A. D., & Woods, S. C. (2005). Gastrointestinal hormones and food intake. *Gastroenterology*, *128*(1), 175-191.

Strecher, V. J., DeVellis, B. M., Becker, M. H., & Rosenstock, I. M. (1986). The role of self-efficacy in achieving health behavior change. *Health Education & Behavior*, *13*(1), 73-92.

Strehl, U. (2014). What learning theories can teach us in designing neurofeedback treatments. *Frontiers in Human Neuroscience 8:*894, doi: 10.3389/fnhum.2014.00894.

Striegel-Moore, R. H., & Franko, D. L. (2003). Epidemiology of binge eating disorder. *International Journal of Eating Disorders*, *34*(S1), S19-S29.

Striegel-Moore, R. H., Rosselli, F., Perrin, N., DeBar, L., Wilson, G. T., May, A., & Kraemer, H. C. (2009). Gender difference in the prevalence of eating disorder symptoms. *The International Journal of Eating Disorders*, *42*(5), 471-474.

Stroebe, W., Mensink, W., Aarts, H., Schut, H., & Kruglanski, A. W. (2008). Why dieters fail: Testing the goal conflict model of eating. *Journal of Experimental Social Psychology*, *44*(1), 26-36.

Stroebe, W., Papies, E. K., & Aarts, H. (2008). From homeostatic to hedonic theories of eating: Self-regulatory failure in food-rich environments. *Applied Psychology*, *57*(S1), 172-193.

Stunkard, A. J., & Messick, S. (1985). The Three-Factor Eating Questionnaire to measure dietary restraint, disinhibition and hunger. *Journal of Psychosomatic Research*, *29*(1), 71-83.

Styn, M. A., Bovbjerg, D. H., Lipsky, S., & Erblich, J. (2013). Cue-induced cigarette and food craving: A common effect?. *Addictive Behaviors*, *38*(3), 1840-1843.

Svaldi, J., Tuschen-Caffier, B., Peyk, P., & Blechert, J. (2010). Information processing of food pictures in binge eating disorder. *Appetite*, *55*(3), 685-694.

Swinburn, B. A., Sacks, G., Hall, K. D., McPherson, K., Finegood, D. T., Moodie, M. L., & Gortmaker, S. L. (2011). The global obesity pandemic: Shaped by global drivers and local environments. *The Lancet*, *378*(9793), 804-814.

Swinburn, B. A., Sacks, G., Lo, S. K., Westerterp, K. R., Rush, E. C., Rosenbaum, M., ... & Ravussin, E. (2009). Estimating the changes in energy flux that characterize the rise in obesity prevalence. *The American Journal of Clinical Nutrition*, *89*(6), 1723-1728.

Swinburn, B., Egger, G., & Raza, F. (1999). Dissecting obesogenic environments: The development and application of a framework for identifying and prioritizing environmental interventions for obesity. *Preventive Medicine*, *29*(6), 563-570.

Swinburn, B., Sacks, G., & Ravussin, E. (2009). Increased food energy supply is more than sufficient to explain the US epidemic of obesity. *The American Journal of Clinical Nutrition*, *90*(6), 1453-1456.

Tammela, L. I., Pääkkönen, A., Karhunen, L. J., Karhu, J., Uusitupa, M. I., & Kuikka, J. T. (2010). Brain electrical activity during food presentation in obese binge-eating women. *Clinical Physiology and Functional Imaging*, *30*(2), 135-140.

Tan, G., Thornby, J., Hammond, D. C., Strehl, U., Canady, B., Arnemann, K., & Kaiser, D. A. (2009). Meta-analysis of EEG biofeedback in treating epilepsy. *Clinical EEG and Neuroscience*, *40*(3), 173-179.

Taren, D. L., Tobar, M., Hill, A., Howell, W., Shisslak, C., Bell, I., & Ritenbaugh, C. (1999). The association of energy intake bias with psychological scores of women. *European Journal of Clinical Nutrition*, *53*(7), 570-578.

Tatham, M. (2011). The role of imagery-based techniques in cognitive–behavioural therapy for adults with eating disorders. *Clinical Psychology Review*, *31*(7), 1101-1109.

Telch, C. F. (1997). Skills training treatment for adaptive affect regulation in a woman with binge-eating disorder. *International Journal of Eating Disorders*, *22*(1), 77-81.

Tetley, A., Brunstrom, J., & Griffiths, P. (2009). Individual differences in food-cue reactivity. The role of BMI and everyday portion-size selections. *Appetite*, *52*(3), 614-620.

Teufel, M., Stephan, K., Kowalski, A., Käsberger, S., Enck, P., Zipfel, S., & Giel, K. E. (2013). Impact of biofeedback on self-efficacy and stress reduction in obesity: A randomized controlled pilot study. *Applied Psychophysiology and Biofeedback*, *38*(3), 177-184.

Thibault, R. T., Lifshitz, M., Birbaumer, N., & Raz, A. (2015). Neurofeedback, self-regulation, and brain imaging: Clinical science and fad in the service of mental disorders. *Psychotherapy and Psychosomatics*, *84*(4), 193-207.

Thibault, R. T., Lifshitz, M., & Raz, A. (2016). The self-regulating brain and neurofeedback: Experimental science and clinical promise. *Cortex*, *74*, 247-261.

Thompson, M., & Thompson, L. (2007). Neurofeedback for stress management. In P. M. Lehrer, R. L. Woolfolk, & W. E. Sime (Eds.), *Principles and practice of stress management* (3rd ed., pp. 249-287). New York, NY: Guilford.

Thomsen, D. K. (2006). The association between rumination and negative affect: A review. *Cognition and Emotion*, *20*(8), 1216-1235.

Thomsen, D. K., Tønnesvang, J., Schnieber, A., & Olesen, M. H. (2011). Do people ruminate because they haven't digested their goals? The relations of rumination and reflection to goal internalization and ambivalence. *Motivation and Emotion*, *35*(2), 105-117.

Tice, D. M., Bratslavsky, E., & Baumeister, R. F. (2001). Emotional distress regulation takes precedence over impulse control: If you feel bad, do it!. *Journal of Personality and Social Psychology*, *80*(1), 53-67.

Tiffany, S. T. (1990). A cognitive model of drug urges and drug-use behavior: Role of automatic and nonautomatic processes. *Psychological Review*, *97*(2), 147-168.

Tiggemann, M., & Kemps, E. (2005). The phenomenology of food cravings: The role of mental imagery. *Appetite, 45*(3), 305-313.

Toro, J., Cervera, M., Feliu, M. H., Garriga, N., Jou, M., Martinez, E., & Toro, E. (2003). Cue exposure in the treatment of resistant bulimia nervosa. *International Journal of Eating Disorders, 34*(2), 227-234.

Tuschen-Caffier, B., & Vögele, C. (1999). Psychological and physiological reactivity to stress: An experimental study on bulimic patients, restrained eaters and controls. *Psychotherapy and Psychosomatics, 68*(6), 333-340.

Udo, T., Weinberger, A. H., Grilo, C. M., Brownell, K. D., DiLeone, R. J., Lampert, R., ... & McKee, S. A. (2014). Heightened vagal activity during high-calorie food presentation in obese compared with non-obese individuals—results of a pilot study. *Obesity Research & Clinical Practice, 8*(3), e258-e265.

Vainik, U., Neseliler, S., Konstabel, K., Fellows, L. K., & Dagher, A. (2015). Eating traits questionnaires as a continuum of a single concept. Uncontrolled eating. *Appetite, 90*, 229-239.

Valck, E. D., Cluydts, R., & Pirrera, S. (2004). Effect of cognitive arousal on sleep latency, somatic and cortical arousal following partial sleep deprivation. *Journal of sleep research*, *13*(4), 295-304.

Val-Laillet, D., Aarts, E., Weber, B., Ferrari, M., Quaresima, V., Stoeckel, L. E., ... & Stice, E. (2015). Neuroimaging and neuromodulation approaches to study eating behavior and prevent and treat eating disorders and obesity. *NeuroImage: Clinical, 8*(1), 1-31.

Van Gucht, D., Vansteenwegen, D., Beckers, T., Hermans, D., Baeyens, F., & Van den Bergh, O. (2008). Repeated cue exposure effects on subjective and physiological indices of chocolate craving. *Appetite, 50*(1), 19-24.

Van Son, G. E., Van Hoeken, D., Bartelds, A. I., Van Furth, E. F., & Hoek, H. W. (2006). Urbanisation and the incidence of eating disorders. *The British Journal of Psychiatry*, *189*(6), 562-563.

Van Strien, T. (1999). Success and failure in the measurement of restraint: Notes and data. *International Journal of Eating Disorders*, *25*(4), 441-449.

Van Strien, T., Frijters, J. E., Bergers, G., & Defares, P. B. (1986). The Dutch Eating Behavior Questionnaire (DEBQ) for assessment of restrained, emotional, and external eating behavior. *International Journal of Eating Disorders*, *5*(2), 295-315.

Van Strien, T., Herman, C. P., & Verheijden, M. W. (2012). Eating style, overeating and weight gain. A prospective 2-year follow-up study in a representative Dutch sample. *Appetite*, *59*(3), 782-789.

Vanderlinden, J., Dalle Grave, R., Fernandez, F., Vandereycken, W., Pieters, G., & Noorduin, C. (2004). Which factors do provoke binge eating? An exploratory study in eating disorder patients. *Eating and Weight Disorders-Studies on Anorexia, Bulimia and Obesity, 9*(4), 300-305.

Vanderlinden, J., Dalle Grave, R., Vandereycken, W., & Noorduin, C. (2001). Which factors do provoke binge-eating? An exploratory study in female students. *Eating Behaviors*, *2*(1), 79-83.

Vannucci, A., Tanofsky-Kraff, M., Crosby, R. D., Ranzenhofer, L. M., Shomaker, L. B., Field, S. E., ... & Yanovski, J. A. (2013). Latent profile analysis to determine the typology of disinhibited eating behaviors in children and adolescents. *Journal of Consulting and Clinical Psychology*, *81*(3), 494-507.

Verheul, R., van den Brink, W., & Geerlings, P. (1999). A three-pathway psychobiological model of craving for alcohol. *Alcohol and Alcoholism*, *34*(2), 197-222.

Vernon, D. J. (2005). Can neurofeedback training enhance performance? An evaluation of the evidence with implications for future research. *Applied Psychophysiology and Biofeedback*, *30*(4), 347-364.

Vernon, D. J., Egner, T., Cooper, N., Compton, T., Neilands, C., Sheri, A., & Gruzelier, J. (2003). The effect of training distinct neurofeedback protocols on aspects of cognitive performance. *International Journal of Psychophysiology, 47*(1), 75-85.

Vidaurre, C., & Blankertz, B. (2010). Towards a cure for BCI illiteracy. *Brain Topography, 23*(2), 194-198.

Vocks, S., Tuschen-Caffier, B., Pietrowsky, R., Rustenbach, S. J., Kersting, A., & Herpertz, S. (2010). Meta-analysis of the effectiveness of psychological and pharmacological treatments for binge eating disorder. *International Journal of Eating Disorders, 43*(3), 205-217.

Vögele, C., & Florin, I. (1997). Psychophysiological responses to food exposure: An experimental study in binge eaters. *International Journal of Eating Disorders, 21*(2), 147-157.

Volkow, N. D., Fowler, J. S., & Wang, G. J. (2004). The addicted human brain viewed in the light of imaging studies: Brain circuits and treatment strategies. *Neuropharmacology, 47*(S1), 3-13.

Volkow, N. D., Wang, G. J., & Baler, R. D. (2011). Reward, dopamine and the control of food intake: implications for obesity. *Trends in Cognitive Sciences, 15*(1), 37-46.

Volkow, N. D., Wang, G. J., Fowler, J. S., Tomasi, D., & Baler, R. (2012). Food and drug reward: Overlapping circuits in human obesity and addiction. *Current Topics in Behavioral Neuroscience, 11*, 1-14.

Walker, J. E. (2011). QEEG-guided neurofeedback for recurrent migraine headaches. *Clinical EEG and Neuroscience, 42*(1), 59-61.

Wallis, D. J., & Hetherington, M. M. (2004). Stress and eating: The effects of ego-threat and cognitive demand on food intake in restrained and emotional eaters. *Appetite, 43*(1), 39-46.

Wallis, D. J., & Hetherington, M. M. (2009). Emotions and eating. Self-reported and experimentally induced changes in food intake under stress. *Appetite, 52*(2), 355-362.

Ward, A., & Mann, T. (2000). Don't mind if I do: Disinhibited eating under cognitive load. *Journal of Personality and Social Psychology, 78*(4), 753.

Wardle, J. (1990). Conditioning processes and cue exposure in the modification of excessive eating. *Addictive Behaviors, 15*(4), 387-393.

Wardle, J., Steptoe, A., Oliver, G., & Lipsey, Z. (2000). Stress, dietary restraint and food intake. *Journal of Psychosomatic Research, 48*(2), 195-202.

Warren, C. S. (2008). Disinhibited eating. In K. Keller (Ed.), *Encyclopedia of Obesity* (pp. 190-191). Los Angeles, CA: Sage.

Waschburger, P., & Kröller, K. (2005). Adipositas im Kindes- und Jugendalter: Was sind Risikofaktoren für die Entstehung einer Binge Eating Disorder. *Zeitschrift für Gesundheitspsychologie, 13*(2), 69-78.

Waters, A., Hill, A., & Waller, G. (2001). Bulimics' responses to food cravings: Is binge-eating a product of hunger or emotional state?. *Behaviour Research and Therapy, 39*(8), 877-886.

Westenhöfer, J. (1992). *Gezügeltes Essen und Störbarkeit des Eßverhaltens.* Göttingen, Germany: Hogrefe.

Westerterp, K. R. (2010). Physical activity, food intake, and body weight regulation: Insights from doubly labeled water studies. *Nutrition Reviews, 68*(3), 148-154.

White, M. A., Kalarchian, M. A., Masheb, R. M., Marcus, M. D., & Grilo, C. M. (2009). Loss of control over eating predicts outcomes in bariatric surgery patients: a prospective, 24-month follow-up study. *The Journal of Clinical Psychiatry, 71*(2), 1-478.

Wickramasekera, I. (1999). How does biofeedback reduce clinical symptoms and do memories and beliefs have biological consequences? Toward a model of mind-body healing. *Applied Psychophysiology and Biofeedback, 24*(2), 91-105.

Wilfley, D. E., Friedman, M. A., Dounchis, J. Z., Stein, R. I., Welch, R. R., & Ball, S. A. (2000). Comorbid psychopathology in binge eating disorder: Relation to eating disorder severity at baseline and following treatment. *Journal of Consulting and Clinical Psychology, 68*(4), 641-649.

Wilson, G. T., Grilo, C. M., & Vitousek, K. M. (2007). Psychological treatment of eating disorders. *American Psychologist, 62*(3), 199-216.

Wilson, G. T., Vitousek, K. M., & Loeb, K. L. (2000). Stepped care treatment for eating disorders. *Journal of Consulting and Clinical Psychology*, *68*(4), 564-572.

Wing, R. R., Tate, D. F., Gorin, A. A., Raynor, H. A., & Fava, J. L. (2006). A self-regulation program for maintenance of weight loss. *New England Journal of Medicine*, *355*(15), 1563-1571.

Wolfe, B. E., Baker, C. W., Smith, A. T., & Kelly-Weeder, S. (2009). Validity and utility of the current definition of binge eating. *International Journal of Eating Disorders*, *42*(8), 674-686.

Wolff, G. E., & Clark, M. M. (2001). Changes in eating self-efficacy and body image following cognitive–behavioral group therapy for binge eating disorder: A clinical study. *Eating Behaviors*, *2*(2), 97-104.

Wolz, I., Fagundo, A. B., Treasure, J., & Fernández-Aranda, F. (2015). The processing of food stimuli in abnormal eating: A systematic review of electrophysiology. *European Eating Disorders Review*, *23*(4), 251-261.

Woods, S. C., Seeley, R. J., Porte, D., & Schwartz, M. W. (1998). Signals that regulate food intake and energy homeostasis. *Science*, *280*(5368), 1378-1383.

Yokum, S., & Stice, E. (2013). Cognitive regulation of food craving: Effects of three cognitive reappraisal strategies on neural response to palatable foods. *International Journal of Obesity*, *37*(12), 1565-1570.

Zocca, J. M., Shomaker, L. B., Tanofsky-Kraff, M., Columbo, K. M., Raciti, G. R., Brady, S. M., ... & Yanovski, J. A. (2011). Links between mothers' and children's disinhibited eating and children's adiposity. *Appetite*, *56*(2), 324-331.

Zotev, V., Phillips, R., Yuan, H., Misaki, M., & Bodurka, J. (2014). Self-regulation of human brain activity using simultaneous real-time fMRI and EEG neurofeedback. *NeuroImage*, *85*(3), 985-995.

9.1 List of figures

9.2 List of tables

10. Appendix

A.1 Vote of the institutional ethics committee – Study 1

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Frau
Prof. Dr. Alexandra Martin
FB G
Klinische Psychologie und Psychotherapie

Gebäude Z.02.09

Votum der Ethik-Kommission

Sehr geehrte Frau Prof. Martin,

hiermit teile ich Ihnen in meiner Eigenschaft als zuständiger Prorektor mit, dass die Ethik-Kommission der Bergischen Universität Wuppertal ausführlich über den von Ihnen gestellten Antrag zur Durchführung des Forschungsvorhabens „Neurofeedbacktraining zur Reduktion von Heißhungeranfällen bei gezügeltem Essverhalten" beraten und positiv, d. h. für die Unbedenklichkeit der projektierten Studie votiert hat.

Mit freundlichen Grüßen

Prof. Dr. Michael Scheffel

A.2 Vote of the institutional ethics committee – Studies 2 & 3

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DATUM 12. Mai 2014

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Frau
Prof. Dr. Alexandra Martin
Fachbereich G/
Klinische Psychologie und Psychotherape

Z.02.09

Im Hause

Votum der Ethik-Kommission

Sehr geehrter Frau Prof. Dr. Matin,

hiermit teile ich Ihnen in meiner Eigenschaft als zuständiger Prorektor mit, dass die Ethik-Kommission der Bergischen Universität Wuppertal ausführlich über den von Ihnen gestellten Antrag zur Durchführung des Forschungsvorhabens „Neurofeedback zur Reduktion von Heißhungeranfällen bei gezügeltem Essverhalten im Vergleich zu einem Imagery-basierten psychologischen Training", beraten und positiv, d. h. für die Unbedenklichkeit der projektierten Studie votiert hat.

Mit freundlichen Grüßen

Prof. Dr. Michael Scheffel

B.1 Participant information – Study 1

Allgemeine Aufklärung zur Studie
„Neurofeedback als Interventionsmethode bei Heißhungeranfällen“

Liebe Teilnehmerin,

wir freuen uns über Ihre Bereitschaft, als Probandin in unserer wissenschaftlichen Studie **„Neurofeedback als Interventionsmethode bei Heißhungeranfällen“** teilzunehmen. In dieser Aufklärung möchten wir Ihnen vorab genauer mitteilen, was Sie im Rahmen der Studie erwarten wird. Bei der Studie handelt es sich um eine psychologische Interventionsstudie, deren Ergebnisse in zwei Abschlussarbeiten des Psychologie-Studiums wissenschaftlich ausgewertet werden. Wir bedanken uns daher sehr herzlich, dass Sie unsere Forschung durch Ihr Interesse und Ihre Teilnahme unterstützen.

Sie haben sich entschlossen, die **Trainingsmethode des Neurofeedbacks** auszuprobieren. Das Neurofeedback ist eine Methode, die Ihre Gehirnströme absolut ungefährlich und schmerzfrei mithilfe von Elektroden an ihrer Kopfhaut erfasst. Für das Neurofeedback wird Ihre Kopfhaut in jeder Sitzung mit einem leichten Peeling vorbehandelt. Die Elektroden werden mit einem in Salzwasser befeuchteten Stoffbezug bezogen. Alle verwendeten Vorbereitungs-Produkte sind dermatologisch getestet und alle verwendeten Geräte medizinisch zertifiziert. Die Signale werden direkt im Computer verrechnet und in einzelne Bestandteile zerlegt. Der Anteil bestimmter Komponenten (Frequenzbänder) ihrer Hirnströme wird Ihnen sodann direkt in Form einer Animation zurückmeldet.

Diese Animation lernen Sie in insgesamt zehn Trainingssitzungen so zu kontrollieren, dass sie eigene Strategien zur bewussten Kontrolle Ihrer Gehirnströme entwickeln. Heißhungeranfälle gehen mit einem Muster eines besonders gestressten Zustands im Gehirn einher. Sie sollen in unserer Studie lernen, dieses Muster so zu beeinflussen, dass Sie wieder einen Zustand der Entspannung erreichen, wenn sie mit Lebensmitteln in Kontakt kommen, die bei Ihnen Heißhunger auslösen. Diese Strategie können Sie dauerhaft in Ihren Alltag integrieren.

Wir würden Sie im Rahmen der Studie bitten, ihre Ernährung (abgesehen natürlich von den Effekten auf Ihre Heißhungeranfälle) nicht gezielt auf eine Diät zur Gewichtsreduktion umzustellen (z.B. Shake-Diäten, FDH, Dinner Cancelling, Atkins-Diät o.Ä.). Sollten sie bisher bereits dauerhaft eine bestimmte Ernährungsform einhalten (wie z.B. nach Weight Watchers, Low Fat 30 oder vegatarische/vegane Ernährung), behalten Sie diese einfach nach wie vor bei.

Der Trainingszeitraum der ersten Gruppe liegt voraussichtlich im **Mai-Juli 2013**, der Trainingszeitraum der zweiten Gruppe ca. im **Juli-September 2013**.

Damit wir ihre Trainingssitzungen individuell auf Sie zuschneiden können, brauchen wir vorab von Ihnen eine möglichst genaue **Auflistung der Lebensmittel**, die bei Ihnen Heißhunger auslösen. Diese Auflistung sollten Sie uns bitte schnellstmöglich zur Verfügung stellen (schriftlich oder per E-Mail). Die aufgezählten Lebensmittel werden Ihnen in jeder Trainingssitzung als Bilder präsentiert. Sollte es sich bei Ihren persönlichen Heißhunger-Lebensmitteln um sehr ungewöhnliche oder spezielle Produkte handeln, lassen Sie uns bitte wenn möglich ein Foto davon zukommen. In der ersten Sitzung werden wir zur Sicherheit zudem einen Test auf mögliche unerkannte Essstörungen bei Ihnen durchführen. Dies ist im Rahmen von Vorschriften zur Durchführung psychologischer Interventionen notwendig.

Um den Fortschritt des Trainings bei Ihnen festzustellen, werden wir zu verschiedenen Zeitpunkten in der ersten Sitzung, in der fünften Sitzung, in der zehnten Sitzung, sowie drei Monate nach der zehnten Sitzung, sofern Sie damit einverstanden sind, folgende Daten von Ihnen erheben: Gewicht, Angaben zur Häufigkeit Ihrer Heißhungeranfälle und Ihrem Essverhalten sowie Angaben zu ihrem Stressempfinden und ihrer Lebenszufriedenheit. Um wissenschaftlich haltbare Ergebnisse zu erzielen, sind uns ehrliche Auskünfte ihrerseits, sowie die **zuverlässige Teilnahme** an Terminen zum Training und zur Datenerfassung wichtig. Sollten Sie einen Termin nicht wahrnehmen können, würden wir gern zu einem schnellstmöglichen Zeitpunkt einen Ersatztermin mit Ihnen vereinbaren. Sollten Sie zu einem bereits abgesprochenen Termin verhindert sein (Krankheit, Notfälle o.Ä.), geben Sie uns bitte

umgehend, bestenfalls bis 24 Stunden vor Ihrem Termin, Bescheid. Unsere Kontaktdaten finden Sie am Ende dieses Informationsschreibens.

Sie erhalten im Rahmen der Studie zehn kostenlose Neurofeedback-Sitzungen. Die Studie wird nicht anderweitig vergütet.

Datenschutz:
Alle erhobenen Daten werden anonymisiert und streng vertraulich nach Vorgaben der Datenschutzrichtlinien behandelt. Von Ihnen bereitgestellte Kontaktdaten und personenbezogene Informationen werden lediglich für die Dauer der Studiendurchführung zur Absprache von Terminen erhoben und gespeichert und im Anschluss, oder nach Aufforderung ihrerseits gelöscht. Die Erhebung der Daten in Form von Fragebögen und in den Neurofeedbacksitzungen erfolgt pseudonymisiert, d. h. in namentlich nicht gekennzeichneter Form. Ihre Antworten und Ergebnisse werden unter einer Nummer gespeichert. Es existiert eine Kodierliste auf Papier, die Ihren Namen mit der Nummer verbindet, was für die Auswertung der Daten erforderlich sein könnte. Die Kodierliste ist nur den Projektmitarbeitern zugänglich; sie wird in einem abschließbaren Schrank aufbewahrt und nach Abschluss der Datenerhebung vernichtet. Nach Vernichtung der Kodierliste liegen die Daten nur noch als Nummer in vollständig anonymisierter Form vor; ein Rückschluss auf den einzelnen Probanden ist dann nicht mehr möglich. Die anonymisierten Daten werden mindestens 10 Jahre gespeichert. Sie können, wann immer Sie möchten, die Löschung aller von Ihnen erhobenen Daten verlangen. Wenn die Kodierliste aber erst einmal gelöscht ist, können wir Ihren Datensatz nicht mehr identifizieren. Deshalb können wir Ihrem Verlangen nach Löschung Ihrer Daten nur solange nachkommen, wie die Kodierliste existiert. Die im Rahmen dieser Studie erhobenen Daten und persönlichen Mitteilungen werden vertraulich behandelt. Mitarbeiter, die durch direkten Kontakt mit Ihnen über personenbezogene Daten verfügen, sind verpflichtet, diese nicht an Dritte weiterzugeben. Desweiteren wird die Veröffentlichung der Ergebnisse der Studie in anonymisierter Form erfolgen, d. h. ohne dass Ihre Daten Ihrer Person zugeordnet werden können.

Die Teilnahme an der Studie ist freiwillig. Sie können jederzeit und ohne Angabe von Gründen Ihre Einwilligung zur Teilnahme an dieser Studie widerrufen, ohne dass Ihnen daraus Nachteile entstehen. **Falls Sie zu irgendeinem Zeitpunkt - aus welchen Gründen auch immer - eine Sitzung oder die gesamte Behandlung abbrechen möchten, steht Ihnen dies völlig frei.** Teilen Sie uns dies in dem Fall bitte mit. Sie werden zur Studienteilnahme eine Einverständniserklärung ausfüllen, in der Sie uns bestätigen, dass Sie über diese Möglichkeit aufgeklärt wurden und freiwillig an der Studie teilnehmen.

Es handelt sich bei dieser Studie um eine erste Untersuchung dieser Methodik bei Heißhungeranfällen. Nach gründlicher Recherche können wir sagen, dass das Auftreten negativer Effekte sehr unwahrscheinlich ist. Sollten sich bei Ihnen dennoch negative Effekte des Neurofeedback-Trainings zeigen, können Sie dieses jederzeit abbrechen. Wir können Ihnen für diesen Fall ein Training in „Positiven Imagery Techniken“ als alternative Behandlung anbieten.

Bei weiteren Fragen stehen wir Ihnen selbstverständlich gern zur Verfügung.

Ansprechpartner:

Studien-Informationen allgemein:
Dipl.-Psych. Jennifer Schmidt, Mobil: ….

Terminabsprachen für Neurofeedbacksitzungen:
Kamila Lewicki, Mobil: ….
Rahel Kuttner, Mobil: ….

psyrecon

psyrecon research & consulting GmbH
Bergische Universität Wuppertal
Heirich Heine Universität Düsseldorf

Projektleitung: Dipl.-Psych. Jennifer Schmidt

Ansprechpartner für eventuelle Rückfragen:
Dipl.-Psych. Jennifer Schmidt, Mobil: ….

Spezielle Informationen zum Neurofeedback und psychologischer Testdiagnostik

Studie: „Neurofeedback als Interventionsmethode bei Heißhungeranfällen“

Liebe Teilnehmerin,

im Rahmen einer wissenschaftlichen Studie möchten werden wir Neurofeedback-Sitzungen mit Ihnen durchführen. Das Neurofeedback arbeitet mit den Messungen der Gehirnströme, also eines Elektroenzephalogramms (EEG). Zudem arbeiten wir mit testdiagnostischen Fragebögen, um eventuelle, nicht erkannte Essstörungen festzustellen. In den folgenden Abschnitten erfahren Sie Näheres über beide Verfahren. Fragen Sie uns gerne, wenn Sie etwas nicht verstanden haben oder mehr über die Untersuchungsmethode erfahren möchten.

Neurofeedback – Methodik des EEG

Die Untersuchung dient der Erforschung der Funktionsweise des menschlichen Gehirns.

Während der Neurofeedbacksitzungen wird mit Hilfe von Elektroden, die *mit Hilfe einer elastischen Befestigungs-Kappe* auf der Kopfoberfläche befestigt werden, das EEG aufgezeichnet. Hierbei handelt es sich um die elektrische Aktivität des Gehirns, die an der Kopfoberfläche gemessen werden kann.

Die Aufzeichnung des EEGs ist beim Menschen mit keinen Risiken verknüpft. Da die Potentialfelder des Gehirns an der Kopfoberfläche sehr schwach sind, ist es erforderlich, dass Stellen, an denen Elektroden angebracht werden, mit Hilfe einer speziellen Paste und Alkohol gereinigt werden. Der Kontakt zwischen Elektrode und Kopfoberfläche wird über ein in Salzwasser (NaCl-Lösung) getränktes Stoffstück hergestellt. Alle verwendeten Produkte sind klinisch getestet und Rückstände lassen sich nach Abschluss jeder Sitzung leicht entfernen. In seltenen Fällen können trotzdem Hautirritationen auftreten. Manchmal bleiben noch für eine Weile Druckstellen an den Orten zurück, an denen die Elektroden bzw. die Elektrodenkappe befestigt wurden; in ganz seltenen Fällen sind die Stellen, an denen die Elektroden saßen, noch für ein paar Tage sichtbar (z. B. Rötungen). Bitte teilen Sie uns mit, falls Sie an bestimmten Hautallergien oder Überempfindlichkeiten der Haut leiden.

Testdiagnostik für Essstörungen

Wir werden Ihnen zwei verschiedene Fragebögen zur Diagnose möglicher unerkannter Essstörungen vorlegen. Bitte füllen Sie diese ehrlich aus. Alle Angaben werden natürlich streng vertraulich behandelt. Die Testverfahren erfassen, ob bei Ihnen eventuell klinisch relevante Essstörungen vorliegen könnten. Sollten wir auf den Fragebögen bedenklich hohe Werte feststellen, werden wir mit Ihnen ein intensiveres Interview (Dauer ca. 1 Stunde) in einer individuellen Sitzung durchführen, um die Ergebnisse zu überprüfen. Dafür werden wir sie in dem Falle vertraulich kontaktieren. Sollten wir in allen Verfahren feststellen, dass Ihre Fragebogenwerte die existierenden Schwellen für kritische Werte überschreiten, können wir Sie leider nicht in die Studie aufnehmen, da wir nicht autorisiert sind, Essstörungen zu behandeln.

Auffällige Befunde

Die Untersuchung dient ausschließlich Forschungszwecken. Eine medizinische oder psychologische Beurteilung Ihrer Daten erfolgt nicht. Es könnte uns jedoch sowohl im EEG, als auch in den Fragebögen zur Essstörungs-Diagnostik, ungewöhnliche Untersuchungsergebnisse auffallen. In diesem Fall werden wir Sie vertraulich auf die Auffälligkeiten hinweisen und Ihnen eine Kurzberatung zu möglichem weiteren Vorgehen geben. Wir empfehlen in diesem Falle, das Ergebnis bei Ihrem Hausarzt, einem Facharzt oder einem approbierten Psychotherapeuten professionell diagnostisch weiter abklären zu lassen.

Nur wenn Sie damit einverstanden sind, dass wir Sie ggf. über einen auffälligen Befund informieren, können Sie an dieser Studie teilnehmen. Ob Sie diesen weiter abklären lassen, steht Ihnen völlig frei.

Wir möchten Sie im Voraus darauf hinweisen, dass Ihnen, sollte bei der späteren diagnostischen Abklärung eine Erkrankung festgestellt werden, unter Umständen Nachteile entstehen können. So kann z.B. der Abschluss einer privaten Krankenversicherung, Berufsunfähigkeitsversicherung oder einer Lebensversicherung erschwert werden, mit höheren Beiträgen oder Ausschluss des Versicherungsschutzes für bestimmte Krankheitsfälle verbunden sein. Mit dem Unterzeichnen der Einverständniserklärung bestätigen Sie, diesen Hinweis zur Kenntnis genommen zu haben und mit dem Vorgehen einverstanden zu sein.

B.2 Declaration of consent – Study 1

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Heirich Heine Universität Düsseldorf

Projektleitung: Dipl.-Psych. Jennifer Schmidt

Ansprechpartner für eventuelle Rückfragen:
Dipl.-Psych. Jennifer Schmidt, Mobil: ….

Einwilligungserklärung „Neurofeedback als Interventionsmethode bei Heißhungeranfällen“

Ich _____ bin mündlich und schriftlich über die Studie und den Versuchsablauf aufgeklärt worden. Ich habe alle Informationen vollständig gelesen und verstanden. Sofern ich Fragen zu dieser vorgesehenen Studie hatte, wurden sie von Frau Jennifer Schmidt vollständig und zu meiner Zufriedenheit beantwortet.

Mit der beschriebenen Handhabung der erhobenen Daten bin ich einverstanden. Die Aufzeichnung und Auswertung der Daten erfolgt pseudonymisiert, d. h. unter Verwendung einer Nummer und ohne Angabe meines Namens. Es existiert eine Kodierliste auf Papier, die meinen Namen mit dieser Nummer verbindet. Diese Kodierliste ist nur dem Versuchsleiter zugänglich und wird nach Abschluss der Datenerhebung oder der Datenauswertung gelöscht. Mir ist bekannt, dass ich mein Einverständnis zur Aufbewahrung bzw. Speicherung dieser Daten widerrufen kann, ohne dass mir daraus Nachteile entstehen. Ich bin darüber informiert worden, dass ich jederzeit eine Löschung all meiner Daten verlangen kann. Wenn allerdings die Kodierliste bereits gelöscht ist, kann mein Datensatz nicht mehr identifiziert und also auch nicht mehr gelöscht werden. Meine Daten sind dann anonymisiert. Ich bin einverstanden, dass meine anonymisierten Daten zu Forschungszwecken weiter verwendet werden können und mindestens 10 Jahre gespeichert bleiben.

Sollten Auffälligkeiten im EEG oder in der Testdiagnostik erkannt werden, bin ich damit einverstanden, dass mir diese vertraulich mitgeteilt werden, so dass ich diese ggf. freiwillig weiter abklären lassen kann. Ich wurde darauf hingewiesen, dass eine spätere ärztliche Abklärung auffälliger Befunde bei der Diagnose eines Krankheitsbildes u.U. mit versicherungsrechtlichen Konsequenzen verbunden sein kann.

Ich wurde darüber aufgeklärt, dass das Auftreten negativer Effekte im Rahmen der Neurofeedback-Trainings sehr unwahrscheinlich ist, aber nicht völlig ausgeschlossen werden kann. Ich wurde über alternative Möglichkeiten informiert, die ich im Falle negativer Effekte in Anspruch nehmen kann. Ich hatte genügend Zeit für eine Entscheidung und bin bereit, an der o.g. Studie teilzunehmen. Ich weiß, dass die Teilnahme an der Studie freiwillig ist und ich die Teilnahme jederzeit ohne Angaben von Gründen beenden kann.

Eine Ausfertigung der Teilnehmerinformation über die Untersuchung, über EEG-Studien und Testdiagnostik und eine Ausfertigung der Einwilligungserklärung habe ich erhalten.

Ich stimme zudem der Verwendung meiner persönlichen Daten nach oben beschriebenem Vorgehen zu. O JA O NEIN.

Ich nehme zur Kenntnis, dass ich die Möglichkeit habe, die Untersuchung oder einzelne Sitzungen jederzeit abzubrechen und meine Teilnahme völlig freiwillig erfolgt. O JA O NEIN

Ort, Datum & Unterschrift des Teilnehmers: Name des Teilnehmers in Druckschrift:

_____ _____

Ort, Datum & Unterschrift des Versuchsleiters: Name des Versuchsleiters in Druckschrift:

_____ _____

Zusatzvereinbarung für künftige Kontaktaufnahmen im Rahmen dieser Studie

Ich gebe mein Einverständnis, dass im Falle einer Fortführung dieser Studie oder von Anschlussstudien meine personenbezogenen Daten für eine erneute Kontaktaufnahme weiter verwendet werden dürfen. Ich bin darüber informiert, dass meine Daten bis zum endgültigen Abschluss der Datenerhebung und/oder Auswertung weiterhin in pseudonymisierter Form (Kodierliste) vorliegen und nur die Studienleitung darauf Zugriff hat. Nach spätestens 10 Jahren werden meine personenbezogenen Daten gelöscht. Bis dahin kann ich jederzeit Auskunft über meine personenbezogenen Daten erhalten und die Löschung meiner Daten verlangen.

O JA O NEIN.

Rückmeldung von Ergebnissen

Ich bin daran interessiert, etwas über die Ergebnisse der Studie zu erfahren, und bitte hierzu um Übersendung entsprechender Informationen.

O JA O NEIN.

Bei Fragen oder anderen Anliegen kann ich mich an folgende Person wenden:

Frau Dipl.-Psych. Jennifer Schmidt

c/o psyrecon research & consulting
Institut für angewandte Psychophysiologie GmbH
Alte Freiheit 1
42103 Wuppertal

Tel.: ….

Mobil: ….

E-Mail: ….

B.3 Neurofeedback evaluation questionnaire – Study 1

VP: Datum: Gruppe:

Allgemeines Feedback zur Studie Neurofeedback bei Heißhungeranfällen

Sie haben nun ihre zehnte Neurofeedback-Sitzung abgeschlossen! Für die Auswertung unserer Studie und zukünftige Verbesserungen, hilft es uns sehr, wenn Sie uns ein ehrliches Feedback zu Ihren Erfahrungen mit dem Neurofeedback-Training geben. Auch Kritik hilft uns, unser Vorgehen in Zukunft zu verbessern. Bitte beantworten Sie daher die nachfolgenden Fragen ehrlich und offen.

Wie haben Sie das Neurofeedback-Training **insgesamt** empfunden?

O	O	O	O	O
sehr negativ	negativ	mittelmäßig	positiv	sehr positiv

Das Neurofeedback-Training hatte Auswirkungen auf mein **Essverhalten**.

O	O	O	O	O
überhaupt nicht	etwas	ziemlich	stark	sehr stark

Das Neurofeedback-Training hatte für mich persönlich Auswirkungen **auf das Auftreten von Heißhungeranfällen.**

O	O	O	O	O
überhaupt nicht	etwas	ziemlich	stark	sehr stark

Das Neurofeedback-Training hatte für mich persönlich Auswirkung **auf meinen Umgang mit Heißhungeranfällen.**

O	O	O	O	O
überhaupt nicht	etwas	ziemlich	stark	sehr stark

Das Neurofeedback-Training hatte für mich persönlich Auswirkung auf mein **Stressempfinden.**

O	O	O	O	O
überhaupt nicht	etwas	ziemlich	stark	sehr stark

Das Neurofeedback-Training hatte für mich persönlich Auswirkung auf mein **Wohlbefinden.**

O	O	O	O	O
überhaupt nicht	etwas	ziemlich	stark	sehr stark

Ich selbst habe während meiner Neurofeedback-Trainingsphase Veränderungen in meinem Verhalten gemerkt.

O	O	O	O	O
überhaupt nicht	etwas	ziemlich	stark	sehr stark

Mein Umfeld hat während meiner Neurofeedback-Trainingsphase Veränderungen in meinem Verhalten bemerkt.

O	O	O	O	O
überhaupt nicht	etwas	ziemlich	stark	sehr stark

VP: Datum: Gruppe:

Ich sehe Neurofeedback-Training für mich als **geeignet** an.

O	O	O	O	O
überhaupt nicht	etwas	ziemlich	stark	sehr stark

Ich war mit dem Neurofeedback-Training insgesamt **zufrieden.**

O	O	O	O	O
überhaupt nicht	etwas	ziemlich	stark	sehr stark

Das Neurofeedback-Training hat sich für mich **gelohnt**.

O	O	O	O	O
überhaupt nicht	etwas	ziemlich	stark	sehr stark

Ich kann mir vorstellen, die Strategien, die ich im Neurofeedback-Training gelernt habe zukünftig **im Alltag** einzusetzen.

O	O	O	O	O
überhaupt nicht	etwas	ziemlich	stark	sehr stark

Ich würde das Neurofeedback-Training Menschen mit Heißhungeranfällen **weiterempfehlen.**

O	O	O	O	O
überhaupt nicht	etwas	ziemlich	stark	sehr stark

Bitte beantworten Sie nachfolgend noch diese offenen Fragen. Jeder Kommentar, egal ob positiv oder negativ, hilft uns sehr weiter!

Was genau hat Ihnen am besten geholfen?

_____
_____
_____

Was hat Sie am meisten gestört?

_____
_____
_____

Haben Sie Verbesserungsvorschläge für uns?

_____
_____
_____
_____

B.4 Participant information – Studies 2 & 3

Allgemeine Aufklärung zur Studie
„Psychologisches Training bei Heißhungeranfällen“

Liebe Teilnehmerin,

wir freuen uns über Ihre Bereitschaft, als Probandin in unserer wissenschaftlichen Studie **„Psychologisches Training bei Heißhungeranfällen“** unter Leitung von Prof. Dr. Alexandra Martin, (Lehrstuhl für Klinische Psychologie und Psychotherapie, Bergische Universität Wuppertal) teilzunehmen. In dieser Aufklärung möchten wir Ihnen vorab genauer mitteilen, was Sie im Rahmen der Studie erwarten wird. Bei der Studie handelt es sich um eine psychologische Interventionsstudie, deren Ergebnisse in zwei Abschlussarbeiten des Psychologie-Studiums und einer Doktorarbeit Fach Psychologie wissenschaftlich ausgewertet werden. Wir bedanken uns daher sehr herzlich, dass Sie unsere Forschung durch Ihr Interesse und Ihre Teilnahme unterstützen.

Sie haben sich entschlossen, eine psychologische **Trainingsmethode** auszuprobieren. Es wird zwei verschiedene Gruppen im Rahmen des Trainings geben, sowie eine Wartelistengruppe, welche mit dem Training mithilfe einer der beiden Methoden zu einem späteren Zeitpunkt startet. Welcher Gruppe sie zugeordnet werden erfolgt nach dem Zufallsprinzip. In einer der beiden Trainingsgruppen wird das Neurofeedback als hirnstrombasiertes Verfahren (EEG) angewandt. Dies ist eine Methode, die Ihre Gehirnströme absolut ungefährlich und schmerzfrei mithilfe von Elektroden an ihrer Kopfhaut erfasst. Für das EEG wird Ihre Kopfhaut in jeder Sitzung mit einem leichten Peeling vorbehandelt. Die Elektroden werden mit einem in Salzwasser befeuchteten Stoffbezug bezogen. Alle verwendeten Vorbereitungs-Produkte sind dermatologisch getestet und alle verwendeten Geräte medizinisch zertifiziert. Der Schwerpunkt in der anderen Gruppe wird auf der Fokussierung des inneren Erlebens und der gezielten Entspannung während der Konfrontation mit Bildern von Heißhungerlebensmitteln liegen (Imagery-Training).

Heißhungeranfälle gehen mit einem Muster eines besonders gestressten Zustands im Gehirn einher. In beiden Trainingsgruppen lernen Sie dieses Muster gezielt zu beeinflussen.

Datenschutz:
Alle erhobenen Daten werden anonymisiert und streng vertraulich nach Vorgaben der Datenschutzrichtlinien behandelt. Von Ihnen bereitgestellte Kontaktdaten und personenbezogene Informationen werden lediglich für die Dauer der Studiendurchführung zur Absprache von Terminen erhoben und gespeichert und im Anschluss, oder nach Aufforderung ihrerseits gelöscht. Die Erhebung der Daten in Form von Fragebögen und in den Trainingssitzungen erfolgt pseudonymisiert, d. h. in namentlich nicht gekennzeichneter Form. Ihre Antworten und Ergebnisse werden unter einer Nummer gespeichert. Es existiert eine Kodierliste auf Papier, die Ihren Namen mit der Nummer verbindet, was für die Auswertung der Daten erforderlich sein könnte. Die Kodierliste ist nur den Projektmitarbeitern zugänglich; sie wird in einem abschließbaren Schrank aufbewahrt und nach Abschluss der Datenerhebung vernichtet. Nach Vernichtung der Kodierliste liegen die Daten nur noch als Nummer in vollständig anonymisierter Form vor; ein Rückschluss auf den einzelnen Probanden ist dann nicht mehr möglich. Die anonymisierten Daten werden mindestens 10 Jahre gespeichert. Sie können, wann immer Sie möchten, die Löschung aller von Ihnen erhobenen Daten verlangen. Wenn die Kodierliste aber erst einmal gelöscht ist, können wir Ihren Datensatz nicht mehr identifizieren. Deshalb können wir Ihrem Verlangen nach Löschung Ihrer Daten nur solange nachkommen, wie die Kodierliste existiert. Die im Rahmen dieser Studie erhobenen Daten und persönlichen Mitteilungen werden vertraulich behandelt. Mitarbeiter, die durch direkten Kontakt mit Ihnen über personenbezogene Daten verfügen, sind verpflichtet, diese nicht an Dritte weiterzugeben. Desweiteren wird die Veröffentlichung der Ergebnisse der Studie in anonymisierter Form erfolgen, d. h. ohne dass Ihre Daten Ihrer Person zugeordnet werden können.

Die Teilnahme an der Studie ist freiwillig. Sie können jederzeit und ohne Angabe von Gründen Ihre Einwilligung zur Teilnahme an dieser Studie widerrufen, ohne dass Ihnen daraus Nachteile entstehen. **Falls Sie zu irgendeinem Zeitpunkt - aus welchen Gründen auch immer - eine Sitzung oder die**

gesamte Behandlung abbrechen möchten, steht Ihnen dies völlig frei. Teilen Sie uns dies in dem Fall bitte mit. Sie werden zur Studienteilnahme eine Einverständniserklärung ausfüllen, in der Sie uns bestätigen, dass Sie über diese Möglichkeit aufgeklärt wurden und freiwillig an der Studie teilnehmen.

Es handelt sich bei dieser Studie um eine zweite Untersuchung dieser Methodik bei Heißhungeranfällen. Nach gründlicher Recherche können wir sagen, dass das Auftreten negativer Effekte sehr unwahrscheinlich ist. Sollten sich bei Ihnen dennoch negative Effekte des Trainings zeigen, können Sie dieses jederzeit abbrechen. Wir können Ihnen für diesen Fall das Ausprobieren der jeweils anderen Methode bzw. eine individualisierte Ernährungsberatung anbieten.

Bei weiteren Fragen stehen wir Ihnen selbstverständlich gern zur Verfügung.

Ansprechpartner:

Wissenschaftliche Leitung:
Prof. Dr. Alexandra Martin (Lehrstuhl für Klinische Psychologie und Psychotherapie, Bergische Universität Wuppertal)

Studien-Informationen allgemein:
Dipl.-Psych. Jennifer Schmidt, Mobil: ….

Terminabsprachen für Trainingssitzungen:
Dilek Soysal, ….@uni-wuppertal.de,
Jenny Bullerjahn, ….@uni-wuppertal.de .

Aufklärung zum Studienablauf

Die Studie unterteilt sich in folgende Bestandteile:

- Teilnahme an der Einführungsveranstaltung sowie ein letztes Screening der Teilnahmevoraussetzungen
- Aufteilung in Gruppen (Neurofeedback, Imagery, Warteliste)
- Terminvereinbarung für die Sitzungen
- Erstellung der individuellen Trainingsmaterialien
- **10 Sitzungen** nach individueller Terminvereinbarung (je 45-50 Min)

    1. Sitzung: Psychophysiologische Messung, Fragebögen und Training
    2.-4. Sitzung: Training
    5. Sitzung: Fragebögen und Training
    6.-9. Sitzung: Training
    10. Sitzung: Psychophysiologische Messung, Fragebögen und Training

    Follow-Up (3 Monate nach letzter Sitzung)

In der ersten und zehnten Sitzung werden Messungen der Hirnströme, des Herzschlag und der Hautleitfähigkeit vorgenommen. Dies bedeutet einen Mehraufwand von circa 30 Minuten. Da wir während dieser beiden Sitzungen eine gelartige Paste auf Ihrer Kopfhaut verwenden werden, bringen Sie am besten eine Kopfbedeckung mit!

Um wissenschaftlich haltbare Ergebnisse zu erzielen, sind uns ehrliche Auskünfte ihrerseits, sowie die **zuverlässige Teilnahme** an Terminen zum Training und zur Datenerfassung wichtig. Sollten Sie einen Termin nicht wahrnehmen können, würden wir gern zu einem schnellstmöglichen Zeitpunkt einen Ersatztermin mit Ihnen vereinbaren. Sollten Sie zu einem bereits abgesprochenen Termin verhindert sein (Krankheit, Notfälle o.Ä.), geben Sie uns bitte umgehend, bestenfalls bis 24 Stunden vor Ihrem Termin, Bescheid. Unsere Kontaktdaten finden Sie am Ende dieses Informationsschreibens.

Der „Allgemeinen Aufklärung zur Studie“ (Anlage 1a) können Sie entnehmen, dass das Training aus zwei verschiedenen Gruppen bestehen wird. Der Trainingszeitraum der ersten beiden Gruppen findet über einen Zeitraum von 8 Wochen von circa **April** bis **Mai 2014** statt. Der Trainingszeitraum der Wartelistengruppe wird circa von **Mai** bis **Ende Juni 2014** stattfinden.

Wir würden Sie im Rahmen der Studie bitten, ihre Ernährung (abgesehen natürlich von den Effekten auf Ihre Heißhungeranfälle) nicht gezielt auf eine Diät zur Gewichtsreduktion umzustellen (z.B. Shake-Diäten, FDH, Dinner Cancelling, Atkins-Diät o.Ä.). Sollten sie bisher bereits dauerhaft eine bestimmte Ernährungsform einhalten (wie z.B. nach Weight Watchers, Low Fat 30 oder vegatarische/vegane Ernährung), behalten Sie diese einfach nach wie vor bei.

Damit wir ihre Trainingssitzungen individuell auf Sie zuschneiden können, brauchen wir vorab von Ihnen eine möglichst genaue **Auflistung der Lebensmittel**, die bei Ihnen Heißhunger auslösen. Diese Auflistung sollten Sie uns bitte schnellstmöglich zur Verfügung stellen (schriftlich oder per E-Mail). Die aufgezählten Lebensmittel werden Ihnen in jeder Trainingssitzung als Bilder präsentiert. Sollte es sich bei Ihren persönlichen Heißhunger-Lebensmitteln um sehr ungewöhnliche oder spezielle Produkte handeln, lassen Sie uns bitte wenn möglich ein Foto davon zukommen. In der ersten Sitzung werden wir zur Sicherheit zudem einen Test auf mögliche unerkannte Essstörungen bei Ihnen durchführen. Dies ist im Rahmen von Vorschriften zur Durchführung psychologischer Interventionen notwendig.

Um den Fortschritt des Trainings bei Ihnen festzustellen, werden wir zu verschiedenen Zeitpunkten in der ersten Sitzung, in der fünften Sitzung, in der zehnten Sitzung, sowie drei Monate nach der zehnten Sitzung, sofern Sie damit einverstanden sind, folgende Daten von Ihnen erheben: Gewicht, Angaben

zur Häufigkeit Ihrer Heißhungeranfälle und Ihrem Essverhalten sowie Angaben in einigen Fragebögen (z.B. zum individuellen Stress).

Sie erhalten im Rahmen der Studie zehn kostenlose Trainings-Sitzungen. Die Studie wird nicht anderweitig vergütet.

Bei weiteren Fragen stehen wir Ihnen selbstverständlich gern zur Verfügung.

Ansprechpartner:

Wissenschaftliche Leitung:
Prof. Dr. Alexandra Martin (Lehrstuhl für Klinische Psychologie und Psychotherapie, Bergische Universität Wuppertal)

Studien-Informationen allgemein:
Dipl.-Psych. Jennifer Schmidt, Mobil: ….

Terminabsprachen für Trainingssitzungen:
Dilek Soysal, ….@uni-wuppertal.de ,
Jenny Bullerjahn, ….@uni-wuppertal.de .

Bergische Universität Wuppertal
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Wissenschaftliche Leitung: Prof. Dr. Alexandra Martin
Lehrstuhl für Klinische Psychologie und Psychotherapie
Bergische Universität Wuppertal

Projektleitung: Dipl.-Psych. Jennifer Schmidt

Ansprechpartner für eventuelle Rückfragen:
Dipl.-Psych. Jennifer Schmidt, Mobil: ….

Spezielle Informationen zum EEG und psychologischer Testdiagnostik

Studie: „Psychologisches Training bei Heißhungeranfällen“

Liebe Teilnehmerin,

im Rahmen einer wissenschaftlichen Studie möchten wir EEG-Sitzungen mit Ihnen durchführen. Das EEG arbeitet mit den Messungen der Gehirnströme, also eines Elektroenzephalogramms (EEG). Zudem arbeiten wir mit einem testdiagnostischen Verfahren, um eventuelle, nicht erkannte Essstörungen festzustellen. In den folgenden Abschnitten erfahren Sie Näheres über beide Verfahren. Fragen Sie uns gerne, wenn Sie etwas nicht verstanden haben oder mehr über die Untersuchungsmethode erfahren möchten.

Methodik des EEG

Die Untersuchung dient der Erforschung der Funktionsweise des menschlichen Gehirns.

Während der EEG-Sitzungen wird mit Hilfe von Elektroden, die *mit Hilfe einer elastischen Befestigungs-Kappe* auf der Kopfoberfläche befestigt werden, das EEG aufgezeichnet. Hierbei handelt es sich um die elektrische Aktivität des Gehirns, die an der Kopfoberfläche gemessen werden kann.

Die Aufzeichnung des EEGs ist beim Menschen mit keinen Risiken verknüpft. Da die Potentialfelder des Gehirns an der Kopfoberfläche sehr schwach sind, ist es erforderlich, dass Stellen, an denen Elektroden angebracht werden, mit Hilfe einer speziellen Paste und Alkohol gereinigt werden. Der Kontakt zwischen Elektrode und Kopfoberfläche wird über eine spezielle Leitpaste hergestellt. Alle verwendeten Produkte sind klinisch getestet und Rückstände lassen sich nach Abschluss jeder Sitzung leicht entfernen. In seltenen Fällen können trotzdem Hautirritationen auftreten. Manchmal bleiben noch für eine Weile Druckstellen an den Orten zurück, an denen die Elektroden bzw. die Elektrodenkappe befestigt wurden; in ganz seltenen Fällen sind die Stellen, an denen die Elektroden saßen, noch für eine kurze Zeit sichtbar (z. B. Rötungen). Bitte teilen Sie uns mit, falls Sie an bestimmten Hautallergien oder Überempfindlichkeiten der Haut leiden.

Testdiagnostik für Essstörungen

Wir werden Ihnen einen Fragebogen zur Diagnose möglicher unerkannter Essstörungen vorlegen. Bitte füllen Sie diesen ehrlich aus. Alle Angaben werden natürlich streng vertraulich behandelt. Das Testverfahren erfasst, ob bei Ihnen eventuell klinisch relevante Essstörungen vorliegen könnten. Sollten wir auf dem Fragebogen bedenklich hohe Werte feststellen, werden wir mit Ihnen ein intensiveres Interview (Dauer ca. 1 Stunde) in einer individuellen Sitzung durchführen, um die Ergebnisse zu überprüfen. Dafür werden wir sie in dem Falle vertraulich kontaktieren. Sollten wir in den Verfahren feststellen, dass Ihre Angaben auf das Vorliegen einer Essstörung hindeuten, können wir Sie leider nicht in die Studie aufnehmen, da wir nicht autorisiert sind, Essstörungen zu behandeln.

Auffällige Befunde

Die Untersuchung dient ausschließlich Forschungszwecken. Eine medizinische oder psychologische Beurteilung Ihrer Daten erfolgt nicht. Es könnte uns jedoch sowohl im EEG, als auch im Fragebogen zur Essstörungs-Diagnostik, ungewöhnliche Untersuchungsergebnisse auffallen. In diesem Fall werden wir Sie vertraulich auf die Auffälligkeiten hinweisen und Ihnen eine Kurzberatung zu möglichem weiteren Vorgehen geben. Wir empfehlen in diesem Falle, das Ergebnis bei Ihrem Hausarzt, einem Facharzt oder einem approbierten Psychotherapeuten professionell diagnostisch weiter abklären zu lassen.

Nur wenn Sie damit einverstanden sind, dass wir Sie ggf. über einen auffälligen Befund informieren, können Sie an dieser Studie teilnehmen. Ob Sie diesen weiter abklären lassen, steht Ihnen völlig frei.

Wir möchten Sie im Voraus darauf hinweisen, dass Ihnen, sollte bei der späteren diagnostischen Abklärung eine Erkrankung festgestellt werden, unter Umständen Nachteile entstehen können. So kann z.B. der Abschluss einer privaten Krankenversicherung, Berufsunfähigkeitsversicherung oder einer Lebensversicherung erschwert werden, mit höheren Beiträgen oder Ausschluss des Versicherungsschutzes für bestimmte Krankheitsfälle verbunden sein. Mit dem Unterzeichnen der Einverständniserklärung bestätigen Sie, diesen Hinweis zur Kenntnis genommen zu haben und mit dem Vorgehen einverstanden zu sein.

B.5 Declaration of consent – Studies 2 & 3

Bergische Universität Wuppertal
psyrecon research & consulting GmbH

Wissenschaftliche Leitung: Prof. Dr. Alexandra Martin
Lehrstuhl für Klinische Psychologie und Psychotherapie
Bergische Universität Wuppertal

Projektleitung: Dipl.-Psych. Jennifer Schmidt

Ansprechpartner für eventuelle Rückfragen:
Dipl.-Psych. Jennifer Schmidt, Mobil: ….

Einwilligungserklärung „Psychologisches Training bei Heißhungeranfällen“

Ich _____ bin mündlich und schriftlich über die Studie und den Versuchsablauf aufgeklärt worden. Ich habe alle Informationen vollständig gelesen und verstanden. Sofern ich Fragen zu dieser vorgesehenen Studie hatte, wurden sie von Frau Jennifer Schmidt vollständig und zu meiner Zufriedenheit beantwortet.

Mit der beschriebenen Handhabung der erhobenen Daten bin ich einverstanden. Die Aufzeichnung und Auswertung der Daten erfolgt pseudonymisiert, d. h. unter Verwendung einer Nummer und ohne Angabe meines Namens. Es existiert eine Kodierliste auf Papier, die meinen Namen mit dieser Nummer verbindet. Diese Kodierliste ist nur dem Versuchsleiter zugänglich und wird nach Abschluss der Datenerhebung oder der Datenauswertung gelöscht. Mir ist bekannt, dass ich mein Einverständnis zur Aufbewahrung bzw. Speicherung dieser Daten widerrufen kann, ohne dass mir daraus Nachteile entstehen. Ich bin darüber informiert worden, dass ich jederzeit eine Löschung all meiner Daten verlangen kann. Wenn allerdings die Kodierliste bereits gelöscht ist, kann mein Datensatz nicht mehr identifiziert und also auch nicht mehr gelöscht werden. Meine Daten sind dann anonymisiert. Ich bin einverstanden, dass meine anonymisierten Daten zu Forschungszwecken weiter verwendet werden können und mindestens 10 Jahre gespeichert bleiben.

Sollten behandlungsbedürftige Auffälligkeiten im EEG oder in der Testdiagnostik erkannt werden, bin ich damit einverstanden, dass mir diese mitgeteilt werden, so dass ich diese ggf. weiter abklären lassen kann. Ich wurde darüber informiert, dass die weitere Abklärung auffälliger Befunde u.U. mit versicherungsrechtlichen Konsequenzen verbunden sein kann.

Ich wurde darüber aufgeklärt, dass das Auftreten negativer Effekte im Rahmen der Intervention sehr unwahrscheinlich ist, aber nicht völlig ausgeschlossen werden kann. Ich wurde über alternative Möglichkeiten informiert, die ich im Falle negativer Effekte in Anspruch nehmen kann. Ich hatte genügend Zeit für eine Entscheidung und bin bereit, an der o.g. Studie teilzunehmen. Ich weiß, dass die Teilnahme an der Studie freiwillig ist und ich die Teilnahme jederzeit ohne Angaben von Gründen beenden kann.

Eine Ausfertigung der Teilnehmerinformation über die Untersuchung, über EEG-Studien und Testdiagnostik und eine Ausfertigung der Einwilligungserklärung habe ich erhalten.

Ich stimme zudem der Verwendung meiner persönlichen Daten nach oben beschriebenem Vorgehen zu. O JA O NEIN.

Ich nehme zur Kenntnis, dass ich die Möglichkeit habe, die Untersuchung oder einzelne Sitzungen jederzeit abzubrechen und meine Teilnahme völlig freiwillig erfolgt. O JA O NEIN.

Ort, Datum & Unterschrift des Teilnehmers: Name des Teilnehmers in Druckschrift:

Ort, Datum & Unterschrift des Versuchsleiters: Name des Versuchsleiters in Druckschrift:

Zusatzvereinbarung für künftige Kontaktaufnahmen im Rahmen dieser Studie

Ich gebe mein Einverständnis, dass im Falle einer Fortführung dieser Studie oder von Anschlussstudien meine personenbezogenen Daten durch die Bergische Universität Wuppertal für eine erneute Kontaktaufnahme weiter verwendet werden dürfen. Ich bin darüber informiert, dass meine Daten bis zum endgültigen Abschluss der Datenerhebung und/oder Auswertung weiterhin in pseudonymisierter Form (Kodierliste) vorliegen und nur die Studienleitung darauf Zugriff hat. Nach spätestens 10 Jahren werden meine personenbezogenen Daten gelöscht. Bis dahin kann ich jederzeit Auskunft über meine personenbezogenen Daten erhalten und die Löschung meiner Daten verlangen.

O JA O NEIN.

Rückmeldung von Ergebnissen

Ich bin daran interessiert, etwas über die Ergebnisse der Studie zu erfahren, und bitte hierzu um Übersendung entsprechender Informationen.

O JA O NEIN.

Bei Fragen oder anderen Anliegen kann ich mich an folgende Person wenden:

Frau Dipl.-Psych. Jennifer Schmidt

c/o psyrecon research & consulting
Institut für angewandte Psychophysiologie GmbH
Alte Freiheit 1
42103 Wuppertal

Tel.: ….

Mobil: ….

E-Mail: ….

B.6 Treatment evaluation questionnaire – Study 2

VP:	Datum:	Sitzung:	Gruppe:

Allgemeines Feedback zur Studie Training gegen Heißhungeranfälle

Sie haben nun ihre zehnte Trainings-Sitzung abgeschlossen! Für die Auswertung unserer Studie und zukünftige Verbesserungen, hilft es uns sehr, wenn Sie uns ein ehrliches Feedback zu Ihren Erfahrungen mit dem Training geben. Auch Kritik hilft uns, unser Vorgehen in Zukunft zu verbessern. Bitte beantworten Sie daher die nachfolgenden Fragen ehrlich und offen.

Wie haben Sie das Training **insgesamt** empfunden?

O	O	O	O	O
sehr negativ	negativ	mittelmäßig	positiv	sehr positiv

Das Training hatte Auswirkungen auf mein **Essverhalten**.

O	O	O	O	O
überhaupt nicht	etwas	ziemlich	stark	sehr stark

Das Training hatte für mich persönlich Auswirkungen **auf das Auftreten von Heißhungeranfällen.**

O	O	O	O	O
überhaupt nicht	etwas	ziemlich	stark	sehr stark

Das Training hatte für mich persönlich Auswirkung **auf meinen Umgang mit Heißhungeranfällen.**

O	O	O	O	O
überhaupt nicht	etwas	ziemlich	stark	sehr stark

Das Training hatte für mich persönlich Auswirkung auf mein **Stressempfinden.**

O	O	O	O	O
überhaupt nicht	etwas	ziemlich	stark	sehr stark

Das Training hatte für mich persönlich Auswirkung auf mein **Wohlbefinden.**

O	O	O	O	O
überhaupt nicht	etwas	ziemlich	stark	sehr stark

Ich selbst habe während meiner Trainingsphase Veränderungen in meinem Verhalten gemerkt.

O	O	O	O	O
überhaupt nicht	etwas	ziemlich	stark	sehr stark

Mein Umfeld hat während meiner Trainingsphase Veränderungen in meinem Verhalten bemerkt.

O	O	O	O	O
überhaupt nicht	etwas	ziemlich	stark	sehr stark

VP:	Datum:	Sitzung:	Gruppe:

Ich sehe Training für mich als **geeignet** an.

O	O	O	O	O
überhaupt nicht	etwas	ziemlich	stark	sehr stark

Ich war mit dem Training insgesamt **zufrieden.**

O	O	O	O	O
überhaupt nicht	etwas	ziemlich	stark	sehr stark

Das Training hat sich für mich **gelohnt**.

O	O	O	O	O
überhaupt nicht	etwas	ziemlich	stark	sehr stark

Ich kann mir vorstellen, die Strategien, die ich im Training gelernt habe zukünftig **im Alltag** einzusetzen.

O	O	O	O	O
überhaupt nicht	etwas	ziemlich	stark	sehr stark

Ich würde das Training Menschen mit Heißhungeranfällen **weiterempfehlen.**

O	O	O	O	O
überhaupt nicht	etwas	ziemlich	stark	sehr stark

Bitte beantworten Sie nachfolgend noch diese offenen Fragen. Jeder Kommentar, egal ob positiv oder negativ, hilft uns sehr weiter!

Was genau hat Ihnen am besten geholfen?

_____

_____

_____

_____

Was hat Sie am meisten gestört?

_____

_____

_____

Haben Sie Verbesserungsvorschläge für uns?

_____

_____

_____

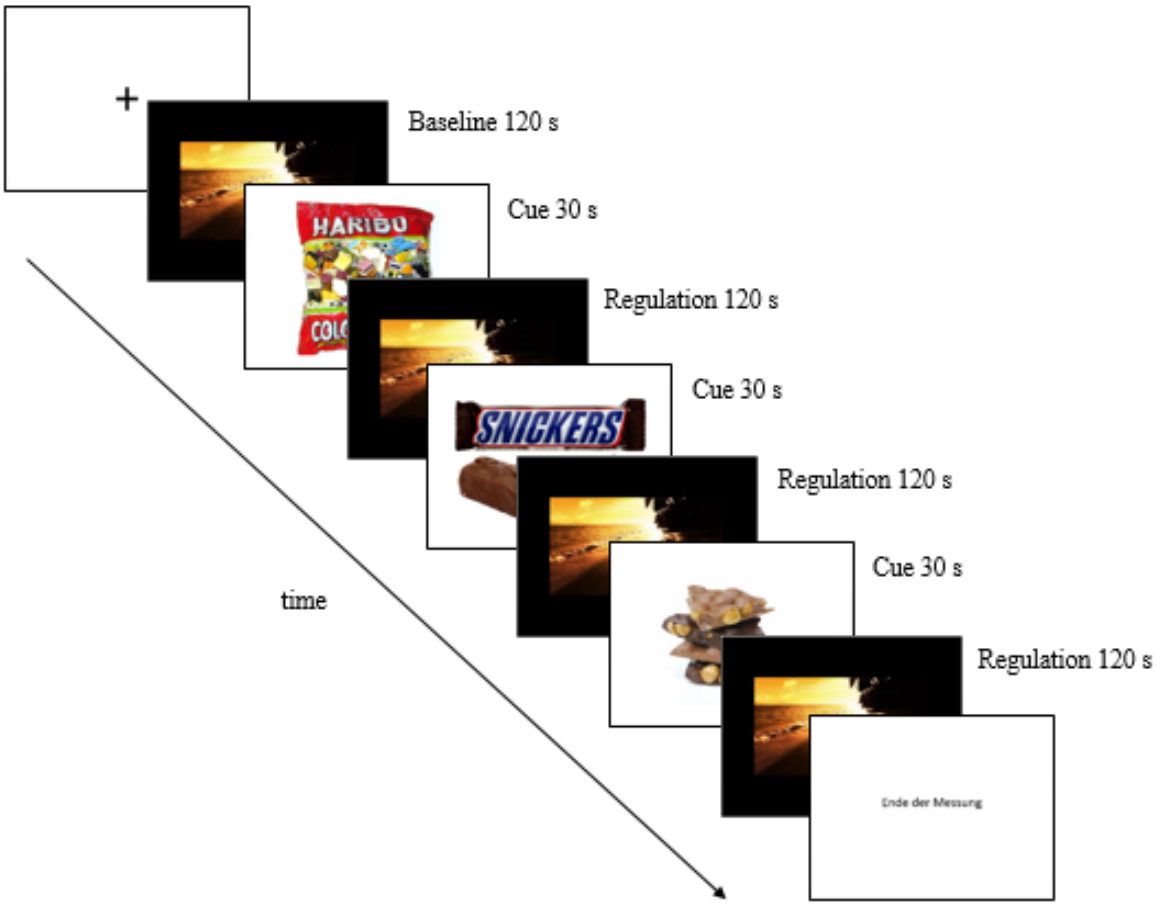
B.7 Somatic self-efficacy questionnaire – Studies 2 & 3

KK	Stimme gar nicht zu						Stimme absolut zu
Ich habe eine starke Kontrolle über meine körperlichen Reaktionen.	(0)	(1)	(2)	(3)	(4)	(5)	(6)
In der Regel tut mein Körper, was mein Geist befiehlt.	(0)	(1)	(2)	(3)	(4)	(5)	(6)
Es fällt leicht, mich wieder zu beruhigen, wenn ich aufgeregt bin.	(0)	(1)	(2)	(3)	(4)	(5)	(6)
Ich verfüge über Strategien, um mich gezielt zu entspannen, wenn es nötig ist.	(0)	(1)	(2)	(3)	(4)	(5)	(6)
Ich kann meine körperlichen Reaktionen beeinflussen, wenn ich es möchte.	(0)	(1)	(2)	(3)	(4)	(5)	(6)

B.8 Experimental design – Study 3

Instruction:

> **Liebe Teilnehmerin**
>
> Wir werden Ihnen gleich für jeweils 30 Sekunden die drei Lebensmittel präsentieren, die in Ihnen am stärksten Heißhungeranfälle auslösen.
>
> Bitte stellen Sie sich die Lebensmittel bei Betrachtung der Bilder so lebhaft wie möglich vor (Geruch, Geschmack, Konsistenz etc.)!
>
> Anschließend sollen Sie sich jeweils nach Ihrer individuellen Strategie für 2 Minuten entspannen.
>
> Vor der ersten Bildpräsentation werden wir eine 2-minütige Ruhemessung durchführen. Insgesamt dauert die Messung ca. 10 Minuten.
>
> Bitte bleiben Sie während der gesamten Messung möglichst ruhig sitzen und sprechen Sie nicht.



C. Declaration

Name, Vorname: Schmidt, Jennifer Sabrina

Anschrift: ….

E-Mail / Tel.: jschmidt@uni-wuppertal.de / 0202-439 5057

Erklärung

Gem. § 10 der Promotionsordnung der Fakultät 2, Human- und Sozialwissenschaften, vom 21.05.2015

Hiermit erkläre ich, Jennifer Sabrina Schmidt,

1. dass ich die von mir eingereichte Dissertation selbständig und ohne fremde Hilfe verfasst habe,
2. dass ich bei der Abfassung der Arbeit nur die in der Dissertation angegebenen Hilfsmittel benutzt und alle wortwörtlich oder inhaltlich übernommenen Stellen als solche gekennzeichnet habe,
3. dass die Dissertation in der gegenwärtigen oder einer anderen Fassung keinem anderen Fachbereich einer wissenschaftlichen Hochschule vorgelegen hat.

Ich bin damit einverstanden, dass meine Dissertation wissenschaftlich interessierten Personen oder Institutionen zur Einsichtnahme zur Verfügung gestellt werden kann und unter Wahrung urheberrechtlicher Grundsätze zitiert werden darf. Korrektur- oder Bewertungshinweise in meiner Arbeit dürfen nicht zitiert werden.

Wuppertal, 13.07.2016

_____

(Unterschrift)

D. Curriculum Vitae

For privacy reasons, the curriculum vitae is not included in the online-version of this dissertation.

Der Lebenslauf ist in der Online-Version der Dissertation aus Gründen des Datenschutzes nicht enthalten.

Publications

***Schmidt, J.,** & Martin, A. (submitted). Physiological vs. psychological learning in neurofeedback against binge eating.

Schmidt, J. & Martin, A. (2017). "Smile away your cravings" – Facial feedback modulates cue-induced food cravings. *Appetite, 116*, 536-543.

Schmidt, J., Kärgel, C. & Opwis, M. (2017). Neurofeedback in Substance Use and Overeating: Current Applications and Future Directions. *Current Addiction Reports, 4*(2), 116-131.

Opwis, M.[a], **Schmidt, J.**[a], Martin, A. & Salewski, C. (2017). Gender differences in eating behavior and eating pathology: The mediating role of rumination. Appetite, 110, 103-107. ([a] shared first authorship).

*** 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.

Stürmer, R., Blaak, J., Opwis, M., **Schmidt, J**., Staib, P., Wohlfart, R., & Boucsein, W. (2015). A psychophysiological approach to substantiate efficacy of bath additives. *IFSCC Magazine, 18*(3), 23-30.

Eisfeld, W., Prinz, D., Schröder, B., **Schmidt, J.,** & Stürmer, R. (2015). Investigation of consumers' hair shine perception by eye tracking technology in combination with assessment of physiological body reactions. *IFSCC Magazine, 18*(3), 3-9.

***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.

Langner, T., **Schmidt, J.,** & Fischer, A. (2015). Is it really love? A comparative investigation of the emotional nature of brand and interpersonal love. *Psychology & Marketing, 32*(6), 624-634.

Stürmer, R., & **Schmidt, J.** (2014). *Erfolgreiches Marketing durch Emotionsforschung. Messung, Analyse, Best-Practice.* Freiburg, Germany: Haufe.

Stürmer, R., & **Schmidt, J.** (2014). Wie man den Emotionswert von Produkten messen kann. In H.-G. Häusel (Ed.), *Neuromarketing - Erkenntnisse der Hirnforschung für Markenführung, Werbung und Verkauf,* (3rd Ed.) (pp. 115-132). Freiburg, Germany: Haufe.

Schmidt, J., Stürmer, R., & Wohlfart, R. (2012). Aromachology – The scientific side of aromatherapy. *COSSMA, 7-8/2012*, 24-25.

Schmidt, J., & Opwis, M. (2011). Insight inside – Was der Körper verrät. Alternativen zur bildgebenden Hirnforschung. *planung & analyse 06/2011*, 34-37.

(*****Article is included in this doctoral thesis)

Conference contributions (selection)

Schmidt, J., & Martin, A. (2016). Efficacy of neurofeedback vs. mental imagery for subclinical binge eating is differentially affected by impulsivity. *30th Conference of the European Health Psychology Society / British Psychological Society Division Health Psychology,* Aberdeen, Scotland. (Scientific talk)

Schmidt, J., & Martin, A. (2016). Impulsivität und Trait-Food Craving beeinflussen die elektrophysiologische Cue Reaktivität auf Heißhunger-Lebensmittel – Eine Analyse des EEG

Spektrums. *34. Symposium der Fachgruppe Klinische Psychologie und Psychotherapie der DGPs*, Bielefeld, Germany. (Scientific talk)

Schmidt, J., & Martin, A. (2016). Neurofeedback gegen Heißhungeranfälle bei gezügelten Esserinnen – Eine randomisierte kontrollierte Studie mit zwei Kontrollgruppen. *5. Wissenschaftlicher Kongress der Deutschen Gesellschaft für Essstörungen*, Essen, Germany. (Scientific talk)

Schmidt, J. (2016). Der Geist ist willig, aber das Fleisch ist schwach? Psychophysiologische Mechanismen bei dysfunktionaler Essens- und Gewichtsregulation. *15. Kongress der Deutschen Gesellschaft für Verhaltensmedizin und Verhaltensmodifikation*, Mainz, Germany. (Chair)

Schmidt, J., & Martin, A. (2016). Physiologische vs. psychologische Lernmechanismen in einem Neurofeedback-Training gegen Heißhungeranfälle. *15. Kongress der Deutschen Gesellschaft für Verhaltensmedizin und Verhaltensmodifikation*, Mainz, Germany. (Scientific talk)

Schmidt, J., & Martin, A. (2015). Psychophysiologische Analyse zum Sitzungsprotokoll für Neurofeedbacktraining gegen Heißhungeranfälle. *15. Jahrestagung der Deutschen Gesellschaft für Biofeedback e.V. 2015*, Heidelberg, Germany. (Poster)

Schmidt, J., & Martin, A. (2015). Neurofeedback against subclinical binge eating in women: A randomized controlled trial with two control groups. *29th Conference of the European Health Psychology Society*. Limassol, Cyprus. (Scientific talk)

Schmidt, J., & Martin, A. (2015). Neurofeedback gegen Heißhungeranfälle bei gezügelten Esserinnen – Eine randomisierte kontrollierte Studie mit zwei Kontrollgruppen. *33. Symposium der Fachgruppe Klinische Psychologie und Psychotherapie der DGPs*, Dresden, Germany. (E-Poster)

Schmidt, J. (2014). Neurofeedback gegen Heißhungerattacken. *14. Jahrestagung der Deutschen Gesellschaft für Biofeedback e.V. 2014*, Hamburg, Germany. (Scientific talk)

Schmidt, J., & Martin, A. (2014). Neurofeedback-training reduces disinhibited food craving epochs in female restrained eaters. *Psychologie & Gehirn 2014*, Lübeck. (Poster)

Schmidt, J., & Werner, B. (2014). Weniger ist (emotional) mehr: Wie man mit einfachen Reizen den Erfolg von (Marken-)Kommunikation steigern kann. *7. Neuromarketing Kongress*, Munich, Germany. (Scientific talk)

Schmidt, J., Stürmer, R., Theuerzeit, C., Opwis, M., Lambeck, J.-P., Schweitzer, N., & Werner, B. (2013). Empirical validation of psychophysiological patterns related to the "Limbic® Map". *2013 NeuroPsychoEconomics Conference*, Bonn, Germany. (Poster)

Langner, T., **Schmidt, J.,** & Fischer, A. (2012). How deep is your love? A psychophysiological comparison of brand love and interpersonal love. *11th International Conference on Research in Advertising*, Stockholm, Sweden. (Scientific talk)

Schmidt, J., & Stürmer, R. (2012). Psychophysiologische Reaktionen gezügelter Esser auf hoch- und niedrigkalorische Desserts. *Psychologie & Gehirn 2012*, Jena, Germany. (Poster)

Stürmer, R., & **Schmidt, J.** (2012). Product-Experience. Den unbewussten Kaufgründen auf der Spur. *5. Neuromarketing Kongress*, Munich, Germany. (Scientific talk)

Schmidt, J., Opwis, M., & Stürmer, R. (2012). Physio-HeatMaps. Visualizing complex psychophysiological assessment of market research stimuli. *2012 NeuroPsychoEconomics Conference*, Rotterdam, the Netherlands. (Poster)