



PhD-FLSHASE-2015-40  
The Faculty of Language and Literature, Humanities, Arts and Education

## DISSERTATION

Defense held on 18/12/2015 in Luxembourg

to obtain the degree of

DOCTEUR DE L'UNIVERSITÉ DU LUXEMBOURG

EN PSYCHOLOGIE

by

**Zoé van Dyck**

Born on 21 February 1986 in Luxembourg (Luxembourg)

## THE ROLE OF EXTERNAL AND INTERNAL STIMULI IN THE REGULATION OF EATING BEHAVIOUR

### Dissertation defense committee

Prof. Dr. Claus Vögele, dissertation supervisor  
*Professor, University of Luxembourg*

Prof. Dr. Dieter Ferring, Chairman  
*Professor, University of Luxembourg*

Dr. André Schulz, Vice Chairman  
*University of Luxembourg*

Prof. Dr. Simone Munsch  
*Professor, University of Fribourg*

Prof. Dr. Jens Blechert  
*Professor, University of Salzburg*

**Dissertation Defence Committee**

Prof. Dr. Claus Vögele, University of Luxembourg (Supervisor)

Prof. Dr. Dieter Ferring, University of Luxembourg (Chair)

Dr. André Schulz, University of Luxembourg

Prof. Dr. Simone Munsch, University of Fribourg, Switzerland

Prof. Dr. Jens Blechert, University of Salzburg, Austria



Curiosity is the key to problem solving.

(Galileo Galilei, 1564-1642)



## **Acknowledgements**

I would like to thank and acknowledge everyone who helped and supported me and who contributed to my research. You all made my PhD years an exciting, valuable and great experience.

First and foremost I would like to thank my supervisor, Claus Vögele, for all your support and for believing in my work as a researcher. Thank you for trusting in my autonomy, and at the same time always being there when I needed your help, advice, and opinion. I could not have imagined a better way of scientific guidance.

I also highly appreciate the support and advice offered by Jens Blechert. Thank you for your great ideas and suggestions in the planning phase and your valuable contribution and feedback during the writing process. I have learned a lot from you. I owe special thanks to Dieter Ferring; this doctoral project was possible on account of your support and approval. My appreciation also goes to Simone Munsch, for having agreed to be part of my dissertation defence committee and taking up the long journey to Luxembourg. I heartily thank my Scientific Advisors: Beate Herbert for the inspiring discussions and valuable contribution and feedback during the planning and writing of my studies, and André Schulz for the scientific and moral support and important input to this project – I have learned a great deal from you.

I would also like to thank everyone at the INSIDE Research Unit and especially the Self-Regulation and Health team. Apart from the more formal professional interactions, I will mostly remember the time we have spent together informally. A very special thanks to Annika and Silke, who have always been there to support me both professionally and personally. You are not only great colleagues but also wonderful friends. Anne and Claudia, my dear former colleagues: Our time together in Fribourg was short, but a long friendship results from it. You understand like no one else the ups and downs of doing a PhD. Thank you for all your support and the great weekends in Leipzig, Gerlafingen, and Luxembourg. I am already looking

forward to the next one. I also want to thank Alan Murdock and his wonderful wife Elke.

Alan, thank you for proof-reading all of my boring (now you can admit it) manuscripts. If one day you should write a thesis in Luxembourgish, don't hesitate to ask for my help.

A big thank you for all the students who helped me in data collection and supported me managing this mammoth project (Sonja, Jessica, Gillian, Annabel, Marie, Mareike, Elise, Constanze, and many more). This work would not have been possible without your outstanding help and commitment.

I would also like to thank all those who participated in my studies and endured the up to eight hours testing for the sake of research. It was a wonderful experience meeting you all and without you this thesis would not have been possible!

I want to thank my family and friends for their open ears and minds and their patience with me during these work-loaded last four years.

E ganz groussen Merci geet virun allem un meng Elteren, Pia an Marc, fir hier onersetzbar an inconditionnel Ënnerstëtzung mäin Liewen laang. Et ass en wonnerbart Gefill ze wëssen, dass ech ëmmer eng Platz bei iech hunn an heem kommen kann, egal wei fuerchtbar schrecklech grad alles ass. Menger Schwëster, dem Tammy, villmools Merci dass du ëmmer en oppent Ouer fir mech has (an hues) an mech op aner Gedanken bruecht hues, virun allem wann 's de mat engem Owe siessen an enger Fläsch Wäin laanscht komm bass fir mech vum Computer ewech ze zeien. Nanni, Merci fir déi flott Freideg Mëttesstonnen. Ech hoffen, dass mir déi Traditioun nach laang wäerten weider féieren.

Sarah, Géraldine G. an Géraldine B., mir ginn lo schonn säit esou villen Joeren zesummen duerch all Déiften a Héichten an ech kann mech wierklech glécklech schätzen esou Frëndinnen un menger Säit ze hunn. Dir hutt mir virun allem an deenen leschten ustrengenden Wochen vill Kraaft ginn. Ech war sécher net ëmmer einfach ze erdroen, hunn mech seelen

gemellt, ma trotzdem hutt dir mech während der ganzer Zäit bedengungslos ënnerstëtzt. Dir sidd super! Mengem Matbewunner dem Sébastien och e groussen Merci fir déi super Zäit. Dofir, dass du mech moies mat Orangejus versuert hues an ëmmer en oppent Ouer has wann ech mol erëm soueren misst.

Aber der größte Dank gilt Christian, der mich die ganzen Jahre hindurch sowohl privat als auch fachlich unterstützt hat wie kein anderer. Ich weiß, dass es auch für dich nicht immer einfach war und trotzdem warst du immer da wenn ich dich brauchte. Du bist der beste Partner, Freund und Mitarbeiter den man sich vorstellen kann. Danke!



## **Abstract**

Obesity and eating disorders are growing public health problems that affect many individuals world-wide. Research suggests that this obesity epidemic is attributable to our obesogenic environment, where appealing but energy-dense foods are everywhere and anytime available. Yet, within the same environment, there is considerable variation between individuals regarding their susceptibility to overeat and gain weight. It has been argued that a selective attention to food could be an important factor contributing to susceptibility for overeating and binge eating. Another line of research suggests that disordered eating behaviours are characterised by altered responsiveness to internal hunger and satiety signals. The present project was designed to investigate these ‘external’ and ‘internal’ stimuli and their role in regulating eating behaviour. In Study 1, we experimentally investigated the causal role of attentional bias for eating behaviours in a nonclinical sample. Results showed a sustained effect of attentional training on attentional bias and food choice, but not on craving. Moreover, training effects differed according to whether or not participants were aware of the experimental contingencies. In Study 2, we translated and validated a German version of the Intuitive Eating Scale (IES-2) and applied it to a sample of eating disordered women. The German IES-2 yielded good psychometric properties and women with an eating disorder displayed significantly lower intuitive eating scores compared to healthy participants. Study 3 described the development and evaluation of a standardised drink test to measure gastric interoception. Results showed that this two-step Water Load Test (WLT-II) is a simple and reliable test that distinguishes well between sensations of comfortable satiation and unpleasant fullness. Furthermore, the volume of water ingested until satiation was found to be related to bulimic symptoms. In Study 4, the WLT-II was applied to a sample of patients with bulimia nervosa (BN) or binge-eating disorder (BED). Eating disordered patients displayed delayed satiation compared to healthy control participants. In addition, gastric myoelectrical activity

was measured by electrogastrography (EGG) before and after water ingestion. BN and BED patients had increased gastric dysrhythmia and percentage of normogastria was negatively related to frequency of binge eating. We conclude that enhanced attentional processing of food stimuli and a dysfunctional perceptual processing of physiological mechanisms signalling meal termination are key factors that may contribute to overeating or binge eating.

## Contents

1. INTRODUCTION .....	12
1.1. Obesity and binge eating .....	14
1.1.1. Overweight and obesity.....	14
1.1.2. Bulimia Nervosa and Binge-Eating Disorder.....	15
1.2. External stimuli: Food cue reactivity.....	24
1.2.1. The ‘toxic’ food environment .....	24
1.2.2. Food cue reactivity .....	25
1.2.3. Eating and addiction.....	26
1.2.4. Attentional bias for food cues .....	28
1.3. Internal stimuli: Visceroception and the perception of hunger and satiety .....	33
1.3.1. Intuitive eating.....	33
1.3.2. Interoceptive processing.....	36
1.3.3. Interoception and theories of emotion.....	38
1.3.4. Interoception and eating behaviour .....	39
1.3.5. Regulation of hunger and satiety.....	41
1.3.6. Gastric processing and binge eating.....	44
1.3.7. Gastric motility and gastric myoelectrical activity.....	46
2. STUDY AIMS .....	49
3. EMPIRICAL STUDIES.....	51
<b>Chapter 1:</b> Experimental manipulation of attentional bias for food cues: Effects on appetite and the role of contingency awareness .....	51
<b>Chapter 2:</b> German version of the Intuitive Eating Scale: Psychometric evaluation and application to an eating disordered population.....	73
<b>Chapter 3:</b> The Water Load Test as a measure of gastric interoception: Development of a two-stage protocol and application to a healthy population. ....	104

<b>Chapter 4:</b> Gastric interoception and gastric myoelectrical activity in bulimia nervosa and binge-eating disorder. ....	125
4. GENERAL DISCUSSION .....	149
4.1. Summary of findings .....	149
4.1.1. Experimental manipulation of attentional bias for food cues: Effects on appetite and the role of contingency awareness (Study 1) .....	149
4.1.2. German version of the Intuitive Eating Scale: Psychometric evaluation and application to an eating disordered population (Study 2) .....	150
4.1.3. The Water Load Test as a measure of gastric interoception: Development of a two-stage protocol and application to a healthy population. (Study 3) .....	151
4.1.4. Gastric interoception and gastric myoelectrical activity in bulimia nervosa and binge-eating disorder. (Study 4).....	152
4.2. Integration with the existing literature.....	153
4.3. Implications for the treatment of obesity and binge eating .....	159
4.4. Limitations.....	161
4.5. Concluding remarks.....	162
5. REFERENCES .....	164
6. APPENDICES .....	189

## 1. INTRODUCTION

*“My whole life has been about eating and food. When I wake up in the morning, my first thought is about the food I am going to have that day. When something good happens, I reward myself with food. When something bad happens, I comfort myself with food. I wish I could have a normal relationship with food.”*

Participant, 43 years old, ATTEND study

One of the most alarming public health problems of the 21<sup>st</sup> century is the increasing prevalence of overweight and obesity. In the European Union alone, the proportion of the population that is overweight or obese has risen considerably since the 1980s, also in countries with traditionally low rates, resulting in more than half of the EU population being overweight or obese (Eurostat, 2011). This is a matter of grave concern, because obesity is associated with a range of serious health consequences. While it is generally acknowledged that the aetiology of obesity is multifactorial, disordered eating patterns, especially binge eating, are common and associated with body mass index (BMI; Yanovski, 2002). Binge eating is characterised by eating, in a discrete period of time, an amount that is larger than most other people would consume in similar circumstances, and is accompanied by a sense of loss of control over eating (American Psychiatric Association, 2013).

The emergence of an international obesity problem and increasing prevalence of eating disorders have led to a heightened interest in the factors that influence and regulate eating behaviour. To a large extent, the obesity epidemic has been attributed to an energy balance problem, with energy intake exceeding energy expenditure, resulting in weight gain (Epstein, 1995). This excess caloric intake is triggered by ever present and easily available sweet and fatty foods in a society of plenty. Yet, within the same environment, there is considerable variation between individuals regarding their susceptibility to overeat and gain

weight, suggesting that there is individual variability in the intensity of responsiveness to food and the motivation to eat.

The brain has emerged as the key regulator of food intake, receiving and integrating a variety of signals regarding energy status with environmental information, and then translating them into complex eating behaviour. With energy availability being essential to our survival, it is little surprising that we have powerful hunger signals that are aimed at constantly replenishing and building energy reserves. The role for satiation signals that limit food intake is, however, less evident. These signals seem to be comparably weak, especially in the context of our current obesogenic environment where they are not powerful enough to avoid excessive energy intake. Hence, although our understanding of the factors regulating eating behaviour has improved considerably over the last few decades, we are still unable to tackle the growing obesity problem effectively. Therefore, the aim of this thesis was to further investigate the processes underlying eating behaviour characterised by excessive energy intake, with an emphasis on examining how food intake may be regulated by environmental and interoceptive stimuli.

## 1.1. Obesity and binge eating

### 1.1.1. Overweight and obesity

Over the past decades, the prevalence of overweight and obesity has risen considerably. The World Health Organization (WHO) describes obesity as ‘a global epidemic’, with more than 1.4 billion adults worldwide being overweight, half a million of whom are clinically obese (World Health Organization, 2013b). Thus, obesity is a worldwide problem that affects most countries, but is particularly prevalent in Western societies.

Overweight and obesity are most commonly defined by the BMI, which is calculated as body weight in kilograms divided by height in metres squared ( $\text{kg}/\text{m}^2$ ). The WHO has recommended the following classification system based on the BMI:

Underweight	$< 18.5$
Normal weight	$18.5 - 24.9$
Overweight	$25.0 - 29.9$
Obese	$\geq 30.0$

The obese category is further divided into three subcategories, according to the severity of the associated health risk. Class I obesity refers to a BMI ranging between 30.0 and 34.9, associated with moderately increased health risks compared to the normal weight category. Class II obesity refers to a BMI ranging between 35.0 and 39.9, and the associated health risks are severe. Class III obesity refers to a BMI of 40 or higher, and is also named ‘morbid obesity’ because of the associated extremely severe health risks.

It has to be noted, however, that the BMI can lead to an erroneous estimation of body fat due to increased weight associated with lean body mass (i.e., in athletic body types), and that the borderlines are not necessarily valid for all ethnic groups. Other measures of body fat distribution include the waist to hip circumference ratio and skinfold measurements (Björntorp, 2002). Nevertheless, the BMI is the most commonly used measure both in

practice and in research, as it is convenient and readily accessible also in self-report data collection.

In Luxembourg, overweight and obesity rates have risen considerably during the last 20 years. Prevalence estimates from 2008 showed that 60.6% of the adult population (> 20 years old) were overweight and 26.0% were obese (World Health Organization, 2013a). The prevalence was higher among men (overweight: 66.7%; obesity: 26.3%) than among women (overweight: 54.7%; obesity: 25.8%). Similarly, a nationally representative study involving individuals aged 16 years and older found that 55.1% were overweight and 17.7% were obese (Tchicaya & Lorentz, 2010). This is a matter of grave concern, because overweight and obesity are associated with a range of serious health consequences, such as increased risk of type 2 diabetes, cardiovascular disease, and some types of cancer (Mitchell, Catenacci, Wyatt, & Hill, 2011; Visscher & Seidell, 2001). The WHO estimates that 2.8 million people die each year as a direct or indirect consequence of being overweight or obese (World Health Organization, 2013b).

### **1.1.2. Bulimia Nervosa and Binge-Eating Disorder**

Obesity is frequently associated with disordered eating patterns, especially binge eating, which refers to the consumption of large amounts of food in a discrete period of time. Studies have shown that a large number of obese people also report binge eating, and these people appear to be especially prone to further weight gain (Yanovski, 2002). Historically, the first 'bulimic-like' eating behaviours have already been described more than 2000 years ago (Habermas, 2008). Overeating was for a long time looked upon as an aetiologically non-specific symptom, which was observed in a diversity of illnesses, predominantly gastrointestinal disorders (Vandereycken, 2002). It is only since the early 1970s that a discrete cluster of symptoms was identified, distinguishing bulimic eating behaviours from Anorexia



Nervosa (AN) and obesity. The symptom of binge eating was first identified in 1959 (Stunkard, 1959), in conjunction with obesity, but was proposed as an independent diagnosis only in the 1990s (Vandereycken, 2002). Binge eating behaviours, which were by then mostly described in men, started to be considered a rather female problem (Habermas, 2008).

Since eating disorders have only emerged as a serious health problem in the late 1970s, it is probably not surprising that eating disorder research has still not revealed their entire aetiology (Polivy & Herman, 2002). The present thesis will focus on the two officially recognised eating disorders that are characterised by recurrent binge eating episodes: Bulimia Nervosa (BN) and Binge-Eating Disorder (BED). They have to be distinguished from AN, which is characterised by low body weight achieved by strict fasting, exercise or compensatory behaviours, as well as body image disturbances (American Psychiatric Association, 2013).

While BN is marked by the oscillation between uncontrollable binge eating and compensatory behaviour to prevent weight gain (e.g., vomiting), together with an undue influence of shape on self-evaluation, BED entails uncontrollable binge eating in the absence of such compensatory behaviours (American Psychiatric Association, 2013). Although binge eating has been recognised as a distinct eating pattern in obese people as early as 1959, extensive BED research started only in the 1990s (Raymond, Mussell, Mitchell, de Zwaan, & Crosby, 1995). The question of whether BED is a syndrome of clinical significance has been debated since 1994, when BED first appeared in the DSM-IV as a disorder with research criteria requiring further study (American Psychiatric Association, 1994). Nevertheless, research over the past decade has shown that individuals with BED differ meaningfully from individuals without eating disorders and share important similarities to, yet are distinct from, individuals with AN and BN (Wilfley, Wilson, & Agras, 2003).

Binge eating behaviours are both highly prevalent and are found among a wide range of individuals. In a representative adolescent high school sample, 46% of women and 30% of men reported ever having gone on an eating binge in their life (Ross & Ivis, 1999). Similarly, in a study by Saules et al. (2009), 37.2% of college students reported having engaged in binge eating during the past six months. Furthermore, binge eating behaviours are equally prevalent among a large array of racial and ethnic groups, unlike other disordered eating patterns (Johnson, Rohan, & Kirk, 2002). It is important to note that especially BED is strongly related to obesity, and that the prevalence of BED seems to increase with BMI (Dingemans, Bruna, & Van Furth, 2002). The prevalence of BED has been found to be up to 30% among individuals who seek weight control treatment (de Zwaan, 2001). A recent review reported lifetime prevalence rates for BN of 1.5% for women and 0.5% for men. The point prevalence was on average 0.3%, with rates of 0.1 % for men and 0.5% for women (Hudson, Hiripi, Pope, & Kessler, 2007). For BED, the lifetime prevalence was 2% for men and 3.5% for women, whereas the BED twelve-month-prevalence was 1.6% for women and 0.8% for men. Thus, BED is more equally distributed between the sexes than the other eating disorders (de Zwaan, 2001; Dingemans et al., 2002).

### **Diagnostic Criteria**

To meet DSM-5 (American Psychiatric Association, 2013) diagnostic criteria for BN or BED, binge eating (i.e., consumption of unusually large quantities of food accompanied by subjective feelings of loss of control) must occur at least once weekly during three months. In contrast to BN, binge eating in BED occurs in the absence of regular inappropriate purging or non-purging compensatory behaviours (i.e., self-induced vomiting, laxative abuse or excessive exercise). Binge eating episodes in BED are rather accompanied by behavioural features including eating much more rapidly than normal, or eating until feeling

uncomfortably full, in addition to marked distress regarding binge eating (American Psychiatric Association, 2013). As a consequence, the afflicted individuals are often overweight or even obese, although the diagnosis is not limited to overweight individuals, whereas patients with BN are usually of normal weight. As research examining the DSM-IV requirements generated mixed results, necessary changes were made in the DSM-5 (e.g., definition of binge eating, introduction of a ‘severity specifier’; Grilo, Ivezaj, & White, 2015). Other than for BN, the BED diagnosis is lacking a cognitive criterion pertaining to body image. The exact diagnostic criteria for BN and BED in DSM-IV are contrasted with those in DSM-5 in Table 1.

Table 1

*Diagnostic criteria for BN and BED in DSM-IV and DSM-5*

	DSM-IV	DSM-5
Bulimia Nervosa	<p>A. Recurrent episodes of binge eating. An episode of binge eating is characterised by both of the following:</p> <ol style="list-style-type: none"> <li>1. Eating, in a discrete period of time (e.g., within any 2 hour period), an amount of food that is definitely larger than most people would eat during a similar period of time and under similar circumstances.</li> <li>2. A sense of lack of control over eating during the episode (e.g., a feeling that one cannot stop eating or control what or how much one is eating).</li> </ol> <p>B. Recurrent inappropriate compensatory behaviour in order to prevent weight gain, such as self-induced vomiting; misuse of laxatives, diuretics, enemas or other medications; fasting; or excessive exercise.</p> <p>C. The binge eating and inappropriate compensatory behaviours both occur, on average, at least twice a week for three months.</p> <p>D. Self-evaluation is unduly influenced by body shape and weight.</p> <p>E. The disturbance does not occur exclusively during episodes of anorexia nervosa.</p>	<p>A. Recurrent episodes of binge eating. An episode of binge eating is characterized by both of the following:</p> <ol style="list-style-type: none"> <li>1. Eating, in a discrete period of time (e.g., within any 2-hour period), an amount of food that is definitely larger than most people would eat during a similar period of time under similar circumstances.</li> <li>2. A sense of lack of control over eating during the episode (e.g., a feeling that one cannot stop eating or control what or how much one is eating).</li> </ol> <p>B. Recurrent inappropriate compensatory behaviours in order to prevent weight gain, such as self-induced vomiting; misuse of laxatives, diuretics, or other medications, fasting; or excessive exercise.</p> <p>C. The binge eating and inappropriate compensatory behaviours both occur, on average, at least once per week for three months.</p> <p>D. Self-evaluation is unduly influenced by body shape and weight.</p> <p>E. The disturbance does not occur exclusively during episodes of anorexia nervosa.</p>
Subtype	<p><u>Purging Type</u>: During the current episode of bulimia nervosa, the person has regularly engaged in self-induced vomiting or the misuse of laxatives, diuretics or enemas.</p> <p><u>Non-purging Type</u>: During the current episode of bulimia nervosa, the person has used other inappropriate compensatory behaviours, such as fasting or excessive exercise, but has not regularly engaged in self-induced vomiting or the misuse of laxatives, diuretics or enemas.</p>	

	DSM-IV	DSM-5
Binge Eating Disorder	<p>A. Recurrent episodes of binge eating. An episode of binge eating is characterised by both of the following:</p> <ol style="list-style-type: none"> <li>1. Eating, in a discrete period of time (e.g., within any 2-hour period), an amount of food that is definitely larger than most people would eat in a similar period of time under similar circumstances.</li> <li>2. A sense of lack of control over eating during the episode (e.g., a feeling that one cannot stop eating or control what or how much one is eating).</li> </ol> <p>B. The binge eating episodes are associated with three (or more) of the following:</p> <ol style="list-style-type: none"> <li>1. Eating much more rapidly than normal.</li> <li>2. Eating until feeling uncomfortably full.</li> <li>3. Eating large amounts of food when not feeling physically hungry.</li> <li>4. Eating alone because of being embarrassed by how much one is eating.</li> <li>5. Feeling disgusted with oneself, depressed, or very guilty after overeating.</li> </ol> <p>C. Marked distress regarding binge eating is present.</p> <p>D. The binge eating occurs, on average, at least 2 days a week for 6 months.</p> <p>E. The binge eating is not associated with the regular use of inappropriate compensatory behaviours (e.g., purging, fasting, excessive exercise) and does not occur exclusively during the course of anorexia nervosa or bulimia nervosa.</p>	<p>A. Recurrent episodes of binge eating. An episode of binge eating is characterised by both of the following:</p> <ol style="list-style-type: none"> <li>1. Eating, in a discrete period of time (e.g., within any 2-hour period), an amount of food that is definitely larger than most people would eat in a similar period of time under similar circumstances.</li> <li>2. A sense of lack of control over eating during the episode (e.g., a feeling that one cannot stop eating or control what or how much one is eating).</li> </ol> <p>B. The binge eating episodes are associated with three (or more) of the following:</p> <ol style="list-style-type: none"> <li>1. Eating much more rapidly than normal.</li> <li>2. Eating until feeling uncomfortably full.</li> <li>3. Eating large amounts of food when not feeling physically hungry</li> <li>4. Eating alone because of feeling embarrassed by how much one is eating.</li> <li>5. Feeling disgusted with oneself, depressed, or very guilty afterward.</li> </ol> <p>C. Marked distress regarding binge eating is present.</p> <p>D. The binge eating occurs, on average, at least once a week for 3 months.</p> <p>E. The binge eating is not associated with the recurrent use of inappropriate compensatory behaviour as in bulimia nervosa and does not occur exclusively during the course of bulimia nervosa or anorexia nervosa.</p>

## **Course and Outcome**

Generally, there is no easy treatment for eating disorders, and both BN and BED often become chronic disorders. Although spontaneous remission can occur, most individuals continue to have residual features of the disorder, or migrate into other eating disorder diagnostic categories (Sullivan, 2002). About a third of patients continue to meet diagnostic criteria five years and longer after initial treatment (Fairburn, Cooper, Doll, Norman, & O'Connor, 2000; Keel, Mitchell, Miller, Davis, & Crow, 1999). Because BED has only recently been acknowledged as an eating disorder in its own right in the DSM-5, less is known about its long-term outcome (Keel & Herzog, 2004). Overall, the course of eating disorders is far from being completely understood, but the existence of a single pathway is very unlikely (Sullivan, 2002).

Across eating disorder diagnoses, estimates of mortality rates (including suicide) range from 5% to 8%, but these high rates are mostly due to AN (Polivy & Herman, 2002). Smink, van Hoeken, and Hoek (2012) report a mortality rate of 0.2% for BN and 2.9% for BED, unfortunately with only few studies included in the BED sample. The high rate of obese patients with BED makes these finding also very difficult to interpret (Smink et al., 2012). Steinhausen and Weber (2009) report recovery rates of 45% and a mean chronicity rate of 23% for BN. For BED, cognitive behaviour therapy (CBT) or interpersonal psychotherapy (IPT) improves binge eating (i.e., abstinence rates of about 50%). Furthermore, antidepressants and standard weight loss treatments (e.g., bariatric surgery) may also be effective when imposed after eating disorder treatment (de Zwaan, 2001).

Comorbidity is generally very high for all eating disorders, so that 94.5% of individuals with BN and 78.9% of patients with BED meet criteria for at least one additional psychological disorder (e.g., mood, anxiety, impulse-control, substance use; Hudson et al.,

2007). The most common comorbid disorders are major depression, anxiety disorders, and psychoactive substance use disorders (Sullivan, 2002).

### **Aetiological and Maintaining Factors**

It is generally recognised that there are a multitude of risk and resilience factors for eating disorders. Most of these are common for all eating disorders, and only few factors seem to differentiate between BN and BED. Factors specific for the development of BED are rarely found. In the following, we give a brief account of the current evidence on aetiological and risk factors for BN and BED.

BN and BED result most likely from a combination of biological and psychosocial risk factors. Twin and family studies provide evidence for a role of genetic transmission of binge eating behaviour (Hudson et al., 2006; Kendler et al., 1991). Psychosocial risk factors that have been identified in retrospective studies include adverse premorbid experiences (e.g., physical or sexual abuse), family problems (e.g., low parental contact or critical comments about eating, shape, or weight), and psychological vulnerability (e.g., negative self-esteem, shyness, premorbid depression), although their specificity for the development of eating disorders remains unclear (Jacobi, Hayward, de Zwaan, Kraemer, & Agras, 2004). Childhood obesity may also contribute to the development of BN or BED (Fairburn & Harrison, 2003). Importantly, no aetiological differences were found between BN and BED (Hilbert & Tuschen-Caffier, 2010).

The cognitive-behavioural model has identified several maintenance factors. Internalisation of a societal thin ideal and a low self-esteem have been shown to be strongly linked with body dissatisfaction and eating pathology (Thompson & Stice, 2001). Individuals with eating disorders tend to focus on their outer appearance, which may lead to body dissatisfaction, as they perceive themselves as not being able to live up to their unrealistic

body ideals (Stice, 2002). Body dissatisfaction has been shown to promote eating pathology (e.g., dieting), as individuals attempt to restrict food intake to try to achieve an appearance similar to this cultural ideal (Garner, 1991). Minor transgressions of these dietary rules can trigger all-or-nothing reactions, resulting in bulimic eating behaviours (Stice & Shaw, 2002; Stice, 2002). Binge eating episodes can also be provoked by negative affect, and be negatively reinforced through their temporary capacity to neutralise such affective states (Fairburn, 2002). Ensuing negative emotions, such as disgust and self-loathing, further decrease self-esteem and perpetuate the cycle (Fairburn, 2002; Hilbert & Tuschen-Caffier, 2010; Vögele & Gibson, 2010).



## **1.2. External stimuli: Food cue reactivity**

### **1.2.1. The ‘toxic’ food environment**

The increase in prevalence of obesity and overeating over the last few decades as outlined in chapter one of the present thesis has gone hand in hand with environmental changes. Today’s food environment is characterised by an abundance of palatable, energy-dense foods that are anywhere and anytime available, and are promoted in unprecedented ways. Brownell (2002) points out that the yearly marketing budgets of McDonald’s and Coca-Cola are \$1.1 billion and \$866 million, respectively, compared to the \$1 million budget of the U.S. National Cancer Institute to promote healthy eating. And these are only two big food companies. Thus, it is hardly surprising that our environment is rather characterised by advertisements for fast food and soft drinks than by healthy eating campaigns.

Advertising alone, however, is not a direct cause for obesity. The advertised sweet and fatty foods also need to be available, which is the case in our environment. Indeed, high-calorie, palatable foods have never been more varied, cheap, and easy to obtain, and portion sizes have never been larger (Hill & Peters, 1998). One may just think of the proliferation of fast-food restaurants, 24-hour services, or drive-in windows in the past 20 years. This omnipresence of tasty food activates the hedonic appetite system that promotes food intake not due to physiological needs, but due to environmental or emotional reasons (Lowe & Levine, 2005). Yet, despite the potency of the obesogenic environment, a substantial number of individuals manage to maintain a healthy body weight, leading to the assumption that the environment does not have an equal effect on everyone. But what explains these differences in susceptibility to overeat and gain weight? In addition to interindividual differences in metabolism and build, it has been suggested that psychological factors, such as the sensitivity and responsivity to environmental food temptations, may explain some of this variation.

### **1.2.2. Food cue reactivity**

The idea of increased responsiveness to external food-related cues in eating disordered or obese individuals is not new. Already in the 1960's, Schachter observed that the eating behaviour of obese individuals is comparatively unresponsive to internal physiological signals, but rather influenced by salient external food-related stimuli (for a review see Schachter, 1971). This tendency to eat in response to food-related cues (i.e., the sight, smell, or taste of food) was termed 'external eating'. According to Schachter, obesity is the result of a relative abundance of attractive external food cues, which the external eater can hardly resist (Herman, 1974). It has to be noted, however, that normal-weight eaters also have a preference for tasty food. What differentiates obese from normal-weight individuals, according to the externality theory, is that in obese individuals food cue reactivity is identical in states of hunger and satiety, whereas in normal-weight individuals the responsiveness to food cues is enhanced during hunger as compared to satiety (Herman & Polivy, 2008).

During the years following its publication, the externality theory has been applied to different groups of people, not only obese individuals (Herman & Polivy, 2008). It has been suggested that heightened external responsiveness is not confined to obese individuals, but that it is rather related to certain eating behaviours, such as dieting (Nisbett, 1972). In line with this hypothesis, enhanced food cue reactivity has been found in normal-weight restrained eaters as compared to non-restrained individuals (Herman & Mack, 1975). As a result, and also because the concept of obesity is a complex and heterogeneous one (Rodin, 1981), food cue reactivity research started to become more directed towards specific eating behaviours, such as dietary restraint and overeating. Today, externality is rather considered a trait that is not exclusively a characteristic of obese individuals (Nijs & Franken, 2012).

Various theoretical models have suggested that individual differences in food cue responsiveness depend considerably on learning mechanisms (Jansen, 1998; Wardle, 1990;

Weingarten, 1985; Woods, 1991). These models have in common that, through classical conditioning, exposure to food-related cues can trigger cephalic phase responses, that is, anticipatory physiological responses preparing the organism for food intake (e.g., insulin release and increased salivation; Coelho, Jansen, Roefs, & Nederkoorn, 2009). It has further been suggested that individuals with disordered eating behaviours, such as restrained eating or binge eating, display enhanced classically conditioned physiological (i.e., cephalic phase responses) and psychological (i.e., cravings) responses to food-related stimuli (Jansen, 1998). Therefore, food-related cues could act as disinhibiting for individuals with disordered eating behaviours (Coelho et al., 2009).

### **1.2.3. Eating and addiction**

Conditioned cue reactivity models for overeating behaviours have their roots in classical addiction theories, which assume that drug cue reactivity and craving play an important role in the development and maintenance of addictive disorders (Franken, 2003; Robinson & Berridge, 1993). Research has suggested that addictive behaviours and overeating share common mechanisms that reflect imbalances in the responses of the brain to rewarding stimuli (e.g., food or drug stimuli). In the following, this theoretical account will be briefly summarised.

As previously stated, the obesity epidemic has until recently been primarily attributed to an energy balance problem, with energy intake exceeding energy expenditure, resulting in weight gain (Epstein, 1995). Eating when deprived of energy and abstaining from eating when satiated serves as a 'homeostatic' model for the regulation of energy balance (Berthoud, 2011). Most of the work based on this model has focused on the role of circulating hormones on eating behaviour (e.g., leptin and ghrelin), which transmit information on peripheral energy levels to the brain, with the central brain structure being the hypothalamus. Thus,

according to the homeostatic system, food intake occurs when energy stores are depleted and a meal is terminated when the stomach is 'full', that is, when feeling satiated (Benelam, 2009; Berthoud, 2011).

The homeostatic model is, however, not sufficient to explain the observation that some humans override their internal satiety signals and continue to eat despite being satiated. This behaviour is better explained by the brain reward system, which is the second system involved in food motivation (Nijs & Franken, 2012), and consists of a network of mesolimbic and frontal brain regions, as well as the nucleus accumbens (Franken, 2003). Within the brain reward network, communication occurs through neurotransmitters, such as dopamine, which is assumed to be primarily involved in appetite ('wanting') responses to rewarding cues (Berridge, 2009).

It is well known that the reward system is involved in drug addiction. Several addiction theories state that drug-related cue reactivity, and in particular subjective craving, is a central characteristic of addiction, which plays a role in the inability of addicted individuals to stop drug use, and in relapse (Drummond, 2001). One of these theories is the incentive sensitisation theory (Berridge, 2009; Robinson & Berridge, 1993). According to this theory, addictive cues signalling imminent drug consumption acquire motivational properties, or incentive salience, through a conditioning process of repeated association between the cue and consumption of the rewarding substance. As a result of this process, the dopamine-driven brain reward system becomes hypersensitised to the perception and mental representation of these addictive cues, resulting in a strong conditioned dopaminergic response when exposed to them. Reward-related cues become highly salient and 'attention-grabbing', trigger 'wanting' and craving, and ultimately lead to substance acquisition and consumption (Berridge, 2009; Franken, 2003; Robinson & Berridge, 1993). This model has been extended and adapted to explain overeating (Nijs & Franken, 2012), assuming that food cues (such as

the sight or smell of palatable foods) can gain incentive salience through a conditioning process and thereby induce a motivational state of craving. This motivational state can be measured as attentional bias for food-related cues.

#### **1.2.4. Attentional bias for food cues**

Attentional bias (AB), defined as the selective attentional processing of salient stimuli (MacLeod, Mathews, & Tata, 1986), is a central concept in the understanding of overeating and obesity (for a review, see Werthmann, Jansen, & Roefs, 2014). There exists a large variety of experimental paradigms that measure allocation of attention, which makes it difficult to summarise the current state of the art in the field of eating behaviours. There are four different methodologies that have been primarily applied to assess AB for food stimuli: 1) the Stroop task, 2) the dot probe task, 3) eye movement monitoring using eye-tracking, and 4) the monitoring of event-related potentials (ERPs) in the electroencephalogram (EEG). These four methods will be considered in the following, and their understanding is important for the interpretation of results on food-related AB, because study results depend to a large extent on methodological choices.

In the food-modified Stroop task, coloured food-related and non-food related words are presented, and participants are required to report the colour in which each word is printed as fast as possible, while ignoring the meaning of the words. The idea behind this task is that emotionally relevant words interfere more with colour naming than neutral words, leading to longer reaction times in the colour naming of salient words. Thus, if the reaction times in the colour naming of the food-related as compared to the non-food-related words are delayed, this is interpreted as AB for food-related cues. The usefulness of the Stroop task to assess AB is, however, debatable, as it may reflect multiple underlying attentional processes. The colour-naming interference effects for food words could be the result of both heightened attention for

the semantic meaning or avoidance of food-related material (Field & Cox, 2008).

Furthermore, it is not clear if the interference effect reflects early or late components of attention (Phaf & Kan, 2007).

In the visual probe task (MacLeod, Mathews, & Tata, 1986), a food-related stimulus (pictures or words) and a matched control stimulus are simultaneously presented on a computer screen for a fixed duration (typically 2000 ms). When both stimuli disappear, a target stimulus (i.e., visual probe) appears in the position of one of the stimuli. The participant is instructed to respond to the location of the probe by pressing a corresponding key on the keyboard. The logic behind this task is that participants respond faster if their attention was already allocated to the location (and thus the stimulus) where the probe appears. AB is indexed as the difference between reaction times in congruent trials (when the probe replaces the food stimulus) and reaction times in incongruent trials (when the probe replaces the control stimulus). The advantage of this task is that it is possible to distinguish early and late components of attention by modifying the presentation time of the stimuli: A presentation duration of 100 - 500 ms is considered a measure of initial orientation, whereas longer presentation durations (500 ms and above) reflect maintained attention (e.g., Mogg, Bradley, Miles, & Dixon, 2004; Mogg, Bradley, De Bono, & Painter, 1997).

While the Stroop task and the visual probe task are indirect, behavioural measures, more direct assessments of information processing can be obtained by recording electrocortical brain activity or eye movements. Using different EEG-ERP components (e.g., P200, P300 or LPP), it is possible to disentangle early and late stages of information processing, while at the same time measuring the intensity by which attentional resources are allocated to food-related stimuli (Nijs & Franken, 2012; Olofsson, Nordin, Sequeira, & Polich, 2008). Similarly, the recording of eye movements during exposure to food stimuli is a direct measure of visual attention, allowing for the assessment of the time course and the

direction of attention (Bradley, Mogg, Wright, & Field, 2003; Field, Mogg, & Bradley, 2006; Werthmann, Jansen, et al., 2014). Thus, using eye-tracking measures it is possible to determine whether a person focuses on (attentional approach) or ignores (attentional avoidance) disorder-specific stimuli.

AB towards food cues has been reported for AN (e.g., Giel et al., 2011) and BN patients (e.g., Shafran, Lee, Cooper, Palmer, & Fairburn, 2008), and individuals with BED (e.g., Schmitz, Naumann, Trentowska, & Svaldi, 2014; Svaldi, Tuschen-Caffier, Peyk, & Blechert, 2010), although using different methods (for a recent review see Werthmann et al., 2014). Some of these studies found that eating disordered patients exhibited attentional avoidance of food cues (e.g., Giel et al., 2011), whereas others reported enhanced attentional approach towards food stimuli in eating disordered compared to control participants (e.g., Smeets, Roefs, van Furth, & Jansen, 2008). Veenstra and de Jong (2012) even reported attentional avoidance patterns in both eating disordered and healthy control participants. Regarding studies examining food-related ABs in relation to BMI and/or obesity, findings are also mixed. Some studies found enhanced attentional approach bias to food cues in obese individuals compared to healthy-weight participants (Castellanos et al., 2009; Nijs, Muris, Euser, & Franken, 2010), whereas others suggested that a higher BMI was associated with attentional avoidance of food cues, or increased attention towards low-energy food (Graham, Hoover, Ceballos, & Komogortsev, 2011; Nummenmaa, Hietanen, Calvo, & Hyönä, 2011). It has been suggested that these seemingly contradictory findings might be better explained by an approach-avoidance pattern of attentional processing, consisting in an enhanced initial automatic orientation of attention towards food stimuli, followed by a more strategic attentional disengagement from these stimuli (Nijs & Franken, 2012; Werthmann, Jansen, et al., 2014; Werthmann et al., 2011). ABs towards food-related stimuli have also been documented in non-clinical populations at risk for eating disorders, such as restrained eaters

(Long, Hinton, & Gillespie, 1994; Papies, Stroebe, & Aarts, 2008; Tapper, Pothos, Fadardi, & Ziori, 2008) and external eaters (Brignell, Griffiths, Bradley, & Mogg, 2009; Hou et al., 2011; Nijs, Franken, & Muris, 2009).

It should be noted, however, that most of the previously cited studies used a cross-sectional design, making it impossible to draw conclusions about any causal relationships between AB and eating disorder psychopathology. Establishing a causal relationship between AB and disordered eating is difficult and requires the successful experimental manipulation of AB, and the demonstration that the induction of a (temporary) AB in a non-clinical sample results in increased food craving and intake (i.e., increased symptoms of overeating). Using a modified form of the dot probe task (MacLeod, Rutherford, Campbell, Ebsworthy, & Holker, 2002), several authors have attempted to induce AB towards food cues in healthy participants and to assess its effect on subsequent eating behaviour. While some studies found increased AB for and consumption of high-calorie food in the experimental compared to a control group (Kakoschke, Kemps, & Tiggemann, 2014; Kemps, Tiggemann, & Elford, 2014; Kemps, Tiggemann, Orr, & Grear, 2014), other studies reported no effects of cognitive trainings on food-seeking behaviours (Becker, Jostmann, Wiers, & Holland, 2015; Hardman, Rogers, Etchells, Houstoun, & Munafò, 2013). Also, most of these studies compared two active trainings without a no-bias-induction control condition, which makes it impossible to determine whether post-training effects resulted from the AB training or from the control training.

In summary, there is evidence suggesting a link between cognitive processing of food-related stimuli and eating behaviours; however, we cannot yet provide a definite answer on the question whether obese or eating disordered individuals demonstrate enhanced attention towards food stimuli, and to what degree this influences food responsiveness, such as craving or consumption. Additional research is warranted to investigate key issues, such as the



directional or causal relationship between food-related AB and eating behaviours. This knowledge might be important for clarifying the contribution of attentional mechanisms and environmental factors to overeating and obesity and may have implications for the development of intervention and prevention programs.

### **1.3. Internal stimuli: Visceroception and the perception of hunger and satiety**

In addition to enhanced responsiveness to ‘external’ environmental cues, there is evidence that overeating and obesity might be affected by a decreased sensitivity to ‘internal’ physiological signals. The following chapter addresses mechanisms of internal appetite control, starting by discussing intuitive eating, a recently introduced concept that describes eating in response to one’s physiological hunger and satiety signals rather than external and emotional cues (Tribble & Resch, 2003; Tylka, 2006). The second part of this chapter will focus on interoceptive processing and the role of afferent sensory impulses arising from the viscera in the regulation of food intake, with a particular emphasis on gastric processing.

#### **1.3.1. Intuitive eating**

The concept of intuitive eating has been developed with the idea of assessing adaptive eating behaviours, as a counterpart to eating disorder symptomatology that has traditionally been the focus of research (e.g., Tylka & Kroon Van Diest, 2013; Tylka, 2006). In the light of the alarmingly high levels of obesity and disordered eating behaviours in Western societies (e.g., Mintz & Betz, 1988), research on the topic has exploded during the last decades. While this growing literature is promising and encouraging, the vast majority of studies have maintained an almost exclusive pathology-based focus (Avalos & Tylka, 2006). In accordance with the approach of positive psychology (Seligman & Csikszentmihalyi, 2000) that examines adaptive behaviours contributing to and maintaining psychological health, Tylka (2006) has argued that eating disorder treatments and prevention would benefit from maintaining healthy eating behaviours alongside preventing and treating maladaptive eating.

Intuitive eating is defined as the ability to perceive and use internal bodily sensations (i.e., internal physiological hunger and satiety cues) to regulate food intake, combined with a low

preoccupation with food (Tribole & Resch, 1995; Tylka, 2006). Thus, intuitive eaters neither eat for emotional or external reasons, nor do they eat according to restrictive diet plans. Moreover, they are very attentive to their body responses to certain foods and their food choices are based on both health and body functioning, as well as taste (Tylka & Kroon Van Diest, 2013).

Intuitive eating consists of several dimensions, all of them supposedly being closely related (Tribole & Resch, 2003; Tylka & Kroon Van Diest, 2013; Tylka, 2006). The first dimension is termed unconditional permission to eat (UPE) and describes the willingness to eat according to internal physiological hunger signals and the food desired (Tylka, 2006). Individuals high in UPE do not follow dieting rules, nor do they categorise food into ‘bad’ or ‘good’. It has been shown that individuals who try to restrict their food intake tend to overindulge in food given a disinhibiting situation (Herman & Polivy, 1988; Polivy & Herman, 1999). When they perceive that their dietary rules have been broken, that is, when they eat forbidden foods, restrained eaters tend to lose control over their eating, which results in excessive food intake (Herman & Polivy, 1988; Tylka, 2006). Individuals who allow themselves to eat unconditionally are less likely to overeat or feel guilty when eating (Polivy & Herman, 1999).

The second dimension, eating for physical rather than emotional reasons (EPR), refers to the inclination to use food to satisfy physical hunger signals, rather than to regulate (negative) emotions and/or distress (Tribole & Resch, 1995; Tylka, 2006). The relation between emotions and eating behaviours has been addressed by Herman and Polivy (1983) and reviewed by Vögele and Gibson (2010). While a frequent reaction to emotional distress is loss of appetite, individuals who endorse in frequent dieting tend to show disinhibited eating when coping with emotions (Herman, Polivy, Lank, & Heatherton, 1987; Herman & Polivy, 1983), probably in an effort to provide comfort and distraction from adverse emotions (Stice

& Shaw, 2002). In support of these findings, EPR has been shown to be negatively associated with binge eating and food preoccupation (Tylka & Wilcox, 2006).

The third dimension is referred to as reliance on internal hunger and satiety cues to determine when and how much to eat (RHSC) and represents awareness of internal hunger and satiety cues and the tendency to trust these signals to guide food intake (Tribole & Resch, 2003; Tylka, 2006). The premise behind this factor is that everybody is born with an intrinsic knowledge of the quantity and type of food to eat to remain healthy and maintain weight (Gast & Hawks, 1998). With development, however, this innate 'body wisdom' could be thwarted by an environment that imposes external rules for eating, such as diets or eating at dinner time (Gast & Hawks, 1998; Van Dyke & Drinkwater, 2014). Previous findings suggest that individuals who do not eat according to their internal hunger and satiety cues are more likely to experience dietary restraint, weight gain, and emotional eating (Birch & Fisher, 2000; Tylka & Kroon Van Diest, 2013).

Recently, a fourth dimension has been added: Body-food choice congruence (B-FCC) is based on the concept of 'gentle nutrition' as articulated by Tribole and Resch (2003), referring to the tendency to choose foods that honour health and body functioning, while at the same time eating palatable foods (Tylka & Kroon Van Diest, 2013).

Intuitive eating is typically assessed via self-report questionnaires, the most recent, well-evaluated and widely used measure being the second version of the Intuitive Eating Scale (IES-2; Tylka & Kroon Van Diest, 2013). Using the IES-2, Tylka and Wilcox (2006) were able to show that intuitive eating is not only the absence of eating disorder symptomatology, but also contains unique constructs not captured in the disordered eating continuum framework, which provides justification for exploring potential contributors to this construct. In other words, eating intuitively is not synonymous with low levels of eating disorder symptomatology.

In support of the adaptive properties of intuitive eating, cross-sectional research has found intuitive eating to be associated with lower BMI (Denny, Loth, Eisenberg, & Neumark-Sztainer, 2013; Gast, Campbell Nielson, Hunt, & Leiker, 2014; Herbert, Blechert, Hautzinger, Matthias, & Herbert, 2013; Tylka & Kroon Van Diest, 2013; Tylka & Wilcox, 2006), lower triglyceride levels and cardiovascular risk (Hawks, Madanat, Hawks, & Harris, 2005), reduced eating disorder symptomatology (Denny et al., 2013; Tylka & Kroon Van Diest, 2013; Tylka, 2006), and increased self-esteem and reduced negative affect (Tylka & Wilcox, 2006). Furthermore, interventions based on intuitive eating principles have been shown to have a positive impact on psychological health outcomes and eating behaviours, such as body image, self-esteem, interoceptive awareness, and anti-dieting attitudes, while decreasing dietary restraint, drive for thinness, depressive symptoms, and disinhibited eating (Bacon, Stern, Van Loan, & Keim, 2005; Cole & Horacek, 2010; Hawks, Madanat, Smith, & De La Cruz, 2008). At a metabolic level, there is evidence that non-diet interventions decrease blood lipids, and systolic blood pressure (Bacon et al., 2002).

### **1.3.2. Interoceptive processing**

As presented in the previous chapter, a crucial key feature of intuitive eating assumes that it is primarily based on one's internal hunger and satiety signals rather than environmental cues, as well as on the ability to clearly recognise the physiological signs of hunger and fullness. Thus, individuals who eat intuitively consume food in response to hunger and stop eating when satiated (Tylka, 2006). This links the concept of intuitive eating with the concept of 'interoception', defined as the body-to-brain axis of sensation concerning stimuli generated from within the body, including the viscera (Cameron, 2002). The term 'viscera' refers to the internal organs inside the thoracic, abdominal, and pelvic body cavities. In its broader sense, interoception refers to the ability of visceral afferent information to directly or

indirectly influence behaviour, irrespective of conscious awareness (Cameron, 2002). It has to be distinguished from exteroception (i.e., the perception of the external environment) and proprioception (i.e., limb position; Sherrington, 1948). A more inclusive definition has been suggested by Craig (2002), who argued that the concept of interoception should include the sense of the current physiological state of the entire body, not just the viscera.

Viscerosensitive signals are encoded by mechanoreceptors, thermoreceptors, chemoreceptors, and nociceptors, and are transferred via sympathetic and parasympathetic fibres to brainstem and thalamic areas (Craig, 2002; Vaitl, 1996). These afferents converge to 'interoceptive centres' in the insular and orbitofrontal cortices and give rise to interpretation and conscious awareness of the internal state. Concomitant activation of the anterior cingulate cortex adds a motivational component that generates a behavioural response. According to Craig (2002), this interoceptive processing is necessary for the body to maintain homeostasis, that is, to respond to interior and exterior stimuli in a way that maintains optimal physiological conditions in the body. Conscious perception of the afferent signals is not required for homeostatic functions, and only occurs when the information reaches cortical areas (Craig, 2002). A major role in the conscious perception of visceral signals seems to be played by the right anterior insula (Craig, 2002; Critchley, Wiens, Rotshtein, Ohman, & Dolan, 2004; Pollatos, Kirsch, & Schandry, 2005), which has been put forward as the basis of re-representation and integration of the interoceptive state of the body with higher order and emotional processes (Craig, 2002). In this context, Pennebaker's (1982) 'competition of cues' model indicates that exteroceptive and interoceptive stimuli compete for the organism's limited capacity of cognitive information processing, with preference being given to the more salient cue. As an increased focus on external food-related cues has been demonstrated in eating disordered patients, this theory is in support of the idea of a blunted processing of internal body signals in eating disorders.

### **1.3.3. Interoception and theories of emotion**

It is generally acknowledged that cognitive and physiological processes influence each other, wherefore it is not surprising that the perception of signals arising from the body plays an important role in many theories of emotions (i.e., ‘peripheral theories of emotion’). Experiencing changes in bodily states may influence our thoughts, feelings and behaviours. The three most popular and influential theories dealing with the interaction of perceived arousal and emotions are the ones by James (1884), Schachter & Singer (1962), and Damasio (1994, 1996, 1999).

William James suggested that emotions are related to physiological responses, stating that ‘bodily changes follow directly the perception of the exciting fact, and that our feelings of the same changes as they occur IS the emotion’ (James, 1884, p. 189f.). His theory is often called the James-Lange theory, because Carl Lange introduced a similar theory in 1887 (Lange, 1887). The James-Lange theory further argues that different bodily states accompany different subjective emotions and that individuals differ in their perception of bodily sensations and, thus, also differ regarding their emotional experience and the emotion’s intensity. Cannon (1927) disagreed by arguing that the same emotion may occur in various bodily states, and that a general physiological arousal would be too unspecific to account for the richness and variety of emotional experiences. Schachter and Singer (1962) proposed a resolution, as they also believed in the primacy of arousal. They argued, however, that changes in the body must be interpreted cognitively to determine which emotion is experienced. The theory was supported by their laboratory experiment, in which participants who were physiologically aroused by adrenaline reported experiencing more irritation or happiness, depending on the emotion of a confederate in the room.

Nonetheless, the underlying unidimensional model of bodily arousal cannot explain the variety of organ-specific responses regulated by the autonomic nervous system and within

the last decades the notion that emotions have a peripheral origin has been reinvigorated by Damasio and colleagues and their ‘somatic marker hypothesis’ (Damasio, 1994, 1996, 1999). The hypothesis claims that when individuals make decisions, they must judge the incentive value of the available choices using both cognitive and emotional processes. Confronted with complex and conflicting choices, individuals may be unable to decide using only cognitive processes. In these cases, so-called somatic markers can help to decide, as they bias one’s cognitive processing. Located in the ventromedial prefrontal cortex, these markers induce physiological affective states. Hence, because both emotion and cognition are subjected to bodily biases (Damasio, 1994, 1999), a comprehensive understanding of neural, cognitive and affective processes, with the appropriate methods and techniques (e.g., functional brain imaging), must further investigate the integration of mental and bodily processes.

#### **1.3.4. Interoception and eating behaviour**

Several lines of research have suggested that interoceptive processes that are supposed to maintain homeostasis are dysfunctional in disordered eating behaviours, particularly with respect to the regulation of food intake and energy balance. Bruch (1962) was the first to describe deficits in the perception of hunger and satiety in eating disordered patients. A few years later, Garfinkel, Moldofsky, Garner, Stancer, and Coscina (1978) referred to these deficits as ‘disturbances in interoception’. More recently, interoceptive deficits have primarily been investigated in relation to, or by proxy of, awareness of emotional states. A fundamental role has been played by the Eating Disorder Inventory (EDI; Garner, Olmsted, & Polivy, 1983), a self-report questionnaire that has become a standard tool in the field of eating behaviours.

The EDI assesses the specific psychopathology of eating disorders, such as drive for thinness, bulimia, body dissatisfaction, and interoceptive awareness, the latter of which refers



to the ability to accurately recognise and respond to emotional states (Garner et al., 1983). A few items also measure uncertainty in the identification of specific visceral sensations relating to hunger and satiety, therefore not differentiating between emotional and appetite awareness (Brown, Smith, & Craighead, 2010; Pollatos et al., 2008).

A substantial body of research has provided evidence that interoceptive awareness, as measured by the EDI, is dysfunctional in people with disordered eating behaviours, ranging from severe dietary restriction to uncontrollable overeating (Fassino, Pierò, Gramaglia, & Abbate-Daga, 2004; Garner et al., 1983; Lilenfeld, Wonderlich, Riso, Crosby, & Mitchell, 2006; Matsumoto et al., 2006). Thus, poor interoceptive awareness seems to be a core psychopathological element which might play a role in the onset and maintenance of eating disorders (Fassino et al., 2004; Pollatos et al., 2008). Matsumoto et al. (2006) further found that interoceptive awareness deficits improved after treatment. Using self-report instruments, however, it is not possible to determine whether these interoceptive deficits in eating disordered populations are due to detection abnormalities or reflect disturbances in higher order processes.

Recently, more objective measures have been used to investigate interoceptive processes, most of which have focussed on people's ability to consciously perceive their own heartbeats (Brener & Kluitse, 1988; Critchley et al., 2004; Schandry, 1981; Whitehead, Drescher, Heiman, & Blackwell, 1977). The predominant focus on cardiac accuracy is largely due to pragmatic reasons: Heartbeats are distinct and frequent internal events that can be easily discriminated and measured (Garfinkel, Seth, Barrett, Suzuki, & Critchley, 2015). These so-called heartbeat perception tasks are well validated measures, as evidenced by relationships between cardiac perception accuracy and activation in brain structures responsible for the mapping of internal bodily responses, in particular the right anterior insula, and the somatomotor and cingulate cortices (Critchley et al., 2004).

Using heartbeat detection tasks, it has been suggested that variations in the ability to accurately perceive body signals could be an important contributing factor to the onset and maintenance of psychopathology, including anxiety (Domschke, Stevens, Pfleiderer, & Gerlach, 2010; Ehlers & Breuer, 1996), depression (Dunn, Dalgleish, Ogilvie, & Lawrence, 2007; Terhaar, Viola, Bär, & Debener, 2012), and somatoform disorders (Pollatos et al., 2011). In the field of eating disorders, however, studies using heartbeat detection tasks have yielded inconsistent results. Attenuated interoceptive accuracy compared to healthy controls has been reported in AN patients (Pollatos et al., 2008) and in women recovered from BN (Klabunde et al., 2013). Both studies used the mental tracking task, which requires participants to count their own heartbeats during specific time intervals (Schandry, 1981). In another study, Eshkeviri, Rieger, Musiat, and Treasure (2014) assessed interoceptive sensitivity in patients with current AN or BN using a version of the heartbeat perception task where a series of tones are presented to the participant, who is required to judge whether his or her heartbeats were simultaneous with the tones. The authors found no differences between eating disordered and control participants. Another study with AN patients even reported an enhanced cortical representation of afferent signals from the cardiovascular system, as indicated by increased heartbeat evoked potentials in the EEG (Lutz, Schulz, Voderholzer, Koch, & Vögele, 2015). The same study found that AN patients performed slightly better in the mental tracking task than control participants. Hence, findings from studies using heartbeat perception tasks with eating disordered patients provide inconclusive results and, hence, no definite conclusion can be drawn.

### **1.3.5. Regulation of hunger and satiety**

As outlined above, interoceptive processes have frequently been investigated in relation to, or by proxy of, perception of bodily states related to emotional experience. In

eating disorders, however, an important interoceptive deficit concerns the accurate detection and interpretation of physiological hunger and satiety signals. Satiety is commonly defined as ‘sufficiency’, and as ‘full gratification of appetite or thirst, with abolition of the desire to eat or drink’ (Dorland, 2000). Two components of satiety can be distinguished, namely short-term satiety and long-term satiety. Short-term satiety (referred to as ‘satiation’ in the following) has been defined as ‘the process that leads to the termination of eating, which may be accompanied by a feeling of satisfaction’ (Benelam, 2009, p. 129). Long-term satiety (referred to as ‘satiety’ in the following) designates ‘the feeling of fullness that persists after eating, suppressing further energy intake until hunger returns’ (Benelam, 2009, p. 129). Over the course of a day, people typically have several eating episodes, including meals, drinks, and snacks. According to previous definitions, satiation controls the amount of food consumed at, and the duration of, each single eating occasion, while satiety regulates the duration of the periods between eating episodes (i.e., inter-meal intervals). In more simple terms, satiation controls meal termination and satiety determines meal initiation (De Graaf, Blom, Smeets, Stafleu, & Hendriks, 2004). Different peripheral physiological mechanisms play a role in satiety and satiation. The most well-known and widely studied ones will be briefly described in the following.

The gastrointestinal tract plays a key role in feelings of satiation. Powley and Phillips (2004) differentiate between gastric and intestinal satiation, suggesting that gastric satiation is of mechanical (volumetric) nature, whereas intestinal satiation is of chemical (nutritional) nature. When food enters the stomach, gastric stretch receptors and mechanoreceptors are activated and transmit signals via vagal afferent fibres to the brain (Hellström et al., 2004). Vagal involvement in satiation has been confirmed by van de Wall, Pomp, Strubbe, Scheurink, and Koolhaas (2005), who found that capsaicin-induced vagal deafferentiation resulted in overconsumption of sucrose in rats. Findings from several short-term intake

studies indicate that gastric distention (i.e., meal volume), rather than the energy content, promotes satiation (e.g., Phillips & Powley, 2000; Poppitt & Prentice, 1996). The role of stomach distention in satiation and meal termination has also been demonstrated by a series of studies of Geliebter and colleagues (Geliebter, 1988; Geliebter et al., 1992; Geliebter, Schachter, Lohmann-Walter, Feldman, & Hashim, 1996).

Intestinal satiation, on the other hand, is rather sensitive to the nutrients ingested. From the stomach, food is released into the small intestine, where the gut hormones cholecystokinin (CCK) and glucagon-like peptide-1 (GLP-1) are released into the blood. CCK and GLP-1 manage the transit of nutrients from the stomach into the gut, acting on pyloric pressure, stomach motility, and stomach relaxation, which cause a delay in gastric emptying and a subsequent increase in gastric distention (De Graaf et al., 2004; Flint, Raben, Ersbøll, Holst, & Astrup, 2001). Thus, CCK and GLP-1 have an important role in the causal chain leading to satiation or meal termination (De Graaf et al., 2004).

Long-term satiety is mostly regulated by insulin and leptin, which are also implicated in the long-term regulation of energy balance (i.e., the balance of energy intake and expenditure, so that a relatively constant bodyweight is maintained; Schwartz, Woods, Porte, Seeley, & Baskin, 2000). Insulin is produced by the pancreas and secreted in the blood in response to increases in blood glucose concentrations. In healthy subjects it controls blood glucose levels by stimulating its uptake by peripheral tissues. Leptin is a peptide hormone, synthesised mainly by adipose tissue and providing information on the availability of body fat stores to the hypothalamus. Consequently, circulating leptin levels are proportional to fat mass and BMI, and are decreased by weight loss (Benelam, 2009). However, the amount of leptin produced at a given percentage of body fat varies between individuals (Maffei et al., 1995). Interestingly, the leptin receptor is expressed in the pancreatic  $\beta$  cells that produce insulin, suggesting that leptin may also affect insulin production (Kieffer & Habener, 2000).

Ghrelin also contributes to satiety, although it acts as a meal initiator, stimulating appetite rather than satiety (Benelam, 2009). Ghrelin is primarily produced in the fundus of the stomach (Ariyasu et al., 2001) and it is the first and only peripherally secreted hormone with confirmed orexigenic properties. Thus, ghrelin stimulates appetite, probably by inhibiting activity of vagal afferents (Berthoud, 2008). Plasma ghrelin concentrations decline quickly after a meal and return to preprandial concentrations before the next meal is initiated (Cummings et al., 2001). Importantly, although ghrelin is mainly synthesised in the stomach, it is not affected by stomach distention, as suggested by findings showing that non-caloric water intake does not influence ghrelin concentrations (Shiia et al., 2002). Other hormones involved in satiety are the PP fold peptides peptide YY (PYY) and pancreatic polypeptide (PP). They are both produced in the gut and have been found to reduce food intake in humans (Batterham, Cohen, et al., 2003; Batterham, Le Roux, et al., 2003).

### **1.3.6. Gastric processing and binge eating**

As described in the previous chapter, food intake is influenced by multiple internal feedback signals, including distention of the stomach and gastrointestinal peptide hormones that inhibit feeding and promote meal termination. Reduced sensitivity to these signals could contribute to obesity and binge eating (Wang et al., 2008). A central characteristic of BN and BED is the ingestion of binge meals that are larger than what most people would eat under similar circumstances (American Psychiatric Association, 2013). Numerous studies have shown that patients experiencing binge eating eat larger meals than healthy subjects (e.g., Kissileff, Walsh, Kral, & Cassidy, 1986) and don't stop eating when satiated (Woolley et al., 2007), suggesting that they encounter difficulties in meal termination.

As previously stated, one of the key satiation mechanisms is stomach distention during food intake. Stomach distention is typically investigated using gastric balloons and barostats,

which are medical devices used to give some pressure or maintain constant pressure in a closed chamber. For the assessment of sensory thresholds, the pressure inside the balloon is increased progressively and the individual sensory perceptions are recorded (Andresen, 2009). Several studies have examined brain activation during gastric distention using  $^{15}\text{O}$ -water positron emission tomography (PET; Ladabaum et al., 2001; Stephan et al., 2003; Vandenberg et al., 2005) or functional magnetic resonance imaging (fMRI) with blood oxygenation level-dependent (BOLD) contrast (Ladabaum, Roberts, & McGonigle, 2007; Lu et al., 2004; Wang et al., 2008). They found that gastric distention activated the anterior and posterior insula, sensorimotor cortices, and left amygdala. Thus, these brain regions seem to play a role in processing interoceptive gastric stimuli, which could influence the volumes of food consumed in a given meal.

Gastric distention studies in eating disordered patients have found that participants with BN have larger stomach capacities compared to healthy controls and obese individuals (Geliebter & Hashim, 2001; Geliebter et al., 1992). Similar results were reported for BED patients (Geliebter, Yahav, Gluck, & Hashim, 2004). In addition, meal-induced gastric relaxation was diminished in patients with BN (Walsh, Zimmerli, Devlin, Guss, & Kissileff, 2003). Gastric relaxation refers to the reduction in gastric tone in response to meal ingestion, thereby facilitating the ingestion of larger volumes (Kim et al., 2001). In healthy individuals, this reflex is prominent in the proximal stomach and requires intact vagal pathways (Coulie, Tack, Sifrim, Andrioli, & Janssens, 1999). BN patients further display substantial delay in gastric emptying of a meal into the duodenum (Devlin et al., 1997; Geliebter et al., 1992). Both BN and BED patients commonly report a variety of upper gastrointestinal symptoms, such as early satiety, bloating, nausea, and upper abdominal discomfort (Cremonini et al., 2009; Koch, Bingaman, Tan, & Stern, 1998). Taken together, these abnormalities in gastric

function may give rise to important medical complications and could play a role in the perpetuation of compulsive overeating.

Gastric processing may also be investigated by measuring gastric motor activity. Stomach motility not only regulates the rates at which nutrients are being processed, but also contributes to the development of satiation. As such, it is intimately linked to the gastric functions and sensations described above. A non-invasive measure to evaluate gastric motor activity is the EGG, which will be introduced in the next chapter.

### **1.3.7. Gastric motility and gastric myoelectrical activity**

The functions of the stomach in the digestive phase consist of receiving ingested foodstuffs, mixing them with secretions of acid and enzymes, mechanically breaking down the contents to smaller particles and subsequently emptying the chyme to the small intestine (Stern, Koch, Levine, & Muth, 2007). The stomach can be divided into three major regions: the fundus, the body, and the antrum. The gastric fundus receives the food and must relax to accommodate the particular ingested volume, while at the same time maintaining a continuous contractile tone. After ingestion, foods are moved from the gastric fundus into the gastric body and antrum for mixing and emptying (Stern et al., 2007). Healthy individuals experience pleasant, satisfying sensations in the epigastrium after meals, that is, when the stomach is performing the muscular work of gastric emptying (Koch, 2001). In contrast, many eating disordered patients experience upper gastrointestinal symptoms after a meal, such as nausea, abdominal discomfort, or bloating (Cremonini et al., 2009; Diamanti et al., 2003; Koch et al., 1998).

Gastric peristaltic contractions of the body/antrum are the basis for emptying of solids from the stomach (Camilleri, Malagelada, Brown, Becker, & Zinsmeister, 1985). These events start in the gastric body and move down into the antrum where the waves dissipate in

the prepyloric region (Stern et al., 2007). As a result of these contractions, the contents of the stomach are pushed toward the pyloric sphincter and into the duodenum (Meyer, 1987). The postprandial motility pattern and gastric emptying rate depend to a large extent on the physical state and nutrient content of the ingested food. For example, hyperosmolar and fatty foods result in a delayed gastric emptying and longer lag phase (Koch, 2001). The electrical events that control gastric contractions are the gastric slow waves that originate from a gastric pacemaker area in the region of the juncture of the fundus and body. From this region, the slow wave moves circumferentially and distally towards the pylorus (Koch & Stern, 2004). Slow waves regulate the frequency and propagation of gastric contractions, and their normal frequency in humans is 3 cycles per minute (cpm). In the postprandial condition, the slow waves are linked to action potentials (the spike potentials), which evoke contractions of the antral muscles. Gastric slow waves and spike potentials are the myoelectrical components of gastric contractile activity that may be non-invasively recorded from electrodes positioned on the skin – the EGG (Brown, Smallwood, Duthie, & Stoddard, 1975; Hamilton, Bellahsene, Reichelderfer, Webster, & Bass, 1986; Koch & Stern, 2004).

There are only few studies, which have investigated gastric myoelectrical activity in eating disordered patients. Ogawa et al. (2004) recorded the EGG from 36 patients with eating disorders before and after water ingestion. They reported a significantly smaller percentage of normal 3 cpm gastric activity and increased 1 - 2.4 cpm bradygastria in eating disordered compared to healthy control participants, both before and after water ingestion. When comparing different eating disorder diagnoses (i.e., AN vs BN vs eating disorder not otherwise specified), they found no differences in terms of EGG spectrum distribution. Interestingly, the degree of EGG abnormality before water ingestion was positively related to the (self-reported) duration of the eating disorder. Similarly, Diamanti et al. (2003) found that adolescents with BN displayed less normal gastric electrical activity and more bradygastria,



together with delayed gastric emptying time. In their study, however, EGG activity did not differ between patients with AN and healthy controls. Altered gastric myoelectrical activity in BN was also reported by Koch et al. (1998). No study has yet investigated gastric myoelectrical activity in BED patients.

## 2. STUDY AIMS

This thesis consists of four experimental chapters. The literature summarised above suggests that, although it is not clear if overeating and obesity are linked to attentional approach or avoidance of food-related cues, there is evidence for an altered attentional processing of external food stimuli. Yet, it is still an open question as to whether AB plays a causal role for overeating and obesity. Therefore, Study 1 experimentally investigates the directional relationship between AB for food cues and eating behaviours in a nonclinical sample.

Studies 2-4 investigate the role of perceiving and relying on internal bodily signals in disordered eating behaviours. As outlined in chapter 1.3., different measures have been used to investigate interoceptive processing and, consequently, studies yield inconsistent results. First of all, a distinction has to be made between subjective and objective measures. Self-reported interoceptive processes have been mostly assessed using the interoceptive awareness subscale of the EDI. This measure, however, confounds emotional and appetite awareness, which have been demonstrated to play separate roles in the development and maintenance of disordered eating behaviour (Brown et al., 2010; Trenary, Craighead, & Hill, 2005). In the present thesis, the focus will be put on the perception of visceral sensations relating to hunger and satiety, while largely neglecting emotional states, which have been the focus of many previous studies on interoception in the eating domain. As an alternative to the pathology-focussed EDI, we will use the IES-2, which is a positively framed instrument investigating the ability to perceive and eat in response to physiological hunger and satiety signals. Until now, however, most studies on intuitive eating have been conducted with nonclinical, North American female college students. Accordingly, the IES-2 is not yet available for other cultures and in different languages. Therefore, the first aim of Study 2 is to develop and validate a German version of the IES-2. In a second step, intuitive eating will be assessed in

participants with an eating disorder diagnosis and different types of eating disorders will be compared.

Although self-report measures are of interest and necessary to gauge individual differences in the subjectively perceived tendency to rely on physiological signals to regulate food intake, they can be biased and do not inherently address whether this subjective interoceptive sensibility reflects objective interoceptive processing. Therefore, Study 3 of the present thesis aims at developing a more objective measure to investigate sensitivity to internal body signals. Specifically, because most previous studies have focused on cardiac accuracy, thereby only partially capturing a core aspect of interoceptive deficits in eating disorders, that is, an impaired perception of hunger and satiety cues, the primary aim of study 3 is to develop a measure investigating interoceptive processing in the gastric system. Hence, the objective of Study 3 is to establish a noninvasive and standardised test to measure gastric interoception by loading the stomach with water. Building on results from Study 3, Study 4 aims at examining gastric interoception in patients with binge eating behaviours, that is, BN and BED. Furthermore, because gastric motor activity participates in the development of satiation, gastric myoelectrical activity will be assessed before and after ingestion of non-caloric water.

### **3. EMPIRICAL STUDIES**

#### **Chapter 1**

#### **Experimental manipulation of attentional bias for food cues: Effects on appetite and the role of contingency awareness**

*Submitted as:*

van Dyck, Z., Blechert, J., Reiter, S., & Vögele, C.

*Experimental manipulation of attentional bias for food cues: Effects on appetite and the role of contingency awareness.* Manuscript in revision.

## **Abstract**

Theoretical models ascribe a causal role to attentional bias (AB) for food cues in the maintenance of overeating and obesity. Yet, current research provides only preliminary empirical support for this hypothetical causal link, mostly by experimentally manipulating specific food-related biases. Unresolved issues addressed in the current study concern the role of procedural (contingency awareness) and temporal (duration of effects) aspects of attentional bias modification (ABM), and the breadth of affected behaviors. Forty-six healthy women were randomly allocated to either an ABM group, trained to attend toward food, or sham training (control group). Three to four days after the four-session ABM training, AB, food craving, food choice, and perceived self-regulatory success were assessed. Participants in the ABM group showed an increased AB for food cues, chose more high-calorie (HC) food items compared to the no-bias-induction control group, and demonstrated a negative association between dieting intention and subsequent self-regulatory success. Moreover, contingency awareness influenced the effects of ABM on AB and craving. The results show sustained effects of ABM on AB and food-seeking behavior and thus support the notion of a causal role of AB in eating behavior. They further demonstrate the importance of contingency awareness in ABM protocols.

*Keywords:* Attentional Bias Modification; Food Cues; Contingency Awareness; Overeating; Self-Regulation.

## INTRODUCTION

One of the most alarming public health problems of the 21st century is the increasing prevalence of overweight and obesity (Eurostat, 2011). Research suggests that this obesity epidemic may be attributed to our obesogenic environment, where appealing but energy-dense foods are anywhere and anytime available (e.g., Swinburn et al., 2011). Yet, within the same environment, there is considerable variation between individuals regarding their susceptibility to overeat and gain weight, which raises the important question of why this is the case.

In addition to interindividual differences in metabolism and build, psychological factors such as the (in-)capacity to resist the temptation of tasty food may explain some of this variation. The increased susceptibility to succumb to food temptations has been shown to play a role in eating disorders and overweight/obesity (Ouweland & Papies, 2010; Stice, Spoor, Ng, & Zald, 2010) and is related to altered cognitive processing of food stimuli (Berridge, 2009; Rolls, 2011). In eating behaviors, an extensively researched cognitive process concerns attentional bias (AB; for a recent review see Werthmann et al., 2014), referring to the selective attentional processing of salient (i.e., disorder-related) stimuli (MacLeod et al., 1986). A marked AB toward food has been demonstrated for eating disordered or obese individuals (Castellanos et al., 2009; Nijs & Franken, 2012; Shafran, Lee, Cooper, Palmer, & Fairburn, 2007; Werthmann et al., 2011), and has been documented in nonclinical populations at risk for eating disorders, such as restrained eaters (Long et al., 1994; Papies et al., 2008; Tapper et al., 2008) and external eaters (Brignell et al., 2009; Hou et al., 2011; Nijs et al., 2009). Similar results have been reported for short-term food-deprived healthy participants (Castellanos et al., 2009; Mogg, Bradley, Hyare, & Lee, 1998; Nijs & Franken, 2012). Despite its obvious relevance, actual food consumption has been a neglected dependent variable in some of this research. Two studies reported positive correlations between AB for high-calorie (HC) snack foods and subsequent food intake (Nijs et al., 2010; Werthmann et

al., 2011). In a similar vein, Calitri, Pothos, Tapper, Brunstrom, and Rogers (2010) found that AB for unhealthy foods predicted an increase in body mass index (BMI) over a 1-year period.

Although the link between AB to food cues and unhealthy eating behaviors now stands beyond contention, none of these studies supports firm conclusions concerning the causal relationship between AB and eating disorder psychopathology. Causality inference requires the successful experimental manipulation of AB, and that the demonstration that the induction of a temporary AB in a nonclinical sample results in increased food craving and intake (i.e., increased symptoms of overeating). To this end, the dot probe paradigm, which has initially been developed to assess AB, was transformed into a “training paradigm”, where the probe consistently replaces either the disorder-relevant or the neutral stimulus, thereby modifying attention towards or away from these cues (MacLeod et al., 2002). Attentional bias modification (ABM) has primarily been applied in the areas of anxiety (Amir, Beard, Burns, & Bomyea, 2009; Eldar et al., 2012) and substance abuse (Attwood, O’Sullivan, Leonards, Mackintosh, & Munafò, 2008; Field & Eastwood, 2005), with medium to large effects (Beard, Sawyer, & Hofmann, 2012). ABM trainings have further been found to modify eye tracking-measured gaze patterns (Wadlinger & Isaacowitz, 2008), and to influence prefrontal cortical activity to emotional stimuli, suggesting that improved top down attentional control of attention underlies the observed training effects (Browning, Holmes, Murphy, Goodwin, & Harmer, 2010).

Recently, ABM has been adapted for the food context. Three studies conducted with undergraduate female students report increased AB for and consumption of HC food in participants trained to attend towards food cues compared to participants trained to avoid food stimuli (Kakoschke et al., 2014; Kemps, Tiggemann, & Elford, 2014; Kemps, Tiggemann, Orr, et al., 2014). Similarly, using a food approach avoidance task, Brockmeyer, Hahn, Reetz, Schmidt, and Friederich (2015) found moderate to large effects on cognitive biases, food

craving, and eating disorder symptoms. Other studies, however, reported no effects of cognitive trainings on food-seeking behaviors (Becker et al., 2015; Hardman et al., 2013). First results from studies with obese participants suggest that the experimental manipulation of AB for food may influence subsequent food intake in children (Boutelle, Kuckertz, Carlson, & Amir, 2014) and that effects on attention generalize to an independent measure of biased information processing (Kemps, Tiggemann, & Hollitt, 2014).

Together, these studies suggest that changes in AB for food cues can lead to changes in eating behavior. Nevertheless, findings are inconsistent and several issues need further examination. *First*, most of these studies lacked a no-bias-induction control condition, making it impossible to determine whether posttraining group differences resulted from increased food intake in participants who were trained to attend to food stimuli, from decreased intake in participants whose attention was modified away from food cues, or from a combination of both.

*Second*, many of the previous studies focused on specific food items, such as chocolate or cake. It would be important to investigate whether these findings also apply to other HC food stimuli and, most importantly, if modifying attention to some food types generalizes to other food items. Such generalization effects would be important if ABM is to have any real-world application, as individuals need to be able to withstand multiple types of unwanted foods in various situations, not just the particular ones learned during training.

*Third*, ABM effects seem to largely depend on the type of dependent variable chosen. Meta-analyses show that ABM has a reliable effect on AB and laboratory challenge tasks (e.g., a taste test to measure food intake), but only small effects have been reported for motivational states (e.g., craving; Beard et al., 2012). To further investigate this issue, the present study included both food choice (postchallenge task) and food craving as dependent variables. Furthermore, with the aim to include a real-life challenge, we measured self-



perceived self-regulatory success after the training in participants with an intention to diet. The latter is important, as previous studies have solely investigated effects of ABM on eating attitudes and behaviors in laboratory settings, lacking information on participants' eating behavior in their natural environment.

*Last*, we also measured participants' awareness of the experimental contingencies during attentional training (i.e., the relation between picture type and probe location) and examined its role on training effects. Findings from previous studies concerning this issue are mixed. Two ABM studies on alcohol and cigarette cravings found training effects to be restricted to participants who became aware of the experimental contingencies (Attwood et al., 2008; Field et al., 2007). In contrast, some studies modifying food-related AB found that awareness did not impact training effects (Kakoschke et al., 2014; Kemps, Tiggemann, & Hollitt, 2014).

In summary, we predicted that: (1) ABM training would result in changes in AB between groups, such that participants trained towards food-related stimuli (ABM group) would show an increased AB for HC foods at posttest compared to control participants; (2) ABM training would result in increased subjective craving and more unhealthy food choices compared to the sham training condition; (3) the impact of dieting intention on self-regulatory success would be moderated by the type of training received; (4) contingency awareness would influence the effects of ABM on AB at posttest in the ABM group.

## **METHOD**

### **Participants**

A total of 46 participants were recruited from students and staff at the University of Luxembourg. To preclude possible gender effects on AB and food intake, participation was restricted to women (Rolls, Fedoroff, & Guthrie, 1991). Participants were required to be aged between 18 and 40 years and to have normal or corrected-to-normal vision. Exclusion criteria

were current or past mental disorder, and current physical disease affecting diet or weight (i.e., diabetes or hyperthyreosis). Participants were randomly allocated to one of the two attentional training conditions: trained towards food (ABM group) or sham training (control group). Since bias for food is influenced by participants' hunger levels (Mogg et al., 1998), we tried to restrict the impact of this variable by instructing participants to refrain from eating for at least three hours prior to the sessions where AB for food cues was measured (i.e., at pretest and posttest). All participants reported having complied with this instruction. The study received ethical approval from the University's ethics review panel. Participant characteristics for both groups are presented in Table 1.

### **Stimuli**

Stimuli consisted of 96 digital colored photographs from the food-pics database ([www.food-pics.sbg.ac.at](http://www.food-pics.sbg.ac.at)), providing metadata on calorie content, subjective palatability, and physical features (Blechert, Meule, Busch, & Ohla, 2014). Forty-eight pictures of HC foods were individually paired with 48 pictures of neutral stationery and household items (e.g., a stapler, a watering can) and the images were matched for perceptual characteristics (brightness, contrast, and complexity). All pictures were 136 mm high and 169 mm wide. They were presented on a 22-inch monitor with a resolution of 600 x 450 pixels and at a viewing distance of 60 cm.

Four subsets of pictures were created: Set 1 consisted of 32 image pairs that were used to measure baseline AB at pretest. For the purposes of training, these 32 picture pairs were split into two subsets (set 2 and set 3), with 16 pairs per subset. At posttest, 16 novel image pairs were added to the subset that was not used during the training (i.e., subset 2 or 3) to create subset 4, which was used to measure AB at posttest. That way we ensured that different picture pairs were used during training and posttest. Additionally, eight neutral picture pairs (with no food-related content) were developed for use in task practice trials. Stimulus

presentation was controlled through Eprime software (Eprime 2.0; Psychology Software Tools Inc., Pittsburgh, PA, USA).

## **Questionnaires**

Self-report questionnaires comprised the Dutch Eating Behavior Questionnaire (DEBQ; van Strien, Frijters, Bergers, & Defares, 1986; German version by Grunert, 1989), the Attentional Control Scale (ACQ; Derryberry & Reed, 2002), and the “intense desire to eat”-subscale of the Food Craving Questionnaire-State (FCQ-S; Cepeda-Benito, Gleaves, Williams, & Erath, 2000; German version by Meule, Lutz, Vögele, & Kübler, 2012). Following a procedure by Kroese, Adriaanse, Evers, and De Ridder (2011), participants’ intention to diet was assessed at pretest using four questions: “I [am] determined/intend/want/expect to diet the next period of time” (Cronbach’s  $\alpha = .97$ ) that were answered on an 8-point Likert scale from 1 (*strongly disagree*) to 7 (*strongly agree*). They were further asked to determine a personal temptation, which was described as “an unhealthy snack that you really like but that you would rather eat less of” (i.e., an attractive food that in conflict with the dieting goal; Kroese et al., 2011). After the training sessions (at posttest), three items were administered to assess perceived self-regulatory success since the last training session, which constituted one of the dependent variables: “Compared to the week before you participated in the experiment, (1) how often did you succeed in resisting (personal temptation) during the last three days; (2) how well did you succeed in resisting (personal temptation) during the last three days; and (3) how often did you give in to (personal temptation) during the last three days [reverse coded]” (Cronbach’s  $\alpha = .92$ ). Items were answered on a scale ranging from 1 (*never/not at all*) to 7 (*very often/ very well*).

## **Procedure**

According to previous ABM protocols (Field & Eastwood, 2005; Kemps, Tiggemann, Orr, et al., 2014), the modified dot probe procedure used in the present study consisted of

three phases: (a) a pretraining baseline assessment of participants' AB for HC food cues (pretest); (b) a training phase in which half of the participants were trained to attend to HC food cues, whereas the other half of the participants received a sham training; (c) a posttraining assessment of participants' AB for HC food cues similar to the pretest (posttest). Participants were trained and tested individually and sessions took place in a quiet room of the Clinical Psychophysiology Laboratory.

### *Pretest*

Upon arrival, participants were informed about the study and signed informed consent. Then they rated their momentary craving using the "intense desire to eat"-subscale of the FCQ-S. After providing general demographic information, they completed the DEBQ and the ACQ.

Participants then completed a standard dot probe task, the purpose of which was to measure, not manipulate, AB for food cues. Each trial began with a fixation cross that was displayed in the middle of the screen for 500ms, followed by the presentation of a picture pair for 500ms. The pictures were presented on the left and on the right side of the screen, with 60mm apart. After picture offset, a visual probe consisting of an arrow pointing up or down appeared in the location of one of the preceding pictures and remained there until participants pressed the corresponding button on the computer keyboard ("↑" or "↓"). The premise behind the traditional dot probe task is that the latency to respond to the probe provides an index of attention at image offset. A subject is faster to respond to a probe if it replaces the picture he or she was attending to previously. There was an intertrial interval of 500 ms before the onset of the next trial. Participants were instructed to look at the fixation cross at the start of each trial and respond to the visual probe as quickly and accurately as possible.

The task started with eight practice trials, followed by two buffer trials and 126 critical trials. Each of the 32 critical image pairs was presented four times, with each food picture

presented twice on each side. Probes replaced food and neutral images with equal frequency, and there was an equal number of probes of each type. All pairs were presented in random order for each participant. After the dot probe task, participants rated their intention to diet during the next period of time and listed a personal temptation.

### *Training*

Results from meta-analyses revealed that ABM studies including multiple training sessions yield larger and perhaps more reliable effects than single session ABM trainings (Beard et al., 2012; Hakamata et al., 2010). Hence, participants from both groups received four training sessions over a 2-week period. Following Schoenmakers et al. (2010), training sessions were scheduled twice per week, if feasible on the same time of the day. To keep expectations equal between groups, all participants were told that the training aimed at improving attention control (attentional shifting and attentional focusing), with the goal to decrease RTs and increase accuracy in the course of the training sessions.

Attentional training consisted of a modified dot probe task, in which 16 critical picture pairs were presented 16 times, resulting in a total of 256 trials for each session. For the ABM group, probes replaced the food-related picture on 100% of trials and never replaced neutral pictures, thus training attention toward food cues. For the control group, probes replaced food and neutral pictures with equal frequency (50/50).

In accordance with the procedure by Schoenmakers et al. (2010), all participants were given positive feedback on their training performance (i.e., mean reaction time (RT) and percentage of correct responses) after the ABM or control training, and new goals were set for performance in the next session. Providing participants with immediate feedback in order to improve their performance has been found to increase the effectiveness of ABM interventions (Fadardi & Cox, 2009; Schoenmakers et al., 2010; Wiers et al., 2006) The duration of one training session was approximately 15 minutes.

### *Posttest*

Both groups attended the posttest 3-4 days after the last ABM or control training session (Schoenmakers et al., 2010). Participants again started by rating their momentary craving using the FCQ-S subscale “desire.” They then completed a standard dot probe task that was similar to the pretest in that two buffer trials were followed by 126 critical trials and probes replaced food-related and neutral images with equal frequency. Practice trials were omitted. Upon completion of the final dot probe task, participants responded to the three items assessing perceived self-regulatory success during the past week.

Participants were then offered a range of different snacks (tangerines, chocolate bars, and crisps) as an additional “reward for their achievement.” They were allowed to pick as many snacks as they wanted and the number of chosen healthy/unhealthy items was noted unbeknownst to the participants. Finally, participants were thanked and an oral funneled debriefing method as recommended by Bargh and Chartrand (2000) was used to test whether participants from the ABM group were aware of the aim of the study and the manipulation (i.e., that arrows always replaced food-related pictures). All participants received 30 € as a compensation for their participation.

### **Data reduction**

Consistent with previous studies (e.g., Shafran, Lee, Cooper, Palmer, & Fairburn, 2007), RT data from incorrect trials were discarded, as were outliers defined as extreme RTs ( $< 200$  ms or  $> 2000$  ms), or RTs that were more than 2 standard deviations above or below each participant’s own mean. Pre- and posttest AB scores were then calculated by subtracting the mean latency to respond to probes replacing food-related images (congruent trials) from the mean latency to respond to probes replacing control images (incongruent trials). Positive values indicate AB towards food-associated stimuli (i.e., faster RTs to food pictures compared to neutral pictures) and negative values a bias away from food pictures.

## RESULTS

### Group characteristics

A series of independent samples *t* tests showed no statistically significant group differences in demographics, eating behaviors, attentional control, or baseline hunger (all *p*'s > .05; see Table 1).

Table 1

*Sample characteristics for participants in the ABM group and control group, respectively.*

	ABM group ( <i>n</i> = 23)		Control group ( <i>n</i> = 23)		<i>t</i> (44)	<i>p</i>
	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>		
Age	22.65	3.02	24.30	5.70	1.23	.23
BMI	22.29	2.55	23.98	3.84	1.76	.09
SES	3.35	0.78	3.54	0.98	0.75	.46
Hunger at baseline	1.65	1.97	1.96	1.74	0.56	.58
Restrained eating	28.57	8.06	30.61	8.33	0.85	.40
Emotional eating	23.43	9.59	23.96	10.10	0.18	.86
External Eating	32.57	6.57	33.30	6.88	0.37	.71
Attentional focusing	2.67	0.36	2.57	0.39	-0.96	.34
Attentional shifting	2.78	0.37	2.57	0.42	-1.82	.08

*Note.* BMI = Body mass index; SES = Socioeconomic status.

### **Attentional bias**

To assess whether the ABM training successfully modified AB for HC food stimuli, a Time (pretest vs posttest) x Group (ABM vs control) mixed-design analysis of variance (ANOVA) was conducted on AB scores. Results indicated significant main effects of time,  $F(1,44) = 8.36, p = .006, \eta_p^2 = .16$  and group,  $F(1,44) = 5.99, p = .019, \eta_p^2 = .12$ , which were qualified by a significant Time x Group interaction,  $F(1,44) = 8.61, p = .005, \eta_p^2 = .17$ .

Follow-up independent samples *t* tests indicated that there was no difference in AB between the two groups at pretest,  $t(44) = 0.50, p = .620$ , but at posttest the ABM group had a significantly larger AB than the control group,  $t(44) = -3.05, p = .004, d = 0.92$ . Additional paired samples *t* tests performed separately on each group revealed that AB scores significantly increased from pretest to posttest in the ABM group,  $t(22) = -3.40, p = .003, d = 0.71$ , but not in the control group,  $t(22) = 0.04, p = .966$ . Pretest and posttest AB scores in the two groups are shown in Table 2.

### **Craving**

To assess whether the ABM training had an effect on subjective food craving, another Time (pretest vs posttest) x Group (ABM vs control) mixed-design ANOVA was conducted. There were no significant main effects for group,  $F(1,44) = 1.97, p = .167$ , or time,  $F(1,44) = 1.31, p = .259$ , and there was no interaction between time and group,  $F(1,44) = 0.02, p = .878$ . Pretest and posttest subjective craving scores in the two groups are shown in Table 2.

### **Behavioral choice**

Since the number of unhealthy food items chosen was highly skewed, group differences at posttest were analyzed using a nonparametric Mann-Whitney *U* test for independent samples. As can be seen in Table 2, individuals from the ABM group chose significantly more unhealthy food items than individuals in the control group,  $U = 158.50, p = .012, d = 0.79$ .



Table 2

*Pretest and posttest means (SD) for multiple outcome measures in ABM and control groups.*

	ABM group ( <i>n</i> = 23)		Control group ( <i>n</i> = 23)	
	Pretest	Posttest	Pretest	Posttest
AB score	2.21 (9.79)	22.81 (28.78)	3.32 (13.31)	3.17 (9.48)
Craving	2.87 (1.20)	3.10 (1.28)	3.20 (1.27)	3.51 (1.01)
Number of food items	-	2.17 (2.06)	-	0.96 (0.71)

### **Self-regulatory success**

To explore whether the ABM training moderated the effect of intention to diet on self-regulatory success, we excluded participants who indicated no intention to diet (i.e., participants with a mean value of 1 on the intention to diet scale; *n* = 6), as it is meaningless to analyze self-regulatory success in individuals who do not intend to diet. A regression analysis was conducted with self-regulatory success as the dependent variable and intention to diet, group, and their interaction term as predictors. There was no significant effect of intention to diet ( $p = .798$ ) or group ( $p = .381$ ), but there was a significant interaction effect of intention to diet and group,  $\beta = -.46$ ,  $p = .005$ . Simple slopes analyses revealed that for the control group, the effect of intention to diet on self-regulatory success did not reach significance ( $p = .083$ ). For the ABM group, however, the effect of intention to diet on self-regulatory success was significant ( $p = .015$ ). The interaction is depicted in Fig. 1, showing that in the ABM group, self-regulatory success significantly decreased as a function of intention to diet.

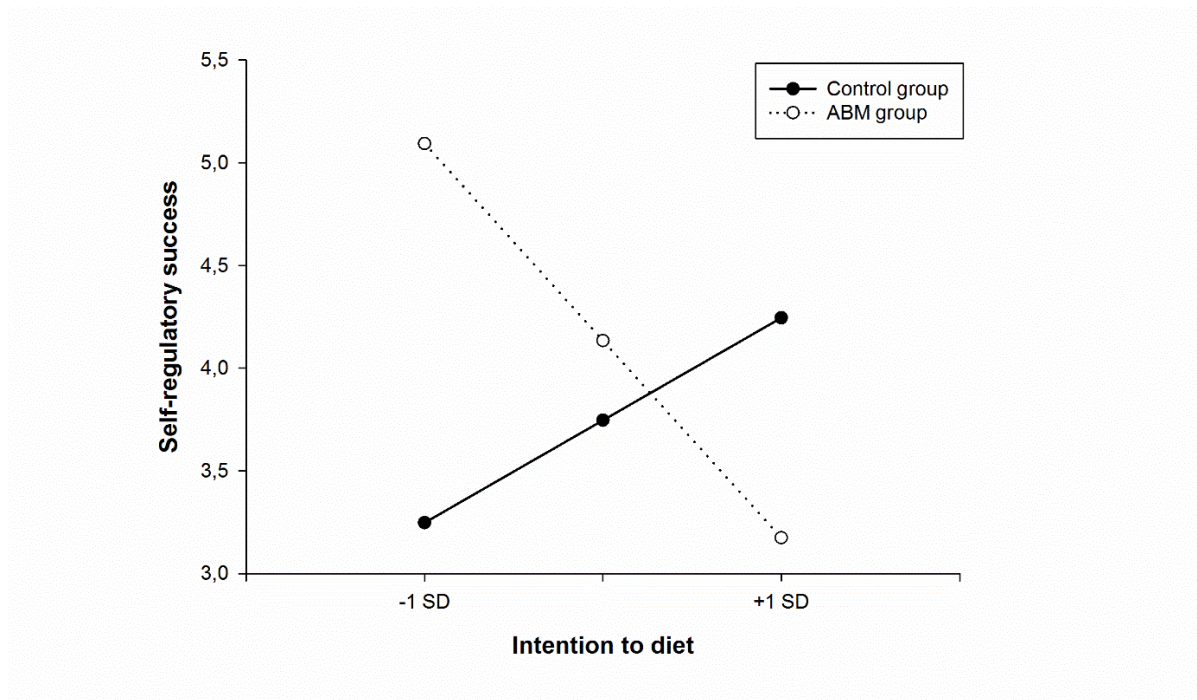


Figure 1. Self-regulatory success as a function of high (+1SD) and low (-1SD) intention to diet for the ABM group and the control group, respectively.

### Awareness of experimental contingencies

As there were no experimental contingencies to be aware of in the control condition, the analysis of contingency awareness was limited to the ABM group. Seven participants (30.4%) from the ABM group correctly recalled or recognized the relationship between the type of pictures and the location of the probes during the training phase. The other 16 participants (69.6%) were not aware of the experimental contingencies. To examine the effect of contingency awareness on AB scores, subjective craving, and behavioral choice, the previous analyses were repeated with awareness (aware, unaware) as between-subjects factor.

### Attentional bias and contingency awareness

A Time (pretest vs posttest) x Awareness (aware vs unaware) mixed-design ANOVA was conducted on AB scores in the ABM group. Results indicated a significant main effect of time,  $F(1,21) = 17.35, p = .000, \eta_p^2 = .45$ , confirming the efficacy of the training. More

interestingly, the Time x Awareness interaction approached significance,  $F(1,21) = 4.29, p = .051, \eta_p^2 = .17$ . Independent samples  $t$  tests showed no difference in AB between the aware group and the unaware group at pretest,  $t(21) = 0.76, p = .457$ , whereas at posttest there was a trend towards higher AB in participants who were aware of the experimental contingencies compared to the unaware group,  $t(21) = -1.77, p = .091, d = 0.69$ . Additional paired samples  $t$  tests performed separately on aware and unaware participants revealed that AB scores significantly increased from pretest to posttest in the aware group,  $t(6) = -2.49, p = .047, d = 0.94$  and in the unaware group,  $t(15) = -2.69, p = .017, d = 0.67$ . Altogether, these results indicate that training effects were present in both groups, but that the increase in AB scores was more pronounced in aware participants. Pretest and posttest AB scores in the aware and the unaware group are shown in Table 3.

#### *Craving and contingency awareness*

To assess whether contingency awareness had an effect on subjective food craving, again, a Time (pretest vs posttest) x Awareness (aware vs unaware) mixed-design ANOVA was conducted. There were no significant main effects of time,  $F(1,21) = 0.41, p = .528$ , or awareness,  $F(1,21) = 0.33, p = .573$ , but there was a significant interaction between time and awareness,  $F(1,21) = 13.92, p = .001, \eta_p^2 = .40$ . As can be seen in Table 3, pretest subjective craving scores were higher in aware compared to unaware participants, but this difference was not significant,  $t(21) = -1.52, p = .143$ . At posttest, unaware participants had significantly higher subjective craving scores than the aware group,  $t(21) = 2.51, p = .020, d = 1.13$ . Subsequent paired samples  $t$  tests performed separately on aware and unaware participants revealed a significant decrease of subjective craving scores from pretest to posttest in the aware group,  $t(6) = 3.02, p = .023, d = 1.15$ , and a significant increase of scores in the unaware group,  $t(15) = -2.67, p = .017, d = 0.66$ .

### *Behavioral choice and contingency awareness*

The nonparametric Mann-Whitney  $U$  test for independent samples revealed no effect of awareness on the number of unhealthy food items chosen after the ABM training,  $U = 49.50, p = .654$ .

Table 3

*Pretest and posttest means (SD) for multiple outcomes in the aware group and the unaware group, respectively.*

	Aware group ( $n = 7$ )		Unaware group ( $n = 16$ )	
	Pretest	Posttest	Pretest	Posttest
AB score	-0.15 (8.13)	38.14 (40.82)	3.24 (10.50)	16.07 (19.76)
Craving	3.43 (1.05)	2.19 (1.18)	2.63 (1.21)	3.50 (1.14)
Number of food items	-	2.86 (2.97)	-	1.88 (0.39)

## **DISCUSSION**

The present study experimentally investigated the causal role of AB for eating behaviors in a nonclinical sample. Consistent with recent studies (Kakoschke et al., 2014; Kemps, Tiggemann, & Elford, 2014; Kemps, Tiggemann, Orr, et al., 2014), results show a successful induction of an AB for food cues using a modified dot probe paradigm. After four sessions of attentional training, participants in the ABM group showed an increase in AB from baseline, whereas there were no changes in the control group. Importantly, these effects were found three to four days after the last training session, indicating that ABM produces changes in AB for food cues that may be maintained beyond the duration of the training protocol. Up to now, there is only one study that has examined sustained effects of food-related ABM. Kemps, Tiggemann, and Elford (2014) found that effects of multiple attentional

training sessions on AB for chocolate cues were maintained at one-week follow-up. However, they compared two active trainings, thus lacking a no-training control group and making it impossible to determine which condition contributed to these effects. In the present study, the magnitude of AB for food stimuli increased in the ABM group and did not change in the control group. Furthermore, results suggest that sustained effects of ABM are not limited to the specific chocolate stimuli and, importantly, that these effects generalize to novel, previously unseen food items. Such generalization is important, as individuals are likely to encounter a multitude of novel food cues in an obesogenic environment, not just the particular ones used during training.

While the attentional training resulted in appropriate changes in AB, it did not affect food craving. This finding is in line with several other studies that have found no effects of ABM on chocolate craving (Werthmann, Field, Roefs, Nederkoorn, & Jansen, 2014) and craving for snack foods (Boutelle et al., 2014). Interestingly, Kemps, Tiggemann, Orr, et al. (2014) found a significant effect of ABM on chocolate craving in one of their two studies. These inconsistencies are in line with the mixed reports of training effects on craving for alcohol and tobacco and the weak effects of ABM on craving reported in a recent meta-analysis (Beard et al., 2012). In the present study, the time that elapsed between the last training session and the posttest, when craving was measured, may have contributed to these null findings. Future research should also assess subjective craving immediately after training.

Nevertheless, ABM clearly affected behavioral food choice. Three to four days after the last training session, participants in the ABM group picked more HC food items than those in the control group. These results add to recent findings showing that ABM affects food intake (Boutelle et al., 2014; Kakoschke et al., 2014; Kemps, Tiggemann, & Elford, 2014; Kemps, Tiggemann, Orr, et al., 2014; Werthmann, Field, et al., 2014) and they are consistent with cognitive-motivational models of craving and consumption, suggesting that

AB plays a causal role for eating behavior (Franken, 2003; Ryan, 2002). While previous studies measured food intake directly, the current study focused on food choice. Two previous studies have used food choice as a dependent variable, yet with somewhat different methods. Using an approach avoidance task, Becker and colleagues (2015) found that the training had an effect on snack choices in successfully trained participants only. In their study, participants who were successfully trained to avoid unhealthy foods more often chose the healthy snack option compared to those having received sham training. In contrast to the present results, Smith and Rieger (2009) found that, after a body image challenge, participants trained to attend to HC food words more frequently chose the low-caloric over the high-caloric cookies compared to control participants. In accordance with Werthmann et al. (2014), it could be argued that these divergences are due to the body image challenge that might have activated dieting or restraint associations, thereby leading to more calorie-conscious subsequent food decisions (Anschutz, Van Strien, & Engels, 2008; Papies & Hamstra, 2010).

To our knowledge, this is the first study to investigate the effects of ABM training on self-regulatory success outside the laboratory. Results revealed that in the ABM group, intention to diet negatively predicted self-regulatory success, indicating that the experimental induction of AB for food cues may lead to counterintentional effects in dieters. In contrast, diet intention was positively related to self-regulatory success in the control group, although this correlation failed to reach significance. In future studies, it would be important to examine the effects of ABM on a broader range of outcome measures from outside the laboratory. Moreover, in the present study self-regulatory success was assessed via self-report. It might be interesting for future research to use more objective behavioral measures.

Interestingly, and unlike previous studies from the food domain (Kakoschke et al., 2014; Kemps, Tiggemann, & Hollitt, 2014; Kemps, Tiggemann, Orr, et al., 2014), training effects on AB and craving differed according to whether or not participants from the ABM

group were aware of the experimental contingencies. These discrepancies may be explained by methodological differences, such as the number of training sessions (single session vs multiple sessions), the type of stimuli used during posttest dot probe task (same stimuli than during training vs novel stimuli), or the assessment method for contingency awareness. In previous studies, awareness was typically measured using an open-ended recall question, followed by a multiple-choice recognition question (i.e., Field & Eastwood, 2005). In the present study, a funneled debriefing procedure with questions with open-answer format was used (Bargh & Chartrand, 2000), in which participants were asked increasingly specific questions to explore their awareness of the experimental contingency. The omission of multiple-choice questions could thus be responsible for these discrepancies and may also be the reason why in the present study fewer individuals were considered aware of the experimental contingency compared to previous studies. Indeed, previous studies stated that more than half of their participants were aware of the manipulation, compared to about a third of the participants in the present study. Thus, a noteworthy avenue for future research would be to determine the most appropriate method to assess contingency awareness in ABM studies. Also, as cell sizes in the present study were small, findings on contingency awareness should be considered preliminary and need further replication.

Unlike food-related studies, two ABM studies on alcohol and cigarettes have reported a role of contingency awareness on training effects, and in both studies it was found that training effects on AB and craving were restricted to aware participants (Attwood et al., 2008; Field et al., 2007). The present results show a significant increase in AB in both groups, even though it was stronger in aware than in unaware participants. Aware participants knew that they had to direct their attention towards food stimuli in order to reduce reaction times and most likely used this knowledge to improve their task performance. Thus, individuals who noticed the “rule” predicting probe location during the training phase may have continued

using this rule during posttest measurement of AB, thereby decreasing their RTs in congruent trials. However, unlike in previous studies, we found that subjective craving scores decreased in participants who were aware of the experimental contingency, whereas an increase was observed in the unaware group. This discrepancy may have occurred because participants who were aware that their attention was being manipulated during training may have been even more motivated to prevent appetitive reactions towards palatable food items. Awareness of the manipulation may have triggered an “alarm bell” response so as to maintain self-control and not succumb to manipulation. Thus, aware participants might have registered the aim of the intervention (to influence food craving and intake), resulting in activation of defensive mechanisms to counter the threat (i.e., palatable foods). If so, more indirect measures of the outcome variables might be necessary, such as automatic or neural/peripheral responses during food picture viewing. Finally, the fact that craving, but not subsequent food choice, was affected by contingency awareness further supports the idea that craving and substance-seeking behavior can be decoupled under certain conditions (Tiffany, 1990).

As in all studies, this study has both strengths and limitations that need to be acknowledged. Strengths include the multiple-session protocol and examination of sustained effects (3-4 days after training), the inclusion of a sham-training control group, the variety of outcome variables, and the use of many different food stimuli during training. Limitations include the comparatively small and exclusively female sample size and the laboratory environment in which training and testing took place. Another important point is that, unlike most ABM studies in the food domain, we did not include a laboratory taste test. When conducting taste tests with student samples in preliminary trials, many of our participants reported awareness that their food intake was being measured. This might be problematic, as individuals who are aware that the amount of food they eat is being monitored are likely to restrict food intake (Robinson, Kersbergen, Brunstrom, & Field, 2014). Nevertheless, further



research is warranted to examine effects of ABM on both food choice and the amount of food consumed, and to extend research to the natural environment.

In conclusion, the present study adds to a growing body of research on the causal relationship between AB and eating behaviors. We could demonstrate that food-related AB can be modified and that this modification affects food-seeking behaviors 3-4 days after the training. Importantly, these maintained training effects generalized to novel food-related stimuli, and group differences were found using a no-bias-induction group. The current study also provides first evidence for effects of ABM on eating behaviors outside the laboratory. Furthermore, the present study suggests that ABM training effects are influenced by participants' awareness of the experimental contingencies. Further research with larger sample sizes and including both women and men is warranted to more closely examine the influence of contingency awareness on ABM training effects. Additional data along these lines might ultimately justify the addition of attentional training procedures to dietary interventions in healthy and eating disordered populations.

## **Chapter 2**

### **German version of the Intuitive Eating Scale: Psychometric evaluation and application to an eating disordered population**

*Submitted as:*

van Dyck, Z., Herbert, B.M., Happ, C., Kleveman, G.V., & Vögele, C.

*German version of the Intuitive Eating Scale: Psychometric evaluation and application to an eating disordered population. Manuscript in revision.*

## **Abstract**

Intuitive eating has been described to represent an adaptive eating behaviour that is characterised by eating in response to physiological hunger and satiety cues, rather than situational and emotional stimuli. The Intuitive Eating Scale-2 (IES-2) has been developed to measure such attitudes and behaviours on four subscales: unconditional permission to eat (UPE), eating for physical rather than emotional reasons (EPR), reliance on internal hunger and satiety cues (RHSC), and body-food choice congruence (B-FCC). The present study aimed at validating the psychometric properties of the German translations of the IES-2 in a large German-speaking sample. A second objective was to assess levels of intuitive eating in participants with an eating disorder diagnosis. The proposed factor structure of the IES-2 could be confirmed for the German translation of the questionnaire. The total score and most subscale scores were negatively related to eating disorder symptomatology, problems in appetite and emotional awareness, body dissatisfaction, focus on outer appearance, and self-objectification. Women with eating disorders had significantly lower values on all IES-2 subscale scores and the total score than healthy female participants. Women with a binge-eating disorder (BED) diagnosis had higher scores on the UPE subscale compared to participants with anorexia nervosa (AN) or bulimia nervosa (BN) and those diagnosed with AN had higher scores on the EPR subscale than individuals with BN or BED. We conclude that the German IES-2 constitutes a useful self-report instrument for the assessment of intuitive eating in German-speaking samples. Further studies are warranted to evaluate psychometric properties of the IES-2 in different samples, and to investigate its application in a clinical setting.

*Keywords:* Intuitive Eating; German Translation; Psychometric Properties; Eating Disorders.

## INTRODUCTION

Research on eating behaviours has traditionally focused on understanding maladaptive eating behaviours, such as dietary restriction and binge eating (e.g., Fairburn et al., 1998; Polivy & Herman, 2002; Stice, 2001, 2002; Vanderlinden et al., 2004), while largely neglecting people's strengths and their areas of resilience (Avalos & Tylka, 2006). Some studies have addressed positive eating behaviours as part of the eating disorders continuum (Mintz & Betz, 1988; Tylka & Subich, 2004); however, they tend to conceptualise these behaviours as the mere absence of eating disorder symptoms (cf. Tylka, 2006). It has been argued that adaptive eating represents more than just low levels of disordered eating behaviours (Tylka & Wilcox, 2006), demonstrating that it is associated with psychological well-being above and beyond eating disorder symptoms. The identification of such unique components has important implications for interventions, as they may foster and maintain healthy eating behaviours alongside preventing and treating maladaptive eating (Tylka & Wilcox, 2006; Tylka, 2006).

One adaptive form of eating that has recently attracted attention is the concept of 'intuitive eating', defined as a strong awareness of, and eating in response to physiological hunger and satiety cues, combined with a low preoccupation with food (Tribole & Resch, 1995; Tylka, 2006). Intuitive eaters neither eat for emotional or external reasons, nor do they rely on diet plans that instruct them what, when and how much to eat. They are attentive to how their body responds to certain foods and they make food choices that promote their health and body functioning, while at the same time tasting good (Tylka & Kroon Van Diest, 2013). Cross-sectional studies have found intuitive eating to be associated with lower body mass index (BMI; Denny et al., 2013; Gast et al., 2015; Herbert et al., 2013; Tylka & Kroon Van Diest, 2013; Tylka & Wilcox, 2006), lower triglyceride levels and cardiovascular risk (Hawks, Madanat, Hawks, & Harris, 2005), reduced eating disorder symptomatology (Denny

et al., 2013; Tylka & Kroon Van Diest, 2013; Tylka, 2006), and increased self-esteem and reduced negative affect (Tylka & Wilcox, 2006). Furthermore, interventions based on intuitive eating principles have been shown to have a positive impact on psychological health outcomes and eating behaviours, such as body image, self-esteem, interoceptive awareness, and anti-dieting attitudes, while decreasing dietary restraint, drive for thinness, depressive symptoms, and disinhibited eating (Bacon, Stern, Van Loan, & Keim, 2005; Cole & Horacek, 2010; Hawks, Madanat, Smith, & De La Cruz, 2008). At a metabolic level, there is evidence that non-diet interventions decrease blood lipids, and systolic blood pressure (Bacon et al., 2002).

Intuitive eating is typically assessed using self-report questionnaires. The first instrument measuring intuitive eating has been developed by Hawks, Merrill, and Madanat (2004) and consists of 27 items clustered into four dimensions: intrinsic eating, extrinsic eating, anti-dieting, and self-care. Two years later, Tylka published the initial 'Intuitive Eating Scale' (IES-1; Tylka, 2006), which was developed based on the ten principles of intuitive eating by Tribole and Resch (1995). These principles were combined into three separate, but interrelated factors (c.f., Tylka, 2006): (a) unconditional permission to eat (UPE; i.e., readiness to eat in response to internal physiological hunger signals and what food is desired at the moment), (b) eating for physical rather than emotional reasons (EPR; i.e., using food to satisfy physiological hunger drives rather than as a coping mechanism for emotional distress), and (c) reliance on internal hunger and satiety cues to determine when and how much to eat (RHSC; i.e., awareness and use of physiological hunger and satiety cues to guide one's food intake). The IES-1 was validated in a sample of college women, demonstrating good psychometric properties (Tylka, 2006).

More recently, the Intuitive Eating Scale has been improved (IES-2; Tylka & Kroon Van Diest, 2013). In its latest version it consists of 11 items from the original scale and 12

newly developed, positively scored items to assess the presence, rather than the absence of intuitive eating attitudes. The IES-2 has a four factor solution, including the three original factors and the newly developed three-item ‘body-food choice congruence’ (B-FCC) subscale measuring the inclination to make food choices according to one’s body’s needs. The B-FCC subscale was developed according to the component of ‘gentle nutrition’ as articulated by Tribole and Resch (2003), referring to the tendency to choose foods that honour health and body functioning, while at the same time eating palatable foods (Tylka & Kroon Van Diest, 2013). Similar to its precursor, the IES-2 yields good psychometric properties and construct validity (Tylka & Kroon Van Diest, 2013). Exploratory and second-order confirmatory factor analyses (CFAs) confirmed its four-factor structure in both female and male college students. IES-2 scores showed good to excellent internal consistency reliabilities and proved to be stable over a 3-week period. Several studies supported the construct validity of the IES-2 in both men and women, showing that overall intuitive eating and most subscale scores were linked with other eating, body-related, and well-being measures (Camilleri et al., 2015; Tylka, Calogero, & Daníelsdóttir, 2015; Tylka & Kroon Van Diest, 2013). The IES-1 and the IES-2 total and subscale scores were highly correlated, indicating a considerable construct overlap (Tylka & Kroon Van Diest, 2013). Furthermore, the IES-2 has been shown to be sensitive to the detection of treatment-related changes (Schaefer, 2015).

Although these results are very promising, studies included primarily small female student samples, raising questions concerning the generalizability to other populations. Also, to our knowledge, only two studies have investigated intuitive eating principles in other samples than North American college students (Camilleri et al., 2015; Madden, Leong, Gray, & Horwath, 2012). It seems mandatory, therefore, to further investigate intuitive eating principles and their associations with eating- and body-related constructs in different cultures, in the general public, and in clinical samples.

The primary aim of the present study was to validate a German version of the IES-2. European countries share a number of important sociocultural factors related to eating and weight with the USA; nevertheless, important differences exist between the US and Europe concerning languages, traditions, and eating patterns (López-Guimerà et al., 2013). Hence, psychometric properties and norms reported for North American college students do not necessarily apply to a German-speaking population and it is important to investigate whether intuitive eating comprises similar dimensions and is associated with the same eating- and body-related constructs in both cultures.

To further validate the IES, we used the diagnostic status of the participants (i.e., with or without an eating disorder) and analysed how individuals with different types of eating disorders responded to the IES subscales. The fundamental assumption behind intuitive eating is that, if listened to, the body ‘tells us’ when, what and how much to eat to maintain both nutritional health and an appropriate weight (Gast & Hawks, 1998). Intuitive eaters utilise this internal bodily information to guide food choices, rather than following external rules, such as diet plans to adhere to a culturally imposed thin-ideal stereotype (Augustus-Horvath & Tylka, 2011; Avalos & Tylka, 2006). Eating disorders, on the contrary, are characterised by restrictive food intake and/or overeating, as well as preoccupation with food, weight and body shape (American Psychiatric Association, 2013). Individuals with eating disorders focus on their outer appearance (rather than on internal bodily signals), which may lead to body dissatisfaction, as they perceive themselves not being able to live up to the unrealistic body ideals (Stice, 2002). Body dissatisfaction has been shown to promote eating pathology (e.g., dieting), as individuals attempt to restrict food intake to try to achieve an appearance similar to this cultural ideal (Garner, 1991). This dieting in turn increases the risk for the onset and maintenance of anorexic and bulimic symptoms (Stice & Shaw, 2002; Stice, 2002), and may lead to a repetitive cycle of episodes of food restriction followed by excessive food intake

(Polivy & Herman, 1999). Indeed, chronic dieters often oscillate between periods of food restriction, and excessive caloric intake (Heatherton, Herman, & Polivy, 1991, 1992; Polivy & Herman, 1985, 2002), which may dampen internal appetitive signals that would normally guide food intake.

In line with these findings, there is evidence suggesting the prominence of disturbed perception of internal bodily signals in individuals with eating disorders, both using self-report questionnaires (Fassino et al., 2004; Garner et al., 1983), and objective measures of the perception and processing of bodily signals (Klabunde et al., 2013; Pollatos et al., 2008). According to these results, we predicted lower intuitive eating scores in individuals diagnosed with an eating disorder compared to healthy controls.

In summary, the primary aim of the present study was to evaluate psychometric properties of a German version of the IES-2 in a large sample including both men and women. This included the investigation of its factor structure, internal consistencies, and correlations with other scales assessing maladaptive eating behaviours, attitudes towards physical appearance, perception of internal bodily sensations, and self-objectification. Second, we aimed at comparing intuitive eating scores between participants with and without eating disorder diagnosis and between different types of eating disorders. To our knowledge, this is the first study assessing intuitive eating principles in an eating disordered population.

## **METHOD**

### **Procedure**

Participants were recruited through online and print advertisements circulated at the University of Luxembourg, as well as by sending invitation emails to students and employees via electronic mailing lists. Participants were free to forward the link to their acquaintances in order to recruit additional participants. Furthermore, student councils of several universities in Germany and Switzerland were requested to distribute the online link to the survey among their



students. To recruit participants with eating disorders, the link was also posted on various eating disorder and obesity community forums.

The survey started with the participant information statement and consent form, followed by a series of socio-demographic questions. First, participants were asked to confirm that they were at least 18 years of age. This was followed by a set of various eating- and body-related measures, including intuitive eating scales, as well as questionnaires on interoceptive awareness and other psychological symptoms (for details on questionnaires, please see below). As an incentive, Amazon gift vouchers of a total value of 200 € were raffled among participants who completed the entire set of questionnaires and provided their email address. The online-survey was established and assembled using the survey platform SoSciSurvey (<https://www.soscisurvey.de>; Leiner, 2014). Questionnaire completion took approximately 30 - 45 minutes.

## **Participants**

The entire set of questionnaires was completed by 1134 participants. Most participants were women (81.3%;  $n = 922$ ) and they ranged in age from 18 to 77 years ( $M = 26.35$ ;  $SD = 9.11$ ). Their self-reported height and weight were used to calculate BMI ( $\text{kg}/\text{m}^2$ ). Women's mean BMI was 22.79 ( $SD = 5.77$ ; range: 13.01 - 70.15) and men's average BMI was 24.02 ( $SD = 4.13$ ; range: 16.05 - 52.24). The majority of participants were either students (62.3%;  $n = 707$ ) or currently working (22.8%;  $n = 259$ ). They self-identified as German (59.1%,  $n = 670$ ), Luxembourgish (21.7%,  $n = 246$ ), Swiss (7.8%,  $n = 89$ ), or Austrian (1.4%,  $n = 16$ ). 10% endorsed as 'other'.

Eating disorder diagnoses were assessed via self-report. Participants were asked if they had ever been diagnosed with an eating disorder and – if so – who had made the diagnosis. Only participants who indicated that they had received an official diagnosis by a mental health professional, for example, a psychiatrist or a clinical psychologist, were included in the eating

disorder subsample. They were also asked whether they were recovered or whether the eating disorder was current. Possible diagnoses were anorexia nervosa (AN), bulimia nervosa (BN), binge-eating disorder (BED), and Eating Disorders Not Otherwise Specified (EDNOS). A total of 174 participants (16.4%) stated having a history of eating disorder, with only one male, whose data was excluded from further analyses. Of the remaining 173 female participants, 47.4% ( $n = 82$ ) indicated that they had been diagnosed with AN, 36.4% ( $n = 63$ ) with BN, 11.0% ( $n = 19$ ) with BED, and 5.2% ( $n = 9$ ) with EDNOS. 93 participants (53.8%) indicated a current eating disorder, wherefrom 40.9% ( $n = 38$ ) indicated AN, 40.9% ( $n = 38$ ) BN, 11.8% ( $n = 11$ ) BED, and 6.5% ( $n = 6$ ) EDNOS. Because of the small sample size and the heterogeneity of the group, individuals with EDNOS diagnoses were discarded from further analyses.

## **Measures**

### *Intuitive Eating Scale (IES)*

Both the 21 items of the IES-1 (Tylka, 2006) and the 23 items of the IES-2 (Tylka & Kroon Van Diest, 2013) were administered. As the purpose of the present study was to validate a German version of the updated and improved IES, results from the IES-1 will not be reported in the present article. They can, however, be found in the supplemental materials (Appendix C).

The IES-2 consists of four subscales that can be added to form a total score measuring the overall level of intuitive eating: 1) unconditional permission to eat when hungry and what food is desired (e.g., ‘If I am craving a certain food, I allow myself to have it’; six items); 2) eating for physical rather than emotional reasons (e.g., ‘I stop eating when I feel full’; eight items), 3) reliance on internal hunger and satiety cues to determine when and how much to eat (e.g., ‘I trust my body to tell me when to eat’; six items), and 4) body-food choice congruence (e.g., ‘I mostly eat foods that make my body perform efficiently (well)’; three items). With samples of college women and men, IES-2 internal consistency reliabilities ranged between .87 and .89 for the total score and between .81 and .93 for the four subscales (Tylka & Kroon Van

Diest, 2013). IES-2 scores were stable over a 3-week period, with correlation coefficients ranging between .77 and .92 in different samples (Tylka & Kroon Van Diest, 2013). Items are rated on a 5-point Likert scale from 1 (*strongly disagree*) to 5 (*strongly agree*), with high values suggesting higher levels of intuitive eating. For the present study, German versions of the IES-1 (Herbert et al., 2013) and IES-2 were used.<sup>1</sup>

#### *Dutch Eating Behavior Questionnaire (DEBQ)*

The DEBQ (van Strien, Frijters, Bergers, & Defares, 1986; German version by Grunert, 1989) is a 33-item questionnaire measuring restrained eating, emotional eating and external eating. Restrained eating refers to the intention to eat less, and to maintain or lose weight (e.g., ‘How often do you refuse food or drink offered because you are concerned about your weight?’; 10 items). Emotional eating assesses the desire to eat in response to several distinct emotions (e.g., ‘Do you have a desire to eat when you are emotionally upset?’; 13 items). External eating measures eating in response to external food-related stimuli, regardless of the internal state of hunger or satiety (e.g., ‘If food smells and looks good, do you eat more than usual?’; 10 items). Responses are given on a 5-point Likert-type scale from 1 (*never*) to 5 (*very often*), with higher values indicating higher levels of restrained, external or emotional eating. All subscales are reported to have high internal consistencies, which was also found in the present sample: restrained eating (Cronbach's  $\alpha = .91$  in Grunert, 1989 vs  $\alpha = .92$  in the present sample), external eating ( $\alpha = .80$  vs  $\alpha = .84$ ) and emotional eating ( $\alpha = .91$  vs  $\alpha = .95$ ).

#### *Eating Disorder Inventory -2 (EDI-2)*

The EDI-2 (Garner, 1991; German version by Paul & Thiel, 2005) assesses the specific psychopathology of eating disorders. It consists of 91 items, each of which is answered along a scale ranging from 1 (*never*) to 6 (*always*). In the present study, only the first three subscales were used: Drive for thinness (e.g., ‘I exaggerate or magnify the importance of weight’; seven

---

<sup>1</sup> For more information on the IES-1, or to obtain the German IES-1 items, please contact B.M. Herbert (beate.herbert@gmx.de)

items), measuring excessive concerns with dieting, preoccupation with weight, and fear of gaining weight; bulimia (e.g., ‘I stuff myself with food’; seven items), assessing the tendency to consider and engage in episodes of uncontrollable overeating; Body dissatisfaction (e.g., ‘I think my thighs are too large’; nine items), referring to the degree of dissatisfaction with overall body appearance, as well as the size of different body parts. These three subscales were chosen because they assess core eating pathology typical of eating disorders, whereas the other subscales assess psychopathology commonly associated with, but not unique to, eating disorders (Garner, 1991). The first three EDI-2 subscales have been shown to have sound psychometric properties (Eberenz & Gleaves, 1994; Garner, 1991), with internal consistencies ranging between .81 and .91 in different samples (alphas = .89 - .92 in the current sample). Furthermore, Thiel and Paul (2006) reported test-retest reliabilities between  $r = .81$  and  $r = .89$  for eating disordered participants.

#### *Body Consciousness Questionnaire (BCQ)*

The BCQ (Miller, Murphy, & Buss, 1981) measures the habitual tendency to focus attention on the body using three subscales: private body consciousness, public body consciousness and body competence. Private body consciousness emphasizes symptoms and measures the tendency to be attentive to internal bodily sensations (e.g., ‘I am sensitive to internal bodily tensions’; five items). Public body consciousness assesses the tendency to focus on observable aspects of the body, that is, on exterior physical appearance (e.g., ‘I’m concerned about my posture’; six items). Body competence refers to the perceived efficacy of bodily actions (e.g., ‘For my size, I’m pretty strong’; four items). Items are rated on a 5-point Likert scale from 0 (*not at all characteristic*) to 4 (*extremely characteristic*), with higher scores indicating a greater body consciousness. Moderately high internal consistencies were found for both the private and public body consciousness subscales (alphas = .62 and .77, respectively), as well as for body competence ( $\alpha = .74$ ) (Montepare, 2006). Comparable levels of internal

consistency were found in the present sample (private body consciousness:  $\alpha = .67$ ; public body consciousness:  $\alpha = .74$ ; body competence:  $\alpha = .70$ ).

#### *Interoceptive Awareness Questionnaire – Expanded (IAQ-E)*

The IAQ-E (Craighead & Niemeier, 2002) is an expanded version of the interoceptive awareness subscale of the EDI-2 (Garner, 1983), which measures one's attention towards internal body states, such as emotions, hunger, and satiety. The interoceptive awareness subscale of the EDI-2 encompasses items assessing attentiveness to both physiological and emotional internal sensations. Eight new items were added to form the IAQ-E, with the objective to differentiate between the components of appetite and emotion awareness (Hill, Craighead, & Safer, 2011; Trenary et al., 2005). The IAQ-E consists of three subscales: appetite (e.g., 'I continue to eat after I feel full'; seven items), emotion (e.g. 'I get frightened when my feelings are too strong'; five items) and restrict (e.g. 'I try not to eat until I am very hungry'; three items). For the present study, only the subscales appetite and emotion were used. Items are rated on a 6-point Likert scale from 1 (*never*) to 6 (*always*), with higher scores indicating poorer interoceptive awareness. Previous research has supported the IAQ-E's internal consistency reliabilities (Cronbach's  $\alpha = .90$ ; Trenary et al., 2005; current study  $\alpha = .70 - .87$ ).

#### *Self-Objectification Questionnaire (SOQ)*

The SOQ (Fredrickson, Roberts, Noll, Quinn, & Twenge, 1998) measures the tendency of an individual to view his or her body in an observable, appearance-based (i.e., objectified) way, versus a non-observable competence-based way. It allows examining to what extent the person is concerned with his or her own appearance, without taking into account the level of satisfaction. The scale consists of 10 body attributes, five of which refer to physical appearance (e.g., 'weight') and five of which are based on physical ability (e.g., 'strength'). Respondents are instructed to rank these body attributes according to their impact on their own physical self-concept, from 0 (*lowest impact*) to 9 (*highest impact*). Self-objectification scores are calculated

by subtracting the summed ranks given to the ability items from the summed ranks of the appearance items. Scores range from -25 to 25, with positive scores indicating greater emphasis on appearance, which is interpreted as higher self-objectification. The SOQ shows satisfactory validity (Fredrickson et al., 1998) and good test-retest reliability (Miner-Rubino, Twenge, & Fredrickson, 2002).

#### *State-Trait-Anxiety Inventory – Trait Scale (STAI-T)*

Trait anxiety was measured with the trait form of the STAI (Spielberger, Gorsuch, & Lushene, 1970; for the German version, see Laux, Glanzmann, Schaffner, & Spielberger, 1981). The STAI-T comprises 20 items referring to how the respondent feels in general. Seventeen statements are formulated in the direction of anxiety (e.g., ‘I get in a state of tension or turmoil as I think over my recent concerns and interests’) and seven items are reverse scored (e.g., ‘I feel rested’). Items are rated on a 4-point Likert scale from 1 (*almost never*) to 4 (*almost always*). The STAI-T is found to have high internal consistency, with reported  $\alpha$ 's varying between .89 and .90 (Barnes, Harp, & Jung, 2002). A high internal consistency was also found in the present sample (Cronbach's  $\alpha = .93$ ).

#### *Body mass index (BMI)*

BMI ( $\text{kg}/\text{m}^2$ ) was calculated using self-reported weight and height.

#### **Translation of the IES**

Translation of the intuitive eating scales was carried out with the consent of the authors of the original scale. Both questionnaires were translated from English to German by bilingual researchers in the field of eating disorders. Thereafter, another bilingual researcher provided a back-translation into English. The back-translation was examined and compared with the original English version. In the case of incongruence between the two versions, item formulation was discussed and necessary adjustments were made (for the IES-1, see also Herbert et al., 2013).

## Data analysis

Statistical analyses were performed using the Statistical Package for the Social Sciences (SPSS; Version 22.0) and MPlus (Version 6.12, Muthén & Muthén, 2010). Due to the large sample size, distributions of all variables were examined by visually inspecting stem-and-leaf plots, boxplots, and Q-Q plots, and by checking values of the skewness and kurtosis statistics (Field, 2009). No obvious signs of deviation from a normal distribution were detected, so that parametric tests were used for further data analyses.

Differences between men and women on IES-2 total and subscale scores were calculated using Multivariate Analyses of Variance (MANOVA) with IES scales as dependent variables and gender as between-subject factor. CFA with maximum likelihood estimation and the covariance matrix as input was performed to investigate the factor structure of the German IES-2. Goodness-of-fit for the models was evaluated by the  $\chi^2$ -statistic. However, as the chi-square test is sensitive to sample size and usually significant in samples with more than 200 cases (Browne & Cudeck, 1993), we also used the comparative fit index (CFI), the root mean square error of approximation (RMSEA) and the standardised root mean square residual (SRMR) as guides in assessing fit. Levels of .90 or higher for CFI and .08 or lower for RMSEA and SRMR indicate a good fit of the model to the data (Browne & Cudeck, 1993; Hu & Bentler, 1999). Cronbach's  $\alpha$  was calculated for all subscales and the total score, for men and women separately. Pearson correlation coefficients were then used to determine construct validity. We examined the associations with measures of eating disorder symptoms, perception of internal bodily sensations, and trait anxiety, for women and men separately.

For comparisons between eating disordered and healthy individuals and between different types of eating disorders, only the female subgroup of the non-clinical sample was included in the analysis, because the eating disorder subgroup included only female participants. Differences on demographic variables were calculated using Bonferroni-

corrected independent samples *t* tests or one-way analyses of variance (ANOVAs). Given the differences in sample sizes, the distribution of variance was carefully examined. When variances were heterogeneous and when the larger variance was found in the smaller group, the Welch test was used (Glass & Hopkins, 1996). Differences between eating disordered and healthy individuals and between different types of eating disorders regarding IES-2 scales were calculated using MANOVAs with IES-2 scales as dependent variables and diagnostic group as between-subject factor. Pillai's trace was used as test statistic for the *F* tests, as it is more robust with unequal cell sizes (Hair, Black, Babin, Anderson, & Tatham, 2006). When *F* tests for the comparison of more than two groups were significant, post hoc analyses were conducted with the Games-Howell multiple comparison procedure for unequal sample sizes and variances (Games & Howell, 1976). Partial  $\eta^2$ , describing the proportion of total variability attributable to a factor, was displayed for estimation of effect sizes ( $\eta_p^2$ : small, 0.01; medium, 0.06; large, 0.14; see Cohen, 1988). When *t* tests were conducted, Cohen's *d* was used for estimation of effect sizes, with a value of .30 representing a small effect size, .50 a medium effect size, and .80 a large effect size (Cohen, 1988).

## **RESULTS**

### **Sex differences**

Mean total and subscale scores for the IES-2 are presented in Table 1. The MANOVA with IES-2 subscales as dependent variables and gender as between-subject factor was significant,  $V = 0.09$ ,  $F(5, 1128) = 21.75$ ,  $p < .001$ ,  $\eta_p^2 = .09$ . Men scored generally higher than women on the total score,  $F(1, 1132) = 61.24$ ,  $p < .001$ ,  $\eta_p^2 = .05$ , UPE score,  $F(1, 1132) = 19.00$ ,  $p < .001$ ,  $\eta_p^2 = .02$ , EPR score,  $F(1, 1132) = 90.72$ ,  $p < .001$ ,  $\eta_p^2 = .07$ , RHSC score,  $F(1, 1132) = 10.53$ ,  $p < .01$ ,  $\eta_p^2 = .01$ , and B-FCC score,  $F(1, 1132) = 9.58$ ,  $p < .01$ ,  $\eta_p^2 = .01$ .



Table 1

*Means and SD of IES-2 scores for women and men.*

	Women ( <i>n</i> = 922)	Men ( <i>n</i> = 212)	<i>F</i>	<i>p</i>
Total score	3.36 (0.69)	3.75 (0.48)	61.24	.000
UPE	3.30 (0.97)	3.61 (0.81)	19.00	.000
EPR	3.46 (0.94)	4.12 (0.68)	90.72	.000
RHSC	3.27 (0.97)	3.50 (0.80)	10.53	.001
B-FCC	3.36 (0.77)	3.54 (0.73)	9.58	.002

*Note.* UPE = Unconditional Permission to Eat, EPR = Eating for Physical Rather Than Emotional Reasons, RHSC = Reliance on Hunger and Satiety Cues, B-FCC = Body-Food Choice Congruence.

### **Confirmatory Factor Analysis**

Each IES-2 item was specified to load only on its latent first-order factor.

Relationships between the four hypothesised latent factors were estimated, and these four latent factors were specified to load on a second-order intuitive eating factor. Due to similar wording, items were expected to share method variance, wherefore correlated errors between these similarly phrased items were estimated. This decision was based on recommendations from scholars (e.g., Brown, 2006; Kline, 2005), who have argued that it is both acceptable and preferable to correlate the errors between certain items that share method effects. This model provided a good fit to the data for the combined sample, CFI = .962, SRMR = .062, RMSEA = .047, 90% CI [.043, .051],  $\chi^2(1, 218) = 728.97, p < .001$ . Not high, but only satisfactory loadings above .30 (Matsunaga, 2010) were observed for Items 13 ('When I am bored, I do not eat just for something to do.') and 18 ('Most of the time, I desire to eat nutritious foods.') in the total sample, and for Items 3 ('If I am craving a certain food, I allow myself to have it'), 9 ('I have forbidden foods that I don't allow myself to eat.'), and 12 ('I am able to cope with

my negative emotions (e.g., anxiety, sadness) without turning to food for comfort.’) in the male sample.

Altered only slightly (UPE was highly correlated with the second-order factor, so the mean of RHSC was fixed to 1, which is a commonly used constraint to ensure identifiability in CFA; Muthén & Muthén, 2010), this model also provided an acceptable fit to the data for women, CFI = .938, SRMR = .116, RMSEA = .061, 90% CI [.057, .067],  $\chi^2(1, 219) = 910.457, p < .001$ . Even though the SRMR score was above the conventional level, RMSEA and CFI indicated a good fit of the data. For men, RHSC was highly correlated with the second-order factor. Thus, the mean of UPE was fixed to 1 and the model showed also an acceptable fit to the data, CFI = .925, SRMR = .074, RMSEA = .056, 90% CI [.045, .066],  $\chi^2(1, 219) = 363.15, p < .001$ . Table 2 includes the item–factor loadings as well as the first-order factor loadings on the second-order intuitive eating factor for the overall and sex-specific models. Corrected Item-Total correlations can be found online in the supplemental material (Appendix C).

Table 2

*Standardised item and factor loadings of the IES-2 obtained by confirmatory factor analyses of the overall sample, women (n = 922), and men (n = 212).*

Factor and IES-2 item	First-order			Second-order		
	Overall	Women	Men	Overall	Women	Men
<b>UPE</b>				.74	.88	.71
Item 1	.96	.83	.97			
Item 3	.62	.65	.48			
Item 4	.63	.64	.59			
Item 9	.59	.64	.41			
Item 16	.78	.78	.72			
Item 17	.70	.71	.56			
<b>EPR</b>				.52	.43	.26
Item 2	.77	.78	.62			
Item 5	.85	.85	.82			
Item 10	.86	.86	.75			
Item 11	.76	.76	.67			
Item 12	.56	.56	.37			
Item 13	.45	.45	.43			
Item 14	.80	.82	.58			
Item 15	.68	.68	.52			
<b>RHSC</b>				.97	.58	.88
Item 6	.77	.86	.70			
Item 7	.60	.64	.63			
Item 8	.87	.90	.88			
Item 21	.82	.86	.76			
Item 22	.78	.81	.75			
Item 23	.86	.88	.80			
<b>B-FCC</b>				.33	.26	.03
Item 18	.31	.29	.42			
Item 19	.88	.89	.95			
Item 20	.94	.93	.86			

*Note.* IES-2 = Intuitive Eating Scale-2, UPE = Unconditional Permission to Eat, EPR = Eating for Physical Rather Than Emotional Reasons, RHSC = Reliance on Hunger and Satiety Cues, B-FCC = Body-Food Choice Congruence.

### **Internal consistencies**

For women and men, respectively, Cronbach's alphas for the IES-2 scores were .91 and .82 for the total score, .85 and .74 for UPE, .90 and .81 for EPR, .91 and .88 for RHSC, and .71 and .77 for B-FCC.

### **Construct validity**

The strength of the correlations between the IES-2 scores and other study variables were determined using Cohen's (1992) criteria (i.e.,  $r_s \geq .50$  indicate a large/strong effect size;  $r_s$  around .30, a medium/moderate effect size; and  $r_s$  around .10, a slight/negligible effect size). These correlations are presented in Tables 3 and 4.

Overall, IES-2 scores were moderately to strongly inversely correlated with eating disorder-related behaviours and cognitions in both men and women. Specifically, IES-2 total scores showed large negative correlations with restrained eating, emotional eating, drive for thinness, bulimia, and body dissatisfaction; however, it was only weakly negatively correlated with external eating. UPE was largely correlated in a negative direction with restrained eating and drive for thinness in both sexes. The negative correlation between UPE and body dissatisfaction was large in women and moderate in men. EPR was strongly inversely related to emotional eating and bulimia in both women and men. In women, RHSC was strongly negatively related to all eating disorder-related behaviours and cognitions, except for external eating. In men, RHSC was moderately negatively related to all eating-related variables, except for emotional and external eating.

Awareness of internal bodily sensations was assessed using the IAQ-E subscales appetite and emotion, as well as the private body consciousness subscale of the BCQ. In

women, IES-2 total score and UPE, EPR and RHSC subscale scores were moderately to strongly negatively related to appetite and emotional awareness, whereas weak correlations were found for the B-FCC subscale. In men, IES-2 total score and EPR and RHSC subscale scores had moderate to large negative correlations with both IAQ-E subscales. UPE was moderately negatively related to appetite awareness only and no correlations were found for B-FCC. As for private body consciousness, a medium correlation was found with RHSC in men and a weak positive correlation with B-FCC in both sexes.

Attitudes towards one's own physical appearance were assessed using the SOQ (i.e., the tendency to view one's body in an appearance-based way) and the public body consciousness subscale of the BCQ (i.e., general focus on outer appearance). Moderately sized negative links were observed between all IES-2 scores and self-objectification in women, whereas no correlations were found in men. Concerning public body consciousness, small to moderate negative correlations were found with the total score, UPE, and RHSC in women, and in men, public body consciousness was moderately negatively related to UPE only.

Furthermore, all IES-2 scores had moderate to strong negative links to anxiety in women, and in men, correlations with anxiety were observed for the total score, EPR, and RHSC. In men, IES-2 total and EPR were moderately associated with BMI, whereas in women, no noteworthy correlations between IES-2 scores and BMI were found.

Table 3

*Intercorrelations of the measured variables in women (n = 922).*

	IES-2				
	Total	UPE	EPR	RHSC	B-FCC
1. IES-2 Total					
2. IES-2 UPE	.72**				
3. IES-2 EPR	.77**	.27**			
4. IES-2 RHSC	.85**	.56**	.49**		
5. IES-2 B-FCC	.40**	.16**	.16**	.34**	
6. BCQ-Public	-.27**	-.33**	-.15**	-.21**	-.01
7. BCQ-Private	.02	-.04	-.04	.07*	.15**
8. BCQ-Body Competence	.20**	.06	.16**	.17**	.22**
9. DEBQ-Restrained Eating	-.66**	-.82**	-.28**	-.57**	-.17**
10. DEBQ-Emotional Eating	-.71**	-.28**	-.86**	-.48**	-.13**
11. DEBQ-External Eating	-.17**	.12**	-.31**	-.16**	-.06
12. EDI-Drive for Thinness	-.77**	-.78**	-.45**	-.67**	-.24**
13. EDI-Bulimia	-.76**	-.47**	-.69**	-.63**	-.23**
14. EDI-Body Dissatisfaction	-.65**	-.57**	-.43**	-.56**	-.25**
15. IAQ-Appetite	-.72**	-.40**	-.62**	-.67**	-.21**
16. IAQ-Emotion	-.50**	-.33**	-.41**	-.43**	-.20**
17. Self-Objectification	-.36**	-.32**	-.22**	-.31**	-.21**
18. STAI-Trait	-.61**	-.43**	-.47**	-.52**	-.29**
19. Age	.06	.00	.04	.08*	.06
20. BMI	-.11**	-.01	-.18**	-.07*	-.03

\*  $p < .05$ .

\*\*  $p < .01$ .

Table 4

*Intercorrelations of the measured variables in men (n = 212).*

	IES-2				
	Total	UPE	EPR	RHSC	B-FCC
1. IES-2 Total					
2. IES-2 UPE	.67**				
3. IES-2 EPR	.68**	.16*			
4. IES-2 RHSC	.73**	.40**	.20**		
5. IES-2 B-FCC	.26**	-.13	.15*	.11	
6. BCQ-Public	-.13	-.32**	.01	-.04	.16*
7. BCQ-Private	.09	-.03	-.08	.26**	.16*
8. BCQ-Body Competence	.12	-.08	.13	.08	.26**
9. DEBQ-Restrained Eating	-.58**	-.68**	-.18**	-.41**	-.06
10. DEBQ-Emotional Eating	-.57**	-.23**	-.77**	-.17*	-.11
11. DEBQ-External Eating	-.09	.11	-.20**	-.16*	.12
12. EDI-Drive for Thinness	-.67**	-.54**	-.36**	-.47**	-.23**
13. EDI-Bulimia	-.49**	-.25**	-.49**	-.34**	.01
14. EDI-Body Dissatisfaction	-.53**	-.28**	-.38**	-.36**	-.32**
15. IAQ-Appetite	-.53**	-.30**	-.33**	-.51**	-.03
16. IAQ-Emotion	-.32**	-.10	-.30**	-.24**	-.10
17. Self-Objectification	-.12	-.09	-.03	-.11	-.09
18. STAI	-.39**	-.17*	-.37**	-.23**	-.15*
19. Age	-.10	-.11	-.06	.01	-.12
20. BMI	-.34**	-.13	-.33**	-.22**	-.19**

\*  $p < .05$ .

\*\*  $p < .01$ .

## Comparison of eating disorder groups

Women with and without an eating disorder diagnosis did not differ regarding age ( $p = .22$ ) or BMI ( $p = .30$ ). The MANOVA with IES-2 scales as dependent variables and diagnostic status as between-subject factor revealed an overall effect of diagnostic status,  $V = 0.25$ ,  $F(4, 917) = 74.27$ ,  $p < .001$ ,  $\eta_p^2 = .25$ . Women with and without eating disorders differed significantly on all subscale scores and the total score (for means and standard deviations, see Table 5) in that those with self-reported eating disorders had significantly lower total scores,  $F(1, 920) = 256.77$ ,  $p < .001$ ,  $\eta_p^2 = .22$ , UPE scores,  $F(1,920) = 165.85$ ,  $p < .001$ ,  $\eta_p^2 = .15$ , EPR scores,  $F(1,920) = 68.54$ ,  $p < .001$ ,  $\eta_p^2 = .07$ , RHSC scores,  $F(1,920) = 250.70$ ,  $p < .001$ ,  $\eta_p^2 = .21$ , and B-FCC scores,  $F(1,920) = 43.38$ ,  $p < .001$ ,  $\eta_p^2 = .05$ .

Table 5

*Mean scores and SD for age, BMI, and IES-2 subscales and total score for women with and without eating disorders.*

	Eating disorders ( $n = 87$ )	Healthy controls ( $n = 835$ )	$F$	$t$	$p$
Age	26.32 (9.17)	25.06 (7.95)		1.24	.216
BMI	22.88 (5.54)	21.96 (7.68)		1.04	.299
<b>IES-2</b>					
Total	2.36 (0.60)	3.46 (0.61)	256.77*		.000
UPE	2.13 (0.92)	3.42 (0.89)	165.85*		.000
EPR	2.70 (1.05)	3.54 (0.89)	68.54*		.000
RHSC	1.88 (0.83)	3.42 (0.86)	250.70*		.000
B-FCC	2.85 (0.87)	3.41 (0.74)	43.38*		.000

*Note.* BMI = Body Mass Index, IES-2 = Intuitive Eating Scale-2, UPE = Unconditional Permission to Eat, EPR = Eating for Physical Rather Than Emotional Reasons, RHSC = Reliance on Hunger and Satiety Cues, B-FCC = Body-Food Choice Congruence.



Means and standard deviations for eating disorder categories are presented in Table 6. Subgroups of eating disorder diagnoses (AN, BN, BED) differed significantly in BMI  $F(2, 20.26) = 33.88, p < .001, \eta_p^2 = .61$ . Games-Howell post hoc tests revealed significant differences between all three groups. Women from the BED group had a significantly higher BMI than those with BN ( $p = .006$ ) or AN ( $p < .001$ ), whereas women with BN had a higher BMI than those with AN ( $p < .001$ ). There were no significant group differences in age ( $p = .13$ ). The MANOVA with IES-2 scales as dependent variables and type of eating disorder as between-subject factor was also significant,  $V = 0.29, F(8, 164) = 3.48, p < .001, \eta_p^2 = .15$ . Participants with different eating disorder diagnoses differed significantly on the UPE subscale,  $F(2,84) = 5.79, p = .004, \eta_p^2 = .12$ , and on the EPR subscale,  $F(2, 84) = 9.22, p < .001, \eta_p^2 = .18$ . Games-Howell post hoc tests revealed significantly higher UPE scores for participants with BED compared to participants with AN ( $p = .004$ ) or BN ( $p = .013$ ). Also, individuals with AN showed significantly higher EPR scores than participants with BN ( $p = .002$ ) or BED ( $p < .001$ ). No other post hoc comparisons reached statistical significance.

Table 6

*Mean scores and SD for age, BMI, and IES-2 subscales and total score by eating disorder diagnosis.*

	Anorexia nervosa ( <i>n</i> = 38)	Bulimia nervosa ( <i>n</i> = 38)	Binge-Eating Disorder ( <i>n</i> = 11)	<i>F</i>	<i>p</i>
Age	24.13 (7.00)	23.95 (6.39)	32.09 (12.23)	2.24	.128
BMI	17.31 (1.70)	22.31 (4.38)	36.50 (10.73)	33.88	.000
<b>IES-2</b>					
Total	2.47 (0.59)	2.22 (0.62)	2.43 (0.47)	1.89	.158
UPE	1.93 (0.15)	2.09 (0.14)	2.94 (0.23)	5.79	.004
EPR	3.19 (0.18)	2.37 (0.15)	2.10 (0.18)	9.22	.000
RHSC	1.84 (0.13)	1.82 (0.13)	2.21 (0.27)	1.01	.370
B-FCC	2.91 (0.15)	2.84 (0.15)	2.70 (0.22)	0.26	.770

*Note.* BMI = Body Mass Index, IES-2 = Intuitive Eating Scale-2, UPE = Unconditional Permission to Eat, EPR = Eating for Physical Rather Than Emotional Reasons, RHSC = Reliance on Hunger and Satiety Cues, B-FCC = Body-Food Choice Congruence.

## DISCUSSION

Translating questionnaires and administering them in another cultural context than the original within which they were developed, may change their psychometric properties. This seems to be of particular relevance for research on eating behaviours, where most instruments have only been validated in female samples from relatively homogeneous English-speaking populations (Striegel-Moore & Smolak, 2002). We, therefore, investigated the factor structure and psychometric properties of a German version of the IES-2 in a large German-speaking sample of women and men. A second objective of the study was to assess levels of intuitive eating in participants with an eating disorder diagnosis and to compare different types of eating disorders. To our knowledge, no study has yet investigated intuitive eating in an eating disordered population.

The proposed factor structure of the IES-2 could be largely confirmed for the German translation of the questionnaires. Maximum likelihood estimations and covariance matrices as input were used to conduct second-order CFAs. The original four-factor structure could be replicated and the four subscales loaded on a higher order intuitive eating factor. As expected the  $\chi^2$  goodness-of-fit index was significant given the large sample size. Nonetheless, the CFI, the RMSEA and the SRMR indicated a sufficient to good model fit for the total sample and the male and female subsamples. The second-order factor (Intuitive Eating) was either strongly interrelated with UPE (for females) or RHSC (for males), suggesting that Intuitive Eating has a slightly different emphasis in males and females. Except for item 18 ('Most of the time, I desire to eat nutritious foods') and the respective first-order factor (B-FCC), all factor loadings of the single items and first-order factors were high. Low B-FCC item loadings have also been reported for the French translation of the scale (Camilleri et al., 2015). The low second-order loading of the B-FCC was mainly due to the male subsample. Thus, for men, Body-Food Choice Congruence seems not to be a major part of Intuitive Eating. However, as omitting item 18 or the first-order factor did not improve the model fit, the original structure was kept for the IES-2. In summary, results underline the convincing factor structure of the German IES-2. The similar patterns of results found in the U.S. and in Germany indicate sufficient similarity between the two countries regarding the factor structure of the questionnaire. Reliabilities of the German IES-2 scores were upheld for both women and men, and internal consistencies were comparable to those reported in previous validation studies (Tylka & Kroon Van Diest, 2013; Tylka, 2006).

In a second step, convergent and divergent subscale and total score correlations were investigated. Interrelations between factors were moderate to large for most scales, except for the B-FCC factor that was only slightly to moderately related to the other scores. Most interestingly, the B-FCC subscale was slightly negatively related to UPE in men. A moderate

negative correlation was also found by Tylka and Kroon Van Diest (2013) in both sexes. The authors' explanation was that in some cases UPE and B-FCC might be incompatible.

Individuals who allow themselves to eat the foods they are craving (UPE) may sometimes crave (and consequently choose) foods that do not necessarily give their body energy and stamina (B-FCC). In the absence of craving, intuitive eaters will rather choose foods that honour health and body functioning (Tribole & Resch, 2003). The fact that intuitive eaters strike a balance between these two attitudes towards food might be the reason for the low or even negative correlations between B-FCC and the other factors.

For both men and women, higher levels of overall intuitive eating, UPE, EPR, and RHSC were related to lower eating disorder symptomatology. More precisely, UPE showed the strongest negative associations with scales referring to dietary restriction, whereas EPR had the highest inverse correlations with scales assessing emotional and bulimic eating behaviours and cognitions. This is not surprising since, by definition, individuals who allow themselves to eat a wide variety of foods and the food desired do not follow any dieting rules. Similarly, those who eat for physical rather than emotional reasons avoid using food to soothe emotions and are less prone to indulge in overeating (Tylka & Kroon Van Diest, 2013; Tylka, 2006). These findings are in line with previous research suggesting that intuitive eating should be negatively related to eating disorder symptomatology (e.g., Camilleri et al., 2015; Polivy & Herman, 1999; Tribole & Resch, 1995; Tylka & Kroon Van Diest, 2013; Tylka, 2006).

Two questionnaires were administered to assess awareness of internal bodily sensations: The IAQ-E is an eating disorder-specific instrument measuring problems in appetite and emotional awareness; the private body consciousness subscale of the BCQ measures the habitual tendency to be aware of bodily internal sensations that are not necessarily related to eating disorder symptomatology. For both men and women, overall intuitive eating and the three initial subscales were negatively related to problems in appetite

and emotional awareness, indicating that both hunger/satiety perception and the awareness of emotions are associated with intuitive eating. Correlations with more general internal body consciousness have been found only in men and particularly for the RHSC subscale. Nevertheless, it should be noted that the private body consciousness subscale of the BCQ yielded low internal consistencies.

Based on both humanistic (Rogers, 1961) and feminist body objectification theories (Fredrickson et al., 1998), Augustus-Horvath and Tylka (2011) highlighted the importance of body acceptance and resistance to adopt an observer's perspective of one's body for promoting an intuitive/adaptive eating style. They have argued that women who focus on their outer appearance are less likely to focus on physiological hunger and satiety signals (Augustus-Horvath & Tylka, 2011; Tylka, 2006). Accordingly, we found negative correlations between intuitive eating scales and body dissatisfaction, focus on outer appearance, and self-objectification in women. In men, links between intuitive eating scales and attitudes towards one's own physical appearance were less pronounced. These results corroborate findings by Fredrickson et al. (1998), showing that self-objectification and unhealthy eating behaviours were related in women only, probably due to the greater cultural demands placed on women to meet physical attractiveness ideals.

Interestingly, correlations between IES-2 total and subscale scores and BMI were zero or low in women and slight to moderate in men. Although these results are contrary to those published in some previous studies (e.g., Gast et al., 2015; Herbert et al., 2013; Madden et al., 2012), small correlations have also been reported in the validation study by Tylka and Kroon Van Diest (2013). These diverging results may in part be attributable to differences in sample composition: Whereas most of the previous studies focused on student participants, the present study included a broader range of individuals, also comprising participants with an eating disorder diagnosis. Also, the strength of the link between intuitive eating and BMI was

found to vary with age, showing stronger negative relationships between BMI and intuitive eating in early and/or middle adult women than in emerging adult women (Augustus-Horvath & Tylka, 2011). Further research is needed to better understand the relationship between BMI and intuitive eating, also taking into account potential confounding variables, such as age or sex.

It is also noteworthy that the B-FCC subscale showed overall smaller correlations with eating- and body-related variables compared to the three initial subscales and total score. In line with findings reported by Tylka and Kroon Van Diest (2013), B-FCC was rather linked to body-related variables, such as body dissatisfaction and body competence, than to eating disorder symptoms.

We found that women with an eating disorder had significantly lower values on all IES-2 subscale scores and the total score. This result is consistent with the high correlations found between IES-2 scores and other measures of eating disorder symptoms and further emphasizes the adaptive properties of intuitive eating and its components. When comparing women with different eating disorder diagnoses, significant differences were found for the UPE subscale and for the EPR subscale, but not regarding RHSC, B-FCC and the total score. Women with a BED diagnosis had higher UPE scores compared to AN and BN participants. A possible explanation could be that some UPE items might have been interpreted as ‘authorisation to overeat’ as experienced during a binge eating episode. Dieting or energy delimitation is a key feature in AN (especially in the restricting subtype) and BN (in between overeating episodes), but is certainly not compatible with UPE. Thus, although UPE is generally decreased in eating disorders, it might be even more so in AN and BN. Furthermore, women diagnosed with AN had higher EPR scores than individuals with BN or BED. This is plausible, as bingeing-purging has frequently been linked to higher emotional eating, a finding that could not always be replicated for restrictive eating behaviours (Danner, Evers,

Stok, Van Elburg, & De Ridder, 2012). These results further corroborate our previous findings on the strong negative correlation between UPE and variables assessing dietary restriction on the one hand, and between EPR and scales assessing emotional and bulimic behaviours on the other hand.

### **Strengths and limitations**

To our knowledge, this is the first study to investigate intuitive eating in a sample including eating disordered individuals from a range of diagnostic subgroups. Particular strengths of the current study are the large sample size and heterogeneous composition of the sample in terms of age, sex, and BMI.

Despite these strengths, several limitations need to be mentioned. One limitation of our study is that eating disorder diagnoses were based on self-reports. Although we tried to minimize the risk of including participants without a ‘real’ diagnosis into our clinical sample by only considering those who were diagnosed by a suitably qualified mental health professional, the information provided could not be verified and we could not be sure that the mental health professional in question made a valid diagnosis (e.g., based on DSM criteria). Similarly, body weight and height were based on self-reports, which might have led to inaccurate estimations (Glaesmer & Brähler, 2002). Differences between subjective and objective BMI data might also be responsible for the previously reported diverging results on the relationship between BMI and intuitive eating. Another issue is that internet-based data collection can result in selection or response bias. However, numerous studies show that questionnaire scores obtained online are comparable to those collected using traditional paper-and-pencil formats, and that psychometric properties of questionnaires are not affected by computerised data collection (Mayr et al., 2012; Naus, Philipp, & Samsi, 2009). On the contrary, anonymous, web-based surveys may even be more useful when collecting data on sensitive issues, such as eating- and body-related questions (Kays, Gathercoal, & Buhrow,

2012). Nevertheless, our sample consisted primarily of young, female, student participants, probably because many participants were recruited through Universities, but also because of the internet-based data collection that requires access to the Web and some technology and internet experience (Kays et al., 2012; Mayr et al., 2012). Future studies are warranted to replicate and extend the current findings in more diverse German-speaking samples.

## **Conclusion**

In summary, our findings support the factorial structure of the German version of the IES-2. Psychometric properties and correlates of the English IES-2 could be largely replicated for the German version. Hence, the German IES-2 constitutes a useful self-report instrument for the assessment of intuitive eating in German-speaking countries and makes cross-cultural comparisons possible. The current study also provided first evidence for reduced intuitive eating in individuals with an eating disorder, and the IES-2 may, therefore, prove to be a useful clinical tool that might be administered at different treatment stages to assess peoples' therapeutic progress. Indeed, it would be interesting for clinicians to monitor changes in adaptive eating alongside changes in maladaptive eating throughout therapy. Further studies are warranted to evaluate the psychometric properties of the IES-2 in community and clinical samples.



## **Chapter 3**

**The Water Load Test as a measure of gastric interoception: Development of a two-stage protocol and application to a healthy population.**

van Dyck, Z., Vögele, C., Blechert, J., Lutz, A., Schulz, A., Herbert, B.M.  
*The Water Load Test as a measure of gastric interoception: Development of a two-stage protocol and application to a healthy population.* Manuscript in preparation.

## **Abstract**

The sensitivity for one's own internal body signals (i.e., interoception) has been demonstrated to play an important role in the pathogenesis of eating and weight disorders. Most previous measures assessing interoceptive processing have not, or only partly, captured perception of hunger and satiety cues, which is a core aspect of interoceptive deficits in eating disorders. In addition, methods used to measure sensitivity to gastric signals are heterogeneous and findings inconsistent. The primary aim of the present study was to establish a standardised test to measure gastric interoception, and to provide normative data using a non-clinical adult sample. The two-step Water Load Test (WLT-II) involves ingestion of non-caloric water until perceived satiation (step 1) and until maximum fullness (step 2). The WLT-II consists of several variables: Besides volumes of water ingested until satiation and maximum fullness expressed in ml, the mean percentage of satiation to maximum fullness is calculated as an individual index of gastric interoception that is not confounded with stomach capacity. Ninety-nine healthy women participated in the study. Measures included the WLT-II, a heartbeat detection task, a self-report questionnaire assessing subjective sensations, and the Eating Disorder Inventory-2 (EDI-2). Twenty-eight participants underwent test-retest of the WLT-II. Results suggest that the WLT-II is a valid and reliable measure of gastric interoception. Importantly, satiation volume and percentage of satiation to maximum fullness were strongly positively related to self-reported bulimic symptoms, indicating that the WLT-II could emerge as a useful clinical tool to measure interoceptive processing in the field of eating disorders.

*Keywords:* Gastric interoception; Water Load Test; Eating Disorders; Satiation; Maximum Fullness.

## INTRODUCTION

The sensitivity for one's own internal body signals (i.e., interoception) has been demonstrated to be important for a broad range of cognitive and affective functions (e.g., Craig, 2009). In the literature, interoception is commonly referred to as the psychosomatic connection between the body and the brain, conveying signals regarding the state of the internal body and its visceral organs (Cameron, 2001). Several lines of investigation have suggested that interoception plays an important role in the pathogenesis of eating and weight disorders, in particular with respect to deficits in the perception of hunger and satiety (e.g., Kissileff et al., 1996; Sysko, Devlin, Walsh, Zimmerli, & Kissileff, 2007). These interoceptive deficits have typically been investigated in relation to, or by proxy of, awareness of emotional states. A widely used measure in the field of eating disorders is the Interoceptive Awareness subscale of the Eating Disorder Inventory (EDI; Garner et al., 1983), which measures the self-reported ability to accurately detect and respond to emotional states (Garner et al., 1983). More objective measures of interoception have typically focussed on people's ability to perceive their own heartbeats (e.g., Brener & Kluvitse, 1988; Critchley et al., 2004; Schandry, 1981; Whitehead et al., 1977). These so-called heartbeat perception tasks are well validated measures, as evidenced by relationships between cardiac perception accuracy and activation in brain structures responsible for the mapping of internal bodily responses, in particular the right anterior insula, and the somatomotor and cingulate cortices (Critchley et al., 2004).

In the field of eating disorders, however, studies using heartbeat detection tasks have yielded inconsistent results. Some studies reported attenuated interoceptive cardiac accuracy in anorexia nervosa (Pollatos et al., 2008) and in bulimia nervosa even after recovery (Klabunde et al., 2013), while others found no difference between eating disordered individuals and healthy controls (Eshkevari et al., 2014). One study even reported an

enhanced cortical representation of afferent signals from the cardiovascular system, as indicated by increased heartbeat evoked potentials in the electroencephalogram of participants with anorexia nervosa (Lutz et al., 2015). These inconsistencies might be explained by the increased levels of anxiety and/or depression in eating disordered patients, as evidenced by high rates of comorbid mood and anxiety disorders (Blinder, Cumella, & Sanathara, 2000; Hudson et al., 2007), which are themselves characterised by altered cardioceptive accuracy (Domschke et al., 2010; Terasawa, Shibata, Moriguchi, & Umeda, 2013; Terhaar et al., 2012). In addition, a core aspect of interoceptive deficits in eating disorders, that is a specifically impaired perception of hunger and satiety cues, is only partially captured by measuring cardioceptive accuracy. Hence, to advance our understanding of interoception in the eating domain, more specific interoception measures in the gastric tract are required.

Interoceptive processes in the gastric system can be assessed using different methods of distention of the stomach, most of which have been primarily used in patients with functional gastrointestinal disorders (Cameron, 2001). Gastric distention activates vagal afferents, which send signals from the stomach to the brain and lead to the perception of satiety and fullness, thereby regulating food intake (Hellström et al., 2004). Reduced responsivity to gastric signals could result in disordered eating behaviours, such as excessive food intake (Wang et al., 2008). Previous studies using gastric distention methods found larger gastric volume capacities in bulimic compared to healthy subjects (Geliebter & Hashim, 2001; Geliebter et al., 1992), together with a reduced sensitivity to gastric distention (Zimmerli, Walsh, Guss, Devlin, & Kissileff, 2006). These results are, however, based on invasive measures (e.g., barostat with intragastric balloon), which are cumbersome and unpleasant techniques that lack ecological validity (e.g., Andresen, 2009; Herbert, Muth, Pollatos, & Herbert, 2012; Jones, Hoffman, Shah, Patel, & Ebert, 2003).

A non-invasive and more participant-friendly method is the Water Load Test (WLT), which has been originally developed to induce gastric distention and assess gastrointestinal symptoms in patients with functional digestive disorders. The WLT stimulates the stomach using a natural distention stimulus (i.e., ingestion of water) and without the complex hormonal response of a caloric meal (Andresen, 2009; Koch, Hong, & Xu, 2000). Initial studies have shown that the WLT is acceptable to both healthy individuals and patients with gastrointestinal symptoms, such as nausea, early satiety, and bloating, that are frequently experienced by bulimic patients (Koch et al., 1998). Furthermore, the WLT has been demonstrated to be reproducible (Koch et al., 2000), is related to invasive, barostat measures (Boeckxstaens, Hirsch, Berkhout, & Tytgat, 1999; Li, Zhu, & Hou, 2004), and ingested water volume represents a valid indicator for feelings of subjective fullness (Jones et al., 2003; Koch et al., 2000). Importantly, cross-modal convergence has also been reported: The amount of water consumed during the WLT was negatively related to cardiac accuracy (Herbert et al., 2012), indicating that interoceptive sensitivity for cardiac and gastric signals overlaps in healthy persons, that is that there exists a generalised tendency to be aware of visceral events.

Although these findings suggest that WLT protocols represent a promising and valid way to assess gastric signal perception, it was initially developed to investigate gastric sensation and accommodation in patients with functional digestive disorders and would, therefore, benefit from some adaptations to measure eating-related gastric processing. An important limitation of the current WLT concerns the lack of standardisation in procedures and instructions (Andresen, 2009). For example, terms used to instruct the cessation of water ingestion have been used quite heterogeneously. The standardised definition of drinking thresholds, however, is of major importance to ensure comparability of results from different studies. We will use the term satiation, which is defined as ‘the process that leads to the termination of eating and that is accompanied by a feeling of satisfaction’ (Benelam, 2009).

This definition implies that, at least in healthy individuals, satiation is associated with a positive sensation of satisfaction, in contrast to the fullness that may be induced by gastric distention and has been assessed in most previous studies. Eating disordered or obese individuals, in contrast, tend to override satiation signals; they do not stop eating when they are comfortably full and continue to eat until feeling overstuffed (Blundell & Finlayson, 2004). Hence, from a clinical point of view, detection of satiation signals might be even more relevant than the stomach fullness assessed in the initial WLT.

Another important limitation of the current WLT is that it does not control for interindividual differences in gastrointestinal capacity. Research has shown that a stomach with a large capacity requires a bigger meal to trigger satiation (Geliebter et al., 1992), wherefore it remains unclear if increased water ingestion during the WLT is attributable to a larger gastric capacity, to a less accurate perception of gastric changes, or both. Introduction of two separate drinking thresholds can, at least in part, circumvent this issue through the calculation of the percentage distribution of the volumes. Hence, we decided to include a second drinking step until subjectively perceived maximum stomach fullness. The relation of satiation to fullness allows us to determine the percentage of maximum fullness at which satiation occurs. In other words, it represents a subjectively scaled measure of the interoceptive distance between the two thresholds, independent of absolute gastric capacity. To our knowledge, there is only one study that has directly compared two indices of perceived gastric fullness. Using a gastric balloon procedure, Geliebter and Hashim (2001) assessed maximum fullness alongside maximum capacity. They found that, although bulimic patients needed larger volumes to produce maximum fullness and capacity, the ratio of the volumes did not differ between groups. The authors concluded that satiety as such may not be altered in bulimic patients, but that bulimics rather have larger gastric capacities. These results

suggest that absolute volumes and their ratio provide differential and complementary information.

In light of this background, the primary purpose of the present study was to establish a standardised, two-step drink test to measure gastric interoception, consisting of two drinking periods assessing sensitivity for satiation and for maximum stomach fullness, and to provide normative data using a non-clinical adult sample. As previous research has been inconsistent with regard to the concepts measured, we included a short self-report questionnaire assessing subjective sensations related to ingestion and gastric distention. In accordance with studies showing that physiological responses arising from different visceral systems activate overlapping brain areas (e.g., Craig, 2009), and to further validate the WLT as a measure of gastric interoception, we investigated correlations between volumes of water ingested and cardiac accuracy. In addition, relationships between WLT-II variables and eating disorder symptoms were explored.

## **METHOD**

### **Participants**

Volunteer participants were recruited via advertisement from staff and students of the University of Luxembourg. To preclude gender effects on ingested water volumes during WLT (Boeckxstaens, Hirsch, van den Elzen, Heisterkamp, & Tytgat, 2001; Jones et al., 2003) and heartbeat perception (Vaitl, 1996), we only included women in the current sample. Demographic data were collected for age, socioeconomic status, body weight and height, current and former illnesses, medication use, and physical activity. Exclusion criteria comprised intense physical activity, current or past mental disorders, and current physical conditions or medication that affect diet or weight. The final sample consisted of 99 healthy female participants aged between 18 and 35 years ( $M = 22.86$ ;  $SD = 3.41$ ) with a mean body

mass index (BMI) of 22.33 ( $SD = 3.34$ ). All participants provided written informed consent with all procedures being approved by the University's Ethics Review Panel.

### **Cardiac accuracy**

Cardiac accuracy was assessed using a heartbeat detection task as described by Schandry (1981). A training interval of 25 seconds was followed by four experimental intervals of 25, 35, 45, and 55 seconds that occurred in a random order. During each interval, participants silently counted their heartbeats, while seated in a comfortable chair and instructed not to take their pulse or engage in any other manipulation that could facilitate heartbeat detection. Timing of counting phases and recording of participants' reports of heartbeats was controlled by a stimulus presentation software. Electrocardiogram (ECG) was recorded throughout the whole procedure at a sampling rate of 1000 Hz through a BIOPAC™ MP150 (Biopac Systems Inc., USA). The ECG raw signal was processed using the software Acqknowledge 4.2. Cardioceptive accuracy was determined across the four intervals using the following formula: Heartbeat detection score =  $1/4 \sum [1 - (|recorded\ heartbeats - reported\ heartbeats|)/recorded\ heartbeats]$ . Heartbeat detection scores vary between 0 and 1, with higher scores representing a smaller difference between the numbers of reported and recorded heartbeats, that is higher cardioceptive accuracy. The heartbeat detection task is a commonly used method to quantify interoceptive accuracy (Garfinkel et al., 2015; Herbert et al., 2013; Pollatos et al., 2008; Pollatos, Schandry, Auer, & Kaufmann, 2007; Tsakiris, Tajadura-Jiménez, & Costantini, 2011) that has shown good test-retest reliability (Knoll & Hodapp, 1992) and has been found to correlate well with other heartbeat detection tasks (Vaitl, 1996).

### **Water Load Test-II**

The WLT-II was performed by asking participants to drink non-carbonated water at room temperature over two successive 5-min periods. During the first period, participants were instructed to drink water ad libitum until reaching the point of perceived satiation, that



is, the sensation that determines meal termination. During the second period, participants were asked to drink again, this time until reaching the point of maximum stomach fullness.

Participants were not told that there would be a second drinking phase in order not to influence their first water intake (i.e., to avoid that they would drink less to ‘leave some room’ in the stomach). This 2-step drink test allows for differentiating between sensations of satiation and maximum fullness and, via calculation of the percentage distributions, creating an individual index of gastric interoception that is not confounded with stomach capacity.

Water was administered in non-transparent 5-litre flasks from which participants drank through a long straw to control for swallowing sizes. However, unbeknownst to the participants, the flask was filled with only 1.5 litres of water. This procedure blinded participants to the amount they consumed and gave them the impression of barely unlimited water supply while at the same time ensuring safety through the 1.5 litres maximum. After the first drinking period, the flask was substituted by a new but identically looking flask, again filled with 1.5 litres of water. The volume consumed from each flask in millilitres was recorded unobtrusively. The experimenter left the room during each drinking period to minimize experimenter effects.

## **Questionnaires**

### *WLT-II questionnaire*

Items to assess subjective sensations related to the WLT were chosen based on previous studies using distension methods and on the eating disorder literature. Participants were asked to concentrate on their current abdominal sensations, especially if their stomach felt full or empty. They then rated their momentary feelings of satiation and fullness, and completed eight WLT questionnaire items measuring sensations of thirst, stomach tension, immobility, discomfort, guilt, sluggishness, nausea, and arousal. All items were answered on a 7-point scale ranging from 1 (*no sensation/not at all*) to 7 (*extremely*). Ratings were

obtained before the first water intake (t0, baseline), and after the first (t1) and second (t2) drinking period.

#### *Eating Disorder Inventory-2 (EDI-2)*

The EDI-2 (Garner, 1991; German version by Paul & Thiel, 2005) assesses the specific psychopathology of eating disorders. It consists of 91 items, each of which is answered on a scale ranging from 1 (*never*) to 6 (*always*). In the present study, only the first three subscales were used: Drive for thinness (e.g., ‘I exaggerate or magnify the importance of weight’; seven items), measuring excessive concerns with dieting, preoccupation with weight, and fear of gaining weight; Bulimia (e.g., ‘I stuff myself with food’; seven items), assessing the tendency to consider and engage in episodes of uncontrollable overeating; Body dissatisfaction (e.g., ‘I think my thighs are too large’; nine items), referring to the degree of dissatisfaction with overall body appearance, as well as the size of different body parts. These three subscales were chosen because they assess core eating pathology typical of eating disorders, whereas the other subscales assess psychopathology commonly associated with, but not unique to, eating disorders (Garner, 1991). The EDI-2 has been shown to have sound psychometric properties (Eberenz & Gleaves, 1994; Garner, 1991): Drive for thinness had internal consistencies between .81 and .85 in different samples ( $\alpha = .87$  in our sample). Bulimia had internal consistencies between .83 and .87 ( $\alpha = .79$  in the present sample). For the subscale body dissatisfaction, internal consistencies of .91 have been reported ( $\alpha = .88$  in the current sample). Furthermore, Thiel and Paul, (2006) reported test-retest reliabilities between  $r = .81$  and  $r = .89$  for eating disordered participants.

#### *Body Consciousness Questionnaire (BCQ)*

The BCQ (Miller et al., 1981) measures the habitual tendency to focus attention on the body using three subscales: private body consciousness, public body consciousness and body competence. In the present study, only the private body consciousness (PBC) subscale was

used. PBC emphasizes symptoms and measures the tendency to be attentive to internal bodily sensations (e.g., ‘I am sensitive to internal bodily tensions’; five items). Items are rated on a 5-point Likert scale from 0 (*not at all characteristic*) to 4 (*extremely characteristic*), with higher scores indicating a greater focus on internal bodily sensations. Moderately high internal consistencies were found for the PBC subscale in previous studies ( $\alpha = .62$ ; Montepare, 2006) and in the present sample ( $\alpha = .62$ ).

### **Procedure**

Participants were instructed to refrain from eating at least three hours before taking part in the experiment, and from drinking during the two hours prior to the session. All participants reported having complied with this instruction. Upon arrival, they were led into a sound-attenuated room, where a short interview was conducted to assess sociodemographic characteristics. They were then weighed and measured without shoes, before completing the heartbeat detection task. Participants were encouraged to use the rest room before the WLT-II was conducted. Subsequently, participants performed the WLT-II and completed the WLT-II questionnaires in the order described previously. Participants maintained a half-supine position throughout the experimental session. Finally, they completed the EDI and the BCQ, together with other questionnaires not described here. A subset of participants ( $n = 28$ ) underwent the WLT on a second occasion one week after the initial testing session to investigate test-retest reliability.

### **Data analysis**

For normally distributed data, correlations were calculated using Pearson’s  $r$ , whereas correlations between non-normally distributed data were determined using Spearman’s rho. To analyse the factorial structure of the WLT questionnaire, a principle component analysis (PCA) and varimax rotation was conducted. The number of factors was determined by Horn’s parallel analysis (Horn, 1965) in conjunction with visual inspection of the scree plot in order

to prevent overfactoring (Fabrigar, Wegener, MacCallum, & Strahan, 1999). In parallel analysis, the eigenvalues of empirical components are compared with those of components derived from random datasets with identical specifications (i.e., sample size, number of variables). Only factors with greater corresponding eigenvalues than those estimated from the random data were retained (O'Connor, 2000). The effects of water intake on subjective sensations were analysed using repeated-measures analyses of variance (ANOVA), followed, where appropriate, by Bonferroni-corrected paired-samples *t* tests. Greenhouse-Geisser adjusted degrees of freedom were used in case of violation of the sphericity assumption.

## **RESULTS**

### **Normal values for ingested water volumes**

The mean volume (ml) required to produce satiation (sat\_ml) was 417.63 ml (*SD* = 174.63; range: 136 – 1231 ml) and the mean additional volume needed to produce maximum fullness ( $\Delta$ full\_ml) was 332.44 ml (*SD* = 184.70; range: 59 – 868), adding up to a mean total water volume (total\_ml; i.e., the sum of both volumes) of  $M = 750.06$  (*SD* = 277.66; range: 277 – 1683 ml). The mean percentage of satiation to total volume (sat\_%) was  $M = 58.25$  (*SD* = 16.38; range: 24.61 – 93.50).

### **Ingested water volumes and heartbeat perception**

Cardiac accuracy data from four participants was missing due to technical problems. The remaining 95 participants had a mean heartbeat detection score of .60 (*SD* = .18; range: .19 - .94). This distribution of cardiac accuracy as measured by the heartbeat detection task is comparable to the distribution found in earlier studies in nonclinical samples (Herbert et al., 2012; Herbert, Pollatos, Flor, Enck, & Schandry, 2010).

Heartbeat detection scores correlated significantly although weakly negatively with sat\_ml ( $r = -.26$ ,  $p = .011$ ) and moderately negatively with  $\Delta$ full\_ml ( $r = -.41$ ,  $p < .001$ ), and total\_ml ( $r = -.42$ ,  $p < .001$ ). BMI was positively correlated with sat\_ml ( $r = .28$ ,  $p = .005$ ),

$\Delta\text{full\_ml}$  ( $r = .29, p = .004$ ), and  $\text{total\_ml}$  ( $r = .35, p < .001$ ). After adjustment for BMI, cardiac accuracy continued to be significantly correlated with  $\text{sat\_ml}$  ( $r = -.22, p = .032$ ),  $\Delta\text{full\_ml}$  ( $r = -.38, p < .001$ ), and  $\text{total\_ml}$  ( $r = -.35, p < .001$ ). The heartbeat detection score was not related to  $\text{sat\_}\%$  ( $r = .19, p = .065$ ).

### **Ingested water volumes and private body consciousness**

PBC scores ranged from 0.60 to 4.00 ( $M = 2.38, SD = 0.74$ ). There was no significant correlation between self-reported PBC and  $\text{sat\_ml}$  ( $r = -.05, p = .623$ ),  $\Delta\text{full\_ml}$  ( $r = -.08, p = .431$ ) or  $\text{sat\_}\%$  ( $r = .10, p = .325$ ).

### **Test retest of the WLT**

Twenty-eight subjects underwent WLT-II on two occasions separated by one week. Correlations between  $\text{sat\_ml}$  at the first session (WLT1) and at the second session (WLT2) were high ( $r = .67, p < .001$ ). Participants drank significantly less water until feeling satiated at WLT2 compared to WLT1,  $t(27) = 2.82, p = .009, d = 0.53$ . Correlations between  $\Delta\text{full\_ml}$  at WLT1 and WLT2 were also large ( $r = .70, p < .001$ ), and there was no significant difference between water volumes at WLT1 and WLT2,  $t(27) = 1.42, p = .169$ .

### **Subjective ratings**

For the WLT-II questionnaire, the Kaiser–Meyer–Olkin measure (Kaiser, 1974) revealed a value of .73 and Bartlett’s test of sphericity (Lawley, 1956) was significant ( $p < .001$ ), both suggesting that the dataset was adequate for factor analysis (Tabachnick & Fidell, 2007). Parallel analysis and inspection of the scree plot indicated a one-factor solution. Only items with factor loadings  $\geq .40$  were considered (Stevens, 1996), leading to the deletion of three items (thirst, stomach tension and immobility). The five remaining items (discomfort, guilt, sluggishness, nausea, and arousal) were subjected to another PCA, which confirmed the unidimensional structure. The final scale accounted for 51.75% of the variance and was labeled Negative Affect (NA). Its factor loadings ranged from .67 to .79. The corresponding

Cronbach's alpha values were .76 at baseline (t0), .77 after the first water intake (t1), and .72 after the second water intake (t2).

Mean values for satiety, fullness, and NA are depicted in Figure 1. Repeated measures ANOVA and post-hoc *t* tests revealed increasing satiety and fullness ratings from t0 to t1 and from t1 to t2 (all *ps* < .001), thereby confirming sensitivity to the changes in ingested water volumes. Interestingly, NA was not affected by the first drinking period (*p* = .68), but a significant increase was observed after the second water intake (*p* < .001). These findings suggest that drinking until satiation was not experienced as aversive in the present sample and lend further credibility to the idea that ingestion until satiation and until fullness are conceptually different.

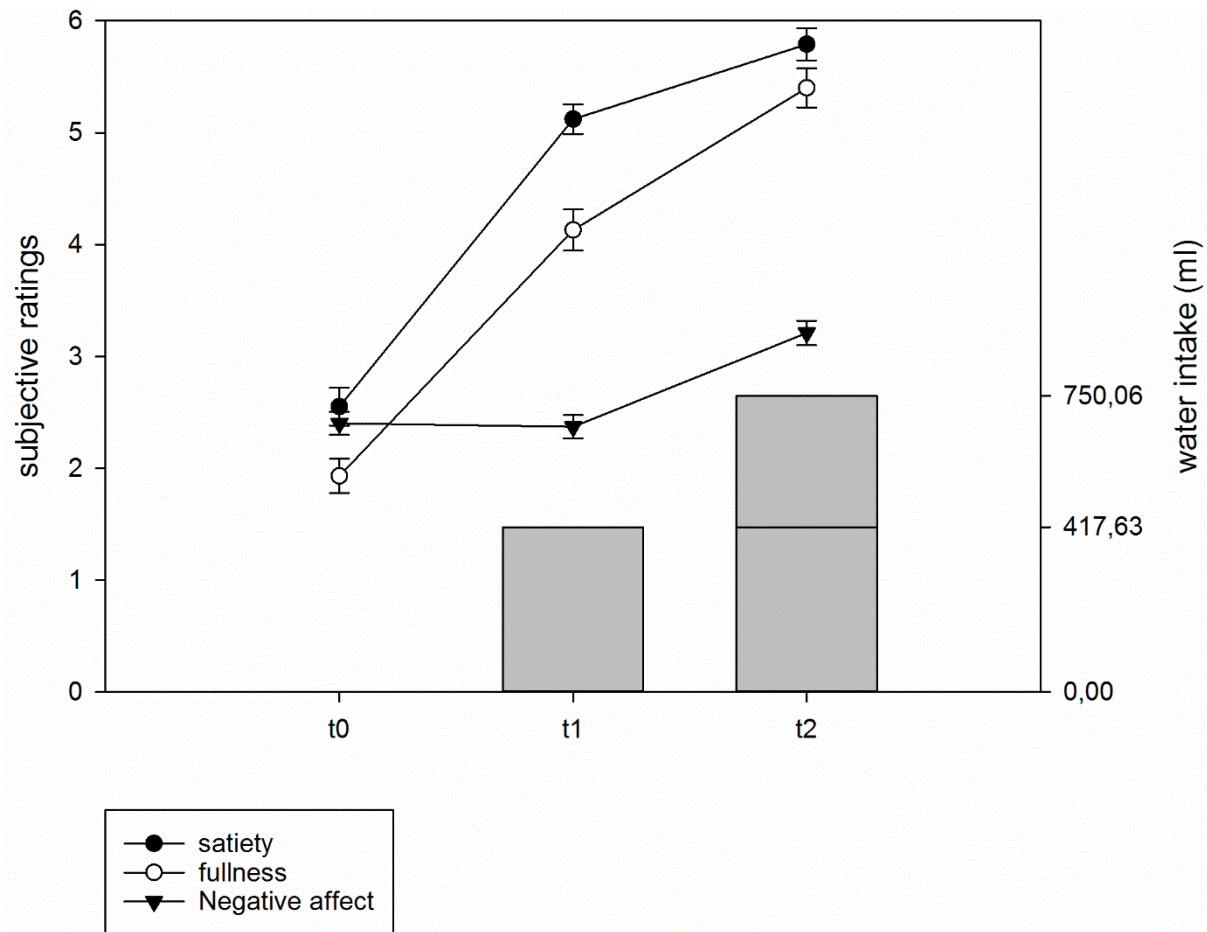


Figure 1. Mean ( $\pm$  SE) subjective sensations related to the WLT-II and ingested water volumes at t0, t1, and t2.

### Correlations with eating disorder pathology

Sat\_ml was strongly positively related to the EDI subscale bulimia ( $r = .53, p < .001$ ), and moderately positively correlated with drive for thinness ( $r = .32, p = .001$ ) and body dissatisfaction ( $r = .27, p = .006$ ). As for  $\Delta$ full\_ml, a moderate positive correlation was found with drive for thinness ( $r = .29, p = .003$ ), but not with body dissatisfaction ( $r = .16, p = .114$ ), or bulimia ( $r = .14, p = .176$ ). Sat\_% was positively related to bulimia ( $r = .44, p < .001$ ), but not to drive for thinness ( $r = -.04, p = .694$ ) or body dissatisfaction ( $r = .08, p = .431$ ).

## DISCUSSION

Impaired perception of interoceptive cues is a frequently suggested abnormality in eating disordered patients. However, the currently available tests to quantify interoception in the eating domain are nonspecific, unstandardised, and yield heterogeneous results. In the present study, we aimed at developing a standardised test to measure gastric interoception and to evaluate it in a non-clinical sample. Importantly, the WLT-II was designed to be easy to apply, well tolerated, and should improve upon some of the limitations noted for the initial version.

Our first main observation, in many ways a test of construct validity, was that both the ingested volume of water required to produce satiation and maximum fullness were significantly negatively related to cardiac accuracy. It has to be noted, however, that the association between satiation volume and heartbeat detection scores was weak, whereas moderate correlations were observed with maximum fullness volume. These results suggest that interoceptive cardiac accuracy measured with the heartbeat detection task is rather associated with the threshold to maximum fullness than with comfortable satiation. In addition, both ingested volumes were related to BMI. A positive association between BMI and maximal ingested volume in healthy controls has also been reported in previous research (Boeckxstaens et al., 2001; Mejía-Rivas, Remes-Troche, Montañó-Loza, Herrera, & Valdovinos-Díaz, 2009).

Our findings reinforce and extend results from previous studies, which indicate that sensitivity for interoceptive processes may be generalised, at least across cardiovascular and gastric domains (Herbert et al., 2012; Whitehead & Drescher, 1980). Similar to the present findings, Herbert and colleagues (2012) reported that individuals with high heartbeat detection scores consumed significantly less water until reaching fullness. In their study, however, they only used one drinking threshold, thereby not differentiating levels of satiation



and stomach fullness. Our results extend these findings by indicating that a correspondence between gastric and cardiac sensitivities emerges primarily after a comfortable satiation threshold is overcome. These findings may be explained by the fact that water quickly empties from the stomach, which could have contributed to the weak association between the first drinking volume and cardiac sensitivity. Indeed, gastric emptying of a liquid starts within minutes of water ingestion (Hausken, Gilja, Odegaard, & Berstad, 1998), so that part of the water ingested at the beginning of the WLT-II (i.e., during the first drinking period) may have emptied immediately to the duodenum, which could have influenced gut perception and thereby intake until satiation (Serra, Azpiroz, & Malagelada, 1998).

Validity of the WLT-II is also supported by self-reported subjective sensations in response to drinking periods. Satiety and fullness ratings increased as a consequence of water consumption, which further substantiates the suitability of the WLT-II to gradually induce satiation and gastric fullness. Similarly, previous studies have demonstrated that gastric distention in the absence of caloric intake (i.e., by inserting gastric balloons and filling them with water) increases sensations of fullness (Geliebter & Hashim, 2001; Geliebter et al., 1996). It has, however, been questioned whether these findings were due to the discomfort associated with the placement and inflation of the balloons, rather than normal feelings of fullness. The present findings add to this line of research by showing that sensations of satiation and fullness may be influenced by non-invasively loading the stomach with water. Furthermore, we found that NA was not affected by drinking until satiated, yet a significant increase in NA was observed after the second drinking period. This is not surprising, as the NA subscale is mainly composed by items referring to states of discomfort, including both psychological and physiological factors. This result further confirms the distinction between satiation reflecting a positive, comfortable sensation, in contrast to maximum fullness, which has clearly been found to cause negative subjective sensations. In addition, no participant

refused to drink during the second period, which further confirms that satiation and maximal fullness are well discriminable interoceptive states.

The amount of water ingested was uncorrelated with the PBC subscale measuring the general tendency of an individual to be attentive to internal bodily sensations. This finding is in line with previous research on cardiac accuracy that failed to find an association between self-reported awareness and experimentally measured visceral sensitivity (Ainley & Tsakiris, 2013; Dunn et al., 2007). To our knowledge, this study is among the first to directly investigate the relationship between interoceptive performance and self-reported interoceptive sensibility using visceral sensations that are not triggered by heartbeat signals. According to the theoretical framework of interoception by Vaitl (1996), it could be argued that the WLT-II reflects representations of visceral-afferent signal transmission in the central nervous system, whereas questionnaires represent individual beliefs that are a result of learning processes. Thus, it is unlikely that sensitivity for gastric stimuli measured using the WLT-II and questionnaire data reflect the same psychobiological constructs. Nonetheless, there is a need for future studies to investigate the correspondence between objective interoceptive performance and self-perceived interoception in other visceral bodily systems (for example respiration) and using different questionnaire measures.

Although results were based on a small sample size and should be considered preliminary, they indicated satisfactory repeatability over time for both drinking periods. Test-retest correlations for the WLT-II were slightly lower than those reported for the heartbeat detection task (Ehlers & Breuer, 1992) and comparable to the values achieved using a standard 5-minutes WLT (Jones et al., 2003). Stability of the WLT-II was, however, challenged by the differences in water volumes consumed between the first and the second session. Indeed, participants drank significantly less water until feeling satiated at the second compared to the first session. This is most probably due to the fact that, during the first

session, participants were unaware that they would have to drink twice, whereas at re-test they remembered the procedure. Hence, knowing that there would be a second drinking phase, some individuals might have consumed less water until satiation at re-test. Lower water volumes at re-test were also reported by Jones and colleagues (2003) using a traditional 5-minutes WLT. These results suggest that the WLT-II is prone to the within-subject variation observed with other measures of gastric function, especially using liquid stimuli (Kong, Perkins, King, Blackshaw, & Macdonald, 1998; Roland, Dobbeleir, Vandevivere, & Ham, 1990).

Across this non-clinical sample of young adults, our data shows positive correlations between WLT-II variables and eating disorder psychopathology. Most notably, satiation volume was strongly positively related to bulimic symptoms, whereas no noteworthy correlations were found for fullness volume. These findings suggest that individuals with bulimic symptoms (i.e., binge eating and purging behaviours) either need considerably larger volumes until the onset of satiation, or are less sensitive to gastric signals and therefore drink beyond the satiation threshold. This question may be addressed by examining the percentage distribution of the volumes. Percentage of satiation to total volume was positively related to bulimic symptoms, indicating that in individuals with high bulimia scores, satiation is reached at a larger proportion of maximum stomach fullness. These results suggest that satiation may be disordered in bulimic individuals, as they stopped drinking at a point that was closer to their (subjective) gastric capacity. In contrast, Geliebter and Hashim (2001) found no difference in the ratio of the volumes between binge eating disordered patients and control subjects. In their study, however, they assessed maximum fullness and maximum discomfort volumes, in contrast to satiation and maximum stomach fullness in the present study. Also, they induced gastric distention using an invasive, gastric balloon method, which could account for the differing findings.

As outlined in the introduction, previous research investigating interoceptive processing in the eating domain mostly relied on emotional and cardiac perception and yielded contradictory findings. Those studies directly measuring gastric perception have used differing methods. For example, development of satiation and meal termination have frequently been examined using different laboratory test meals (e.g., Kissileff et al., 1996; Sysko et al., 2007). Caloric meals introduce a variety of factors that are difficult to control for, such as caloric composition, osmolality, palatability, and consistency (Jones et al., 2003). Water, on the other hand, offers a modality that restricts satiation determinants to gastrointestinal distention, visceral sensations, and psychological concomitants of fullness. Especially during and shortly after food intake, when overeating may occur, sensations of satiation are primarily determined by gastric distention rather than by the nutrient content of a meal (Janssen et al., 2011). Gastric distension triggers mechanosensitive receptors that in turn relay their information via vagal afferents (Sengupta & Gebhart, 1994) to the central nervous system (Molinari et al., 2006), thereby regulating satiation and food intake. Accordingly, it has been demonstrated that meal volume, but not energy content, affected perceptions of fullness and satiety in healthy participants (Goetze et al., 2007). These observations, together with the present findings, suggest that loading the stomach with water represents a standardised and non-invasive method to investigate the development and perception of satiation and stomach fullness.

We do not claim that the WLT-II is an accurate measure of gastric volume, because there is a number of confounding variables that this test cannot account for. Hence, the WLT-II was not designed to assess stomach capacity, but it was rather developed as a standardised and non-invasive test to objectively measure sensitivity for gastric functions, irrespective of stomach volume. The present results suggest that the WLT-II is an easily performed, well tolerated and reliable test that correlates well with the commonly utilised heartbeat detection

task to measure objective interoceptive sensitivity. Importantly, the WLT-II could emerge as a useful clinical tool to measure interoceptive processing in eating disorders. Nevertheless, these results are preliminary and further research is warranted to investigate the clinical utility and reproducibility of the test.

## **Chapter 4**

### **Gastric interoception and gastric myoelectrical activity in bulimia nervosa and binge-eating disorder.**

van Dyck, Z., Schulz, A., Blechert, J., Herbert, B.M., Lutz, A., Vögele, C.  
*Gastric interoception and gastric myoelectrical activity in bulimia nervosa and binge-eating disorder.* Manuscript in preparation.

## **Abstract**

Identifying factors that control food intake and meal termination is crucial to the understanding and treatment of eating disorders characterised by binge eating. As a digestive organ and periodic reservoir for ingested food and drinks, the stomach plays an important role in the development of satiation. Nevertheless, little is known about the processing of gastric stimuli and gastric motility in patients with binge eating behaviours, that is, bulimia nervosa (BN) and binge-eating disorder (BED). The present study aimed at investigating the onset of satiation and maximum stomach fullness in patients with BN or BED (eating disordered group;  $n = 29$ ) and healthy controls (healthy control group;  $n = 32$ ). In addition, gastric myoelectrical activity was measured before and after ingestion of non-caloric water. The onset of satiation and stomach fullness was assessed using a novel 2-step Water Load Test (WLT-II). Gastric myoelectrical activity was measured by electrogastrography (EGG). ED participants drank significantly more water until feeling satiated during the WLT-II. The percentage of normal gastric myoelectrical power was significantly smaller in ED compared to HC participants. Power in the bradygastria range was greater in ED than in HC subjects. Percentage of normogastria was strongly negatively related to the number of objective binge eating episodes per week in bulimic patients. ED patients have a delayed response to satiation compared to HC participants, together with abnormal gastric myoelectrical activity. Repeated binge eating episodes may induce disturbances to gastric motor function.

*Keywords:* gastric interoception; binge-eating disorder; bulimia nervosa; electrogastrography; gastric myoelectrical activity.

## INTRODUCTION

Bulimia nervosa (BN) and binge-eating disorder (BED) are prevalent and serious mental health problems that are marked by medical complications, comorbid psychopathology, and increased all-cause mortality (Crow et al., 2009; Mond & Hay, 2007; Steinhausen & Weber, 2009). Their key characteristic is the occurrence of recurrent binge eating episodes, including consumption of large amounts of food in a discrete period of time, accompanied by a sense of loss of control over eating (American Psychiatric Association, 2013). These episodes have also been termed objective bulimic episodes (Fairburn & Cooper, 1993). A better understanding of the factors that control food intake and meal termination are crucial to the etiological models of BN and BED and their treatment. Therefore, the present study focuses on the role of gastric activity, associated sensations, and their relationship with BN and BED.

As a digestive organ and periodic reservoir for ingested food, the stomach is important for the regulation of food intake. Gastric distention during intake activates vagal and spinal mechanosensitive afferents, which transmit signals from the stomach to the brain and lead to the perception of satiation and fullness (Hellström et al., 2004). Reduced sensitivity to these signals may result in excessive food intake (Wang et al., 2008). Several lines of research suggest that patients with eating disorders in which binge eating is a central characteristic have disturbances in the development of satiation and an accompanying difficulty in stopping eating. According to DSM-5 criteria, binge meals are larger than what most people would eat under similar circumstances (American Psychiatric Association, 2013), and laboratory studies have shown that, even under controlled circumstances, bulimic patients eat larger meals than control subjects (Kissileff, Walsh, Kral, & Cassidy, 1986). Furthermore, patients require much larger amounts of food than controls to reach similar levels of fullness (Kissileff et al., 1996). Nevertheless, the macronutrient composition of binge meals was found to be similar to



that of normal meals (Kissileff et al., 1996), suggesting that the key problem in binge eating episodes is the amount of food consumed, rather than the kinds of foods chosen (Walsh et al., 2003).

Because gastric volume, rather than nutrient content influences meal termination (e.g., Phillips & Powley, 2000), several studies have investigated stomach function in patients with BN or BED. It has been found that BN patients exhibit numerous gastric abnormalities, including larger gastric capacities (Geliebter & Hashim, 2001; Geliebter et al., 1992), reduced sensitivity to gastric distention (Zimmerli et al., 2006), delayed gastric emptying (Devlin et al., 1997; Geliebter et al., 1992), and diminished gastric relaxation reflex (Walsh et al., 2003). BED patients also had greater gastric capacities (Geliebter et al., 2004), as well as lower postprandial ghrelin, but gastric emptying rate was similar to control participants (Geliebter, Gluck, & Hashim, 2005; Geliebter et al., 2004). In addition, BN and BED patients commonly report a variety of upper gastrointestinal symptoms, such as early satiety, bloating, nausea, and upper abdominal discomfort (Cremonini et al., 2009; Koch et al., 1998).

These findings on altered stomach function and dyspeptic symptoms suggest that individuals who binge eat may have disturbances of their gastric motor activity. Stomach motility not only regulates the rates at which nutrients are being processed, but also participates in the development of satiation. A non-invasive measure to evaluate gastric motor activity is the electrogastrogram (EGG), which captures the myoelectric activity of gastric smooth muscles through electrodes attached to the outer abdominal wall. The frequency of the cutaneously recorded EGG signal has been demonstrated to be identical to the frequency of the postprandial stomach contractions and to the frequency of the signal recorded from the serosal surface (Hamilton et al., 1986; Levine, 2005). In healthy humans, the stomach normally contracts approximately three times every minute; the EGG signal that reflects this normal gastric activity is generally referred to as 3 cycles per minute (cpm).

There are only few studies, which have investigated gastric myoelectrical activity in eating disordered patients. Ogawa et al. (2004) recorded the EGG from 36 patients with eating disorders before and after water ingestion. The percentage of normal 3 cpm gastric activity was significantly smaller for patients than for healthy controls. Diamanti et al. (2003) found that adolescents with BN displayed abnormal gastric activity, whereas patients with anorexia nervosa did not. Altered gastric myoelectrical activity in BN was also reported by Koch et al. (1998). These results suggest that gastric motility alterations could play a role in the development and/or maintenance of binge eating. Up to date, however, no study has directly investigated the influence of repeated binge eating with and without purging behaviours on gastric myoelectrical activity.

As gastric distention plays a major role in the regulation of food intake, and because individuals who binge eat seem to demonstrate a blunted development of satiation, several studies have investigated the perception of stomach fullness in BN and BED. Most of these studies were performed using invasive barostat measures, which are cumbersome and unpleasant techniques with limited ecological validity (Andresen, 2009; Herbert et al., 2012; Jones et al., 2003). To circumvent such limitations, the Water Load Test (WLT) was developed as a more participant-friendly, non-invasive tool to induce gastric distension using a natural distention stimulus (water) and without the complex hormonal response of a caloric test meal (Andresen, 2009; Koch et al., 2000). During the WLT, participants are typically instructed to drink water until perceiving their stomach to be 'full'. Research in healthy samples and in participants with functional digestive disorders has shown that the WLT is well tolerated, reproducible, and related to invasive, barostat measures (Boeckxstaens et al., 1999; Koch et al., 1998, 2000; Li et al., 2004). Furthermore, the ingested water volume represents a valid indicator for subjective fullness (Jones et al., 2003; Koch et al., 2000).

Recently, the WLT has been adapted to measure sensitivity for eating-related sensations (van Dyck et al., 2015). The new WLT-II is a two-step drink test that measures water intake until eating-related satiation, in addition to the experience of maximum stomach fullness recorded in the initial version. The introduction of an intermediate ‘satiation step’ between start and maximum fullness is based on the following reasoning: While satiation is associated with a positive feeling of satisfaction in healthy individuals (Benelam, 2009), eating disordered or obese patients tend to override these signals and continue to eat until feeling overstuffed (Blundell & Finlayson, 2004). Initial results in a non-clinical sample suggest that these two steps are reliable, distinguishable, and differentially related to the affective quality of gastric sensations and eating disorder symptoms (van Dyck et al., 2015). Specifically, higher volumes ingested until satiation went along with subclinical bulimic symptoms, corroborating the clinical utility of such an additional step in the WLT-II. Moreover, any water load stimulates gastric neuromuscular activity, allowing the measurement of meaningful EGG without posing a caloric threat to participants (Koch & Stern, 2004).

The present study aimed at investigating whether patients with BN or BED show altered perceptions of satiation and stomach fullness, and abnormal gastric electric activity, as assessed by EGG. We also examined differences in eating-related gastric sensations and gastric myoelectrical activity between patients with and without regular purging behaviours (i.e., BN and BED). The results might shed light on gastric interoception and its role in binge eating, overeating and dysfunctional meal termination.

## **METHODS**

### **Participants**

Thirty-two individuals meeting current DSM-5 criteria for either BN or BED and 32 healthy control participants without any previous history of eating disorders were recruited

through announcements in local newspapers, flyers, and at the university campus. Participants from both groups were required to be at least 18 years of age, free from psychotropic medications, and have no history of gastrointestinal illnesses or surgeries. Additional exclusion criteria included bipolar disorder, current or past psychotic disorder or schizophrenia, current suicidal ideation, and physical conditions or treatments known to affect eating or weight. Based on these criteria, three eating disordered individuals were excluded, leaving a final sample of 29 eating disordered (ED) and 32 healthy control (HC) individuals. Of these 29 ED participants, 18 met criteria for BED and 11 for BN.

Telephone screenings were conducted to determine initial eligibility, which was later confirmed via face-to-face structured clinical interview. Lifetime and present eating disorders were assessed using the Eating Disorder Examination (EDE; Fairburn & Cooper, 1993; Hilbert, Tuschen-Caffier, & Ohms, 2004). All other diagnoses were determined using the Structured Clinical Interview for DSM-IV Axis I (SCID; First, Spitzer, Gibbon, & Williams, 1995; Wittchen, Zaudig, & Fydrich, 1997). The study was approved by the Ethics Review Panel of the University of Luxembourg. All participants provided written informed consent and received compensation for their participation.

### **Water Load Test-II**

The WLT-II was performed according to a procedure described in detail elsewhere (van Dyck et al., 2015). In short, participants were asked to drink non-carbonated water at room temperature ad libitum over two consecutive 5-min periods. During the first period, they were instructed to drink water until reaching the point of perceived satiation, that is, the sensation that determines meal termination. During the second period, participants were asked to drink again, this time until reaching the point of maximum stomach fullness. Water was administered in a non-transparent 5-litre flask from which participants drank through a straw to control for swallowing sizes. Unbeknownst to the participants, the flask was filled with

only 1.5 litres of water. This procedure blinded participants to the amount they consumed while at the same time ensuring safety through the 1.5 litres maximum. The volumes consumed during both drinking periods were recorded unobtrusively. Absolute amounts of water ingested in millilitres and the percentage of satiation to maximum fullness were determined as measures of gastric interoception.

## **Questionnaires**

### *WLT-II questionnaire*

The WLT-II questionnaire (van Dyck et al., 2015) assesses subjective sensations related to water ingestion. Participants are asked to concentrate on their current abdominal sensations, especially if their stomach feels full or empty. They then rate their momentary feelings of satiation and fullness, together with five items measuring negative affect (NA). All items are answered on a 7-point scale ranging from 1 (*no sensation/not at all*) to 7 (*extremely*). Ratings are obtained before the first water intake (t0, baseline), and after the first (t1) and second (t2) drinking period. Internal consistency reliabilities for the subscale NA were .71, .77 and .81 for t0, t1 and t2, respectively.

### *Eating Disorder Inventory-2 (EDI-2)*

The EDI-2 (Garner, 1991; German version by Paul & Thiel, 2005) assesses the specific psychopathology of eating disorders. It consists of 91 items, each of which is answered along a scale ranging from 1 (*never*) to 6 (*always*). For the present study, only the subscales drive for thinness, bulimia, and body dissatisfaction are reported, as they assess core eating pathology typical of eating disorders (Garner, 1991). The EDI-2 has been shown to have sound psychometric properties (Eberenz & Gleaves, 1994; Garner, 1991), with internal consistencies ranging between .81 and .91 in different samples ( $\alpha$ 's = .77 - .92 in the current sample). Furthermore, Thiel and Paul (2006) reported test-retest reliabilities between  $r = .81$  and  $r = .89$  for eating disordered participants.

## **Educational level**

As an indicator of socio-economic status, participants' educational level was coded according to the International Standard Classification of Education (ISCED; UNESCO, 1997), to enable valid comparison between degrees obtained in different countries. The ISCED-97 distinguishes between 7 levels of education, ranging from '*not completed primary education*' (= 0) to '*second stage of tertiary education*' (= 6).

## **Electrogastrography**

Gastric myoelectrical activity was measured by EGG in combination with the WLT-II. Participants were tested in the morning, after an overnight fast. The EGG signal was recorded by placing three disposable, ConMed Cleartrace cutaneous electrodes over the region of the antrum of the stomach. The first active electrode was placed on the midpoint between the xiphoid notch and the umbilicus. The second active electrode was placed approximately 6 cm left from the abdominal midline. The reference electrode was placed approximately 10 cm to the right of the midline electrode (Koch & Stern, 2004). Electrode sites were prepared by shaving excess hair, gently abrading the skin (Nuprep, D.O. Weaver and Co., Aurora, CO, USA), and cleaning the area with alcohol. EGG data was recorded on a hard disk with a BrainAmp ExG amplifier (Brain Products, Gilching, Germany). Participants were seated in a half-reclined position at 30 to 45 degrees in a comfortable chair, and were instructed to minimize talking and movement during EGG recording. After signal stabilisation, baseline tracing was performed for 15 minutes. This was followed by the WLT-II. After completion of the drink test, recording continued for an additional 15 minutes.

EGG analysis was performed separately for the two 15-minute measurement segments ('pre' and 'post' water ingestion) using WinCPRS 1.160 software (Absolute Aliens Oy, Turku,

Finland). The raw EGG signal was visually inspected to determine the general quality of the signal and the presence of artefacts. Only continuous artefact-free recordings with visually recognizable waveforms were submitted to computer analysis. The digitalised signal was software-filtered with 0.016 - 0.25 Hz (1 - 15 cpm; Koch & Stern, 2004), before being analysed using fast-Fourier transformations of 240-second runs with an overlap of 75% (Koch & Stern, 2004). Power spectral density was determined for the following electrogastric bands: the bradygastria (1.0 - 2.5 cpm), the normogastria (2.5 - 3.75 cpm); the tachygastria (3.75 - 10.0 cpm) and the duodenal power band (10.1 - 15.0 cpm). Relative power in each EGG band was calculated as percentage of power in the respective frequency band relative to the total EGG band power.

### **Data analysis**

After examination for normality, differences between ED patients and HC participants in demographic characteristics and ingested water volumes were examined using independent samples *t* tests. To evaluate the effect of water ingestion on subjective ratings, we calculated three separate 3 (Time: t0, t1, t2) x 2 (Group: ED, HC) mixed-design analyses of variance (ANOVAs) for each subjective rating (satiety, fullness, NA). To evaluate whether water ingestion caused changes in EGG activity, we conducted four separate 2 (Time: pre, post) x 2 (Group: ED, HC) mixed-design ANOVAs, with the power bands (bradygastria, normogastria, tachygastria, duodenal-respiratory) as dependent variables. Significant effects and interactions were assessed with additional Bonferroni-corrected post hoc independent and paired samples *t* tests. Where appropriate (i.e., Mauchly's Sphericity Test  $p < .05$ ), the Greenhouse-Geisser procedure was applied to correct for potential violations of the sphericity assumption. Pearson correlations were used for calculating correlations between frequency of binge eating and EGG power bands. All results are expressed as mean  $\pm$  standard deviation. A value of  $p < .05$

was considered statistically significant. SPSS version 22.0 (SPSS Inc., Chicago, IL, USA) was used for statistical data analysis.

## RESULTS

### Demographic characteristics

The demographic and clinical details of both groups are reported in Table 1. ED and HC participants did not differ in age, sex or BMI. ED participants had lower educational levels and reported significantly higher drive for thinness and bulimia compared to HC subjects. Groups did not differ in body dissatisfaction.

Table 1

#### *Demographic characteristics*

	ED ( <i>n</i> = 29)	HC ( <i>n</i> = 32)	<i>t</i> (59)	<i>p</i>
Age, <i>M</i> ( <i>SD</i> )	39.39 (12.88)	37.33 (13.59)	0.61	.547
Sex, female, <i>n</i> (%)	26 (89.66)	29 (90.63)		
BMI, <i>M</i> ( <i>SD</i> )	30.19 (8.34)	29.02 (8.31)	0.55	.587
Education, <i>M</i> ( <i>SD</i> )	3.62 (1.05)	4.38 (1.16)	-2.66	.010
Duration of illness (years), <i>M</i> ( <i>SD</i> )	13.05 (8.95) <sup>a</sup>	-		
OBE frequency, <i>M</i> ( <i>SD</i> )	2.74 (1.00)	-		
Current comorbid diagnoses, <i>n</i> (%)				
Any	9 (31.0)	2 (6.3)		
Affective disorder	4 (13.8)	1 (3.1)		
Anxiety disorder	2 (6.9)	1 (3.1)		
Substance use disorder	3 (10.3)	0		
EDI, <i>M</i> ( <i>SD</i> )				
Drive for thinness	4.07 (1.15)	2.92 (1.13)	3.92	< .001
Bulimia	3.57 (0.85)	1.67 (0.51)	10.70	< .001
Body dissatisfaction	3.60 (1.19)	3.49 (1.02)	0.41	.685

Note. BMI = body mass index; OBE frequency = mean weekly frequency of objective binge eating during the past 3 months; EDI = eating disorder inventory.

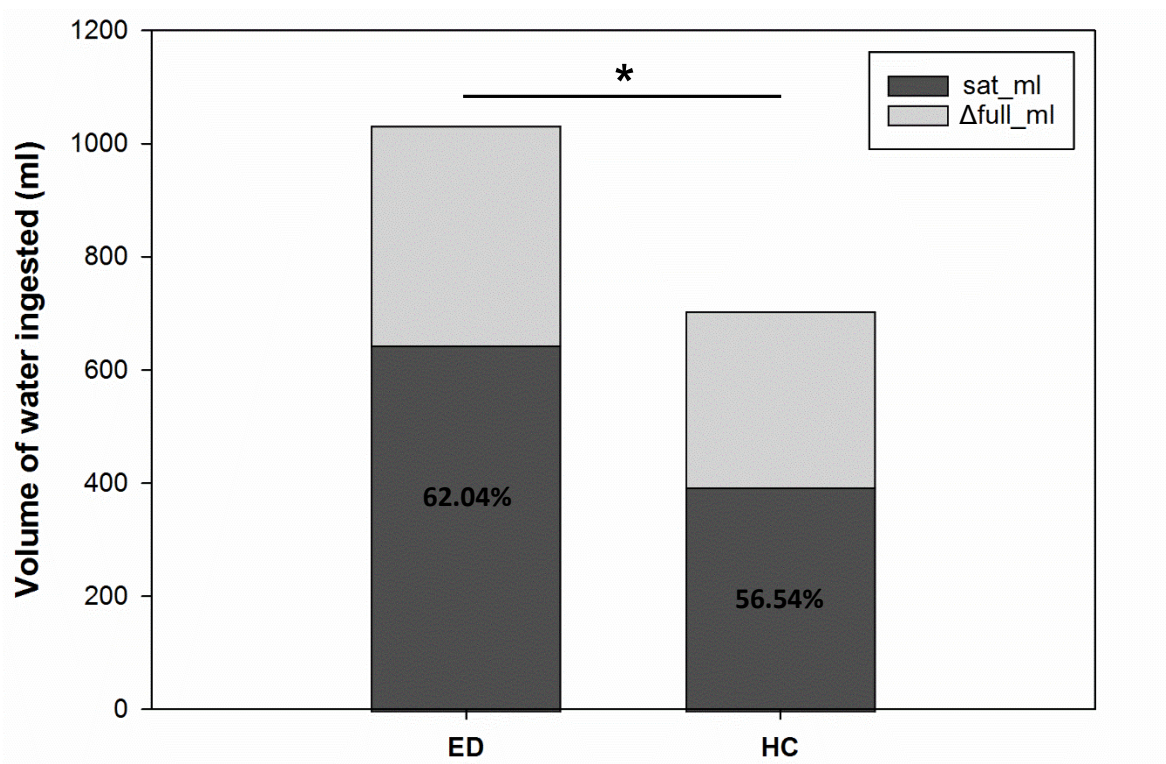
<sup>a</sup> Based on *n* = 28, because data was missing for one participant.



## Ingested water volumes

Mean volumes of water ingested until satiation (sat\_ml), additional water volumes consumed until maximum fullness ( $\Delta$ full\_ml), total water volumes ingested (total\_ml), and mean percentages of satiation relative to total volume (sat\_%) are depicted in Figure 1.

Participants from the ED group and HC individuals differed significantly in sat\_ml ( $645 \pm 311$  ml vs  $395 \pm 169$  ml,  $p < .001$ ) and in total\_ml ( $1031 \pm 414$  ml vs  $705 \pm 273$  ml,  $p = .001$ ), but not in  $\Delta$ full\_ml ( $386 \pm 196$  ml vs  $309 \pm 167$  ml,  $p = .104$ ). ED patients had a higher sat\_% than HC individuals, but this difference failed to reach significance ( $p = .091$ ).



*Figure 1.* Mean volumes of water ingested until satiation and until maximum fullness in eating disordered individuals and in healthy controls. Percentages indicate mean percentages of satiation to total amount of water consumed.

## Subjective ratings

Scores for satiety, fullness, and NA before the water load and after the first and second drinking period are shown in Figure 2. A 3 (Time) x 2 (Group) mixed-design ANOVA on satiety scores revealed significant main effects for Time,  $F(2, 59) = 182.85, p < .001, \eta_p^2 = .76$ , and Group,  $F(1, 59) = 4.85, p = .032, \eta_p^2 = .08$ , but no interaction,  $F(2, 59) = 0.85, p = .432, \eta_p^2 = .01$ . Paired samples  $t$  tests showed that satiety ratings significantly increased from t0 to t1 and from t1 to t2 in both groups ( $ps < .001$ ). The 3 x 2 mixed-design ANOVA on fullness ratings similarly showed significant main effects for Time,  $F(2, 59) = 205.16, p < .001, \eta_p^2 = .78$  and Group,  $F(1, 59) = 4.82, p = .032, \eta_p^2 = .08$ , and no interaction,  $F(2, 59) = 0.06, p = .943, \eta_p^2 = .00$ . As before, paired samples  $t$  tests showed that fullness ratings increased from t0 to t1 and from t1 to t2 in both groups ( $ps < .001$ ). For NA, the 3 x 2 mixed-design ANOVA indicated significant main effects for Time,  $F(1.60, 94.11) = 22.50, p < .001, \eta_p^2 = .28$  and Group,  $F(1, 59) = 8.14, p = .006, \eta_p^2 = .12$ , that were superseded by a significant Time x Group interaction,  $F(1.60, 94.11) = 4.85, p = .015, \eta_p^2 = .08$ . Paired samples  $t$  tests revealed a marginally significant increase in NA from t0 to t1 in the ED group ( $p = .036$ ), but not in HC participants ( $p = .177$ ). NA significantly increased from t1 to t2 in both groups ( $ps < .001$ ). ED participants reported higher NA compared to HC at t1 ( $2.88 \pm 1.11$  vs  $2.09 \pm 0.83, p = .002$ ) and at t2 ( $3.59 \pm 1.43$  vs  $2.63 \pm 0.95, p = .003$ ), but not at baseline ( $2.55 \pm 1.10$  vs  $2.27 \pm 0.92, p = .278$ ).

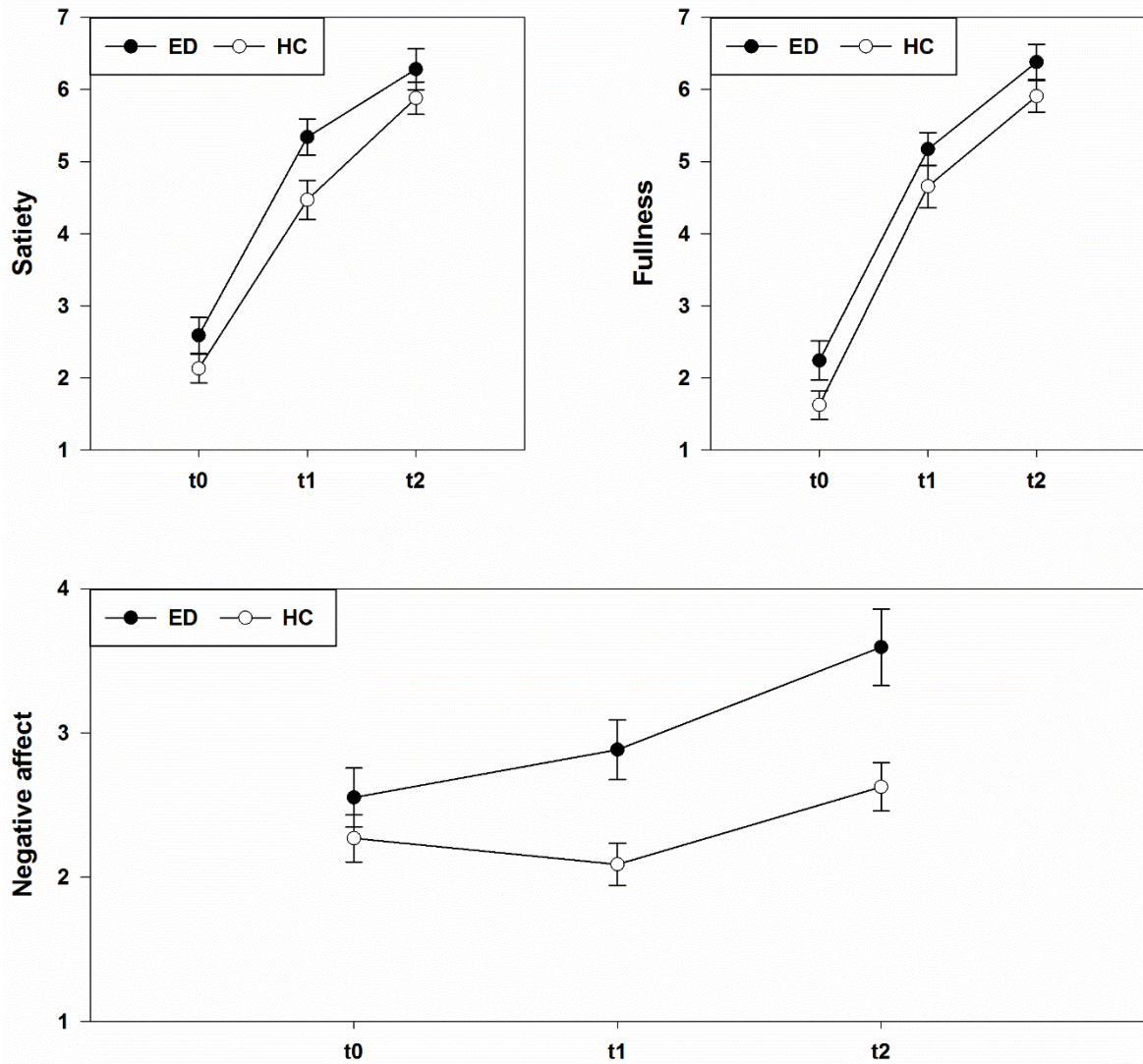


Figure 2. Satiety ratings, fullness ratings, and negative affect before water ingestion, and after the first and second drinking period, in eating disordered patients (ED) and in healthy controls (HC). Figures represent group means. Error bars are  $\pm$  SE.

## Electrogastrographic response to water loads

Due to poor quality of gastric myoelectric signals, data from 9 participants (5 ED and 4 HC) had to be excluded from EGG analysis. Figure 3 shows the mean percentage distributions of total EGG power in the four frequency ranges, for both the ED group ( $n = 24$ ) and the HC group ( $n = 28$ ). The 2 (Time) x 2 (Group) mixed-design ANOVA conducted on percentages of power in the bradygastric range revealed a significant main effect for Group,  $F(1, 50) = 12.98, p < .001, \eta_p^2 = .21$ , but not for Time,  $F(1, 50) = 0.82, p = .370, \eta_p^2 = .02$ . The interaction between time and group was not significant,  $F(1, 50) = 2.02, p = .162, \eta_p^2 = .04$ . Another 2 x 2 mixed-design ANOVA on percentages of normogastric power showed a significant main effect for Group,  $F(1, 50) = 7.93, p = .007, \eta_p^2 = .14$ , and a marginally significant Time x Group interaction,  $F(1, 50) = 4.02, p = .050, \eta_p^2 = .07$ , but no main effect for Time,  $F(1, 50) = 0.00, p = .968, \eta_p^2 = .00$ . Data from the fasting state showed no difference between ED and HC participants ( $36.45 \pm 15.45$  vs  $41.49 \pm 16.52, p = .264$ ), but 2.5 - 3.75 cpm activity was significantly lower in ED compared to HC subjects after water ingestion ( $31.30 \pm 11.55$  vs  $46.45 \pm 18.15, p < .001$ ). The percentages of power in the tachygastric and duodenal-respiratory ranges were similar in each group of participants throughout the study.

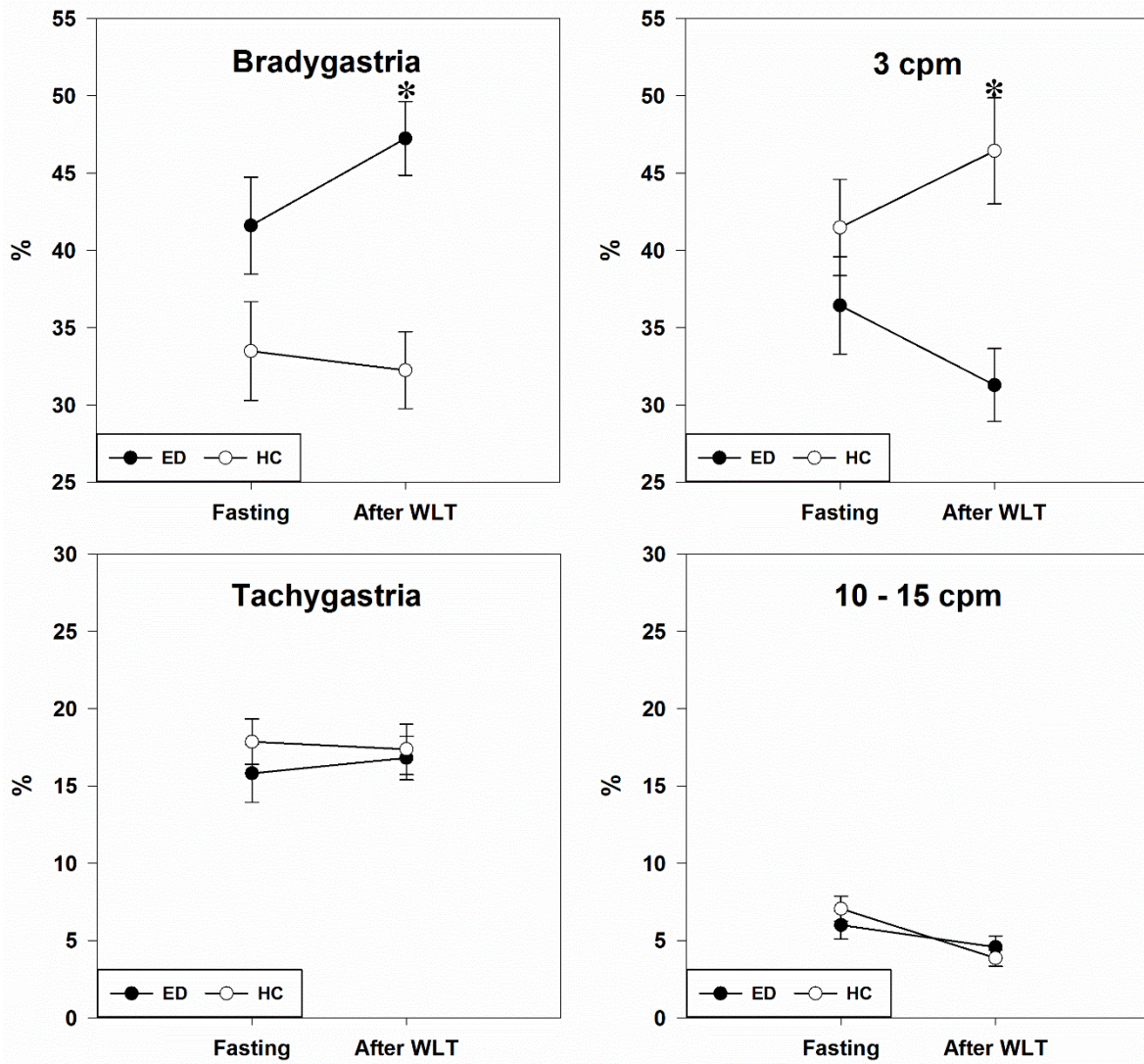


Figure 3. Percentages of total EGG power in the four frequency ranges in eating disordered patients (ED) and in healthy controls (HC), before and after the WLT-II. Figures represent group means. Error bars are  $\pm$  SE. \*  $p < .001$ , ED vs HC.



### Correlations with binge eating frequency in the ED group

OBE frequency was not related to the percentages of EGG power in the four frequency bands before water ingestion (1.0 - 2.5 cpm:  $r = .06$ ,  $p = .768$ ; 2.5 - 3.75 cpm:  $r = .03$ ,  $p = .874$ ; 3.75 - 10.0 cpm:  $r = -.05$ ,  $p = .808$ ; 10.0 - 15.0 cpm:  $r = -.21$ ,  $p = .336$ ). After water ingestion, OBE frequency was moderately negatively related to the percentage of normogastria ( $r = -.48$ ,  $p = .017$ ; see Figure 4). A moderate positive correlation was also found between OBE frequency and percentage of bradygastria after the WLT, although it failed to reach significance ( $r = .35$ ,  $p = .093$ ). OBE frequency was not correlated with percentages of tachygastria or duodenal-respiratory activity relative to total EGG power after the drink test (3.75 - 10.0 cpm:  $r = .11$ ,  $p = .611$ ; 10.0 - 15.0 cpm:  $r = .21$ ,  $p = .321$ ).

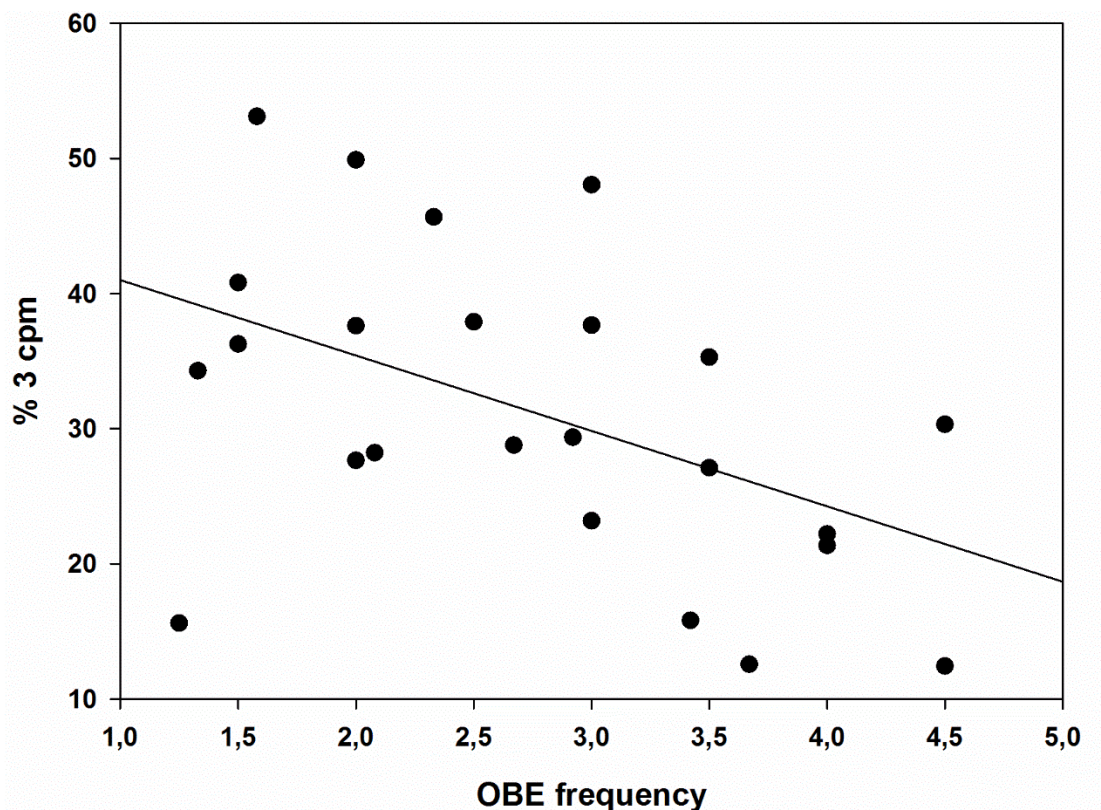


Figure 4. Correlation between the number of objective binge eating episodes within the three months prior to the study and percentage of normogastric activity relative to the total power. OBE = Mean number of objective binge-eating episodes.

### **Between-ED-group analyses**

Additional analyses were performed to examine differences between eating disorder diagnoses (BED vs BN). There were no differences between the two groups in ingested water volumes during the WLT-II, nor were there any group differences in subjective ratings before or after water ingestion. There were also no differences between participants with BN and BED in gastric myoelectrical activity before or after the WLT-II (all  $ps > .05$ ).

### **DISCUSSION**

The aim of the current study was to investigate differences in the onset of satiation and stomach fullness between ED individuals who binge eat and healthy controls, and to assess gastric myoelectrical activity before and after gastric stimulation through water ingestion.

Our first main finding was that ED patients ingested more water until satiated compared to controls, and that the percentage of satiation to maximal fullness was larger in patients than in healthy participants, although this difference failed to reach significance. These findings suggest that individuals experiencing regular binge eating episodes are less responsive to gastric satiation signals and, therefore, may drink beyond this threshold. Indeed, the larger percentage of satiation to maximum fullness in ED patients indicates that satiation was reached at a larger proportion of maximum stomach fullness, that is, they stopped drinking at a point that was closer to their gastric fullness threshold. Our results add to previous evidence suggesting that patients with BN or BED exhibit deficient satiation sensation, possibly contributing to difficulties in meal termination. For example, Sysko et al. (2007) found binge eaters to require substantially more food to reach similar levels of satiation compared to obese and normal-weight controls. In addition, the difference between groups in total water volume ingested until maximum fullness may reflect a larger stomach capacity in ED patients, as suggested by studies using intragastric balloon methods (Geliebter & Hashim, 2001; Geliebter et al., 1992, 2004). Compared to intragastric balloon inflation,

however, the WLT-II is a physiologic and non-invasive test of distensibility of the entire upper gastrointestinal tract, not only the fundus (Koch et al., 2000). Nevertheless, absolute water volume intakes should be interpreted with caution since they are most likely also influenced by other factors than stomach volume, such as sensory and psychological determinants.

Perceptions of stomach fullness and satiety were slightly elevated across the three time points in ED participants compared to the HC group. Thus, although ED patients subjectively felt fuller and more satiated already at baseline, they still ingested larger water volumes compared to healthy controls. Koch and colleagues (1998) also observed increased perceptions of stomach fullness and satiety in BN patients at baseline and after water ingestion, but differences were much larger, although their participants ingested a smaller but fixed amount of water (240 ml). In the present study, self-reported satiety and fullness increased at the same rate in ED and in HC participants, although ED patients ingested larger water volumes and despite the baseline differences in subjective ratings. Thus, ED patients needed larger water volumes to induce the same increase in satiety and fullness perceptions, which is consistent with the idea of an altered development of satiety in individuals who binge eat. In a previous study, Kissileff et al. (1996) also observed comparable changes in subjective fullness over the course of a meal in BN and control subjects, but BN patients consumed a larger amount of food than controls. Hence, the change in fullness per unit of food ingested was lower in BN than in control participants, similar to the present findings with non-caloric water.

Interestingly, our ED patients experienced a significant increase in NA after drinking water until satiated. The NA subscale consists of five items assessing states of discomfort that can be related to food intake (van Dyck et al., 2015). In healthy individuals, satiation reflects a positive, comfortable sensation (Benelam, 2009), which concurs with findings from our



control group. Our ED patients, however, experienced water intake until satiated as uncomfortable, which is consistent with the idea of disturbed sensory responses to intake in BN and BED. In an older study, Turner et al. (1991) also observed that bulimic patients interpreted ingestion of a carbohydrate drink as nauseating rather than as satiating like the control group.

The second main objective of the present study concerned the assessment of gastric myoelectrical activity in ED patients and healthy individuals before and after the WLT-II. Gastric peristalsis is controlled by intrinsic pacesetter potentials or slow waves, which regulate the frequency and propagation of gastric contractions (Koch & Stern, 2004). The normal frequency of the gastric slow waves is approximately 3 cpm or 0.05 Hz in humans. In the postprandial condition, the slow waves are linked to action potentials (the spike potentials), which evoke contractions of the antral muscles. Gastric slow waves and spike potentials are the myoelectrical components of gastric contractile activity that may be non-invasively recorded from electrodes positioned on the skin – the EGG (Brown et al., 1975; Hamilton et al., 1986; Koch & Stern, 2004). In the present study, we used the WLT-II as a non-invasive and highly reproducible method to stimulate the neuromuscular activity of the stomach (Koch & Stern, 2004). The EGG pattern evoked by water ingestion reflects pure neuromuscular activity, without the complex hormonal response of a caloric meal (Koch et al., 2000).

The present study results show significantly increased bradygastria in ED patients compared to controls, primarily after water ingestion, together with significantly less normogastric activity. Mechanoreceptors in the proximal and distal stomach transmit sensory information regarding stomach distention and gastric contractions via afferent vagal activity to the brain (Hellström et al., 2004). Changes in EGG rhythm from normogastria to gastric dysrhythmia (i.e., bradygastria and/or tachygastria) often result in epigastric distress,

especially nausea (Koch & Stern, 2004). Thus, these altered gastric neuromuscular responses to the water load and/or abnormal visceral afferent sensory pathways may be related and contribute to the altered subjective sensations experienced by the patients.

Our EGG findings in BN and BED patients concur with previous studies reporting altered gastric myoelectrical patterns in BN (Diamanti et al., 2003; Koch et al., 1998; Ogawa et al., 2004). Yet, to the best of our knowledge, this is the first study investigating gastric myoelectrical activity in a sample including BED patients. In their study, Ogawa et al. (2004) tested eight patients with a DSM-IV eating disorder not otherwise specified (EDNOS) diagnosis, which might have included some patients with BED. They observed predominant bradygastria in both EDNOS and BN patients and found no significant differences between patient groups. In the present study, no differences were found between participants with a BN and BED diagnosis. This finding suggests that BN and BED patients share gastric rhythm abnormality, and that binge eating (i.e., the excessive stimulation with large volumes of food) might be responsible for the altered gastric myoelectrical responses in BN and BED patients.

We found that the degree of normal myoelectrical activity decreased with the number of binge eating episodes per week. Also, a marginally significant positive association was found between the percentage of bradygastria and OBE frequency. These findings suggest a systematic relationship between the severity of the eating disorder and the extent of disturbances to gastric motor function. Nevertheless, it remains unclear whether the altered gastric myoelectrical activity is responsible for the development of binge eating or simply a result of an adaptive response to repeated overeating.

The present results should be interpreted in combination with findings on other abnormalities in the functioning of the upper gastrointestinal tract observed in BN and BED patients. Several studies have reported that the postprandial release of cholecystokinin (CCK), a gut hormone whose release is an important contributor to satiation (Degen, Matzinger,

Drewe, & Beglinger, 2001), is diminished in patients with BN (Devlin et al., 1997; Geraciotti & Liddle, 1988). In addition, some, but not all, studies on gastric emptying suggest that it is delayed among bulimic patients (Devlin et al., 1997; Geliebter et al., 1992, 2004). Also, Walsh et al. (2003) reported on diminished gastric relaxation reflex in BN patients. This raises the important question of how these abnormalities are related to one another. It has been suggested that the abnormal gastric motor pattern in bulimics might result in, or be coincident with, delayed gastric emptying (Chen, Lin, Pan, & McCallum, 1996; Diamanti et al., 2003). The delayed gastric emptying may in turn result in reduced CCK release (Walsh et al., 2003), which has been demonstrated to affect satiation and enhance gastric relaxation. Thus, a blunted postprandial CCK release in bulimics might be responsible for the diminished satiation and gastric relaxation reflex in response to a meal. Another possibility is that these disturbances in gastrointestinal functioning are manifestations of an underlying physiological disturbance, such as impaired activation of the afferent vagus nerve that carries signals from the gut to higher brain areas (Faris et al., 2000; Wang et al., 2008). This hypothesis is supported by findings on increased pain threshold in BN and BED patients, since somatosensory pain is also transmitted via vagal afferents (de Zwaan, Biener, Bach, Wiesnagrotzki, & Stacher, 1996; Faris et al., 2000; Lautenbacher, Pauls, Strian, Pirke, & Krieg, 1991; Raymond et al., 1995). Overall, these findings support the theory that satiation signals that inhibit ingestion are reduced in patients experiencing regular binge eating episodes.

Nevertheless, most of the aforementioned studies investigating gastrointestinal functioning were restricted to BN patients, while data of this kind from BED patients is still quite limited. While the present results indicate no differences in the experience of satiation and associated physiological mechanism between BN and BED, these null findings are based on rather small samples and it seems premature to draw conclusions.

## **Limitations**

The results of this study need to be viewed with its limitations in mind. First of all, nine participants had to be excluded because of their poor quality EGG signals. This might be due to the high number of overweight or obese participants in our sample, because fat layer under the electrodes acts as an insulator and decreases the amplitude of the EGG signal (Koch & Stern, 2004; Xing & Chen, 2004). However, as the number of participants excluded was almost identical between groups, and as groups were matched for BMI, it is unlikely that this would have affected the results on group differences in EGG patterns. Nevertheless, future studies should take extra caution to ensure the validity of their data when recording EGG from obese individuals.

A second limitation concerns the small proportion of male participants enrolled in this study. We decided to recruit both women and men, because binge eating is common in both sexes (Hudson et al., 2007) and levels of impairment are similar in men and in women (Striegel, Bedrosian, Wang, & Schwartz, 2012), suggesting that men and women with binge eating are comparable and that inclusion of both sexes increases ecological validity. Nevertheless, only few men participated, probably because more women are interested in, and seek treatment for, eating disorders. By rigorously matching groups for sex, however, we ensured that they were comparable. Future research should identify larger and more representative samples that are less likely to be biased by self-selection factors.

Furthermore, the EGG recording periods in the present study were comparatively short (i.e., 15 minutes before and after the WLT-II). In line with our results, Koch et al. (1998) found less normogastria and more bradygastria in BN patients at baseline and during the first 20 minutes after water ingestion. By 21-30 minutes, however, the percentage of normogastric and bradygastric power was equivalent in both groups. Thus, future studies should consider using longer recording periods of at least 30 minutes.

## **Conclusion**

The present results show that BN and BED patients differ from healthy controls with regards to their gastric myoelectrical pattern and water load. Patients had a decreased sensitivity to gastric distention in response to the WLT-II and more gastric dysrhythmias than control participants. These non-invasive measures may be a promising route to elucidating the pathophysiology of eating disorders, and to developing effective therapeutic approaches, as well as monitoring the progress of treatments. Nevertheless, it remains a matter of debate whether impaired intestinal motility is a clinical manifestation of the eating disorder itself or a consequence thereof.

## **4. GENERAL DISCUSSION**

The current research project aimed to investigate two main research questions, both addressing unanswered methodological and theoretical issues in the literature regarding processes underlying overeating and binge eating. More precisely, the present project examined the responsivity to external and internal information and their association with eating behaviour. The four studies presented in this thesis were conducted to investigate the causal role of attentional bias (AB) for eating behaviours, to adapt and develop measures assessing the perception of visceral sensations related to hunger and satiety, and to apply these measures to eating disordered and non-eating disordered samples.

In the first part of this general discussion, the main findings of the present thesis will be briefly summarised. Then the results of the four studies will be integrated, before implications for the treatment of obesity and overeating will be described. After discussing some general strengths and limitations of the present work, we will conclude with some final remarks.

### **4.1. Summary of findings**

#### **4.1.1. Experimental manipulation of attentional bias for food cues: Effects on appetite and the role of contingency awareness (Study 1)**

The primary aim of Study 1 was to investigate whether experimentally enhancing AB towards food cues affects food responsiveness, that is, AB, subjective craving, and unhealthy food choices. Study 1 addressed some unresolved issues and limitations encountered in previous attentional bias modification (ABM) studies, such as choice of the control group, duration of the effects, and the role of contingency awareness. Results show that AB for food cues can be successfully induced using a modified dot probe paradigm. Three to four days after the last training session, participants in the experimental group

showed increased AB for food cues and chose more high-calorie food items compared to the no-bias-induction control group. The attentional training, however, did not affect subjective food craving. These findings are in line with results from meta-analyses (e.g., Beard et al., 2012), suggesting that ABM has a reliable effect on AB and laboratory challenge tasks, but only small effects on motivational states. Additional analyses examined, the role of contingency awareness on training effects. Results showed that training effects on AB and craving differed according to whether or not participants from the ABM group were aware of the experimental contingencies. Although cell sizes were comparatively small and findings will, therefore, need further replication, it is suggested that contingency awareness plays an important role in ABM protocols.

#### **4.1.2. German version of the Intuitive Eating Scale: Psychometric evaluation and application to an eating disordered population (Study 2)**

Study 2 aimed at translating and validating a German version of the second version of the Intuitive Eating Scale (IES-2). Results showed that the proposed factor structure of the questionnaire could be largely confirmed in a large German-speaking sample of women and men. Nevertheless, it should be noted that second-order loadings of the newly introduced ‘body-food choice congruence’ (B-FCC) subscale were low, particularly for the male subsample, suggesting that B-FCC may not be a major part of intuitive eating and that further studies are warranted to investigate whether this component should be kept as part of the construct. Supporting its construct validity, IES-2 scores were negatively related to eating disorder symptomatology, problems in appetite and emotional awareness, body dissatisfaction, focus on outer appearance, and self-objectification. Contrary to some previously published studies (e.g., Gast et al., 2015; Herbert et al., 2013; Madden et al., 2012), no noteworthy correlations were found between IES-2 total and subscale scores and body

mass index (BMI). As a second aim of Study 2, intuitive eating scores were compared between participants with and without self-reported eating disorder diagnoses and between different types of eating disorders. Results showed that women with eating disorders had significantly lower total scores and lower scores on all IES-2 subscales compared to healthy female participants. Furthermore, women with binge-eating disorder (BED) had higher scores on the 'unconditional permission to eat' (UPE) subscale compared to participants with anorexia nervosa (AN) or bulimia nervosa (BN); those diagnosed with AN had higher scores on the 'eating for physical rather than emotional reasons' (EPR) subscale than individuals with BN or BED. Thus, results from Study 2 support the notion of reduced capacities to perceive and use internal bodily sensations to regulate food intake in eating disordered patients.

#### **4.1.3. The Water Load Test as a measure of gastric interoception: Development of a two-stage protocol and application to a healthy population. (Study 3)**

The primary purpose of Study 3 was to establish a standardised, two-step drink test to measure gastric interoception (the WLT-II), and to provide normative data using a non-clinical adult sample. Secondary aims were the development of a short self-report questionnaire assessing subjective sensations related to ingestion and gastric distention, as well as the examination of correlations between WLT-II variables and eating disorder symptoms. The WLT-II consists of several variables: In addition to volumes of water ingested until satiation and maximum fullness expressed in ml, the mean percentage of satiation to maximum fullness is calculated as an individual index of gastric interoception that is not confounded with stomach capacity. We found that both the ingested volume of water required to result in satiation and maximum fullness were significantly negatively related to cardiac accuracy. The WLT-II was well tolerated by all participants and no one refused to drink



during the second period, confirming that satiation and maximum fullness thresholds are well discriminable interoceptive states. Results further demonstrated satisfactory test-retest correlations of ingested water volumes. Consistent with definitions of satiation and maximum fullness, findings on subjective ratings showed increased satiety and fullness ratings as a consequence of water consumption, whereas negative affect (NA) only increased as a consequence of the second drinking period until maximum fullness. Importantly, strong positive correlations were found between satiation volume and percentage of satiation to maximum fullness on the one hand, and bulimic symptoms on the other hand. These results corroborate the suitability of the WLT-II for the investigation of gastric interoception in eating disorder research.

#### **4.1.4. Gastric interoception and gastric myoelectrical activity in bulimia nervosa and binge-eating disorder. (Study 4)**

Study 4 aimed at investigating gastric interoception and gastric myoelectrical activity in BN and BED patients. Results indicated that eating disordered patients show delayed satiation compared to healthy control participants, together with abnormal gastric myoelectrical activity. Eating disordered patients ingested more water until satiated compared to controls. Also, the difference in the percentage of satiation to maximum fullness between patients and healthy participants was marginally significant, with BN and BED patients displaying a larger satiation percentage compared to healthy controls. Results on subjective ratings showed that perceptions of stomach fullness and satiety were slightly elevated in eating disordered participants compared to the healthy control group. Also, eating disordered patients reported significantly increased negative affect after water ingestion until satiated, while no differences were observed in healthy controls. In healthy individuals, satiation reflects a positive, comfortable sensation, which concurs with findings from the control group.

Eating disordered patients, however, experienced water intake until satiated as uncomfortable, which is consistent with the idea of disturbed sensory responses to intake in BN and BED. The second main finding of Study 4 concerns differences in gastric myoelectrical activity between eating disordered patients and healthy controls. Results showed that BN and BED patients exhibited significantly more bradygastria and less normogastria than controls, especially after water ingestion. Furthermore, the degree of normal myoelectrical activity was negatively correlated with the number of binge eating episodes per week, indicating that there may be a systematic relationship between the severity of the eating disorder and the extent of disturbances to gastric motor function.

## **4.2. Integration with the existing literature**

Results from the present project integrate and expand on previous research in a number of ways. During the past two decades, numerous studies have suggested that AB to food cues might be associated with overconsumption, demonstrating a marked AB to food in obese (Castellanos et al., 2009; Nijs et al., 2010; Nummenmaa et al., 2011; Werthmann et al., 2011), bulimic (Blechert, Feige, Joos, Zeeck, & Tuschen-Caffier, 2011; Brooks et al., 2011; Brooks, Prince, Stahl, Campbell, & Treasure, 2011; Shafran, Lee, Cooper, Palmer, & Fairburn, 2007, 2008), and binge-eating disordered individuals (Schmitz et al., 2014; Svaldi et al., 2010). Recently, research has started to investigate if food-related AB could lead to changes in food responsiveness, using ABM tasks. Three studies conducted with undergraduate female students report increased AB for and consumption of high-calorie food in participants trained to attend towards food cues compared to participants trained to avoid food stimuli (Kakoschke et al., 2014; Kemps, Tiggemann, & Elford, 2014; Kemps, Tiggemann, Orr, et al., 2014). Other studies, however, reported no effects of cognitive trainings on food-seeking behaviours (Becker et al., 2015; Hardman et al., 2013). The present

results add to and extend these findings by demonstrating that: (1) ABM training affected AB and food choice in the ABM group compared to a no-bias-induction control group, (2) training effects were maintained three to four days after the last training session, (3) training effects are not equally strong for all outcome measures, (4) these effects of attentional training generalised to novel food stimuli, and (5) the effects of ABM are influenced by participants' awareness of the experimental contingencies.

The present results and findings from previous studies suggest that experimentally changing AB for food cues affects food-seeking behaviour, such as attention to food, choice, and consumption. Training effects on food craving are less pronounced and need further investigation. Indeed, up to now only one study has found an effect of ABM on self-reported craving (Kemps, Tiggemann, Orr, et al., 2014), while all other studies, including the present one, observed no change in craving in response to ABM (Werthmann, Jansen, et al., 2014). These results are in line with the weak effects of ABM on craving reported in a recent meta-analysis (Beard et al., 2012). One explanation might be that all studies conceptualized craving through self-report measures of subjective experience. It is well known that self-reported eating- and weight-related issues tend to be underreported (Dauphinot et al., 2009; Glaesmer & Brähler, 2002; Goris, Westerberp-Plantenga, & Westerberp, 2000). Hence, it is conceivable that people also underreport feelings of hunger or craving, for instance due to feelings of shame or social desirability. It is, therefore, of particular interest for future ABM studies to assess a wider range of different outcomes, including objective behavioural measures and measures from outside the laboratory.

An important finding of Study 1 is that training effects on some outcome measures (here AB and subjective craving) seem to differ according to whether or not participants are aware of the experimental contingencies. Similar results have also been reported in ABM studies from the addiction domain (Attwood et al., 2008; Field et al., 2007). In the discussion

of Study 1, we argued that the increased training effect on AB in aware participants might be explained by the fact that individuals who noticed the ‘rule’ predicting probe location during the training might have used this knowledge to improve their task performance, and continued using this rule during post-test. Consequently, AB itself is probably not the most adequate outcome measure to investigate whether ABM trainings have differential effects for aware and unaware individuals. In addition, methods used to assess contingency awareness differ between studies, which could be accountable for the inconsistent findings. Further research is needed to investigate the role of contingency awareness in ABM trainings, and to establish a standard assessment method.

In summary, inconsistencies in ABM findings seem to be largely the result of methodological differences between studies. ABM protocols differ on crucial issues, such as the number of training sessions (single session vs multiple sessions), the type of stimuli used during the post-test dot probe task (same stimuli than during training vs novel stimuli), the choice of dependent variables, and the method used for assessing contingency awareness. Future experimental studies and meta-analyses should seek to identify the ABM protocol that is most appropriate and effective in changing eating behaviour, and to develop standardised procedures. Although research on AB for food stimuli is complex and challenging, ABM study results present a promising first step in tackling AB to environmental food cues in eating disordered and obese individuals. Attentional retraining could be an effective, feasible, and acceptable way of reducing AB for food and binge eating, thereby improving the success of eating disorder treatments.

While Study 1 addressed some unresolved questions in the literature on AB to external food stimuli, Studies 2-4 investigated the role of detecting and relying on internal bodily signals in food intake regulation. Despite the long-standing and intense interest in interoception, the literature to date remains inconsistent, and methods used to investigate

interoceptive processing have been quite heterogeneous, which makes it difficult to compare study results. Important work has been carried out by Garfinkel and colleagues (Garfinkel & Critchley, 2013; Garfinkel et al., 2015), who proposed a dimensional construct of interoception that distinguishes between different levels of interoception. Amongst others, they introduced the distinction between interoceptive accuracy (i.e., the accurate detection of bodily sensations, as measured by objective tests of interoceptive proficiency) and interoceptive sensibility (i.e., the overall tendency to focus on internal bodily sensations, measured using self-report questionnaires), which are two components that seem to be unrelated and therefore represent distinct processes that should not be conflated (Garfinkel et al., 2015).

In the domain of disordered eating behaviours, the concept of interoception has traditionally included the recognition and accurate identification of both emotional states and internal hunger and satiety signals. This conceptualisation is based on the notion of emotional eating, which corresponds with a major theory on the aetiology of overeating, that is, psychosomatic theory (Bruch, 1973). Psychosomatic theory states that emotional eaters have difficulties to distinguish hunger from the physiological state accompanying negative emotions (Bruch, 1964), and as a result respond by eating when experiencing negative emotions. The interoceptive awareness subscale of the EDI, a widely used measure of self-reported interoceptive processing, assesses both awareness of appetite sensations and awareness of emotions in one and the same subscale. Recent research, however, suggests that sensitivity to appetite sensations and emotional feelings might play separate roles in the development and maintenance of eating disorders and obesity (Brown et al., 2010). For the present research project, we decided to focus on the perception of visceral sensations relating to hunger and satiety.

An important contribution of the present thesis lies in the adaptation and development of two instruments to investigate intuitive eating and gastric interoception. In Study 2, we translated and validated a German version of the IES-2 in a large sample of both men and women. The sample included a large age and BMI range, supporting generalisability and ecological validity of the present results. Importantly, we demonstrated that women with an eating disorder had lower intuitive eating scores compared to healthy participants, which was further supported by the negative correlations found between IES-2 scores and measures of eating disorder symptoms in the entire sample. Furthermore, we found differences in IES-2 subscale scores between women with different eating disorder diagnoses. These results suggest that different components of intuitive eating might be of relevance in AN, BN, and BED patients. Such findings are of utmost importance and this information could even be used in clinical contexts. For example, the information that UPE is decreased in AN and BN, and that EPR seems to be impaired in BN and BED, could be used to adapt treatments and interventions accordingly.

The WLT-II is the second instrument developed in the present research project (Study 3 and Study 4). Because previous studies investigating interoceptive accuracy in the eating domain yielded inconsistent results, and because traditional measures only partially reflect disturbances in the perception of hunger and satiety cues, Study 3 aimed at developing a measure of interoceptive processing in the gastric tract. The WLT-II was developed with the objective of providing a non-invasive, ecologically valid and standardised test of gastric interoception.

The potential importance of the WLT-II in investigating binge eating behaviours was demonstrated by the positive correlations between WLT-II variables and bulimic symptoms (Study 3), as well as differences in ingested water volumes between eating disordered (BN or BED) and healthy participants (Study 4). In Study 3, water volume ingested until satiation and

percentage of satiation to total volume were strongly positively related to bulimic symptoms. Similarly, in Study 4, BN and BED patients ingested more water until satiated compared to controls, and the percentage of satiation to maximal fullness was larger in patients than in healthy participants, although this difference failed to reach significance. Taken together, these results suggest that individuals with binge eating symptoms are less responsive to gastric satiation signals and, therefore, may drink beyond this threshold. This interpretation is corroborated by self-reported subjective sensations in response to drinking periods. In BN and BED patients, drinking until satiated, a feeling that is normally perceived as positive and comfortable, was accompanied by negative subjective sensations, such as discomfort and nausea.

In addition, Study 4 investigated gastric myoelectrical activity before and after water ingestion. It was found that, compared to healthy controls, eating disordered patients exhibited significantly increased bradygastria and decreased 3 cpm normogastria. These differences were particularly prominent after water ingestion. Furthermore, the degree of normal myoelectrical activity was negatively correlated with the number of binge eating episodes per week, indicating that there may be a systematic relationship between the severity of the eating disorder and the extent of disturbances to gastric motor function. Overall, these findings further speak against a role of gastric capacity in the increased water consumption in eating disordered individuals, and in favour of blunted satiation responses. Gastric dysrhythmia may vary in response to changes in gastric volume, and individuals with smaller gastric volumes need less water to develop nausea, a sensation that can be reflected in gastric dysrhythmia (Koch & Stern, 2004). One might argue that, if eating disordered patients had larger gastric volumes than control participants, they would also tolerate larger water volumes until developing gastric dysrhythmia. A resulting hypothetical explanation could be that, if eating disordered patients had stopped drinking when comfortably satiated, irrespective of the

amount of water consumed, no group differences in EGG power bands would have been observed after water ingestion.

Collectively, findings from Studies 2-4 support the notion of altered perception of, and reliance on, internal bodily signals in eating disordered individuals. While Study 2 provides evidence for decreased self-reported intuitive eating scores in eating disordered women, Studies 3 and 4 suggest that individuals experiencing binge eating may show blunted detection of gastric sensations in response to the WLT-II. However, results have been obtained in different study samples and it might be of interest for future research to investigate intuitive eating and gastric interoception within one and the same sample.

### **4.3. Implications for the treatment of obesity and binge eating**

The current findings have implications for the treatment of overeating and binge eating. The literature outlined in the introduction of this work and findings from Study 1 suggest that AB for food could be a cognitive factor that contributes to the development of overeating and binge eating. The learning model of binge eating and obesity states that this enhanced food cue reactivity follows from the systematic association of food cues (e.g., the sight or smell of palatable foods) and the rewarding effects of food intake. This leads to the important question whether it is possible to *break* these learned associations. It has been suggested that food exposure, an intervention that has been introduced in the context of cognitive behaviour therapy, might affect these conditioned responses and reduce or extinguish them (Fairburn, Marcus, & Wilson, 1993; Jansen, 1998). In patients with BN or BED, food exposure usually focuses on the prevention of bingeing. This intervention is based on the idea that, if a (conditioned) food stimulus triggers bingeing and elicits physiological responses such as craving, then repeated or prolonged exposure to this food stimulus in the absence of consumption will result in extinction of craving (Hilbert & Tuschen-Caffier, 2010;



Jansen, 1998). During food exposure, patients are exposed to their personal binge food and prevented from bingeing while they look at, describe, touch, smell, and taste the food. They are, however, not allowed to eat it (Hilbert & Tuschen-Caffier, 2010). Patients rate their actual craving and repeat the exposure again and again, until subjective craving has significantly decreased. Studies on food exposure have generally reported positive outcomes and the procedure seems to be well tolerated (Martinez-Mallén et al., 2007; Toro et al., 2003), suggesting that it might be worthwhile to integrate them into, or offer them in addition to, traditional eating disorder treatments.

Findings from Studies 2-4 suggest that eating disordered patients have difficulties perceiving visceral sensations related to hunger and satiety. Several implications for the treatment of BN or BED might follow from these findings on appetitive abnormalities. Similar to food exposure, these implications have to a large degree already been addressed by current cognitive behaviour therapies. For example, most of these interventions target maladaptive dieting patterns, such as limiting calorie intake, leaving excessive time between meals, and introducing forbidden foods (Fairburn et al., 1993; Hilbert & Tuschen-Caffier, 2010). Patients are required to follow a prescribed pattern of eating, until their hunger and satiety signals are re-established. This pattern of eating also aims at preventing binge eating (Latner & Wilson, 2000).

Another line of interventions that could be proposed is rather derived from awareness trainings, trying to improve the patient's ability to regulate eating through heightened responsivity to internal hunger and satiety cues. The goal of these interventions is to sensitize patients to their feelings of hunger and satiety and encourage them to start and stop eating in response to those feelings, rather than to eat in response to environmental, affective, or cognitive cues (Craighead & Allen, 1995). For example, patients can be instructed to rate their hunger and satiety levels repeatedly before, during, and after a meal (Hilbert & Tuschen-

Caffier, 2010). They are encouraged to consciously focus on their body signals and try to realise when they are satiated. In a similar vein, findings from Study 2 suggest that interventions based on intuitive eating principles may have a positive impact on eating behaviours and more general psychological health outcomes.

#### **4.4. Limitations**

Although the present studies contribute to extant literature, several limitations need to be acknowledged. A variety of alternative or additional measures could be considered to replicate findings and accumulate further evidence to support their theoretical implications. For example, the focus of study 1 was on biasing attention towards unhealthy foods, which is, however, not of direct applied value. Indeed, to establish ABM trainings as a treatment for eating disorders, an equal induction of a bias away from unhealthy foods or towards healthy ones has to be demonstrated. Furthermore, other methods of measuring attentional bias could have been included to investigate whether the effects of our ABM training generalise to these tasks. As described in the introduction, there are numerous tasks available to assess selective attention towards food-related cues, including the modified Stroop, the visual probe task, and measurement of eye movements or event-related potentials. It has been argued that these different tasks might tap into different components of attention (Field et al., 2007), wherefore it would be of interest to examine if ABM training using the visual probe task can influence different measures of attentional bias.

Concerning Studies 3 and 4, other measures of gastric function could be obtained to extend and strengthen the presented results. For example, it would be important to directly measure gastric capacity as a control variable for ingested water volumes. Also, we did not measure gastric emptying, gastric accommodation and gastrointestinal peptide hormones in our participants; therefore, we cannot be certain that the abnormalities that have been

described in the literature for other eating disordered patients would also have been observed in our sample. Moreover, to further validate the WLT-II, it would be of interest to investigate correlations with well-established barostat measures. Although it has been suggested that both measures are related (Boeckxstaens et al., 1999; Li et al., 2004), these findings need to be replicated for the WLT-II.

Lastly, attentional bias and interoception have not been investigated within the same sample, as yet. Therefore, it is impossible to know if enhanced reactivity towards external food stimuli goes along with reduced interoceptive processing within the same person. Further studies should investigate these processes within one and the same study sample.

#### **4.5. Concluding remarks**

Preferential attentional processing of food stimuli reflects probably an evolutionary mechanism to safeguard survival by increasing the chances to restore depleted energy stores. In the context of an environment in which palatable, high-calorie food is omnipresent, such an attentional bias to food may contribute to overeating or obesity. The present research suggests that this motivational regulation of attention, together with a dysfunctional perceptual processing of physiological mechanisms signalling meal termination, are key factors that may contribute to individual differences in susceptibility to overeat and gain weight.

Up to now, most efforts to treat and prevent overeating consisted in promoting healthy diets or divulging guidelines on nutrition. Nevertheless, in view of the unrelenting growth of the obesity epidemic, the efficacy and/or effectiveness of such information-based interventions and their underlying assumptions are questionable. The present findings suggest that more attention should be paid to the positive effects that following one's 'gut feelings' can have on eating behaviour. Individuals with high sensitivity to interoceptive signals may be particularly proficient at utilizing this bodily information to guide behavioural food

choices. After all, hunger and satiety have evolved phylogenetically in order to secure the survival of the human species. Therefore, listening to one's body and following its advice may be a starting point for shaping healthy eating behaviour. In modern Western society, where unhealthy, fattening food is omnipresent, as well as the societal demand for slimness and restricted eating, we seem to have forgotten what hunger and satiety sensations are there for.

## 5. REFERENCES

- Ainley, V., & Tsakiris, M. (2013). Body conscious? Interoceptive awareness, measured by heartbeat perception, is negatively correlated with self-objectification. *PloS One*, *8*(2), e55568. <http://doi.org/10.1371/journal.pone.0055568>
- American Psychiatric Association. (1994). *Diagnostic and Statistical Manual of Mental Disorders (DSM-IV)* (4th ed.). Washington, D.C.: American Psychiatric Association.
- American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders (DSM-5)* (5th ed.). Washington, D.C.: American Psychiatric Association.
- Amir, N., Beard, C., Burns, M., & Bomyea, J. (2009). Attention modification program in individuals with generalized anxiety disorder. *Journal of Abnormal Psychology*, *118*(1), 28–33. <http://doi.org/10.1037/a0012589>
- Andresen, V. (2009). Visceral sensitivity testing. *Best Practice and Research: Clinical Gastroenterology*, *23*(3), 313–324. <http://doi.org/10.1016/j.bpg.2009.04.007>
- Anschutz, D. J., Van Strien, T., & Engels, R. C. M. E. (2008). Exposure to slim images in mass media: Television commercials as reminders of restriction in restrained eaters. *Health Psychology*, *27*(4), 401–408. <http://doi.org/10.1037/2160-4134.1.S.48>
- Ariyasu, H., Takaya, K., Tagami, T., Ogawa, Y., Hosoda, K., Akamizu, T., ... Nakao, K. (2001). Stomach is a major source of circulating ghrelin, and feeding state determines plasma ghrelin-like immunoreactivity levels in humans. *Journal of Clinical Endocrinology and Metabolism*, *86*(10), 4753–4758. <http://doi.org/10.1210/jc.86.10.4753>
- Attwood, A. S., O’Sullivan, H., Leonards, U., Mackintosh, B., & Munafò, M. R. (2008). Attentional bias training and cue reactivity in cigarette smokers. *Addiction*, *103*(11), 1875–1882. <http://doi.org/10.1111/j.1360-0443.2008.02335.x>
- Augustus-Horvath, C. L., & Tylka, T. L. (2011). The acceptance model of intuitive eating: A comparison of women in emerging adulthood, early adulthood, and middle adulthood. *Journal of Counseling Psychology*, *58*(1), 110–125. <http://doi.org/10.1037/a0022129>
- Avalos, L. C., & Tylka, T. L. (2006). Exploring a model of intuitive eating with college women. *Journal of Counseling Psychology*, *53*(4), 486–497. <http://doi.org/10.1037/0022-0167.53.4.486>
- Bacon, L., Keim, N. L., Van Loan, M. D., Derricote, M., Gale, B., Kazaks, A., & Stern, J. S. (2002). Evaluating a “non-diet” wellness intervention for improvement of metabolic fitness, psychological well-being and eating and activity behaviors. *International Journal of Obesity and Related Metabolic Disorders: Journal of the International Association for the Study of Obesity*, *26*(6), 854–865. <http://doi.org/10.1038/sj.ijo.0802012>
- Bacon, L., Stern, J. S., Van Loan, M. D., & Keim, N. L. (2005). Size acceptance and intuitive eating improve health for obese, female chronic dieters. *Journal of the American Dietetic Association*, *105*(6), 929–936. <http://doi.org/10.1016/j.jada.2005.03.011>
- Bargh, J. A., & Chartrand, T. L. (2000). The mind in the middle: A practical guide to priming and automaticity research. In H. T. Reis & C. M. Judd (Eds.), *Handbook of research methods in social and personality psychology* (pp. 253–285). New York: Cambridge University Press.

- Barnes, L. L. B., Harp, D., & Jung, W. S. (2002). Reliability generalization of scores on the Spielberger State-Trait Anxiety Inventory. *Educational and Psychological Measurement*, 62(4), 603–618. <http://doi.org/10.1177/0013164402062004005>
- Batterham, R. L., Cohen, M. A., Ellis, S. M., Le Roux, C. W., Withers, D. J., Frost, G. S., ... Bloom, S. R. (2003). Inhibition of food intake in obese subjects by peptide YY 3–36. *New England Journal of Medicine*, 349(10), 941–948. <http://doi.org/10.1056/NEJMoa030204>
- Batterham, R. L., Le Roux, C. W., Cohen, M. A., Park, A. J., Ellis, S. M., Patterson, M., ... Bloom, S. R. (2003). Pancreatic polypeptide reduces appetite and food intake in humans. *The Journal of Clinical Endocrinology & Metabolism*, 88(8), 3989–3992. <http://doi.org/10.1210/jc.2003-030630>
- Beard, C., Sawyer, A. T., & Hofmann, S. G. (2012). Efficacy of attention bias modification using threat and appetitive stimuli: A meta-analytic review. *Behavior Therapy*, 43(4), 724–740. <http://doi.org/10.1016/j.beth.2012.01.002>
- Becker, D., Jostmann, N. B., Wiers, R. W., & Holland, R. W. (2015). Approach avoidance training in the eating domain: Testing the effectiveness across three single session studies. *Appetite*, 85, 58–65. <http://doi.org/10.1016/j.appet.2014.11.017>
- Benelam, B. (2009). Satiety, satiety and their effects on eating behaviour. *Nutrition Bulletin*, 34(2), 126–173. <http://doi.org/10.1111/j.1467-3010.2009.01753.x>
- Berridge, K. C. (2009). “Liking” and “wanting” food rewards: Brain substrates and roles in eating disorders. *Physiology & Behavior*, 97(5), 537–550. <http://doi.org/10.1016/j.physbeh.2009.02.044>
- Berthoud, H.-R. (2008). The vagus nerve, food intake and obesity. *Regulatory Peptides*, 149(1-3), 15–25. <http://doi.org/10.1016/j.regpep.2007.08.024>
- 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. <http://doi.org/10.1016/j.conb.2011.09.004>
- Birch, L. L., & Fisher, J. O. (2000). Mothers’ child-feeding practices influence daughters’ eating and weight. *American Journal of Clinical Nutrition*, 71(5), 1054–1061.
- Björntorp, P. (2002). Definition and classification of obesity. In C. G. Fairburn & K. D. Brownell (Eds.), *Eating disorders and obesity: A comprehensive handbook* (2nd ed., pp. 377–381). New York: Guilford Press.
- Blechert, J., Feige, B., Joos, A., Zeeck, A., & Tuschen-Caffier, B. (2011). Electrocortical Processing of Food and Emotional Pictures in Anorexia Nervosa and Bulimia Nervosa. *Psychosomatic Medicine*, 73(5), 415–421. <http://doi.org/10.1097/PSY.0b013e318211b871>
- Blechert, J., Meule, A., Busch, N. A., & Ohla, K. (2014). Food-pics: An image database for experimental research on eating and appetite. *Frontiers in Psychology*, 5, 1–10. <http://doi.org/10.3389/fpsyg.2014.00617>
- Blinder, B. J., Cumella, E. J., & Sanathara, V. A. (2000). Psychiatric comorbidities of female inpatients with eating disorders. *Psychosomatic Medicine*, 68(3), 454–462. <http://doi.org/10.1097/01.psy.0000221254.77675.f5>
- Blundell, J. E., & Finlayson, G. (2004). Is susceptibility to weight gain characterized by

- homeostatic or hedonic risk factors for overconsumption? *Physiology and Behavior*, 82(1), 21–25. <http://doi.org/10.1016/j.physbeh.2004.04.021>
- Boeckxstaens, G. E., Hirsch, D. P., Berkhout, B., & Tytgat, G. N. (1999). Is a drink test a valuable tool to study proximal study function? *Gastroenterology*, 116, A960.
- Boeckxstaens, G. E., Hirsch, D. P., van den Elzen, B. D., Heisterkamp, S. H., & Tytgat, G. N. (2001). Impaired drinking capacity in patients with functional dyspepsia: Relationship with proximal stomach function. *Gastroenterology*, 121(5), 1054–1063. <http://doi.org/10.1053/gast.2001.28656>
- Boutelle, K. N., Kuckertz, J. M., Carlson, J., & Amir, N. (2014). A pilot study evaluating a one-session attention modification training to decrease overeating in obese children. *Appetite*, 76, 180–185. <http://doi.org/10.1016/j.appet.2014.01.075>
- Bradley, B. P., Mogg, K., Wright, T., & Field, M. (2003). Attentional bias in drug dependence: Vigilance for cigarette-related cues in smokers. *Psychology of Addictive Behaviors*, 17(1), 66–72. <http://doi.org/10.1037/0893-164X.17.1.66>
- Brener, J., & Kluvitse, C. (1988). Heartbeat detection: Judgments of the simultaneity of external stimuli and heartbeats. *Psychophysiology*, 25(5), 554–561.
- Brignell, C., Griffiths, T., Bradley, B. P., & Mogg, K. (2009). Attentional and approach biases for pictorial food cues. Influence of external eating. *Appetite*, 52(2), 299–306. <http://doi.org/10.1016/j.appet.2008.10.007>
- Brockmeyer, T., Hahn, C., Reetz, C., Schmidt, U., & Friederich, H.-C. (2015). Approach bias modification in food craving – A proof-of-concept study. *European Eating Disorders Review*, 23(5), 352–360. <http://doi.org/10.1002/erv.2382>
- Brooks, S. J., O'Daly, O. G., Uher, R., Friederich, H.-C., Giampietro, V., Brammer, M., ... Campbell, I. C. (2011). Differential neural responses to food images in women with bulimia versus anorexia nervosa. *PloS One*, 6(7), e22259. <http://doi.org/10.1371/journal.pone.0022259>
- Brooks, S., Prince, A., Stahl, D., Campbell, I. C., & Treasure, J. (2011). A systematic review and meta-analysis of cognitive bias to food stimuli in people with disordered eating behaviour. *Clinical Psychology Review*, 31(1), 37–51. <http://doi.org/10.1016/j.cpr.2010.09.006>
- Brown, A. J., Smith, L. T., & Craighead, L. W. (2010). Appetite awareness as a mediator in an eating disorders prevention program. *Eating Disorders*, 18(4), 286–301. <http://doi.org/10.1080/10640266.2010.490118>
- Brown, B. H., Smallwood, R. H., Duthie, H. L., & Stoddard, C. J. (1975). Intestinal smooth muscle electrical potentials recorded from surface electrodes. *Medical & Biological Engineering*, 13(1), 97–103. <http://doi.org/10.1007/bf02478194>
- Browne, M. W., & Cudeck, R. (1993). Alternative ways of assessing model fit. In K. A. Bollen & J. S. Long (Eds.), *Testing structural equation models* (pp. 136–162). Newbury Park, CA: Sage.
- Brownell, K. D. (2002). The environment and obesity. In C. G. Fairburn & K. D. Brownell (Eds.), *Eating disorders and obesity: A comprehensive handbook* (2nd ed., pp. 433–438). New York: Guilford Press.
- Browning, M., Holmes, E. A., Murphy, S. E., Goodwin, G. M., & Harmer, C. J. (2010).

- Lateral prefrontal cortex mediates the cognitive modification of attentional bias. *Biological Psychiatry*, 67(10), 919–925. <http://doi.org/10.1016/j.biopsych.2009.10.031>
- Bruch, H. (1962). Perceptual and conceptual disturbances in anorexia nervosa. *Psychosomatic Medicine*, 24(2), 187–194. <http://doi.org/10.1097/00006254-196210000-00037>
- Bruch, H. (1964). Psychological Aspects of Overeating And Obesity. *Psychosomatics*, 5(5), 269–274. [http://doi.org/10.1016/S0033-3182\(64\)72385-7](http://doi.org/10.1016/S0033-3182(64)72385-7)
- Bruch, H. (1973). *Eating disorders: Obesity, anorexia nervosa, and the person within*. New York: Basic Books.
- Calitri, R., Pothos, E. M., Tapper, K., Brunstrom, J. M., & Rogers, P. J. (2010). Cognitive biases to healthy and unhealthy food words predict change in BMI. *Obesity*, 18(12), 2282–2287. <http://doi.org/10.1038/oby.2010.78>
- Cameron, O. G. (2001). Interoception: The inside story-A model for psychosomatic processes. *Psychosomatic Medicine*, 63(5), 697–710. <http://doi.org/0033-3174/01/6305-0697>
- Cameron, O. G. (2002). *Visceral sensory neuroscience: Interoception*. New York: Oxford University Press.
- Camilleri, G. M., Méjean, C., Bellisle, F., Andreeva, V. A., Sautron, V., Hercberg, S., & Péneau, S. (2015). Cross-cultural validity of the Intuitive Eating Scale-2. Psychometric evaluation in a sample of the general French population. *Appetite*, 84, 34–42. <http://doi.org/10.1016/j.appet.2014.09.009>
- Camilleri, M., Malagelada, J. R., Brown, M. L., Becker, G., & Zinsmeister, A. R. (1985). Relation between antral motility and gastric emptying of solids and liquids in humans. *The American Journal of Physiology*, 249(5 Pt 1), G580–G585.
- Cannon, W. B. (1927). *Bodily changes in pain, hunger, fear and rage: An account of recent researches into the function of emotional excitement*. New York: D. Appleton.
- 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. <http://doi.org/10.1038/ijo.2009.138>
- 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. [http://doi.org/10.1016/S0005-7894\(00\)80009-X](http://doi.org/10.1016/S0005-7894(00)80009-X)
- Chen, J. D., Lin, Z., Pan, J., & McCallum, R. W. (1996). Abnormal gastric myoelectrical activity and delayed gastric emptying in patients with symptoms suggestive of gastroparesis. *Digestive Diseases and Sciences*, 41(8), 1538–1545. <http://doi.org/10.1007/BF02087897>
- Coelho, J. S., Jansen, A., Roefs, A., & Nederkoorn, C. (2009). Eating behavior in response to food-cue exposure: Examining the cue-reactivity and counteractive-control models. *Psychology of Addictive Behaviors: Journal of the Society of Psychologists in Addictive Behaviors*, 23(1), 131–139. <http://doi.org/10.1037/a0013610>
- Cohen, J. (1988). *Statistical power analysis for the behavioral sciences*. (2nd ed.). Hillsdale, NJ: Erlbaum.
- Cohen, J. (1992). A power primer. *Psychological Bulletin*, 112(1), 155–159.



<http://doi.org/10.1037/0033-2909.112.1.155>

- Cole, R. E., & Horacek, T. (2010). Effectiveness of the “My Body Knows When” intuitive-eating pilot program. *American Journal of Health Behavior, 34*(3), 286–297. Retrieved from <http://www.ncbi.nlm.nih.gov/pubmed/20001186>
- Coulie, B., Tack, J., Sifrim, D., Andrioli, A., & Janssens, J. (1999). Role of nitric oxide in fasting gastric fundus tone and in 5-HT 1 receptor-mediated relaxation of gastric fundus. *American Journal of Physiology, 276*, G373–G377.
- Craig, A. D. (2002). How do you feel? Interoception: The sense of the physiological condition of the body. *Nature Reviews. Neuroscience, 3*(8), 655–666. <http://doi.org/10.1038/nrn894>
- Craig, A. D. (2009). How do you feel — now? The anterior insula and human awareness. *Nature Reviews Neuroscience, 10*(1), 59–70. <http://doi.org/10.1038/nrn2555>
- Craighead, L. W., & Allen, H. N. (1995). Appetite awareness training: A cognitive behavioral intervention for binge eating. *Cognitive and Behavioral Practice, 2*, 249–270. Retrieved from <papers2://publication/uuid/556ECFE6-C1FD-427C-8AA9-927E2DB44F2B>
- Craighead, L. W., & Niemeier, H. M. (2002). *The Interoceptive Awareness Scale-Expanded*. Boulder, CO: University of Colorado.
- Cremonini, F., Camilleri, M., Clark, M. M., Beebe, T. J., Locke, G. R., Zinsmeister, A. R., ... Talley, N. J. (2009). Associations among binge eating behavior patterns and gastrointestinal symptoms: A population-based study. *International Journal of Obesity, 33*(3), 342–353. <http://doi.org/10.1038/ijo.2008.272>
- Critchley, H. D., Wiens, S., Rotshtein, P., Ohman, A., & Dolan, R. J. (2004). Neural systems supporting interoceptive awareness. *Nature Neuroscience, 7*(2), 189–195. <http://doi.org/10.1038/nn1176>
- Crow, S. J., Peterson, C. B., Swanson, S. a, Raymond, N. C., Specker, S., Eckert, E. D., & Mitchell, J. E. (2009). Increased mortality in bulimia nervosa and other eating disorders. *The American Journal of Psychiatry, 166*(12), 1342–1346. <http://doi.org/10.1176/appi.ajp.2009.09020247>
- Cummings, D. E., Purnell, J. Q., Frayo, R. S., Schmidova, K., Wisse, B. E., & Weigle, D. S. (2001). A preprandial rise in plasma ghrelin levels suggests a role in meal initiation in humans. *Diabetes, 50*(8), 1714–1719. <http://doi.org/10.2337/diabetes.50.8.1714>
- Damasio, A. R. (1994). *Descartes' error: Emotion, reason, and the human brain*. New York: Avon Books.
- Damasio, A. R. (1996). The somatic marker hypothesis and the possible functions of the prefrontal cortex. *Philosophical Transactions: Biological Sciences, 351*, 1413–1420.
- Damasio, A. R. (1999). *The feeling of what happens: Body and emotion in the making of consciousness*. New York: Harcourt Brace.
- Danner, U. N., Evers, C., Stok, F. M., Van Elburg, A. A., & De Ridder, D. T. D. (2012). A double burden: Emotional eating and lack of cognitive reappraisal in eating disordered women. *European Eating Disorders Review, 20*(6), 490–495. <http://doi.org/10.1002/erv.2184>
- Dauphinot, V., Wolff, H., Naudin, F., Guéguen, R., Sermet, C., Gaspoz, J.-M., & Kossovsky, M. P. (2009). New obesity body mass index threshold for self-reported data. *Journal of*

*Epidemiology and Community Health*, 63(2), 128–132.  
<http://doi.org/10.1136/jech.2008.077800>

- De Graaf, C., Blom, W. A. M., Smeets, P. A. M., Stafleu, A., & Hendriks, H. F. J. (2004). Biomarkers of satiation and satiety. *American Journal of Clinical Nutrition*, 79(6), 946–961.
- de Zwaan, M. (2001). Binge eating disorder and obesity. *International Journal of Obesity and Related Metabolic Disorders: Journal of the International Association for the Study of Obesity*, 25(1), 51–55. <http://doi.org/10.1038/sj.ijo.0801699>
- de Zwaan, M., Biener, D., Bach, M., Wiesnagrotzki, S., & Stacher, G. (1996). Pain sensitivity, alexithymia, and depression in patients with eating disorders: Are they related? *Journal of Psychosomatic Research*, 41(1), 65–70. [http://doi.org/10.1016/0022-3999\(96\)00088-8](http://doi.org/10.1016/0022-3999(96)00088-8)
- Degen, L., Matzinger, D., Drewe, J., & Beglinger, C. (2001). The effect of cholecystokinin in controlling appetite and food intake in humans. *Peptides*, 22(8), 1265–1269. [http://doi.org/10.1016/S0196-9781\(01\)00450-8](http://doi.org/10.1016/S0196-9781(01)00450-8)
- Denny, K. N., Loth, K., Eisenberg, M. E., & Neumark-Sztainer, D. (2013). Intuitive eating in young adults. Who is doing it, and how is it related to disordered eating behaviors? *Appetite*, 60(1), 13–19. <http://doi.org/10.1016/j.appet.2012.09.029>
- Derryberry, D., & Reed, M. A. (2002). Anxiety-related attentional biases and their regulation by attentional control. *Journal of Abnormal Psychology*, 111(2), 225–236. <http://doi.org/10.1037/0021-843X.111.2.225>
- Devlin, M. J., Walsh, B. T., Guss, J. L., Kissileff, H. R., Liddle, R. A., & Petkova, E. (1997). Postprandial cholecystokinin release and gastric emptying in patients with bulimia nervosa. *American Journal of Clinical Nutrition*, 65(1), 114–120.
- Diamanti, A., Bracci, F., Gambarara, M., Ciofetta, G. C., Sabbi, T., Ponticelli, A., ... Castro, M. (2003). Gastric electric activity assessed by electrogastrography and gastric emptying scintigraphy in adolescents with eating disorders. *Journal of Pediatric Gastroenterology and Nutrition*, 37(1), 35–41. <http://doi.org/10.1097/00005176-200307000-00006>
- Dingemans, A. E., Bruna, M. J., & Van Furth, E. F. (2002). Binge eating disorder: A review. *International Journal of Obesity*, 26(3), 299–307.
- Domschke, K., Stevens, S., Pfleiderer, B., & Gerlach, A. L. (2010). Interoceptive sensitivity in anxiety and anxiety disorders: An overview and integration of neurobiological findings. *Clinical Psychology Review*, 30(1), 1–11. <http://doi.org/10.1016/j.cpr.2009.08.008>
- Dorland, W. A. N. (2000). *Dorland's illustrated medical dictionary* (29th ed.). Philadelphia, PA: W.B. Saunders Company.
- Drummond, D. C. (2001). Theories of drugs craving, ancient and modern. *Addiction*, 96(1), 33–46. <http://doi.org/10.1080/09652140020016941>
- Dunn, B. D., Dalgleish, T., Ogilvie, A. D., & Lawrence, A. D. (2007). Heartbeat perception in depression. *Behaviour Research and Therapy*, 45(8), 1921–1930. <http://doi.org/10.1016/j.brat.2006.09.008>
- Eberenz, K. P., & Gleaves, D. H. (1994). An examination of the internal consistency and factor structure of the eating disorder inventory-2 in a clinical sample. *The International*

- Journal of Eating Disorders*, 16(4), 371–379. [http://doi.org/10.1002/1098-108X\(199412\)16:43.0.CO;2-W](http://doi.org/10.1002/1098-108X(199412)16:43.0.CO;2-W)
- Ehlers, A., & Breuer, P. (1992). Increased cardiac awareness in panic disorder. *Journal of Abnormal Psychology*, 101(3), 371–382. <http://doi.org/10.1037/0021-843X.101.3.371>
- Ehlers, A., & Breuer, P. (1996). How good are patients with panic disorder at perceiving their heartbeats? *Biological Psychology*, 42(1-2), 165–182. [http://doi.org/10.1016/0301-0511\(95\)05153-8](http://doi.org/10.1016/0301-0511(95)05153-8)
- Eldar, S., Apter, A., Lotan, D., Edgar, K. P., Naim, R., Fox, N. A., ... Bar-Haim, Y. (2012). Attention bias modification treatment for pediatric anxiety disorders: A randomized controlled trial. *American Journal of Psychiatry*, 169(2), 213–220. <http://doi.org/10.1176/appi.ajp.2011.11060886>
- Epstein, L. H. (1995). Application of behavioral economic principles to treatment of childhood obesity. *Obesity Treatment*, 278, 113–119.
- Eshkevari, E., Rieger, E., Musiat, P., & Treasure, J. (2014). An investigation of interoceptive sensitivity in eating disorders using a heartbeat detection task and a self-report measure. *European Eating Disorders Review*, 22(5), 383–388. <http://doi.org/10.1002/erv.2305>
- Eurostat. (2011). *Overweight and obesity - BMI statistics - Statistics explained*. Luxembourg.
- Fabrigar, L. R., Wegener, D. T., MacCallum, R. C., & Strahan, E. J. (1999). Evaluating the use of exploratory factor analysis in psychological research. *Psychological Methods*, 4(3), 272–299. <http://doi.org/10.1037/1082-989X.4.3.272>
- Fadardi, J. S., & Cox, W. M. (2009). Reversing the sequence: Reducing alcohol consumption by overcoming alcohol attentional bias. *Drug and Alcohol Dependence*, 101(3), 137–145. <http://doi.org/10.1016/j.drugalcdep.2008.11.015>
- Fairburn, C. G. (2002). Cognitive-behavioral therapy for bulimia nervosa. In C. G. Fairburn & K. D. Brownell (Eds.), *Eating disorders and obesity: A comprehensive handbook* (2nd ed., pp. 302–307). New York: Guilford Press.
- Fairburn, C. G., & Cooper, Z. (1993). The eating disorder examination. In C. G. Fairburn & G. T. Wilson (Eds.), *Binge eating: Nature, assessment, and treatment* (12th ed., pp. 317–360). New York: Guilford Press.
- 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. Retrieved from <http://archpsyc.jamanetwork.com/article.aspx?articleid=481632>
- Fairburn, C. G., Doll, H. A., Welch, S. L., Hay, P. J., Davies, B. A., & Connor, M. E. O. (1998). Risk factors for binge eating disorder. *Archives of General Psychiatry*, 55(5), 425–432. <http://doi.org/10.1001/55.5.425>
- Fairburn, C. G., & Harrison, P. J. (2003). Eating disorders. *The Lancet*, 361(9355), 407–416. [http://doi.org/10.1016/S0140-6736\(03\)12378-1](http://doi.org/10.1016/S0140-6736(03)12378-1)
- Fairburn, C. G., Marcus, M. D., & Wilson, G. T. (1993). Cognitive-behavioral therapy for binge eating and bulimia nervosa: A comprehensive treatment manual. In C. G. Fairburn & G. T. Wilson (Eds.), *Binge eating. Nature, assessment, and treatment* (pp. 361–404). New York: Guilford Press.
- Faris, P. L., Kim, S. W., Meller, W. H., Goodale, R. L., Oakman, S. a, Hofbauer, R. D., ...

- Hartman, B. K. (2000). Effect of decreasing afferent vagal activity with ondansetron on symptoms of bulimia nervosa: A randomised, double-blind trial. *Lancet*, *355*, 792–797. [http://doi.org/10.1016/S0140-6736\(99\)09062-5](http://doi.org/10.1016/S0140-6736(99)09062-5)
- Fassino, S., Pierò, A., Gramaglia, C., & Abbate-Daga, G. (2004). Clinical, psychopathological and personality correlates of interoceptive awareness in anorexia nervosa, bulimia nervosa and obesity. *Psychopathology*, *37*, 168–174. <http://doi.org/10.1159/000079420>
- Field, A. (2009). *Discovering statistics using SPSS*. Thousand Oaks, CA: Sage.
- Field, M., & Cox, W. M. (2008). Attentional bias in addictive behaviors: A review of its development, causes, and consequences. *Drug and Alcohol Dependence*, *97*(1-2), 1–20. <http://doi.org/10.1016/j.drugalcdep.2008.03.030>
- Field, M., Duka, T., Eastwood, B., Child, R., Santarcangelo, M., & Gayton, M. (2007). Experimental manipulation of attentional biases in heavy drinkers: Do the effects generalise? *Psychopharmacology*, *192*(4), 593–608. <http://doi.org/10.1007/s00213-007-0760-9>
- Field, M., & Eastwood, B. (2005). Experimental manipulation of attentional bias increases the motivation to drink alcohol. *Psychopharmacology*, *183*(3), 350–357. <http://doi.org/10.1007/s00213-005-0202-5>
- Field, M., Mogg, K., & Bradley, B. P. (2006). Attention to drug-related cues in drug abuses and addiction: Component processes. In R. W. Wiers & A. W. Stacy (Eds.), *Handbook of implicit cognition and addiction* (pp. 151–163). Thousand Oaks, CA: Sage Publications.
- First, M. B., Spitzer, R. L., Gibbon, M., & Williams, J. B. W. (1995). *Structured clinical interview for DSM-IV Axis I disorders*. Washington, D.C.: American Psychiatric Press.
- Flint, A., Raben, A., Ersbøll, A. K., Holst, J. J., & Astrup, A. (2001). The effect of physiological levels of glucagon-like peptide-1 on appetite, gastric emptying, energy and substrate metabolism in obesity. *International Journal of Obesity and Related Metabolic Disorders: Journal of the International Association for the Study of Obesity*, *25*(6), 781–792. <http://doi.org/10.1038/sj.ijo.0801627>
- Franken, I. H. A. (2003). Drug craving and addiction: Integrating psychological and neuropsychopharmacological approaches. *Progress in Neuro-Psychopharmacology and Biological Psychiatry*, *27*(4), 563–579. [http://doi.org/10.1016/S0278-5846\(03\)00081-2](http://doi.org/10.1016/S0278-5846(03)00081-2)
- Fredrickson, B. L., Roberts, T. A., Noll, S. M., Quinn, D. M., & Twenge, J. M. (1998). That swimsuit becomes you: Sex differences in self-objectification, restrained eating, and math performance. *Journal of Personality and Social Psychology*, *75*(1), 269–284. <http://doi.org/10.1037/h0090332>
- Games, P. A., & Howell, J. F. (1976). Pairwise multiple comparison procedures with unequal N's and/or variances: A Monte Carlo study. *Journal of Educational and Behavioral Statistics*, *1*(2), 113–125. <http://doi.org/10.3102/10769986001002113>
- Garfinkel, P. E., Moldofsky, H., Garner, D. M., Stancer, H. C., & Coscina, D. V. (1978). Body awareness in anorexia nervosa: disturbances in “body image” and “satiety.” *Psychosomatic Medicine*, *40*(6), 487–498.
- Garfinkel, S. N., & Critchley, H. D. (2013). Interoception, emotion and brain: new insights link internal physiology to social behaviour. Commentary on: “Anterior insular cortex mediates bodily sensibility and social anxiety” by Terasawa et al. (2012). *Social Cognitive and Affective Neuroscience*, *8*(3), 231–4. <http://doi.org/10.1093/scan/nss140>

- Garfinkel, S. N., Seth, A. K., Barrett, A. B., Suzuki, K., & Critchley, H. D. (2015). Knowing your own heart: Distinguishing interoceptive accuracy from interoceptive awareness. *Biological Psychology, 104*, 65–74. <http://doi.org/10.1016/j.biopsycho.2014.11.004>
- Garner, D. M. (1991). *Eating Disorders Inventory 2: Professional manual*. FL: Psycho.
- Garner, D. M., Olmsted, M. P., & Polivy, J. (1983). Development and validation of a multidimensional eating disorders inventory for anorexia nervosa and bulimia. *International Journal of Eating Disorders, 2*(2), 15–34.
- Gast, J., Campbell Nielson, A., Hunt, A., & Leiker, J. J. (2015). Intuitive eating: Associations with physical activity motivation and BMI. *American Journal of Health Promotion, 29*(3), 91–99. <http://doi.org/10.4278/ajhp.130305-QUAN-97>
- Gast, J., & Hawks, S. R. (1998). Weight loss education: The challenge of a new paradigm. *Health Education & Behavior, 25*(4), 464–473. <http://doi.org/10.1177/109019819802500405>
- Geliebter, A. (1988). Gastric distension and gastric capacity in relation to food intake in humans. *Physiology & Behavior, 44*(4-5), 665–668. [http://doi.org/10.1016/0031-9384\(88\)90333-2](http://doi.org/10.1016/0031-9384(88)90333-2)
- Geliebter, A., Gluck, M. E., & Hashim, S. A. (2005). Plasma ghrelin concentrations are lower in binge-eating disorder. *The Journal of Nutrition, 135*(5), 1326–1330. <http://doi.org/10.1093/ajph.135.5.1326> [pii]
- Geliebter, A., & Hashim, S. A. (2001). Gastric capacity in normal, obese, and bulimic women. *Physiology & Behavior, 74*(4-5), 743–746. [http://doi.org/10.1016/S0031-9384\(01\)00619-9](http://doi.org/10.1016/S0031-9384(01)00619-9)
- Geliebter, A., Melton, P. M., McCray, R. S., Gallagher, D. R., Gage, D., & Hashim, S. A. (1992). Gastric capacity, gastric emptying, and test-meal intake in normal and bulimic women. *American Journal of Clinical Nutrition, 56*(4), 656–661.
- Geliebter, A., Schachter, S., Lohmann-Walter, C., Feldman, H., & Hashim, S. A. (1996). Reduced stomach capacity in obese subjects after dieting. *The American Journal of Clinical Nutrition, 63*(2), 170–173. Retrieved from <http://www.ncbi.nlm.nih.gov/pubmed/11707540>
- Geliebter, A., Yahav, E. K., Gluck, M. E., & Hashim, S. A. (2004). Gastric capacity, test meal intake, and appetitive hormones in binge eating disorder. *Physiology and Behavior, 81*(5), 735–740. <http://doi.org/10.1016/j.physbeh.2004.04.014>
- Geraciotti, T. D., & Liddle, R. A. (1988). Impaired cholecystokinin secretion in bulimia nervosa. *New England Journal of Medicine, 319*(11), 683–688. <http://doi.org/10.1056/NEJM198809153191105>
- Giel, K. E., Friederich, H.-C., Teufel, M., Hautzinger, M., Enck, P., & Zipfel, S. (2011). Attentional processing of food pictures in individuals with anorexia nervosa-An eye-tracking study. *Biological Psychiatry, 69*(7), 661–7. <http://doi.org/10.1016/j.biopsycho.2010.09.047>
- Glaesmer, H., & Brähler, E. (2002). Schätzung der Prävalenz von Übergewicht und Adipositas auf der Grundlage subjektiver Daten zum Body-Mass-Index (BMI). *Gesundheitswesen, 64*(3), 133–138. <http://doi.org/10.1055/s-2002-22317>
- Glass, G. V., & Hopkins, K. D. (1996). *Statistical methods in education and psychology* (3rd.

- ed.). Boston: Allyn & Bacon.
- Goetze, O., Steingoetter, A., Menne, D., van der Voort, I. R., Kwiatek, M. A., Boesiger, P., ... Schwizer, W. (2007). The effect of macronutrients on gastric volume responses and gastric emptying in humans: A magnetic resonance imaging study. *American Journal of Physiology. Gastrointestinal and Liver Physiology*, 292(1), G11–G17. <http://doi.org/10.1152/ajpgi.00498.2005>
- Goris, A. H. C., Westerterp-Plantenga, M. S., & Westerterp, K. R. (2000). Undereating and underrecording of habitual food intake in obese men: Selective underreporting of fat intake. *American Journal of Clinical Nutrition*, 71(1), 130–134.
- Graham, R., Hoover, A., Ceballos, N. A., & Komogortsev, O. (2011). Body mass index moderates gaze orienting biases and pupil diameter to high and low calorie food images. *Appetite*, 56(3), 577–586. <http://doi.org/10.1016/j.appet.2011.01.029>
- Grilo, C. M., Ivezaj, V., & White, M. A. (2015). Evaluation of the DSM-5 severity indicator for binge eating disorder in a community sample. *Behaviour Research and Therapy*, 66, 72–76. <http://doi.org/10.1016/j.brat.2015.01.004>
- Grunert, S. C. (1989). Ein Inventar zur Erfassung von Selbstaussagen zum Ernährungsverhalten. *Diagnostica*, 35(2), 167–179.
- Habermas, T. (2008). Klassifikation und Diagnose: Eine historische Betrachtung. In S. Herpertz, M. de Zwaan, & S. Zipfel (Eds.), *Handbuch Essstörungen und Adipositas* (pp. 4–8). Berlin, Heidelberg: Springer Berlin Heidelberg. <http://doi.org/10.1007/978-3-540-76882-1>
- Hair, J., Black, B., Babin, B., Anderson, R., & Tatham, R. (2006). *Multivariate data analysis* (6th ed.). NJ: Pearson.
- Hakamata, Y., Lissek, S., Bar-Haim, Y., Britton, J. C., Fox, N. A., Leibenluft, E., ... Pine, D. S. (2010). Attention bias modification treatment: A meta-analysis toward the establishment of novel treatment for anxiety. *Biological Psychiatry*, 68(11), 982–990. <http://doi.org/10.1016/j.biopsych.2010.07.021>
- Hamilton, J. W., Bellahsene, B. E., Reichelderfer, M., Webster, J. G., & Bass, P. (1986). Human electrogastragrams. *Digestive Diseases and Sciences*, 31(1), 33–39. <http://doi.org/10.1007/BF01347907>
- Hardman, C. A., Rogers, P. J., Etchells, K. A., Houstoun, K. V. E., & Munafò, M. R. (2013). The effects of food-related attentional bias training on appetite and food intake. *Appetite*, 71, 295–300. <http://doi.org/10.1016/j.appet.2013.08.021>
- Hausken, T., Gilja, O. H., Odegaard, S., & Berstad, A. (1998). Flow across the human pylorus soon after ingestion of food, studied with duplex sonography. Effect of glyceryl trinitrate. *Scandinavian Journal of Gastroenterology*, 33(5), 484–490. <http://doi.org/10.1080/00365529850172034>
- Hawks, S., Madanat, H., Hawks, J., & Harris, A. (2005). The relationship between intuitive eating and health indicators among college women. *American Journal of Health Education*, 36(6), 331. <http://doi.org/10.1080/19325037.2005.10608206>
- Hawks, S., Merrill, R. M., & Madanat, H. N. (2004). The Intuitive Eating Scale: Development and preliminary validation. *American Journal of Health Education*, 35(2), 90–99. <http://doi.org/10.1080/19325037.2004.10603615>

- Hawks, S. R., Madanat, H., Smith, T., & De La Cruz, N. (2008). Classroom approach for managing dietary restraint, negative eating styles, and body image concerns among college women. *Journal of American College Health, 56*(4), 359–366. <http://doi.org/10.3200/JACH.56.44.359-368>
- Heatherton, T. F., Herman, C. P., & Polivy, J. (1991). Effects of physical threat and ego threat on eating behavior. *Journal of Personality and Social Psychology, 60*(1), 138–143. <http://doi.org/10.1037/0022-3514.60.1.138>
- Heatherton, T. F., Herman, C. P., & Polivy, J. (1992). Effects of distress on eating: The importance of ego-involvement. *Journal of Personality and Social Psychology, 62*(5), 801–803. <http://doi.org/10.1037/0022-3514.62.5.801>
- Hellström, P. M., Geliebter, A., Näslund, E., Schmidt, P. T., Yahav, E. K., Hashim, S. A., & Yeomans, M. R. (2004). Peripheral and central signals in the control of eating in normal, obese and binge-eating human subjects. *British Journal of Nutrition, 92*(S1), 47–57. <http://doi.org/10.1079/BJN20041142>
- Herbert, B. M., Blechert, J., Hautzinger, M., Matthias, E., & Herbert, C. (2013). Intuitive eating is associated with interoceptive sensitivity. Effects on body mass index. *Appetite, 70*, 22–30. <http://doi.org/10.1016/j.appet.2013.06.082>
- Herbert, B. M., Muth, E. R., Pollatos, O., & Herbert, C. (2012). Interoception across modalities: On the relationship between cardiac awareness and the sensitivity for gastric functions. *PLoS One, 7*(5), e36646. <http://doi.org/10.1371/journal.pone.0036646>
- Herbert, B. M., Pollatos, O., Flor, H., Enck, P., & Schandry, R. (2010). Cardiac awareness and autonomic cardiac reactivity during emotional picture viewing and mental stress. *Psychophysiology, 47*(2), 342–54. <http://doi.org/10.1111/j.1469-8986.2009.00931.x>
- Herman, C. P. (1974). External and internal cues as determinants of the smoking behavior of light and heavy smokers. *Journal of Personality and Social Psychology, 30*(5), 664–672. <http://doi.org/10.1037/h0037440>
- Herman, C. P., & Mack, D. (1975). Restrained and unrestrained eating. *Journal of Personality, 43*(4), 647–660. Retrieved from <http://www.ncbi.nlm.nih.gov/pubmed/1206453>
- Herman, C. P., & Polivy, J. (1983). A boundary model for the regulation of eating. *Psychiatric Annals, 13*(12), 918–927.
- Herman, C. P., & Polivy, J. (1988). Studies of eating in normal dieters. In B. T. Walsh (Ed.), *Eating behavior in eating disorders* (pp. 95–112). Washington, D.C.: American Psychiatric Association.
- Herman, C. P., & Polivy, J. (2008). External cues in the control of food intake in humans: The sensory-normative distinction. *Physiology & Behavior, 94*(5), 722–728. <http://doi.org/10.1016/j.physbeh.2008.04.014>
- Herman, C. P., Polivy, J., Lank, C. N., & Heatherton, T. F. (1987). Anxiety, hunger, and eating behavior. *Journal of Abnormal Psychology, 96*(3), 264–269. Retrieved from <http://www.ncbi.nlm.nih.gov/pubmed/3680767>
- Hilbert, A., & Tuschen-Caffier, B. (2010). *Essanfälle und Adipositas*. Göttingen: Hogrefe.
- Hilbert, A., Tuschen-Caffier, B., & Ohms, M. (2004). Eating Disorder Examination: Deutschsprachige Version des strukturierten Essstörungeninterviews. *Diagnostica, 50*(2),

98–106.

- Hill, D. M., Craighead, L. W., & Safer, D. L. (2011). Appetite-focused dialectical behavior therapy for the treatment of binge eating with purging: A preliminary trial. *International Journal of Eating Disorders, 44*(3), 249–261. <http://doi.org/10.1002/eat.20812>
- Hill, J. O., & Peters, J. C. (1998). Environmental contributions to the obesity epidemic. *Science, 280*(5368), 1371–1374. <http://doi.org/10.1126/science.280.5368.1371>
- Horn, J. L. (1965). A rationale and test for the number of factors in factor analysis. *Psychometrika, 30*(2), 179–185. <http://doi.org/10.1007/BF02289447>
- Hou, R., Mogg, K., Bradley, B. P., Moss-Morris, R., Peveler, R., & Roefs, A. (2011). External eating, impulsivity and attentional bias to food cues. *Appetite, 56*(2), 424–427. <http://doi.org/10.1016/j.appet.2011.01.019>
- Hu, L., & Bentler, P. M. (1999). Cutoff criteria for fit indexes in covariance structure analysis: Conventional criteria versus new alternatives. *Structural Equation Modeling: A Multidisciplinary Journal, 6*(1), 1–55. <http://doi.org/10.1080/10705519909540118>
- 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. <http://doi.org/10.1016/j.biopsych.2006.03.040>
- Hudson, J. I., Lalonde, J. K., Berry, J. M., Pindyck, L. J., Bulik, C. M., Crow, S. J., ... Pope, H. G. (2006). Binge-eating disorder as a distinct familial phenotype in obese individuals. *Archives of General Psychiatry, 63*, 313–319. <http://doi.org/10.1001/archpsyc.63.3.313>
- Jacobi, C., Hayward, C., de Zwaan, M., Kraemer, H. C., & Agras, W. S. (2004). Coming to Terms With Risk Factors for Eating Disorders: Application of Risk Terminology and Suggestions for a General Taxonomy. *Psychological Bulletin, 130*(1), 19–65. <http://doi.org/10.1037/0033-2909.130.1.19>
- James, W. (1884). What is an emotion? *Mind, 9*, 188–205.
- Jansen, A. (1998). A learning model of binge eating: Cue reactivity and cue exposure. *Behaviour Research and Therapy, 36*(3), 257–272. [http://doi.org/10.1016/S0005-7967\(98\)00055-2](http://doi.org/10.1016/S0005-7967(98)00055-2)
- Janssen, P., Vanden Berghe, P., Verschueren, S., Lehmann, A., Depoortere, I., & Tack, J. (2011). Review article: The role of gastric motility in the control of food intake. *Alimentary Pharmacology & Therapeutics, 33*(8), 880–894. <http://doi.org/10.1111/j.1365-2036.2011.04609.x>
- Johnson, W. G., Rohan, K. J., & Kirk, A. A. (2002). Prevalence and correlates of binge eating in white and African American adolescents. *Eating Behaviors, 3*(2), 179–189. [http://doi.org/10.1016/S1471-0153\(01\)00057-5](http://doi.org/10.1016/S1471-0153(01)00057-5)
- Jones, M. P., Hoffman, S., Shah, D., Patel, K., & Ebert, C. C. (2003). The Water Load Test: Observations from healthy controls and patients with functional dyspepsia. *American Journal of Physiology. Gastrointestinal and Liver Physiology, 284*(6), G896–904. <http://doi.org/10.1152/ajpgi.00361.2002>
- Kakoschke, N., Kemps, E., & Tiggemann, M. (2014). Attentional bias modification encourages healthy eating. *Eating Behaviors, 15*(1), 120–124. <http://doi.org/10.1016/j.eatbeh.2013.11.001>
- Kays, K., Gathercoal, K., & Buhrow, W. (2012). Does survey format influence self-disclosure



- on sensitive question items? *Computers in Human Behavior*, 28(1), 251–256.  
<http://doi.org/10.1016/j.chb.2011.09.007>
- Keel, P. K., & Herzog, D. B. (2004). Long-term outcome, course of illness, and mortality in anorexia nervosa, bulimia nervosa, and binge eating disorder. In T. D. Brewerton (Ed.), *Clinical handbook of eating disorders: An integrated approach* (pp. 97–116). New York: Marcel Dekker, Inc.
- Keel, P. K., Mitchell, J. E., Miller, K. B., Davis, T. L., & Crow, S. J. (1999). Long-term Outcome of Bulimia Nervosa. *Archives of General Psychiatry*, 56(1), 63.  
<http://doi.org/10.1001/archpsyc.56.1.63>
- Kemps, E., Tiggemann, M., & Elford, J. (2014). Sustained effects of attentional re-training on chocolate consumption. *Journal of Behavior Therapy and Experimental Psychiatry*, 49, 1–7. <http://doi.org/10.1016/j.jbtep.2014.12.001>
- Kemps, E., Tiggemann, M., & Hollitt, S. (2014). Biased attentional processing of food cues and modification in obese individuals. *Health Psychology*, 33(11), 1391–1401.  
<http://doi.org/http://dx.doi.org/10.1037/hea0000069>
- Kemps, E., Tiggemann, M., Orr, J., & Grear, J. (2014). Attentional retraining can reduce chocolate consumption. *Journal of Experimental Psychology: Applied*, 20(1), 94–102.  
<http://doi.org/10.1037/xap0000005>
- Kendler, K. S., MacLean, C., Neale, M., Kessler, R., Heath, A., & Eaves, L. (1991). The genetic epidemiology of bulimia nervosa. *The American Journal of Psychiatry*, 148(12), 1627–1637.
- Kieffer, T. J., & Habener, J. F. (2000). The adipoinular axis: Effects of leptin on pancreatic  $\beta$ -cells. *American Journal of Physiology-Endocrinology and Metabolism*, 278(1), E1–E14.
- Kim, D. Y., Camilleri, M., Murray, J. A., Stephens, D. A., Levine, J. A., & Burton, D. D. (2001). Is there a role for gastric accommodation and satiety in asymptomatic obese people? *Obesity Research*, 9(11), 655–661. <http://doi.org/10.1038/oby.2001.89>
- Kissileff, H. R., Walsh, B. T., Kral, J. G., & Cassidy, S. M. (1986). Laboratory studies of eating behavior in women with bulimia. *Physiology & Behavior*, 38(4), 563–570.  
[http://doi.org/10.1016/0031-9384\(86\)90426-9](http://doi.org/10.1016/0031-9384(86)90426-9)
- Kissileff, H. R., Wentzlaff, T. H., Guss, J. L., Walsh, B. T., Devlin, M. J., & Thornton, J. C. (1996). A direct measure of satiety disturbance in patients with bulimia nervosa. *Physiology & Behavior*, 60(4), 1077–1085. Retrieved from <http://www.ncbi.nlm.nih.gov/pubmed/8884936>
- Klabunde, M., Acheson, D. T., Boutelle, K. N., Matthews, S. C., & Kaye, W. H. (2013). Interoceptive sensitivity deficits in women recovered from bulimia nervosa. *Eating Behaviors*, 14(4), 488–492. <http://doi.org/10.1016/j.eatbeh.2013.08.002>
- Knoll, J. F., & Hodapp, V. (1992). A comparison between two methods for assessing heartbeat perception. *Psychophysiology*, 29(2), 218–222.
- Koch, K. L. (2001). Electrogastrography: Physiological basis and clinical application in diabetic gastropathy. *Diabetes Technology & Therapeutics*, 3(1), 51–62.
- Koch, K. L., Bingaman, S., Tan, L., & Stern, R. M. (1998). Visceral perceptions and gastric myoelectrical activity in healthy women and in patients with bulimia nervosa.

- Neurogastroenterol Motil*, 10(1), 3–10. Retrieved from <http://www.ncbi.nlm.nih.gov/pubmed/9507247>
- Koch, K. L., Hong, S. P., & Xu, L. (2000). Reproducibility of gastric myoelectrical activity and the Water Load Test in patients with dysmotility-like dyspepsia symptoms and in control subjects. *Journal of Clinical Gastroenterology*, 31(2), 125–129. <http://doi.org/10.1097/00004836-200009000-00007>
- Koch, K. L., & Stern, R. M. (2004). *Handbook of electrogastronomy*. Oxford: Oxford University Press.
- Kong, M. F., Perkins, A. C., King, P., Blackshaw, P. E., & Macdonald, I. A. (1998). Reproducibility of gastric emptying of a pancake and milkshake meal in normal subjects. *Nuclear Medicine Communications*, 19(1), 77–82. <http://doi.org/10.1097/00006231-199801000-00011>
- Kroese, F. M., Adriaanse, M. A., Evers, C., & De Ridder, D. T. D. (2011). “Instant success”: Turning temptations into cues for goal-directed behavior. *Personality & Social Psychology Bulletin*, 37(10), 1389–1397. <http://doi.org/10.1177/0146167211410889>
- Ladabaum, U., Minoshima, S., Hasler, W. L., Cross, D., Chey, W. D., & Owyang, C. (2001). Gastric distention correlates with activation of multiple cortical and subcortical regions. *Gastroenterology*, 120, 369–376. <http://doi.org/10.1053/gast.2001.21201>
- Ladabaum, U., Roberts, T. P., & McGonigle, D. J. (2007). Gastric fundic distension activates fronto-limbic structures but not primary somatosensory cortex: A functional magnetic resonance imaging study. *NeuroImage*, 34(2), 724–732. [http://doi.org/S1053-8119\(06\)00798-1](http://doi.org/S1053-8119(06)00798-1) [pii]r10.1016/j.neuroimage.2006.07.033
- Lange, C. (1887). *Ueber Gemüthsbewegungen*. Leipzig: Verlag von Theodor Thomas.
- Latner, J. D., & Wilson, G. T. (2000). Cognitive-behavioral therapy and nutritional counseling in the treatment of bulimia nervosa and binge eating. *Eating Behaviors*, 1(1), 3–21. [http://doi.org/10.1016/S1471-0153\(00\)00008-8](http://doi.org/10.1016/S1471-0153(00)00008-8)
- Lautenbacher, S., Pauls, A. M., Strian, F., Pirke, K., & Krieg, J.-C. (1991). Pain sensitivity in anorexia nervosa and bulimia nervosa. *Biological Psychiatry*, 29(11), 1073–1078. [http://doi.org/10.1016/0006-3223\(91\)90249-L](http://doi.org/10.1016/0006-3223(91)90249-L)
- Laux, L., Glanzmann, P., Schaffner, P., & Spielberger, C. D. (1981). *Das State-Trait-Angstinventar (STAI) [The State-Trait Anxiety Inventory]*. Weinheim: Beltz.
- Leiner, D. J. (2014). SoSci Survey. *SoSci Survey (Version 2.5.00-1)*. Retrieved from <http://www.sosicisurvey.com>
- Levine, M. E. (2005). Sickness and satiety: Physiological mechanisms underlying perceptions of nausea and stomach fullness. *Current Gastroenterology Reports*, 7(4), 280–288. <http://doi.org/10.1007/s11894-005-0020-2>
- Li, Q., Zhu, L., & Hou, X. (2004). The clinical study of water loading in gastric sensation thresholds testing. *Zhonghua Nei Ke Za Zhi [Chinese Journal of Internal Medicine]*, 43(6), 436–438.
- Lilenfeld, L. R. R., Wonderlich, S., Riso, L. P., Crosby, R., & Mitchell, J. (2006). Eating disorders and personality: A methodological and empirical review. *Clinical Psychology Review*, 26(3), 299–320. <http://doi.org/10.1016/j.cpr.2005.10.003>
- Long, C. G., Hinton, C., & Gillespie, N. K. (1994). Selective processing of food and body size

- words: Application of the Stroop Test with obese restrained eaters, anorexics, and normals. *The International Journal of Eating Disorders*, 15(3), 279–283.
- López-Guimerà, G., Neumark-Sztainer, D., Hannan, P., Fauquet, J., Loth, K., & Sánchez-Carracedo, D. (2013). Unhealthy weight-control behaviours, dieting and weight status: A cross-cultural comparison between North American and Spanish adolescents. *European Eating Disorders Review*, 21(4), 276–283. <http://doi.org/10.1002/erv.2206>
- Lowe, M. R., & Levine, A. S. (2005). Eating motives and the controversy over dieting: Eating less than needed versus less than wanted. *Obesity Research*, 13(5), 797–806. <http://doi.org/10.1038/oby.2005.90>
- Lu, C. L., Wu, Y. T., Yeh, T. C., Chen, L. F., Chang, F. Y., Lee, S. D., ... Hsieh, J. C. (2004). Neuronal correlates of gastric pain induced by fundus distension: A 3T-fMRI study. *Neurogastroenterology and Motility*, 16, 575–587. <http://doi.org/10.1111/j.1365-2982.2004.00562.x>
- Lutz, A. P. C., Schulz, A., Voderholzer, U., Koch, S., & Vögele, C. (2015). Interoception in anorexia nervosa: Evidence at cortical and self-report levels. In *Paper presented at 45th Annual EABCT Congress*. Jerusalem, Israel.
- MacLeod, C., Mathews, A., & Tata, P. (1986). Attentional bias in emotional disorders. *Journal of Abnormal Psychology*, 95(1), 15–20.
- MacLeod, C., Rutherford, E., Campbell, L., Ebsworthy, G., & Holker, L. (2002). Selective attention and emotional vulnerability: Assessing the causal basis of their association through the experimental manipulation of attentional bias. *Journal of Abnormal Psychology*, 111(1), 107–123. <http://doi.org/10.1037//0021-843X.111.1.107>
- Madden, C. E., Leong, S. L., Gray, A., & Horwath, C. C. (2012). Eating in response to hunger and satiety signals is related to BMI in a nationwide sample of 1601 mid-age New Zealand women. *Public Health Nutrition*, 15(12), 1–8. <http://doi.org/10.1017/S1368980012000882>
- Maffei, M., Halaas, J., Ravussin, E., Pratley, R. E., Lee, G. H., Zhang, Y., ... Friedman, J. M. (1995). Leptin levels in human and rodent: Measurement of plasma leptin and ob RNA in obese and weight-reduced subjects. *Nature Medicine*, 1(11), 1155–1161. <http://doi.org/10.1038/nm1195-1155>
- Martinez-Mallén, E., Castro-Fornieles, J., Lázaro, L., Moreno, E., Morer, A., Font, E., ... Toro, J. (2007). Cue exposure in the treatment of resistant adolescent bulimia nervosa. *International Journal of Eating Disorders*, 40(7), 596–601. <http://doi.org/10.1002/eat.20423>
- Matsumoto, R., Kitabayashi, Y., Narumoto, J., Wada, Y., Okamoto, A., Ushijima, Y., ... Fukui, K. (2006). Regional cerebral blood flow changes associated with interoceptive awareness in the recovery process of anorexia nervosa. *Progress in Neuro-Psychopharmacology and Biological Psychiatry*, 30(7), 1265–1270. <http://doi.org/10.1016/j.pnpbp.2006.03.042>
- Matsunaga, M. (2010). How to factor-analyze your data right: Do's, don'ts, and how-to's. *International Journal of Psychological Research*, 3(1), 97–110.
- Mayr, A., Gefeller, O., Prokosch, H. U., Pirkl, A., Fröhlich, A., & De Zwaan, M. (2012). Web-based data collection yielded an additional response bias - But had no direct effect on outcome scales. *Journal of Clinical Epidemiology*, 65(9), 970–977.

<http://doi.org/10.1016/j.jclinepi.2012.03.005>

- Mejía-Rivas, M., Remes-Troche, J. M., Montaña-Loza, A., Herrera, M., & Valdovinos-Díaz, M. A. (2009). Gastric capacity is related to body mass index in obese patients. A study using the Water Load Test. *Revista de Gastroenterología de México*, 74(1), 71–73.
- Meule, A., Lutz, A., Vögele, C., & Kübler, A. (2012). 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.  
<http://doi.org/10.1016/j.appet.2011.09.010>
- Meyer, J. H. (1987). Motility of the stomach and gastroduodenal junction. In L. R. Johnson, J. Christensen, E. D. Jacobsen, & S. G. Schultz (Eds.), *Physiology of the gastrointestinal tract* (pp. 613–630). New York: Raven Press.
- Miller, L. C., Murphy, R., & Buss, A. H. (1981). Consciousness of body: Private and public. *Journal of Personality and Social Psychology*, 41(2), 397–406.  
<http://doi.org/10.1037/0022-3514.41.2.397>
- Miner-Rubino, K., Twenge, J. M., & Fredrickson, B. L. (2002). Trait self-objectification in women: Affective and personality correlates. *Journal of Research in Personality*, 36(2), 147–172. <http://doi.org/10.1006/jrpe.2001.2343>
- Mintz, L. B., & Betz, N. E. (1988). Prevalence and correlates of eating disordered behaviors among undergraduate women. *Journal of Counseling Psychology*, 35(4), 463–471.  
<http://doi.org/10.1037/0022-0167.35.4.463>
- Mitchell, N. S., Catenacci, V. A., Wyatt, H. R., & Hill, J. O. (2011). Obesity: Overview of an epidemic. *Psychiatric Clinics of North America*, 34(4), 717–732.  
<http://doi.org/10.1016/j.psc.2011.08.005>
- Mogg, K., Bradley, B., Miles, F., & Dixon, R. (2004). Time course of attentional bias for threat scenes: Testing the vigilance-avoidance hypothesis. *Cognition & Emotion*, 18(5), 689–700. <http://doi.org/10.1080/02699930341000158>
- Mogg, K., Bradley, B. P., De Bono, J., & Painter, M. (1997). Time course of attentional bias for threat information in non-clinical anxiety. *Behaviour Research and Therapy*, 35(4), 297–303. [http://doi.org/10.1016/S0005-7967\(96\)00109-X](http://doi.org/10.1016/S0005-7967(96)00109-X)
- Mogg, K., Bradley, B. P., Hyare, H., & Lee, S. (1998). Selective attention to food-related stimuli in hunger: Are attentional biases specific to emotional and psychopathological states, or are they also found in normal drive states? *Behaviour Research and Therapy*, 36, 227–237.
- Molinari, C., Sabbatini, M., Grossini, E., Mary, D. A. S. G., Cannas, M., & Vacca, G. (2006). Cardiovascular effects and c-Fos expression in the rat hindbrain in response to innocuous stomach distension. *Brain Research Bulletin*, 69(2), 140–146.  
<http://doi.org/10.1016/j.brainresbull.2005.11.014>
- Mond, J. M., & Hay, P. J. (2007). Functional impairment associated with bulimic behaviors in a community sample of men and women. *International Journal of Eating Disorders*, 40(5), 391–398. <http://doi.org/10.1002/eat.20380>
- Montepare, J. M. (2006). Body consciousness across the adult years: Variations with actual and subjective age. *Journal of Adult Development*, 13(2), 102–107.  
<http://doi.org/10.1007/s10804-007-9010-y>

- Muthén, L. K., & Muthén, B. O. (2010). *Mplus User's Guide* (6th ed.). Los Angeles, CA: Muthén & Muthén.
- Naus, M. J., Philipp, L. M., & Samsi, M. (2009). From paper to pixels: A comparison of paper and computer formats in psychological assessment. *Computers in Human Behavior*, 25(1), 1–7. <http://doi.org/10.1016/j.chb.2008.05.012>
- Nijs, I. M. T., & Franken, I. H. A. (2012). Attentional processing of food cues in overweight and obese individuals. *Current Obesity Reports*, 1(2), 106–113. <http://doi.org/10.1007/s13679-012-0011-1>
- Nijs, I. M. T., Franken, I. H. A., & Muris, P. (2009). Enhanced processing of food-related pictures in female external eaters. *Appetite*, 53(3), 376–383. <http://doi.org/10.1016/j.appet.2009.07.022>
- Nijs, I. M. T., Muris, P., Euser, A. S., & Franken, I. H. A. (2010). Differences in attention to food and food intake between overweight/obese and normal-weight females under conditions of hunger and satiety. *Appetite*, 54(2), 243–254. <http://doi.org/10.1016/j.appet.2009.11.004>
- Nisbett, R. E. (1972). Hunger, obesity, and the ventromedial hypothalamus. *Psychological Review*, 79(6), 433–453. <http://doi.org/10.1037/h0033519>
- Nummenmaa, L., Hietanen, J. K., Calvo, M. G., & Hyönä, J. (2011). Food catches the eye but not for everyone: A BMI–contingent attentional bias in rapid detection of nutrients. *PLoS ONE*, 6(5), e19215. <http://doi.org/10.1371/journal.pone.0019215>
- O'Connor, B. P. (2000). SPSS and SAS programs for determining the number of components using parallel analysis and Velicer's MAP test. *Behavior Research Methods, Instruments, & Computers*, 32(3), 396–402. <http://doi.org/10.3758/BF03200807>
- Ogawa, A., Mizuta, I., Fukunaga, T., Takeuchi, N., Honaga, E., Sugita, Y., ... Takeda, M. (2004). Electrogastrography abnormality in eating disorders. *Psychiatry and Clinical Neurosciences*, 58(3), 300–310. <http://doi.org/10.1111/j.1440-1819.2004.01236.x>
- Olofsson, J. K., Nordin, S., Sequeira, H., & Polich, J. (2008). Affective picture processing: An integrative review of ERP findings. *Biological Psychology*, 77(3), 247–265. <http://doi.org/10.1016/j.biopsycho.2007.11.006>
- Ouweland, C., & Papiés, E. K. (2010). Eat it or beat it. The differential effects of food temptations on overweight and normal-weight restrained eaters. *Appetite*, 55(1), 56–60. <http://doi.org/10.1016/j.appet.2010.04.009>
- Papiés, E. K., & Hamstra, P. (2010). Goal priming and eating behavior: Enhancing self-regulation by environmental cues. *Health Psychology*, 29(4), 384–388. <http://doi.org/10.1037/a0019877>
- Papiés, E. K., Stroebe, W., & Aarts, H. (2008). The allure of forbidden food: On the role of attention in self-regulation. *Journal of Experimental Social Psychology*, 44(5), 1283–1292. <http://doi.org/10.1016/j.jesp.2008.04.008>
- Paul, T., & Thiel, A. (2005). *EDI-2. Eating Disorder Inventory-2. Deutsche Version*. Göttingen: Hogrefe.
- Pennebaker, J. W. (1982). *The psychology of physical symptoms*. New York: Springer.
- Phaf, R. H., & Kan, K. J. (2007). The automaticity of emotional Stroop: A meta-analysis. *Journal of Behavior Therapy and Experimental Psychiatry*, 38(2), 184–199.

<http://doi.org/10.1016/j.jbtep.2006.10.008>

- Phillips, R. J., & Powley, T. L. (2000). Tension and stretch receptors in gastrointestinal smooth muscle: Re-evaluating vagal mechanoreceptor electrophysiology. *Brain Research Reviews*, *34*(1-2), 1–26. <http://doi.org/S0165017300000369> [pii]
- Polivy, J., & Herman, C. P. (1985). Dieting and bingeing. A causal analysis. *The American Psychologist*, *40*(2), 193–201. Retrieved from <http://www.ncbi.nlm.nih.gov/pubmed/3857016>
- Polivy, J., & Herman, C. P. (1999). Distress and eating: Why do dieters overeat? *International Journal of Eating Disorders*, *26*(2), 153–164. [http://doi.org/10.1002/\(SICI\)1098-108X\(199909\)26:23.0.CO;2-R](http://doi.org/10.1002/(SICI)1098-108X(199909)26:23.0.CO;2-R)
- Polivy, J., & Herman, C. P. (2002). Causes of eating disorders. *Annual Review of Psychology*, *53*, 187–213. <http://doi.org/10.1146/annurev.psych.53.100901.135103>
- Pollatos, O., Herbert, B. M., Wankner, S., Dietel, A., Wachsmuth, C., Henningsen, P., & Sack, M. (2011). Autonomic imbalance is associated with reduced facial recognition in somatoform disorders. *Journal of Psychosomatic Research*, *71*(4), 232–239. <http://doi.org/10.1016/j.jpsychores.2011.03.012>
- Pollatos, O., Kirsch, W., & Schandry, R. (2005). Brain structures involved in interoceptive awareness and cardioafferent signal processing: A dipole source localization study. *Human Brain Mapping*, *26*(1), 54–64. <http://doi.org/10.1002/hbm.20121>
- Pollatos, O., Kurz, A.-L., Albrecht, J., Schreder, T., Kleemann, A. M., Schöpf, V., ... Schandry, R. (2008). Reduced perception of bodily signals in anorexia nervosa. *Eating Behaviors*, *9*(4), 381–388. <http://doi.org/10.1016/j.eatbeh.2008.02.001>
- Pollatos, O., Schandry, R., Auer, D. P., & Kaufmann, C. (2007). Brain structures mediating cardiovascular arousal and interoceptive awareness. *Brain Research*, *1141*, 178–87. <http://doi.org/10.1016/j.brainres.2007.01.026>
- Poppitt, S. D., & Prentice, A. M. (1996). Energy density and its role in the control of food intake: Evidence from metabolic and community studies. *Appetite*, *26*(2), 153–174. [http://doi.org/S0195-6663\(96\)90013-6](http://doi.org/S0195-6663(96)90013-6) [pii] \n10.1006/appe.1996.0013
- Powley, T. L., & Phillips, R. J. (2004). Gastric satiation is volumetric, intestinal satiation is nutritive. *Physiology and Behavior*, *82*(1), 69–74. <http://doi.org/10.1016/j.physbeh.2004.04.037>
- Raymond, N. C., de Zwaan, M., Faris, P. L., Nugent, S. M., Ackard, D. M., Crosby, R. D., & Mitchell, J. E. (1995). Pain thresholds in obese binge-eating disorder subjects. *Biological Psychiatry*, *37*(3), 202–204. Retrieved from <http://www.ncbi.nlm.nih.gov/pubmed/7727630>
- Raymond, N. C., Mussell, M. P., Mitchell, J. E., de Zwaan, M., & Crosby, R. D. (1995). An age-matched comparison of subjects with binge eating disorder and bulimia nervosa. *The International Journal of Eating Disorders*, *18*(2), 135–143. Retrieved from <http://www.ncbi.nlm.nih.gov/pubmed/7581415>
- Robinson, E., Kersbergen, I., Brunstrom, J. M., & Field, M. (2014). I'm watching you. Awareness that food consumption is being monitored is a demand characteristic in eating-behaviour experiments. *Appetite*, *83*, 19–25. <http://doi.org/10.1016/j.appet.2014.07.029>

- Robinson, T. E., & Berridge, K. C. (1993). The neural basis of drug craving: An incentive-sensitization theory of addiction. *Brain Research Reviews*, *18*(3), 247–291. [http://doi.org/10.1016/0165-0173\(93\)90013-P](http://doi.org/10.1016/0165-0173(93)90013-P)
- Rodin, J. (1981). Current status of the internal-external hypothesis for obesity: What went wrong? *American Psychologist*, *36*(4), 361–372. <http://doi.org/10.1037/0003-066X.36.4.361>
- Rogers, C. R. (1961). *On becoming a person*. Boston, MA: Houghton Mifflin.
- Roland, J., Dobbeleir, A., Vandevivere, J., & Ham, H. R. (1990). Evaluation of reproducibility of solid-phase gastric emptying in healthy subjects. *European Journal of Nuclear Medicine*, *17*(3-4), 130–133. <http://doi.org/10.1007/BF00811440>
- Rolls, B. J., Fedoroff, I. C., & Guthrie, J. F. (1991). Gender differences in eating behavior and body weight regulation. *Health Psychology*, *10*(2), 133–142. <http://doi.org/10.1037/0278-6133.10.2.133>
- Rolls, E. T. (2011). Taste, olfactory and food texture reward processing in the brain and obesity. *International Journal of Obesity*, *35*(4), 550–561. <http://doi.org/10.1038/ijo.2010.155>
- Ross, H. E., & Ivis, F. (1999). Binge eating and substance use among male and female adolescents. *International Journal of Eating Disorders*, *26*, 245–260. [http://doi.org/10.1002/\(SICI\)1098-108X\(199911\)26:3<245::AID-EAT2>3.0.CO;2-R](http://doi.org/10.1002/(SICI)1098-108X(199911)26:3<245::AID-EAT2>3.0.CO;2-R)
- Ryan, F. (2002). Detected, selected, and sometimes neglected: Cognitive processing of cues in addiction. *Experimental and Clinical Psychopharmacology*, *10*(2), 67–76. <http://doi.org/10.1037/1064-1297.10.2.67>
- Saules, K. K., Collings, A. S., Hoodin, F., Angelella, N. E., Alschuler, K., Ivezaj, V., ... Wiedemann, A. A. (2009). The contributions of weight problem perception, BMI, gender, mood, and smoking status to binge eating among college students. *Eating Behaviors*, *10*(1), 1–9. <http://doi.org/10.1016/j.eatbeh.2008.07.010>
- Schachter, S. (1971). Some extraordinary facts about obese humans and rats. *The American Psychologist*, *26*(2), 129–144. <http://doi.org/10.1037/h0030817>
- Schachter, S., & Singer, J. (1962). Cognitive, social, and physiological determinants of emotional state. *Psychological Review*, *69*(5), 379–399. <http://doi.org/10.1037/h0046234>
- Schaefer, J. T. (2015). *Intuitive eating: Expanding the research & describing the state of practice*. Kent State University.
- Schandry, R. (1981). Heart beat perception and emotional experience. *Psychophysiology*, *18*(4), 483–488. Retrieved from <http://www.ncbi.nlm.nih.gov/pubmed/7267933>
- Schmitz, F., Naumann, E., Trentowska, M., & Svaldi, J. (2014). Attentional bias for food cues in binge eating disorder. *Appetite*, *80*, 70–80. <http://doi.org/10.1016/j.appet.2014.04.023>
- Schoenmakers, T. M., de Bruin, M., Lux, I. F. M., Goertz, A. G., Van Kerkhof, D. H. A. T., & Wiers, R. W. (2010). Clinical effectiveness of attentional bias modification training in abstinent alcoholic patients. *Drug and Alcohol Dependence*, *109*(1-3), 30–36. <http://doi.org/10.1016/j.drugalcdep.2009.11.022>
- Schwartz, M. W., Woods, S. C., Porte, D., Seeley, R. J., & Baskin, D. G. (2000). Central nervous system control of food intake. *Nature*, *404*(6778), 661–671.

- Seligman, M. E. P., & Csikszentmihalyi, M. (2000). Positive psychology: An introduction. *American Psychologist*. <http://doi.org/10.1037/0003-066X.55.1.5>
- Sengupta, J. N., & Gebhart, G. F. (1994). Gastrointestinal afferents fibers and sensation. In J. L. R (Ed.), *Physiology of the gastrointestinal tract* (pp. 483–519). New York: Raven.
- Serra, J., Azpiroz, F., & Malagelada, J. R. (1998). Modulation of gut perception in humans by spatial summation phenomena. *Journal of Physiology*, *506*(2), 579–587. <http://doi.org/10.1111/j.1469-7793.1998.579bw.x>
- Shafran, R., Lee, M., Cooper, Z., Palmer, R. L., & Fairburn, C. G. (2007). Attentional bias in eating disorders. *The International Journal of Eating Disorders*, *40*(4), 369–380. <http://doi.org/10.1002/eat>
- Shafran, R., Lee, M., Cooper, Z., Palmer, R. L., & Fairburn, C. G. (2008). Effect of psychological treatment on attentional bias in eating disorders. *The International Journal of Eating Disorders*, *41*(4), 348–354. <http://doi.org/10.1002/eat.20500>
- Sherrington, C. S. (1948). *The integrative action of the nervous system*. Cambridge, UK: Cambridge University Press.
- Shiiba, T., Nakazato, M., Mizuta, M., Date, Y., Mondal, M. S., Tanaka, M., ... Matsukura, S. (2002). Plasma ghrelin levels in lean and obese humans and the effect of glucose on ghrelin secretion. *The Journal of Clinical Endocrinology and Metabolism*, *87*(1), 240–244. <http://doi.org/10.1210/jcem.87.1.8129>
- Smeets, E., Roefs, A., van Furth, E., & Jansen, A. (2008). Attentional bias for body and food in eating disorders: increased distraction, speeded detection, or both? *Behaviour Research and Therapy*, *46*(2), 229–38. <http://doi.org/10.1016/j.brat.2007.12.003>
- Smink, F. R. E., van Hoeken, D., & Hoek, H. W. (2012). Epidemiology of eating disorders: Incidence, prevalence and mortality rates. *Current Psychiatry Reports*, *14*(4), 406–414. <http://doi.org/10.1007/s11920-012-0282-y>
- Smith, E., & Rieger, E. (2009). The effect of attentional training on body dissatisfaction and dietary restriction. *European Eating Disorders Review*, *17*(3), 169–176. <http://doi.org/10.1002/erv.921>
- Spielberger, C. D., Gorsuch, R. C., & Lushene, R. E. (1970). *STAI. Manual for the State-Trait Anxiety Inventory*. Palo Alto, CA: Consulting Psychologist Press.
- Steinhausen, H. C., & Weber, S. (2009). The outcome of bulimia nervosa: Findings from one-quarter century of research. *American Journal of Psychiatry*, *166*(12), 1331–1341. <http://doi.org/10.1176/appi.ajp.2009.09040582>
- Stephan, E., Pardo, J. V., Faris, P. L., Hartman, B. K., Kim, S. W., Ivanov, E. H., ... Goodale, R. L. (2003). Functional neuroimaging of gastric distention. *Journal of Gastrointestinal Surgery*, *7*(6), 740–749. [http://doi.org/10.1016/S1091-255X\(03\)00071-4](http://doi.org/10.1016/S1091-255X(03)00071-4)
- Stern, R. M., Koch, K. L., Levine, M. E., & Muth, E. R. (2007). Gastrointestinal response. In J. T. Cacioppo, L. G. Tassinary, & G. G. Berntson (Eds.), *Handbook of psychophysiology* (3rd ed., pp. 211–230). Cambridge, UK: Cambridge University Press.
- Stevens, J. (1996). *Applied multivariate statistics for the social sciences* (3rd ed.). Mahwah, NJ: Lawrence Erlbaum Associates.
- Stice, E. (2001). Risk factors for eating pathology: Recent advances and future directions. In R. H. Striegel-Moore & L. Smolak (Eds.), *Eating disorders: Innovative directions in*



- research and practice* (pp. 51–73). Washington, D.C.
- Stice, E. (2002). Risk and maintenance factors for eating pathology: A meta-analytic review. *Psychological Bulletin*, *128*(5), 825–848. <http://doi.org/10.1037/0033-2909.128.5.825>
- Stice, E., & Shaw, H. E. (2002). Role of body dissatisfaction in the onset and maintenance of eating pathology: A synthesis of research findings. *Journal of Psychosomatic Research*, *53*(5), 985–993. Retrieved from <http://www.ncbi.nlm.nih.gov/pubmed/12445588>
- Stice, E., Spoor, S., Ng, J., & Zald, D. H. (2010). Relation of obesity to consummatory and anticipatory food reward. *Physiology & Behavior*, *97*(5), 551–560. <http://doi.org/10.1016/j.physbeh.2009.03.020.Relation>
- Striegel, R. H., Bedrosian, R., Wang, C., & Schwartz, S. (2012). Why men should be included in research on binge eating: Results from a comparison of psychosocial impairment in men and women. *International Journal of Eating Disorders*, *45*(2), 233–240. <http://doi.org/10.1002/eat.20962>
- Striegel-Moore, R. H., & Smolak, L. (2002). Gender, ethnicity, and eating disorders. In C. G. Fairburn & K. D. Brownell (Eds.), *Eating disorders and obesity: A comprehensive handbook* (2nd ed., pp. 251–255). New York: Guilford Press.
- Stunkard, A. J. (1959). Eating patterns and obesity. *The Psychiatric Quarterly*, *33*(2), 284–295. <http://doi.org/10.1007/BF01575455>
- Sullivan, P. F. (2002). Course and outcome of anorexia nervosa and bulimia nervosa. In C. G. Fairburn & K. D. Brownell (Eds.), *Eating disorders and obesity: A comprehensive handbook* (2nd ed., pp. 226–230). New York: Guilford Press.
- Svaldi, J., Tuschen-Caffier, B., Peyk, P., & Blechert, J. (2010). Information processing of food pictures in binge eating disorder. *Appetite*, *55*(3), 685–694. <http://doi.org/10.1016/j.appet.2010.10.002>
- 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. [http://doi.org/10.1016/S0140-6736\(11\)60813-1](http://doi.org/10.1016/S0140-6736(11)60813-1)
- Sysko, R., Devlin, M. J., Walsh, B. T., Zimmerli, E., & Kissileff, H. R. (2007). Satiety and test meal intake among women with binge eating disorder. *International Journal of Eating Disorders*, *40*(6), 554–561. <http://doi.org/10.1002/eat>
- Tabachnick, B. G., & Fidell, L. S. (2007). *Using multivariate statistics* (5th ed.). Boston: Allyn & Bacon.
- Tapper, K., Pothos, E. M., Fadardi, J. S., & Ziori, E. (2008). Restraint, disinhibition and food-related processing bias. *Appetite*, *51*(2), 335–338. <http://doi.org/10.1016/j.appet.2008.03.006>
- Tchicaya, A., & Lorentz, N. (2010). Prévalence du surpoids et de l'obésité de 1995 à 2008. *Vivre Au Luxembourg, Chroniques de L'enquête (PSELL-3/2007-CEPS INSTEAD)*, *66*, 1–2.
- Terasawa, Y., Shibata, M., Moriguchi, Y., & Umeda, S. (2013). Anterior insular cortex mediates bodily sensibility and social anxiety. *Social Cognitive and Affective Neuroscience*, *8*(3), 259–266. <http://doi.org/10.1093/scan/nss108>
- Terhaar, J., Viola, F. C., Bär, K. J., & Debener, S. (2012). Heartbeat evoked potentials mirror

- altered body perception in depressed patients. *Clinical Neurophysiology*, 123(10), 1950–1957. <http://doi.org/10.1016/j.clinph.2012.02.086>
- Thiel, A., & Paul, T. (2006). Test-retest reliability of the Eating Disorder Inventory 2. *Journal of Psychosomatic Research*, 61(4), 567–569. <http://doi.org/10.1016/j.jpsychores.2006.02.015>
- Thompson, J. K., & Stice, E. (2001). Thin-Ideal Internalization: Mounting Evidence for a New Risk Factor for Body-Image Disturbance and Eating Pathology. *Current Directions in Psychological Science*, 10(5), 181–183. <http://doi.org/10.1111/1467-8721.00144>
- 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. <http://doi.org/10.1037/0033-295X.97.2.147>
- 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. *The International Journal of Eating Disorders*, 34(2), 227–34. <http://doi.org/10.1002/eat.10186>
- Trenary, L., Craighead, L. W., & Hill, D. M. (2005). Validation of the Interoceptive Awareness Questionnaire-Expanded (IAQ-E). In *Poster presented at the annual meeting for the Academy of Eating Disorders*. Montreal, Canada.
- Tribole, E., & Resch, E. (1995). *Intuitive eating: A recovery book for the chronic dieter*. New York: St. Martin's Press.
- Tribole, E., & Resch, E. (2003). *Intuitive eating* (2nd ed.). New York: St. Martin's Griffin.
- Tsakiris, M., Tajadura-Jiménez, A., & Costantini, M. (2011). Just a heartbeat away from one's body: Interoceptive sensitivity predicts malleability of body-representations. *Proceedings. Biological Sciences / The Royal Society*, 278(1717), 2470–2476. <http://doi.org/10.1098/rspb.2010.2547>
- Turner, M. S. J., Foggo, M., Bennie, J., Carroll, S., Dick, H., & Goodwin, G. M. (1991). Psychological, hormonal and biochemical changes following carbohydrate binging: A placebo-controlled study in bulimia nervosa and matched controls. *Psychological Medicine*, 21, 123–133.
- Tylka, T. L. (2006). Development and psychometric evaluation of a measure of intuitive eating. *Journal of Counseling Psychology*, 53(2), 226–240. <http://doi.org/10.1037/0022-0167.53.2.226>
- Tylka, T. L., Calogero, R. M., & Daniélsdóttir, S. (2015). Is intuitive eating the same as flexible dietary control? Their links to each other and well-being could provide an answer. *Appetite*, 95, 166–175. <http://doi.org/10.1016/j.appet.2015.07.004>
- Tylka, T. L., & Kroon Van Diest, A. M. (2013). The Intuitive Eating Scale-2: Item refinement and psychometric evaluation with college women and men. *Journal of Counseling Psychology*, 60(1), 137–153. <http://doi.org/10.1037/a0030893>
- Tylka, T. L., & Subich, L. M. (2004). Examining a multidimensional model of eating disorder symptomatology among college women. *Journal of Counseling Psychology*, 51(3), 314–328. <http://doi.org/10.1037/0022-0167.51.3.314>
- Tylka, T. L., & Wilcox, J. A. (2006). Are intuitive eating and eating disorder symptomatology opposite poles of the same construct? *Journal of Counseling Psychology*, 53(4), 474–485. <http://doi.org/10.1037/0022-0167.53.4.474>

- UNESCO. (1997). *International standard classification of education*. Paris: UNESCO.
- Vaitl, D. (1996). Interoception. *Biological Psychology*, 42(1-2), 1–27.  
[http://doi.org/10.1016/0301-0511\(95\)05144-9](http://doi.org/10.1016/0301-0511(95)05144-9)
- van de Wall, E. H. E. M., Pomp, E. R., Strubbe, J. H., Scheurink, A. J. W., & Koolhaas, J. M. (2005). Deafferentation affects short-term but not long-term control of food intake. *Physiology & Behavior*, 84(4), 659–667. <http://doi.org/10.1016/j.physbeh.2005.02.017>
- van Dyck, Z., Vögele, C., Blechert, J., Lutz, A. P. C., Schulz, A., & Herbert, B. M. (2015). The water load test as a measure of gastric interoception: Development of a two-stage protocol and application to a healthy population. *Manuscript in Preparation*.
- Van Dyke, N., & Drinkwater, E. J. (2014). Review article relationships between intuitive eating and health indicators: Literature review. *Public Health Nutrition*, 17(8), 1757–1766. <http://doi.org/10.1017/S1368980013002139>
- van Strien, T., Frijters, J. E. R., Bergers, G. P. A., & 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.
- Vandenbergh, J., Dupont, P., Fischler, B., Bormans, G., Persoons, P., Janssens, J., & Tack, J. (2005). Regional brain activation during proximal stomach distention in humans: A positron emission tomography study. *Gastroenterology*, 128, 564–573.  
<http://doi.org/10.1053/j.gastro.2004.11.054>
- Vandereycken, W. (2002). History of anorexia nervosa and bulimia nervosa. In C. G. Fairburn & K. D. Brownell (Eds.), *Eating disorders and obesity: A comprehensive handbook* (2nd ed., pp. 151–154). New York: Guilford Press.
- 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*, 9(4), 300–305.  
<http://doi.org/10.1007/BF03325086>
- Veenstra, E. M., & de Jong, P. J. (2012). Attentional bias in restrictive eating disorders. Stronger attentional avoidance of high-fat food compared to healthy controls? *Appetite*, 58(1), 133–140. <http://doi.org/10.1016/j.appet.2011.09.014>
- Visscher, T. L. S., & Seidell, J. C. (2001). The public health impact of obesity. *Annual Review of Public Health*, 22(1), 355–375.
- Vögele, C., & Gibson, L. (2010). Mood, emotions and eating disorders. In W. S. Agras (Ed.), *Oxford handbook of eating disorders* (pp. 180–205). Oxford University Press.
- Wadlinger, H. A., & Isaacowitz, D. M. (2008). Looking happy: The experimental manipulation of a positive visual attention bias. *Emotion*, 8(1), 121–126.  
<http://doi.org/10.1037/1528-3542.8.1.121>
- Walsh, B. T., Zimmerli, E., Devlin, M. J., Guss, J., & Kissileff, H. R. (2003). A disturbance of gastric function in bulimia nervosa. *Biological Psychiatry*, 54(9), 929–933.  
[http://doi.org/10.1016/S0006-3223\(03\)00176-8](http://doi.org/10.1016/S0006-3223(03)00176-8)
- Wang, G. J., Tomasi, D., Backus, W., Wang, R., Telang, F., Geliebter, A., ... Volkow, N. D. (2008). Gastric distention activates satiety circuitry in the human brain. *NeuroImage*, 39(4), 1824–1831. <http://doi.org/10.1016/j.neuroimage.2007.11.008>
- Wardle, J. (1990). Conditioning processes and cue exposure in the modification of excessive

- eating. *Addictive Behaviors*, 15(4), 387–393. [http://doi.org/10.1016/0306-4603\(90\)90047-2](http://doi.org/10.1016/0306-4603(90)90047-2)
- Weingarten, H. (1985). Stimulus control of eating: Implications for a two-factor theory of hunger. *Appetite*, 6(4), 387–401. [http://doi.org/10.1016/S0195-6663\(85\)80006-4](http://doi.org/10.1016/S0195-6663(85)80006-4)
- Werthmann, J., Field, M., Roefs, A., Nederkoorn, C., & Jansen, A. (2014). Attention bias for chocolate increases chocolate consumption – An attention bias modification study. *Journal of Behavior Therapy and Experimental Psychiatry*, 45(1), 136–143. <http://doi.org/10.1016/j.jbtep.2013.09.009>
- Werthmann, J., Jansen, A., & Roefs, A. (2014). Worry or craving? A selective review of evidence for food-related attention biases in obese individuals, eating-disorder patients, restrained eaters and healthy samples. *Proceedings of the Nutrition Society*, 74(2), 99–114. <http://doi.org/10.1017/S0029665114001451>
- Werthmann, J., Roefs, A., Nederkoorn, C., Mogg, K., Bradley, B. P., & Jansen, A. (2011). Can(not) take my eyes off it: Attention bias for food in overweight participants. *Health Psychology*, 30(5), 561–569. <http://doi.org/10.1037/a0024291>
- Whitehead, W. E., & Drescher, V. M. (1980). Perception of gastric contractions and self-control of gastric motility. *Psychophysiology*, 17(6), 552–558. <http://doi.org/10.1111/j.1469-8986.1980.tb02296.x>
- Whitehead, W. E., Drescher, V. M., Heiman, P., & Blackwell, B. (1977). Relation of heart rate control to heartbeat perception. *Biofeedback and Self-Regulation*, 2(4), 371–392. <http://doi.org/10.1007/BF00998623>
- Wiers, R. W., Cox, W. M., Field, M., Fadardi, J. S., Palfai, T. P., Schoenmakers, T., & Stacy, A. W. (2006). The search for new ways to change implicit alcohol-related cognitions in heavy drinkers. *Alcoholism: Clinical and Experimental Research*, 30(2), 320–331. <http://doi.org/10.1111/j.1530-0277.2006.00037.x>
- Wilfley, D. E., Wilson, G. T., & Agras, W. S. (2003). The clinical significance of binge eating disorder. *The International Journal of Eating Disorders*, 34 Suppl, S96–106. <http://doi.org/10.1002/eat.10209>
- Wittchen, H. U., Zaudig, M., & Fydrich, T. (1997). *Strukturiertes klinisches Interview für DSM-IV: Achse I*. Göttingen: Hogrefe.
- Woods, S. C. (1991). The eating paradox: How we tolerate food. *Psychological Review*, 98(4), 488–505. <http://doi.org/10.1037/0033-295X.98.4.488>
- Woolley, J. D., Gorno-Tempini, M. L., Seeley, W. W., Rankin, K., Lee, S. S., Matthews, B. R., & Miller, B. L. (2007). Binge eating is associated with right orbitofrontal-insular-striatal atrophy in frontotemporal dementia. *Neurology*, 69(14), 1424–1433. <http://doi.org/10.1212/01.wnl.0000277461.06713.23>
- World Health Organization. (2013a). *Global Health Observatory Data Repository*. Geneva: World Health Organization.
- World Health Organization. (2013b). *Obesity and overweight. Fact Sheet 311*. Geneva. Retrieved from <http://amro.who.int/common/Display.asp?Lang=E&RecID=10203>
- Xing, J., & Chen, J. D. Z. (2004). Alterations of gastrointestinal motility in obesity. *Obesity Research*, 12(11), 1723–1732. <http://doi.org/10.1038/oby.2004.213>
- Yanovski, S. Z. (2002). Binge eating in obese persons. In *Eating disorders and obesity: A*

*comprehensive handbook* (2nd ed., pp. 403–407). New York: Guilford Press.

Zimmerli, E. J., Walsh, B. T., Guss, J. L., Devlin, M. J., & Kissileff, H. R. (2006). Gastric compliance in bulimia nervosa. *Physiology & Behavior*, *87*(2), 441–446.  
<http://doi.org/10.1016/j.physbeh.2005.11.010>

## **6. APPENDICES**

<b>Appendix A</b>	Questionnaires used in Study 1
<b>Appendix B</b>	Exemplary high-calorie and neural pictures from the dot probe task (Study 1)
<b>Appendix C</b>	Supplemental tables from Study 2
<b>Appendix D</b>	Questionnaires used in Study 2
<b>Appendix E</b>	Questionnaires used in Study 3
<b>Appendix F</b>	Telephone Screening used in Study 4
<b>Appendix G</b>	Structured sociodemographic interview used in Studies 1-4
<b>Appendix H</b>	Information brochure that was distributed to participants of the ATTEND study before participation (Study 4)

## **APPENDIX A**

### **Questionnaires used in Study 1**

Removed for Copyright Reasons

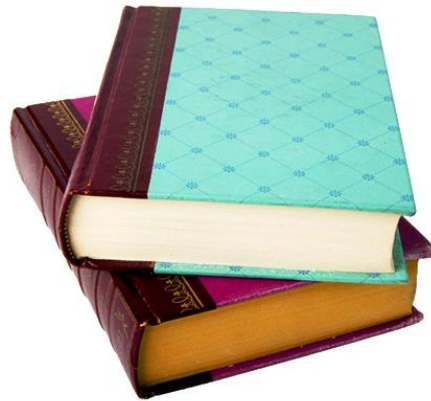
## **APPENDIX B**

**Exemplary high-calorie and neural pictures from the dot probe task**

**(Study 1)**



**Exemplary high-calorie and neutral pictures from the dot probe task (Study 1)**



## **APPENDIX C**

### **Supplemental tables from Study 2**

Supplemental Table 1

*Standardized item and factor loadings of the IES-1 obtained by confirmatory factor analyses of the overall sample, women (n = 922), and men (n = 212).*

Factor and IES-1 item	First-order			Second-order		
	Overall	Women	Men	Overall	Women	Men
<b>UPE</b>				.86	.84	.94
Item 1	.87	.88	.91			
Item 4	.72	.76	.51			
Item 5	.70	.74	.48			
Item 9	.76	.77	.69			
Item 14	.70	.76	.45			
Item 18	.81	.81	.77			
Item 19	.59	.63	.47			
Item 20	.10	.14	-.07			
Item 21	.55	.58	.35			
<b>EPR</b>				.66	.67	.55
Item 2	.21	.26	.12			
Item 3	.78	.79	.66			
Item 6	.61	.60	.56			
Item 10	.84	.84	.77			
Item 16	.84	.85	.70			
Item 17	.79	.79	.73			
<b>RHSC</b>				.73	.79	.40
Item 7	.46	.50	.21			
Item 8	.18	.20	.04			
Item 11	.72	.74	.61			
Item 12	.57	.57	.62			
Item 13	.88	.88	.98			
Item 15	.64	.68	.50			

*Note.* IES-1 = Intuitive Eating Scale-1, UPE = Unconditional Permission to Eat, EPR = Eating for Physical Rather Than Emotional Reasons, RHSC = Reliance on Hunger and Satiety Cues.

Supplemental Table 2

*IES-2 factor loadings and item-factor-correlations corrected for overlap.*

<b>Factor</b>	<b>IES-2 item</b>	<b><i>r</i></b>	<b><i>r.cor</i></b>
<b>UPE</b>	Item 1	.96	.62
	Item 3	.62	.58
	Item 4	.63	.59
	Item 9	.59	.54
	Item 16	.78	.71
	Item 17	.70	.60
<b>EPR</b>	Item 2	.77	.73
	Item 5	.85	.77
	Item 10	.86	.82
	Item 11	.76	.70
	Item 12	.56	.54
	Item 13	.45	.44
	Item 14	.80	.77
	Item 15	.68	.66
<b>RHSC</b>	Item 6	.77	.73
	Item 7	.60	.54
	Item 8	.87	.78
	Item 21	.82	.74
	Item 22	.78	.74
	Item 23	.86	.83
<b>B-FCC</b>	Item 18	.31	.30
	Item 19	.88	.71
	Item 20	.94	.68

*Note.* IES-2 = Intuitive Eating Scale-2, UPE = Unconditional Permission to Eat, EPR = Eating for Physical Rather Than Emotional Reasons, RHSC = Reliance on Hunger and Satiety Cues, B-FCC = Body-Food Choice Congruence.

## **APPENDIX D**

### **Questionnaires used in Study 2**

Removed for Copyright Reasons

## **APPENDIX E**

### **Questionnaires used in Study 3**

Removed for Copyright Reasons

## **APPENDIX F**

### **Telephone Screening used in Study 4**

Removed for Copyright Reasons

## **APPENDIX G**

### **Structured sociodemographic interview used in Studies 1-4**



## Soziodemographisches Interview

Dieses Interview enthält eine Reihe optionaler Kategorien und Unterkategorien, die je nach Bedarf einer Studie ausgewählt werden können. Auch Fragen innerhalb der Kategorien können je nach Bedarf ausgewählt werden. Einige Fragen sind in alternativen Versionen angegeben, die ebenfalls je nach Bedarf ausgewählt werden können. Bitte ändern Sie nicht die Itemnummern, um eine problemlose Zuordnung der Antworten zu den Itemnummern in der SPSS-Datenmaske gewährleisten zu können.

Die Auswahl der Items findet vor der Datenerhebung statt, sodass für den Interviewer nur die relevanten Items im Katalog stehen, die dann vollständig abgefragt werden.

Die Items werden durch eine fünfstellige Nummer gekennzeichnet:

1. Stelle = Kategorie
2. Stelle = Unterkategorie
3. und 4. Stelle = fortlaufende Nummer innerhalb einer Itemkategorie
5. Stelle = Identifikation von Alternativitems oder Identifikation von String-Variablen zur Kodierung der Angaben unter „Sonstige“ (in diesem Fall entspricht die 5. Stelle der Nummer der Kategorie „Sonstige“ (10 wird zu 1))  
Z.B.: 21017 -> Kategorie 2 (Nationalität und Sprache), Unterkategorie 1 (Staatsangehörigkeit und Migrationshintergrund), Item 01, unter Antwortmöglichkeit 7 („Sonstige“) angegebene Bezeichnung.
5. Stelle bei Schwangerschaftsitems: Kennzeichnung der einzelnen Schwangerschaften bei mehreren Schwangerschaften (1., 2. und 3. Schwangerschaft)

Kennzeichnung der Items in der SPSS-Maske:

Die Nummer ist identisch zur Nummer im Fragebogen, jedoch werden die Buchstaben *SD* für soziodemographisches Interview voran gestellt.

Nähere Angaben zur Kategorie „Sonstige“ werden mit einer eigenen Stringvariablen kodiert, die mit Ausnahme der letzten Stelle der übergeordneten Itemnummer entspricht.

### **Instruktion für den/die Interviewende/n**

*Dieses Interview enthält Fragen zum soziodemographischen Hintergrund der Teilnehmenden. Fragen Sie alle Fragen ab, lassen Sie keine aus. Eine Ausnahme stellt die Kategorie „Für Frauen“ dar, die nur bei weiblichen Teilnehmerinnen abgefragt wird. Außerdem werden einige Fragen nur gestellt, wenn die vorherige Frage mit „ja“ beantwortet wurde. Diese bedingten Fragen sind grau unterlegt. In der Kategorie 4 „Ausbildung und Beruf“ gelten zusätzliche Regeln für bedingte Items, die an der entsprechenden Stelle beschrieben werden. Fragen und Antworten sind jeweils normal gedruckt, während Instruktionen und Hinweise für den/die Interviewer/in kursiv gesetzt sind. Die Formulierung der Fragen ist nicht zwingend vorgegeben und kann nach Bedarf umformuliert werden. Stellen Sie so viele zusätzliche Fragen wie nötig, um eine ausreichend ausführliche Antwort zu erhalten. Fragen und notieren Sie lieber zu viel, als zu wenig.*

*Versuchen Sie möglichst ausführliche und genaue Antworten von dem/der Teilnehmenden zu erhalten. Wenn Sie sich bei geschlossenem Antwortformat nicht sicher sind, welche Antwort*

die zutreffende ist, notieren Sie möglichst genau die Aussagen des/der Teilnehmenden und besprechen Sie den Fall mit Ihrem/r Supervidierenden. Notieren Sie bei offenem Antwortformat möglichst ausführlich die Aussagen des/der Teilnehmenden. Umkreisen Sie bei geschlossenem Antwortformat die zutreffende Zahl. Wenn Mehrfachnennungen möglich sind, ist dies angegeben. Notieren Sie offene Antworten auf den vorgegebenen Linien. Nutzen Sie, wenn nötig, die zusätzlichen Notizblätter am Ende des Interviews. Kennzeichnen Sie zusätzliche Notizen immer mit der zugehörigen Itemnummer. Wenn Sie bemerken, dass einem/r Teilnehmenden eine Frage unangenehm ist, sprechen Sie ihn/sie empathisch darauf an. Versuchen Sie Zweifel aus der Welt zu schaffen, indem Sie z.B. nochmals die Anonymität der Daten betonen.

*Besondere Kodierungen:*

999 = nicht zutreffend

888 = weiß nicht/Antwort verweigert

Wenn eine Frage auf den/die Teilnehmende/n nicht zutrifft, z.B. Fragen nur für Frauen bei einem männlichen Teilnehmer oder bedingte Fragen wenn zuvor mit „nein“ geantwortet wurde, geben Sie als Antwort „999“ an. Geben Sie auch in die SPSS-Datenmaske „999“ ein. Wenn der/die Teilnehmende eine Antwort nicht weiß oder nicht antworten möchte, kodieren Sie mit „888“.

### **Einleitung für den/die Teilnehmende/n**

Passen Sie den Text ggf. im Voraus an Ihre Studie an, indem Sie z.B. erklären, welche soziodemographischen Variablen speziell für Ihre Studie besonders wichtig sind. Ändern Sie den Text vor Beginn der Studie, damit jede/r Interviewende dasselbe vorliest.

*Lesen Sie vor:*

<b>lesen</b>	<p>(Vielen Dank für Ihre Teilnahme an dieser Studie.)</p> <p>Wir beginnen nun mit einem kleinen, allgemeinen Interview. Dabei werde ich die meiste Zeit ablesen, so wie jetzt grade auch. Das wird deshalb so gemacht, damit wir sicherstellen können, dass jeder Interviewer auch genau dieselben Fragen stellt und nichts vergisst.</p> <p>Im Folgenden werde ich Ihnen einige Fragen zu Ihrer Person und Ihren Lebensumständen stellen. Diese Informationen sind sehr wichtig für das Gelingen unserer Studie. Insbesondere geht es darum, dass wir die Gruppe der Teilnehmenden als Ganzes beschreiben können, z.B. was war das mittlere Alter der Teilnehmenden, wie viele Männer und wie viele Frauen nahmen teil, aus welchen Ländern kamen die Teilnehmenden. Diese genaue Beschreibung der Gruppe der Teilnehmenden ist außerordentlich wichtig, um Ergebnisse aus verschiedenen Studien miteinander vergleichen zu können. Z.B. können sich Ergebnisse von Studien stark unterscheiden, wenn unterschiedliche Altersgruppen befragt werden. Wenn Ihnen während des Interviews etwas unklar ist oder Sie eine Frage nicht richtig verstanden haben, fragen Sie bitte jederzeit nach.</p> <p>Ihre Antworten werde ich hier in diesem Interview-Bogen notieren und Sie dürfen auch gerne sehen, was ich aufschreibe.</p> <p>Selbstverständlich bleiben alle Ihre Antworten anonym, wie dies für alle Daten der Fall ist, die wir im Rahmen dieser Studie aufzeichnen.</p> <p>Zu unserer eigenen Kontrolle werden wir alle Interviews auf Tonband aufzeichnen. Dies dient dazu, dass wir später überprüfen können, ob alle Fragen korrekt gestellt wurden und wir nichts vergessen</p>
--------------	--

haben. Zudem ermöglicht uns die Aufnahme, noch einmal zu überprüfen, ob wir alles richtig mitgeschrieben haben. Ihre Aufnahme wird nach Abschluss der Studie gelöscht. Um Ihre Anonymität zu gewährleisten, werde ich während des Interviews Ihren Namen nicht nennen.

Gibt es von Ihrer Seite noch Fragen bezüglich des Interviews? Sie können sonst auch gerne jederzeit Fragen stellen.

Sind Sie bereit, mit dem ersten Interview zu beginnen?

**Kategorie 1: Allgemeine Daten**

Nr.	Frage	Antwort
10010	<p>Geschlecht</p> <p><i>Wenn das Geschlecht eindeutig erkennbar ist, kreuzen Sie einfach an, ohne zu fragen.</i></p>	<p>1 weiblich</p> <p>2 männlich</p>
10020	<p>Was ist Ihr Geburtsdatum?</p>	<p><i>Datum: ___ / ___ / ___ (dd/mm/yyyy)</i></p>
10021	<p>(Dann sind Sie heute ___ Jahre alt?)</p> <p><i>Kann auch im Nachhinein berechnet werden.</i></p>	<p><i>-&gt; Alter zum Zeitpunkt der Testung:</i></p> <p>___ Jahre ___ Monate</p>
	<p><i>Geben Sie in SPSS das Alter ein.</i></p>	
10030	<p>Welche Hand verwenden Sie vorzugsweise, z.B. zum Schreiben? Gibt es sonst Sachen, die Sie eher mit der anderen Hand machen?</p> <p><i>Wenn die Teilnehmenden angeben, beide Hände zu verwenden, bzw. sich nicht sicher sind, fragen Sie den Edinburgher Händigkeitinventar ab. Kreuzen Sie dann auf Basis des Lateralitätsquotients entweder 1 oder 2 an. Kreuzen Sie 3 nur an, wenn der Lateralitätsquotient = 0 ist.</i></p>	<p>1 Rechte Hand (LQ &gt; 0)</p> <p>2 Linke Hand (LQ &lt; 0)</p> <p>3 Beide Hände (LQ = 0) -&gt; <i>Edinburgher Händigkeitinventar</i></p>
10031	<p><i>Der Händigkeitindex wird unter Item 10031 eingegeben, wenn er abgefragt wurde.</i></p>	<p><i>Händigkeitindex/Lateralitätsquotient:</i></p> <p><math>LQ = (R - L) / (R + L) \times 100 = \underline{\hspace{2cm}}</math></p>

*Edinburgher Händigkeitinventar → Wenn Händigkeit unklar ist!*

Bitte markieren Sie Ihren bevorzugten Handgebrauch bei den aufgelisteten Tätigkeiten durch ein Kreuz (+) in der entsprechenden Spalte. Wenn die Bevorzugung so stark ist, dass Sie nie versuchen würden, die andere Hand für diese Tätigkeit zu gebrauchen (ohne dazu gezwungen zu sein), dann markieren Sie diese durch zwei Kreuze (++). Wenn es wirklich egal ist, dann setzen Sie bitte in beide Spalten ein Kreuz.

Einige dieser Tätigkeiten erfordern den Einsatz beider Hände. In diesen Fällen ist der Teil der Aufgabe, bzw. das Objekt in Klammern angegeben, wofür die bevorzugte Hand erfragt wird.

Versuchen Sie bitte, alle Fragen zu beantworten, und lassen Sie nur dann einen Freiraum, wenn Sie überhaupt keine Erfahrung mit der Tätigkeit oder mit dem Objekt haben.

<b>Tätigkeit</b>	<b>links</b>	<b>rechts</b>
Schreiben		
Zeichnen		
Werfen		
Schere		
Zahnbürste		
Messer (ohne Gabel)		
Löffel		
Besen (obere Hand)		
Streichholz anzünden (Streichholz)		
Dose öffnen		
*Welchen Fuß bevorzugen Sie beim Kicken?		
*Welches Auge benutzen Sie, wenn Sie nur mit einem sehen?		

*\*Items beziehen sich nicht auf Bevorzugung einer Hand sondern auf allgemeine Lateralität -> je nach Studie ein- oder ausschließen*

**Kategorie 2: Nationalität und Sprache**

21	<b>Staatsangehörigkeit und Migrationshintergrund</b>	
21010          21017 21018	<p>Welche Staatsangehörigkeit(en) haben Sie?  <i>Kodieren Sie bei mehreren Staatsangehörigkeiten „8“ und notieren Sie alle Staatsangehörigkeiten. Geben Sie diese unter der String-Variable 21018 in SPSS ein.</i></p>	<p>1 Luxemburgische            2 Portugiesische            3 Französische            4 Italienische            5 Belgische            6 Deutsche            7 Sonstige: _____            8 Multiple Staatsangehörigkeit:            _____            _____</p>
21040      21045	<p>In welchem Land leben Sie momentan?  <i>Wenn nicht klar zu beantworten: In welchem Land haben Sie Ihren (Haupt-)Wohnsitz angemeldet? In welchem Land verbringen Sie mehr als 50% Ihrer Zeit?</i></p>	<p>1 Luxemburg            2 Deutschland            3 Frankreich            4 Belgien            5 Sonstiges: _____</p>
21050	<p>Wie lange leben Sie bereits (ununterbrochen) in diesem Land?</p>	<p><i>Jahre:</i> ___ __</p>
21060       21067	<p>Welches ist das Geburtsland Ihrer Mutter?</p>	<p>1 Luxemburg            2 Portugal            3 Frankreich            4 Italien            5 Belgien            6 Deutschland            7 Sonstiges: _____</p>
21070	<p>Welches ist das Geburtsland Ihres Vaters?</p>	<p>1 Luxemburg            2 Portugal            3 Frankreich            4 Italien            5 Belgien            6 Deutschland</p>

21077		7 Sonstiges: _____
-------	--	--------------------

<b>22</b>	<b>Sprache</b>	
	<p>Wie gut beherrschen Sie folgende Sprachen?  <i>Umkreisen Sie für die angegebenen Sprachen die entsprechende Zahl aus der Skala rechts. Notieren Sie unter „Sonstige“ die Muttersprache, wenn diese sich nicht unter den anderen Sprachen befindet. Weitere Sprachen sind irrelevant.</i></p>	<p>0 Überhaupt nicht  1 Grundkenntnisse (A1, A2)  2 Mittelmäßig (B1, B2)  3 Fortgeschritten (C1)  4 Fließend/Muttersprache (C2)</p> <p><i>Der Europäische Referenzrahmen wird nicht vorgegeben; die Angaben in Klammern dienen nur der Orientierung des/der Interviewenden.</i></p>
22010	Deutsch	0 1 2 3 4
22040	Luxemburgisch	0 1 2 3 4
22060	Sonstige: _____	
22063		0 1 2 3 4

**Kategorie 3: Aktuelle Lebenssituation**

<p>30010</p> <p>30012</p> <p>30013</p> <p>30014</p> <p>30015</p>	<p>Welchen Familienstand haben Sie? Leben Sie momentan in einer Partnerschaft? Waren Sie einmal gepacst oder haben in einer eingetragenen Lebenspartnerschaft gelebt?  <i>Mehrfachnennungen möglich, z.B. geschieden und neu verheiratet. In diesem Fall aktuelle Lebenssituation mit „A“ kennzeichnen. Diese wird dann in die SPSS-Maske eingetragen, die vergangene Lebenssituation wird ignoriert.</i></p>	<p>1 Ledig</p> <p>2 Partnerschaft</p> <p>    21 Nicht eingetragene Partnerschaft</p> <p>    22 Eingetragene Lebenspartnerschaft (Deutschland)</p> <p>    23 PACS (Luxemburg, Frankreich)</p> <p>    24 Verheiratet</p> <p>    25 Sonstige: _____</p> <p>3 Partnerschaft aufgelöst</p> <p>    31 Geschieden</p> <p>    32 Gesetzlich aufgelöst</p> <p>    33 Sonstige: _____</p> <p>4 Tod des Partners</p> <p>    41 Verwitwet</p> <p>    42 Partnerschaft durch Tod des Partners beendet</p> <p>    43 Sonstige: _____</p> <p>5 Sonstige: _____</p>
<p>30020</p>	<p>Haben Sie Kinder?</p>	<p>0 nein</p> <p>1 ja</p>
<p>30030</p>	<p>Wie viele?</p>	<p><i>Anzahl:</i> __ __</p>
<p>30040</p>	<p>In welchen Jahren wurden Ihre Kinder geboren?  <i>Geben Sie die Geburtsjahre als String in SPSS ein.</i></p>	<p><i>Geburtsjahre:</i> _____          _____</p>



30050	Mit wem leben Sie momentan zusammen?	0 Lebt alleine 1 Eltern oder Elternteil 2 Großeltern oder Großelternteil 3 Stiefeltern oder Stiefelternteil ( <i>hier auch ankreuzen, wenn Kind bei leiblichem Elternteil und dessen Partner/in lebt, ohne dass eine Ehe besteht</i> ) 4 Geschwister 5 Ehegatte/in, Partner/in 6 Eigene Kinder
30058		7 Mitbewohner/in
30059		8 Sonstige Verwandte: _____ _____ 9 Sonstige nicht Verwandte: _____ _____

### Kategorie 4: Ausbildung und Beruf

41	Derzeitige Tätigkeit	
	<p>Welche Tätigkeit üben Sie derzeit aus? Führen Sie daneben noch eine andere Tätigkeit aus?  <i>Mehrfachnennungen möglich, z.B. Student, der nebenher jobbt -&gt; Haupttätigkeit = Student, Nebentätigkeit = berufstätig.</i></p>	<p>1 Schüler/in an einer Primar- oder Sekundarschule</p>
41010	<p><i>Haupttätigkeit unter 41010 kodieren.</i></p>	<p>2 Student/in an einer Hochschule</p>
41020	<p><i>Nebentätigkeit unter 41020 kodieren.</i></p>	<p>3 Auszubildende/r (<i>in der Berufsausbildung, ohne 1 und 2</i>)</p>
41014	<p><i>Bei Mehrfachnennungen im Folgenden alles abfragen und notieren!</i></p>	<p>4 berufstätig (<i>wenn Auszubildende/r: 3 ankreuzen</i>)</p>
		<p>41 momentan arbeitend</p>
		<p>42 vorübergehend nicht arbeitend, wegen: _____</p>
		<p>_____</p>
		<p>(<i>z.B. Mutterschutz/Elternzeit, Krankschreibung, ...</i>)</p>
		<p>5 arbeitslos, arbeitssuchend</p>
		<p>6 Hausfrau/-mann (<i>ausschließlich im eigenen Haushalt tätig, nicht berufstätig</i>)</p>
		<p>7 Rentner/in, Frührentner/in (Pensions- oder Rentenempfänger/in), auch Witwenrente, etc.</p>
		<p>8 Eigentümer/in, vom Vermögen lebend</p>
		<p>9 Dauerhaft erwerbsunfähig</p>
41011		<p>10 Sonstiges: _____</p>

Hinweise zum sozioökonomischen Status und dem Abfragen der Abschlüsse:

Fragen Sie Haupt- und Nebentätigkeit ab! Z.B. Studium und Berufstätigkeit.

Wichtig ist, dass Sie ALLE Abschlüsse, Ausbildungen, etc. abfragen und notieren. Für den sozioökonomischen Status (SÖS) zählt später die höchste Schulausbildung und der höchste berufliche oder Hochschulabschluss.

Fragen Sie immer zuerst das Land ab, in dem ein Abschluss erworben wurde. Sehen Sie nach, ob eine entsprechende ISCED-Liste vorhanden ist. Wenn nicht, fragen Sie den/die Teilnehmende/n nach einem luxemburgischen Äquivalent des Abschlusses. Notieren Sie auf jeden Fall die landesspezifische, originale Bezeichnung des Abschlusses und das entsprechende Land. Wenn für ein Land eine ISCED-Liste vorliegt und Sie mit dem Bildungssystem dieses Landes nicht vertraut sind, prüfen Sie, ggf. gemeinsam mit dem/der

*Teilnehmenden, ob der Abschluss in der Liste aufgeführt ist. Wenn nicht, versuchen Sie einen gleichwertigen Abschluss auf der Liste zu finden.*

*Manche Fragen werden nur gestellt, wenn beim ersten Item der Kategorie 4 (#41010) eine bestimmte Antwort gegeben wurde. Z.B. I=I bedeutet, diese Frage wird nur gestellt, wenn das Item #41010 mit I „Schüler/in“ beantwortet wurde. Alle anderen Fragen werden immer gestellt!*

*Fragen, die immer gestellt werden, ohne dass eine Bedingung gegeben sein muss, sind durch eine unterstrichene Item-Nummer gekennzeichnet!*

*Lesen Sie vor:*

<b>lesen</b>	Nun geht es um Ihre Ausbildung, also Ihre Schul- und Berufsabschlüsse. Da diese sich sehr stark zwischen Ländern unterscheiden können, werde ich Sie im Folgenden immer zuerst nach dem Land fragen, in dem Sie einen Abschluss erworben haben. Für jedes Land haben wir dann eine Liste, in der wir den Abschluss nachschlagen können. Auf dieser Liste sind die Abschlüsse dann in Kategorien eingeteilt, die es uns ermöglichen, die Abschlüsse über die Länder hinweg zu vergleichen.
--------------	---

<b>42</b>	<b>Aktuelle Beschulung</b>	
42020	<i>I=I</i> In welchem Land besuchen Sie die Schule?	<i>Land:</i> _____
42030	<i>I=I</i> Welche Schule/ welchen Schulzweig besuchen Sie?	<i>Notieren Sie die landesspezifische Bezeichnung und sehen Sie in der ISCED-Liste nach.</i> <i>Schule/Schulart/Schulzweig:</i> _____ _____ _____
42040	In welche Klasse gehen Sie?	<i>Klassenstufe:</i> _____ _____
	<i>I=I Hilfsfrage: Haben Sie bereits einen oder mehrere Schulabschlüsse?</i> <i>Wenn ja -&gt; weiter mit nächster Frage (42050)</i> <i>Wenn nein -&gt; Springe zu Unterkategorie 3 Berufsausbildung</i>	

<b>42 Höchster Schulabschluss</b>		
42050	In welchem Land haben Sie Ihren höchsten (allgemeinbildenden) Schulabschluss erworben?	Land: _____
42060	Welchen höchsten allgemeinbildenden Schulabschluss haben Sie? <i>Ohne Hochschulstudium</i>	<i>Notieren Sie die landesspezifischen Bezeichnungen! Schlagen Sie diese in der ISCED-Liste nach.</i>
42070	Wenn unklar: Auf welcher Schule haben Sie den Abschluss gemacht? <i>Sammeln Sie ausreichend Informationen, um den Abschluss in der ISCED-Liste finden zu können! Gehen Sie ggf. gemeinsam mit dem/der Teilnehmenden die Liste durch!</i>	Abschluss (genaue Bezeichnung!): _____ Schulart: _____
42080	Wenn Schulabschluss unklar: Wie viele Jahre sind Sie insgesamt zur Schule gegangen? <i>Ohne Hochschulstudium</i>	Jahre: __ __

<b>43 Aktuelle Berufsausbildung</b>		
43010	I=3 In welchem Land machen Sie Ihre Ausbildung?	Land: _____
43020	I=3 Welche Ausbildung machen Sie momentan?	Ausbildung: _____
43030	Evtl. Schule/Schulart abfragen	Schulart: _____

<b>43 Abgeschlossene Berufsausbildung</b>		
43040	Haben Sie eine oder mehrere Berufsausbildung(en) abgeschlossen? <i>Hochschulstudium -&gt; Unterkategorie 44</i>	0 nein 1 ja
43050	In welchem Land haben Sie diese abgeschlossen?	Land: _____
43060	Welche Ausbildung haben Sie abgeschlossen? <i>Evtl. Schulart abfragen</i>	Ausbildungsbezeichnung(en): _____ _____

43070		Schulart: _____
-------	--	-----------------

<b>44</b>	<b>Aktuelles Hochschulstudium</b>	
44010	I=2 In welchem Land besuchen Sie die Hochschule?	Land: _____
44020	I=2 An welcher Art von Hochschule studieren Sie? Z.B. Universität, Fachhochschule, ...	Hochschule: _____
44030	I=2 Für welchen Studiengang sind Sie eingeschrieben?	Studiengang: _____
44040	I=2 Im wievielten Semester studieren Sie dieses Fach?	Semesterzahl: _____
44050	I=2 Auf welchem Niveau?	1 Bachelor
44052	Geben Sie die Bezeichnung für einen dem Master äquivalenten Abschluss unter Item 44052 ein.	2 Master (oder Äquivalent: _____)
44054		3 Doktorat
		4 Sonstige: ggf. länderspezifisch _____

<b>44</b>	<b>Abgeschlossenes Hochschulstudium</b>	
44060	Haben Sie ein oder mehrere Hochschulstudien abgeschlossen?	0 nein 1 ja
44070	In welchem Land war das?	Land: _____
44080	Welches/welche?	Studiengang/-gänge: _____ _____
44090	Auf welcher Art von Hochschule? Z.B. Universität, Fachhochschule, ...	Hochschule: _____
44100	Mit welchem Abschluss? Was ist insgesamt Ihr	1 Bachelor
44102	höchster akademischer Grad?	2 Master (oder Äquivalent: _____)
44104	Mehrfachnennungen möglich. Markieren Sie alle Abschlüsse für alle Studiengänge. Für die	3 Doktor

Bestimmung des SÖS wichtig ist der insgesamt höchste Abschluss. Nur dieser wird in SPSS eingegeben.	4 Sonstige: ggf. länderspezifisch _____ _____
---	--

<b>45</b>	<b>Aktuelle <u>berufliche</u> Tätigkeit</b>	
45010	<i>I=4</i> Welche berufliche Tätigkeit führen Sie momentan aus? <i>Fragen Sie nach der genauen Bezeichnung des Berufs (z.B. nicht „Lehrer“ sondern „Grundschullehrer“)</i>	<i>Berufsbezeichnung:</i> _____ _____ _____
45020	<i>I=4</i> In welchem Land arbeiten Sie?	<i>Land:</i> _____
45030	<i>I=4</i> Wie viele Stunden pro Woche oder wie viel Prozent arbeiten Sie?	<i>Stunden:</i> _____
45040		<i>Prozent:</i> _____
45050	<i>I=4</i> Wie viele Stunden sind Regelarbeitszeit im Land, in dem Sie arbeiten?	<i>Regelarbeitszeit [h/Woche]:</i> _____

### **Kategorie 5: Sozio-ökonomischer Status (Eltern/Partner)**

*Fragen Sie bei Minderjährigen und Volljährigen in der Ausbildung (Berufsausbildung, Studium), also allen, die noch im Haushalt der Eltern leben bzw. von diesem abhängig sind, die Unterkategorien 51-Mutter und 52-Vater ab. Wenn Minderjährige bei anderen Verwandten oder Pflegeeltern leben, fragen Sie diese ab und notieren Sie, um wen es sich handelt (z.B. Großmutter, Großvater). Es geht um die Personen, mit denen das Kind in einem Haushalt lebt bzw. von denen das Kind materiell abhängig ist. Fragen Sie ggf. mehr als 2 Personen ab, wenn die Familiensituation kompliziert ist, z.B.: Ein Kind lebt mit einem leiblichen Elternteil und einem Stiefelternteil zusammen und der zweite leibliche Elternteil ist weiterhin sorgeberechtigt/unterhaltspflichtig. Fragen Sie in solchen Fällen alle beteiligten Personen ab und beschreiben Sie die Rollen der einzelnen Personen genau. Klären Sie später mit Ihrem/r Supervidierenden, welche Person zur Bestimmung des SÖS herangezogen wird. Fragen Sie bei Volljährigen (mit eigenem Haushalt) die Unterkategorie 53-Partner ab (wenn Partner vorhanden). Klären Sie ggf. ab, ob der Partner/ die Partnerin im selben Haushalt wie der/die Teilnehmende lebt und notieren Sie dies.*

*→ Der Elternteil oder Partner mit dem höchsten sozialen Status bestimmt den Status des Haushalts und dieser ist somit der Status aller im Haushalt lebenden Personen, incl. des/der Teilnehmenden.*

*Wichtig ist, dass Sie ALLE Abschlüsse, Ausbildungen, etc. abfragen und notieren. Für den SÖS zählt später die höchste Schulausbildung und der höchste berufliche oder Hochschulabschluss.*

*Fragen Sie immer zuerst das Land ab, in dem ein Abschluss erworben wurde. Sehen Sie nach, ob eine entsprechende ISCED-Liste vorhanden ist. Wenn nicht, fragen Sie den/die Teilnehmende/n nach einem luxemburgischen Äquivalent seines Abschlusses. Notieren Sie auf jeden Fall die landesspezifische, originale Bezeichnung des Abschlusses und das entsprechende Land. Wenn für ein Land eine ISCED-Liste vorliegt und sie mit dem Bildungssystem dieses Landes nicht vertraut sind, prüfen Sie, ggf. gemeinsam mit dem/der Teilnehmende/n, ob der Abschluss in der Liste aufgeführt ist. Wenn nicht, versuchen Sie einen gleichwertigen Abschluss auf der Liste zu finden.*

*Lesen Sie vor:*

<b>lesen</b>	<p>Nun geht es um die Ausbildung Ihrer Eltern/ Ihres Partners/ Ihrer Partnerin. Diese Informationen sind wichtig für diese Studie, damit wir nicht nur die teilnehmenden Personen selbst, sondern auch ihr Lebensumfeld beschreiben können. Auch dies sind wichtige Faktoren, die die Ergebnisse unserer Studie beeinflussen können.</p> <p>Wie gerade eben, werde ich Sie wieder zuerst nach dem Land fragen, in dem ein Abschluss erworben wurde, und diesen dann in der Liste nachschlagen.</p>
--------------	--

51	Mutter	
51010	In welchem Land hat Ihre Mutter ihren höchsten Schulabschluss gemacht? <i>Je nach Land wird die entsprechende Liste gewählt und nach ISCED kodiert.</i>	Land: _____
51020 51030	Welchen höchsten allgemeinbildenden Schulabschluss hat Ihre Mutter? Evtl. noch Schule/Schulart abfragen.	Abschluss: _____ Schulart: _____
51040	Hat Ihre Mutter eine Berufsausbildung abgeschlossen?	0 nein 1 ja
51050	In welchem Land?	Land: _____
51060 51070	Welche Ausbildung? Evtl. Schule/Schulart abfragen. <i>Wiederum je nach Land die entsprechende ISCED-Liste wählen.</i>	Ausbildung: _____ Schulart: _____
51080	Hat Ihre Mutter ein Hochschulstudium abgeschlossen?	0 nein 1 ja
51090	In welchem Land?	Land: _____
51100	An welcher Art von Hochschule?	Hochschule: _____
51110	Welches Studium?	Studienfach: _____
51120	Mit welchem Abschluss? Z.B. Bachelor, Master,... <i>Wiederum je nach Land die entsprechende ISCED-Liste wählen (Hochschulabschluss ist gewöhnlich ISCED 5).</i>	Abschluss: _____ _____
51130	Hat Ihre Mutter einen Dokortitel?	0 nein 1 ja
51140	In welchem Land wurde dieser erworben?	Land: _____



51150	In welchem Fach? <i>Wiederum je nach Land die entsprechende ISCED-Liste wählen (Dokortitel ist gewöhnlich ISCED 6).</i>	<i>Studienfach:</i> _____ _____
51160	Welche Tätigkeit führt Ihre Mutter momentan aus? <i>Wenn berufstätig: genauere Beschreibung der beruflichen Tätigkeit abfragen und notieren</i>	<ol style="list-style-type: none"> <li>1 Schüler/in an einer Primar- oder Sekundarschule</li> <li>2 Student/in an einer Hochschule</li> <li>3 Auszubildende/r (<i>in der Berufsausbildung, ohne 1 und 2</i>)</li> <li>4 berufstätig (<i>wenn Auszubildende/r: 3 ankreuzen</i>) <ol style="list-style-type: none"> <li>41 momentan arbeitend</li> <li>42 vorübergehend nicht arbeitend, wegen: _____ _____</li> </ol> <i>(z.B. Mutterschutz/Elternzeit, Krankschreibung, ...)</i> </li> <li>5 arbeitslos, arbeitssuchend</li> <li>6 Hausfrau/-mann (ausschließlich im eigenen Haushalt tätig)</li> <li>7 Rentner/in, Frührentner/in (Pensions- oder Rentenempfänger/in), auch Witwenrente, etc.</li> <li>8 Eigentümer/in, vom Vermögen lebend</li> <li>9 Dauerhaft erwerbsunfähig</li> <li>10 Sonstiges: _____</li> </ol>
51164		
51161		
51170	In welchem Land?	<i>Land:</i> _____

<b>52</b>	<b>Vater</b>	
52010	In welchem Land hat Ihr Vater seinen höchsten Schulabschluss gemacht? <i>Je nach Land wird die entsprechende Liste gewählt und nach ISCED kodiert.</i>	<i>Land:</i> _____
52020	Welchen höchsten allgemeinbildenden Schulabschluss hat Ihr Vater? <i>Evtl. noch</i>	<i>Abschluss:</i> _____
52030	<i>Schule/Schulart abfragen.</i>	<i>Schulart:</i> _____
52040	Hat Ihr Vater eine Berufsausbildung abgeschlossen?	0 nein 1 ja
52050	In welchem Land?	<i>Land:</i> _____
52060	Welche Ausbildung? <i>Evtl. Schule/Schulart abfragen.</i>	<i>Ausbildung:</i> _____
52070	<i>Wiederum je nach Land die entsprechende ISCED-Liste wählen.</i>	<i>Schulart:</i> _____
52080	Hat Ihr Vater ein Hochschulstudium abgeschlossen?	0 nein 1 ja
52090	In welchem Land?	<i>Land:</i> _____
52100	An welcher Art von Hochschule?	<i>Hochschule:</i> _____
52110	Welches Studium?	<i>Studienfach:</i> _____
52120	Mit welchem Abschluss? Z.B. Bachelor, Master,... <i>Wiederum je nach Land die entsprechende ISCED-Liste wählen (Hochschulabschluss ist gewöhnlich ISCED 5).</i>	<i>Abschluss:</i> _____
52130	Hat Ihr Vater einen Dokortitel?	0 nein 1 ja
52140	In welchem Land wurde dieser erworben?	<i>Land:</i> _____

52150	In welchem Fach? <i>Wiederum je nach Land die entsprechende ISCED-Liste wählen (Dokortitel ist gewöhnlich ISCED 6).</i>	<i>Studienfach:</i> _____ _____ _____
52160	Welche Tätigkeit führt Ihr Vater momentan aus? <i>Wenn berufstätig: genauere Beschreibung der beruflichen Tätigkeit abfragen und notieren</i>	1 Schüler/in an einer Primar- oder Sekundarschule 2 Student/in an einer Hochschule 3 Auszubildende/r ( <i>in der Berufsausbildung, ohne 1 und 2</i> ) 4 berufstätig ( <i>wenn Auszubildende/r: 3 ankreuzen</i> )
52164		41 momentan arbeitend 42 vorübergehend nicht arbeitend, wegen: _____ _____ <i>(z.B. Mutterschutz/Elternzeit, Krankschreibung, ...)</i> 5 arbeitslos, arbeitssuchend 6 Hausfrau/-mann (ausschließlich im eigenen Haushalt tätig) 7 Rentner/in, Frührentner/in (Pensions- oder Rentenempfänger/in), auch Witwenrente, etc. 8 Eigentümer/in, vom Vermögen lebend 9 Dauerhaft erwerbsunfähig 10 Sonstiges: _____
52161		
52170	In welchem Land?	<i>Land:</i> _____

53	Partner/in	
53010	In welchem Land hat Ihr/e Partner/in seinen/ihren höchsten Schulabschluss gemacht? <i>Je nach Land wird die entsprechende Liste gewählt und nach ISCED kodiert.</i>	Land: _____
53020 53030	Welchen höchsten allgemeinbildenden Schulabschluss hat Ihr/e Partner/in? Evtl. noch Schule/Schulart abfragen.	Abschluss: _____ Schulart: _____
53040	Hat Ihr/e Partner/in eine Berufsausbildung abgeschlossen?	0 nein 1 ja
53050	In welchem Land?	Land: _____
53060 53070	Welche Ausbildung? Evtl. Schule/Schulart abfragen. <i>Wiederum je nach Land die entsprechende ISCED-Liste wählen.</i>	Ausbildung: _____ Schulart: _____
53080	Hat Ihr/e Partner/in ein Hochschulstudium abgeschlossen?	0 nein 1 ja
53090	In welchem Land?	Land: _____
53100	An welcher Art von Hochschule?	Hochschule: _____
53110	Welches Studium?	Studienfach: _____
53120	Mit welchem Abschluss? Z.B. Bachelor, Master, ... <i>Wiederum je nach Land die entsprechende ISCED-Liste wählen (Hochschulabschluss ist gewöhnlich ISCED 5).</i>	Abschluss: _____ _____
53130	Hat Ihr/e Partner/in einen Dokortitel?	0 nein 1 ja
53140	In welchem Land wurde dieser erworben?	Land: _____

53150	<p>In welchem Fach?</p> <p><i>Wiederum je nach Land die entsprechende ISCED-Liste wählen (Dokortitel ist gewöhnlich ISCED 6).</i></p>	<p><i>Studienfach:</i> _____</p>
53160	<p>Welche Tätigkeit führt Ihr/e Partner/in momentan aus?</p> <p><i>Wenn berufstätig: genauere Beschreibung der beruflichen Tätigkeit abfragen und notieren</i></p>	<p>1 Schüler/in an einer Primar- oder Sekundarschule</p> <p>2 Student/in an einer Hochschule</p> <p>3 Auszubildende/r (<i>in der Berufsausbildung, ohne 1 und 2</i>)</p> <p>4 berufstätig (<i>wenn Auszubildende/r: 3 ankreuzen</i>)</p>
53164		<p>41 momentan arbeitend</p> <p>42 vorübergehend nicht arbeitend, wegen: _____</p> <p>_____</p> <p><i>(z.B. Mutterschutz/Elternzeit, Krankschreibung, ...)</i></p> <p>5 arbeitslos, arbeitssuchend</p> <p>6 Hausfrau/-mann (ausschließlich im eigenen Haushalt tätig)</p> <p>7 Rentner/in, Frührentner/in (Pensions- oder Rentenempfänger/in), auch Witwenrente, etc.</p> <p>8 Eigentümer/in, vom Vermögen lebend</p> <p>9 Dauerhaft erwerbsunfähig</p> <p>10 Sonstiges: _____</p>
53161		
53170	<p>In welchem Land?</p>	<p><i>Land:</i> _____</p>

<b>54</b>	<b>Auswertung des SÖS nach ISCED</b>
	<i>Tragen Sie hier die oben ermittelten ISCED-Werte ein!</i>
	<i>In Ausbildung/ohne eigenes Einkommen/bei den Eltern lebend:</i>
54010	<i>ISCED Mutter: __</i>
54020	<i>ISCED Vater: __</i>
	<i>Nicht mehr vom elterlichen Haushalt abhängig (eigener Haushalt):</i>
54030	<i>ISCED Teilnehmer/in: __</i>
54040	<i>ISCED Partner/in: __</i>
54050	<i>Höchster ISCED im Haushalt = SÖS = ____</i>

**Kategorie 6: Gesundheit**

61	Ausschlusskriterien	
61010	<p>Sind Sie in Ihrer Sehfähigkeit eingeschränkt?            Tragen Sie eine Brille oder Kontaktlinsen?  <i>Wenn ja: Ist die Sehfähigkeit zum jetzigen Zeitpunkt bzw. zum Zeitpunkt der Teilnahme durch eine Brille oder Kontaktlinsen korrigiert?</i></p>	<p>0 nein            1 ja, eingeschränkt aber korrigiert            2 ja, eingeschränkt und <u>nicht</u> korrigiert  <i>Entscheidend ist, dass die Sehfähigkeit zum Zeitpunkt der Testung korrigiert ist, nicht zum Zeitpunkt des Interviews.</i></p>
61020	<p>Sind Sie farbenblind?  <i>Achtung: bei Präsentation von farbigen Bildern</i></p>	<p>0 nein            1 ja</p>
61050	<p>Haben Sie Allergien oder Unverträglichkeiten, z.B. Erdnussallergie, Laktoseunverträglichkeit, Hautallergien?  <i>Es sind alle Allergien gemeint, nicht nur in Bezug auf Nahrungsmittel!</i></p>	<p>0 nein            1 ja</p>
61051	<p>Welche?  <i>Hautallergien: Vorsicht bei Elektroden</i>  <i>Nahrungsmittelallergien: Vorsicht bei Studien mit Nahrungsaufnahme</i></p>	<p>Allergie(n): _____            _____            _____            _____</p>
61060	<p>Gibt es Nahrungsmittel, die Sie aus Prinzip nicht essen, z.B. Fleisch?  <i>Hierunter fallen Vegetarier, Veganer, Muslime die kein Schweinefleisch essen, etc.</i></p>	<p>0 nein            1 ja</p>
61061	<p>Welche Nahrungsmittel sind es, die Sie nicht essen?  <i>Fragen Sie möglichst genau nach!</i>  <i>Achtung: bei Studien mit Nahrungsaufnahme</i></p>	<p>Nahrungsmittel: _____            _____            _____            _____</p>
61080	<p>Leiden Sie momentan unter chronischen oder akuten körperlichen Erkrankungen?</p>	<p>0 nein            1 ja</p>

61081	<p>An welchen?</p> <p><i>Chronisch: dauert meist mehrere Jahre an, mind. 4 Wochen (z.B. Herz-Kreislaufkrankungen, Asthma, Epilepsie, ...)</i></p> <p><i>Akut: dauert nur eine kurze Zeit an, meist bis 14 Tage (z.B. Erkältung)</i></p> <p><i>Achtung bei Hauterkrankungen, neurologischen Erkrankungen, Herz-Kreislauf-Erkrankungen (HRV!)</i></p>	<p>Erkrankung(en): _____</p> <p>_____</p> <p>_____</p> <p>_____</p> <p>_____</p> <p>_____</p> <p>_____</p>
61090	<p>Hatten Sie jemals einen epileptischen Anfall?</p> <p><i>Achtung: Keine Teilnahme an Studien mit Bildpräsentation möglich (kann Anfall auslösen)</i></p>	<p>0 nein</p> <p>1 ja</p>

<b>62</b>	<b>Psychische Störungen und Psychopharmaka</b>	
62010	<p>Wurde bei Ihnen jemals eine psychische Störung diagnostiziert?</p>	<p>0 nein</p> <p>1 ja</p>
<p><i>Die folgenden Fragen in dieser Unterkategorie werden nur abgefragt, wenn bei der ersten Frage (62010) mit „ja“ geantwortet wurde. Wenn mit „nein“ geantwortet wurde, springen Sie zur nächsten Unterkategorie.</i></p> <p><i>Wenn mehrere psychische Erkrankungen oder Episoden angegeben werden, fragen Sie alle ab.</i></p>		
62020	<p>Welche war das?</p>	<p>Störung: _____</p>
62030	<p>Wann war das?</p>	<p>Jahr: _____</p>
62040	<p>Waren Sie aufgrund dieser Erkrankung in Behandlung?</p>	<p>0 nein</p> <p>1 ja</p>
62050	<p>Wurde die Behandlung erfolgreich abgeschlossen?</p>	<p>0 nein, immer noch in Behandlung</p> <p>1 ja, erfolgreich abgeschlossen</p> <p>2 abgeschlossen, aber ohne Erfolg</p>
62060	<p>Nahmen Sie aufgrund der Erkrankung Medikamente ein? <i>Gemeint sind Psychopharmaka</i></p>	<p>0 nein</p> <p>1 ja</p>



	<p>Welche waren das?  <i>Möglichst genaue Angaben: Name des Produkts oder Wirkstoff (soweit bekannt)</i></p> <p>In welcher Dosierung? Also, welche Menge nahmen Sie ein und wie oft? <i>Falls bekannt. Möglichst Angabe in ml oder mg. Bei Tabletten o. Ä. nachfragen, wie viel 1 Tablette in mg ist.</i></p> <p><i>Wenn nicht bekannt -&gt; Zuhause nachschauen lassen und nachtragen</i></p>	<p><i>Tragen Sie die Antworten unter den nachfolgenden Items ein.</i></p> <p><i>Wenn mehr Medikamente eingenommen wurden, als Items vorhanden sind, nummerieren Sie die zusätzlichen Items nach dem Muster der vorhandenen.</i></p>
62070	<i>Name Medikament 1</i>	
62071	<i>Dosierung Medikament 1</i>	
62080	<i>Name Medikament 2</i>	
62081	<i>Dosierung Medikament 2</i>	
62090	<i>Name Medikament 3</i>	
62091	<i>Dosierung Medikament 3</i>	
62100	Leiden Sie gegenwärtig unter dieser Erkrankung?	<p>0 nein</p> <p>1 ja</p>
62110	Nehmen Sie gegenwärtig Medikamente aufgrund dieser Erkrankung ein? <i>Gemeint sind Psychopharmaka.</i>	<p>0 nein</p> <p>1 ja</p>
	<p>Welche?  <i>Möglichst genaue Angaben: Name des Produkts oder Wirkstoff (soweit bekannt)</i></p> <p>In welcher Dosierung? Also, welche Menge nehmen Sie ein und wie oft?  <i>Möglichst Angabe in ml oder mg. Bei Tabletten o. Ä. nachfragen, wie viel 1 Tablette in mg ist.</i></p> <p><i>Wenn nicht bekannt -&gt; Zuhause nachschauen lassen und nachtragen</i></p>	<p><i>Tragen Sie die Antworten unter den nachfolgenden Items ein.</i></p> <p><i>Wenn mehr Medikamente eingenommen werden, als Items vorhanden sind, nummerieren Sie die zusätzlichen Items nach dem Muster der vorhandenen.</i></p>
62120	<i>Name Medikament 1</i>	

62121	<i>Dosierung Medikament 1</i>	
62130	<i>Name Medikament 2</i>	
62131	<i>Dosierung Medikament 2</i>	
62140	<i>Name Medikament 3</i>	
62141	<i>Dosierung Medikament 3</i>	

<b>63</b>	<b>Frühere Essstörungen und Psychopharmaka</b>	
63010	Wurde bei Ihnen jemals eine Essstörung diagnostiziert?	0 nein 1 ja
	<p><i>Die folgenden Fragen in dieser Unterkategorie werden nur abgefragt, wenn bei der ersten Frage (63010) mit ja geantwortet wurde. Wenn mit nein geantwortet wurde, springen Sie zur nächsten Unterkategorie.</i></p> <p><i>Wenn mehrere Essstörungen oder Episoden angegeben werden, fragen Sie alle ab.</i></p>	
63020	Welche war das? <i>Wenn die Teilnehmenden sich an die genaue Diagnose oder sogar die DSM oder ICD Kodierung erinnern, notieren Sie diese! Subtypen müssen nicht abgefragt werden, wenn dies für die Studie nicht relevant ist.</i>	1 Anorexia Nervosa 11 Restriktiver Typus 12 Binge-Eating/Purging-Typus 2 Bulimia Nervosa 21 Purging 22 Non-Purging 3 Binge-Eating-Störung 4 Essstörung NNB (EDNOS) 5 Sonstige: _____
63025		
63030	Wann war das?	<i>Jahr:</i> _____
63040	Wer hat diese Essstörung bei Ihnen diagnostiziert?	1 Psychiater 2 Psychologe 3 Hausarzt/Allgemeinmediziner 4 Neurologe 5 Ernährungsberater 6 Andere Person: _____
63046		

63050	Waren Sie aufgrund dieser Erkrankung in Behandlung?	0 nein 1 ja
63060	Wurde die Behandlung erfolgreich abgeschlossen?	0 nein, immer noch in Behandlung 1 ja, erfolgreich abgeschlossen 2 abgeschlossen, aber ohne Erfolg
63070	Nahmen Sie aufgrund der Erkrankung Medikamente ein? <i>Gemeint sind Psychopharmaka</i>	0 nein 1 ja
	<p>Welche waren das?  <i>Möglichst genaue Angaben: Name des Produkts oder Wirkstoff (soweit bekannt)</i>  In welcher Dosierung? Also, welche Menge nahmen Sie ein und wie oft? <i>Falls bekannt.</i>  <i>Möglichst Angabe in ml oder mg. Bei Tabletten o. Ä. nachfragen, wie viel 1 Tablette in mg ist.</i>  <i>Wenn nicht bekannt -&gt; Zuhause nachschauen lassen und nachtragen</i></p>	<p><i>Tragen Sie die Antworten unter den nachfolgenden Items ein.</i>  <i>Wenn mehr Medikamente eingenommen wurden, als Items vorhanden sind, nummerieren Sie die zusätzlichen Items nach dem Muster der vorhandenen.</i></p>
63080	<i>Name Medikament 1</i>	
63081	<i>Dosierung Medikament 1</i>	
63090	<i>Name Medikament 2</i>	
63091	<i>Dosierung Medikament 2</i>	
63100	<i>Name Medikament 3</i>	
63101	<i>Dosierung Medikament 3</i>	
63110	Leiden Sie gegenwärtig unter dieser Erkrankung?	0 nein 1 ja
63120	Nehmen Sie gegenwärtig Medikamente aufgrund dieser Erkrankung ein? <i>Gemeint sind Psychopharmaka.</i>	0 nein 1 ja

	<p>Welche?  Möglichst genaue Angaben: Name des Produkts oder Wirkstoff (soweit bekannt)  In welcher Dosierung? Also, welche Menge nehmen Sie ein und wie oft?  Möglichst Angabe in ml oder mg. Bei Tabletten o. Ä. nachfragen, wie viel 1 Tablette in mg ist.  Wenn nicht bekannt -&gt; Zuhause nachschauen lassen und nachtragen</p>	<p><i>Tragen Sie die Antworten unter den nachfolgenden Items ein.</i>  <i>Wenn mehr Medikamente eingenommen werden, als Items vorhanden sind, nummerieren Sie die zusätzlichen Items nach dem Muster der vorhandenen.</i></p>
63130	Name Medikament 1	
63131	Dosierung Medikament 1	
63140	Name Medikament 2	
63141	Dosierung Medikament 2	
63150	Name Medikament 3	
63151	Dosierung Medikament 3	

<b>64</b>	<b>Weitere Medikamente</b>	
64010	<p>Nehmen Sie momentan regelmäßig Medikamente ein? <i>Ohne Psychopharmaka und hormonelle Empfängnisverhütung (Pille).</i></p>	<p>0 nein  1 ja</p>
	<p>Welche?  Möglichst genaue Angaben: Name des Produkts oder Wirkstoff (soweit bekannt)  In welcher Dosierung? Also, welche Menge nehmen Sie ein und wie oft?  Möglichst Angabe in ml oder mg. Bei Tabletten o. Ä. nachfragen, wie viel 1 Tablette in mg ist.  Wenn nicht bekannt -&gt; Zuhause nachschauen lassen und nachtragen</p>	<p><i>Tragen Sie die Antworten unter den nachfolgenden Items ein.</i>  <i>Wenn mehr Medikamente eingenommen werden, als Items vorhanden sind, nummerieren Sie die zusätzlichen Items nach dem Muster der vorhandenen.</i></p>
64020	Name Medikament 1	

64021	<i>Dosierung Medikament 1</i>	
64030	<i>Name Medikament 2</i>	
64031	<i>Dosierung Medikament 2</i>	
64040	<i>Name Medikament 3</i>	
64041	<i>Dosierung Medikament 3</i>	

<b>65</b>	<b>Sport</b>	
65000	<i>Fragen Sie den IPAQ ab. Notieren Sie evtl.</i>	<i>MET-Minuten/Woche: _____</i>
65001	<i>Wichtiges hier. Halten Sie einen Taschenrechner bereit zur Umrechnung von Stunden in Minuten.</i>	<i>Aktivitätskategorie:</i> 1 niedrig 2 mittel 3 hoch

**VORLESEN:** Ich werde Sie zu der Zeit befragen, die Sie während der letzten 7 Tage in körperlicher Aktivität verbracht haben. Bitte beantworten Sie alle Fragen, auch wenn Sie sich selbst nicht als aktive Person ansehen. Bitte berücksichtigen Sie die Aktivitäten im Rahmen Ihrer Arbeit/Schule/Studium, in Haus und Garten, um von einem Ort zum anderen zu gelangen und in Ihrer Freizeit für Erholung, Bewegung und Sport.

**VORLESEN:** Denken Sie nun an alle *anstrengenden* Aktivitäten, die Sie in den vergangenen 7 Tagen ausgeführt haben. Anstrengende Aktivitäten bezeichnen Aktivitäten, die *starke körperliche* Anstrengungen erfordern und bei denen Sie deutlich stärker atmen als normal. Dies beinhaltet z.B. Aktivitäten wie das Tragen schwerer Lasten, Erdarbeiten, Aerobic oder schnelles Fahrradfahren. Denken Sie nur an die körperlichen Aktivitäten, die Sie für mindestens 10 Minuten ohne Unterbrechung ausgeführt haben.

65010	1. Während der <b>letzten 7 Tage</b> , an wie vielen Tagen haben Sie <b>anstrengende</b> körperliche Aktivitäten ausgeführt?	__ Tage pro Woche [Range 0-7, 888, 999] 888 weiß nicht 999 keine Angabe
-------	--	---

**[Interviewer Verdeutlichung:** Denken Sie nur an die körperlichen Aktivitäten, die Sie für mindestens 10 Minuten ohne Unterbrechung ausführen.]

**[Interviewer Anmerkung:** Wenn der/die Befragte mit null antwortet, sich weigert zu antworten oder die Antwort nicht weiß, springen Sie zu Frage 3.]

65020	2. Wie viel Zeit insgesamt haben Sie für gewöhnlich an einem dieser Tage mit <b>anstrengenden</b> körperlichen Aktivitäten verbracht?	__ __ Stunden pro Tag [Range 0-16] __ __ __ Minuten pro Tag [Range 0-960, 888, 999] 888 weiß nicht 999 keine Angabe <i>Nur die Minuten werden in SPSS eingegeben -&gt; umrechnen!</i>
-------	---	---

**[Interviewer Verdeutlichung:** Denken Sie nur an die körperlichen Aktivitäten, die Sie für mindestens 10 Minuten ohne Unterbrechung ausgeführt haben.]

65021	<b>[Interviewer Klarstellung:</b> Wir suchen eine durchschnittliche Zeit für einen der Tage, an denen Sie anstrengende körperliche Aktivitäten ausführen. Wenn der/die Befragte nicht antworten kann, da die Zeitverteilung sehr stark von Tag zu	__ __ Stunden pro Woche [Range 0-112] __ __ __ Minuten pro Woche [Range 0-6720, 888, 999] 888 weiß nicht 999 keine Angabe
-------	---	--

	Tag variiert, fragen Sie: „Wie viel Zeit haben Sie insgesamt über <b>die letzten 7 Tage</b> mit anstrengenden körperlichen Aktivitäten verbracht?“	<i>Nur die Minuten werden in SPSS eingegeben -&gt; umrechnen!</i>
--	--	---

**VORLESEN: Denken Sie nun an alle moderaten Aktivitäten, die Sie in den vergangenen 7 Tagen ausgeführt haben. Moderate Aktivitäten bezeichnen Aktivitäten mit moderater körperlicher Anstrengung, bei denen Sie ein wenig stärker atmen als normal. Dies beinhaltet z.B. Aktivitäten wie das Tragen leichter Lasten, Fahrradfahren bei gewöhnlicher Geschwindigkeit oder Doppel-Tennis. Schliessen Sie Gehen nicht mit ein. Denken Sie wiederum nur an die körperlichen Aktivitäten, die Sie für mindestens 10 Minuten ohne Unterbrechung ausgeführt haben.**

65030	3. Während der <b>letzten 7 Tage</b> , an wie vielen Tagen haben Sie <b>moderate</b> körperliche Aktivitäten ausgeführt?	__ Tage pro Woche [Range 0-7, 888, 999] 888 weiß nicht 999 keine Angabe
-------	--	---

**[Interviewer Verdeutlichung:** Denken Sie nur an die körperlichen Aktivitäten, die Sie für mindestens 10 Minuten ohne Unterbrechung ausführen.]

**[Interviewer Anmerkung:** Wenn der/die Befragte mit null antwortet, sich weigert zu antworten oder die Antwort nicht weiß, springen Sie zu Frage 5.]

65040	4. Wie viel Zeit insgesamt haben Sie für gewöhnlich an einem dieser Tage mit <b>moderaten</b> körperlichen Aktivitäten verbracht?	__ __ Stunden pro Tag [Range 0-16] __ __ __ Minuten pro Tag [Range 0-960, 888, 999] 888 weiß nicht 999 keine Angabe <i>Nur die Minuten werden in SPSS eingegeben -&gt; umrechnen!</i>
-------	---	---

**[Interviewer Verdeutlichung:** Denken Sie nur an die körperlichen Aktivitäten, die Sie für mindestens 10 Minuten ohne Unterbrechung ausführen.]

65041	<b>[Interviewer Klarstellung:</b> Wir suchen eine durchschnittliche Zeit für einen der Tage, an denen Sie moderate körperliche Aktivitäten ausführen. Wenn der/die Befragte nicht antworten kann, da die Zeitverteilung sehr stark von Tag zu	__ __ Stunden pro Woche [Range 0-112] __ __ __ __ Minuten pro Woche [Range 0-6720, 888, 999] 888 weiß nicht 999 keine Angabe
-------	---	---

	Tag variiert, fragen Sie: „Wie viel Zeit haben Sie insgesamt über <b>die letzten 7 Tage</b> mit moderaten körperlichen Aktivitäten verbracht?“	<i>Nur die Minuten werden in SPSS eingegeben -&gt; umrechnen!</i>
--	--	---

**VORLESEN: Denken Sie nun an die Zeit, die Sie in den letzten 7 Tagen mit zu Fuß gehen verbracht haben. Dies schliesst das zu Fuss gehen auf der Arbeit und zu Hause, das Gehen, um von einem Ort zum anderen zu gelangen, und alles andere Gehen, was Sie ausschliesslich als Entspannung, Sport, Bewegung und Freizeit ausgeführt haben, ein.**

65050	5. Während der vergangenen 7 Tage, an wie vielen Tagen sind Sie mindestens 10 Minuten ohne Unterbrechung <b>zu Fuss gegangen</b> ?	__ Tage pro Woche [Range 0-7, 888, 999] 888 weiß nicht 999 keine Angabe
-------	--	---

**[Interviewer Verdeutlichung:** Denken Sie nur an das zu Fuß gehen, das Sie für mindestens 10 Minuten ohne Unterbrechung ausgeführt haben.]

**[Interviewer Anmerkung:** Wenn der/die Befragte mit null antwortet, sich weigert zu antworten oder die Antwort nicht weiß, springen Sie zu Frage 7.]

65060	6. Wie viel Zeit haben Sie für gewöhnlich an einem dieser Tage mit <b>zu Fuss gehen</b> verbracht?	__ __ Stunden pro Tag [Range 0-16] __ __ __ Minuten pro Tag [Range 0-960, 888, 999] 888 weiß nicht 999 keine Angabe <i>Nur die Minuten werden in SPSS eingegeben -&gt; umrechnen!</i>
-------	--	---

65061	<b>[Interviewer Klarstellung:</b> Wir suchen eine durchschnittliche Zeit für einen der Tage, an denen Sie zu Fuß gehen. Wenn der/die Befragte nicht antworten kann, da die Zeitverteilung sehr stark von Tag zu Tag variiert, fragen Sie: „Wie	__ __ Stunden pro Woche [Range 0-112] __ __ __ Minuten pro Woche [Range 0-6720, 888, 999] 888 weiß nicht 999 keine Angabe
-------	--	--



	viel Zeit haben Sie insgesamt über <b>die letzten 7 Tage</b> mit zu Fuß gehen verbracht?“	<i>Nur die Minuten werden in SPSS eingegeben -&gt; umrechnen!</i>
--	---	---

**VORLESEN: Denken Sie nun an die Zeit, die Sie während der letzten 7 Tage an Wochentagen sitzend verbracht haben, z.B. bei der Arbeit, zu Hause, beim Studieren und in der Freizeit. Dies schliesst ein das Sitzen am Schreibtisch, den Besuch bei Freunden, das Lesen oder das Sitzen oder Liegen vor dem Fernseher.**

65070	7. Während der vergangenen 7 Tage, wie viel Zeit haben Sie für gewöhnlich an einem <b>Wochentag</b> im <b>Sitzen</b> verbracht?	__ __ Stunden pro Tag [Range 0-16] __ __ __ Minuten pro Tag [Range 0-960, 888, 999] 888 weiß nicht 999 keine Angabe <i>Nur die Minuten werden in SPSS eingegeben -&gt; umrechnen!</i>
-------	---	---

**[Interviewer Verdeutlichung:** Schließen Sie Zeit, in der Sie gelegen haben (und dabei wach waren), ebenso ein wie Sitzen.]

65071	<b>[Interviewer Klarstellung:</b> Wir suchen eine durchschnittliche Zeit, die Sie pro Tag sitzend verbringen. Wenn der/die Befragte nicht antworten kann, da die Zeitverteilung sehr stark von Tag zu Tag variiert, fragen Sie: „Wie viel Zeit haben Sie insgesamt letzten <b>Mittwoch</b> mit <b>Sitzen</b> verbracht?“	__ __ Stunden am Mittwoch [Range 0-16] __ __ __ Minuten am Mittwoch [Range 0-960, 888, 999] 888 weiß nicht 999 keine Angabe <i>Nur die Minuten werden in SPSS eingegeben -&gt; umrechnen!</i>
-------	--	---

### **IPAQ-Auswertung – Berechnung von MET-Minuten/Woche:**

MET-Minuten/Woche Gehen =  $3.3 * \text{Minuten Gehen} * \text{Tage Gehen}$

MET-Minuten/Woche moderat =  $4.0 * \text{Minuten moderate Aktivität} * \text{Tage moderate Aktivität}$

MET-Minuten/Woche anstrengend =  $8.0 * \text{Minuten anstrengende Aktivität} * \text{Tage anstrengende Aktivität}$

Gesamtscore physische Aktivität in MET-Minuten/Woche = Summe von MET-Minuten/Woche Gehen + moderat + anstrengend

### **IPAQ-Auswertung – Kategorien:**

#### **Kategorie 1: niedrig**

Niedrigstes Level physischer Aktivität. In diese Kategorie fallen Personen, die die Kriterien für die Kategorien 2 und 3 nicht erfüllen.

#### **Kategorie 2: mittel**

Ein moderates Aktivitätsmuster wird anhand der folgenden Kriterien definiert:

a) 3 oder mehr Tage mit anstrengender Aktivität von mindestens 20 Minuten am Tag

**oder**

b) 5 oder mehr Tage mit moderater Aktivität und/oder Gehen von mindestens 30 Minuten pro Tag

**oder**

c) 5 oder mehr Tage mit einer beliebigen Kombination aus Gehen, moderaten oder anstrengenden Aktivitäten von insgesamt mindestens 600 MET-Minuten/Woche

Bei Personen, bei denen mindestens eines dieser Kriterien zutrifft, geht man von einem Mindestmaß an Aktivität aus und klassifiziert sie deshalb als „moderat“.

#### **Kategorie 3: hoch**

Diese Kategorie wird zur Beschreibung von hohem Aktivitätsniveau verwendet.

Die beiden Kriterien zur Klassifikation sind:

a) anstrengende Aktivität an mindestens 3 Tagen mit einer Gesamtaktivität von mindestens 1500 MET-Minuten/Woche

**oder**

b) 7 oder mehr Tage mit einer beliebigen Kombination aus Gehen, moderaten oder anstrengenden Aktivitäten von insgesamt mindestens 3000 MET-Minuten/Woche

<b>66</b>	<b>Substanzkonsum</b>	
66010	Rauchen Sie?	0 nein 1 ja
66011	Wie viele Zigaretten rauchen Sie durchschnittlich am Tag?	Zigaretten/Tag: _____ Ggf. umrechnen, z.B. 1 in der Woche -> 0,14 pro Tag
66030	Wie oft trinken Sie gewöhnlich Alkohol?	_____ mal pro Woche Ggf. umrechnen, z.B. 1 mal im Monat -> 0,25 mal pro Woche. Kodieren Sie nur mit 0, wenn die Person angibt, überhaupt nie Alkohol zu trinken (1 mal im Jahr = 0,0027)
66080	Nehmen Sie andere Drogen, z.B. Cannabis?	0 nein 1 ja
66081	Welche Drogen?	Droge(n): _____
66082	Wie oft?	_____ mal pro Woche

<b>67</b>	<b>Gewicht und Diät</b>	
67010	Was war Ihr <u>niedrigstes</u> (Erwachsenen-) Gewicht?	Gewicht [kg]: _ _ _
67020	Wann hatten Sie dieses Gewicht?	Jahr: _ _ _ _
67030	Das war also vor _____ Monaten? Ggf. im Nachhinein berechnen.	Monate: _ _ _
67040	Was war Ihr <u>höchstes</u> (Erwachsenen-) Gewicht?	Gewicht [kg]: _ _ _
67050	Wann hatten Sie dieses Gewicht?	Jahr: _ _ _ _
67060	Das war also vor _____ Monaten? Ggf. im Nachhinein berechnen.	Monate: _ _ _
67070	Haben Sie bereits an einer ambulanten Ernährungsberatung teilgenommen?	0 nein 1 ja
67080	Wie häufig?	_____ mal

67090	Wann haben Sie zum ersten Mal an einer ambulanten Ernährungsberatung teilgenommen?	Jahr: _____ z.B. 1993										
67100	Wann haben Sie zum letzten Mal an einer ambulanten Ernährungsberatung teilgenommen?	Jahr: _____ z.B. 2010										
67110	Wie viel würden Sie gerne wiegen? Was ist Ihr Idealgewicht?	Gewicht [kg]: _____										
67120	Machen Sie momentan eine Diät oder versuchen Sie, sich beim Essen einzuschränken, um Ihr Gewicht oder Ihre Figur zu beeinflussen?	0 nein 1 ja										
Wie häufig in Ihrem Leben haben Sie die folgende Anzahl an Kilogramm absichtlich abgenommen?												
67130	bis 5 kg	0x	1x	2x	3x	4x	5x	6x	7x	8x	9x	≥10x
67140	6 bis 10 kg	0x	1x	2x	3x	4x	5x	6x	7x	8x	9x	≥10x
67150	11 bis 20 kg	0x	1x	2x	3x	4x	5x	6x	7x	8x	9x	≥10x
67160	21 bis 30 kg	0x	1x	2x	3x	4x	5x	6x	7x	8x	9x	≥10x
67170	31 bis 40 kg	0x	1x	2x	3x	4x	5x	6x	7x	8x	9x	≥10x
67180	mehr als 40 kg	0x	1x	2x	3x	4x	5x	6x	7x	8x	9x	≥10x
67200	Wenn Sie abgenommen haben, wie lange haben Sie dieses Gewicht gewöhnlich gehalten?	Zeitdauer: _____ Wochen										

### **Kategorie 7: weibliche Gesundheit**

<b>71</b>	<b>Nur für Frauen: Menstruation und Verhütung</b>											
71010	Wann begann Ihre letzte Periode? (Vor der Studienteilnahme)	Datum: ____ / ____ / _____ (dd/mm/yyyy)										
71030	Verwenden Sie eine hormonelle Empfängnisverhütung (z.B. <i>Beispiele siehe nächstes Item</i> )?	0 nein 1 ja										

71040	Welche Art von hormoneller Empfängnisverhütung?	1 Antibabypille ( <i>enthält Östrogen und Gestagene</i> ) 2 Minipille ( <i>enthält Gestagene</i> ) 3 Dreimonatsspritze 4 Vaginalring 5 Hormonpflaster 6 Verhütungsstäbchen (Hormonimplantat) 7 Hormonspirale 8 Sonstige: _____
71048		
71050	Wie ist der Name des Verhütungsmittels? <i>Möglichst genaue Bezeichnung des Präparats</i>	<i>Präparat:</i> _____
71060	Mit welchem Wirkstoff? <i>Wenn nicht bekannt, im Nachhinein anhand des Präparatnamens nachschlagen.</i>	<i>Wirkstoff:</i> _____
71070	Sind Sie momentan schwanger?	0 nein 1 ja
71080	Sind Sie in der (Post-)Menopause?	0 nein 1 ja

**Kategorie 8: Fragen zum heutigen Tag**

<i>Diese Fragen beziehen sich, wenn nicht anders angegeben, auf die letzten 24 Stunden.</i>		
80010	Wie lange haben Sie in der letzten Nacht geschlafen?	Stunden: __ __
80020	Wie lange schlafen Sie für gewöhnlich?	Stunden: __ __
80030	Wann haben Sie, bevor Sie hierher kamen, zum letzten Mal etwas gegessen?	Uhrzeit: __ __ : __ __ (hh:mm, 24-Std.-Format)
80040	<i>Zeitspanne vom Beginn der Testung bis zur letzten Mahlzeit davor berechnen.</i> (Das war also vor ____ Stunden/Minuten?)	__ __ : __ __ (hh:mm)
80050	Was haben Sie gegessen und wie viel?	<i>Gegessene Nahrungsmittel und Menge notieren</i> _____ _____ _____ _____
80060	<i>Wenn keine richtige Mahlzeit angegeben wird, sondern z.B. nur ein Apfel:</i> Wann haben Sie, bevor Sie hierher kamen, zum letzten Mal eine richtige Mahlzeit gegessen?	Uhrzeit: __ __ : __ __ (hh:mm, 24-Std.-Format)
80070	Was haben Sie gegessen und wie viel?	<i>Gegessene Nahrungsmittel und Menge notieren</i> _____ _____ _____ _____
80080	Haben Sie in den letzten 24 Stunden koffeinhaltige Getränke getrunken, z.B. Kaffee, schwarzer oder grüner Tee, Cola, Red Bull?	0 nein 1 ja
80090	Wann haben Sie, bevor Sie hierher kamen, zum letzten Mal ein koffeinhaltiges Getränk zu sich genommen?	Uhrzeit: __ __ : __ __ (hh:mm, 24-Std.-Format)

80100	<i>Zeitspanne vom Beginn der Testung bis zum letzten koffeinhaltigen Getränk davor berechnen.</i> (Das war also vor ____ Stunden/Minuten?)	__ __ : __ __ (hh:mm)
80110	Welches Getränk/ welche Getränke?	Getränk(e): _____
80120	Wie viel?	Menge: __ __ , __ __ l
80130	Haben Sie in den letzten 24 Stunden alkoholhaltige Getränke getrunken?	0 nein 1 ja
80140	Wann haben Sie, bevor Sie hierher kamen, zum letzten Mal ein alkoholhaltiges Getränk zu sich genommen?	Uhrzeit: __ __ : __ __ (hh:mm, 24-Std.-Format)
80150	<i>Zeitspanne vom Beginn der Testung bis zum letzten alkoholhaltigen Getränk davor berechnen.</i> (Das war also vor ____ Stunden/Minuten?)	__ __ : __ __ (hh:mm)
80160	Welches Getränk/ welche Getränke?	Getränk(e): _____
80170	Wie viel?	Menge, möglichst in ml: __ __ __ __
80180	Haben Sie in den letzten 24 Stunden Zigaretten geraucht oder in anderer Form Nikotin zu sich genommen?	0 nein 1 ja
80190	Wann haben Sie, bevor Sie hierher kamen, zum letzten Mal eine Zigarette geraucht oder in anderer Form Nikotin zu sich genommen?	Uhrzeit: __ __ : __ __ (hh:mm, 24-Std.-Format)
80200	<i>Zeitspanne vom Beginn der Testung bis zur letzten Zigarette davor berechnen.</i> (Das war also vor ____ Stunden/Minuten?)	__ __ : __ __ (hh:mm)
80210	Haben Sie innerhalb der letzten 24 Stunden Cannabis oder andere Drogen eingenommen?	0 nein 1 ja
80220	Welche?	Bezeichnung: _____
80230	Wann war das?	Uhrzeit: __ __ : __ __ (hh:mm, 24-Std.-Format)

80240	<i>Zeitspanne vom Beginn der Testung bis zur letzten Drogeneinnahme davor berechnen. (Das war also vor ____ Stunden/Minuten?)</i>	__ __ : __ __ (hh:mm)
80210	Haben Sie in den letzten 4 Tagen sonstige Medikamente eingenommen?	0 nein 1 ja
	Welche Medikamente waren das? In welcher Dosierung haben Sie diese eingenommen? Wann haben Sie diese zum letzten Mal eingenommen? <i>Wenn nicht bekannt -&gt; Zuhause Nachschauen lassen und nachtragen!</i>	
80220	<i>Name/Präparat</i>	_____
80221	<i>Dosierung</i>	__ __ __ __ ml/mg (angeben!)
80222	<i>Letzte Einnahme Datum</i>	<i>Datum:</i> __ __ / __ __ / __ __ __ __ (dd/mm/yyyy)
80223	<i>Letzte Einnahme Uhrzeit</i>	<i>Uhrzeit:</i> __ __ : __ __ (hh:mm, 24-Std.-Format)
80230	<i>Name/Präparat</i>	_____
80231	<i>Dosierung</i>	__ __ __ __ ml/mg (angeben!)
80232	<i>Letzte Einnahme Datum</i>	<i>Datum:</i> __ __ / __ __ / __ __ __ __ (dd/mm/yyyy)
80233	<i>Letzte Einnahme Uhrzeit</i>	<i>Uhrzeit:</i> __ __ : __ __ (hh:mm, 24-Std.-Format)
80240	<i>Name/Präparat</i>	_____
80241	<i>Dosierung</i>	__ __ __ __ ml/mg (angeben!)
80242	<i>Letzte Einnahme Datum</i>	<i>Datum:</i> __ __ / __ __ / __ __ __ __ (dd/mm/yyyy)
80243	<i>Letzte Einnahme Uhrzeit</i>	<i>Uhrzeit:</i> __ __ : __ __ (hh:mm, 24-Std.-Format)
80250	<i>Name/Präparat</i>	_____
80251	<i>Dosierung</i>	__ __ __ __ ml/mg (angeben!)
80252	<i>Letzte Einnahme Datum</i>	<i>Datum:</i> __ __ / __ __ / __ __ __ __ (dd/mm/yyyy)
80253	<i>Letzte Einnahme Uhrzeit</i>	<i>Uhrzeit:</i> __ __ : __ __ (hh:mm, 24-Std.-Format)



80260	<i>Name/Präparat</i>	_____
80261	<i>Dosierung</i>	_____ ml/mg ( <i>angeben!</i> )
80262	<i>Letzte Einnahme Datum</i>	<i>Datum:</i> ____ / ____ / ____ (dd/mm/yyyy)
80263	<i>Letzte Einnahme Uhrzeit</i>	<i>Uhrzeit:</i> ____ : ____ (hh:mm, 24-Std.-Format)

## **APPENDIX H**

**Information brochure that was distributed to participants of the**

**ATTEND study before participation (Study 4)**

- Informationsbroschüre für Teilnehmerinnen und Teilnehmer -

# PROJEKT ZUR UNTERSUCHUNG VON ESSVERHALTEN

Doktorarbeitsprojekt von M.Sc. Zoé van Dyck,  
Universität Luxemburg



## Sehr geehrte Teilnehmerin, sehr geehrter Teilnehmer,

Wir leben heute in einer Gesellschaft des Überflusses, in der Nahrungsmittel mit einer hohen Energiedichte leicht verfügbar sind. Unsere Umgebung ist geprägt durch eine ständige Konfrontation mit fettreichem Essen, sowohl auf Bildern (z.B. in der Werbung) als auch in der Realität. In unserer Studie interessieren wir uns dafür, **wie diese Essensreize von den Menschen wahrgenommen und bewertet werden.**

Unser Essverhalten wird zu einem großen Teil durch die Empfindung von **Hunger und Sätttheit** gesteuert: Wir essen wenn wir hungrig sind und hören auf wenn wir satt sind. Allerdings kann man feststellen, dass viele Menschen auch aufgrund anderer Faktoren essen. Nicht selten setzt man sich z.B. bestimmte Regeln hinsichtlich des Essens fest. Diese **Diätregeln** bestimmen was man essen sollte, wann man essen sollte und wie viel man essen darf, unabhängig davon ob man hungrig ist. Ein anderes Problem stellt das heutige **Überangebot** an Nahrungsmitteln dar. So kann man durch die ständige Verfügbarkeit schmackhafter Nahrungsmittel zum Essen verleitet werden, ohne dass man wirkliche Hungergefühle empfindet.

## Ziele der Studie

In unserer Studie untersuchen wir die **Wahrnehmung und Beurteilung von Essensreizen** (Bilder von Essen und richtiges Essen), sowie die **Empfindung von Hunger und Sätttheit**. Wir interessieren uns dafür, wie sich Personen mit Essanfällen und Personen ohne Essanfälle bezüglich dieser Mechanismen voneinander unterscheiden. Zwei offiziell anerkannte Essstörungen kennzeichnen sich durch Essanfälle: die Bulimia Nervosa („Ess-Brechsucht“) und die Binge-Eating-Störung („Esssucht“).

## Bulimia Nervosa und Binge-Eating-Störung

Bulimie und Binge-Eating-Störung sind neben der Anorexie (Magersucht) eine von drei anerkannten Essstörungen. Wo die Bulimie schon länger als offizielle psychische Erkrankung gilt, da ist die Binge-Eating-Störung erst kürzlich zu einer medizinisch anerkannten Essstörung geworden. Nichtsdestotrotz handelt es sich bei der Binge-Eating-Störung um die am weitesten verbreitete Essstörung: 2-5% der Bevölkerung leiden darunter. Außerdem ist das Verhältnis zwischen Frauen und Männern fast gleich, anders als bei sonstigen Essstörungen.

Sowohl Bulimie als auch die Binge-Eating-Störung sind gekennzeichnet durch regelmäßige Heißhungeranfälle mit dem Verlust der bewussten Kontrolle über die Nahrungsaufnahme. Bei diesen Anfällen werden vorrangig sehr kalorienreiche, süße bzw. fette Lebensmittel „verschlungen“. **Im Gegensatz zur Bulimie werden bei der Binge-Eating-Störung nach den Essanfällen keine Maßnahmen zur Eindämmung der Gewichtszunahme (Erbrechen, Abführmittel, exzessiver Sport) ergriffen.** Demnach geht die Binge-Eating-Störung auch oft mit Übergewicht oder Adipositas einher.

## Nutzen der Studie

Durch die Ergebnisse dieser Studie werden wir neue Erkenntnisse über das Wesen und die Auslöser von Essanfällen erhalten. Diese Erkenntnisse sind wichtig für die **Entwicklung wirksamer Therapieprogramme** zur Behandlung von Patienten mit Essstörungen. Außerdem können wir durch das gewonnene Wissen neue Ansätze zur Vorbeugung von Essstörungen entwickeln.

Derzeitige Behandlungen sind zwar erfolgreich in der kurzfristigen Reduktion von Essanfällen, langfristig erleiden jedoch 30-50% der behandelten Personen einen Rückfall. Dies ist teilweise darauf zurückzuführen, dass die **Entstehungs- und Aufrechterhaltungsbedingungen** von Essstörungen bisher noch nicht hinreichend bekannt sind. Es ist daher äußerst wichtig mehr über diese Faktoren zu erfahren, um die Behandlungsmöglichkeiten für Betroffene zu verbessern. Genauso wichtig ist die Entwicklung effektiver **Präventionsmaßnahmen**, damit sich Essstörungen gar nicht erst entwickeln.



## Studiendurchführung

Sie werden an drei unterschiedlichen Tagen getestet:

Am ersten Tag wird eine **ausführliche persönliche Diagnostik** durchgeführt. Diese umfasst mehrere Fragebögen und zwei Interviews zur Erfassung des Essverhaltens und der seelischen Gesundheit (Dauer ca. 2 Stunden). Mit dieser umfassenden Diagnostik wird abgeklärt ob eine Essstörung vorliegt. Zusätzlich können aber auch weitere psychische Problembereiche erkannt werden und eine angemessene Behandlung eingeleitet werden. An diesem Termin wird auch das Gewicht gemessen.

Die Interviews werden **auf Tonband aufgenommen**. Diese Tonbandaufzeichnungen werden mit einem Code versehen, sodass keine Rückverfolgung auf Ihre Person mehr möglich ist. Sie dienen lediglich der Qualitätssicherung, was bedeutet dass ein unabhängiger Beurteiler überprüfen wird, ob sich der Interviewer an die Interviewrichtlinien gehalten hat.

In der zweiten und dritten Sitzung finden jeweils die selben Aufgaben statt, einmal nachdem die Sie etwas gegessen haben und einmal wenn Sie hungrig sind. Beide Sitzungen dauern etwa **2 Stunden**. Wir wollen untersuchen, ob die Menschen unterschiedlich reagieren wenn sie **hungrig oder satt** sind. Das ist deshalb wichtig, weil Diäten meist mit Phasen des Hungerns einhergehen.

An beiden Testtagen werden wir Sie bitten, vor der Testung nichts zu essen. Es ist für uns wichtig sicherzustellen, dass sich alle Teilnehmer am Versuchstag ungefähr identisch ernähren, da die Reaktionen in den Tests wesentlich davon abhängig ist, was jemand bereits gegessen hat. Wir können die Ergebnisse der Teilnehmer die sich nicht daran halten nicht benutzen, deshalb werden wir mit Hilfe eines **Speicheltests** überprüfen müssen, ob Sie tatsächlich nichts gegessen haben. Es wird an beiden Terminen Essen zur Verfügung gestellt, damit niemand die Testung hungrig verlassen muss.

An der zweiten und dritten Sitzung finden jeweils folgende Aufgaben statt:

	WAS MÜSSEN SIE DABEI TUN?	WAS WIRD DABEI GEMESSEN?	WAS IST DAS ZIEL DAVON?
<b>Teil 1</b> <b>Wahrnehmung von Essensreizen</b>	Sie betrachten Bilder von Essen am PC und treffen Entscheidungen zwischen jeweils zwei Nahrungsmitteln.	Wir zeichnen Ihre Entscheidungen auf und messen die Bewegungen, die Sie mit der Computermaus machen.	Wir interessieren uns für Entscheidungsprozesse und wollen untersuchen, weshalb Personen gute oder schlechte Entscheidungen beim Essen treffen.
<b>Teil 2</b> <b>Innere Wahrnehmung des Körpers</b>	1. Sie achten auf Ihren eigenen Herzschlag.  2. Sie achten auf Ihr Völlegefühl während Sie stilles Wasser trinken.	1. Wir messen Ihren Herzschlag mithilfe eines Elektrokardiogramms (EKG). Hierzu werden Sensoren unterhalb des Schlüsselbeins und am Bauch auf die Haut geklebt, ähnlich einem Pflaster.  2. Wir untersuchen wann Sättigungssignale einsetzen und wie diese beurteilt werden.	Wir interessieren uns dafür, wie Vorgänge aus dem Körperinneren (Herzschlag und Magenaktivität) wahrgenommen werden. Diese Wahrnehmung ist wichtig, damit wir unser Verhalten an den Zustand unseres Körpers anpassen können.  Z.B.: Wir hören auf zu Essen wenn wir satt sind und spüren dass der Magen voll ist; Wir legen beim Sport eine Pause ein wenn die Anstrengung zu groß wird und das Herz zu schnell schlägt.
<b>Teil 3</b> <b>Geschmackstest</b>	Sie wählen verschiedene Nahrungsmittel aus, die Sie im Nachhinein probieren und beurteilen.	Wir untersuchen wie der Geschmack, das Aussehen und die Konsistenz verschiedener Nahrungsmittel eingeschätzt wird.	Wir interessieren uns dafür, wie Nahrungsmittel mit unterschiedlichem Geschmack und Kaloriengehalt bewertet werden. Außerdem wollen wir wissen, ob das Essen anders bewertet wird wenn man hungrig oder satt ist.

## PRAKTISCHE INFORMATIONEN

### Studiensprache

Sie haben die Möglichkeit zu wählen, in welcher Sprache Ihre Teilnahme ablaufen soll. Dies betrifft sowohl die Interviews und Fragebögen, als auch die Sprache des Versuchsleiters. Wir können Ihnen folgende Sprachen für die schriftlichen Unterlagen zur Auswahl stellen: **Deutsch**, **Französisch** oder **Englisch**. Falls Sie diese Informationsbroschüre nicht in der von Ihnen bevorzugten Sprache erhalten haben, dann teilen Sie uns dies bitte mit.

### Wie werden Sie während der Teilnahme betreut?

Während Ihrer Teilnahme werden Sie zu jedem Zeitpunkt durch die Studienleiterin, eine diplomierte Psychologin, und eine assistierende Psychologiestudentin betreut. Alle Beteiligten sprechen Luxemburgisch, Deutsch, Französisch und Englisch und sind gerne bereit, Ihnen jegliche Fragen zu beantworten.

### Wie wird Ihr Aufwand entschädigt?

Als Aufwandsentschädigung erhalten Sie 80€, die für Teilnehmer aus Luxemburg in Form von Sodexo-Geschenkgutscheinen abgegeben werden.

### Wo findet die Studie statt?

Alle Termine finden in den Räumlichkeiten der Forschungseinheit INSIDE der Universität Luxemburg statt. Diese befinden sich auf dem **Campus Walferdange**. Bei Ihrer Ankunft können Sie sich an der Rezeption melden, wo Sie von der Studienleiterin abgeholt werden. Wenn Sie mit dem Auto anreisen dann können Sie auch die hauseigenen Parkplätze nutzen. Mit öffentlichen Verkehrsmitteln erreichen Sie den Campus von Luxemburg-Stadt aus mit der Buslinie 11, sowie der Eisenbahnlinie Luxemburg-Mersch. Einen Lageplan des Campus finden Sie auf der letzten Seite dieser Broschüre.



### Wer führt die Studie durch?

Die Studie findet an der Universität Luxemburg statt, im Rahmen der Doktorarbeit von Zoé van Dyck, M.Sc. Das Projekt wird von der Arbeitsgruppe Selbstregulation und Gesundheit“ (Leitung: Prof. Dr. Claus Vögele) durchgeführt; Sie ist Teil der Integrativen Forschungseinheit für soziale und individuelle Entwicklung (INSIDE) der geisteswissenschaftlichen Fakultät (FLSHASE) der Universität Luxemburg.

Folgende Mitarbeiter der Universität Luxemburg und anderer Universitäten sind an dieser Studie beteiligt:

Zoé van Dyck, M.Sc. (Universität Luxemburg): Studienleitung

Prof. Dr. Claus Vögele (Universität Luxemburg): Supervision

Dr. Jens Blechert (Universität Salzburg): Wissenschaftlicher Berater

Dr. André Schulz (Universität Luxemburg): Wissenschaftlicher Berater

Annabel Stender, Sonja Reiter, Jessica Schrauf (Universität Luxemburg): Wissenschaftliche Hilfskräfte

### Freiwilligkeit der Studienteilnahme

Die Teilnahme an der Untersuchung ist vollkommen freiwillig und das Einverständnis des Teilnehmers kann jederzeit ohne Angabe von Gründen und ohne Nachteile widerrufen werden. Alle im Rahmen der Studie erhobenen Daten unterliegen der Schweigepflicht und werden anonymisiert gespeichert und streng vertraulich behandelt.



## Kontakt

Zoé VAN DYCK, M.Sc.

Université du Luxembourg  
Campus Walferdange  
Route de Diekirch (BP 2)  
L-7220 Walferdange

Tel.: (+352) 46 66 44 9799

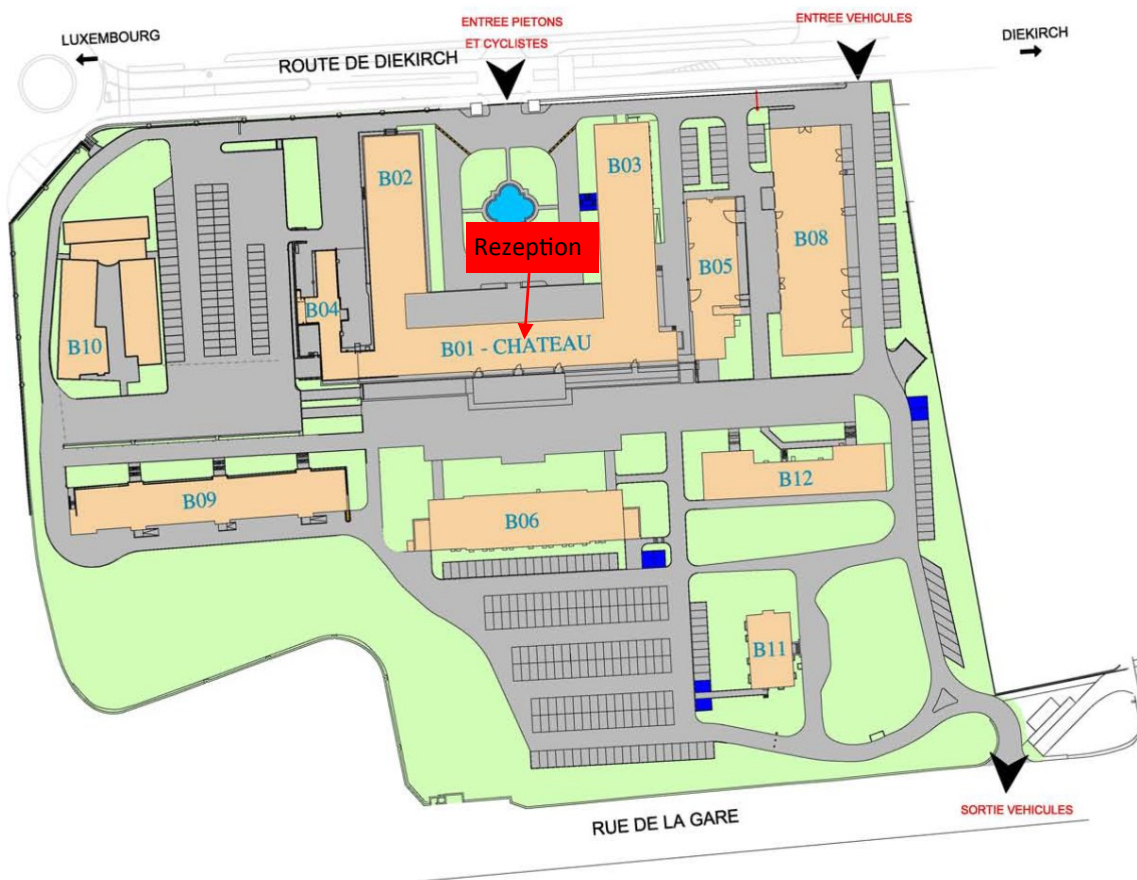
Fax: (+352) 46 66 44 9535

E-mail: zoe.vandyck@uni.lu



## Anfahrt

### Université du Luxembourg - Campus Walferdange



UNIVERSITY OF LUXEMBOURG  
Integrative Research Unit on Social  
and Individual Development (INSIDE)