

Training Response Inhibition to Reduce Food Consumption

Rachel C. Adams

PhD Thesis

School of Psychology
Cardiff University
2014

Abstract

Response inhibition refers to our ability to stop or interrupt impulsive actions. This cognitive process is essential for goal-directed behaviour, and deficits in inhibitory control have been associated with various impulse control disorders including substance use and obesity. However, recent research has demonstrated the potential of response inhibition training as a therapeutic tool for reducing impulsive behaviours. Theoretical models argue that training inhibition towards tempting stimuli may prime general self-control or cause the devaluation of inhibited stimuli. Here, I investigated the effectiveness of a single session of food-related inhibition training on food consumption in restrained eaters. Furthermore, I examined the role of different training protocols, stimulus-specific associations and underlying cognitive mechanisms. Participants received either inhibition or control training using a modified version of either the stop-signal or go/no-go task. During training the associations between stop and go responses were manipulated for particular foods. The consumption of and attitudes towards trained and untrained foods were then measured. Results for food consumption showed a greater difference in intake between inhibition and control groups on the go/no-go task compared to the stop-signal task. There was also evidence to show that the effect generalised to a novel unhealthy food following go/no-go training. However, the inclusion of an additional control group, who did not make any responses during training, provided evidence to suggest that differences in intake were the result of increased consumption in the 'control' group rather than decreased consumption in the inhibition group. Furthermore, I found limited evidence to suggest an effect of inhibition training on either implicit or explicit attitudes towards food. These results cast doubt on the effects of inhibition training on behaviour and demonstrate that more appropriate control tasks and dependent measures are required in future research to fully explore its potential.

DECLARATION

This work has not been submitted in substance for any other degree or award at this or any other university or place of learning, nor is being submitted concurrently in candidature for any degree or other award.

Signed (candidate) Date ...2/11/2014

STATEMENT 1

This thesis is being submitted in partial fulfillment of the requirements for the degree of PhD.

Signed (candidate) Date ...2/11/2014

STATEMENT 2

This thesis is the result of my own independent work/investigation, except where otherwise stated. Other sources are acknowledged by explicit references. The views expressed are my own.

Signed (candidate) Date ...2/11/2014

STATEMENT 3

I hereby give consent for my thesis, if accepted, to be available for photocopying and for inter-library loan, and for the title and summary to be made available to outside organisations.

Signed (candidate) Date ...2/11/2014

STATEMENT 4: PREVIOUSLY APPROVED BAR ON ACCESS

I hereby give consent for my thesis, if accepted, to be available for photocopying and for inter-library loans after expiry of a bar on access previously approved by the Academic Standards & Quality Committee.

Signed (candidate) Date ...2/11/2014

Acknowledgements

First and foremost I thank my primary supervisor, Chris Chambers, for all his help, support and guidance throughout my PhD. I believe his remarkable passion and enthusiasm for ‘good’ science has helped me to become a better researcher. Secondly, I thank my supervisors, Frederick Verbruggen and Natalia Lawrence for all their help and expertise. I am also grateful to the School of Psychology at Cardiff University for funding this research and to all the participants who devoted their time.

I would also like to thank everyone from our lab – who have all contributed in some way. Special thanks to Leah for being a massive support and a great friend over the last few years, to Sinéad and Charlotte for assistance with data collection, to Jemma for logistics, to Chris for help with Bayes and Veldri for programming advice. I am also greatly indebted to Justin Savage and Spiro Stathakis for all their help with Java and implementation of the online study – this project would not have been possible without them.

Finally, a huge thank you to all my family, especially to my parents for their unconditional support, and to Lewis... to whom there are too many things for which I am grateful...

thank you.

Table of Contents

Abstract	i
Declaration	ii
Acknowledgements	iii
<u>Chapter 1. Literature Review</u>	1
1.1. Dual Process Models and Addiction	2
1.1.1. Applying Dual Process Models to Behavioural Interventions	4
1.2. Food Addiction: Can We View Food as Addictive, and How Can This Inform Intervention?	9
1.2.1. Considering the Evidence for Food Addiction Based on the DSM-V Diagnostic Criteria for Substance Use Disorder	10
1.2.1.1. Impaired Control	11
1.2.1.1.1. Impulsivity	13
1.2.1.1.2. Reward Sensitivity	15
1.2.1.1.3. Craving	18
1.2.1.2. Social Impairment	19
1.2.1.3. Risky Use	20
1.2.1.4. Pharmacological Criteria	21
1.2.2. Foods with Addictive Potential	22
1.2.3. Neurobiological Similarities between Palatable Foods and Drugs of Abuse	24
1.2.3.1. Reward Sensitivity	24
1.2.3.2. Inhibitory Control	27
1.2.4. Assessment of ‘Food Addiction’	29
1.2.5. Treatment Implications	30
1.2.5.1. Cognitive Interventions	31
1.2.5.2. Neuromodulation Interventions	35
1.2.6. Conclusions	38
1.3. Inhibitory Control and Obesity	40
1.3.1. Response Inhibition Training and Food Consumption	45
1.3.2. Response Inhibition Training: Potential Mechanisms	52
1.4. Dietary Restraint: A Cautionary Tale	62

1.4.1. Dietary Restraint and Increased Food Motivation	63
1.4.2. Dietary Restraint and Poor Self-Control	65
1.4.3. Dietary Restraint Versus Dietary Disinhibition	66
1.5. Synopsis	69
<u>Chapter 2. Study 1</u>	72
<i>Dietary Restraint and Disinhibited Eating: A Comparison of the Restraint Scale and the Restrained Eating Scale of Dutch Eating Behaviour Questionnaire</i>	
2.1. Introduction	73
2.2. Method	77
2.2.1. Participants	77
2.2.2. Materials / Measures	78
2.2.2.1. The Restraint Scale	78
2.2.2.2. The Dutch Eating Behaviour Questionnaire – Restrained Eating Scale	78
2.2.2.3. The Dutch Eating Behaviour Questionnaire – External Eating Scale	78
2.2.2.4. The Attitudes to Chocolate Questionnaire – Craving Scale	79
2.2.2.5. Ratings of Healthy and Unhealthy Food Liking	79
2.2.2.6. The General Food Craving Questionnaire- Trait Version	79
2.2.2.7. Body Mass Index (BMI)	80
2.2.3. Procedure	80
2.2.4. Statistical Analysis	80
2.3. Results	81
2.3.1. Internal Consistency	81
2.3.2. Factor Structure	81
2.2.3. Demographic Differences	84
2.2.4. Comparison of the RS and DEBQRE	85
2.2.4.1. Restraint Correlations	85
2.2.4.2. External Eating	86
2.2.4.3. Food Liking	87
2.2.4.4. Food Craving	88
2.2.4.5. BMI	90
2.4. Discussion	90

<u>Chapter 3. Study 2</u>	96
<i>Training Response Inhibition in Trait Chocolate Lovers: Effects on Implicit Attitudes and Consumption</i>	
3.1. Introduction	96
3.2. Method	101
3.2.1. Participants	101
3.2.2. Materials / Measures	102
3.2.2.1. Training Task	102
3.2.2.2. Unipolar, Single-Category Implicit Association Test	104
3.2.2.3. Taste Test	107
3.2.2.4. Questionnaires	107
3.2.3. Procedure	108
3.2.4. Statistical Analysis	110
3.3. Results	114
3.3.1. Group Differences	114
3.3.2. Training Data Analysis	114
3.3.3. Consumption Data Analysis	116
3.3.4. Unipolar, SC-IAT Data Analysis	117
3.3.5. Debrief Analysis	120
3.4. Discussion	121
<u>Chapter 4. Study 3</u>	128
<i>Training Response Inhibition to Reduce Food Consumption: A Comparison of the Stop-Signal and Go/No-Go Paradigms</i>	
4.1. Introduction	128
4.1.1. The Stop-Signal Task and the Go/No-Go Task	129
4.1.2. Response Inhibition and Healthy Food Consumption	131
4.1.3. Increased Inhibition or Increased Approach?	135
4.2. Method	137
4.2.1. Participants	137
4.2.2. Materials / Measures	137
4.2.2.1. Training Tasks	138
4.2.2.1.1. Stop-Signal Training	139

4.2.2.1.2. Go/No-Go Training	140
4.2.2.1.3. Observe Training	141
4.2.2.2. Snack Buffet	141
4.2.2.3. Questionnaires	142
4.2.2.4. Recognition Task	143
4.2.3. Procedure	144
4.2.4. Statistical Analysis	145
4.3. Results	148
4.3.1. Group Differences	148
4.3.2. Training Data Analysis	150
4.3.3. Recognition Task Analysis	152
4.3.4. Consumption Data Analysis	152
4.3.4.1. Stop-signal Training Results	152
4.3.4.2. Go/No-Go Training Results	155
4.3.4.3. Comparison of all Groups, Including the Observe Group	157
4.3.5. Debrief Analysis	159
4.4. Discussion	161
<u>Chapter 5. Study 4</u>	167
<i>The Effect of Food-Related Go/No-go Training on Implicit and Explicit Attitudes Towards Palatable Snacks: An Internet-Based Study</i>	
5.1. Introduction	167
5.2. Method	172
5.2.1. Participants	172
5.2.2. Experimental Distribution and Procedure	173
5.2.3. Materials / Measures	175
5.2.3.1. Go/No-Go Training	175
5.2.3.2. Unipolar, Single-Category Implicit Association Test (Study 4a)	176
5.2.3.3. Explicit Stimulus Evaluation Task (Study 4b)	178
5.2.3.4. Questionnaires	180
5.2.4. Statistical Analysis	180
5.3. Results	183
5.3.1. Study 4a: Implicit Evaluation Results	183

5.3.1.1. Group Differences	183
5.3.1.2. Training Data Analysis	184
5.3.1.3. Unipolar, SC-IAT Data Analysis	185
5.3.2. Study 4b: Explicit Evaluation Results	187
5.3.2.1. Group Differences	187
5.3.2.2. Training Data Analysis	188
5.3.2.3. Explicit Stimulus Evaluation Task Analysis	190
5.4. Discussion	193
<u>Chapter 6. General Discussion</u>	198
6.1. Summary and Discussion of Findings	198
6.2. Limitations and Directions for Further Research	208
6.2.1. Sample Selection and Generalisability	208
6.2.1.1. Psychology Undergraduates	208
6.2.1.2. Self-Selection	209
6.2.1.3. Menstrual Cycle and Food Cravings	210
6.2.1.4. Restrained Eaters	210
6.2.1.5. Alternative Samples	211
6.2.2. Training Protocols	213
6.2.2.1. Task Parameters	213
6.2.2.2. Control Tasks	214
6.2.3. Measuring Food Consumption	215
6.2.3.1. The Cover Story	215
6.2.3.2. Ecological Validity	216
6.2.4. Demand Characteristics and Placebo Effects	217
6.2.5. Summary	219
6.3. Conclusion	220
<u>References</u>	221
<u>Appendices</u>	269

Chapter 1. Literature Review

Our capacity for self-regulation is perhaps one of the most remarkable and adaptive features of the human condition. The ability to manage our emotions, cognitions and behaviours enables us to restrain our immediate impulses for the benefit of our long-term goals in ways that other animals are not capable of doing. For example, self-regulation enables us to plan for the future and persist at difficult tasks such as studying for exams, saving money or maintaining a healthy lifestyle. The term ‘self-regulation’ has been used broadly to refer to goal-directed behaviour, whereas the term ‘self-control’ typically defines the ability to inhibit unwanted impulses or urges (Hofmann, Schmeichel & Baddeley, 2012). Good self-control has been implicated in a number of positive outcomes such as academic and interpersonal success (Tangney, Baumeister & Boone, 2004) and better health behaviours (Griffin, Scheier, Acevedo, Grenard & Botvin, 2012; Quinn & Fromme, 2010). Failures of self-control, however, have been associated with a number of personal and behavioural problems such as procrastination (Steel, 2007), violence and crime (Denson, Pedersen, Friese, Hahm & Roberts, 2011b; DeWall, Baumeister, Stillman & Gailliot, 2007; Hirshi, 2004), risky sexual behaviour (Wiederman, 2004), financial and gambling problems (Bergen, Newby-Clarke & Brown, 2012; Faber & Vohs, 2004), substance use (Hull & Slone, 2004; King, Fleming, Monahan & Catalano, 2011; Wills & Stoolmiller, 2002; Wills, Walker, Mendoza & AINETTE, 2006) and obesity (Bryant, King & Blundell, 2008; Francis & Susman, 2009; Herman & Polivy, 2004). These failures generally occur when our self-control is unable to suppress our hedonic impulses.

The following section describes how these two processes interact according to dual process models and particularly focuses on how they predict addictive behaviours and inform interventions. The application of these theories and interventions are then considered in the context of overeating and obesity. Firstly I present a detailed review on the concept of ‘food addiction’ before discussing how an addiction model has been used to inform treatments for overeating. Although an understanding of

overeating and obesity, in the majority of cases, does not require a model of food addiction, this model provides a good framework for understanding the underlying causes and wider implications of overeating beyond a dual process account. The remainder of this thesis focuses on one potential intervention for overeating – response inhibition training. Relevant literature for this intervention and the potential underlying mechanisms are reviewed. Finally, I discuss the concept of dietary restraint and why individuals who score highly on this dimension are of particular interest for training food-related inhibitory control. The literature review concludes with a synopsis of the four studies presented in this thesis.

1.1. Dual Process Models and Addiction

The idea of a conflict between ‘passion’ and ‘reason’ dates back to Greek philosophy. On the one hand we possess unconscious impulses and desires, and on the other we have a conscious obligation to behave according to cultural and societal norms (Hofmann, Friese & Strack, 2009b). These ideas have evolved into contemporary dual-system models that have been used to explain behaviour across various domains (Hofmann, Friese & Wiers, 2008; Metcalfe & Mischel, 1999; Smith & DeCoster, 2000; Shiffrin & Schneider, 1977; Strack & Deutsch, 2004). One such model that particularly focuses on how behaviour is determined by the interaction of these systems is the reflective impulsive model (RIM; Strack & Deutsch, 2004). According to this model, behaviour emerges from the joint functioning of an automatic, impulsive system and a controlled, reflective system. The impulsive system consists of an associative network of spreading activation where associative weights are created and strengthened based on temporal and spatial proximity. These associative clusters link elements of the environment with affective, cognitive and behavioural reactions. The impulsive system generates behaviour through the activation of behavioural schema that form when stimuli are regularly coactivated with motor reactions. Moreover, these links are inflexible, enduring and readily accessible. The reflective system, by contrast, is based on symbolic representations and semantic links between stimuli. These links are highly flexible but are slow to develop, short-lived and require rehearsal. Whereas the impulsive system is concerned with automatic, approach-avoidance behaviours, the reflective system is

responsible for behaviour that is in accordance with our personal standards and long-term goals. Behaviour in the reflective system is a consequence of conscious deliberation through reasoning and intending. This provides the reflective system with some degree of control, but only when cognitive capacity is high. The cost of such a flexible system is the demand on cognitive resources; whereas the impulsive system is always engaged in processing, the reflective system is easily disrupted. Ultimately, therefore, behaviour is determined by the relative strength of both systems.

This ‘tug-of-war’ scenario between conflicting systems has been applied to addictive behaviours (Bechara, 2005; Deutsch & Strack, 2006; Evans & Coventry, 2006; Friese, Hofmann & Wiers, 2011; Hofmann *et al.*, 2008; Wiers, Ames, Hofmann, Krank & Stacy, 2010a; Wiers *et al.*, 2007; Wiers & Stacy, 2006). Firstly, changes in these systems may occur as a result of addiction. For example, chronic consumption of illicit substances will lead to changes in the associative structure of the impulsive system. The incentive-sensitisation theory of addiction (Robinson & Berridge, 1993, 2001, 2003) describes how drug use disrupts the dopamine system, increasing the motivational and rewarding properties of these substances. Substance abuse has also been linked to changes in the reflective system, generally through alterations in executive functioning (for a review see Fernández-Serrano, Pérez-García & Verdejo-García, 2011). Secondly, individual differences in the strength of these systems have been implicated in the development of and vulnerability to addiction (Bardo, Fishbein & Milich, 2011; Dawe & Loxton, 2004; de Wit, 2008; Giancola & Tarter, 1999; Iacono, Malone & McGue, 2008; Verdejo-García, Lawrence & Clark, 2008; Wills, Ainette, Stoolmiller, Gibbons & Shinar, 2008). Research in this area is not only of theoretical interest but has also helped to identify vulnerable populations, such as adolescents and those with attention-deficit/ hyperactivity disorder (ADHD; Carroll & Rounsaville, 1993; Clure, Brady, Saladin, Johnson, Waid & Rittenbury, 1999; Gullo & Dawe, 2008; Molina & Pelham, 2003; Vitaro, Ferland, Jacques & Ladouceur, 1998). Furthermore, this model and associated processes have implications for potential behavioural interventions (for an overview see Friese *et al.*, 2011; Wiers, Galdwin, Hofmann, Salemink & Ridderinkhof, 2013; see Figure 1.1.).

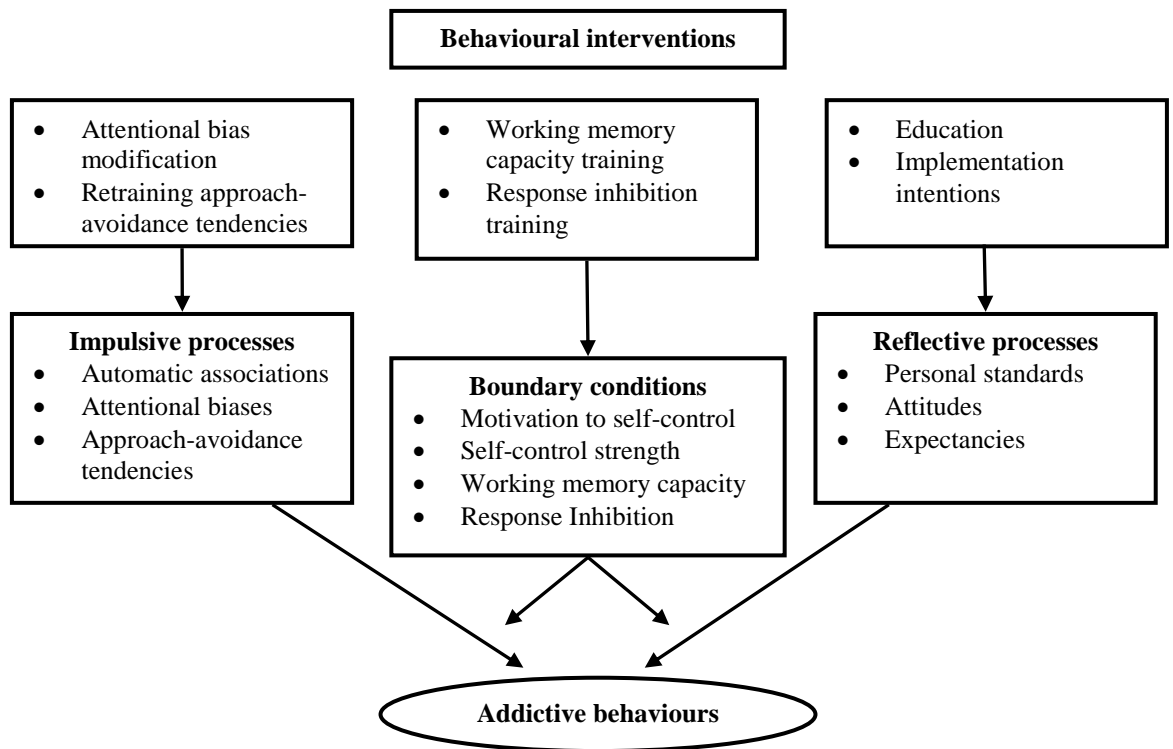


Figure 1.1. A dual process model illustrating the interaction between impulsive and reflective processes on addictive behaviours. Behavioural interventions to reduce addictive behaviours and improve outcomes may focus on either of these systems or on the boundary conditions that improve the ability to exert self-control (adapted from Friese *et al.*, 2011).

1.1.1. Applying Dual Process Models to Behavioural Interventions

One approach for reducing addictive behaviours is to target the overactive impulsive system and the strong associative structures. Substance abuse has been linked to a number of cognitive biases that increase the likelihood of drug-seeking and drug-taking behaviours. An attentional bias for drug-related cues has been found with alcohol (Fadardi & Cox, 2009; Field, Mogg, Zetteler & Bradley, 2004; Miller & Fillmore, 2010; Townshend & Duka, 2001), cannabis (Field, Eastwood, Bradley & Mogg, 2006), opiates (Lubman, Peters, Mogg, Bradley & Deakin, 2000) and tobacco (Bradley, Field, Mogg & De Houwer, 2004; Bradley, Mogg, Wright & Field, 2003;

Ehrman, Robbins, Bromwell, Lankford, Monterosso & O'Brien, 2002; for reviews see Field & Cox, 2008; Franken, 2003). Typically, dependent individuals will orient attention towards a drug-related stimulus more easily than they will a neutral stimulus. This has led to attempts to train these individuals to redirect their attention towards a more appropriate neutral stimulus (Attwood, O'Sullivan, Leonards, Mackintosh & Munafò, 2008; Fadardi & Cox, 2009; Field, Duka, Eastwood, Child, Santarcangelo & Gayton, 2007; Field, Duka, Tyler & Schoenmakers, 2009; Field & Eastwood, 2005; Schoenmakers, de Bruin, Lux, Goertz, Van Kerkhof & Wiers, 2010; Schoenmakers, Wiers, Jones, Bruce & Jansen, 2007). For example, Field *et al.* (2007) used a modified version of the visual probe task to train heavy drinking individuals to avoid alcohol. In this task two images are presented simultaneously to either side of a central fixation. These images then disappear and one of them is replaced by a probe; participants are required to respond as quickly as possible to the location of the probe. To measure an attentional bias towards alcohol the picture pairs would typically include one alcohol-related image and one neutral, control stimulus (such as a soft drink) with the probe appearing with equal probability behind each stimulus type. To estimate the attentional bias for alcohol stimuli the reaction time for alcohol trials is subtracted from the reaction time for neutral trials. In the modified version of the task, however, Field *et al.* always presented the probe behind the neutral stimulus in the 'avoid alcohol' group and behind the alcohol stimulus in the 'attend alcohol' group. Whereas the attentional bias towards alcohol increased for the attend group, this bias decreased in the avoid group. Although this effect did not generalise to new stimuli, more encouraging results have been found with repeated training sessions (Schoenmakers *et al.*, 2010).

Heavy drinkers (Field, Kiernan, Eastwood & Child, 2008), cannabis users (Cousijn, Goudriaan & Wiers, 2011; Field *et al.*, 2006) and smokers (Bradley *et al.*, 2004; Mogg, Bradley, Field, & De Houwer, 2003) have also shown a strong approach response for drug-related stimuli (for a thorough review on the theoretical background and empirical data for approach-avoidance tendencies in addiction see Watson, de Wit, Hommel & Wiers, 2012). Individuals typically find it more congruent to approach positively valenced stimuli and avoid negatively valenced stimuli than vice versa. Using the same logic as the attentional bias modification

studies, researchers have attempted to retrain this approach bias by having participants move a joystick or manikin towards neutral stimuli and away from substance-related stimuli (Fishbach & Shah, 2006; Wiers, Rinck, Kordts, Houben & Strack, 2010b; Wiers, Eberl, Rinck, Becker & Lindenmeyer, 2011). This training has demonstrated some success with relapse rates in clinical trials with alcohol-dependent patients. Patients who received just four training sessions, alongside normal treatment, were 13% less likely to relapse one year later compared with control patients (Wiers *et al.*, 2011). Together these training techniques demonstrate that the associative clusters within the impulsive system can be altered and that these training techniques can have valuable consequences for addictive behaviours.

Another way to influence behaviour is through changes in the ability of the reflective system to exert control. One method is to address an individual's personal standards and goals directly through education of knowledge, ability and expectancies. For example, Connor and Higgins (2010) found a reduction in smoking for adolescents who formed implementation intentions (i.e. specific if-then plans) of how, when and where to refuse a cigarette (for a review on implementation intentions and goal attainment see Gollwitzer & Sheeran, 2006). A substantial literature on these interventions already exists and is beyond the scope of this chapter (see the following comprehensive reviews: Carey, Scott-Sheldon, Carey & DeMartini, 2007; Christensen, Low & Anstey, 2006; Irvin, Bowers, Dunn & Wang, 1999; Toumbourou, Stockwell, Neighbors, Marlatt, Sturge & Rehm, 2007; Viswesvaran & Schmidt, 1992). Another method involves improving self-control ability. The strength model of self-control views self-regulation as a limited energy or resource (Baumeister, Bratslavsky, Muraven, & Tice, 1998; Hagger, Wood, Stiff & Chatzisarantis, 2009; Muraven & Baumeister, 2000; Muraven, Tice, & Baumeister, 1998). According to this model our ability to exert self-control is finite and task-independent; exerting self-control on one task, such as emotion suppression, depletes our general resource and leaves us less able to exert self-control on a second, unrelated task, such as solving anagrams (Baumeister *et al.*, 1998; Muraven *et al.*, 1998). This effect is referred to as 'ego-depletion' and has been found across a wide variety of tasks and measures (see Hagger, Wood, Stiff and Chatzisarantis (2010) for a meta-analysis; for an alternative explanation to the resource model see Inzlicht &

Schmeichel, 2012; Inzlicht, Schmeichel & Macrae, 2014). Moreover, effects of ego-depletion have been reported in relation to substance use. Christiansen, Cole and Field (2012) found that participants who suppressed emotions while watching a video clip consumed more alcohol during a taste test than participants in the control group. In addition, this effect was mediated by self-reported effort during the depletion task.

Muraven and Baumeister (2000) have also referred to this self-regulatory resource as a muscle. Just as a muscle can become fatigued with over-exertion, it is also possible to strengthen that muscle with repeated exercise (Baumeister, Gailliot, DeWall & Oaten, 2006; Denson, Capper, Oaten, Friese & Schofield, 2011a; Finkel, DeWall, Slotter, Oaten & Foshee, 2009; Gailliot, Plant, Butz & Baumeister, 2007; Hagger & Chatzisarantis, 2013; Muraven, 2010; Muraven, Baumeister & Tice, 1999; Oaten & Cheng, 2006a, 2006b, 2007). This 'training hypothesis' has been applied to behavioural interventions for health-related behaviours. In a recent study Oaten and Cheng (2006a) had participants adhere to a two month exercise programme in which they were required to regularly partake in physical exercise. They found that time spent in the programme was associated with changes across a wide range of health behaviours; participants reduced their consumption of cigarettes, alcohol, caffeine and junk food and increased their consumption of healthy foods. They also showed improvements in other areas such as spending and time-keeping. The generalisation of effects in this study is impressive and also supports the idea of a domain-general self-regulatory resource. However, other explanations such as improvements in mood and stress management cannot be fully dismissed; future research is therefore needed to replicate these effects and explore the exact mechanisms underlying them.

It is also possible to improve the ability to self-control by targeting training at more specific executive functions such as working memory capacity (WMC) and response inhibition. Working memory has been implicated in self-control functioning not through its memory structure per se, but due to its ability to control attention and prioritise information (Engle, 2002). The finding that working memory is responsive to training and can improve other aspects of executive functioning (Klingberg, 2010; Klingberg, Forssberg & Westerberg, 2002) led Houben, Wiers and Jansen (2011b) to

attempt to train WMC in a sample of problem drinkers. Heavy drinking participants performed working memory tasks over twenty-five training sessions; for the training group task difficulty was increased on a trial-by-trial basis, whereas the control group remained at the baseline difficulty level. This resulted in an improvement in WMC for the training group but not the control group. Furthermore, this improvement mediated an effect of training on weekly alcohol consumption, especially for those who showed strong implicit preferences for alcohol.

Another executive function that has been prominent in studies of behavioural training is response inhibition. Response inhibition refers to our ability to interrupt or override impulsive reactions in accordance with new information, and plays a key role in goal-directed behaviour (Aron, Robbins & Poldrack, 2004, 2014; Logan, 1985; Logan & Cowan, 1984; Logan, Schachar & Tannock, 1997; Miyake, Friedman, Emerson, Witzki & Howerter, 2000). Deficits in response inhibition have been linked to various addictions (e.g. Hester, Bell, Foxe & Garavan, 2013; Luijten, Littel & Franken, 2011; Monterosso, Aron, Cordova, Xu & London, 2005; Murphy & Garavan, 2011; for a review see Jentsch & Pennington, 2014) and simple tasks designed to train response inhibition have already been shown to reduce gambling behaviour (Stevens *et al.*, under review; Verbruggen, Adams & Chambers, 2012) and alcohol consumption (Bowley *et al.*, 2013; Houben, Nederkoorn, Wiers & Jansen, 2011a; Houben, Havermans, Nederkoorn & Jansen, 2012a; Jones, Christiansen, Nederkoorn, Houben & Field, 2013; Jones & Field, 2013). These training tasks have also been applied to overeating and obesity and are showing encouraging effects across a range of eating-related behaviours (e.g. Houben, 2011; Houben & Jansen, 2011; Lawrence, Verbruggen, Morrison, Adams & Chambers, under review; van Koningsbruggen, Veling, Stroebe & Aarts, 2013a; Veling, Aarts & Papies, 2011; Veling, Aarts & Stroebe, 2013a; Veling, van Koningsbruggen, Aarts & Stroebe, 2014). Intuitively, it is clear to see how increased impulsivity and reduced self-control can play a role in excess calorie intake, especially when considering the obesogenic environment in which we live (Hill & Peters, 1998). The next section discusses these potential mechanisms under a model of food addiction which also includes the effects of overeating on social and physical well-being. Moreover, considering the addictive potential of certain foods should also increase

our understanding of why these foods are so strongly associated with the failure of self-control.

1.2. Food Addiction: Can We View Food as Addictive, and How Can This Inform Intervention?

In 2003 obesity was declared a global epidemic by the World Health Organisation (WHO, 2003), and the prevalence of overweight and obesity in Western societies continues to increase (Hedley, Ogden, Johnson, Carroll, Curtin & Flegal, 2004; Lobstein & Frelut, 2003; Lobstein, James & Cole, 2003; Ogden, Carroll, Curtin, McDowell, Tabak & Flegal, 2006; Wang & Beydoun, 2007). Obesity levels also present a great economic burden; for the European Union it was estimated in 2002 that at least half the member states had obesity levels of more than 20%, costing an estimated €33 billion (Fry & Finley, 2005). One of the common explanations for the increase in obesity over recent decades is the environment and the availability of highly varied, palatable and fattening foods (Cummins & Macintyre, 2006; French, Story & Jeffery, 2001; Jeffery & Utter, 2003; Levitsky, 2005; McCrory *et al.*, 1999). While many individuals manage to resist these temptations and maintain a healthy weight, obese individuals have been shown to have a preference for such energy dense foods (Blundell, Burley, Cotton & Lawton, 1993; Drewnowski, Brunzell, Sande, Iverius & Greenwood, 1985; Drewnowski, Kurth, Holden-Wiltse & Saari, 1992), which is positively correlated with adiposity measures (Mela & Sacchetti, 1991). The critical question of why some individuals are able to resist overeating while others cannot has led to the question of whether some individuals experience an addictive-type relationship with food.

The concept of 'food addiction' has been evident in the media and general public for some time and is now gaining increasing interest in the scientific literature (Gearhardt, Davis, Kushner & Brownell, 2011b). There are now many reviews discussing the diagnostic, neurobiological and practical aspects of food addiction, with arguments both for and against its utility and validity (Avena, Gearhardt, Gold, Wang & Potenza, 2012; Barry, Clarke & Petry, 2009; Corsica & Pelchat, 2010; Davis & Carter, 2009; Del Parigi, Chen, Salbe, Reiman & Tataranni, 2003; DiLeone,

Taylor & Picciotto, 2012; Gearhardt, Corbin & Brownell, 2009a; Gold, Graham, Cocores, & Nixon, 2009; Ifland *et al.*, 2009; Meule, 2014; Pelchat, 2009; Rogers & Smit, 2000; Smith & Robbins, 2013; Volkow & Wise, 2005; Volkow, Wang, Tomasi & Baler, 2013; Wilson, 1991, 2010; Ziauddeen & Fletcher, 2013; Ziauddeen, Farooqi & Fletcher, 2012a, 2012b). This surge of interest comes with a new perspective that addiction can be conceptualised as an excessive appetite for a particular behaviour or substance without the need to focus on psychoactive substances (Frascella, Potenza, Brown, & Childress, 2010; Orford, 2001). This shift in perspective has now been acknowledged in the fifth version of the Diagnostic and Statistical Manual (DSM-V; American Psychiatric Association, 2013) with the addition of gambling disorder as the first behavioural addiction. Acceptance of this disorder was based on evidence that gambling can produce behavioural symptoms that parallel those of substance addiction and can activate the same neural reward circuits as drugs of abuse. There is now a large body of research demonstrating that these findings are also true of overeating and obesity. Moreover, treatments developed for addictive disorders have also shown some efficacy for the treatment of overeating. These findings highlight how a model of food addiction may help us to understand elements of overweight/obesity beyond a simple lack of willpower and can also be used to inform effective interventions and policy (Barry *et al.*, 2009; Gearhardt, Grilo, DiLeone, Brownell & Potenza, 2011c; Volkow & Wise, 2005; Wilson, 2010).

1.2.1. Considering the Evidence for Food Addiction Based on the DSM-V Diagnostic Criteria for Substance Use Disorder

Although food addiction has not been recognised in the DSM-V (APA, 2013), the similarities between some feeding and eating disorders and substance use disorders (SUDs) have been acknowledged. These similarities include the experience of cravings, reduced control over intake, increased impulsivity and altered reward-sensitivity. Binge eating disorder (BED) has been proposed as a phenotype that may reflect these similarities to the greatest extent (Davis & Carter, 2009; Gold, Frost-Pineda & Jacobs, 2003; Smith & Robbins, 2013). BED is characterised by recurrent episodes of binge eating in which vast quantities of food are consumed, accompanied

by feelings of a lack of control, despite physical and emotional distress. Reports of food addiction have been shown to be particularly high amongst individuals with BED (Cassin & von Ranson, 2007; Curtis & Davis, 2014; Gearhardt, White, Masheb, Morgan, Crosby & Grilo, 2012). For example, using the DSM-V criteria for SUDs, Curtis and Davis (2014) found that women with BED endorsed more addictive symptoms towards food and were twice as likely to meet the diagnosis compared to their BMI-matched controls. Here, the same DSM-V diagnostic criteria for SUDs (see DSM-V manual for full criteria) are used to discuss the evidence for food addiction. These criteria are defined as ‘a cluster of cognitive, behavioural and physiological symptoms’ (p483; APA, 2013). More specifically the following categories are considered: impaired control, social impairment, risky use and pharmacological criteria. However, it should be noted that the pharmacological criteria of tolerance and withdrawal are not necessary for a diagnosis. The DSM-V also states that although changes in neural functioning are a key characteristic of SUDs, the diagnosis is based on a pathological pattern of behaviours. Hence, the diagnostic criteria are discussed initially followed by other relevant topics including neurobiological evidence.

1.2.1.1. Impaired Control

C1 – Use of the substance beyond that intended (larger amounts or for longer periods)

C2 – A persistent desire to cut down, often with multiple unsuccessful attempts

C3 – A great deal of time spent acquiring, using and recovering from substance use

C4 – Experience of intense cravings for the substance

Taking larger amounts of the substance for longer periods than intended has been cited as one of the most commonly reported symptoms in overweight/obese and BED individuals (Curtis & Davis, 2014; Pretlow, 2011). Excessive and uncontrolled eating also forms the definition of binge eating in BED (DSM-V; APA, 2013). Although bingeing can be a planned behaviour, it is argued that planned binges still result in a greater intake than initially intended (Curtis & Davis, 2014). Binge eating has also been documented in non-clinical samples (French, Jeffery, Sherwood & Neumark-Sztainer, 1999); however, in these individuals occasions of impaired

control are more likely to reflect unintentional snacking and excessive portion sizes (Curtis & Davis, 2014; Levitsky, 2005; Macdiarmid, Loe, Kyle & McNeill, 2013).

Unsuccessful efforts to restrict food intake are also well documented with many dieters failing to maintain their diet or even gaining weight in the long term (Bacon & Aphramor, 2011; Dansinger, Gleason, Griffith, Selker, & Schaefer, 2005; Jeffery *et al.*, 2000; Lowe *et al.*, 2006; Mann, Tomiyama, Westling, Lew, Samuels & Chatman, 2007; Pietiläinen, Saarni, Kaprio & Rissanen, 2012). In their paper reviewing evidence for refined food addiction (i.e. processed foods with high levels of sugars or sweeteners, refined carbohydrates, fat, salt and caffeine), Ifland *et al.* (2009) report that “Every refined food addict reports a series of attempts to cut back on eating. They have used a variety of techniques” (pg. 521). Curtis and Davis (2014) also report similar anecdotes in women with BED who describe avoiding certain trigger foods to control their binges.

The third criterion of time spent obtaining, using and recovering from substance use also translates to BED. These individuals may spend a lot of their time thinking about, engaging in and recovering from binge episodes. As mentioned earlier, bingeing is often a planned behaviour which may require a great deal of effort to purchase and store foods ready for a binge episode (Curtis & Davis, 2014). In addition, the criteria for BED emphasise the time spent bingeing with the number of binge episodes per week determining the severity of the disorder (DSM-V; APA, 2013). Moreover, these individuals often experience physical and emotional distress following a binge eating episode. Recovery from food consumption has also been reported in self-identified food addicts with references to feeling sleepy or ‘hung-over’ (Ifland *et al.*, 2009; Russell-Mayhew, von Ranson & Masson, 2010). Although evidence of food addiction directly related to the DSM-V diagnostic criteria for impaired control is largely anecdotal, there is a considerable amount of empirical evidence for an association between overeating/ obesity and impaired control generally. Two aspects of self-regulatory failure that are particularly pertinent in the case of substance use and overeating are impulsivity and reward sensitivity (Dawe & Loxton, 2004; Gullo & Dawe, 2008).

1.2.1.1.1. *Impulsivity*

Although impulsivity is a multi-faceted construct, it can broadly be defined as the tendency to think and act without sufficient forethought, which often results in behaviour that is discordant with one's long-term goals. The role of impulsivity in SUDs is well documented (for reviews see Dawe & Loxton, 2004; de Wit, 2008; Gullo & Dawe, 2008; Iacono *et al.*, 2008; Verdejo-García *et al.*, 2008). Many studies have reported higher impulsivity levels with substance use across a wide range of questionnaires and behavioural tasks, for a variety of different substances including ecstasy (Butler & Montgomery, 2004), cocaine (Coffey, Gudleski, Saladin & Brady, 2003; Verdejo-García, Perales & Pérez-García, 2007), methamphetamine (Monterosso *et al.*, 2005), tobacco (Billieux, Gay, Rochat, Khazaal, Zullino & Van der Linden, 2010; Glass *et al.*, 2009; Golding, Harpur & Brent-Smith, 1983; Spillane, Smith & Kahler, 2010; Spinella, 2002) and alcohol (Grau & Ortet, 1999; Jorm, Christensen, Henderson, Jacomb, Korten, & Rodgers, 1999; Lawrence, Luty, Bogdan, Sahakian & Clark, 2009; Noël *et al.*, 2013; Papachristou, Nederkoorn, Havermans, van der Horst & Jansen, 2012). For example Noël *et al.* (2013) performed a series of cognitive tests assessing the ability to overcome irrelevant responses (response inhibition) and irrelevant information (proactive interference) in a group of detoxified alcohol-dependent individuals and matched healthy controls. They found a significant group difference for all three tests assessing response inhibition but no differences for proactive interference.

Impulsivity has also been implicated in overeating and obesity (for reviews see Dawe & Loxton, 2004; Guerrieri, Nederkoorn & Jansen, 2008b; Jentsch & Pennington, 2014; Meule, 2013). Overweight/obese individuals score higher on self-reported (Chalmers, Bowyer & Olenick, 1990; Davis *et al.*, 2008a; Rydén, Sullivan, Torgerson, Karlsson, Lindroos & Taft, 2003) and behavioural measures of impulsivity (Batterink, Yokum & Stice, 2010; Braet, Claus, Verbeken & Van Vlierberghe, 2007; Davis, Patte, Curtis & Reid, 2010; Nederkoorn, Braet, Van Eijs, Tanghe & Jansen, 2006a; Nederkoorn, Jansen, Mulkens & Jansen, 2006b; Nederkoorn, Smulders, Havermans, Roefs & Jansen, 2006c; Pauli-Pott, Albayrak, Hebebrand & Pott, 2010; Weller, Cook, Avsar & Cox, 2008), whereas those high in

self-control have been shown to be less likely to give in to temptation (Friese, Hofmann & Wänke, 2008; Friese & Hofmann, 2009; Hofmann, Friese & Roefs, 2009a) and are more likely to maintain a healthy diet and engage in physical exercise (Crescioni *et al.*, 2011; de Boer, van Hooft & Bakker 2011; Gerrits *et al.*, 2010; Wills, Isasi, Mendoza & Ainette, 2007). Impulsivity scores have also been shown to predict poor food choices (Jasinska *et al.*, 2012) and correlate positively with food consumption (Allan, Johnston & Campbell, 2010; Allom & Mullan, 2014; Churchill & Jessop, 2011; Galanti, Gluck & Geliebter, 2007; Guerrieri, Nederkoorn & Jansen, 2007b; Guerrieri *et al.*, 2007a). For example, Guerrieri *et al.* (2007b) found that, in a sample of normal-weight women, those with higher impulsivity scores ate more candy during a ‘bogus’ taste test than those with lower impulsivity scores. Churchill and Jessop (2011) also showed a predictive relationship between impulsivity and snacking of high fat foods over a two week period. Further causal evidence for the effect of impulsivity on food intake comes from Rotenberg, Lancaster, Marsden, Pryce, Williams and Lattimore (2005; see also Guerrieri, Nederkoorn, Schrooten, Martijn & Jansen, 2009). They demonstrated that when participants were primed with impulsive words in a memory task, they ate 30% more ice cream during a bogus taste test than participants primed with self-control words. However, without a neutral control group it is unclear whether these results are due to an increase in consumption in the impulsive group or a decrease in consumption in the self-control group (see Figure 1.2).

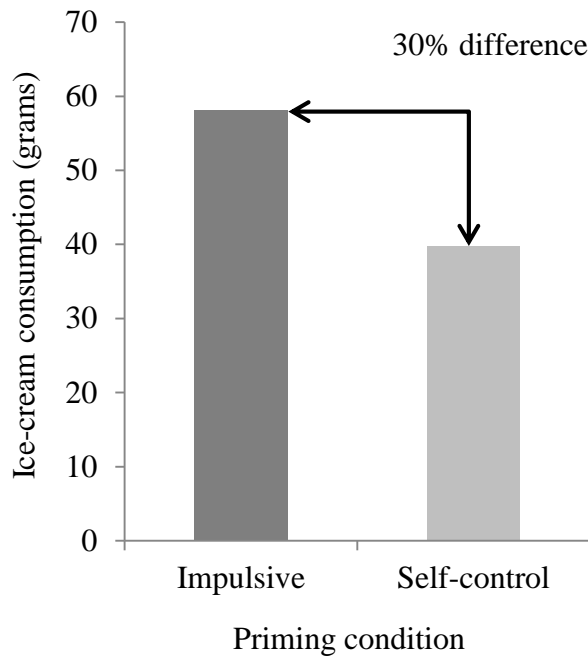


Figure 1.2. Results from Rotenberg *et al.* (2005) showing the effect of memory priming on food intake. Priming participants with impulsive words resulted in a 30% increase in ice-cream consumption compared with those who were primed with self-control words.

1.2.1.1.2. Reward Sensitivity

A heightened sensitivity to reward (STR) has also been linked to both substance use and overeating (Appelhans, Woolf, Pagoto, Schneider, Whited, & Liebman, 2011; Davis *et al.*, 2008b; Davis, Patte, Levitan, Reid, Tweed & Curtis, 2007; Davis *et al.*, 2009; Davis, Strachan & Berkson, 2004a; Dissabandara, Loxton, Dias, Dodd, Daghish & Stadlin, 2014; Franken & Muris, 2005; Guerrieri, Nederkoorn & Jansen, 2008a; Kambouropoulos & Staiger, 2001; Kane, Loxton, Staiger & Dawe, 2004; Loxton & Dawe, 2006; Nederkoorn *et al.*, 2006a). In the food literature, self-report measures of reward sensitivity have revealed associations with BMI, food craving and preferences for foods high in fat and sugar (Davis *et al.*, 2007; Franken & Muris, 2005). Using two behavioural tasks, Guerrieri *et al.* (2008a) measured reward sensitivity and response inhibition in children aged 8-10. They subsequently measured food intake in a bogus taste test when the foods were either varied or monotonous. Their results revealed that reward sensitive children consumed significantly more calories than non-reward sensitive children only when the food

was varied. There was no effect of response inhibition on food intake, nor any interaction with variety; however unlike reward sensitivity, deficient response inhibition was associated with being overweight. The authors suggested that reward sensitivity may play a causal role in overeating, whereas deficient inhibitory control may be more of a maintaining factor. This fits well with findings from a recent study demonstrating a role of reward sensitivity in the early onset of heroin use and a role of impulsivity in escalating use (Dissabandara *et al.*, 2014; also see Lawrence, Hinton, Parkinson & Lawrence, 2012).

There is also evidence to suggest that reward sensitivity may decrease in the later stages of overeating with studies showing anhedonia, or hypo-reward sensitivity, in obese participants (Davis *et al.*, 2004a; Volkow, Fowler & Wang, 2003b; Volkow *et al.*, 2008b; Wang *et al.*, 2001). For example, Davis *et al.* (2004a) demonstrated that although overweight women were more sensitive to reward than normal weight women, those who were obese were significantly less reward sensitive than overweight women. Importantly, the earlier mentioned association between reward sensitivity and increased BMI was found in a sample of mainly (83%) healthy weight women, with only 1% classified as obese (Franken & Muris, 2005). Although there is a great deal of evidence to suggest that STR plays a role in substance abuse and overeating, the causal direction of this relationship remains unclear. On the one hand, increasing STR may lead to overeating by increasing motivation towards pleasurable activities. On the other hand, decreased STR may cause individuals to seek out rewarding activities as a form of 'self-medication' in order to boost dopamine functioning (i.e. addictive behaviour is the result of a 'reward deficiency syndrome'; Blum *et al.*, 2000; Bowirrat & Oscar-Berman, 2005). These two arguments, and relevant neuroimaging literature, are discussed further below (see section 1.2.3.) and in more detail by Burger and Stice (2011).

Burger and Stice (2011) offer several theories for how these two causal directions combine to explain obesity and propose that high STR may initially cause individuals to over-consume palatable foods but this sensitivity is then reduced over time as the brain's reward system becomes habituated. According to Robinson and Berridge's (1993, 2001, 2003) incentive-sensitisation theory this process results in

an increased incentive value for these foods, which is subjectively experienced as excessive ‘wanting’ or craving. Moreover, this theory argues that with repeated presentations of palatable foods, the hedonics for the food will decrease while the anticipation of reward increases. Hence, a circularity emerges in which the individual will experience less pleasure from the food (‘liking’), but will simultaneously experience an increased desire (‘wanting’) for the food (see also Berridge, 2009; Berridge, Ho, Richard & DiFeliceantonio, 2010; see Figure 1.3). The experience of intense cravings is the third criterion of impaired control and is another symptom of substance addiction that can be readily applied to overeating and obesity.

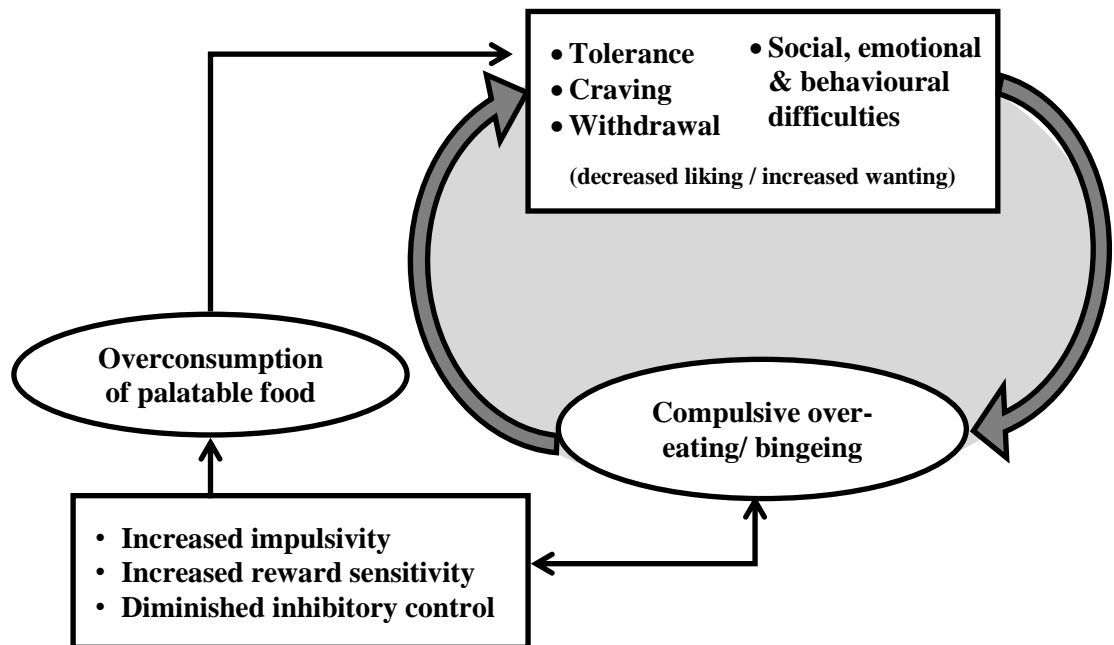


Figure 1.3. The proposed cycle of ‘food addiction’. Initial vulnerability for the overconsumption of palatable consumption is marked by increased impulsivity and reward sensitivity as well as a diminished capacity for inhibitory control. As a consequence of overconsumption, individuals experience tolerance, craving and withdrawal, along with a range of social, emotional and behavioural difficulties such as weight stigmatisation and feelings of guilt and disgust. With repeated consumption of these foods the individual is likely to habituate to the hedonic properties of the food, resulting in reduced enjoyment or liking. These changes are also accompanied by an increased desire or ‘wanting’ for the food (Berridge, 2009; Berridge *et al.*, 2010; Robinson & Berridge, 1993, 2001, 2003). In an

attempt to relieve these symptoms the individual ‘self-medicates’ by increasing food consumption which can result in compulsive or binge eating behaviour, thus creating a cycle of addiction.

1.2.1.1.3. Craving

The term ‘food craving’ typically refers to an intense desire to consume a specific food (Weingarten & Elston, 1990). Food cravings appear to be very common with reports of 100% of young women and 70% of young men experiencing a craving for at least one food in the past year. The most commonly reported craved food is chocolate, although cravings for carbohydrates and salty snacks are also common (Cocores & Gold, 2009; Corsica & Spring, 2008; Hill & Heaton-Brown, 1994; Hill *et al.*, 1991; Massey & Hill, 2012; Rozin, Levine & Stoess, 1991; Weingarten & Elston, 1991; see section 1.2.2. below for a more detailed review on the addictive potential of specific foods). The prevalence of food cravings has prompted the development of several standardised questionnaires that measure food cravings with a good degree of internal consistency and construct validity (Cepeda-Benito, Gleaves, Williams, & Erath, 2000; Hill *et al.*, 1991; Hill & Heaton-Brown, 1994; Nicholls & Hulbert-Williams, 2013; Nijs, Franken & Muris, 2007; White, Whisenhunt, Williamson, Greenway & Netemeyer, 2002), including a specific questionnaire just for chocolate (attitudes to chocolate questionnaire; Benton, Greenfield & Morgan, 1998). Recurrent food cravings are of interest with regards to food addiction as they have been associated with binge eating, increased food intake and increased BMI (Burton, Smit & Lightowler, 2007; Dalton, Blundell & Finlayson, 2013; Hill *et al.*, 1991; Lafay *et al.*, 2001; White, Whisenhunt, Williamson, Greenway & Netemeyer, 2002). Increased reports of food craving have also been demonstrated in individuals who score highly on measures of self-reported food addiction (Davis, Curtis, Levitan, Carter, Kaplan & Kennedy, 2011; Meule & Kübler, 2012) and those with BED (Mussell, Mitchell, deZwaan, Crosby, Seim & Crow, 1996; Ng & Davis, 2013). Furthermore, just as drug craving is associated with an increased likelihood of relapse (Bottlender & Soyka, 2004; Killen & Fortmann, 1997; Litt, Cooney & Morse, 2000; Paliwal, Hyman & Sinha, 2008; Doherty,

Kinnunen, Militello & Garvey, 1995), food craving has been linked to poor dietary success (Gendall, Sullivan, Joyce, Fear & Bulik, 1997; Meule, Lutz, Vögele & Kübler, 2012b; Meule, Westenhöfer, & Kübler, 2011b).

Further support for the similarity between drug and food craving is evident in the findings of cue-reactivity research. The aphorism that cravings are most likely to occur in the presence of substance-related stimuli has been well documented, with cue-exposure paradigms showing significant effects of drug-related cues on self-reported and physiological measures of craving (Carter & Tiffany, 1999; Davidson, Tiffany, Johnston, Flury & Li, 2003; Mahler & de Wit 2010; Miranda, Rohsenow, Monti, Tidey & Ray, 2008; Sinha, Fuse, Aubin & O'Malley, 2000; Styn, Bovbjerg, Lipsky & Erblich, 2013). Similarly, exposure to food cues has also been shown to increase food cravings (Cornell, Rodin & Weingarten, 1989; Nederkoorn, Smulders & Jansen, 2000), especially amongst binge eaters and those with BED (Karhunen, Lappalainen, Tammela, Turpeinen & Uusitupa, 1997; Sobik, Hutchison & Craighead, 2005; see Jansen (1998) for a theoretical review on the role of cue conditioning in binge eating). Furthermore, this heightened reactivity in binge eaters has been correlated with binge eating frequency and BMI (Sobik *et al.*, 2005). It is possible therefore that certain individuals are more susceptible to cue-induced cravings, and also that this susceptibility may transfer across different substances. Both Mahler and de Wit (2010) and Styn *et al.* (2013) found a significant correlation between cue-induced cigarette craving and cue-induced food craving in smokers, suggesting a common mechanism.

1.2.1.2. Social Impairment

C5 – Inability to fulfil major social obligations as a result of substance use

C6 – Social or interpersonal problems that are due to or exacerbated by substance use

C7 – Reduced social activities as a result of substance use

Overeating and obesity have been associated with poor social health especially amongst children and adolescents. When assessing quality of life with child and parent-proxy reports, social functioning is significantly lower for obese compared to

healthy weight children and is inversely correlated with BMI (Schwimmer, Burwinkle & Varni, 2003; Williams, Wake, Hesketh, Maher & Waters, 2005). Poor social functioning in overweight children may be partly due to the overt victimisation and teasing experienced as a direct result of their weight status (Griffiths, Wolke, Page & Horwood, 2006; Hayden-Wade, Stein, Ghaderi, Saelens, Zabinski & Wilfley, 2005; Pearce, Boergers & Prinstein, 2002). Hayden-Wade *et al.* (2005) found that the degree of teasing experienced by overweight children was positively correlated with loneliness, an increased preference for isolative activities and a lower preference for social activities. This preference for being alone, along with the emotional difficulty of being victimised, creates a vicious cycle as these circumstances are likely to promote further overeating and binge-eating which in turn leads to increased weight gain and further teasing (Pretlow, 2011; Neumark-Sztainer, Falkner, Story, Perry, Hannan & Mulert, 2002; see Figure 1.3). Weight stigmatisation may also affect interpersonal friendships and romantic relationships in adulthood with reports of discriminatory attitudes and behaviours in occupational (Puhl & Brownell, 2001; Puhl & Heuer, 2009) and romantic settings (Chen & Brown, 2005; Pearce *et al.*, 2002; Puhl & Heuer, 2009; Puhl & Latner, 2007). For example, Chen and Brown (2005) reported that when making sexual choices about a partner, both male and female college students ranked an obese individual as the least liked.

1.2.1.3. Risky Use

C8 – Continued substance use despite physically hazardous situations

C9 – Substance use despite knowledge of resulting physical or psychological problems

It has been noted that due to the increase in prevalence of obesity and its associated comorbidities, obesity now appears to be a greater threat to the burden of disease than smoking (Jia & Lubetkin, 2010). The physical and psychological effects of overweight and obesity are well documented and include, but are not limited to, depression, an increased risk of diabetes, hypertension, cardiovascular disease and some cancers (Bray, 2004; Carpenter, Hasin, Allison & Faith, 2000; Haslam & James, 2005; Kahn, Hull & Utzschneider, 2006; Lopresti & Drummond, 2013;

Luppino *et al.*, 2010; Mokdad *et al.*, 2003; Van Gaal, Mertens & De Block, 2006). Despite these risks, individuals who undergo weight loss treatment often struggle with compliance. As a consequence, treatment is often ineffective in the long-term with many patients failing to lose weight or even gaining weight following intervention (Bacon & Aphramor, 2011; Jeffery *et al.*, 2000; Mann *et al.*, 2007; Pietiläinen *et al.*, 2012).

1.2.1.4. Pharmacological Criteria

C10 – Tolerance for the substance, defined by increased dosage to achieve the desired effect or a reduced effect with the usual dosage.

C11 – Withdrawal from the substance, defined by the characteristic withdrawal symptoms for the substance or consumption of the substance to relieve the symptoms of withdrawal

Tolerance to a substance occurs when the same amount of the substance has an increasingly diminished effect with repeated use. This usually results in escalated use as the individual increases their dosage in order to recreate the original experience. There is some evidence of food tolerance in animal models of sugar addiction. Rats given intermittent and excessive access to sugar solution increase their intake significantly over time, which is accompanied by neurochemical changes that are similar to those seen in drug abuse (Colantuoni *et al.*, 2001; Rada, Avena & Hoebel, 2005). In humans, there is some indication that tolerance to sugar may occur in the first few years of life. The effectiveness of sucrose as an analgesic in young infants is reported to diminish after 18 months of age as sugar consumption increases (Harrison, 2008; King, 1978; Rossow, Kjaernes & Holst, 1990; but see Slater *et al.*, 2010). The possibility of such early tolerance to palatable foods and the methodological difficulties of diet restriction in humans makes finding empirical evidence of tolerance in adults difficult and unlikely. However, statistics indicating increased consumption and portion sizes for these foods provide indirect evidence of tolerance to high-fat/ high-sugar foods at a population level (Ifland *et al.*, 2009; Nielsen & Popkin, 2003), and also at an individual level based on anecdotal reports (Ifland *et al.*, 2009; Pretlow, 2011). For example, Pretlow (2011) found that 77% of overweight poll respondents reported eating more now than when they originally

became overweight. Furthermore, in response to a follow-up question asking why they believed that they ate more, 15% indicated that they were less satisfied by food.

Withdrawal is the second pharmacological criterion for substance abuse and is defined by the presence of physical or psychological symptoms in response to substance deprivation, or the use of the substance in order to relieve these symptoms. Evidence of withdrawal has also been found in the earlier mentioned animal models of sugar addiction. Under conditions of sugar deprivation these animals show withdrawal symptoms similar to those seen with morphine and nicotine withdrawal, including physical symptoms of teeth chattering, forepaw tremor, head shaking and reduced body temperature (Colantuoni *et al.*, 2002; Wideman, Nadzam & Murphy, 2005; also see Avena, Rada and Hoebel (2008a) for a review) as well as increased aggression (Galic & Persinger, 2002) and anxiety (Avena, Bocarsly, Rada, Kim & Hoebel, 2008b). There are also anecdotal reports of withdrawal-like symptoms in humans including persistent cravings when attempting to reduce food intake (Pretlow, 2011) and the tendency to eat to avoid emotional symptoms such as fatigue, anxiety and depression (Ifland *et al.*, 2009).

1.2.2. Foods with Addictive Potential

If foods are capable of causing addictive-like behaviours then it is important to consider which foods or macronutrients may be particularly problematic. As mentioned earlier, some of the strongest evidence for food meeting the pharmacological criteria for substance addiction comes from animal models of sugar addiction. Rats given intermittent and excessive access to sugar solution show evidence not only of tolerance and withdrawal but also bingeing, craving and cross-sensitisation (Avena *et al.*, 2008a; Colantuoni *et al.*, 2001; Johnson & Kenny, 2010; Rada *et al.*, 2005). Moreover, the neurochemical changes that occur during a binge are similar to those seen in drug consumption (for a review see Avena *et al.*, 2008a). Of particular interest is the finding that dopamine release in the nucleus accumbens (NAc) increases as a function of sucrose concentration not volume (Hajnal, Smith & Norgren, 2004) suggesting that sugar-dense foods may be particularly 'addictive'. One of the most sugar-dense products in the current human diet is sugar-sweetened

soft drinks. Consumption of these drinks has increased rapidly over the last few decades and has been associated with obesity in children and adults (Ludwig, Peterson & Gortmaker, 2001; Malik, Schulze & Hu, 2006; Nielsen & Popkin, 2004). However, despite evidence for their addictive potential, cravings for sweetened soft drinks have not been reported (Weingarten & Elston, 1991). Instead the most commonly craved foods tend to combine high sugar and high fat concentrations. In fact, the most commonly craved food in humans is chocolate, which is made almost entirely of fat and sugar (Drewnowski, 1989; Hill & Heaton-Brown, 1994; Hill *et al.*, 1991; Massey & Hill, 2012; Rozin *et al.*, 1991; Weingarten & Elston, 1991).

There are several cases of chocolate addiction or ‘chocoholism’ discussed in the literature with individuals reporting strong cravings for chocolate that are beyond their control (Benton *et al.*, 1998; Bruinsma & Taren 1999; Hetherington & Macdiarmid, 1993, 1995; Hill & Heaton-Brown, 1994; Massey & Hill, 2012; Rozin *et al.*, 1991; Weingarten & Elston, 1991; for a review see Rogers & Smit, 2000). One possible explanation for the addictive nature of chocolate is the presence of psychoactive stimulants found in cocoa, including theobromine and caffeine. Although concentrations of theobromine are fairly high in chocolate the subjective experience is reported to be relatively modest (Mumford, Evans, Kaminski, Preston, Sannerud, Silverman & Griffiths, 1994). Likewise, the amount of caffeine in chocolate is negligible compared to that of tea and coffee (Barone & Roberts, 1996). However, it is possible that it is the combination of compounds within chocolate that makes it so irresistible. Michener and Rozin (1994) investigated the physiological and sensory satiation of chocolate craving with different chocolate products that varied in the degree of their pharmacological components. Participants were presented with cocoa capsules, a combination of white chocolate (with no pharmacological components) and cocoa capsules, milk chocolate, white chocolate, placebo capsules or nothing. They found that milk chocolate reduced self-reported craving the most, followed by white chocolate and the combination of white chocolate and cocoa capsules. The reduction in craving for the latter did not significantly differ from the reduction for white chocolate alone, thus suggesting that the addition of cocoa had no role in reducing craving. Moreover, the cocoa capsules, placebo capsules and nothing conditions had no effect on craving and did not differ

from one another. These results imply that the psychoactive properties of chocolate play no role in satisfying chocolate craving. This conclusion is supported by reports that milk chocolate is more preferable than dark chocolate, despite its lower cocoa content (Hetherington & Macdiarmid, 1993). Together these findings suggest that chocolate craving and overconsumption are caused by its orosensory properties from the combination of high sugar and fat, which have been argued to have a profound effect on alleviating negative affect (Hetherington & Macdiarmid, 1993). Similarly, carbohydrate craving has also been linked to the alleviation of dysphoric moods following carbohydrate ingestion (Corsica & Spring, 2008; Lieberman, Wurtman & Chew, 1986; Spring *et al.*, 2010).

1.2.3. Neurobiological Similarities Between Palatable Foods and Drugs of Abuse

Just as altered brain functioning has been reported in SUDs, overeating and obesity have also been associated with changes in the neural processing of the motivational properties of food. This includes changes in systems coding the hedonic and rewarding aspects of the substance as well as the systems involved in controlling these motivations (Blumenthal & Gold, 2010; Burger & Stice, 2011; Carnell, Gibson, Benson, Ochner & Geliebter, 2012; Del Parigi, Pannacciulli, Le & Tataranni, 2005; Koob, Sanna & Bloom, 1998; Martin *et al.*, 2010; Parvaz, Alia-Klein, Woicik, Volkow & Goldstein, 2011; Tomkins & Sellers, 2001; Zhang, von Deneen, Tian, Gold & Liu, 2011). Volkow and colleagues (Goldstein & Volkow, 2002, 2011; Volkow *et al.*, 2003b; Volkow, Wang & Baler, 2011a; Volkow, Wang, Fowler & Telang, 2008a; Volkow *et al.*, 2013) have proposed a common model for addiction and obesity that involves two neural circuits that are both modulated by dopamine – increased reward sensitivity and diminished inhibitory control (also see Jentsch & Pennington, 2014).

1.2.3.1. Reward Sensitivity

Addictive drugs directly affect the mesolimbic dopamine system (MDS) which is thought to mediate the processing of pleasure and reward (Pierce & Kumaresan, 2006). Animal studies have shown that, just like drugs of abuse, palatable foods trigger the release of dopamine in the nucleus accumbens (NAc) and ventral

tegmental area (VTA; Hernandez & Hoebel, 1988; Rada *et al.*, 2005; Radhakishun, van Ree & Westerink, 1988; Yoshida *et al.*, 1992). Furthermore, activity in the MDS has been linked to the amount of food ingested and its rewarding properties (Martel & Fantino, 1996a, 1996b). However, distinct patterns of neuronal firing in the NAc to food and illicit substances have also been reported (Carelli, Ijames & Crumling, 2000; see also Caine & Koob, 1994). Higher activation of this reward system has also been shown in human participants during the presentation of food cues and meal consumption (Rothemund *et al.*, 2007; Small *et al.*, 2003; Stoeckel, Weller, Cook, Twieg, Knowlton & Cox, 2008; Volkow *et al.*, 2002a; Volkow *et al.*, 2003a). For example, Small *et al.* (2003) demonstrated that increased dopamine release in the dorsal striatum was correlated with the degree of pleasure reported following a meal. Rothemund *et al.* (2007) also found that viewing high calorie foods activated the dorsal striatum in obese but not healthy weight participants. Obese participants have also demonstrated increased responsivity to food in gustatory and somatosensory regions (Stice, Spoor, Bohon, Veldhuizen & Small, 2008; Wang *et al.*, 2002) suggesting a heightened sensitivity to palatable food that may contribute to overeating and obesity.

Although an increased sensitivity to reward may initially drive individuals to consume calorific foods, it has been speculated that compulsive eating may develop as the pleasure derived from these foods diminishes with increased tolerance (see Figure 1.3). It has been argued that, just as with drugs of abuse, the chronic consumption of such rewarding foods may cause the downregulation of dopamine receptors in order to compensate for their overstimulation (Bello, Lucas & Hajnal, 2002; Johnson & Kenny, 2010; Volkow, Wang, Fowler, Tomasi, Telang & Baler, 2010b). Decreased striatal dopamine receptor availability has frequently been observed in individuals with substance addictions (Fehr *et al.*, 2008; Heinz *et al.*, 2004; Volkow *et al.*, 1993, 1996, 2001), whereas increased receptor availability has been shown to have a protective role against alcoholism (Thanos *et al.*, 2001; Volkow *et al.*, 2006). It has also been shown that striatal D2 receptor availability is significantly lower in severely obese individuals compared to controls and is significantly and negatively correlated with BMI (Volkow *et al.*, 2008b; Wang *et al.*, 2001).

It has been argued therefore that a reduction in dopamine receptor availability may subsequently cause or exacerbate overeating as a form of 'self-medication' whereby the individual attempts to compensate for a diminished experience of reward (Reinholz, Skopp, Breitenstein, Bohr, Winterhoff & Knecht, 2008; Wang *et al.*, 2001; Wang, Volkow, Thanos & Fowler, 2004, but see Hardman, Herbert, Brunstrom, Munafò & Rogers, 2012; See Figure 1.4). For example, Geiger *et al.* (2009) found that rats fed on a cafeteria-style diet showed reduced baseline levels of mesolimbic dopamine activity. This activity was stimulated by cafeteria foods but not by their regular chow, thus suggesting that a preference for palatable food may develop as a consequence of its ability to increase dopamine release compared to other, less palatable, foods. Animal studies have also demonstrated causal effects of D2 receptor agonists and antagonists on overeating. The administration of D2 antagonists has been shown to increase meal size, meal duration and body weight, whereas treatment with D2 agonists can reduce hyperphagia and prevent weight gain (Baptista, Parada & Hernandez, 1987; Clifton, Rusk & Cooper, 1991; Scislowski, Tozzo, Zhang, Phaneuf, Prevelige & Cincotta, 1999). In humans the use of antipsychotic medication which blocks D2 receptors is typically associated with weight gain (Goudie, Cooper & Halford, 2005) whereas some D2 agonists have been found to reduce body weight (Cincotta & Meier, 1996; but see Gibson, Karmally, McMahan, Wardlaw, & Korner, 2012; Pijl *et al.*, 2000). A recent study with gastric bypass patients has also demonstrated increased receptor availability following weight loss indicating that the effects of overeating on dopamine receptor downregulation may be reversible (Steele *et al.*, 2010; see also Ochner *et al.*, 2011).

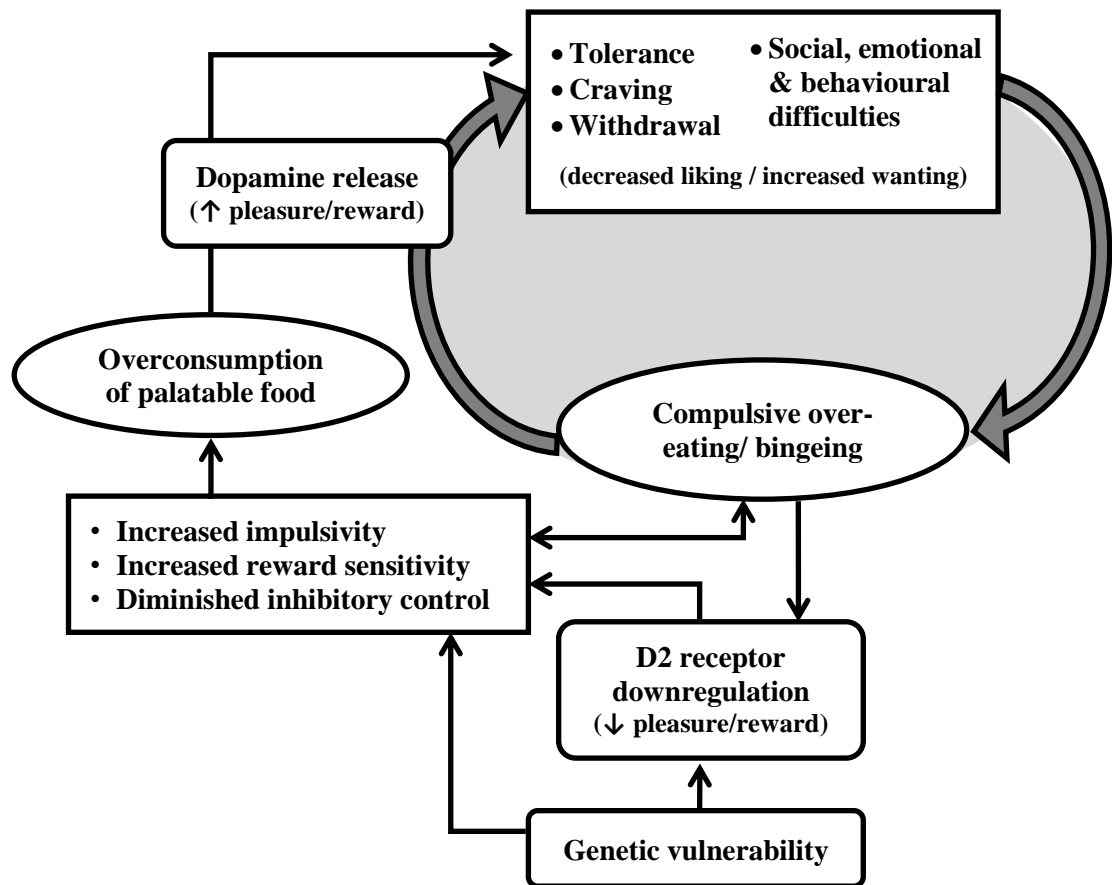


Figure 1.4. The proposed cycle of ‘food addiction’ including the role of dopamine. When palatable food is consumed, the brain releases the hormone dopamine. Over time this increase in dopamine leads to the downregulation of dopamine receptors, causing individuals to experience a reduction in pleasure during palatable food consumption. This decrease in pleasure, combined with symptoms of tolerance, craving, withdrawal and other social, emotional and behavioural difficulties, results in the individual engaging in compensatory behaviour by increasing food consumption. As a consequence, food consumption may become compulsive, thus creating a cycle of food addiction.

1.2.3.2. Inhibitory Control

Dopamine receptor availability in obese individuals has also been shown to correlate positively with metabolism in prefrontal regions involved in inhibitory control (specifically the dorsolateral prefrontal cortex (DLPFC), medial orbitofrontal cortex (OFC) and anterior cingulate gyrus as well as the somatosensory cortices; Volkow *et al.*, 2008b). Similar findings have been observed in healthy weight participants, who

demonstrated a positive correlation between receptor availability and inhibitory control performance on the stop-signal task (Ghahremani *et al.*, 2012). Volkow *et al.* (2008b) hypothesised that altered dopamine functioning may play a role in overeating not only through increasing the rewarding properties of food but also by reducing inhibitory control. A significant negative correlation between BMI and prefrontal activity has also been reported (Batterink *et al.*, 2010; Volkow *et al.*, 2009a) along with reduced prefrontal activation following a meal in obese men and women (Le *et al.*, 2006; Le *et al.*, 2007, but see Gautier *et al.*, 2000). Conversely, successful dieting has been positively associated with frontal activation (Del Parigi *et al.*, 2007; Hollmann *et al.*, 2012; McCaffery *et al.*, 2009; Weygandt *et al.*, 2013).

In a study of healthy women, Lawrence *et al.* (2012) reported an association between food cue reactivity in the NAc and later snack consumption (see also Lopez, Hofmann, Wagner, Kelley & Heatherton, 2014). They also found that this reactivity was associated with increased BMI for individuals who reported low self control. The authors proposed a ‘dual hit’ of increased reward motivation and poor self control in predicting increased food intake (see also Nederkoorn, Houben, Hofmann, Roefs & Jansen, 2010). Similarly, reductions in frontal grey matter volume have also been linked to increased BMI, poor food choices and related deficits in executive functioning (Cohen, Yates, Duong & Convit, 2011; Maayan, Hoogendoorn, Sweat & Convit, 2011; Pannacciulli, Del Parigi, Chen, Le, Reiman & Tataranni, 2006; Taki *et al.*, 2008; Walther, Birdsill, Glisky & Ryan, 2010; Yokum & Stice, 2013). These findings are reflective of a growing literature on the cognitive dysfunction associated with drug abuse and obesity, although research indicates that the causal relationship is bidirectional (Bechara, 2005; Bechara, Dolan, Denburg, Hines, Anderson & Nathan, 2001; Bechara & Martin, 2004; Davis, Levitan, Muglia, Bewell & Kennedy, 2004b; Davis *et al.*, 2010; Elias, Elias, Sullivan, Wolf, D’Agostino, 2005; Gunstad, Paul, Cohen, Tate, Spitznagel & Gordon, 2007).

Although it has been hypothesised that overeating is initially caused by a hyper-responsive reward circuitry and maintained by the subsequent degradation of this system (Burger & Stice, 2011), there is also evidence to suggest that some individuals may be genetically vulnerable to an impaired capacity for reward and

inhibitory control. Genetics studies have revealed that both drug users and obese individuals have a significantly greater prevalence of the *TaqI* A1 allele polymorphism which can cause a 30-40% reduction in striatal D2 receptors (Blum *et al.*, 1996; Comings, Muhleman, Ahn, Gysin & Flanagan, 1994; Han *et al.*, 2008; Jönsson *et al.*, 1999; Noble, 2000; Spitz *et al.*, 2000; Stice, Spoor, Bohon & Small, 2008). In addition, this polymorphism has been associated with behavioural measures of impulsivity and reward sensitivity (Eisenberg *et al.*, 2007; Klein, Neumann, Reuter, Hennig, von Cramon & Ullsperger, 2007). It has also been linked to low grey matter volume in the anterior cingulate cortex (ACC; Montag, Weber, Jentsgens, Elger & Reuter, 2010), an area which is believed to be involved in executive control and reward expectancy (Gasquoine, 2013; Ghahremani *et al.*, 2012; Peoples, 2002), and has been shown to be active during resistance of cigarette craving (Brody *et al.*, 2007). Another recent study found that obese and BED participants carrying the A1 allele reported increased reward sensitivity on two self-report measures, although these measures were not specific to food reward (Davis *et al.*, 2008b). Together these findings demonstrate that obesity and SUDs may share a common neurobiological mechanism involving altered dopamine functioning that subsequently disrupts mechanisms involved in reward sensitivity and inhibitory control.

1.2.4. Assessment of 'Food Addiction'

An important step for furthering research into the possibility of food addiction is the development and use of a valid assessment tool for identifying those who most closely meet the diagnostic criteria for substance addiction. A critical limitation of the current literature is that it has largely relied on individuals' self-reported food addiction. This step has recently been set in motion with the development of the Yale Food Addiction Scale (YFAS; Gearhardt, Corbin & Brownell, 2009b). The YFAS is a questionnaire that was designed to measure food addiction (especially for palatable foods) in a way that parallels the diagnostic criteria for SUDs (according to the DSM-IV-TR). The scale has so far been shown to exhibit good internal reliability as well as convergent, discriminant and incremental validity (Clark & Saules, 2013; Davis *et al.*, 2011; Davis, Loxton, Levitan, Kaplan, Carter & Kennedy, 2013;

Eichen, Lent, Goldbacher & Foster, 2013; Flint, Gearhardt, Corbin, Brownell, Field & Rimm, 2014; Gearhardt *et al.*, 2009b; Gearhardt, Roberto, Seamans, Corbin & Brownell, 2013; Meule, Heckel & Kübler, 2012a; Murphy, Stojek & MacKillop, 2014). It has also been demonstrated to be a useful tool across a range of populations including undergraduate students (Gearhardt *et al.*, 2009b; Murphy *et al.*, 2014), weight loss patients (Clark & Saules, 2013; Eichen *et al.*, 2013; Gearhardt *et al.*, 2011a), bariatric surgery patients (Meule *et al.*, 2012a), overweight/obese women (Davis *et al.*, 2011), BED patients (Gearhardt *et al.*, 2012) and children (Gearhardt *et al.*, 2013; see also Merlo, Klingman, Malasanos and Silverstein (2009) for evidence of food addiction in children). These studies have shown that scores on the YFAS are positively associated with a range of addiction-related symptoms including measures of depression, impulsivity, food cravings, BMI and BED. Moreover, the YFAS has been shown to predict binge-eating behaviour above and beyond other measures (Gearhardt *et al.*, 2009b, 2012). A standardised measure for operationalising ‘food addiction’ should help to homogenise samples in future behavioural, neuroimaging and genetic research, therefore allowing for a more rigorous assessment of the food addiction concept. This tool may also prove to be beneficial for identifying individuals at high-risk of overeating, obesity and BED. Validation of the scale in children (Gearhardt *et al.*, 2013) may also help clinicians to implement early prevention strategies.

1.2.5. Treatment Implications

One of the greatest potential advantages of identifying the similarities between substance addictions and obesity is the development of effective interventions. The standard approach to weight loss, involving maintaining a healthy diet and physical exercise, is often associated with poor adherence rates and overall weight gain (Bacon & Aphramor, 2011; Dansinger *et al.*, 2005; Dishman, 1991; Jeffery *et al.*, 2000; Lowe *et al.*, 2006; Mann *et al.*, 2007; Pietiläinen *et al.*, 2012). One possible reason for the ineffectiveness of dieting is that it is treating the outcome of overeating and not the underlying cause. Approaches that target the criteria of substance addiction, such as increased impulsivity, reward sensitivity and cravings may have more success. For example, Hall, Fong, Epp and Elias (2008) showed that

executive function on the go/no-go task (a measure of response inhibition; see Table 1.1.) predicted unique variance for dietary behaviour and physical exercise, and also moderated the association between intentions and behaviour (see also Hall, 2012). This suggests that individuals who are more capable of controlling their impulsive actions are more likely to successfully meet their goals. This also implies that techniques to improve such abilities may prove to be effective tools for aiding weight loss. The association between response inhibition and overeating, and the potential effectiveness of response inhibition training are discussed in detail later in this review (see sections 1.3. and 1.3.1.). This section discusses other cognitive and neural interventions.

1.2.5.1. Cognitive Interventions

As discussed earlier in this literature review (see section 1.1.1.), increased motivation for illicit substances has been associated with several cognitive biases including an attentional bias (Bradley *et al.*, 2003, 2004; Ehrman *et al.*, 2002; Fadardi & Cox, 2009; Field *et al.*, 2004, 2006; Lubman *et al.*, 2000; Miller & Fillmore, 2010; Townshend & Duka, 2001; for reviews see Field & Cox, 2008; Franken, 2003) and an approach bias (Bradley *et al.*, 2004; Cousijn *et al.*, 2011; Field *et al.*, 2006, 2008; Mogg *et al.*, 2003; for a review see Watson *et al.*, 2012). One method for reducing this motivation, therefore, has been to use training tasks that are designed to reduce these cognitive biases.

Attentional biases towards food have also been demonstrated across various populations including those with disordered eating patterns (Brignell, Griffiths, Bradley & Mogg, 2009; Brooks, Prince, Stahl, Campbell & Treasure, 2011; Hardman, Scott, Field & Jones, 2014; Hollitt, Kemps, Tiggemann, Smeets & Mills, 2010; Hou, Mogg, Bradley, Moss-Morris, Peveler & Roefs, 2011). Although an attentional bias towards food has also been demonstrated in healthy-weight participants, this bias appears to be exaggerated in those who are overweight or obese (Castellanos *et al.*, 2009; Nijs, Muris, Euser & Franken, 2010). For example, Nijs *et al.* (2010) reported that overweight and obese individuals showed an enhanced orientation bias towards food compared to healthy-weight controls. This is

in accordance with other findings indicating that overweight individuals may show an approach-avoidance pattern with food whereby they are faster to direct their attention towards food initially but are also faster to shift their attention away from food – perhaps when these processes are under volitional control (Werthmann, Roefs, Nederkoorn, Mogg, Bradley & Jansen, 2011). Furthermore, Werthmann *et al.* (2011) showed a positive correlation between subjective craving scores and initial orienting bias in overweight individuals. This finding is reflective of the substance addiction literature which has also demonstrated a positive association between craving scores and measures of attentional bias (Field & Cox, 2008). In a later study, these researchers (Werthmann, Roefs, Nederkoorn & Jansen, 2013b) also found a longer initial duration of gaze towards chocolate in a sample of high chocolate cravers, compared to those low in chocolate craving. In addition, craving scores across the whole sample were correlated with both initial gaze duration and dwell time bias, and these measures were also correlated positively with food intake in a bogus taste test. The induction of chocolate craving has also been shown to have an effect on increased attentional bias towards chocolate (Kemps & Tiggemann, 2009) and increased distracter effects of chocolate (Smeets, Roefs & Jansen, 2009), thus demonstrating causal effects. Together, these findings demonstrate a possible initial orienting bias towards food in obese and overweight individuals which appears to be related to increased craving and food intake.

Just as the addiction literature has explored whether the attentional bias can be manipulated to reduce substance intake, this approach has also been explored with food consumption, although with mixed results (Kakoschke, Kemps & Tiggemann, 2014; Hardman, Rogers, Etchells, Houstoun & Munafò, 2013; Kemps, Tiggemann, Orr & Gear, 2013a; Werthmann, Field, Roefs, Nederkoorn & Jansen, 2014). Hardman *et al.* (2013) trained undergraduate students on the visual probe task to either attend or avoid images of cake and stationery. They found a modest increase in attentional bias for the attend-cake group but no effects of bias training on hunger or food consumption; this led them to conclude that any attentional biases with food may be particularly difficult to modify. However, using a female-only sample, Kemps *et al.* (2013a) did manage to manipulate attentional bias towards and away from chocolate using a similar training procedure. Furthermore, this bias generalised

to novel pictures and also had an effect on food consumption – participants in the avoid-chocolate group consumed significantly less chocolate muffin in a subsequent taste test than those in the attend-chocolate group. However, in a second study, those in the avoid group consumed significantly more blueberry muffin than those in the attend group, indicating that any effect of training on consumption may be specific to the trained foods. This latter finding undermines any effect of training on important health outcomes, especially if trained individuals compensate for any training effects with increased consumption of other, equally unhealthy, foods. In another study, Werthmann *et al.* (2014) also found that the effect of training on food consumption was moderated by training performance. Only participants with high accuracy scores during training demonstrated the expected effects whereas those with low accuracy scores demonstrated the reversed pattern. On a more positive note, however, another recent paper has shown that attentional bias modification may be beneficial for adjusting food choices towards healthier options. Kakoschke *et al.* (2014) found that undergraduate women who were trained to attend to healthy food (and ignore unhealthy food) showed an increased attentional bias for these foods and also consumed more healthy than unhealthy snacks compared with those trained to attend to unhealthy foods (and ignore healthy foods). Unfortunately, however, the authors analysed these results as a proportion of healthy to unhealthy snack intake making it difficult to determine any effects on total calorie intake for either healthy or unhealthy foods separately. Since there was no control group, it is also difficult to determine whether these effects were due to a decrease in consumption in the attend-healthy group or an increase in consumption in the attend-unhealthy group.

There is also a small body of evidence demonstrating an approach tendency towards food for individuals with high levels of external, emotional and restrained eating (Brignell *et al.*, 2009; Veenstra & de Jong, 2010; but see Ahern, Field, Yokum, Bohon & Stice, 2010) as well as those who are overweight or obese (Havermans, Giesen, Houben & Jansen, 2011; Mogg *et al.*, 2012). For example, Veenstra and de Jong (2010) showed that those who scored highly on a measure of dietary restraint (a measure for the chronic, cognitive limitation of food intake; see section 1.4. for a detailed review of dietary restraint) were significantly faster to move a manikin towards than away from images of food. Using a similar task, Havermans *et al.*

(2011) found that overweight/obese men were significantly slower to avoid food images, however, overweight/obese women showed a weaker approach bias towards high calorie foods compared to lean females. Consistent with the attentional bias literature the authors argued that overweight women may be more ambivalent towards food, showing an equally strong avoidance tendency. They attributed this to the increased restraint scores for overweight compared to lean women. Although these results directly contradict those of Veenstra and de Jong, the two studies used different measures of dietary restraint that are associated with unsuccessful and successful restraint, respectively (see section 1.4.3. for a discussion of these measures and their relation of disinhibited food intake). Together, these findings suggest that only those scoring highly on measures of unsuccessful dietary restraint show an increased approach bias towards food. However, Van Gucht, Vansteenwegen, Van den Bergh and Beckers (2008) have demonstrated a strong approach tendency for cues previously paired with chocolate following craving induction in a sample of undergraduate students. Using a different measure of approach bias, Kemps, Tiggemann, Martin and Elliott (2013b) also found that participants who liked chocolate were significantly faster at pairing images of chocolate with approach words compared to avoid words. Furthermore, they also demonstrated that participants who were trained to pair images of chocolate with either approach or avoid words increased and decreased their approach bias, respectively. The approach group also demonstrated a significant increase in chocolate cravings, and although the avoid group showed a decrease in reported craving this was not statistically significant from baseline.

Together the findings on substance-related cognitive biases indicate similar cognitive processes in substance addictions and disordered eating. Although the research on their associated interventions is preliminary, and sometimes contradictory, it provides us with a greater understanding of the mechanisms involved in impulsive behaviour and potential methods for effective interventions. As mentioned earlier, another avenue of investigation has explored response inhibition training as a way of decreasing impulsivity. To date this approach has shown encouraging effects, although more research is required to determine their extent and validity (see section 1.3.1.).

1.2.5.2. Neuromodulation Interventions

Non-surgical brain stimulation techniques have also been explored for their potential benefits in reducing craving and addictive behaviours by altering neural activity and increasing dopamine (for reviews see Barr, Fitzgerald, Farzan, George & Daskalakis, 2008; Diana, 2011; Feil & Zangen, 2010; Herremans & Baeken, 2012; Nardone *et al.*, 2012). The most commonly applied stimulation methods are transcranial magnetic stimulation (TMS) and transcranial direct current stimulation (tDCS). These methods are used in awake participants and are generally considered to be safe when used within the recommended guidelines (Loo, McFarquhar & Mitchell, 2008; Maizey *et al.*, 2013; Pascual-Leone *et al.*, 1993; Poreisz, Boros, Antal & Paulus, 2007).

TMS involves the delivery of electromagnetic pulses that penetrate the skull to induce electric current in the underlying cortex and cause short-term changes in cortical excitability. The modulation of cortical excitability can last beyond the period of stimulation by delivering trains of pulses, a technique known as repetitive TMS (rTMS; Fitzgerald, Fountain & Daskalakis, 2006). When applied to the DLPFC, rTMS has been shown to effectively reduce cravings for cigarettes, alcohol and cocaine, especially when applied for multiple sessions (Amiaz, Levy, Vainiger, Grunhaus & Zangen, 2009; Camprodon, Martínez-Raga, Alonso-Alonso, Shih & Pascual-Leone, 2007; Eichhammer *et al.*, 2003; Herremans *et al.*, 2012; Mishra, Nizamie, Das & Praharaj, 2010; Politi, Fauci, Santoro & Smeraldi, 2008). The DLPFC is an area involved extensively in inhibitory control (Beeli, Casutt, Baumgartner & Jäncke, 2008; Garavan, Hester, Murphy, Fassbender & Kelly, 2006; Liddle, Kiehl & Smith, 2001; MacDonald, Cohen, Stenger & Carter, 2000; Wager, Sylvester, Lacey, Nee, Franklin & Jonides, 2005; Zheng, Oka, Bokura & Yamaguchi, 2008) and stimulation of this region may act to boost self control, potentially by increasing dopamine release in the caudate nucleus (Diana, 2011; Strafella, Paus, Barrett & Dagher, 2001).

Reductions in substance craving have also been demonstrated with stimulation of the DLPFC using tDCS (Boggio, Liguori, Sultani, Rezende, Fecteau & Fregni, 2009; Boggio *et al.*, 2008; Boggio, Zaghi, Villani, Fecteau, Pascual-Leone & Fregni, 2010). tDCS involves the application of a weak (typically 1-2mA) direct electrical current to the scalp via a pair of electrodes. The effect of tDCS on brain activity is dependent on the stimulation polarity; anodal stimulation is thought to increase cortical excitability by neuronal depolarisation whereas cathodal stimulation is believed to decrease excitability by hyperpolarising neurons (Antal, Terney, Poreisz & Paulus, 2007; Liebetanz, Nitsche, Tergau & Paulus, 2002; Nitsche & Paulus, 2000; Nitsche *et al.*, 2003a, 2005; Priori, 2003). Specifically, the electrical current is thought to modulate the resting cell membrane potential by opening and closing voltage-gated ion channels (Purpura & McMurtry, 1965; Nitsche *et al.*, 2003b). Long-lasting effects on resting membrane potential have also been shown with longer stimulation durations; for example 13 minutes of anodal tDCS has been shown to increase motor cortical excitability for up to 90 minutes (Nitsche & Paulus, 2001). These after-effects are thought to be regulated by N-methyl-D-aspartate (NMDA) receptor efficiency (Liebetanz *et al.*, 2002; Nitsche *et al.*, 2003b). Compared to TMS, tDCS is a weaker form of stimulation with fewer incidental artefacts and is therefore considered to be safer and more appropriate for reliable double-blinding (Gandiga, Hummel & Cohen, 2006; Nitsche *et al.*, 2008; Poreisz *et al.*, 2007).

These stimulation methods are currently being investigated for their potential to reduce food craving and consumption (Claudino *et al.*, 2011; Fregni *et al.*, 2008; Goldman *et al.*, 2011; Montenegro, Okano, Cunha, Gurgel, Fontes & Farinatti, 2012; Uher *et al.*, 2005; Van den Eynde *et al.*, 2010). Using rTMS to the left DLPFC, Uher *et al.* (2005) found an increase in cue-induced craving for palatable foods in the group who experienced sham stimulation but not the active group. However, no effect was found on ad-libitum food consumption, although this may have been due to a ceiling effect as participants consumed a large amount of calories within a short time period. Using a similar methodology, Van den Eynde *et al.* (2010) demonstrated an increase in craving scores in the sham group, but a decrease in craving scores for the active group in a sample of participants with bulimic-type

eating disorders. In addition, active rTMS was associated with a reduction in binge-eating episodes in the following 24 hour period. However, blinding was only partially successful in this study with most participants correctly guessing whether they were receiving active or sham rTMS. In a later study, Barth *et al.* (2011) used a within-subjects design with an improved sham condition in which they matched the perceived pain of active rTMS with scalp electrodes. They found an equal reduction in cravings for both conditions and attributed this effect to the experience of pain rather than prefrontal stimulation.

As mentioned earlier, tDCS is believed to involve a more reliable sham condition, especially when participants receive active stimulation for a short initial period (Gandiga *et al.*, 2006; Nitsche *et al.*, 2008). When stimulating the DLPFC bilaterally using tDCS, Fregni *et al.* (2008) found a significant increase in cue-induced craving, measured before and after stimulation, in the sham condition and a significant reduction when participants received anodal right/ cathodal left stimulation. Compared to the sham condition, active stimulation was also associated with a reduction in food intake during an ad-libitum eating phase. Although the authors did not assess blinding in this study they did report equal occurrences of mild adverse effects across conditions. These studies offer exciting preliminary findings for the effect of neurostimulation techniques on decreasing craving for addictive substances and palatable foods. Although further exploration and replication is required, a recent meta-analysis (Jansen, Daams, Koeter, Veltman, van den Brink & Goudriaan, 2013) revealed a medium effect-size favouring active over sham stimulation in the reduction of cravings. They further reported no significant difference in effect between food and substance craving.

Another neuromodulation intervention, which is worthy of a brief mention and gaining in popularity for the treatment of SUDs, is real-time fMRI (rt-fMRI) neurofeedback training. Neurofeedback training involves providing participants with feedback of their neural response to certain cues and instructing them to increase or decrease their response so that they may gain volitional control over specific brain regions. In the treatment of SUDs this typically involves increasing activity in control regions, such as the prefrontal cortex, or decreasing activity in regions

associated with craving, such as the ACC. For example, it has been shown that decreasing activity in the ACC with rt-fMRI neurofeedback is significantly correlated with decreased nicotine craving in smokers (Hanlon *et al.*, 2013; Hartwell, Prisciandaro, Borckardt, Li, George & Brady, 2013; Li *et al.*, 2012). Using a similar technique with electroencephalography (EEG) has also shown improvements in cravings, drug use and treatment outcomes for a range of different substances (Dehghani Arani, Rostami & Nostratabadi, 2010; Dehghani Arani, Rostami & Nadali, 2013; Horrell *et al.*, 2010; Scott, Kaiser, Othmer & Sideroff, 2005). Although in its early days, the application of neurofeedback training to food consumption and obesity has already been discussed (Dewiputri & Auer, 2013; Frank, Lee, Preissl, Schultes, Birbaumer & Veit, 2012; Frank, Kullmann & Veit, 2013).

1.2.6. Conclusions

As the prevalence of obesity continues to increase and traditional weight loss methods appear to be largely unsuccessful, researchers and clinicians have begun to consider the addictive potential of food. There is a substantial body of evidence demonstrating the similarities between addictive drugs and food on reward and control pathways in the brain and subsequent behaviour such as craving and impulsivity. There is also some limited evidence to indicate that in some circumstances overeating meets the pharmacological criteria of substance dependence, although more research is necessary to determine the validity of these symptoms in human participants. More research is also required for other behavioural criteria such as social impairment and risky use as the evidence to date is largely anecdotal. However, meeting the pharmacological criteria for addiction is not necessary for a diagnosis, and as food is a legal substance, just like caffeine, tobacco and alcohol, not all criteria associated with SUDs (DSM-V; APA, 2013) readily translate to food addiction. Nevertheless, the criterion of withdrawal in SUDs has been associated with clinical severity and the number of symptoms that an individual endorses is used to determine the disorder's overall severity (DSM-V; APA, 2013).

With a number of these criteria having a limited application to food addiction it seems likely that a severe diagnosis would only be made in a small minority of cases. For the vast majority, a diagnosis of ‘food abuse’ may be deemed more appropriate compared to a diagnosis of ‘food addiction’ (Stice, Figlewicz, Gosnell, Levine & Pratt, 2013; Ziauddeen & Fletcher, 2013). Furthermore, it should be made clear that the concept of food addiction does not equate with obesity. Obesity is a multifactorial condition determined by genetic, environmental, biological and behavioural components. For the majority of cases obesity is caused by a steady increase in excess energy intake and is not characterised by a compulsive drive for food consumption. Instead, it is thought that the concept of food addiction applies most appropriately to those with BED (Davis & Carter, 2009), although the two are not synonymous (Gearhardt *et al.*, 2012). Despite there being considerable parallels between substance use and compulsive overeating there is still some concern regarding the use and validity of the term ‘food addiction’, which is unlikely to apply to the majority of cases (Rogers & Smit, 2000).

There is also concern over the use of such terminology in the wider social context and whether the term may be of more harm than good. While most people would believe that an addiction model reduces individual responsibility, it has also been argued that attributing the problem to a minority of individuals also reduces corporate responsibility (Gearhardt *et al.*, 2009a, 2011c). As the majority of the population would be considered to demonstrate a fair degree of restraint over food intake, there would be less pressure for the food industry to reduce marketing and instead promote healthier alternatives. Likewise, any environmental interventions to reduce access and availability may also seem less critical. There are also implications of such terminology for the diagnosed individual. Obesity is already associated with significant social stigmatisation (Chen & Brown, 2005; Griffiths *et al.*, 2006; Hayden-Wade *et al.*, 2005; Pearce *et al.*, 2002; Pearce *et al.*, 2002; Puhl & Brownell, 2001; Puhl & Heuer, 2009; Puhl & Latner, 2007) and an additional ‘addict’ label, which may invoke stereotypes of a person who is untrustworthy and inferior (Earnshaw, Smith & Copenhaver, 2013), may only serve to heighten the problem. However, a recent study investigating the effect of an addiction model on public perceptions found that it actually reduced stigma, blame and perceived

psychopathology (Latner, Puhl, Murakami & O'Brien, 2014), suggesting that it may be beneficial in reducing weight-related prejudice.

Despite these issues and concerns, it has also been acknowledged that for some individuals, 'food addiction' may be the most appropriate diagnosis for their symptoms and may help to inform their treatment (Smith & Robbins, 2013). As discussed in the previous section (1.2.5.), considering the underlying causes of impulsive overeating has led to the development of some exciting and potentially effective new interventions. Although there are differences between the addictive characteristics of food and illicit substances, there are many parallels that should not be ignored. These parallels have contributed greatly to our current knowledge of compulsive overeating and potential treatments. Both the similarities and differences should encourage more research which is necessary to determine the extent and potential impact of such a disorder. Until then the idea of 'food addiction' is expected to remain hotly debated (Avena *et al.*, 2012; Ziauddeen *et al.*, 2012a, 2012b). However, as levels of obesity continue to rise it seems evident that palatable foods are capable of inducing diminished control despite negative consequences. It seems unsurprising therefore that dieting has such a poor adherence rate when tempting food is capable of so easily overpowering our capacity for self-control.

1.3. Inhibitory Control and Obesity

One possible solution for our inability to resist such tempting foods is to increase self-control capacity. As discussed earlier in this review, the strength model of self-control argues that our capacity for self-control can be increased with repeated exercise (Baumeister *et al.*, 2006; Denson *et al.*, 2011b; Finkel *et al.*, 2009; Gailliot *et al.*, 2007; Muraven, 2010; Muraven *et al.*, 1999; Oaten & Cheng, 2006a, 2006b, 2007). For example, practicing small acts of behavioural control, such as squeezing a handgrip or avoiding sweet foods, has been linked to positive health outcomes such as reduced smoking (Muraven, 2010; for a review see Hagger *et al.*, 2009). Another approach is to target executive functions more directly; response inhibition has been a key target for this line of research due to its central role in goal-directed behaviour (Logan, 1985; Logan & Cowan, 1984; Logan *et al.*, 1997). Response inhibition can

be measured with a variety of different tasks such as the Stroop interference task (Stroop, 1935), and perhaps most commonly, the stop-signal task (SST) and go/no-go (GNG) task (Logan & Cowan, 1984; Logan *et al.*, 1997; see Table 1.1. for an overview of these laboratory measures of response inhibition).

Deficient response inhibition has been linked to the use of several different addictive substances (for a review see Jentsch & Pennington, 2014) such as alcohol (Murphy & Garavan, 2011; Noël, Bechara, Dan, Hanak & Verbanck, 2007, Noël *et al.*, 2013; but see Fernie, Cole, Goudie & Field, 2010), cigarettes (Berkman, Falk, & Lieberman, 2011; Luijten *et al.*, 2011; Reynolds, Patak, Shroff, Penfold, Melanko, & Duhig, 2007; Spinella, 2002; but see Dinn, Aycicegi & Harris, 2004), cocaine (Fillmore & Rush, 2002; Hester *et al.*, 2013; Hester & Garavan, 2004; Streeter *et al.*, 2008; Verdejo-García *et al.*, 2007) and methamphetamine (Monterosso *et al.*, 2005; Salo *et al.*, 2002). In addition it has also been linked to the severity and chronicity of use (Billieux, Gay, Rochat, Khazaal, Zullino & Van der Linden, 2010; Lawrence *et al.*, 2009; Nigg *et al.*, 2006) as well as poor treatment outcomes (Krishnan-Sarin *et al.*, 2007). Houben and Wiers (2009) have also shown that positive implicit attitudes towards alcohol are only related to alcohol consumption when inhibitory control is low. This suggests, therefore, that an increased ability to inhibit responses may enable an individual to exert self-control over their behaviour, even when they possess strong implicit preferences.

Table 1.1. Common laboratory measures of response inhibition.

Task	Description	Measure of inhibitory control	Addiction references	Overweight/ obesity references
Stop-signal task	Speeded choice reaction time task with infrequent signals on a minority of trials indicating that the participant must stop their response. Stop-signals are presented after the go stimulus with a variable delay that is often determined by a tracking procedure (ensuring 50% stop performance). This means that participants must inhibit, or cancel, an initiated response.	The stop-signal reaction time (SSRT) is the estimated latency of the stop process. Calculated by subtracting the required stop delay from the mean, median or n th reaction time for no-signal trials (see Verbruggen & Logan, 2009a). Higher SSRTs indicate poor inhibitory control.	(Billieux <i>et al.</i> , 2010; Fillmore & Rush, 2002; Lawrence <i>et al.</i> , 2009; Monterosso <i>et al.</i> , 2005; Nigg <i>et al.</i> , 2006)	(Hofmann <i>et al.</i> , 2009a; Houben <i>et al.</i> , 2012b; Meule <i>et al.</i> , 2014b; Nederkoorn <i>et al.</i> , 2006a, 2006b, 2006c, 2009a, 2010, 2012)
Go/no-go task	Speeded choice reaction time task with a subset of stop trials. The signal to inhibit a response is presented concurrently with the go stimulus so that the response preparation phase is disrupted. Participants can therefore stop, or restrain, a response before it is initiated.	The rate of commission errors (when participants incorrectly respond on a signal trial). Higher commission error rates indicate poor inhibitory control.	(Hester & Garavan, 2004; Luijten <i>et al.</i> , 2011; Murphy & Garavan, 2011; Spinella, 2002; Verdejo-García <i>et al.</i> , 2007)	(Batterink <i>et al.</i> , 2010; Hall, 2012; Jasinska <i>et al.</i> , 2012; Pauli-Pott <i>et al.</i> , 2010; Rosval <i>et al.</i> , 2006; Wirt <i>et al.</i> , 2014)

Task	Description	Measure of inhibitory control	Addiction references	Overweight/ obesity references
Stroop task	Colour naming reaction time task in which the words are either congruent (e.g. the word 'green' is presented in green colour) or incongruent (e.g. the word 'red' is presented in green colour). To respond correctly on incongruent trials, participants must inhibit their automatic response (e.g. to say 'red') and name the colour of the word rather than read the word.	The interference effect is calculated by the difference in reaction times between incongruent and congruent trials. Poor inhibitory control is reflected in a higher interference effect.	(Houben & Wiers, 2009; Noël <i>et al.</i> , 2013; Salo <i>et al.</i> , 2002; Streeter <i>et al.</i> , 2008; Verdejo-García <i>et al.</i> , 2007)	(Allan <i>et al.</i> , 2010; Cohen <i>et al.</i> , 2011; Hall, 2012)

Similar findings have also been replicated with overeating and obesity. Obese individuals have been shown to demonstrate less efficient response inhibition than their healthy-weight counterparts (Cohen *et al.*, 2011; Guerrieri *et al.*, 2008a; Nederkoorn *et al.*, 2006c) and poor inhibitory control has been associated with increased unhealthy food consumption (Allan *et al.*, 2010; Allom & Mullan, 2014; Hall, 2012; Guerrieri *et al.*, 2007a; Houben, 2011), BMI (Allan *et al.*, 2010; Batterink *et al.*, 2010; Lillis, Levin & Trafton, 2012), and food cravings (Meule, Lutz, Vögele & Kübler, 2014b), as well as unhealthy food choices (Allan, Johnston & Campbell, 2011; Jasinska *et al.*, 2012) and binge-eating (Rosval, Steiger, Bruce, Israël, Richardson & Aubut, 2006). For example, Allan *et al.* (2010) asked participants with healthy dietary intentions to perform three tasks measuring different aspects of executive control; they measured inhibition, planning and cognitive flexibility with the Stroop, tower and fluency tasks, respectively. Of all three measures, inhibitory control performance on the Stroop task was the only one to correlate with body mass, showing a positive relationship between poor inhibition and BMI. Allan *et al.* also measured ad libitum chocolate consumption as part of a consumer product test with different fair-trade products (paper, coffee, handcream and chocolate). They found that both poor inhibition and fluency scores were associated with increased chocolate consumption. However, when entered into a regression model, inhibition remained as the only significant predictor, accounting for 23% of the variance in chocolate intake. The ability to inhibit prepotent responses has also been shown to interact with implicit attitudes towards food; just like the addiction literature, research with food suggests that effective response inhibition may protect against increased food consumption (Hofmann *et al.*, 2009a) and weight gain (Nederkoorn *et al.*, 2010) when implicit food preferences are strong.

Associations between poor inhibitory control and obesity have also been shown in children (Nederkoorn *et al.*, 2006a; Nederkoorn, Coelho, Guerrieri, Houben & Jansen, 2012; Nederkoorn *et al.*, 2006b; Wirt, Hundsdörfer, Schreiber, Keszyüs & Steinacker, 2014). Using the GNG task, Wirt *et al.* (2014) found that response inhibition in primary school children was a significant predictor of body weight above and beyond parent education, migration background, parent weight, TV consumption and breakfast habits. Obese children were also found to show the

poorest inhibition performance compared to healthy-weight and overweight children. Nederkoorn and colleagues (Nederkoorn *et al.*, 2006a, 2006b) have found similar results using the SST. As well as showing a relationship between poor inhibitory control and overweight/obesity, they have also demonstrated that individuals with the poorest inhibitory abilities are the least likely to lose weight following treatment. Furthermore, Nederkoorn *et al.* (2012) have demonstrated that the poor inhibitory control found in overweight children may be exaggerated when having to inhibit responses to food stimuli; they found no difference in performance between overweight and healthy-weight children in a toy-related SST, but overweight children were significantly slower to inhibit their responses towards images of highly palatable foods. A similar finding has also been reported in adults with self-reported unsuccessful weight controllers showing an effect of food exposure on food-related but not general stop performance (Houben, Nederkoorn & Jansen, 2012b; see also Meule, Lutz, Krawietz, Stützer, Vögele & Kübler, 2014a). Together, these findings demonstrate a significant relationship between the inability to inhibit a response, perhaps especially towards food-related stimuli, and overeating, overweight and obesity. Effective inhibitory control, on the other hand, may have a beneficial effect for controlling calorie intake, even when an individual possesses strong implicit preferences for palatable foods.

1.3.1. Response Inhibition Training and Food Consumption

Recent research has therefore begun to investigate whether individuals can be trained on response inhibition tasks as a behavioural intervention to reduce food consumption and overweight/ obesity (see Appendix 1 for a summary of methods and results). Not only does this research open a potential avenue for obesity-related treatment but it also suggests a causal relationship between poor inhibitory control and weight gain. It has previously been shown that consistently pairing a stimulus with stopping improves the ability to inhibit responses to that stimulus on future trials (Verbruggen & Logan, 2008). This is an important finding as it suggests that practicing basic motor control towards certain stimuli may improve self-control towards these stimuli on later encounters. This idea has encouraged several research groups to pair images of problematic substances with inhibition in order to reduce

the consumption of that substance. For example, by pairing stop signals with images of beer, Houben and colleagues significantly reduced weekly alcohol intake in a sample of heavy drinking students (Houben *et al.*, 2011a; Houben *et al.*, 2012a; see also Jones & Field, 2013; see Figure 1.5b for a schematic diagram of an inhibition training task with palatable foods).

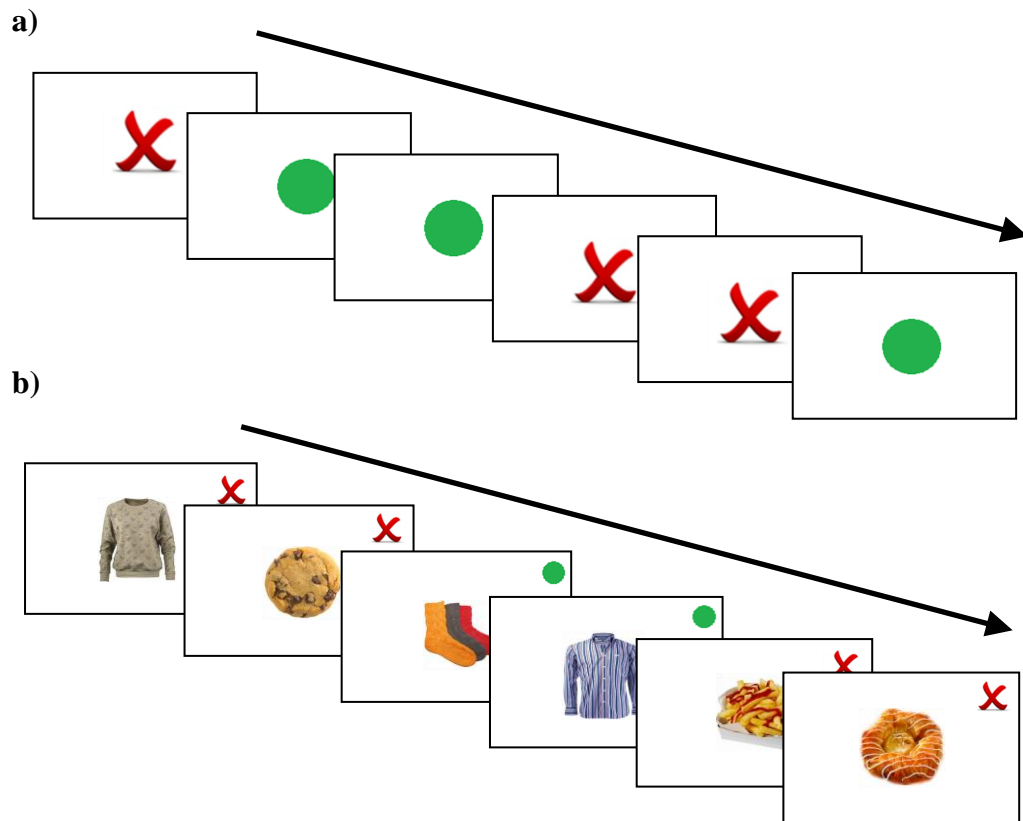


Figure 1.5. Schematic diagram of **a)** a standard go/no-go task and **b)** a food-related go/no-go training task. Participants must respond as quickly as possible when a green circle (go cue) is presented and inhibit their response when a red cross (no-go cue) is presented. In the training task the no-go cue is consistently mapped onto the images of unhealthy foods and filler images are inconsistently paired with both go and no-go cues.

These researchers have also applied the same idea to food consumption. In a within-subjects design, Houben (2011) gave participants a modified version of the SST in which different palatable food items were consistently paired with stopping (100%

inhibition trials), consistently associated with responding (0% inhibition trials) or inconsistently associated with both stopping and responding (50% inhibition trials). After the training task, participants were presented with a bogus taste test to measure food intake; they were presented with the three foods from the training task and were asked to rate the foods along several taste dimensions. They were instructed to consume as much food as they liked and were unaware that the experimenter was only interested in the amount of food consumed and not their rating scores. Prior to the training task, a measure of baseline response inhibition was also recorded with a standard SST. Their results revealed that, consistent with other research (Allan *et al.*, 2010; Allom & Mullan, 2014; Hall, 2012; Guerrieri *et al.*, 2007a), those with low baseline inhibitory control consumed more of the control food (50% inhibition trials) than those who performed well on this task. However, consistently responding to images of food appeared to increase food consumption in those with a good degree of inhibitory control, whereas consistently inhibiting responses to images of food appeared to reduce food consumption for those with poor inhibitory control abilities (see Figure 1.6a.).

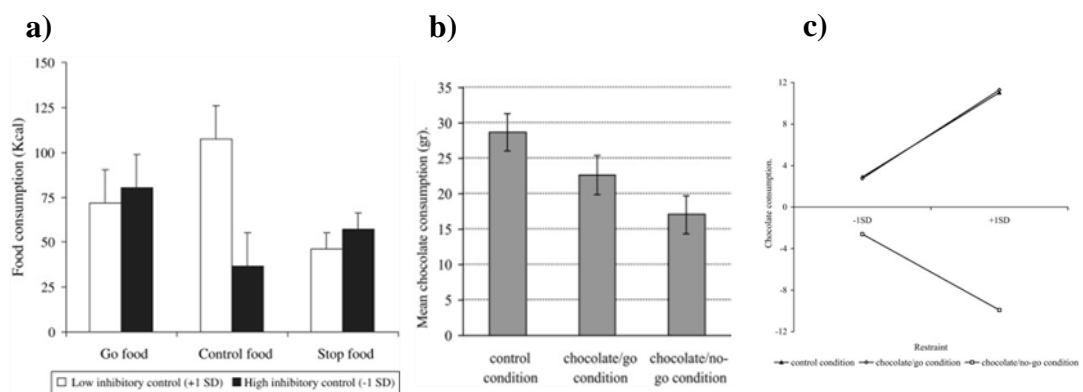


Figure 1.6. a) Results from Houben (2011) showing an effect of inhibitory control training on food consumption. The results reveal an effect of baseline inhibition on consumption of the control food, which was inconsistently paired with stopping, showing reduced consumption for those with high inhibitory control abilities. Consistently pairing a food with responding (go food) appeared to increase consumption in this group to the same level as those who were low in baseline inhibitory control, whereas pairing a food with stopping decreased food consumption in those with poor inhibitory control to the same level as those who scored highly on this measure. b), c) The results of Houben and Jansen (2011) showing

effects of inhibition (100% stop), control (50% stop) and impulsivity (0% stop) training on food consumption; **c)** shows the results after controlling for group differences in dietary restraint. Food consumption increased as a function of restraint in both the control and impulsivity groups but decreased as a function of restraint in the inhibition group.

In a similar study with a between-subjects design, Houben and Jansen (2011) split trait chocolate lovers into three groups to perform a GNG task in which they had to always inhibit their responses to images of chocolate (no-go group), always respond (go group) or inhibit half of their responses to chocolate (control group). They found that those in the control group consumed the most chocolate in a bogus taste test, followed by the go group and then the no-go group who consumed significantly fewer calories than those in the control group (see Figure 1.6b.). However, when controlling for differences in dietary restraint (using the Restraint Scale; Herman & Polivy, 1980) across the three groups, they found that consumption increased as a function of restraint in the go and control groups but decreased as a function of restraint in the no-go group. As dietary restraint is typically associated with an increased motivation towards food (e.g. Herman & Mack, 1975; Hofmann, Rauch & Gawronski, 2007; Spencer & Fremouw, 1979; Veenstra & de Jong, 2010; see section 1.4. for a full discussion of dietary restraint), these results suggest that training motor control in response to food stimuli might help restrained eaters restrict their calorie intake when presented with palatable food (see Figure 1.6c.).

Lawrence *et al.* (under review; Study 2) also found a moderating role of dietary restraint (using the restrained eating scale of the Dutch Eating Behaviour Questionnaire; DEBQRE; van Strien, Frijters, Bergers & Defares, 1986a) for the effect of response inhibition training on food consumption using a modified version of the SST. In this task participants responded to the location of palatable food images and on a subset of trials a visual signal was presented after a variable delay. When this signal was presented, participants in the stop group were required to inhibit their response, participants in the double-response group were required to make an additional response and a third 'ignore' group were instructed to ignore the signals and just made the location responses throughout. The stop signals presented

in this task were differentially mapped onto different foods so that there was one food that was frequently associated with a signal (signal food) and one food that was infrequently associated with a signal (go food). Following training participants were presented with a bogus taste test with both the signal and go foods. The results showed that restrained eaters in the stop group consumed significantly less of the signal food than those in the double-response group; consumption in the ignore group fell between the stop and double-response groups but did not significantly differ from either group. For the unrestrained eaters, however, there were no statistically significant differences between groups. This finding was in contrast to the result of Study 1, which showed a significant main effect of training condition but no moderating role of dietary restraint. It is possible that this difference can be explained by the increased consistency of food-inhibition associations in Study 1 compared to Study 2 – in Study 1 food images were paired with stopping on 87.5% of trials, whereas in Study 2 only 50% of food images were paired with a stop signal. If participants were more likely to learn this association in Study 1 then a greater main effect may be expected. However, previous studies with 100% consistency between food and inhibition have still found a moderating role of dietary restraint (Houben & Jansen, 2011; Veling *et al.*, 2011). For example, Veling *et al.* (2011; study 2) found that chronic dieters (high scorers on the Concern for Dieting subscale of the RS) who were trained to consistently inhibit their responses to images of sweets, compared to those who consistently responded, consumed significantly fewer of those sweets in the home environment over a one day period. These results are particularly encouraging as they demonstrate that a short training task (a few minutes) can influence food consumption over an extended time period in a natural context.

These authors have also investigated whether inhibition training can influence food choice behaviour (Veling *et al.*, 2013a). Participants performed a food-related GNG task in which four unhealthy foods were either always paired with a stop-signal (no-go group) or always paired with a response signal (go group). They were then asked to choose eight foods from an array of sixteen healthy and unhealthy snacks that included the four unhealthy foods from the training task. In their first study, investigating the moderating effect of appetite, their results showed that, for

participants in the go group, a high appetite (they participated in the study before lunch) increased the number of the four palatable foods chosen relative to those with a low appetite (they participated after lunch). This pattern was reversed and marginally significant for those in the no-go group who selected fewer of these foods when they had a high, compared to a low, appetite. Those in the no-go, high appetite condition also selected significantly fewer of these items compared to those in the go, high appetite condition (see also Veling, Aarts & Stroebe, 2013b; see Figure 1.7a.). For their second study, in which they measured the consumption frequency of these foods as a potential moderator, they replicated the effect of inhibition relative to go training on reduced unhealthy food choices for those who reported a high frequency of consumption (see Figure 1.7b.). Moreover, there was a significant positive correlation between consumption frequency and unhealthy snack choices in the go group but not in the inhibition group. In addition to the reduction of unhealthy food choices with stop training, there was also an increase in the number of healthy foods selected when appetite and frequency of consumption were high. These results suggest therefore that food-related inhibition training may enable individuals to make healthier food choices; although, as participants made a forced number of choices it is unclear whether this is due to a voluntary increase in healthy food choices or due to the decreased selection of unhealthy foods. These findings are particularly interesting as they demonstrate that training inhibitory control may not simply result in the reduction of a behaviour, such as food consumption, but may also modify behaviour in favour of more healthy options, potentially by engaging executive decision-making processes.

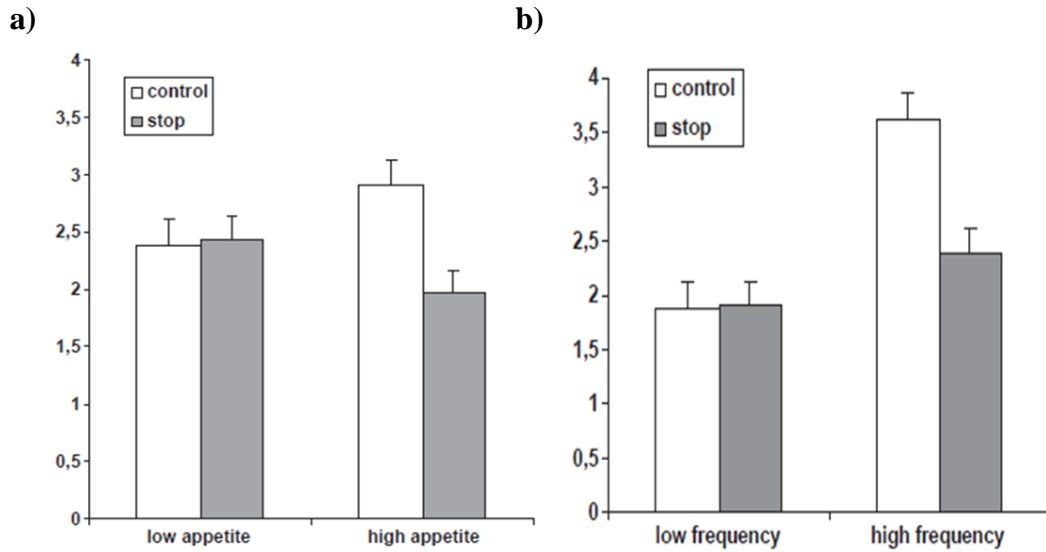


Figure 1.7. Results from Veling *et al.* (2013a) showing the effect of inhibition training on food choice. Inhibition training reduced the number of unhealthy snacks selected when **a)** appetite and **b)** frequency of consumption were high.

Although inhibition training may have the potential to engage top-down decision-making processes, it is still considered to be a bottom-up intervention (van Koningsbruggen *et al.*, 2013a). In their review, Friese *et al.* (2011) discuss behavioural health interventions in terms of a dual-process model in which these interventions target either the strong impulsive desires directly or the ability of the reflective system to control these desires (see Figure 1.1). It may be possible therefore to combine these two approaches to tackle the same maladaptive behaviour from both angles. This was the reasoning behind the study of van Koningsbruggen *et al.* (2013a) who examined whether inhibition training and implementation intentions could have an additive effect on selected portion size. Participants were assigned to one of four groups in which they received either food-related inhibition or control training and were instructed to form implementation intentions that were either diet-related or non-diet-related. Their results revealed that both interventions had a significant effect of reducing selected portion size compared to the double control group; however, there was no additive effect of combining these two interventions on behaviour.

The same design was employed in a similar internet-based study in which they looked at the effect of inhibition training and implementation intentions on weight change (Veling *et al.*, 2014). Participants were required to do the training task and make implementation intentions four times over a four week period, and were weighed at the beginning and end of the study in the lab. In the first session participants also answered several questionnaires, including questions on the strength of their dieting goal (other measures included questions regarding dietary restraint, perceived self-regulatory success, intentions to eat healthily, dieting importance, exercise behaviour, when they last ate/ drank and a series of demographic questions; these variables were analysed to make sure that there were no differences between groups). Their results replicated those of their previous study and revealed that both interventions facilitated weight loss but did not have an additive effect. However, when considering potential moderators they found that diet-related implementation intentions were particularly effective among those with a strong dieting goal, whereas food-related inhibition training influenced weight change independently of goal strength, and facilitated weight loss most effectively for those with a high BMI. The authors argued that implementation intentions may be a suitable aid for facilitating weight loss in dieters as it helps to remind them of their dieting goal (Stroebe, van Koningsbruggen, Papies & Aarts, 2013), whereas inhibition training may be more effective for those with high food-related impulsivity. Again these results are encouraging for response inhibition training as they show an effect on actual weight loss.

1.3.2. Response Inhibition Training: Potential Mechanisms

As well as investigating potential moderators of this effect, understanding the underlying mechanisms is also essential for furthering our knowledge and developing the most effective training paradigms. One possible explanation is an effect of inhibition training on increased general self-control. For example, there is evidence to suggest the existence of a global inhibition mechanism; research has shown that the suppression of manual responses, oculomotor responses and speech can reduce motor evoked potentials (MEPs) in muscles that are irrelevant to the task (Badry *et al.*, 2009; Cai, Oldenkamp & Aron, 2012; Majid, Cai, George, Verbruggen

& Aron, 2012; Wessel, Reynoso & Aron, 2013). Majid *et al.* (2012) argued that motor suppression can be more selective when required; however, when speed is stressed and there is no cost to behaviour, a global suppression mechanism can be recruited (see also Giesen & Rothermund, 2013). Furthermore, it has been argued that this global inhibition mechanism operates across different domains, such that motor inhibition may also ‘spillover’ into affective or cognitive domains (Berkman, Burklund & Lieberman, 2009; Berkman, Graham & Fisher, 2012; Kiss, Raymond, Westoby, Nobre & Eimer, 2008; Spunt, Lieberman, Cohen & Eisenberger, 2012; Tabibnia *et al.*, 2011; see Figure 1.8). Studies have shown that motor inhibition is correlated with affect regulation and reduced amygdala activity – findings which were also associated with grey matter intensity and activity in prefrontal regions believed to be responsible for inhibitory control (Berkman *et al.*, 2009; Tabibnia *et al.*, 2011).

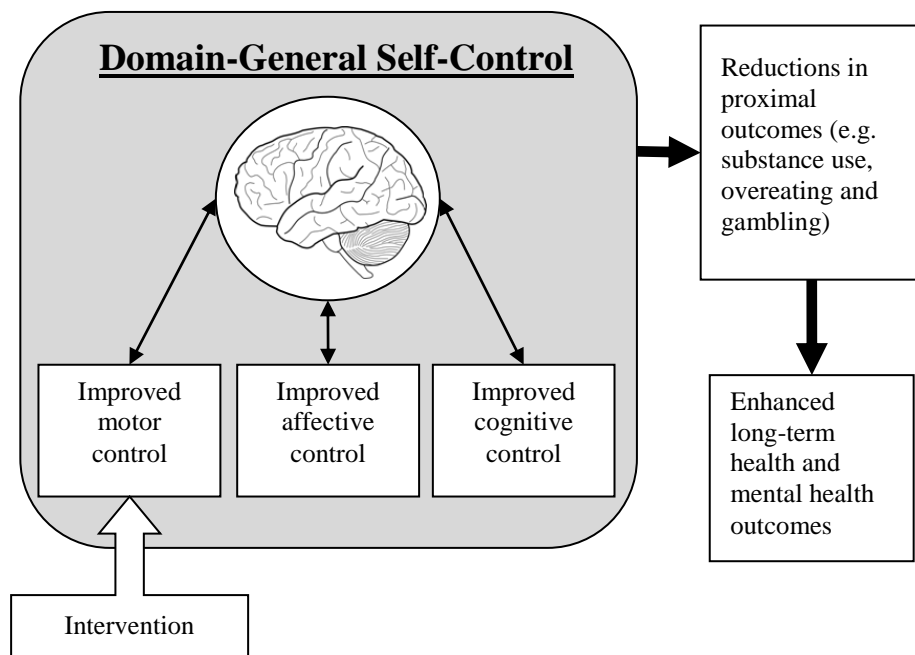


Figure 1.8. A model of domain general self-control. Intervention targeted towards improving motor inhibition will transfer to improvements in affective and cognitive domains; and these improvements will subsequently improve both proximal and long-term health outcomes (adapted from Berkman *et al.*, 2012).

Moreover, activity in the prefrontal cortex has been associated with inhibitory control across various modalities including manual and oculomotor tasks, as well as the inhibition of speech, thought and emotion (Aron, Fletcher, Bullmore, Sahakian & Robbins, 2003; Aron *et al.*, 2004, 2014; Berkman *et al.*, 2009; Casey *et al.*, 1997; Chikazoe, Konishi, Asari, Jimura & Miyashita, 2007; de Zubicaray, Andrew, Zelaya, Williams & Dumanoir, 2000; Dillon & Pizzagalli, 2007; Garavan, Ross & Stein, 1999; Lenartowicz, Verbruggen, Logan & Poldrack, 2011; Leung & Cai, 2007; Li, Huang, Constable & Sinha, 2006; Liddle *et al.*, 2001; MacDonald *et al.*, 2000; Rubia *et al.*, 2001; Swick, Ashley & Turken, 2008; Tabibnia *et al.*, 2011; Watanabe *et al.*, 2002; Xue, Aron & Poldrack, 2008; Zheng *et al.*, 2008). Furthermore, this region has also been linked to self-control across various behavioural domains including gambling, overeating and substance use (Appelhans, 2009; Batterink *et al.*, 2010; Behan, Stone & Garavan, 2014; Camprodon *et al.*, 2007; Del Parigi *et al.*, 2007; Goldstein & Volkow, 2002, 2011; Hare, Camerer & Rangel, 2009; Hare, Malmaud & Rangel, 2011; Knoch *et al.*, 2006; Le *et al.*, 2006, 2007; Lopez *et al.*, 2014). These findings suggest that the prefrontal cortex may be responsible for general self-control; although, it should be noted that this region is not exclusively involved in inhibitory control but is also associated with a range of other cognitive processes such as working memory, attention, response selection, performance monitoring and error detection (Criaud & Boulinguez, 2013; Erika-Florence, Leech & Hampshire, 2014; Hampshire, Chamberlain, Monti, Duncan & Owen, 2010; Mostofsky & Simmonds, 2008; Mostofsky *et al.*, 2003; Ridderinkhof, van den Wildenberg, Segalowitz & Carter, 2004; Rubia, Smith, Brammer & Taylor, 2003; Sharp *et al.*, 2010; Simmonds, Pekar & Mostofsky, 2008).

Verbruggen *et al.* (2012; Studies 2 and 3) have provided some evidence for the idea that inhibition training may influence behaviour by activating a general self-control mechanism. In these studies participants were trained on a SST with arbitrary shape stimuli and their risk taking was measured on a subsequent gambling task. The results showed that participants who had to inhibit their responses during training were significantly more risk averse on the gambling task, compared to participants who had to perform a double-response task or those who received no training (Study

2 only). These results indicate that stopping simple motor responses towards arbitrary stimuli may influence executive processes such as monetary decision-making. A recent series of studies have supported these findings showing that the effect of motor control on gambling generalises across different populations and tasks (Stevens *et al.*, under review). Moreover, these studies also demonstrated that these effects were due to the inhibition of a motor response and not auxiliary processes such as increased arousal or information sampling. However, the authors did not find any evidence to suggest that the effects were due to increased general motor cautiousness as manipulating the speed/accuracy trade off in a secondary task had no influence on gambling preferences.

In a similar study investigating the effect of general inhibition training on food consumption, Guerrieri, Nederkoorn and Jansen (2012) gave participants either a critical reading task (control group) or a standard SST in which the number of stop (inhibition group) or response trials (impulsivity group) was gradually increased. Their results showed that participants in the impulsivity group consumed significantly more calories in a bogus taste test than both the control and inhibition groups, whereas the latter two groups did not differ from one another. Although these findings suggest that disinhibition towards food may be more easily learned than inhibition (a position which is supported by models of associative learning; see Lotz, Uengoer, Koenig, Pearce & Lachnit, 2012), the authors recognise that interpretations regarding the potential influence of inhibitory control training on food consumption is unclear. With previous findings showing an effect of food-related inhibition training on food intake and food choices, it is quite possible that for response inhibition training to be effective in a food context the act of stopping must be targeted towards food stimuli. Lawrence *et al.* (under review) replicated previous findings showing a significant effect of food-related inhibition training on reduced food intake; however, when the food stimuli presented during the training task were replaced with images of household objects, there was no statistically significant difference in intake between the inhibition and control groups. A similar finding has also been reported with alcohol-related inhibition training; Jones and Field (2013) found a significant effect of inhibition training on reduced ad-libitum alcohol consumption, but only when inhibition was paired alcohol images with and not when

participants had to stop to neutral pictures (but see Jones, Guerrieri, Fernie, Cole, Goudie & Field, 2011).

These results imply that inhibitory control training may only be effective for reducing food consumption when response inhibition is specific to food images. This suggests therefore that the underlying mechanism explaining these effects may rely on specific stimulus-stop associations rather than a general inhibition mechanism. Research has shown that consistently pairing a stimulus with stopping can result in that stimulus being 'tagged' with an inhibitory signal (Chiu, Aron & Verbruggen, 2012; Lenartowicz *et al.*, 2011; Verbruggen & Logan, 2008). For example, studies have shown increased slowing and reduced MEPs for stimuli previously paired with stopping (Chiu *et al.*, 2012; Verbruggen & Logan, 2008). Veling *et al.* (2011; Study 1) investigated whether a GNG task led to the automatic inhibition of responses by asking participants to respond to action probes that occasionally and immediately followed stimulus-cue pairs in which food and non-food images were paired with either go or no-go cues. Their results revealed a main effect of cue type with slower responses for no-go cues compared to go cues. However, this main effect was qualified by a significant three-way interaction in which chronic dieters were significantly slower to respond to an action probe when it followed a food/no-go pair compared to a non-food/no-go pair. They argued that the specificity of this finding, for chronic dieters and food images, was due to the dyadic relationship between automatic inhibition and the initial approach response (Nakata *et al.*, 2006). In other words, because chronic dieters have such a strong approach response towards palatable foods (Veenstra & de Jong, 2010) it was possible to detect a reduction in this impulse as a result of inhibition training.

Another potential mechanism that may underlie the effect of inhibitory control training on behaviour is stimulus devaluation. In 2008, Veling, Holland and Knippenberg proposed the Behaviour Stimulus Interaction (BSI) theory. They argued that when a conflict arises between wanting to approach a positive stimulus and having to inhibit that response due to situational demands, the conflict is reduced by devaluing the stimulus. By reducing our desire for an object we can promote self-regulation and redirect our attention towards other goals. In support of this theory,

Veling *et al.* demonstrated that positively valenced images that were paired with no-go signals during a GNG task were subsequently rated as less attractive than images paired with go signals (see also Fenske & Raymond, 2006; Ferrey, Frischen & Fenske, 2012). A similar finding has also been demonstrated using a SST (Wessel, O'Doherty, Berkebile, Linderman & Aron, in press). In this study there were three phases: firstly, there was an implicit learning phase in which eight shapes were paired with four different monetary amounts; secondly, there was a treatment phase in which these shapes were either paired with a quick motor response or the inhibition of this response; and finally, there was an auction phase in which participants valued the stimuli according to how much they were willing to pay for them. In support of the BSI theory, Wessel *et al.* (in press) found that bidding on the auction task was significantly lower for the shapes paired with inhibition compared to those paired with a response. Furthermore, they argued that this effect was specifically due to the inhibition of a response as they found no effects due to the aversiveness, effort, conflict or salience associated with the stop signal.

According to the BSI theory (Veling *et al.*, 2008), stimuli must be initially perceived as positive in order to create a conflict with inhibition and to be devalued. Veling *et al.*'s (2008) studies provided some support for this position showing an effect for positively but not for negatively valenced stimuli. In Wessel *et al.*'s (in press) study, the initial value of stimuli was controlled with the implicit learning task and they found a main effect of value and inhibition on auction bids but no statistically significant interaction between the two. This suggests, therefore, that the initial value does not moderate the effect of inhibition on devaluation – although, it could be argued that all monetary amounts are positive. Nevertheless, other studies have also shown that negatively valenced stimuli can be devalued with inhibition, thus indicating that inhibiting responses towards a stimulus results in a more negative value for that stimulus rather than simply having a neutralising effect (Frischen, Ferrey, Burt, Pistchik & Fenske, 2012).

Veling *et al.* (2013b) investigated whether the devaluation of stimuli mediated the effect of food-related response inhibition training on food choice behaviour. After performing a food-related GNG task in which different foods were either associated

with stopping or going, participants rated the attractiveness of these food images before having to select three of seven palatable foods. Their findings replicated previous results showing that for participants in the high-appetite condition, no-go foods were selected less frequently than go foods. These participants also evaluated no-go foods less positively than go foods, and these evaluations were shown to have a direct effect on food choice. Further analysis revealed that food evaluations mediated the effect of appetite on food choice for no-go foods, whereas evaluations of go foods had no effect on go-food choice. These results suggest that training participants to inhibit their responses to palatable foods may influence food choice by devaluing the explicitly rated appetitive properties of the food. Interestingly, there is also evidence to suggest that this process can operate outside of conscious awareness. Veling and Aarts (2009) found that when images of water were subliminally presented to thirsty participants during a GNG task, participants who had to inhibit their responses rated the perceived size of water objects as significantly smaller than those in the go group, thus suggesting a reduced reward value.

Changes in automatic, or implicit, attitudes following inhibition training have also been demonstrated using the implicit association test (IAT; Greenwald, McGhee & Schwartz, 1998; Houben *et al.*, 2011a; Houben *et al.*, 2012a). The IAT is a categorisation task which is designed to measure the associative strength between two concepts (such as ‘male’ and ‘science’ versus ‘female’ and ‘creative’; see Figure 1.9.) without engaging conscious awareness. Participants are simply required to categorise stimuli according to different concepts as quickly and as accurately as they can. The logic is that if an individual holds a certain attitude regarding two concepts, they will be faster to categorise words using a common response key when the concepts are congruent (‘male’ and ‘science’) compared to when they are incongruent (‘female’ and ‘science’). The advantage of the IAT is that it is believed to be less susceptible to demand characteristics compared to explicit attitude measures (Greenwald *et al.*, 1998; but see Fiedler & Bluemke, 2005; Steffens, 2004). Implicit attitudes may also be a better predictor of behaviour than explicit attitudes when cognitive and self-regulatory resources are low (Friese *et al.*, 2008). Using this task, Houben *et al.* (2011a) measured implicit attitudes towards beer in a sample of

heavy drinking students following a beer-related GNG task. They found that the reduction in weekly alcohol consumption in the beer/no-go group was accompanied by a significant increase in negative implicit attitudes towards beer. The implicit attitudes towards beer in this study were negative initially and increased in this direction, a finding which is consistent with previous studies showing that inhibition can increase the negative valence of both positive and negative stimuli (Frischen *et al.*, 2012).

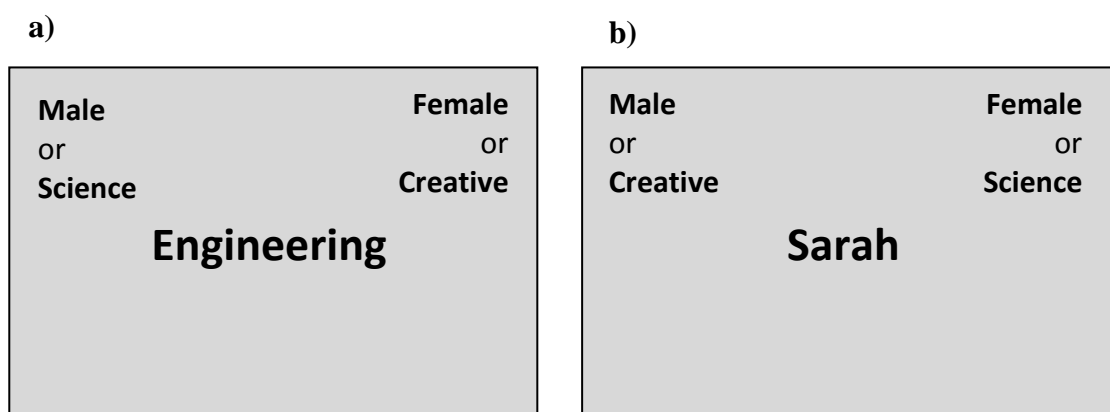


Figure 1.9. The implicit association test (IAT; Greenwald *et al.*, 1998). Participants categorise the central words as quickly as possible according to the attribute labels presented in the top-left and top-right corners of the screen. In this example, if a participant holds a stereotyped attitude that men are scientific and women are creative, they would respond faster in the congruent trials (**a**), when male names are paired with scientific words and female names are paired with creative words, than on the incongruent trials (**b**) when these pairings are reversed. The difference in reaction times provides an indication of the direction and strength of their implicit attitude towards gender stereotypes.

In a second study, Houben *et al.* (2012a) further investigated the potential underlying mechanisms for the effect of inhibition training on self-reported alcohol consumption by measuring changes in implicit attitudes (IAT), approach-avoidance action tendencies (stimulus response compatibility task; Krieglmeier, Deutsch, De Houwer & De Raedt, 2010) and response inhibition (SST) before and after the training procedure. They found no effects of training on either the approach-avoidance or

response inhibition tasks but they did replicate the effects of no-go training on implicit attitudes and weekly alcohol consumption. In this study, implicit attitudes towards beer remained positive following inhibition training, but were significantly less so. Considering all three potential mechanisms in a regression model, Houben *et al.* demonstrated that only changes in IAT scores significantly predicted changes in alcohol use and also mediated the effect of inhibition training on this measure. Bowley *et al.* (2013), however, failed to replicate these findings; while they did show an effect of alcohol-related no-go training on reduced beer consumption during a taste-test, they did not find any effect of training on implicit attitudes. Conversely, they found some evidence to indicate that inhibition training may have resulted in an increased avoidance association. However, there was no statistically significant correlation between post-training avoidance associations and beer consumption but there was a trend towards a positive correlation between implicit attitudes and consumption. Together these results suggest that inhibition training may reduce consumption behaviour by devaluing the stimuli associated with inhibition, although, more research is required to replicate these findings.

One possible explanation for this devaluation is that inhibition activates an approach-avoidance system (McLaren & Verbruggen, submitted; Verbruggen, Best, Bowditch, Stevens & McLaren, in press). Associative learning theories suggest that conditioned inhibitors, which predict the absence of the unconditioned stimulus (US), may excite a 'No-US' representation which in turn excites an appetitive or aversive centre according to whether the US is initially considered to be aversive or appetitive, respectively (see Figure 1.10.). However, although this model can explain the effect of inhibition on stimulus devaluation for positive stimuli, it does not explain findings showing an effect of inhibition on decreased evaluations for negative stimuli. Rather, the model predicts that inhibiting responses towards a negative stimulus should activate the appetitive centre, which would presumably result in more positive evaluations or neutralisation. Another possibility is the idea that approach-good and avoid-bad associations may be hard-wired – an idea which is consistent with theories of embodied cognition (Cacioppo, Priester & Berntson, 1993; Chen & Bargh, 1999; Chiu, Cools & Aron, 2014; Guitart-Masip, Huys, Fuentemilla, Dayan, Duzel & Dolan, 2012; Hung & Labroo, 2011; Phaf & Rotteveel, 2012; Price, Peterson &

Harmon-Jones, 2012; Reimann *et al.*, 2012). For example, Guitart-Masip *et al.* (2012) demonstrated that participants were better at learning to execute a response in anticipation of reward and to withhold a response in anticipation of punishment than when these contingencies were reversed. An inherent avoidance-bad bias would suggest that the act of inhibition itself excites the aversive centre, regardless of the stimulus' valence (see Figure 1.10.). This idea can explain the effect of inhibition on stimulus devaluation for both positively and negatively valenced stimuli, and may also explain both stimulus-specific and domain general increased 'motor cautiousness' (McLaren & Verbruggen, submitted; Verbruggen *et al.*, in press).

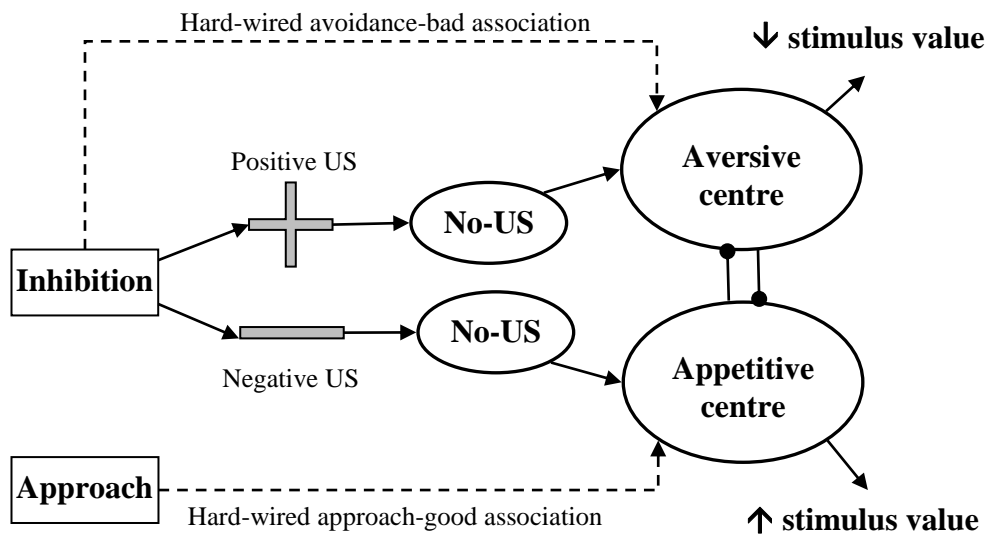


Figure 1.10. Schematic diagram showing how inhibition may activate the appetitive-aversive system. In one model, inhibition of a response towards a positive or negative unconditioned stimulus is believed to excite a 'No-US' centre and subsequently activate either the aversive or appetitive centres, respectively. Excitation of the aversive centre may explain how inhibition results in the devaluation of positive stimuli (Veling *et al.*, 2008; Wessel *et al.*, in press). However, this model predicts increased value for inhibition towards negative stimuli and therefore cannot explain findings demonstrating that inhibition causes devaluation of negative stimuli also (Frischen *et al.*, 2012). A second model (dashed lines) argues that avoidance-bad and approach-good associations are hard-wired and that inhibition will result in the excitation of the aversive centre regardless of stimulus' valence (see McLaren & Verbruggen, submitted).

In summary, studies investigating the potential of response inhibition training as a therapeutic tool for overeating and obesity have so far produced promising results. These studies have shown effects of inhibition training on a range of food-related behaviours, including food consumption, both in and out of the lab, food choices and weight change. Moreover, these effects have been demonstrated with a training task that requires very little time and effort, and can be distributed via the internet with minimal inconvenience and cost. The consideration of potential mediators and moderators of these effects is also essential for further development of these tasks and for targeting them effectively at the most appropriate individuals. However, at present, there are many proposed moderators which are not fully understood and require replication before the utility of this training task can be advocated as a clinical tool. One population who may be particularly suitable for response inhibition training are restrained eaters. Dietary restraint has already been shown to have a moderating role in the effect of inhibition training on food consumption (Houben & Jansen, 2011; Lawrence *et al.*, under review; Veling *et al.*, 2011), and these individuals have also been shown to have low levels of inhibitory control (Nederkoorn, van Eijs & Jansen, 2004; but see Meule, Lukito, Vögele & Kübler, 2011a) and strong preferences for (Hoefling & Strack, 2008; Houben *et al.*, 2010a, 2012b) and approach tendencies towards food (Veenstra & de Jong, 2010). As the experiments presented in this thesis focus on dietary restraint as a key individual difference in this type of research, the next section discusses the concept of dietary restraint in more detail, and aims to outline why, perhaps counterintuitively, these individuals actually experience high levels of dietary disinhibition.

1.4. Dietary Restraint: A Cautionary Tale

Dietary restraint can be defined as the tendency to deliberately restrict food intake with the aim of losing weight or preventing weight gain. To achieve personal standards, restrained eaters ignore internal satiety signals, resulting in a cognitive rather than a physiological control of food intake (Herman & Mack, 1975; Polivy & Herman, 1985). Originally, the theory of restraint was developed to address the difference in eating behaviour between healthy-weight and obese individuals. This

theory was based on the ideas of Schachter (1971), who proposed that obesity was the result of being driven by external, rather than internal cues. The restraint theory therefore hypothesised that obese individuals would be more restrained than normal weight individuals. Although there is evidence to suggest an association between restraint scores and BMI (Guerrieri *et al.*, 2009; Laessle, Tuschl, Kotthaus & Pirke, 1989; Schur, Heckbert & Goldberg, 2010; Veenstra & de Jong, 2010), the hypothesis that restraint underlies differences in obese and healthy-weight individuals remains largely unsupported (see Ruderman, 1986).

A second hypothesis of the restraint theory, the ‘disinhibition hypothesis’, has proved to be more useful. This hypothesis relates to the observation that restrained eaters are prone to over-indulging when circumstances violate self-control. This paradoxical behaviour is illustrated using a ‘preloading’ paradigm whereby participants are given a quantity of calorific and palatable food (a preload) prior to a bogus taste test. Herman and Mack (1975) demonstrated a significant interaction between preload and restraint in predicting calorie consumption. Their results revealed that while unrestrained eaters reduced their intake as a result of the preload (demonstrating regulatory behaviour), restrained eaters actually consumed more calories. This effect of eating more following a preload has been termed ‘counterregulation’ and was believed to be the result of an all-or-nothing approach to dieting in restrained eaters (Herman & Mack, 1975). Evidence suggests that when restrained eaters are ‘in control’ they eat according to their self-imposed standards; however, they are vulnerable to overeating when their self-control is undermined (Hofmann *et al.*, 2007, Spencer & Fremouw, 1979; see Ruderman, 1986 for an overview).

1.4.1. Dietary Restraint and Increased Food Motivation

More recent studies, however, have shown that the consumption of a preload is not always necessary to induce overeating in restrained individuals. In support of the original theory that restrained eaters are highly sensitive to external food cues (Herman & Mack, 1975), it has been shown that mere exposure to palatable food can cause those high in dietary restraint to eat more than their unrestrained counterparts

(Fedoroff, Polivy & Herman, 1997, 2003; Jansen & van den Hout, 1991; Rogers & Hill, 1989; Shimizu & Wansink, 2011). Furthermore, restrained eaters have also been shown to display a greater salivary response in the presence of palatable food (Brunstrom, Yates & Witcomb, 2004; Klajner, Herman, Polivy & Chhabra, 1981; Tepper, 1992) and an attentional bias towards food-related stimuli (Francis, Stewart & Hounsell, 1997; Hollitt *et al.*, 2010; Papies, Stroebe & Aarts, 2008; Stewart & Samoluk, 1997).

As well as showing increased reactivity to food and food-related stimuli, restrained eaters have also been found to demonstrate strong implicit preferences for fattening food (Hoefling & Strack, 2008; Houben, Roefs & Jansen, 2010a, 2012c). Using a task designed to measure implicit associations, Hoefling and Strack (2008) found that compared to unrestrained eaters, restrained eaters showed a stronger positive evaluation of high calorie, but not low calorie food. However, similar studies have failed to find any evidence of a relationship between restraint status and implicit attitudes towards fattening foods (Roefs, Herman, MacLeod, Smulders & Jansen, 2005; Veenstra & de Jong, 2010), or have even found a negative relationship (Papies, Stroebe & Aarts, 2009). One limitation of these studies is that they have all used tasks that directly compared positive and negative affect. Although we may expect restrained individuals to have positive attitudes towards fattening foods, it is also likely that the reverse would be true since these are the foods they try to avoid. Additionally, these studies have compared high and low calorie foods in a relative manner, which may influence results by inadvertently reminding restrained eaters of their dieting goal (Stroebe, Mensink, Aarts, Schut & Kruglanski, 2008; Stroebe *et al.*, 2013).

These issues were addressed by Houben *et al.* (2010a) who asked participants to perform two unipolar versions of the IAT (Greenwald *et al.*, 1998) in Study 1, and two unipolar Single-Category IATs (SC-IAT; Karpinsky & Steinman, 2006) in Study 2. Rather than comparing positive and negative attributes, the unipolar version of the IAT compares either positive or negative attributes with neutral attributes in two separate tasks. The SC-IAT compares these affective associations with just one target category allowing the user to examine attitudes towards that category in

isolation. Houben *et al.* (2010a) found that there was no difference in implicit preferences for low or high calorie foods between restrained and unrestrained eaters when associations were relative to one another (Study 1). Both groups associated high calorie food with negative affect only. However, when snack foods were measured independently (Study 2), there was no evidence of an association with negative affect in either restrained or unrestrained eaters. On the contrary, the results showed that participants in both groups associated high calorie foods more strongly with positive than neutral words, suggesting a positive implicit attitude. Moreover, this association was significantly stronger in restrained compared to unrestrained eaters. This was later replicated by Houben *et al.* (2012c), who further demonstrated that this preference in restrained eaters was due to the palatability of the food rather than the energy density. These results suggest that individuals who score highly in dietary restraint are also likely to possess strong implicit preferences for tasty food.

1.4.2. Dietary Restraint and Poor Self-Control

Along with a hypersensitivity to external food cues and an implicit preference for fattening food, there is also evidence to suggest that restrained eaters are characterised by poor self-control and increased impulsivity (Krahn, Kurth, Gomberg & Drewnowski, 2005; Jansen, Klaver, Merckelbach & van den Hout, 1989; Stewart, Angelopoulos, Baker & Boland, 2000; but see Meule *et al.*, 2011a). Nederkoorn *et al.* (2004) compared inhibitory control in restrained and unrestrained participants using the SST. They found that restrained eaters took significantly longer to inhibit their basic motor responses compared to unrestrained individuals. Furthermore, evidence has shown that impulsivity may moderate the relationship between restraint and food intake. Using self-report measures, van Koningsbruggen, Stroebe and Aarts (2013b) found that trait impulsivity was negatively correlated with perceived dietary success in restrained eaters. This finding has also been demonstrated behaviourally; Jansen, Nederkoorn, van Baak, Keirse, Guerrieri and Havermans (2009) showed that restrained eaters only overate when they also displayed poor response inhibition on the SST. Although these findings are correlational, it is easy to appreciate how a limited ability to control one's actions might play a role in disinhibited eating, especially when coupled with a strong

automatic approach response to food (Veenstra & de Jong, 2010) and a belief that one has little control over food intake (Rotenberg & Flood, 2000; Rotenberg *et al.*, 2005).

Guerrieri *et al.* (2009, Study 1) investigated the causal nature of this relationship by priming participants with either impulsivity or inhibition using a memory task. Their results showed a significant main effect for both priming condition and restraint status; those primed with impulsivity ate significantly more calories than those primed with inhibition, and restrained eaters ate significantly more than unrestrained eaters. Furthermore, these results were qualified by a marginal interaction which revealed that the effect of priming was only significant for participants high in restraint. These results coincide with previous conclusions regarding the sensitivity of restrained eaters to external cues (Brunstrom *et al.*, 2004; Fedoroff *et al.*, 1997, 2003; Jansen & van den Hout, 1991; Klajner *et al.*, 1981; Rogers & Hill, 1989; Schachter, 1971; Shimizu & Wansink, 2011; Tepper, 1992). However, without a control condition it is unclear whether the impulsivity condition caused restrained eaters to eat more or whether the inhibition condition caused them to eat less. This latter suggestion is reasonable given that restrained eaters have been shown to eat less when they are explicitly reminded of their dieting goal (Anschutz, van Strien & Engels, 2008; Papiés & Hamstra, 2010). Moreover, as discussed earlier (see section 1.3.1.), restrained eaters have been shown to be particularly responsive to response inhibition training (Houben & Jansen, 2011; Lawrence *et al.*, under review, Study 2; Veling *et al.*, 2011, study 2).

1.4.3. Dietary Restraint Versus Dietary Disinhibition

While the concept of dietary restraint may be useful for predicting consumption behaviour and food attitudes, there are some conceptual and psychometric issues with dietary restraint that must be discussed (for a review see Heatherton, Herman, Polivy, King & McGree, 1988). As well as issues with incompleteness (Wardle, 1986) and inapplicability to obese samples (see Ruderman, 1983, 1986), the Restraint Scale (RS; Herman & Polivy, 1975), which was the original psychometric tool used in the development of restraint theory, has some limitations concerning criterion

confounding. Generally, use of the RS as a single scale has been discouraged; the scale has been shown to measure two distinct factors – ‘Concern for Dieting’ and ‘Weight Fluctuation’ – although, there is some disagreement over which items correspond to which subscale and which factor has more predictive validity (Blanchard & Frost, 1983; Drewnowski, Riskey & Desor, 1982; Herman & Mack, 1975; van Strien, Breteler & Ouwens, 2002). It has been argued that the correlation between restraint status and overweight is likely to be explained by scores on the weight fluctuation dimension (Drewnowski *et al.*, 1982; Ruderman, 1985; Stunkard & Messick, 1985), which may also overestimate restraint in obese samples (Ruderman, 1985, 1986). The concern for dieting dimension, on the other hand, may be more strongly associated with the goal of weight loss and a greater attentional and emotional association with food (Blanchard & Frost, 1983). This has led some researchers to only consider the concern for dieting subscale when trying to dissociate restrained and unrestrained eaters (e.g. Papiés *et al.*, 2008; van Koningsbruggen *et al.*, 2013b; Veling *et al.*, 2011). Moreover, some researchers have also argued that certain items in the RS (e.g. ‘Do you eat sensibly in front of others and splurge alone?’) measure disinhibited or opportunistic eating (Stice, Ozer & Kees, 1997; Stunkard & Messick, 1985; Wardle & Beales, 1987).

This led to the development of other restraint scales, such as those in the Dutch Eating Behaviour Questionnaire (DEBQRE; van Strien *et al.*, 1986a) and the Three Factor Eating Questionnaire (TFEQ-R; Stunkard & Messick, 1985), which did not include items concerning weight fluctuations or disinhibition. Laessle *et al.* (1989) looked at the construct validity of these three scales with reference to disordered and disinhibited eating, concerns with body image and self-reported calorie intake. Their findings revealed that the RS was positively associated with disinhibited eating and weight fluctuations, while the DEBQRE and TFEQ-R were negatively associated with calorie intake. Furthermore, preload studies have failed to find evidence for the disinhibition hypothesis when using either the DEBQRE (Wardle & Beales, 1987) or the TFEQ-R (Lowe & Kleifield, 1988). In fact, Westenhoefer, Broeckmann, Münch and Pudiel (1994) demonstrated that counterregulation only occurred for participants who scored highly on both the restraint and disinhibition scales of the TFEQ. In addition, participants with a high score on the disinhibition scale consumed more

calories than those with a low score, irrespective of restraint. It is possible therefore that previous results showing an association between scores on the Restraint Scale and counterregulation are the result of a criterion confound with disinhibition. Previous research has shown that disinhibition is also associated with obesity (Bellisle, Clément, Le Barzic, Le Gall, Guy-Grand & Basdevant, 2004; Boschi, Iorio, Margiotta, D’Orsi & Falconi, 2001; Dykes, Brunner, Martikainen & Wardle, 2004; Provencher, Drapeau, Tremblay, Després & Lemieux, 2003), poor food choices (Contento, Zybert & Williams, 2005; Hetherington & Macdiarmid, 1993) and weight gain (Hays, Bathalon, McCrory, Roubenoff, Lipman & Roberts, 2002; Hays & Roberts, 2008).

Together these studies suggest that the TFEQ-R and DEBQRE may be measuring successful restraint whereas the RS measures a form of unsuccessful restraint which also reflects dietary disinhibition and weight fluctuations. An understanding of these differences is necessary for the interpretation of results and experimental design. For example, when planning a study, researchers interested in helping individuals to overcome their food-related disinhibition may benefit from selecting participants based on their RS scores, whereas others interested in how participants maintain a reduced body weight may use the DEBQRE or TFEQ-R. There are three published studies, to date, which have shown an effect of food-related response inhibition training on restrained eaters, however, these three studies have all used different measures of dietary restraint. Houben and Jansen (2011) used the RS, Veling *et al.* (2011) used the concern for dieting subscale of this measure (RSCD), and Lawrence *et al.* (under review) used the DEBQRE. The similarity of findings despite the differences between these measures reflects the fact that these measures are still highly correlated with one another (Allison, Kalinsky & Gorman, 1992; Laessle *et al.*, 1989; Wardle, 1986). Nevertheless, for standardising future research it is important to consider which of these measures may be most appropriate for preselecting individuals who are likely to benefit the most from inhibition training. Consistent with the differences discussed above, the research presented in this thesis used the RS; although, it is important to note that this scale may be more reflective of a disinhibited eating style, rather than what we would intuitively think of as a restrained eating style. In fact, the aim of the first study presented in this thesis was

to compare the RS, RSCD and DEBQ with regards to different measures of disinhibited eating. A synopsis of all the studies in this thesis is presented in the next section.

1.5. Synopsis

The primary aim of this thesis was to investigate the effectiveness of response inhibition training for reducing food consumption in restrained eaters. In four studies I explored whether a single session of food-related inhibition training could reduce the consumption of palatable foods, compared to training on a control task. Furthermore, I examined the role of different training protocols, stimulus-specific associations and underlying cognitive and neural mechanisms.

In the first study I compared three measures of dietary restraint. Restrained eating has previously been shown to moderate the effects of inhibition training on food consumption, however, different measures of this trait have been used. I therefore compared these measures and their relationships with disinhibited eating (specifically, I explored relationships with external eating, measures of food liking and craving and BMI) to examine which measure of restraint was the most suitable screening tool. All participants in my laboratory studies were selected on the basis that they scored highly on the Restraint Scale (Herman & Polivy, 1980); in Study 1 I found evidence to suggest that this measure was reflective of a disinhibited eating style and should therefore help to identify individuals who could benefit the most from self-control training.

In Study 2, restrained, chocolate cravers received a modified version of the stop-signal task in which they had to inhibit or make double-responses to images of chocolate. Following this training task they were presented with two unipolar, Single-Category Implicit Association Tests (SC-IATs) to measure implicit positive and negative attitudes towards chocolate. They were then presented with a bogus taste test. A Bayesian analysis provided evidence for the null hypothesis, showing no effect of inhibition training on food consumption. Furthermore, in contrast to

previous studies, I found no evidence for an effect of inhibition on stimulus devaluation in the SC-IATs.

In the third study I compared two different training protocols, the stop-signal task and the go/no-go task. During training participants responded to images of healthy and unhealthy foods, with the inhibition training targeted towards the unhealthy foods. Consumption was then measured with a snack buffet including healthy and unhealthy foods that were either previously associated with stopping or novel. Training on the go/no-go task resulted in a greater reduction in food consumption compared to stop-signal training. This effect of no-go training on food consumption was specific to the unhealthy foods; however, the effect also transferred to a novel, unhealthy food. An additional group who observed the training task without responding was also included to help determine whether these effects were due to decreased consumption in the inhibition group, or increased consumption in the control group. The results suggest that effects were driven by increased consumption in the control group.

In the final study I ran an online experiment to see whether go/no-go training with snack foods would have an effect on implicit and explicit ratings of these foods. Training was followed by either two SC-IATs to measure implicit positive and negative attitudes or a task in which participants explicitly rated the attractiveness, tastiness and their craving for snack foods. Again I compared foods that were presented during training with novel foods to explore the stimulus-specific nature of these effects. Contrary to expectations, there was some evidence to indicate that no-go training may increase implicit positive attitudes towards snack foods, particularly in restrained eaters. For explicit food ratings there was a statistical trend to suggest that go/no-go training may influence perceptions of the attractiveness of trained foods in restrained eaters. However, there were no effects for ratings of tastiness or desire to eat the foods. Generally, the results of this study did not support the hypothesis that effects of inhibition training on behaviour are due to the devaluation of trained stimuli.

An additional pilot study was also conducted to test the feasibility of a within-subjects design for combining inhibition training with prefrontal brain stimulation (presented in Appendix 11). The aim of this study was to see whether stimulation could augment the effect of training on food consumption. No-go training was paired with either active or sham bilateral stimulation of the dorsolateral prefrontal cortex (DLPFC) using anodal right/ cathodal left transcranial direct current stimulation (tDCS), over two counter-balanced sessions. Food intake was measured using a snack buffet and measures of state food craving and response inhibition were also recorded as potential mediators. Results were generally in the expected direction but indicated substantial complications with repeated sessions due to practice effects and food preferences. These limitations and possible solutions are discussed.

These studies contribute to a growing body of literature investigating whether training inhibitory control can reduce impulsive behaviours such as increased food intake. Together they have implications for standardising methodologies in this field of research based on participant characteristics and suitable training and control tasks.

Chapter 2. Study 1

Dietary Restraint and Disinhibited Eating: A Comparison of the Restraint Scale and the Restrained Eating Scale of Dutch Eating Behaviour Questionnaire

2.1. Introduction

Dietary restraint refers to the tendency to chronically limit food intake in order to lose weight or prevent weight gain. In today's 'obesogenic' environment many individuals are faced with the need to exert greater self-control over their food intake in order to maintain long-term dietary standards. Although this appears to be an adaptive behaviour, high dietary restraint is believed to be governed by increased cognitive control with a reduced reliance on physiological control (Herman & Mack, 1975; Polivy & Herman, 1985). Subsequently, as food intake becomes cognitively rather than physiologically determined¹, individuals become prone to overeating when their cognitive resources are undermined (Hofmann *et al.*, 2007; Lattimore & Maxwell, 2004; Polivy & Herman 1985; Ruderman, 1986). Furthermore, these lapses in self-control are typically associated with increased consumption of the 'forbidden' foods that restrained eaters try so hard to avoid (Boon, Stroebe, Schut & Ijntema, 2002; Knight & Boland, 1989). Paradoxically, therefore, high dietary restraint is strongly related to disinhibited eating – a term that refers to the inability to control intake despite intentions to do so (Goldstein, Forman, Meiran, Herbert, Juarascio, Butryn, 2014; Stunkard & Messick, 1985).

¹This reference to the difference between a cognitive versus physiological control of food intake does not mean to suggest that cognitive processes are not physiological, rather I mean to highlight how restrained eaters 'listen' to their mental rules for food consumption (for example, "I only ate two hours ago and do not need to eat again") rather than their internal satiety signals.

This disinhibited eating in restrained eaters has been demonstrated using the ‘preloading’ paradigm. Studies have shown that when restrained eaters consume a small amount of palatable food they are more likely to overconsume in a later taste test than unrestrained eaters (Herman & Mack, 1975; Spencer & Fremouw, 1979). It was thought that this ‘counterregulation’ of food intake was due to an all-or-nothing approach to dieting; however, this effect has also been shown when restrained eaters are merely exposed to the smell or thought of palatable food (Fedoroff *et al.*, 1997, 2003; Jansen & van den Hout, 1991; Rogers & Hill, 1989; Shimizu & Wansink, 2011), or when they perform a cognitively demanding task prior to the taste test (Lattimore & Maxwell, 2004; Ward & Mann, 2000). These results are consistent with an ego depletion model of self-regulation failure (Baumeister, 2003; Baumeister *et al.*, 1998; Muraven & Baumeister, 2000; Muraven *et al.*, 1998). According to this model, the act of dieting itself may deplete cognitive control resources, therefore leaving dieters more vulnerable to future violations of self-control (Guerrieri *et al.*, 2009; Kahan, Polivy & Herman, 2003; Vohs & Heatherton, 2000).

Support for the idea that restrained eaters are low in cognitive capacity also comes from evidence showing an association between high dietary restraint and impulsivity. Restrained eating has been linked to increased reports of sensation-seeking (Jansen *et al.*, 1989), alcohol use (Krahn *et al.*, 2005; Stewart *et al.*, 2000) and poor decision-making on a gambling task (Kuijjer, de Ridder, Ouwehand, Houx & van den Bos, 2008). Furthermore, Nederkoorn *et al.* (2004) showed that restrained eaters were significantly slower to inhibit their basic motor responses on a SST compared to unrestrained eaters (also see Veling *et al.*, 2011; but see Meule *et al.*, 2011a). This deficit in inhibitory control capacity has also been shown to moderate the effect of restraint on increased food intake; Jansen *et al.* (2009) found that restrained eaters only overate when they also displayed poor inhibitory control (also see van Koningsbruggen *et al.*, 2013b). In addition to being more impulsive generally, restrained eaters may be particularly vulnerable to impulsive overeating due to their increased motivation towards food. Compared to unrestrained individuals, those who score highly on measures of dietary restraint have been shown to display stronger implicit preferences (Hoefling & Strack, 2008; Houben *et*

al., 2010a, 2012c), attentional biases (Francis *et al.*, 1997; Hollitt *et al.*, 2010; Papies *et al.*, 2008; Stewart & Samoluk, 1997), automatic approach tendencies (Veenstra & de Jong, 2010) and salivary responses (Brunstrom *et al.*, 2004; Klajner *et al.*, 1981; Tepper, 1992) towards food.

Although the ego depletion model suggests that dieting may cause an increase in impulsive behaviour, there is also reason to believe that the opposite causal relationship may exist. It is possible that an impulsive disposition causes increased food intake, which therefore results in high dietary restraint as a consequence of weight gain (de Lauzon-Guillain, Basdevant, Romon, Karlsson, Borys & Charles, 2006; Heatherton *et al.*, 1988; Hill, 2004; Johnson, Pratt & Wardle, 2012; Snoek, van Strien, Janssens & Engels, 2008). Indeed, studies have shown that priming impulsivity in both restrained and unrestrained individuals can result in increased food intake (Guerrieri *et al.*, 2009, study 2; Rotenberg *et al.*, 2005). However, Guerrieri *et al.* (2009, study 2) found that restrained eaters who were actively dieting consumed fewer calories when instructed to focus on speed in a SST, compared with those who were instructed to focus on stopping. These results are consistent with the ego depletion model and suggest that additional tasks requiring self-control may cause dieters to overeat. Conversely, providing restrained eaters with food-related self-control training may be an effective intervention for reducing food consumption. Houben and Jansen (2011), Lawrence *et al.*, (under review) and Veling *et al.* (2011) have all shown that a period of response inhibition training, in which individuals must stop their responses towards images of food, can result in decreased food intake compared with performing a control training task. Importantly, the effect of this training procedure on food intake was moderated by dietary restraint: with the exception of Lawrence *et al.*'s first study, unrestrained eaters did not show a decrease in food consumption as a result of inhibition training. Together, these results indicate that individuals who exhibit increased impulsivity and food motivation are likely to benefit the most from food-related response inhibition training.

Although, the above three studies all demonstrated effects of inhibition training on restrained eaters, three different measures of dietary restraint were used. Houben and

Jansen (2011) used the original Restraint Scale (RS; Herman & Polivy, 1980); Veling *et al.* (2011) used only the ‘concern for dieting’ subscale of this measure (RSCD); while Lawrence *et al.* (under review) used the restrained eating subscale of the Dutch Eating Behaviour Questionnaire (DEBQRE; van Strien *et al.*, 1986a). These methodological differences are important to consider as these measures have been associated with different forms of restrained eating (for reviews see Heatherton *et al.*, 1988; Lowe, 1993; Mela, 2001). Generally, increased food motivation and impulsivity in restrained eaters has been linked to the RS, however, this scale has been shown to consist of two distinct factors: the concern for dieting scale and the weight fluctuation scale (Blanchard & Frost, 1983; Drewnowski *et al.*, 1982; Heatherton *et al.*, 1988; Ruderman, 1983; van Strien *et al.*, 2002). The former is believed to reflect a greater attentional and emotional association with food (i.e. feeling conscious of one’s food intake and feeling guilt after overeating; Blanchard & Frost, 1983), whereas the weight fluctuation scale may be responsible for associations between restraint and increased BMI – this could be due to the scale measuring absolute changes in weight gain or may reflect increased attempts to compensate for weight gain in those with a higher BMI (Drewnowski *et al.*, 1982; Ruderman, 1985; Stunkard & Messick, 1985; Stroebe *et al.*, 2008; van Strien *et al.*, 2002). The DEBQRE, on the other hand, has been shown to reflect reduced calorie intake and successful dieting (Brogan & Hevey, 2013; Laessle *et al.*, 1989; Wardle & Beales, 1987; van Strien, Frijters, van Staveren, Defares & Deurenberg, 1986b).

Therefore, despite findings that these three measures are highly correlated with one another (Allison *et al.*, 1992; Laessle *et al.*, 1989; Wardle, 1986), and evidence that all appear to moderate the effect of inhibition training on food consumption (Houben & Jansen, 2011; Lawrence *et al.*, under review; Veling *et al.*, 2011), there are important differences between these measures that should be explored. As this field of research is still developing it is important to understand the potential effects of differing methodologies and ultimately strive towards standardisation, replication, and replicability. Furthermore, as interest grows for the use of response inhibition training as a potential tool for weight loss (Veling *et al.*, 2014; Lawrence, O’Sullivan, Parslow, Javaid, Adams, Chambers, & Verbruggen, in preparation), understanding which populations to target these interventions toward may be of

critical importance. In this study, I therefore explored some potential similarities and differences between the RS (Herman & Polivy, 1980), the RSCD and the DEBQRE (van Strien *et al.*, 1986a). In a series of experiments, presented later in this thesis, participants were recruited for studies investigating the effect of inhibition training on food consumption using the RS (*a priori* using the cut-off score of 15+; Polivy & Herman, 1999). This prescreening process also included demographic information, scores on the DEBQRE, the external eating scale of the DEBQ (DEBQEE) – a scale measuring the extent to which people overeat as a result of external food cues – and the chocolate craving scale of the Attitudes to Chocolate Questionnaire (ACQC; Benton *et al.*, 1998). The measure of chocolate craving was included as a screening tool for Study 2 but is also of interest in the present study as chocolate is reported to be the most commonly craved food (Hill & Heaton-Brown, 1994; Hill *et al.*, 1991; Rozin *et al.*, 1991; Weingarten & Elston, 1991). The DEBQRE was included to explore correlations between this measure and the RS total and RSCD scales, and the DEBQEE was included to explore correlations between external and restrained eating across these three measures.

In addition to the measures discussed above, ratings of food liking for a variety of healthy and unhealthy foods were collected in a second sample of undergraduate psychology students. These ratings were used to explore associations between food preferences and each of the three measures of dietary restraint. Those who met the eligibility criteria during prescreening (scoring 15+ on the RS) and participated in one of the studies formed a third sample for whom data on general food craving (using the General Food Craving Questionnaire – Trait version; G-FCQ-T; Cepeda-Benito *et al.*, 2000; Nijs *et al.*, 2007) and BMI was also collected. In addition, DEBQRE was measured in this sample for a second time at the end of the study. As Lawrence *et al.* (under review) measured DEBQRE after participation in an inhibition task and taste test, it was necessary to rule out the possibility that these tasks may have influenced self-reported restrained eating on this measure. In the analysis for the present study I first examined the internal consistency, factor structure and demographic differences of these measures to see whether they were in accordance with previous research. I then explored correlations within different

measures of restraint and between these measures and external eating, food preferences, food craving and BMI.

2.2. Method

2.2.1. Participants

There were three samples included in this study. The first sample (sample 1) was mainly staff and students from Cardiff University who responded to advertisements for a study investigating food and positive emotion. They were prescreened for other studies using the Restraint Scale (RS; Herman & Polivy, 1980) and the Attitudes to Chocolate Questionnaire Craving scale (ACQC; Benton *et al.*, 1998). During prescreening participants also completed the restrained eating (DEBQRE) and external eating (DEBQEE) scales of the Dutch Eating Behaviour Questionnaire (DEBQ; van Strien *et al.*, 1986a). Thirteen hundred and twenty individuals completed these questionnaires and were included in sample 1 (1031 females; age range: 18-67, $M=22.63$, $SE=0.19$). Sample 2 consisted of 207 first year undergraduate psychology students (185 females; age range: 18-42, $M=18.61$, $SE=0.17$) who participated in an induction session to experimental participation in their first semester. They completed the RS, ACQC, DEBQRE, DEBQEE and measures of liking for unhealthy and healthy foods. The third sample (sample 3) was a subset of sample 1; all 245 participants in sample 3 were eligible for the advertised research (231 females; age range: 18-61, $M=22.26$, $SE=0.46$). These participants all scored highly on the RS (15+) and participated in a study investigating the effect of inhibition training on food consumption. These participants were included in this study due to additional and informative variables that were obtained during the experimental sessions. These included general food craving and BMI and only these variables were analysed for this sample. All methods were approved by the School of Psychology Research Ethics Committee, Cardiff University.

2.2.2. Measures and Materials

2.2.2.1. *The Restraint Scale (RS)*

The RS (Herman & Polivy, 1980) is a 10 item questionnaire; answers are scored from 0-3 or 0-4 and total scores range from 0-35. A total score of 15+ has previously been used as a cut-off to indicate ‘restrained eating’ (e.g. Houben & Jansen, 2011; Polivy & Herman, 1999; Roefs *et al.*, 2005). There are considered to be two subscales of the RS: concern for dieting (RSCD) and weight fluctuations (RSWF; Blanchard & Frost, 1983; Drewnowski *et al.*, 1982; Heatherton *et al.*, 1988; Ruderman, 1983; van Strien *et al.*, 2002). The RSCD subscale includes questions regarding dieting frequency and feelings towards weight gain and overeating (for example, “Do you have feelings of guilt after overeating?”) whereas the RSWF subscale asks questions regarding weight loss and weight gain (for example “What is the maximum amount of weight (in pounds) that you have ever lost within one month?”). There are six questions in the RSCD scale (range 0-19) and four questions in the RSWF scale (range 0-16).

2.2.2.2. *The Dutch Eating Behaviour Questionnaire – Restrained Eating Scale (DEBQRE)*

The DEBQ (van Strien *et al.*, 1986a) is a 33 item questionnaire measuring restrained, emotional and external eating behaviour. The restrained eating scale (DEBQRE) includes ten questions regarding restriction or avoidance of food intake. For example respondents are asked whether they restrict intake when they gain weight and if they eat less than they would like to because they are concerned about weight gain. All questions are scored on a five point scale from 1, ‘Never’ to 5, “Very often” (range 10-50).

2.2.2.3. *The Dutch Eating Behaviour Questionnaire – External Eating Scale (DEBQEE)*

The external eating subscale of the DEBQ (DEBQEE) includes ten questions concerning eating, and overeating, as a result of external food cues such as the taste, sight and smell of food (for example, “If food smells and looks good to you, do you eat more than usual?”) as well as overeating as the result of seeing others eating (for

example, “Do you eat more than usual when you see others eating?”). All questions are scored on a five point scale from 1, ‘Never’ to 5, “Very often” (range 10-50) and one question carries a reverse score (“Can you resist eating delicious foods?”).

2.2.2.4. *The Attitudes to Chocolate Questionnaire – Craving Scale (ACQC)*

The chocolate craving subscale of the ACQ (ACQC; Benton *et al.*, 1998) has 10 items that are scored on a 7 point scale from -3, “not at all like me”, to +3, “very much like me” (range from -30 to +30). The questions concern wanting and desire for chocolate (for example, “My desire for chocolate often seems overpowering”) as well as a lack of control over chocolate consumption (for example, “Even when I do not really want any more chocolate I will often carry on eating it”).

2.2.2.5. *Ratings of Healthy and Unhealthy Food Liking*

Respondents in sample 2 were asked to rate the extent to which they liked various unhealthy (biscuits, cake, chocolate, crisps, ice-cream), healthy (fruit, vegetables) and neutral foods (bread, cheese, rice-cakes, savoury snacks, yogurt) using a ten point scale from 1, “I like very much” to 10, “I don't like very much” (all foods were presented as words). Liking for unhealthy and healthy foods were scored as the mean value for these foods (range 1-10). In accordance with previous studies investigating the relationship between executive function and healthy dietary behaviours, only fruit and vegetables were included as healthy foods (Allan *et al.*, 2011; Collins & Mullan, 2011; Hall *et al.*, 2008; Riggs, Spruijt-Metz, Chou & Pentz, 2012)².

2.2.2.6. *The General Food Craving Questionnaire – Trait Version (G-FCQ-T)*

The G-FCQ-T (Cepeda-Benito *et al.*, 2000; Nijs *et al.*, 2007) is a 21 item questionnaire measuring the strength of food cravings. Respondents are asked to answer the extent to which each question is generally true for them using a six point scale from 1, “Never or not applicable” to 6 “Always” (range 21-126). There are four craving subscales including: preoccupation with food (for example, “I feel like I have food on my mind all the time”; six questions; range 6-36), loss of control over

² The inclusion of rice-cakes and yogurt in this analysis did not influence the results. Neutral foods were included to disguise the purpose of these questions.

food intake (for example, “If I eat what I’m craving, I often lose control and eat too much”; six questions, range 6-36), positive outcome expectancy (for example, “Eating what I crave makes me feel better”; five questions, range 5-30) and emotional craving (for example, “I crave foods when I’m upset”; four questions, range 4-24).

2.2.2.7. Body Mass Index (BMI)

The height and weight of individuals who participated in the experimental studies (sample 3) were measured to calculate their BMI (kg/m²).

2.2.3. Procedure

All participants received the questionnaires in the same order; The DEBQ restrained and external eating measures were followed by the RS and the ACQC. Participants in sample 2 then received the food liking measures. All participants in sample 2 completed the questionnaires via an internet survey whereas those in sample 1 could choose to answer the questionnaires electronically via email, in hard copy or via an internet survey. All participants in sample 3 were recruited for a behavioural study in which they received either inhibition or control training followed by a food consumption phase and a series of questionnaires (the full details of these studies can be found in Chapters 3 and 4). These questionnaires included the DEBQRE, DEBQEE and G-FCQ-T. The height and weight of these participants was recorded at the end of the study to calculate BMI.

2.2.4. Statistical Analysis

First I explored the internal consistency and factor structures of the questionnaire measures to ensure that the data was consistent with previous findings. These analyses were performed on samples 1 and 2 separately to consider the replicability of these findings. Demographic differences for age and gender in restrained eating were explored; for this analysis I collapsed across both samples 1 and 2 as the age range and gender ratio in sample 2 were fairly homogenous (in sample 2 92.75% were aged 18-19, compared to 43% in sample 1 and the sample was only 10% male in sample 2, compared to 20% in sample 1). Subsequently I explored the similarities and differences between the RS, RSCD and DEBQRE. I looked at intercorrelations

between these scales as well as correlations with external eating (using the DEBQEE), food liking (for unhealthy and healthy foods), food craving (ACQC and G-FCQ-T) and BMI. Correlations for sample 1 were well powered; the minimum sample size across all comparisons was 1306. A sensitivity analysis revealed that the smallest detectable effect size with 90% power was $r=0.09$ (using G*Power; Faul, Erdfelder, Lang & Buchner, 2007). Samples 2 and 3 had smaller sample sizes with a minimum N of 206 and 213, respectively, across all comparisons; these sample sizes enabled detection of $r \geq 0.22$ with 90% power and $r \geq 0.19$ with 80% power. All results are reported with unadjusted significance values; corrections for multiple comparisons were calculated for all within-test, within-sample analyses and are only reported where these corrections changed the interpretation of an analysis from statistically significant to non-statistically significant. All analyses were carried out using SPSS.

2.3. Results

2.3.1. Internal Consistency

Internal consistency was calculated using Cronbach's alpha for both samples. The reliability estimates were high ($\alpha > 0.8$) for the RS and DEBQRE across both samples and were satisfactory ($\alpha > 0.7$) for RSCD, RSWF and DEBQEE (see Table 2.1). These values are similar to those of previous studies (Allison *et al.*, 1992; Laessle *et al.*, 1989; Ruderman, 1983).

2.3.2. Factor Structure

The factor structure of the RS and DEBQRE were explored using principle components analysis with varimax rotation in accordance with previous research (Allison *et al.*, 1992; Blanchard & Frost, 1983; Laessle *et al.*, 1989; Ruderman, 1983). For the RS the Kaiser-Meyer-Olkin (KMO) measure of sampling adequacy was 0.85 for sample 1 and 0.88 for sample 2 indicating that factor extraction could be performed for both samples (Kaiser, 1974). Extraction revealed two factors with eigenvalues greater than 1 for both samples. The first factor explained 29.25% of the variance in sample 1 and 35.41% in sample 2 whereas the second factor explained 24.27% in sample 1 and 22.22% in sample 2 (see Table 2.2). These factors combined

explained more variance than when a forced extraction of one factor was used (for sample 1: 53.52% compared to 38.29%; for sample 2: 57.63% compared to 44.29%). The two factors were consistent with the RSCD and RSWF subscales previously found and explained a similar amount of variance (Allison *et al.*, 1992; Blanchard & Frost, 1983; Drewnowski *et al.*, 1982; Heatherton *et al.*, 1988; Ruderman, 1983), although some items loaded onto both factors (factor loadings greater than 0.4 are shown in Table 2.3). The DEBQRE was also suitable for factor analysis: KMO values were 0.94 and 0.95 for samples 1 and 2, respectively. Consistent with previous research the DEBQRE revealed just one factor (Allison *et al.*, 1992; van Strien *et al.*, 1986a) with an eigenvalue of >1 which explained 54.96% of the variance in sample 1 and 64.26% in sample 2.

Table 2.1. Internal consistency for the scales used.

	Cronbachs alpha (α)	
	Sample 1 (min. N=1306)	Sample 2 (min. N=206)
RS	0.82	0.85
RSCD	0.79	0.83
RSWF	0.74	0.75
DEBQRE	0.91	0.94
DEBQEE	0.76	0.75
ACQC	0.93	0.91

Note. RS= Restraint Scale; RSCD= concern for dieting subscale of the RS; RSWF= weight fluctuation scale of the RS; DEBQRE= Dutch Eating Behaviour Questionnaire Restrained Eating scale; DEBQEE= Dutch Eating Behaviour Questionnaire External Eating scale; ACQC= Attitudes to Chocolate Questionnaire Craving scale

Table 2.2. Factor extraction for the RS and DEBQRE.

	Sample 1 (min. N=1306)			Sample 2 (min. N=206)		
	No. principal components	Component	Variance explained (%)	No. principal components	Component	Variance explained (%)
	RS	2	1	29.25	2	1
		2	24.27		2	22.22
DEBQRE	1	1	54.96	1	1	64.26

Note. RS= Restraint Scale; DEBQRE= Dutch Eating Behaviour Questionnaire Restrained Eating scale.

Table 2.3. Factor loadings for the RS across both samples. Only loadings >0.4 are depicted.

	Sample 1 (min. N=1306)		Sample 2 (min. N=206)	
	Factor 1 (RSCD)	Factor 2 (RSWF)	Factor 1 (RSCD)	Factor 2 (RSWF)
	1. How often dieting?	<u>0.66</u>	0.43	<u>0.78</u>
2. Max weight lost 1m?		<u>0.68</u>	0.43	<u>0.52</u>
3. Max weight gain 1wk?		<u>0.83</u>		<u>0.86</u>
4. Weight fluctuate 1wk?		<u>0.77</u>		<u>0.81</u>
5. Effect of 5lb change?	<u>0.73</u>		<u>0.77</u>	
6. Splurge alone?	<u>0.52</u>		<u>0.44</u>	
7. Thought to food?	<u>0.67</u>		<u>0.71</u>	
8. Feelings of guilt?	<u>0.77</u>		<u>0.78</u>	
9. Conscious of eating?	<u>0.68</u>		<u>0.75</u>	
10. Max. overweight?		<u>0.64</u>	0.53	<u>0.56</u>

Note. Loadings underlined are the highest loadings across both factors for each sample. RS= Restraint Scale; RSCD= concern for dieting subscale of the RS; RSWF= weight fluctuation scale of the RS.

2.2.3. Demographic Differences

Correlations revealed a small but significant positive association between age and RS ($r=0.07$, $p=0.01$). This was due to a small, positive correlation between age and the weight fluctuation subscale ($r=0.1$, $p<0.001$); the correlation between age and concern for dieting was not statistically significant ($r=0.03$, $p=0.32$), nor was the correlation between age and DEBQRE ($r= -0.003$, $p=0.9$). Independent t-tests were performed to see whether there were any statistically significant differences between genders across restrained eating measures (see Table 2.4 for means and standard errors). Females scored significantly higher than males across all restraint measures (all $ps<0.001$; all $d>0.4$).

Table 2.4. Means for restrained eating collapsed across samples 1 and 2 according to gender (SE within parentheses).

	Samples 1 & 2	
	Females (min. N=1214)	Males (min. N=278)
RS	13.75 (0.17)	10.28 (0.31)
RSCD	8.78 (0.11)	6.57 (0.19)
RSWF	4.98 (0.09)	3.74 (0.18)
DEBQRE	27.22 (0.24)	21.24 (0.45)

Note. RS= Restraint Scale; RSCD= concern for dieting subscale of the RS; RSWF= weight fluctuation scale of the RS; DEBQRE= DEBQ Restrained Eating scale

Due to the large differences in restraint scores between genders I repeated the correlations between age and restrained eating measures according to gender. The results revealed the same pattern of relationships for female respondents (showing a positive correlation between age and RSWF ($r=0.11$, $p<0.001$)); however, whereas women showed no statistically significant relationships between either RSCD or DEBQRE and age, men showed a significant positive relationship between both RSCD and age ($r=0.27$, $p<0.001$) and DEBQRE and age ($r=0.17$, $p=0.006$). Fisher's Z test for independent correlations revealed that the difference in correlations between RSCD and age ($z=3.89$, $p<0.001$) and DEBQRE and age ($z=2.5$, $p=0.012$)

between men and women were statistically significant. These results suggest that women maintain a fairly high level of dietary restraint across time whereas RSCD and DEBQRE scores increase with age in men.

2.2.4. Comparison of the RS and DEBQRE

Potential similarities and differences were explored between the RS and DEBQRE as previous studies have shown a moderating role of RS, RSCD and DEBQRE for the effect of inhibition training on food consumption (Houben & Jansen, 2011; Lawrence *et al.*, under review; Veling *et al.*, 2011). As Lawrence *et al.* (under review) measured restraint with the DEBQRE following inhibition training and food consumption I first checked the reliability of this measure when administered before and after inhibition training and food consumption in sample 3. I found significant positive correlations between the pre- and post-training measures of DEBQRE for those who performed the control training task ($r=0.66, p<0.001; N=97$) and for those in the inhibition training tasks ($r=0.58, p<0.001; N=98$). Furthermore, a mixed 2x2 ANOVA (between-subjects variable: *training condition*: control or inhibition; within subjects variable: *DEBQ time*, pre- or post-training) revealed a non-significant interaction between training condition and pre- and post-training DEBQRE measures ($F(1,193)=0.42, p=0.52, \eta^2_p=0.002$), providing no evidence that training influenced self-reported restraint on this scale. Veling *et al.* (2011) also administered the RSCD following training; however, unfortunately, pre- and post-training data for this measure was not collected.

2.2.4.1. Restraint Correlations

Significant positive correlations were found between DEBQRE and all three RS measures within samples 1 and 2 (all $ps<0.001$ for sample 1 and all $ps<0.01$ for sample 2; see Table 2.5). Importantly, as Houben and Jansen (2011) used the overall RS score in their regression analysis, the relationship between RS and DEBQRE (used by Lawrence *et al.*, under review) revealed a positive correlation with a large effect size in both samples (sample 1: $r=0.72, p<0.001$; sample 2: $r=0.79, p<0.001$). This relationship appears to rely to a greater extent on the correlation between DEBQRE and RSCD, which yielded larger effect sizes ($r= 0.77$ and 0.84 for samples 1 and 2, respectively) compared to the correlation between DEBQRE and RSWF

($r=0.44$ and 0.53 for samples 1 and 2, respectively). A Steiger's Z test for dependent correlations revealed that the correlation between DEBQRE and RSCD was significantly greater than the correlation between DEBQRE and RSWF for both sample 1 ($z=16.7, p<0.001$) and sample 2 ($z=7.6, p<0.001$). This greater association between DEBQRE and RSCD compared to DEBQRE and RSWF has also been reported previously (Allison *et al.*, 1992).

2.2.4.2. External Eating

Although the RS and DEBQRE were highly correlated with one another, they were differentially correlated with external eating. In sample 1, DEBQEE was positively correlated with RS, RSCD and RSWF (all $ps<0.01$; although effect sizes were small: all $rs<0.12$), but was not significantly correlated with DEBQRE ($r=-0.01, p=0.76$). After correcting for multiple comparisons, Steiger's Z tests revealed that the differences in correlations between these three measures of restraint were not statistically significant. In sample 2, DEBQEE did not significantly correlate with any RS measures (all $ps>0.15$), however, there was a significant negative correlation with a small-medium effect size, between DEBQEE and DEBQRE ($r=-0.18, p=0.008$).

Table 2.5. Correlation matrix for restraint scales and the external eating scale.

	RS	RSCD	RSWF	DEBQRE	DEBQEE	
RS		0.88***	0.83***	0.72***	0.12***	Sample 1 (min. N=1306)
RSCD	0.91***		0.47***	0.77***	0.12***	
RSWF	0.85***	0.57***		0.44***	0.09**	
DEBQRE	0.79***	0.84***	0.53**		-0.01	
DEBQEE	-0.07	-0.10	-0.02	-0.18**		
Sample 2 (min. N=206)						

*** $p<0.001$ ** $p<0.01$

Note. RS= Restraint Scale; RSCD= concern for dieting subscale of the RS; RSWF= weight fluctuation scale of the RS; DEBQRE= Dutch Eating Behaviour Questionnaire Restrained Eating scale; DEBQEE= Dutch Eating Behaviour Questionnaire External Eating scale

2.2.4.3. Food Liking

Measures of liking for unhealthy and healthy foods were collected in sample 2 only. Correlations between these ratings and measures of restrained eating, with unadjusted significance values, revealed that unhealthy food liking was significantly and negatively correlated with both the RS ($r=-0.14$, $p=0.049$) and the DEBQRE ($r=-0.17$, $p=0.017$; see Table 2.6). The relationship between unhealthy food liking and RS scores was due to a significant correlation with the RSCD subscale ($r=-0.15$, $p=0.028$); the relationship between unhealthy food liking and RSWF was not significant ($p=0.25$). The only significant correlation for healthy food liking was a significant positive correlation with DEBQRE ($r=0.16$, $p=0.022$). The RS total score and subscales did not correlate significantly with healthy food liking (all $ps>0.1$). However, after corrections for multiple comparisons were made across each food type, none of these relationships remained statistically significant (with four comparisons per food group, $\alpha=0.0125$)³.

³ Relative preference for unhealthy compared to healthy foods was also analysed by subtracting the mean healthy food liking score from the mean unhealthy food liking score. Correlations revealed a significant negative relationship with RS ($r=-0.15$, $p=0.03$), RSCD ($r=-0.2$, $p=0.004$) and DEBQRE ($r=-0.24$, $p<0.001$) indicating that preferences favour healthy over unhealthy foods as restraint increases across these three indices (the relationship with RSWF was not significant; $r=-0.05$, $p=0.51$). After correcting for multiple comparisons ($\alpha/4=0.0125$) the relationship with total RS score was no longer significant. A Steiger's Z test also showed that the difference between RS and DEBQRE was statistically significant ($z=2.07$, $p<0.05$) suggesting that this association was stronger for the DEBQRE than the RS. Differences between the RSCD and the RS ($z=1.67$, $p>0.05$) and DEBQRE ($z=1.12$, $p>0.05$) were both non-significant.

Table 2.6. Correlations between restrained eating scales and measures of self-reported liking for unhealthy and healthy foods in sample 2.

	Sample 2 (min. N=206)	
	Unhealthy Food Liking	Healthy Food Liking
RS	-0.14*	0.07
RSCD	-0.15*	0.12
RSWF	-0.08	-0.02
DEBQRE	-0.17*	0.16*

* $p < 0.05$

Note. RS= Restraint Scale; RSCD = concern for dieting subscale of the RS; RSWF= weight fluctuation scale of the RS; DEBQRE= Dutch Eating Behaviour Questionnaire Restrained Eating scale

2.2.4.4. Food Craving

Measures of chocolate craving were obtained for samples 1 and 2 (using the ACQC; Benton *et al.*, 1998). For sample 1 this measure was significantly and positively correlated with the RS ($r=0.27, p < 0.001$), RSCD ($r=0.25, p < 0.001$), RSWF ($r=0.20, p < 0.001$) and DEBQRE ($r=0.13, p < 0.001$). After corrections for multiple comparisons, Steiger's Z tests revealed that the correlation between ACQC and RS was significantly greater than that with RSWF ($z=4.49, p < 0.001$) and with DEBQRE ($z=7.24, p < 0.001$). The correlation between RSCD was also significantly greater than the correlation with DEBQRE ($z=6.88, p < 0.001$). These relationships were not significant in sample 2 although the relationship between ACQC and RS showed a trend towards significance ($r=0.12, p=0.079$), however, this was not statistically significant after correcting for multiple comparisons (with four comparisons, $\alpha=0.0125$).

In the third sample, a measure of general food craving was also recorded (using the G-FCQ-T; Cepeda-Benito *et al.*, 2000; Nijs *et al.*, 2007). Correlations between all subscales for this questionnaire and the restrained and external eating measures are given in Table 2.7. All of these results are reported here for completeness; however,

the main relationships of interest are between the restrained eating scales and the G-FCQ-T total score. For the G-FCQ-T subscales, the RS was significantly and positively associated with the preoccupation with food ($r=0.22, p<0.01$), loss of control ($r=0.25, p<0.001$) and emotional craving ($r=0.20, p<0.01$) scales. These correlations were mainly due to associations with the RSCD subscale, although RSWF did show a trend towards a significant relationship with the loss of control subscale ($r=0.13, p=0.057$). For the G-FCQ-T total score, there was a significant positive relationship with the RS ($r=0.22, p<0.01$) and RSCD ($r=0.18, p<0.001$); these two correlations did not statistically differ from one another ($z=0.07, p=0.47$). The correlations with RSWF and DEBQRE did not reach statistical significance (both $ps>0.27$). For external eating, this measure was significantly and positively associated with all food craving scales (all $ps<0.001$).

Table 2.7. Correlations between restrained eating measures and measures of general food craving (G-FCQ-T; Cepeda-Benito *et al.*, 2000; Nijs *et al.*, 2007) for sample 3 who scored highly on the RS (15+) and participated in one of the experimental studies in this thesis.

Sample 3 (min. N=213)					
	FCQ-PWF	FCQ-LoC	FCQ-POE	FCQ-EC	FCQ-total
RS	0.22**	0.25***	0.01	0.20**	0.22**
RSCD	0.24***	0.17*	-0.04	0.18**	0.18**
RSWF	0.00	0.13~	0.07	0.03	0.07
DEBQRE	-0.06	-0.04	-0.10	0.00	-0.06
DEBQEE	0.49***	0.42***	0.35***	0.40***	0.52***

*** $p<0.001$ ** $p<0.01$ * $p<0.05$ ~ $p<0.1$

Note. RS= Restraint Scale; RSCD= concern for dieting subscale of the RS; RSWF= weight fluctuation scale of the RS; DEBQRE= Dutch Eating Behaviour Questionnaire Restrained Eating scale; DEBQEE= Dutch Eating Behaviour Questionnaire External Eating scale; FCQ-PWF= preoccupation with food scale of the G-FCQ-T; FCQ-LoC= loss of control scale of the G-FCQ-T; FCQ-POE= positive outcome expectancy scale of the G-FCQ-T; FCQ-EC= emotional craving scale of G-FCQ-T; FCQ-total= total score for the G-FCQ-T

2.2.4.5. BMI

Measures of BMI were also obtained in sample 3. The mean BMI for these participants was 24.7 ($SE=0.29$; range 16.79 - 40.47) showing that they marginally fell into the healthy weight category (healthy weight range: 18.5–24.9; overweight range: 25–29.9; obese range: 30+). Correlations between restraint measures and BMI revealed a significant positive relationship between BMI and both the RS ($r=0.25$, $p<0.001$) and RSWF ($r=0.34$, $p<0.001$); these two correlations were not statistically different ($z=1.44$, $p=0.15$); the relationship between BMI and RSCD was not statistically significant ($r=-0.03$, $p=0.57$). The correlation between BMI and DEBQRE, conversely, revealed a trend towards a significant negative relationship for this sample ($r=-0.13$, $p=0.051$; the second measure of DEBQRE recorded during the experiment, but before BMI was measured, revealed a significant negative relationship with BMI: $r=-0.15$, $p=0.022$).

2.4. Discussion

This study compared three measures of restrained eating – the Restraint Scale (RS; Herman & Polivy, 1980), the concern for dieting scale of this questionnaire (RSCD) and the restrained eating scale of the DEBQ (DEBQRE; van Strien *et al.*, 1986a). Previous studies have shown a moderating role of dietary restraint for the effect of inhibition training on reduced food intake for the RS (Houben & Jansen, 2011), the RSCD (Veling *et al.*, 2011) and the DEBQRE (Lawrence *et al.*, under review). However, it has been argued that the RS and DEBQRE measure different facets of restrained eating. The RS is believed to be associated with increased food cue reactivity and food intake, whereas the DEBQRE has been linked to successful dietary restriction (Fedoroff *et al.*, 1997, 2003; Francis *et al.*, 1997; Heatherton *et al.*, 1988; Klajner *et al.*, 1981; Laessle *et al.*, 1989; Houben *et al.*, 2010a, 2012c; Veenstra & de Jong, 2010; Wardle & Beales, 1987). In this study I first examined the internal consistency, factor structure and demographic differences for these measures and then explored the similarities and differences between them with regards to external eating, food liking, food craving and BMI.

Consistent with previous research, the results revealed a high degree of internal consistency for the RS, RSCD and DEBQRE across two separate samples (Allison *et al.*, 1992; Laessle *et al.*, 1989; Meule *et al.*, 2012b; Ruderman, 1983; Stroebe *et al.*, 2008). The two-factor structure of the RS, with the concern for dieting and weight fluctuation (RSWF) subscales, and the one factor structure for the DEBQRE (Allison *et al.*, 1992; Blanchard & Frost, 1983; Drewnowski *et al.*, 1982; Heatherton *et al.*, 1988; Ruderman, 1983; van Strien *et al.*, 2002) were also replicated. For the demographic results it was revealed that women scored significantly higher than men across all measures of dietary restraint. This finding has been reported consistently in the literature (Allison *et al.*, 1992; Burton *et al.*, 2007; Drewnowski *et al.*, 1982; Rand & Kulda, 1991; Wardle, 1986), although here there is evidence to suggest that RSCD and DEBQRE scores may increase with age among men.

When comparing the dietary restraint measures I found strong positive correlations between the RS and DEBQRE, the RS and RSCD, and between the RSCD and DEBQRE. These results are consistent with previous findings and suggest that the RS and DEBQRE measure the same construct to a large extent (Allison *et al.*, 1992; Laessle *et al.*, 1989; Wardle, 1986). However, the correlations between restraint measures and food liking were in disagreement with previous research that has demonstrated an increased motivation towards unhealthy foods in restrained eaters (Brunstrom *et al.*, 2004; Houben *et al.*, 2010a, 2012c; Klajner *et al.*, 1981). Here, I found that the RS, RSCD and DEBQRE were all significantly and *negatively* correlated with self-reported liking for unhealthy foods. One possible explanation for these conflicting findings is the way in which participants were asked to rate the extent to which they liked certain foods. Rather than answering these questions based on taste or desirability, it is possible that participants rated these items negatively because of their association with weight gain. As these questions were presented after the RS and DEBQRE, it is likely that these foods would have reminded respondents of their dieting goal (Stroebe *et al.*, 2008, 2013), which would be more likely in those with high levels of dietary restraint. Nevertheless, a similar finding has also been reported when participants were asked to rate the hedonic quality of foods; Papies *et al.* (2008) showed that restrained eaters who scored highly on the RSCD rated palatable foods as significantly less ‘tasty’ than unrestrained

participants. This disparity in findings between positive and negative associations of food liking and restraint may be explained by the use of implicit and explicit measures (although some studies using explicit ratings of liking or palatability have found no difference between restrained and unrestrained eaters; see Roefs *et al.*, 2005; Papies *et al.*, 2008). Indeed, some researchers argue that high dietary restraint is characterised by an ambivalent attitude towards calorific foods, with individuals experiencing positive implicit preferences and both positive and negative explicit attitudes towards these foods (Hoefling & Strack, 2008; Keller & van der Horst, 2013; Stroebe *et al.*, 2008, 2013; Umland & Ito, 2005).

Although the current findings revealed a negative relationship between dietary restraint and explicit ratings for unhealthy food liking, the results for self-reported food cravings showed some positive correlations with restraint. The first sample showed a positive relationship between chocolate craving and all measures of restraint, and a trend towards significance for chocolate craving and RS was found in sample 2. In the third sample of high RS scorers, I found positive relationships between a measure of general food craving and the RS and RSCD but not with the DEBQRE. These results are consistent with previous findings showing a positive relationship between the RS and measures of food craving (Meule *et al.*, 2012b; Polivy, Coleman & Herman, 2005) and findings showing a negative or non-significant association between food craving and the DEBQRE (Burton *et al.*, 2007; Nicholls & Hulbert-Williams, 2013; Tetley, Brunstrom & Griffiths, 2009). The contradictory findings between decreased food liking and increased food craving, particularly for the RS, can be interpreted in light of Robinson and Berridge's (1993, 2001, 2003) incentive salience theory. When applied to food rewards (Berridge, 2009; Berridge *et al.*, 2010), this theory suggests that 'wanting' and 'liking' can become dissociated such that an individual may experience a motivational urge towards food without necessarily experiencing hedonic pleasure from that food. As discussed in Chapter 1 (see section 1.2.1), some individuals may experience an addictive-type relationship with food in which the overconsumption of energy-dense foods causes tolerance (i.e. a decreased effect of the food on sensations of 'pleasure' – often resulting in increased consumption) and sensitisation (i.e. increased reactivity to reward-related cues – often associated with craving and seeking behaviours). The

diminished experience of pleasure and increased craving for these foods can result in compulsive eating, therefore creating a cycle of ‘food addiction’. Although there is no direct evidence linking restrained eating with food addiction, scores on the RS and RSCD have been related to several precursors such as increased impulsivity and reduced inhibitory control (Jansen *et al.*, 1989, 2009; Nederkoorn *et al.*, 2004; Veling *et al.*, 2011). Furthermore, symptoms of food addiction and RSCD scores have both been shown to correlate positively with food craving (Meule *et al.*, 2012b). Food cravings are an important phenomenon with regards to disinhibited eating; as well as being correlated with food addiction scores (Davis *et al.*, 2011; Meule *et al.*, 2012b), they have also been linked to increased food intake, binge eating and BMI (Burton *et al.*, 2007; Dalton *et al.*, 2013; Hill *et al.*, 1991; Lafay *et al.*, 2001; White *et al.*, 2002). Therefore, compared to the DEBQRE, the RS may be more sensitive to individuals who are prone to disinhibited eating.

This suggestion is also supported by the current findings for measures of external eating and BMI. The external eating scale of the DEBQ (DEBQEE) assesses the degree to which individuals overeat as a result of external influences such as the sight or smell of palatable foods. This measure has been associated with an increased attentional bias towards food (Brignell *et al.*, 2009; Hou *et al.*, 2011; Nijs *et al.*, 2010) and increased food craving (Nicholls & Hulbert-Williams, 2013). For example, in the third sample of high RS (15+) participants, the DEBQEE was positively correlated with all measures of general food craving. The correlations between restraint measures and external eating revealed positive associations with RS and RSCD in sample 1 and a negative relationship with DEBQRE in sample 2. These findings were also consistent with the correlations for BMI in our high RS scorers. The RS showed a significant positive correlation with BMI, in accordance with previous findings (Guerrieri *et al.*, 2009; Veenstra & de Jong, 2010), whereas the DEBQRE showed a trend towards a negative relationship. Again these differences indicate that the RS may be more strongly associated with increased food motivation and intake, whereas the DEBQRE is likely to reflect successful dietary restraint. Accordingly, I also found a significant positive relationship between liking for healthy foods and the DEBQRE but not with the RS or RSCD. This finding

reflects previous research showing an association between the DEBQRE and increased health consciousness (Keller & van der Horst, 2013).

Although the relationship between RS scores and BMI appeared to be due to the weight fluctuation scale (RSWF) of this questionnaire, this demonstrates that the total RS score may be more sensitive to individuals with disinhibited eating than the RSCD scale alone. However, it has been argued that because the RSWF scale measures absolute weight changes, the total RS score is likely to be overestimated in those with a high BMI (Rand & Kulda, 1991; Ruderman, 1985, 1986). This was a limitation of this study as BMI data was only collected for high RS scorers and not for the whole sample. To draw any firm conclusions regarding correlations between BMI and the RS and DEBQRE it would be necessary to obtain BMI data across a range of these scores. This was an unfortunate oversight in the current study as generally, correlations between self-reported and measured BMI values are high; although BMI tends to be underestimated across most populations (Brunner Huber, 2007; Kuczmarski, Kuczmarski & Najjar, 2001; Niedhammer, Bugel, Bonenfant, Goldberg & Leclerc, 2000; Nyholm, Gullberg, Merlo, Lundqvist-Persson, Råstam & Lindblad, 2007; Spencer, Appleby, Davey & Key, 2002).

In conclusion, the results of this study suggest that the RS (Herman & Polivy, 1980) may be a more suitable measure for determining 'restrained', or disinhibited, eating in studies investigating the effects of inhibition training on food consumption, compared to the DEBQRE (van Strien *et al.*, 1986a). As the DEBQRE appears to be associated with successful dietary restraint (Brogan & Hevey, 2013; Laessle *et al.*, 1989; Wardle & Beales, 1987; van Strien *et al.*, 1986b), it is less likely that training inhibitory control will have a robust and reliable effect on reduced food intake in individuals who score highly on this measure. Indeed, Lawrence *et al.* (under review) found a significant moderating effect of DEBQRE when measuring food intake in a bogus taste test with two foods, but not when they measured ad-libitum consumption of one food. Conversely, Veling *et al.* (2011) found a moderating role of RSCD scores when participants were provided with a bag of liquorice sweets and invited to eat as many as they liked over a 24 hour period. Houben and Jansen (2011) also found a moderating role of total RS scores in a taste test with three varieties of

chocolate. The current findings indicate that the RS may be a more effective measure for the recruitment of participants who will benefit the most from inhibitory control training due to its associations with increased external eating, food craving and BMI. Although the RSCD subscale is also highly correlated with external eating and food craving, the total RS score explains more variance on these measures. Moreover, the association between RS and BMI may help to identify those who struggle the most to reduce their food intake and maintain a healthy weight.

Chapter 3. Study 2

Training response inhibition in trait chocolate lovers: Effects on implicit attitudes and consumption

3.1. Introduction

Obesity has reached epidemic levels worldwide (WHO, 2003). Despite the huge economic burden (Fry & Finley, 2005) and costs to individual health (Bray, 2004; Carpenter *et al.*, 2000; Van Gaal *et al.*, 2006), there is an increasing abundance of easily available, highly palatable and calorie-dense foods (Hill & Peters, 1998; Jeffery & Utter, 2003). Although the human species has evolved to possess a remarkable capacity for self-control (Hagger & Chatzisarantis, 2013; Hall & Fong, 2007), the ability to resist tempting foods remains a challenge for many people. Yet, this does not appear to be true of everybody; some individuals are able to control their weight and maintain a healthy lifestyle. This leads us to the question of why some individuals are able succeed where others fail?

Dual process models would suggest that the answer lies in individual differences in impulsivity and self-control. These models argue that our behaviour is determined by the interaction of an impulsive system, which is driven by our hedonic needs, and a reflective system, which involves conscious thought and deliberation (Strack & Deutsch, 2004). In the case of overeating and obesity it is possible these individuals possess strong impulsive desires for calorie-dense foods and a lack of control over these desires. Indeed, research has shown that, compared to their healthy-weight counterparts, overweight and obese individuals are more likely to be impulsive across a range of questionnaires and behavioural measures (Batterink *et al.*, 2010; Chalmers *et al.*, 1990; Davis *et al.*, 2008a, 2010; Nederkoorn *et al.*, 2006a, 2006b, 2006c; Rydén *et al.*, 2003). They also report more food cravings (White *et al.*, 2002) and have been shown to demonstrate increased reactivity to food in brain regions associated with pleasure and reward (Rothmund *et al.*, 2007; Small *et al.*, 2003;

Stoeckel *et al.*, 2008; Volkow *et al.*, 2002a, 2003a). Furthermore, this increased motivation towards food appears to be coupled with a diminished capacity for self-control (e.g. Lawrence *et al.*, 2012). Imaging studies have revealed associations between increased BMI and reduced prefrontal activity and grey matter volume (Batterink *et al.*, 2010; Pannacciulli *et al.*, 2006; Taki *et al.*, 2008; Volkow *et al.*, 2009a; Walther *et al.*, 2010; Yokum, Ng & Stice, 2012), as well as reduced activity in these regions in response to a meal in obese men and women (Le *et al.*, 2006, 2007). Behavioural studies have also demonstrated an impaired ability to inhibit prepotent responses in overweight/obese adults and children (Cohen *et al.*, 2011; Guerrieri *et al.*, 2008a; Nederkoorn *et al.*, 2006a, 2006b; Wirt *et al.*, 2014) – a deficit which appears to be particularly potent in the presence of food cues (Houben *et al.*, 2012b; Nederkoorn *et al.*, 2012).

These findings have encouraged researchers to develop new behavioural interventions that are designed to target these individual differences. One of the more promising interventions for overeating and obesity is response inhibition training. Recent studies have shown that training individuals to inhibit simple motor responses to images of food can result in the decreased consumption of that food (Houben, 2011; Houben & Jansen, 2011; Lawrence *et al.*, under review; Veling *et al.*, 2011), healthier food choices (van Koningsbruggen *et al.*, 2013a; Veling *et al.*, 2013a, 2013b) and even weight loss (Veling *et al.*, 2014; Lawrence *et al.*, in preparation). However, the mechanisms underlying these effects are not yet fully understood and are of key interest for the effective development and delivery of these interventions. To date, moderating variables have included baseline response inhibition (Houben, 2011), appetite (Veling *et al.*, 2013a, 2013b), frequency of consumption (Veling *et al.*, 2013a) and dietary restraint (Houben & Jansen, 2011; Lawrence *et al.*, under review; Veling *et al.*, 2011). Although these moderators involve different measures they are all characterised by an increased motivation towards food and reduced control. It has been argued that in order for food-related inhibition training to be successful, the recipient must possess strong impulses towards food at the outset (Veling *et al.*, 2011). It is plausible therefore that inhibition training may influence food behaviour by reducing the strength of this impulse. One way in which this may be possible is through stimulus devaluation.

As discussed in Chapter 1, the Behaviour Stimulus Interaction theory (Veling *et al.*, 2008) argues that when we inhibit our responses towards a desired object, the conflict between approach and avoidance is resolved through the devaluation of that object. Veling *et al.* (2013b) found that when participants performed a food-related go/no-go (GNG) task in which they had to inhibit their responses towards certain food stimuli, the effect of appetite on food choice was mediated by explicit ratings of the attractiveness and tastiness of those foods. Changes in implicit attitudes have also been shown to mediate the effect of alcohol-related inhibition training on weekly alcohol intake in heavy drinkers (Houben *et al.*, 2011a, 2012a). When investigating the potential mediators for the effect of inhibition training on alcohol consumption, Houben *et al.* (2012a) considered changes in implicit attitudes, approach-avoidance tendencies and baseline response inhibition. Their findings showed a significant effect on implicit attitudes only, with no statistically significant change in approach or avoidance tendencies or response inhibition as a result of alcohol-related no-go training (but see Bowley *et al.*, 2013). These findings suggest that training inhibitory control towards certain foods may reduce consumption by altering implicit attitudes towards those foods. However, to date, there are no published studies investigating the effect of food-related inhibition training on changes in implicit food attitudes.

The aim of the current study was to address this gap in the literature by training trait chocolate lovers to inhibit their responses towards images of chocolate and subsequently measuring their implicit attitudes towards, and consumption of chocolate. As previous research has implied that individuals must have a strong impulsive desire towards food for inhibitory control training to be successful, this sample was restricted *a priori* to participants who scored highly on measures of chocolate craving and dietary restraint. Chocolate lovers were chosen for this study as chocolate is considered to be the most commonly craved food (Hill & Heaton-Brown, 1994; Hill *et al.*, 1991; Massey & Hill, 2012; Rozin *et al.*, 1991; Weingarten & Elston, 1991) and previous studies have also demonstrated effects of inhibition training on consumption in this sample, when they scored highly on measures of dietary restraint (Houben & Jansen, 2011). A sample of chocolate cravers also ensures a group of participants who frequently consume the food towards which

inhibition training is targeted; this is important as frequency of consumption has previously been shown to moderate the effects of training on food choice behaviour (Veling *et al.*, 2013a). Dietary restraint, based on the Restraint Scale (RS; Herman & Polivy, 1980; see Chapter 2), is another important consideration with this type of research as it has not only been shown to be an important moderator for effects of training on food consumption (Houben & Jansen, 2011; Veling *et al.*, 2011), but it may also account for findings showing a moderating role of baseline inhibitory control (Houben, 2011). Using the same measure of response inhibition, restrained eaters have demonstrated a reduced ability to stop an on-going motor response on this task, compared to unrestrained eaters (Nederkoorn *et al.*, 2004). Moreover, they have also been shown to demonstrate strong implicit attitudes towards high calorie foods (Hoefling & Strack, 2008; Houben *et al.*, 2010a, 2012c). Individuals who score highly on the RS therefore show both an inability to inhibit responses and a strong impulsive desire towards palatable foods.

Prior to the study participants were asked to refrain from eating for three hours before coming into the lab; therefore controlling for the fourth potential moderator of appetite (Veling *et al.*, 2013a, 2013b). Participants were randomly allocated to either an inhibition training or control group. Those in the stop group performed a stop-signal task (SST) in which they had to inhibit their responses to chocolate stimuli, whereas those in the control group performed the same task but had to make an additional response on chocolate trials (double-response group). As the presentation of the stop signal in the stop training task requires not only response inhibition but also additional error monitoring, attentional control and action updating processes, this double-response task was believed to be an appropriate control task (Tabu, Mima, Aso, Takahashi & Fukuyama, 2011; Verbruggen, Aron, Stevens & Chambers, 2010; Verbruggen *et al.*, 2012; Wessel *et al.*, in press). Furthermore, these training tasks were based on the same training procedure used by Lawrence *et al.* (under review). Following training participants performed two unipolar, Single-Category, Implicit Association Tests (SC-IAT; Greenwald *et al.*, 1998; Houben *et al.*, 2010a; Karpinsky & Steinman, 2006) to measure positive and negative implicit attitudes towards chocolate. It was hypothesised that participants in the stop group would show a reduced positive implicit attitude and/or an increased negative implicit

attitude towards chocolate, compared to the double-response group. Following the SC-IAT, participants completed a bogus taste test. It was expected that those in the stop group would consume significantly fewer calories in the taste test than those in the double-response group.

In addition to these main hypotheses, I was also interested in the specificity of any effects. In a within-subjects design, Houben (2011) demonstrated that the effect of training on the consumption of different foods was dependent on whether the foods were associated with stopping or responding during the training task. Similarly, Lawrence *et al.* (Study 2, under review) found a significant reduction in food intake following inhibition training only for the food that was paired with inhibition (on a majority of trials) during training in a sample of restrained eaters. Veling *et al.* (2013a) also found that the effect of inhibition training on food choice was restricted to the foods that were presented alongside stop-signals in the training task; there was no transfer for the effect of training on choice behaviour to novel foods that were presented in the choice task but not during training. The current study therefore included images of crisps in the training task that were only presented alongside stop-signals on a minority of trials (12.5% compared to 87.5% for chocolate images), and also presented participants with a bowl of crisps in the taste test. It was expected that the stop group would show reduced consumption for the chocolate only. However, any effect of training on crisp intake could imply the occurrence of underlying mechanisms other than stimulus devaluation such as an increase in general self-control or response inhibition (Badry *et al.*, 2009; Baumeister *et al.*, 2006; Berkman *et al.*, 2009, 2012; Cai *et al.*, 2012; Muraven, 2010; Muraven & Baumeister, 2000; Muraven *et al.*, 1999; Verbruggen *et al.*, 2012; Wessel *et al.*, 2013). The specificity of training effects on stimulus devaluation was also investigated using the SC-IAT. During this task participants were presented with images of chocolate that were either identical to those used in the training procedure or novel. A stronger attitude bias for repeated images would suggest that any effect of inhibitory control training on stimulus devaluation may be due to the specific stimulus-stop associations; a transfer of effect to the novel images however would suggest that devaluation effects may generalise to other stimuli within a wider category (e.g. chocolate).

3.2. Method

Figure 3.1 provides a schematic diagram of the study procedure.

Scales	Training Task	SC-IATs	Taste test & questionnaires	Eating-related questionnaires & debrief
Hunger (VAS) Mood (PANAS)	Stop or Double-response training	Positive and negative single-category implicit association test	Bogus taste test (with chocolate & crisps) & non-eating questionnaires	DEBQ G-FCQ-T Debrief BMI
5 mins	20 mins	10 mins	20 mins	5 mins

Figure 3.1. Schematic diagram of the study procedure. Participants answered measures of hunger and mood states (section 3.2.3) before starting the training task (section 3.2.2.1). Participants were randomly allocated to either the stop (inhibition) or double-response (control) training. Following this task participants were presented with two SC-IATs to measure positive and negative implicit attitudes towards chocolate (in a counterbalanced order; see section 3.2.2.2). After the computer tasks participants were presented with a bowl of chocolate buttons and a bowl of crisps (section 3.2.2.3) and were asked to fill out a taste test questionnaire and a battery of personality questionnaires (section 3.2.2.4) while they were left alone for 20 minutes. After 20 minutes had elapsed the food was removed and participants were asked to complete eating-related questionnaires before being debriefed (section 3.2.3).

3.2.1. Participants

One hundred and thirty six participants (127 females; aged 18-61, $M=23.03$, $SE=0.71$) were semi-randomly divided into the stop ($n=68$, 63 females) and double-response groups ($n=68$, 64 females) trying to keep age and gender evenly distributed (sample size was determined according to a Bayes analysis for the main effect of total calorie intake between groups; see section 3.2.4. below). Participants were recruited through posters, electronic advertisements and an online experimental

management system at Cardiff University. They were screened via email using the Attitudes to Chocolate Questionnaire Craving Scale (ACQC; Benton *et al.*, 1998) and the Restraint Scale (RS; Herman & Polivy, 1980) at least one week prior to the study. Participants who were high scoring chocolate cravers (10+ on ACQC; $M=18.79$, $SE=0.49$) and classified as restrained eaters (15+ on RS; $M=19.29$, $SE=0.27$) were included in the study. Participants were not eligible if they were currently dieting (with a weight goal and timeframe in mind) or if they had any history of eating disorders. In accordance with previous studies (Massey & Hill, 2012) a distinction was made between dieting to lose weight and being mindful of not gaining weight; participants were only excluded if they fell into the former category. All participants were reimbursed for their participation; they received course credit or were offered a choice between payment (£6) and entry into a prize draw for a shopping voucher (1 in 100 chance of winning £100). The study was approved by the School of Psychology Research Ethics Committee, Cardiff University.

3.2.2. Materials/ Measures

3.2.2.1. Training Task

The training task was programmed in Matlab (Mathworks, Natick, MA) using Psychophysics Toolbox (www.psychtoolbox.org); all stimuli were presented on a 19-inch flat-panel LCD monitor against a white background. The task consisted of ten blocks of 48 trials and lasted approximately 20 minutes. In each trial a rectangle appeared in the centre of the screen (fixation; 500 ms), within that rectangle a stimulus appeared on either the left or right hand side and remained on screen for 1500 ms (see Figure 3.2). Participants were asked to respond to the location of the stimulus (no-signal trial), as quickly and accurately as possible, using their left and right index fingers ('C' or 'M', respectively, on a standard keyboard). Stimuli consisted of eight images of chocolate, eight images of crisps and 32 filler images (household items). The images of chocolate included solid milk and dark chocolate bars or buttons (see Appendix 3). Food images were selected on the basis that there were no additional ingredients (such as nuts or caramel) or packaging. Efforts were made to match the stimuli as closely as possible for size, shape and visual

complexity (for example, a single bar of chocolate was matched to a single crisp and chocolate buttons were an appropriate match for a portion of crisps). All images were close up shots of the item presented against a white background. Each image was presented once per block and the stimulus type and location of the image were randomly intermixed with equal probability.

Participants were informed that on a subset of trials the rectangle would turn bold after a variable delay (stop-signal delay; SSD), thus indicating a signal trial. Their instructions for signal trials depended on the training condition. Participants in the stop group were informed that they must try to withhold their responses when a signal occurred, whereas those in the double-response group were told that they must make an additional response by pressing the space bar with their thumb. The SSD was initially set at 250ms and was then continuously adjusted using a simulated tracking procedure. If the location response on a no signal trial was less than the SSD plus an estimation of the reaction time to the stop signal (200 ms) the SSD on the following signal trial was decreased by 25 ms; if the response exceeded this time the SSD was increased by 25 ms. A minimum and maximum SSD were also set so that the signal always occurred at least 50ms after the onset of a trial, or 50ms before the end of a trial.

Signals were presented on 25% of trials, with the majority occurring during the presentation of a chocolate stimulus (7/8; 87.5%) and the minority mapped onto the crisp (1/8; 12.5%) and filler (4/32; 12.5%) images. The large number of signals on chocolate trials was to encourage associative learning between chocolate and inhibition in the stop group. At the end of every block participants were given a ten second break and were provided with feedback. Those in the stop group were asked to speed up or slow down their responses depending on their stop performance; those in the double-response condition were only informed if they had missed more than three double-responses, otherwise participants were shown the message “Good”. The researcher was present throughout the training phase and observed the first block to ensure that the participant understood the task instructions and was responding correctly on signal trials.

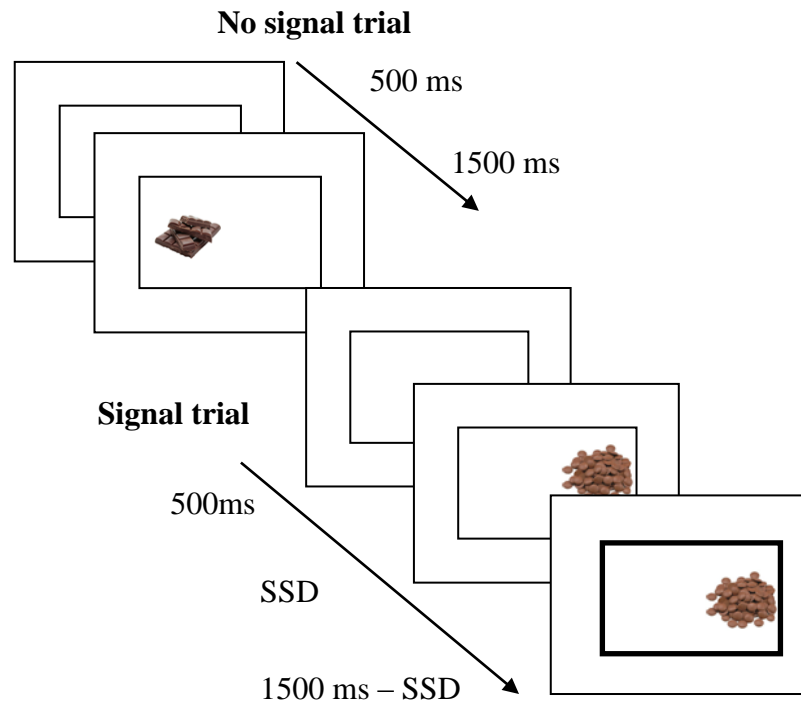


Figure 3.2. Display sequence for the training task. For no-signal trials participants were presented with a rectangle for fixation before seeing a stimulus appear on the left or right hand side. Participants were instructed to respond to the stimulus location using the ‘C’ and ‘M’ keys, with their left and right index fingers, respectively. For signal trials, the lines of the rectangle turned bold after a variable delay (SSD), which was initially 250 ms and was then adjusted using a simulated tracking procedure (see Method). On signal trials, participants were asked to either inhibit their response (stop group) or make an additional response (double-response group).

3.2.2.2. *Unipolar, Single-Category Implicit Association Test (SC-IAT)*

All participants performed two unipolar SC-IATs (Greenwald *et al.*, 1998; Houben *et al.*, 2010a; Karpinsky & Steinman, 2006): a unipolar positive SC-IAT and a unipolar negative SC-IAT. For both tasks the target category was chocolate (six images of chocolate; label ‘chocolate’). Three of the chocolate images were the same as those used in the training procedure (image type: old) and the remainder had not been seen before (image type: new). For the positive SC-IAT the attribute categories were pleasant (words: delicious, delightful, great, heavenly, outstanding, tasty; label ‘pleasant’) and neutral (words: adequate, average, general, moderate, ordinary, undefined; label ‘neutral’), for the negative SC-IAT the attribute categories were

unpleasant (words: awful, bad, disgusting, horrible, nasty, revolting; label ‘unpleasant’) and neutral. Words were selected and matched as closely as possible for scores on concreteness, familiarity, imaginability, number of syllables, verbal frequency (Brown verbal frequency) and written frequency (Kucera-Frances and Thorndike-Lorge written frequency measures) according to the MRC Psycholinguistic Database (see Appendix 4⁴).

Both tasks consisted of three blocks. The first block was for participants to practice categorising the attribute categories (pleasant and neutral for the positive SC-IAT and unpleasant and neutral for the negative SC-IAT) for 24 trials. Participants were instructed to categorise the words as quickly and as accurately as possible using their left and right index fingers (‘C’ and ‘M’ response keys on a standard keyboard). In the second block, chocolate stimuli were paired with one of the attribute categories and were categorised using the same response keys (e.g. pleasant + chocolate vs neutral in the positive SC-IAT). The response assignment of the target category was then reversed in the third block (e.g. pleasant vs neutral + chocolate). There were 72 trials in both the second and third blocks. A 5:2:5 ratio was used to keep the number of responses on each key comparable, so that chocolate images were repeated five times (30 trials), attributes paired with chocolate were repeated twice (12 trials) and attributes not paired with chocolate were repeated five times (30 trials). Each block was preceded by a set of instructions regarding the appropriate responses. Attribute labels were presented throughout the blocks to the bottom-left and bottom-right of the screen and all stimuli appeared centred on the screen (see Figure 3.3). All stimuli remained on screen until a response was given, or for 1500ms. If participants failed to respond within that time the message ‘too slow!’ appeared for 500ms. Participants were provided with feedback after every trial for 150ms (a green or red circle appeared in the centre of the screen for correct and incorrect responses, respectively).

The order of the SC-IATs was counterbalanced across participants (positive-negative or negative-positive). The assignment of the target category was also counterbalanced so that half the sample received the congruent condition (pleasant +

⁴ http://websites.psychology.uwa.edu.au/school/MRCDatabase/uwa_mrc.htm

chocolate *vs.* negative + chocolate *vs.* neutral) followed by the incongruent condition (neutral + chocolate *vs.* pleasant or unpleasant), whereas the other half received them in the reverse order. The assignment of the attribute categories to response keys was also counterbalanced. The tasks were programmed in Matlab (Mathworks, Natick, MA) using Psychophysics Toolbox (www.psychtoolbox.org) and were presented on a 19-inch flat-panel LCD monitor.

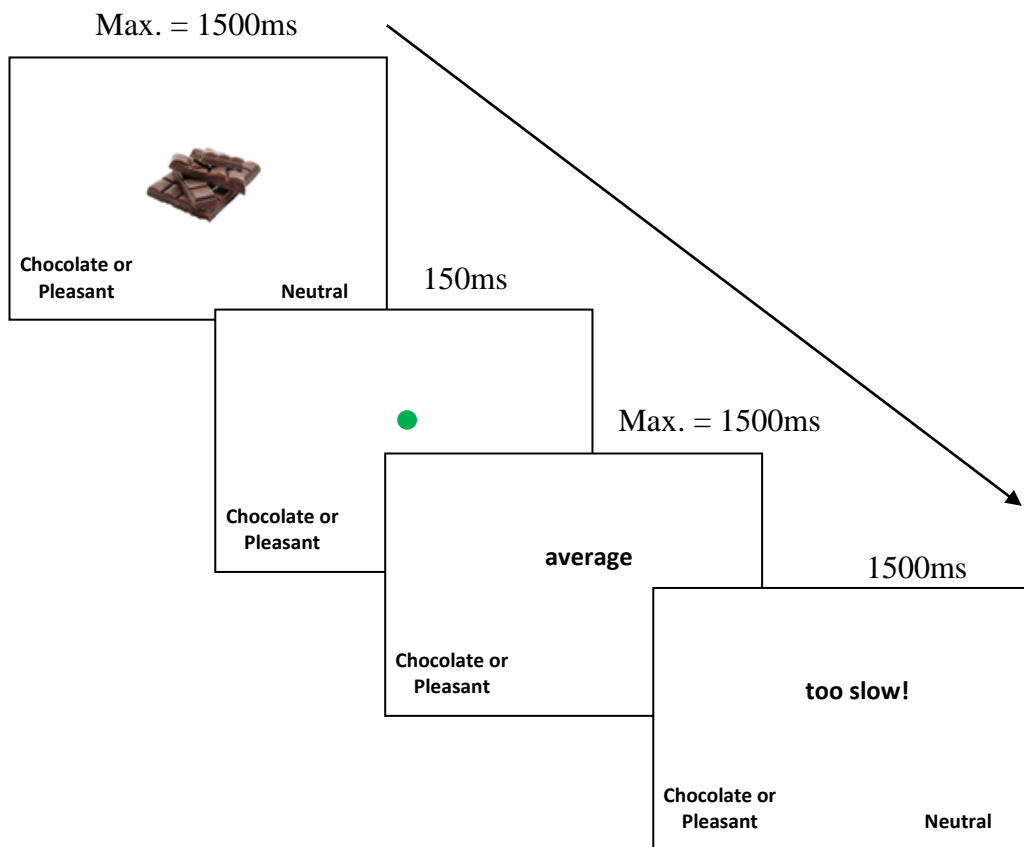


Figure 3.3. Display sequence for the Single-Category Implicit Association Test (SC-IAT). The sequence shows two trials from the positive, congruent block in which pleasant words are paired with chocolate. For the negative condition, pleasant words would be replaced with unpleasant words (along with category labels) and for the incongruent blocks, images of chocolate would be paired with neutral words. The figure shows a chocolate image trial, the correct feedback stimulus (this would follow a ‘C’ response for category label Chocolate or Pleasant), a neutral word trial and a speeded response feedback stimulus (presented for missed trials).

3.2.2.3. Taste Test

Food consumption was measured using a bogus taste test. Participants were presented with two bowls containing milk chocolate buttons ($\pm 210\text{g}$; $M=212.53$, $SE=0.25$; Tesco milk chocolate buttons) and ready salted crisps ($\pm 100\text{g}$; $M=100.7$, $SE=0.24$; Tesco ready salted crisps) and a cup of water ($\pm 150\text{g}$; $M=151.74$, $SE=0.71$). The weight of the foods presented were determined according to their appearance in two identical clear plastic bowls and were the same amounts used in previous research (Lawrence *et al.*, under review). Participants were told that I was interested in how their taste perceptions influenced the data and were invited to consume as much as they liked as the food would be thrown away after the study. They were provided with a questionnaire containing open-ended questions related to the taste of the products and Likert scales measuring the palatability and frequency of consumption for the two foods (e.g. they were asked about the saltiness and sweetness of the product; this questionnaire was identical to that used in previous studies; Lawrence *et al.*, under review; Houben, 2011). Participants were then left alone with the foods for 20 minutes while they completed a battery of personality questionnaires (these were non-eating questionnaires; see Questionnaires below). Participants were left participants alone in a lab room, without windows, for the duration of the taste test to minimise social influences on food intake (e.g. Roth, Herman, Polivy & Pliner, 2001). Furthermore, the cover story of a taste test was used to encourage participants to eat something (Lawrence *et al.*, under review) and also to reduce participants' awareness that food intake was being measured (Robinson, Kersbergen, Brunstrom & Field, 2014). The food products were weighed before and after the taste test without the participants' knowledge. The difference in weight was then converted to calories by multiplying the weight by the caloric density of the food.

3.2.2.4. Questionnaires

Participants were asked to complete a series of filler questionnaires that were provided to occupy the participant whilst leaving them alone with the food for 20 minutes. These questionnaires consisted of the following: The Big Five Inventory (BFI-44; John, Naumann & Soto, 2008), a 44 item questionnaire measuring the Big

Five personality traits (openness, conscientiousness, extraversion, agreeableness and neuroticism); the Brief Self Control Scale (BSCS; Tangney *et al.*, 2004), a 13 item questionnaire assessing dispositional self control (e.g. “I wish I had more self discipline”); the Emotion Regulation Questionnaire (ERQ; Gross & John, 2003), a 10 item questionnaire relating to the use of cognitive reappraisal and expressive suppression as strategies for emotion regulation; the UPPS impulsive behaviour scale (Whiteside & Lynam, 2001), a 45 item questionnaire which measures different facets of impulsivity (premeditation, urgency, sensation seeking, and perseverance); the Attentional Control Questionnaire (ACQ; Derryberry & Reed, 2002), a 20 item questionnaire measuring the ability to focus and shift attention; and the Mood and Anxiety Symptom Questionnaire (MASQ-62; Watson, Clark, Weber, Assenheimer, Strauss & McCormick, 1995), a 62 item questionnaire which asks participants to rate the extent of certain feelings throughout the last week (e.g. “Felt sad”, “Felt tense or ‘high strung’”).

After 20 minutes when the questionnaires and foods were collected, participants were asked to complete two further food-related questionnaires. These were the Dutch Eating Behaviour Questionnaire (DEBQ; Van Strien *et al.*, 1986a) and the General Food Craving Questionnaire – Trait version (G-FCQ-T; Cepeda-Benito *et al.*, 2000; Nijs *et al.*, 2007; see section 2.2.2.6 for full details). These questionnaires were included to examine test-retest reliability for the DEBQRE – to ascertain whether inhibition training and food consumption may have influenced restraint scores in Lawrence *et al.* (under review), and to explore differences in food craving between different measures of dietary restraint (see Study 1).

3.2.3. Procedure

In order to control for levels of appetite, participants were asked to eat something small three hours prior to the study and then to refrain from eating during this time (except water). This approach was consistent with previous studies (Guerrieri *et al.*, 2009; Lawrence *et al.*, under review) and was used to increase food appetite and motivation (Gibson & Desmond, 1999; Veling *et al.*, 2013a, 2013b). Testing therefore only took place between 12-7pm; this timeframe also coincides with an increase in food cravings (Hill *et al.*, 1991). After giving consent participants

answered questions regarding their hunger and mood states. They completed three 100 mm visual analogue scales (Flint, Raben, Blundell & Astrup, 2000; Flint *et al.*, 2000; Yeomans, 2000) to assess feelings of hunger (“How hungry do you feel?”, five anchors from left to right: “Not at all hungry”, “A little hungry”, “Moderately hungry”, “Very hungry” and “As hungry as I have ever felt”), fullness (“How full do you feel?”, five anchors from left to right: “Not at all full”, “A little full”, “Moderately full”, “Very full” and “As full as I have ever felt”) and desire to eat (“How strong is your desire to eat now?”, five anchors from left to right: “Not at all”, “A little”, “Moderately”, “A lot” and “Very much”). They then completed the Positive and Negative Affect Schedule (PANAS; Watson, Clark & Tellegen, 1988) to measure their current mood. These measures were taken at this stage as hunger and mood have been shown to be reliable predictors of food intake (Nederkoorn, Guerrieri, Havermans, Roefs & Jansen, 2009a; Tice, Bratslavsky & Baumeister, 2001); it was important therefore to ensure that there were no group differences prior to the taste test. Participants then completed the training task and unipolar SC-IATs before the taste test and questionnaires.

During the debrief participants were probed for knowledge of the study’s aims and stimulus mappings with open-ended, funnelled questions. Specifically, they were asked a) whether they noticed anything in particular in the computer task, b) whether they noticed anything about when they had to stop or make a double response, c) whether they thought the signals were distributed evenly, randomly or were grouped in any way. Although findings are mixed, some studies have reported training effects only for participants who were aware of the experimental contingencies (Attwood *et al.*, 2008; Field *et al.*, 2007, 2009; Hogarth, Dickinson, Hutton, Bamborough & Duka, 2006; Kakoschke *et al.*, 2014). Furthermore, a moderating role of awareness also has theoretical implications for whether training effects are due to controlled or automatic inhibition (see Verbruggen *et al.*, in press). Participants were also asked whether they thought that performing the first task had any influence on the second task, the taste test or questionnaires. They were then asked if they had participated in any related studies, if they were currently dieting, if they had any history of eating disorders and at what time they last ate to allow for exclusions based on these criteria. Checks for these factors were made prior to testing but were asked again

during the debrief for clarification. At the end of the study participants' height and weight was measured to calculate BMI (kg/m²).

3.2.4. Statistical Analysis

The following demographic, state and trait variables were analysed to ensure that there were no statistically significant differences between training groups: gender ratio, age, BMI, chocolate craving (ACQC), dietary restraint (RS), hunger (VAS measures), mood (PANAS), food palatability ratings, food consumption ratings and hours since last food consumption. As the majority of continuous variables were not normally distributed, and could not be normalised with either a square root or log transformation, nonparametric Mann Whitney U tests were performed.

Data from the training task were also analysed to ensure that participants were performing the task as expected. These analyses were used for participant exclusions and all criteria were decided prior to hypothesis testing. The three variables of interest were the percentage of erroneous responses on signal trials (failed inhibition for the stop group and failure to execute both responses in the double-response group; these errors also included trials in which an incorrect location response was made), the mean reaction time for no-signal trials (GoRT) and the percentage of errors on no-signal trials (including incorrect location responses and missed responses). Participants were excluded if their accuracy on no-signal trials was <85%, their percentage errors on signal trials was >3SDs from the group mean or their GoRT was >3SDs from the group mean. Two participants, one from each training condition, were excluded due to <85% accuracy on no-signal trials and two participants from the double-response condition were excluded because their GoRTs exceeded >3SDs from the group mean. This resulted in a final sample of 132 participants: 67 in the stop group and 65 in the double-response group. A sensitivity analysis (using G*Power; Faul *et al.*, 2007) for the main interaction between training condition and food type revealed that this sample had 80% power to detect a minimum effect size of $f=0.22$ for the main effect of training condition and 80% power to detect an effect size $f=0.11$ for the interaction ($\alpha=0.05$, number of groups =2, number of repetitions =2, correlation among repeated measures =0.58 (calculated post hoc based on the correlation between chocolate and crisp consumption), non-

sphericity correction =1 (i.e. no correction for violations of sphericity with only two levels of the dependent variable)).

For the primary dependent variable of food consumption, outliers were considered as values $>3SDs$ from the group mean for each food type separately. Outlier values were replaced with the nearest non-outlier value +1; this method reduces the impact of a univariate outlier while maintaining the score as the most deviant (Tabachnick & Fidell, 2007). For the consumption data a 2x2 mixed analysis of variance (ANOVA) was performed with the between subjects factor of *training condition* (stop or double-response) and the within-subjects factor of *food type* (chocolate and crisps).

Two unipolar SC-IATs were used to measure the second dependent variable of implicit attitudes. Participants performed the SC-IAT tasks well – only one exclusion was made according to the pre-set criterion of a 20% error rate (Karpinsky & Steinman, 2006). One participant in the stop group was excluded from the unpleasant SC-IAT analysis resulting in a sample size of 66 for this analysis. IAT effects were calculated using a scoring algorithm modelled on the *D*-score algorithm for the IAT (Greenwald, Nosek & Banaji, 2003) and was the same method used by Karpinsky and Steinman (2006). All data from practice blocks were discarded, along with non-responses and responses that were less than 350ms or more than 1000ms. Errors were replaced with the block mean plus an error penalty of 600ms (the higher penalty of 600ms was used in accordance with Houben *et al.*, 2010a, 2011a, 2012a, 2012c). The average response time in the congruent block was subtracted from the average response time in the incongruent block and the result was divided by the standard deviation of all correct response times in both blocks. Higher scores therefore indicate a faster performance on the congruent block relative to the incongruent block, whereas negative scores would reflect the reverse. One-sample t-tests were calculated for the positive and negative bias for each group to see whether attitude scores were significantly different from zero. A 2x2 mixed ANOVA with the between-subjects variable of *training condition* (stop or double-response) and the within-subjects variable of *SC-IAT* (positive and negative) was then performed to see whether attitudes were significantly different according to training condition. The

stimulus-specific effect of training on implicit attitudes was then explored with comparisons between novel chocolate images and those presented during training. One-sample t-tests and a 2x2x2 mixed ANOVA with the between subjects factor of *training condition* (stop or double-response) and within subjects factors of *SC-IAT* (positive or negative) and *image type* (old and new) were calculated.

All results are reported with unadjusted significance values; corrections for multiple comparisons were calculated for all within-test analyses and are only reported where these corrections changed the interpretation of an analysis from statistically significant to non-statistically significant. All statistical analyses were performed with SPSS.

Bayes factors for total food consumption were also calculated for two reasons. Firstly, Bayesian hypothesis testing allows for a flexible stopping rule on data collection without correcting for the elevation of Type I error, as would be required under a frequentist approach (Dienes, 2011, 2014). Secondly, unlike null hypothesis significance testing, Bayes factors can be used to interpret null findings (Dienes, 2011, 2014). A Bayesian analysis can be used to determine the extent to which a result provides evidence for the alternative hypothesis (H1), in this case that inhibition training reduces food consumption compared to control training, or evidence for the null hypothesis (H0; i.e. there is no effect of inhibition training on food intake) according to theoretical predictions or *prior odds*. The prior odds can be defined as the probability of the alternative hypothesis being true over the probability of the null hypothesis being true ($P(H1)/P(H0)$) and can be determined according to theoretical predictions or previous results.

Once data has been collected, the *likelihood* of each hypothesis can be calculated. The likelihood is the probability of obtaining that exact data given the hypothesis ($P(D|H)$). The Bayes factor (B) therefore is the ratio of likelihoods for the two hypotheses:

$$B = \frac{\text{likelihood H1 } (P(D|H1))}{\text{likelihood H0 } (P(D|H0))}$$

Bayes factors can therefore range from 0 to infinity with B values greater than 1 providing evidence for the alternative hypothesis and values less than 1 providing evidence for the null hypothesis. For interpretation, values greater than 3 suggest ‘substantial’ evidence for the alternative hypothesis and values of less than 0.33 indicate ‘substantial’ evidence for the null hypothesis (Dienes, 2011, 2014).

To calculate an expected difference in total calorie intake for restrained eaters in the inhibition training group compared to the control group, available results were entered into a Bayesian meta-analysis (Dienes, 2014⁵). These results were obtained from Houben (2011; the difference in consumption between the go and stop foods for individuals with low inhibitory control; 25.59 kCals), Veling *et al.* (2011; study 2; the difference in consumption between control and no-go groups for chronic dieters; 179.46 kCals) and Lawrence *et al.* (under review; the difference in total consumption between double-response and stop groups for participants with high dietary restraint; 60.09 and 142.01 kCals for studies 1 & 2, respectively). The results of this meta-analysis gave an estimated difference of 49.19 kCals.

To calculate the Bayes factor, following Dienes (2011, 2014), this value was entered as the standard deviation in a half-normal distribution with a mean value of 0; a half-normal distribution was used as smaller effects were considered more probable than larger effects. For the sample mean, a between-subjects t-test was performed for the effect of training condition (stop or double-response) on total food consumption. The mean difference and standard error of the difference for this comparison were entered into Dienes’ online calculator⁶. The result of this calculation was therefore used to guide data collection; data collection was terminated when the Bayes factor provided substantial evidence for either the alternative hypothesis ($B > 3$) or the null hypothesis ($B < 0.33$).

⁵ http://www.lifesci.sussex.ac.uk/home/Zoltan_Dienes/inference/bayes_normalposterior.swf

⁶ http://www.lifesci.sussex.ac.uk/home/Zoltan_Dienes/inference/bayes_factor.swf

3.3. Results

3.3.1. Group Differences

Nonparametric Mann Whitney U tests revealed that there were no significant differences between training groups for measures of age, chocolate craving (ACQC) dietary restraint (RS), BMI and hours since food consumption (all $ps > 0.33$, all $rs < 0.08$; Table 3.1.). The gender ratio was also similar for both groups with five males in the stop group and 4 males in the double-response group; due to the small number of males in each condition a Chi square analysis was not conducted. There were also no significant differences in state measures of hunger (VAS measures) and mood (PANAS; all $ps > 0.24$, all $rs < 0.1$). Participants also reported similar levels of palatability and frequency of consumption for both foods in the taste test (all $ps > 0.34$, all $rs < 0.08$).

3.3.2. Training Data Analysis

The training data show that on average, participants in the stop group incorrectly responded on 45.46% of signal trials ($SE=0.79$; see Figure 3.4a.). This shows that the tracking procedure and feedback were working appropriately to ensure that the probability of responding was ~50%; at this level of performance the stop and go processes are believed to be at a point of maximum competition (Logan *et al.*, 1997). On average, participants in the double-response group made errors on 4.63% ($SE=0.5$; see Figure 3.4b.) of signal trials, demonstrating that they performed the task correctly. For the GoRT there was a significant difference between the two groups, with participants in the stop group ($M=499.27$, $SE=12.45$) responding significantly slower than participants in the double-response group ($M=381.76$, $SE=6.82$; $t(102.05)=8.07$, $p < 0.001$, $d=1.63$; see Figure 3.4c.). This is consistent with proactive slowing as a result of inhibition in the stop group (Verbruggen & Logan, 2009b). Accuracy of performance on no-signal trials was high for both the stop (% errors: $M=1.39$, $SE=0.24$) and double-response (% errors: $M=1.69$, $SE=0.24$) groups ($t(130)=0.89$, $p=0.38$, $d=0.15$; see Figure 3.4d.).

Table 3.1. Group characteristics for gender distribution and mean age, BMI, chocolate craving score (ACQC) restraint score (RS), hours since last food consumption, hunger scores (VAS measures for hunger, fullness and desire to eat), positive and negative affect scores (PANAS) and palatability and frequency of consumption for chocolate and crisps (SE within parentheses).

	Stop (<i>n</i> =67)	Double-response (<i>n</i> =65)	<i>U</i> =	<i>p</i> =
Gender (% female)	92.5	93.8		
Age	23.12 (1.05)	22.18 (0.77)	2074	0.63
BMI	24.39 (0.57) ²	24.81 (0.59) ²	1977.5	0.74
ACQC	18.91 (0.74)	18.52 (0.68)	2123.5	0.81
RS	18.85 (0.3)	19.72 (0.45)	1965	0.33
Hours since food	4.94 (0.36) ¹	5.48 (0.49)	2109.5	0.87
Hunger	5.45 (0.22)	5.43 (0.22)	2115	0.78
Fullness	1.51 (0.18)	1.42 (0.18)	2057.5	0.58
Desire to eat	6.14 (0.26)	5.8 (0.26)	1917.5	0.24
Positive affect	25.55 (0.83) ²	25.29 (0.75)	2060.5	0.81
Negative affect	13.06 (0.47) ²	13.15 (0.49)	2110.5	0.99
Chocolate palatability	8.84 (0.16) ¹	8.6 (0.18) ¹	1916.5	0.34
Chocolate consumption freq.	4.78 (0.07)	4.74 (0.07)	2116.5	0.68
Crisp palatability	7.13 (0.27) ¹	7.33 (0.24) ¹	2094	0.93
Crisp consumption freq.	3.52 (0.18)	3.78 (0.13)	2087.5	0.67

Note. Superscript denotes the number of participants missing for that variable. ACQC= Attitudes to Chocolate Craving subscale; RS=Restraint Scale; BMI=body mass index

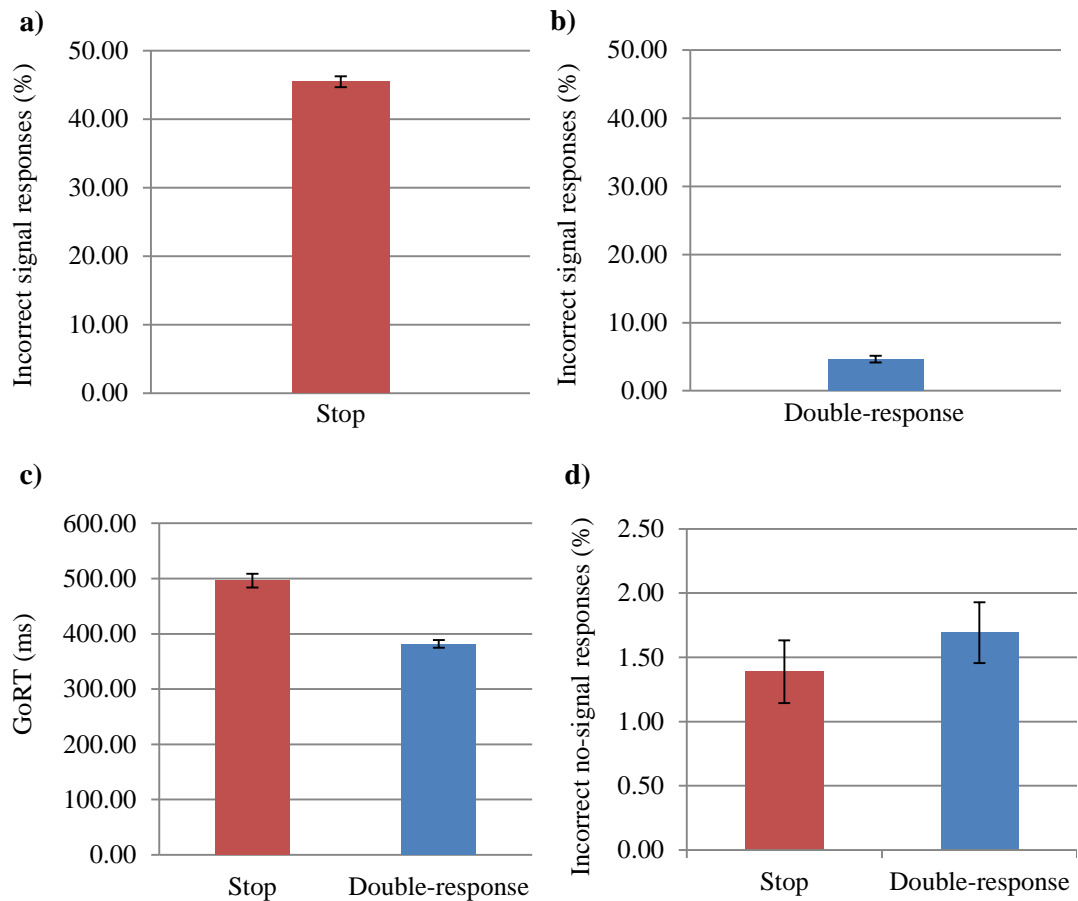


Figure 3.4. Training data showing mean percentage of incorrect responses on a signal trial for the stop group (a); mean number of failed dual responses in the double-response group (b), mean reaction time for no-signal trials (GoRT; c) and mean percentage of incorrect responses on no-signal trials (d). Error bars show $\pm 1SE$.

3.3.3. Consumption Data Analysis

The results for food consumption are presented in Figure 3.5. To correct for positive skew in the consumption data a square root transformation was performed and the statistical analysis was performed on this data; for ease of interpretation all means and standard errors are reported for non-transformed calorie intake. A 2x2 mixed ANOVA revealed a significant main effect of food type showing that participants ate significantly more calories from chocolate ($M=221.78$, $SE=14.01$) than crisps ($M=154.84$, $SE=10.07$; $F(1,130)=8.86$, $p<0.001$, $\eta^2_p=0.18$, $f=0.47$). However, the main effect of training condition ($F(1,130)=1.34$, $p=0.25$, $\eta^2_p=0.01$, $f=0.1$) and the interaction between training condition and food type ($F(1,130)=1.43$, $p=0.23$,

$\eta^2_p=0.01, f=0.1$) were both non-significant. Contrary to the primary hypothesis, the *total* calorie consumption in the stop group ($M=399.8; SE=29.21$) was greater than that for the double-response group ($M=343.39; SE=28.69$). Due to the direction of results, a Bayesian analysis (mean difference = -56.42, SE of the difference = 40.96) revealed a Bayes factor of 0.32, indicating substantial evidence for the null hypothesis ($B<0.33$; Dienes, 2011, 2014).

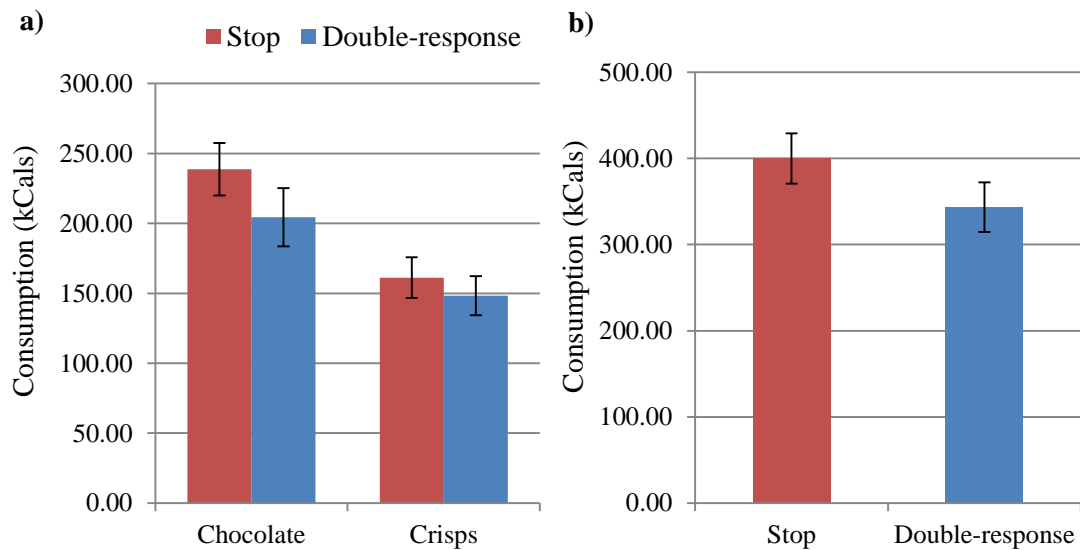


Figure 3.5. Food consumption in calories for the stop and double-response groups: **a)** according to food type (chocolate was paired with a signal on 87.5% of trials and crisps were paired with a signal on 12.5% of trials) and **b)** for total calorie intake. Error bars show $\pm 1SE$.

3.3.4. Unipolar, SC-IAT Data Analysis

For the positive/pleasant SC-IAT, one-sample t-tests (test value of 0) revealed statistically significant positive attitudes towards chocolate for the stop ($M=0.49; SE=0.07; t(66)=7.36, p<0.001, dz=0.9$) and double-response ($M=0.38; SE=0.06; t(64)=6.58, p<0.001, dz=0.81$) groups. For the negative/unpleasant SC-IAT, neither the stop ($M=-0.01; SE=0.06; t(65)=0.27, p=0.79, dz=0.02$) nor the double-response group ($M=-0.09; SE=0.05; t(64)=1.59, p=0.12, dz=0.2$) showed a significant attitude towards chocolate (see Figure 3.6). Contrary to expectation, the mean score on the positive SC-IAT was greater for the stop group compared to the double-response

group, whereas, the scores on the negative SC-IAT were in the expected direction. A 2x2 mixed ANOVA, however, revealed no significant main effect of training condition ($F(1,129)=1.88, p=0.17, \eta^2_p=0.01, f=0.12$), and importantly, no significant interaction between training condition and SC-IAT ($F(1,129)=0.08, p=0.78, \eta^2_p=0.001, f=0.03$). There was a significant main effect of SC-IAT with a positive score on the positive/pleasant SC-IAT ($M=0.43; SE=0.04$) and a negative score on the negative/unpleasant SC-IAT ($M=-0.05; SE=0.04$). With the findings from the one-way t-tests these results indicate that participants demonstrated a significant positive attitude towards chocolate on the pleasant SC-IAT (i.e. the presence of an implicit association between pleasant words and images of chocolate) but did not show a significant attitude in either direction on the unpleasant SC-IAT (i.e. no implicitly held association between unpleasant words and images of chocolate).

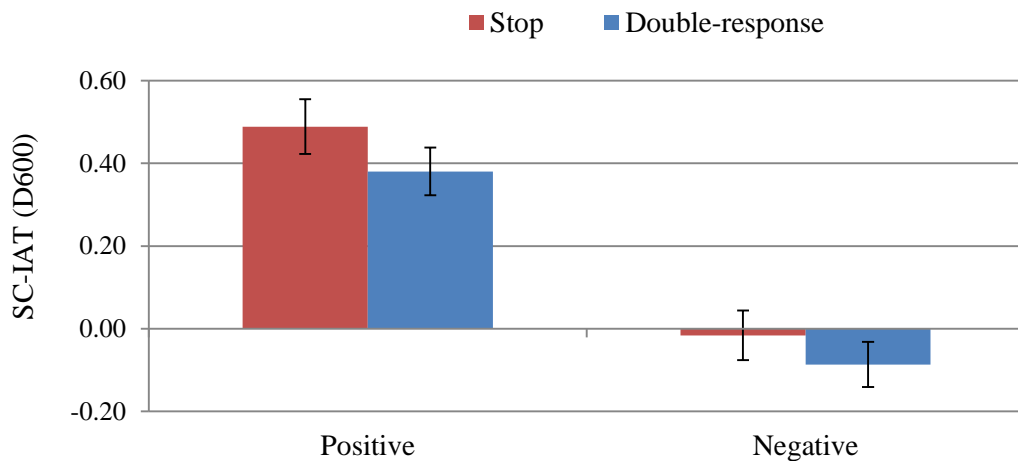


Figure 3.6. Mean bias scores for the pleasant and unpleasant SC-IATs (D600 scoring algorithm) according to training condition. Positive values indicate a faster association for congruent trials (chocolate and pleasant words on the positive SC-IAT and chocolate and unpleasant words on the negative SC-IAT) whereas negative values indicate a faster association in the incongruent trials (chocolate and neutral words). Error bars show ± 1 SE.

Stimulus-specific effects of inhibition training on implicit attitudes were explored with the analysis of trials in which chocolate stimuli appeared (see Figure 3.7). One-sample t-tests (using a test value of 0) revealed that all biases in the positive SC-IAT

were significant, indicating that participants in both groups, and for both old and new image types, were significantly faster to associate images of chocolate with pleasant words than they were with neutral words (all $t_s > 4.42$; all $p_s < 0.001$; all $d_zs > 0.55$). There was also a significant positive bias in the negative SC-IAT for old images in the stop group ($t(65) = 2.43$, $p = 0.02$, $d_z = 0.3$) revealing that participants in the stop group showed a significant association between unpleasant words and images of chocolate that they had previously inhibited responses to. However, this effect was no longer statistically significant after correcting for multiple comparisons ($\alpha/8 = 0.006$). All other effects were non-significant (all $t_s < 1.58$; all $p_s > 0.12$; all $d_zs < 0.19$). The results from the ANOVA revealed a significant effect of SC-IAT, showing that participants associated chocolate more strongly with pleasant words in the positive SC-IAT ($M = 0.63$; $SE = 0.06$) than with unpleasant words in the negative SC-IAT ($M = 0.11$; $SE = 0.05$; $F(1, 129) = 45.39$, $p < 0.001$, $\eta^2_p = 0.26$, $f = 0.59$). There was also a significant main effect of stimulus type with novel chocolate images being more strongly associated with pleasant and unpleasant words than neutral words ($M = 0.42$; $SE = 0.05$) compared to old images ($M = 0.31$; $SE = 0.05$; $F(1, 129) = 4.62$, $p = 0.033$, $\eta^2_p = 0.04$, $f = 0.20$). However, there was no significant main effect of training condition ($F(1, 129) = 1.63$, $p = 0.2$, $\eta^2_p = 0.01$, $f = 0.1$), no significant interaction between SC-IAT and stimulus type ($F(1, 129) = 0.15$, $p = 0.7$, $\eta^2_p = 0.001$, $f = 0.03$) and no significant interaction between condition and stimulus type ($F(1, 129) = 2.63$, $p = 0.11$, $\eta^2_p = 0.02$, $f = 0.14$). Importantly, the interaction between training condition and SC-IAT ($F(1, 129) = 0.09$, $p = 0.77$, $\eta^2_p = 0.001$, $f = 0.03$) and the three-way interaction between training condition, SC-IAT and stimulus-type ($F(1, 129) = 0.89$, $p = 0.35$, $\eta^2_p = 0.01$, $f = 0.1$) were also non-significant.

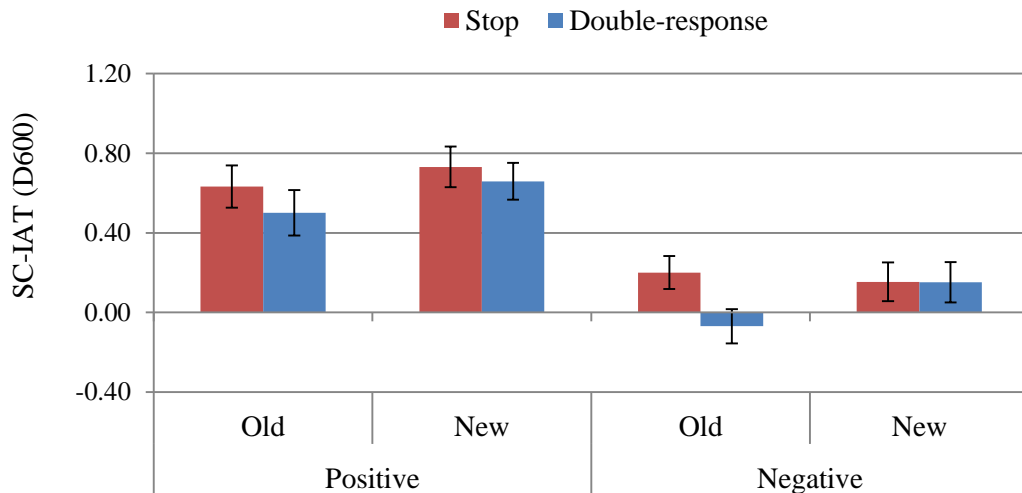


Figure 3.7. Mean bias scores on the positive and negative SC-IATs (D600 scoring algorithm) for the chocolate images that were either presented during training (Old) or novel (New), according to training condition. Positive values indicate a faster association for congruent trials (chocolate and pleasant words on the positive SC-IAT and chocolate and unpleasant words on the negative SC-IAT) and negative values indicate a faster association in the incongruent trials (chocolate and neutral words). Error bars show $\pm 1SE$.

3.3.5. Debrief Analysis

During the debrief participants were probed for awareness of the stimulus mappings using funnelled questions. If they indicated awareness that the majority of signals were mapped onto the chocolate stimuli they were considered ‘aware’, if they mentioned that signals were mapped onto food in general they were considered ‘partially aware’ and if they reported no associations they were considered ‘not aware’. For the whole sample, half of the participants noticed that signals were paired with chocolate (51.1%), 10.7% reported that signals were paired with food in general and 38.2% did not notice any consistent stimulus-signal associations. These values were similar across the two training conditions: 56.7% and 45.3% of the stop and double-response groups were aware, respectively; 7.5% and 14.1% were partially aware for the stop and double-response groups, respectively; and 35.8% and 40.6% were not aware for the stop and double-response groups, respectively. For the chi-square test the aware and partially aware participants were collapsed into one group due to the small number of participants who were partially aware. The test

revealed that the differences between the two groups were not statistically significant ($\chi^2(1)=0.32, p=0.57, \phi=0.05$). Furthermore, a mixed 2x2x2 ANOVA (between subjects factors: *training condition*, stop or double-response and *awareness*, aware or not aware; within subjects factor: *food type*, chocolate and crisps) revealed that awareness of stimulus mappings did not have any discernible effect on food consumption; the main effect of awareness and interactions with awareness were not statistically significant (all $F_s < 1.14$; all $p_s > 0.29$; all $\eta^2_p s < 0.01$).

Importantly, no participants correctly guessed the aim of the study and no participants mentioned an awareness that their food intake was being measured (an awareness that food intake is being monitored can result in floor effects; see Robinson *et al.*, 2014; Roth *et al.*, 2001). On average participants consumed 372 calories ($SE=20.55$) suggesting a moderate degree of intake (with the same taste test procedure and foods used in Lawrence *et al.* (under review) the average intake was ~400 calories). When asked whether they thought that the training had any influence on the food they had, the majority of participants answered that the training had no effect (78%) and a minority of participants reported that the training made them hungry or desire food (22%). These values were similar for the stop and double-response groups: 25.4% and 18.5% reported increased hunger or desire to eat in the stop and double-response groups, respectively. A chi-square test revealed no statistically significant difference between groups ($\chi^2(1)=0.92, p=0.34, \phi=0.08$).

3.4. Discussion

The aim of this study was to investigate whether training inhibitory control towards images of chocolate would influence implicit attitudes towards chocolate and reduce chocolate consumption in a bogus taste test. Consistent with previous research (Houben, 2011; Houben *et al.*, 2011a, 2012a; Houben & Jansen, 2011; Veling *et al.*, 2013b), it was expected that participants in the stop group would consume significantly less chocolate than those in the double-response group, and that this would be mediated by a change in implicit attitudes.

The results revealed a main effect of food in the taste test, with participants consuming significantly more calories from chocolate than crisps. A main effect of SC-IAT task was also found, indicating that participants were faster at associating images of chocolate with pleasant words than they were with unpleasant words. These results are not surprising considering participants were selected on the basis that they scored highly on a measure of chocolate craving. However, there were no significant differences in consumption or attitudes between stop and double-response groups. Although a one sample t-test revealed an indication of a negative bias towards chocolate for the stimuli involved in inhibition training (old images) for the stop group only, this effect did not manifest in a significant interaction in the main analysis. This is only weak evidence that inhibition training may increase negative attitudes towards previously inhibited stimuli compared to control training. Nevertheless, this did not result in a significant reduction in chocolate consumption. Moreover, the results for total calorie intake were in the opposite direction to that predicted, and a Bayesian inferential analysis demonstrated that the results provided substantial evidence for the null hypothesis (Dienes, 2011, 2014). These results are inconsistent with previous studies that have revealed significant effects of inhibitory control training on consumption and implicit attitudes (Houben, 2011; Houben *et al.*, 2011a, 2012a; Houben & Jansen, 2011; Veling *et al.*, 2011).

One possible explanation for the lack of effect on consumption is the intermediate SC-IAT tasks; it is possible that both the time taken to complete the tasks and the nature of the response format in the tasks weakened any training-induced improvements in behavioural control. Previous studies employing a similar design have typically presented participants with the taste test immediately after training (Houben, 2011; Houben & Jansen, 2011). However, the SC-IATs only lasted ten minutes and other studies have found effects of inhibition training on improved self-control over much longer durations (Veling *et al.*, 2011; Verbruggen *et al.*, 2012, Study 3). Although an intermediate IAT has not previously been included in studies investigating the effect of food-related inhibition training, Houben *et al.* (2011a) employed a similar design when training alcohol-related response inhibition. They found that, following training, participants in the inhibition group showed an increase in negative implicit attitudes towards beer on the IAT as well as a statistical

trend for reduced beer consumption in a taste test and a significant decrease in weekly alcohol consumption (using a questionnaire which was completed daily at home; but see Jones & Field, 2013). In a later study investigating whether the effect of training on weekly alcohol intake was mediated by changes in implicit attitudes, Houben *et al.* (2012a) replicated these findings despite having three different intermediate tasks, all of which involved responses towards beer-related images (an IAT, an approach-avoidance task and a SST), suggesting that this effect is fairly robust. Furthermore, Veling *et al.* (2013b) also found that an intermediate task measuring explicit attitudes towards food did not negate the effect of food-related inhibition training on food choice, and even mediated this effect. Together these studies suggest that the lack of an effect of inhibition training on food consumption, shown here, is unlikely to be due to the time delay or response format resulting from the IAT tasks. In addition, as changes in both implicit and explicit attitudes have been argued to underlie the effects of response inhibition training on consumption and choice behaviour, the null effect of inhibition training on food consumption could be explained by the failure to show an effect of training on implicit attitudes. This would imply, therefore, that the training protocol used in the present experiment was insufficient to produce changes in implicit attitudes and subsequently reduce food consumption.

Compared to previous studies showing an effect of inhibition training on attitudes and consumption, for both food and alcohol (Houben, 2011; Houben *et al.*, 2011a, 2012a; Houben & Jansen, 2011; van Koningsbruggen *et al.*, 2013a; Veling *et al.*, 2011, 2013a, 2013b), this training task was fairly long. The number of trials used in other studies has varied from 72 (Veling *et al.*, 2011) to 512 (Lawrence *et al.*, under review, Study 2), whereas this protocol included 480 trials (see Appendix 2). It would be expected therefore that training in the current study was sufficient to produce reliable effects on behaviour. However, the percentage of target food (i.e. the foods that were presented with the majority of signals) trials associated with an inhibition signal and the percentage of total inhibition trials were lower than other studies. Whereas this training procedure was based on the SST and had an overall stop-signal rate of 25% (see also Houben, 2011; Verbruggen *et al.*, 2012), many other studies have shown effects of inhibition training on consumption and attitudes

using the GNG task, which presents a no-go signal on 50% of trials (Houben *et al.*, 2011a, 2012a; Houben & Jansen, 2011; van Koningsbruggen *et al.*, 2013a; Veling *et al.*, 2011, 2013a, 2013b, 2014). However, Veling *et al.* (2013b) found that increasing the number of stimulus-stop pairings during training had no effect on food evaluations, suggesting that a difference in the number of signal trials is unlikely to explain the difference in findings.

Two further differences between the stop-signal training task used in the present study and previous GNG training tasks involve the type of inhibitory process and the consistency of mappings between the stimuli and stop or no-go signals. Firstly, the SST and GNG task have been differentiated based on the form of response inhibition that they require (Eagle, Bari & Robbins, 2008; Schachar, Logan, Robaey, Chen, Ickowicz & Barr, 2007; see Figure 3.8). The SST requires the inhibition of an on-going motor response due to the variable stimulus-onset asynchrony between the stimulus and signal (stop-signal delay; SSD). As the stimulus is presented prior to the signal, the go process has already been initiated and the action must be cancelled during its execution (Logan & Cowan, 1984; Logan *et al.*, 1997; Verbruggen & Logan, 2009a). This form of inhibition has been referred to as ‘action cancellation’ and has been argued to reflect a ‘true’ stopping action (Eagle *et al.*, 2008; Schachar *et al.*, 2007; Wessel *et al.*, in press). The GNG task, on the other hand, presents the stimulus and signal simultaneously so that the stop process is triggered at the same time as the go process. This means that the action is interrupted during the preparation phase; this form of inhibition has been referred to as ‘action restraint’ and is argued to reflect a decision-making paradigm (Eagle *et al.*, 2008; Schachar *et al.*, 2007; Wessel *et al.*, in press). Neuroimaging and stimulation studies have revealed that although these two tasks involve some common neural circuits (e.g. Zheng *et al.*, 2008), they have also been associated with distinct brain regions (e.g. Rubia *et al.*, 2001; Swick, Ashley & Turken, 2011). For example, the inferior frontal gyrus (IFG) has been strongly implicated in the SST (Aron *et al.*, 2003, 2004, 2014; Chambers *et al.*, 2006, 2007; Chevrier, Noseworthy & Schachar, 2007), whereas the GNG task is believed to be more dependent on the DLPFC (Beeli *et al.*, 2008; Garavan *et al.*, 2006; Liddle *et al.*, 2001; Menon, Adleman, White, Glover & Reiss, 2001; Wager, Sylvester, Lacey, Nee, Franklin & Jonides, 2005) – which may reflect

the higher load on decision-making and working memory in some GNG tasks (Criaud & Boulinguez, 2013; Mostofsky *et al.*, 2003; Simmonds *et al.*, 2008).

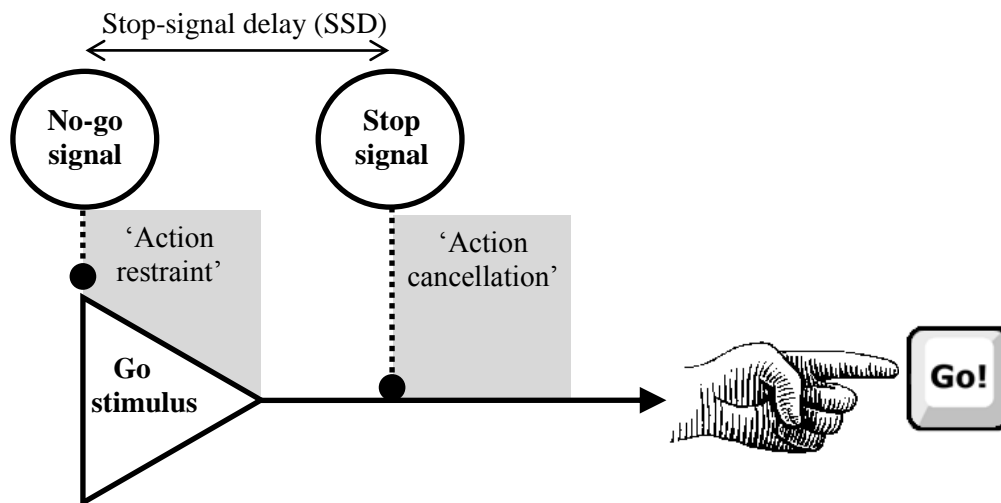


Figure 3.8. Schematic representation of the different inhibitory processes in the go/no-go and stop-signal tasks. In the go/no-go task the presentation of the go stimulus and stop-cue ('no-go signal') is simultaneous, allowing for the stop process to interrupt the action during response preparation – this is referred to as 'action restraint' (Eagle *et al.*, 2008; Schachar *et al.*, 2007). In the stop-signal task the stop-cue ('stop signal') is presented after a variable stop-signal delay (SSD), therefore requiring the inhibition of an already-initiated response – this is referred to as 'action cancellation' (Eagle *et al.*, 2008; Schachar *et al.*, 2007). Figure adapted from Eagle *et al.* (2008).

Secondly, the GNG paradigms previously used to train food-related inhibitory control have all used consistent mapping between the target food and no-go signals (i.e. 100% mapping; Houben & Jansen, 2011; van Koningsbruggen *et al.*, 2013a; Veling *et al.*, 2011; 2013a, 2013b; see Appendix 2), whereas this study included a minority of trials in which participants had to respond to images of chocolate. The consistent mapping in the GNG tasks may have lead to the development of *automatic* inhibitory processes, thereby encouraging fast, bottom-up inhibition and reducing the need for top-down control (Shiffrin & Schneider, 1977; Spierer, Chavan & Manuel, 2013; Verbruggen *et al.*, in press). In a series of experiments, Verbruggen

and Logan (2008) showed that when mappings were consistent, participants were slower and less likely to respond to a stimulus that was previously associated with stopping. Automatic response inhibition is believed to occur when an old stimulus retrieves the stop goal, and as these stimulus-stop associations tend to be more consistent in the GNG task than the SST, automatic inhibition in the GNG task is more likely. Consistent with this idea are findings showing that improvements in inhibitory control as a result of GNG training do not translate to other inhibition or executive function tasks, whereas training on the SST may improve decision-making abilities (Thorell, Lindqvist, Bergman Nutley, Bohlin & Klingberg, 2009; Verbruggen *et al.*, 2012).

Verbruggen and Logan (2008) have also shown that automatic inhibition can occur in the SST when mappings are consistent. This may explain why Houben (2011) found a significant effect of stop-signal training on food consumption as training on this task also used consistent mappings between the target food and no-go signals. Nevertheless, Lawrence *et al.* (under review) also found a significant reduction in food intake following a SST that involved a minority of food-go trials, similar to the training task in the present study. One difference, however, between these two studies was the inclusion of inter-block feedback in the current study; this feedback may explain the lower overall inhibition rate in this study (55%) compared to those in Lawrence *et al.* (76% and 66% in Study 1 and Study 2, respectively). It is likely that encouraging competing go and stop processes in the current training task led to an increased number of failed stops on the chocolate trials – which may have hindered stimulus-stop associations and automatic inhibition from developing (Spierer *et al.*, 2013; Verbruggen & Logan, 2008). Another possibility is that rather than learning an association between the stimulus and the stop goal, participants may have learned an association between the stimulus and the *signal* (Verbruggen *et al.*, in press). If the image of chocolate does not retrieve the stop goal on future encounters, it can be assumed that neither inhibitory processes (neither general nor specific inhibitory control; Berkman *et al.*, 2009, 2012; Chiu *et al.*, 2012; Veling *et al.*, 2011) nor the aversive centre (McLaren & Verbruggen, submitted; Verbruggen *et al.*, in press) will be activated, and therefore neither improved self-control nor stimulus devaluation will occur as a result of training (see Chapter 1 for a full

discussion of potential mediating mechanisms, section 1.3.2.). In conclusion, there are a number of different training parameters that may explain the inconsistent findings between the present study and previous research. However, without comparing these methods within the same study it is difficult to establish which parameters are required for the most effective training protocol. I therefore sought to investigate this in Study 3 by including both a stop-signal training task and a GNG training task.

Chapter 4. Study 3

Training response inhibition to reduce food consumption: A comparison of the stop-signal and go/no-go paradigms

4.1. Introduction

The aim of this study was to again investigate whether training response inhibition could reduce food consumption in restrained eaters. The previous study found evidence for the null hypothesis when training restrained, trait chocolate cravers to inhibit their responses to images of chocolate using a stop-signal task (SST); there was no difference in calorie intake, measured in a bogus taste test, between the inhibition and control groups. These results are inconsistent with earlier studies that have shown an effect of inhibitory control training on decreased food intake and unhealthy snack choices (Houben, 2011; Houben & Jansen, 2011; Lawrence *et al.*, under review; van Koningsbruggen *et al.*, 2013a; Veling *et al.*, 2011, 2013a, 2013b). However, the majority of these studies used a different training protocol, namely the go/no-go (GNG) task, which may explain the difference in findings (see Appendix 1 for a summary of methods and findings). The present study therefore included both the SST and the GNG task, allowing for the effectiveness of these tasks to be compared within the same study. In addition, there were three further aims for the current study; firstly, to investigate the consumption of both healthy and unhealthy foods following training; secondly, to investigate whether these effects were stimulus-specific or whether they would generalise to novel foods not presented during training; and finally, to explore the possibility that these effects are due to increased consumption in the control groups rather than a decrease in consumption in the inhibition groups. This latter aim was based on the possibility that participants in the control groups may be inadvertently receiving a form of approach training (Schonberg *et al.*, 2014), and was explored by adding an additional control group

who made no responses but simply observed the task. These aims and the relevant literature are discussed.

4.1.1. The Stop-Signal Task and the Go/No-Go Task

Although the SST and GNG task are often used interchangeably in the response inhibition literature (Aron *et al.*, 2004, 2014; Jentsch & Pennington, 2014; Oosterlaan, Logan & Sergeant, 1998; Ridderinkhof *et al.*, 2004; Woltering, Liu, Rokeach & Tannock, 2013), and involve some overlapping neural circuits (e.g. Dambacher, Sack, Lobbstaël, Arntz, Brugmann & Schuhmann, 2014; Zheng *et al.*, 2008), they have also been dissociated, both in terms of the inhibitory mechanisms required for each task (Eagle *et al.*, 2008; Schachar *et al.*, 2007) and in the recruitment of different prefrontal regions (Aron *et al.*, 2003; Chambers *et al.*, 2006, 2007; Dambacher *et al.*, 2014; Garavan *et al.*, 2006; Liddle *et al.*, 2001; Rubia *et al.*, 2001; Swick *et al.*, 2011). The SST typically involves a choice reaction-time task in which participants must respond as quickly and as accurately as possible. On a minority of these trials (usually 25-33%) a signal is presented after a varied delay indicating that the participant should try to inhibit their response on that trial (this delay is often adjusted according to the participant's performance so that the likelihood of correctly stopping remains at 0.5; this is when the stop and go processes are believed to be at a point of maximum competition; Logan *et al.*, 1997). The low frequency and unpredictability of the signal, coupled with the delay between the onset of the stimulus and stop-signal means that participants are required to inhibit an already-initiated or 'prepotent' response – this inhibition process has been termed 'action cancellation' (Eagle *et al.*, 2008; Schachar *et al.*, 2007; see Figure 3.8). The GNG task, conversely, presents stimuli alongside either a go-signal or a no-go signal⁷. These trials are often presented with equal probability

⁷ In some GNG tasks participants are asked to respond to some stimuli and withhold responses from others and therefore the stimulus acts as the stop-signal (see Figure 1.5a). For example, Verbruggen and Logan (2008) presented participants with words of living and non-living objects and participants were asked to respond only to living but not non-living objects, or vice versa.

so that participants must try to inhibit their responses on 50% of trials. This increased rate of inhibition and the simultaneous presentation of the stimulus and the signal allows for the stop process to be triggered during the response preparation phase, thus allowing for participants to withhold their response. This form of inhibition has therefore been termed ‘action restraint’ (Eagle *et al.*, 2008; Schachar *et al.*, 2007; see Figure 3.8).

Perhaps of most importance for studies training response inhibition is the degree to which these two tasks require automatic versus controlled inhibition (for a review see Spierer *et al.*, 2013). Automatic inhibition is believed to develop through practice as an association between a stimulus and the stop goal is formed in memory (Verbruggen & Logan, 2008; Verbruggen *et al.*, in press). The rapid retrieval of this memory during stimulus presentation therefore allows for an earlier recruitment of the inhibitory control network and successful stopping (Berkman, Kahn & Merchant, 2014). Although it has been argued that the SST and GNG task both involve a combination of automatic and controlled processes, automatic inhibition is thought to be more likely in the GNG task due to the consistent stimulus-no-go associations (Shiffrin & Schneider, 1977; Verbruggen & Logan, 2008). Consistent associations are likely to be acquired more readily than inconsistent associations are also more likely to result in the successful inhibition of a response. Inconsistent associations, on the other hand, tend to result in more inhibition failures which may maintain the engagement of top-down control and result in stimulus-*signal* rather than stimulus-*stop* associations (Spierer *et al.*, 2013; Verbruggen *et al.*, in press). For research investigating the effects of inhibition training on food consumption and choice behaviour, these differences may have important implications for developing an optimum training procedure.

A GNG task with consistent mappings between the stimulus and the stop-signal suggests two potential advantages over the SST. Due to the greater involvement of automatic inhibition with the GNG task, the first advantage is the increased likelihood of rapidly retrieving the stop goal when encountering foods previously paired with stopping. Secondly, if the effect of inhibition training on behaviour is due to stimulus devaluation as a result of stimulus-stop associations (Houben *et al.*,

2011a, 2012a; Veling & Aarts, 2009; Veling *et al.*, 2008, 2013b), it is possible that stimuli presented during GNG training will be devalued to a greater extent than those on the SST due to more frequent stimulus-stop pairings (although it has previously been shown that increasing the number of pairings had little effect on stimulus evaluations; Veling *et al.*, 2013b). Conversely, it is also theoretically possible that inhibition training may benefit from a greater involvement of top-down control. If training on the SST promotes the activation of a more global stop goal, which is not dependent on associations with certain stimuli, then transfer of effects to novel stimuli, or foods, should be more likely with stop-signal compared to GNG training (Berkman *et al.*, 2009, 2012; Tabibnia *et al.*, 2011; Verbruggen *et al.*, 2012). There is a caveat, however, as increasing inhibition generally is not synonymous with optimal behaviour; for example, although inhibition training has been shown to reduce risky decision-making on a gambling task, continuously placing ‘safer bets’ in this task did not result in maximum financial gains (Verbruggen *et al.*, 2012). In the context of dietary health behaviours, increasing general inhibition could result in the decreased consumption of all foods, including healthy foods such as fruits and vegetables, which would not be a desired outcome for this line of research. On the other hand, there is some evidence to suggest that improved inhibitory control is associated with increased healthy food choices (Hall *et al.*, 2008; Veling *et al.*, 2013b).

4.1.2. Response Inhibition and Healthy Food Consumption

A few studies have already investigated whether response inhibition and executive functioning in general have any role in healthy dietary behaviours. Most of these have considered executive functioning as a potential moderator in the gap between dietary intentions and behaviour (Allan *et al.*, 2011; Collins & Mullan, 2011; Hall, 2012; Hall *et al.*, 2008; Riggs *et al.*, 2012). For example, Hall *et al.* (2008) found that intentions to exercise and eat healthily (fruit and vegetable consumption) were more predictive of behaviour for those with efficient, compared to poor, inhibitory control on the GNG task. However, in a later study, Hall (2012) found that while GNG performance predicted reduced consumption of fatty foods, even after controlling for demographic variables and BMI, there was no relationship between

GNG performance and the consumption of non-fatty foods. A limitation of this measure of non-fatty food consumption, however, was the inclusion of staple foods, such as cereal, milk and rice, which may have dampened this effect compared to the aforementioned study in which only fruit and vegetable consumption was considered. In a similar study, Allan *et al.* (2011) investigated the role of executive functions in the intention-behaviour gap for snacking and fruit and vegetable intake. They showed that although measures of cognitive switching and flexibility were predictive of reduced snack intake and increased fruit and vegetable consumption, performance on the GNG task did not correlate with either of these measures (see also Collins & Mullan, 2011). Conversely, a self-report measure known to reflect poor inhibitory control was positively associated with both reduced healthy food consumption and increased snacking (see also Riggs *et al.*, 2012).

Allom and Mullan (2014) explored whether these conflicting findings between inhibitory control performance and healthy versus unhealthy food consumption could be explained by differences in inhibitory and initiatory control. It has been shown that inhibitory control ability, which requires stopping a response, is negatively associated with undesirable behaviours such as smoking and alcohol consumption, whereas initiatory or 'start' control is predictive of desired behaviours such as studying or exercising (de Boer *et al.*, 2011; de Ridder, de Boer, Lugtig, Bakker & van Hooft, 2011). Allom and Mullan therefore measured two dimensions of executive functioning that were associated with inhibitory and initiatory control to see whether they were predictive of self-reported unhealthy and healthy food intake, respectively. For inhibitory control they analysed performance on two tasks measuring response inhibition (the SST and Stroop task) and for initiatory control they measured the ability to update and monitor goals (using the n-back and operation span tasks). As predicted, they found that poor inhibitory control correlated with increased saturated fat intake but was not predictive of fruit and vegetable consumption. Updating, on the other hand, was predictive of fruit and vegetable intake, but was not related to the consumption of saturated fat. These results suggest that inhibition training is unlikely to affect the consumption of healthy foods, as desired behaviours are more likely to benefit from improved start control than improved stop control (de Boer *et al.*, 2011; de Ridder *et al.*, 2011).

However, these studies have all used standard versions of response inhibition tasks, with arbitrary stimuli – it is possible that food-related inhibitory control will be more likely to correlate with healthy food intake (Nederkoorn *et al.*, 2012; Houben *et al.*, 2012b; Meule *et al.*, 2014a).

To date, the only published study that has explored the effect of food-related response inhibition training on both unhealthy and healthy food-related behaviour is that of Veling *et al.* (2013a). In this study participants were trained to inhibit responses to palatable foods on a GNG task before being asked to select eight snacks from a variety of sixteen healthy and unhealthy foods. These snack choices were hypothetical and did not involve the consumption of these foods. Compared to participants who responded on all trials, those in the inhibition group selected significantly fewer unhealthy snacks and significantly more healthy snacks (these effects were statistically significant only when comparing individuals with a high appetite or frequency of consumption; see Figure 1.7). Although this result appears promising for the effect of inhibition training on healthy food choices, due to the forced number of selected snacks, it is unclear whether this result reflects a voluntary increase in healthy food choices or an inevitable shift due to a decreased selection of unhealthy foods. To draw any firm conclusions regarding inhibitory control training and increased healthy food behaviours it would be necessary to replicate this finding with an unforced number of choices or, alternatively, to measure actual food consumption.

For the present study I therefore measured the consumption of healthy and unhealthy foods following inhibition or control training using both the SST and the GNG task. Although the consumption of healthy food was of interest, inhibition training was targeted towards unhealthy foods only, as these are the foods most commonly associated with obesity and weight problems (Blundell *et al.*, 1993; Drewnowski *et al.*, 1985; Drewnowski *et al.*, 1992). Healthy foods were mainly associated with a go response, with the exception of the stop training task in which a minority were paired with stop-signals. At present it is unclear whether this type of training would have any additional influence on healthy food intake, especially as effects on reduced unhealthy food behaviours are believed to depend upon stimulus devaluation as a

result of specific stimulus-stop associations (Veling *et al.*, 2013b). Firstly, it is possible that response inhibition training would have no effect on healthy food consumption; this would be consistent with previous findings demonstrating no relationship between response inhibition and self-reported healthy food intake for either the SST or GNG task (Allan *et al.*, 2011; Allom & Mullan, 2014; Collins & Mullan, 2011; Hall, 2012). Secondly, consistent with Veling *et al.*'s (2013a) findings for increased food choice, it is possible that inhibition training may result in the increased consumption of healthy foods. One possible explanation for this finding would be an effect of inhibition training on the engagement of high-level decision-making processes, whereby an individual compensates for a decreased intake of unhealthy foods with an increased intake of healthy foods. Alternatively, it is possible that consistently responding to healthy foods may increase evaluations of these foods by priming an approach response or activating an appetitive centre (see section 4.1.3. below; McLaren & Verbruggen, submitted). Moreover, any effect of training on increased healthy food intake would be a key finding from a clinical perspective, especially if paired with a decrease in unhealthy food intake. Finally, it is possible that training may result in *decreased* healthy food consumption, possibly through the activation of a global stop goal (Berkman *et al.*, 2009, 2012; Tabibnia *et al.*, 2011; Verbruggen *et al.*, 2012).

For the unhealthy foods, it was hypothesised that although inhibition training would reduce consumption in both training tasks, this effect would be greater in the GNG task due to the higher consistency of mappings in this task (100%) compared to the SST (89%). However, a larger decrease in unhealthy food intake in the SST compared to the GNG would indicate a greater role of top-down control. To see whether these effects were stimulus-specific, or whether they generalised to novel stimuli, the food consumption phase included foods that were presented during the training task ('old' foods) as well as one novel unhealthy and one novel healthy food ('new' foods). If the effects of training on behaviour are dependent upon specific stimulus-stop associations, the only expected difference would be for the consumption of foods encountered during training. Previous studies have indeed shown that effects of inhibition training on food consumption and food choice were specific to the trained foods (Houben, 2011; Lawrence *et al.*, under review; Veling *et*

al., 2013a). Any transfer of effect to the novel food items, however, may indicate either an effect of inhibition training on increased general self-control or a transfer effect of stimulus devaluation.

4.1.3. Increased Inhibition or Increased Approach?

A final aim of the present study was to consider the possibility that any significant difference in food intake between the inhibition and control groups could be a result of increased consumption in the control group rather than decreased consumption in the inhibition group. Previous studies have all included control conditions in which participants have been required to consistently respond to images of food (Houben, 2011; Houben & Jansen, 2011; Lawrence *et al.*, under review; Veling *et al.*, 2011; see Appendices 1 and 2). Houben (2011) and Houben and Jansen (2011) also included a control condition in which half of the images were paired with a response and half of them were paired with inhibition. The authors believed that this task served as a baseline “without inducing impulsivity of inhibition” (p.386, Houben, 2011). However, there is evidence in both of these studies that participants consumed more calories in the control condition than the impulsivity condition. These unexpected results may be explained by findings indicating that associative uncertainty can increase incentive salience and responding for conditioned stimuli (Anselme, Robinson & Berridge, 2013; Collins & Pearce, 1985; Collins, Young, Davies & Pearce, 1983; Pearce & Hall, 1980). It is possible therefore that both the consistent and inconsistent ‘control’ conditions may act to increase dietary disinhibition as participants learn an approach response towards food. This concern seems reasonable given findings demonstrating the presence of an approach bias for both foods and addictive substances (Bradley *et al.*, 2004; Brignell *et al.*, 2009; Cousijn *et al.*, 2011; Field *et al.*, 2006, 2008; Havermans *et al.*, 2011; Kemps *et al.*, 2013b; Mogg *et al.*, 2003, 2012; Van Gucht *et al.*, 2008; Veenstra & de Jong, 2010; Watson *et al.*, 2012; Wiers *et al.*, 2011).

Moreover, recent research has shown that training participants to respond to unhealthy foods can bias choice behaviour in favour of these foods (Schonberg *et al.*, 2014). In this study participants rated the value of palatable junk food items, using

an auction task, before receiving cue-approach training in which certain foods were always paired with a go response cue (go foods) and other foods were never paired with a response (no-go foods). Following training, participants were asked to choose between two equally valued foods in which there was one go food and one no-go food in each pair. Their results revealed a significant effect of go training on food choice for highly-valued foods; across four separate studies the high value go foods were selected on 60-65% of trials. The lack of an effect for low value foods indicates that these findings were not the result of demand characteristics to strategically select all go foods. Moreover, the same effect was observed in a separate study in which all items were below the median auction value, suggesting that training was effective for changing values relatively, regardless of the item's absolute value. Schonberg *et al.* (2014) also demonstrated that the motor response was crucial for this effect; there was no effect of training on food choice when participants heard the go cue but did not respond. Furthermore, for participants who received the longest training task, these preferences for go foods were still present at follow-up testing two months later. Based on these findings, it seems reasonable to hypothesise that pairing palatable foods with a go response may also affect food consumption. Interestingly, however, when the training instructions were reversed so that participants responded on all trials and inhibited their response when a cue was presented, Schonberg *et al.* found no difference in food choice. This latter finding may suggest that the effect of approach training is not only dependent on the motor response, but also the association with the approach cue. This is an important distinction as the control group for the SST (double-response group) in the current study received trials in which responses were paired with signals whereas those in the control group for the GNG (go group) did not see any signals throughout the training task. Nevertheless, an additional control group was added to the present study in which participants simply observed the task; participants therefore received the same level of exposure to palatable foods but did not make any responses (observe group). If both inhibition and approach processes occur together during training, it would be expected that consumption in the observe group would be intermediate between the inhibition and control groups. If differences in intake reflect either an effect of inhibition or approach training only, the observe group is expected to significantly differ from either the inhibition or control groups, respectively.

4.2. Method

4.2.1. Participants

One hundred and ninety three participants (177 females; aged 18-47, $M=21.69$, $SE=0.39$) were randomly divided into the five training groups: stop ($n=45$, 42 females), double-response ($n=46$, 42 females), no-go ($n=35$, 32 females), go ($n=35$, 32 females) and observe groups ($n=32$, 29 females; sample sizes were determined according to Bayesian analyses for the main difference in total calorie intake between the inhibition group and the respective control group; see below, section 4.2.4). University staff and students were recruited through posters, electronic advertisements and an online experimental management system. They were screened with the Restraint Scale (RS; Herman & Polivy, 1980; see section 2.2.2.1) at least one week prior to the study and were only eligible if they met the cut-off for restrained eating (score of 15+; $M=18.46$, $SE=0.22$). Participants were not eligible if they reported any history of eating disorders or if they were on a particular diet (with certain types of foods or a strict limitation of food intake with a weight goal and timeframe in mind). All participants were reimbursed for their participation; they received course credit or were offered either £6 or entry into a prize draw (for a £100 Amazon voucher). The study was approved by the School of Psychology Research Ethics Committee, Cardiff University.

4.2.2. Materials/ Measures

Figure 4.1 provides a schematic diagram of the study procedure.

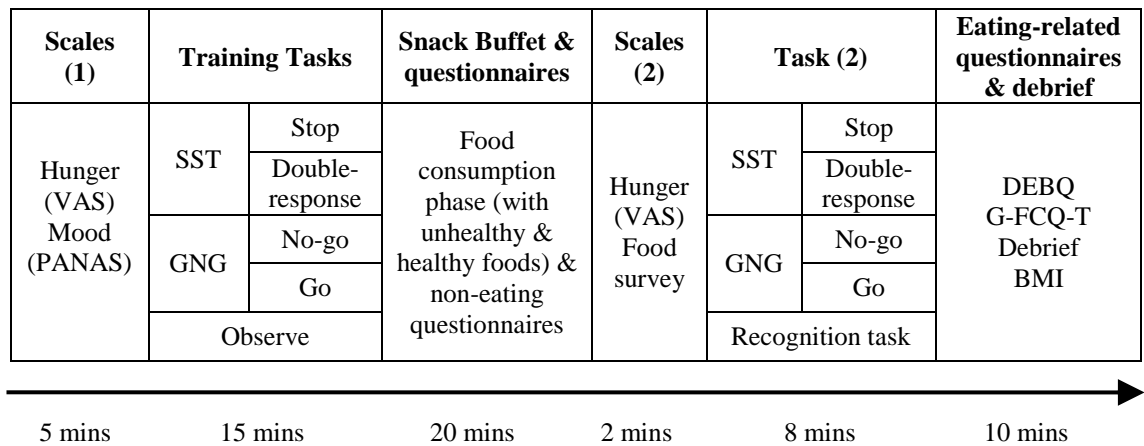


Figure 4.1. Schematic diagram of the study procedure. Participants answered measures of hunger and mood states (section 4.2.3) before the training task (section 4.2.2.1). Participants in the stop-signal groups received either a stop task or a double-response task and participants in the GNG groups received either a no-go or go task. An additional control group was also included who simply observed the training task (without signals). Following this task participants were taken to another room and were presented with a snack buffet with various unhealthy and healthy foods for consumption (section 4.2.2.2). They also completed a series of questionnaires to keep them occupied for 20 minutes (section 4.2.2.3). Participants were then brought back to the original testing room and completed hunger scales, a food survey (section 4.2.2.3) and the training task again – this additional task was included as part of the cover story for investigating cognitive performance at low and high blood glucose levels (section 4.2.3). Participants in the observe group performed a recognition task to ensure that they were paying attention in the first training task (section 4.2.2.4). Participants then completed eating-related questionnaires, were debriefed and their height and weight was recorded to calculate BMI.

4.2.2.1. Training Tasks

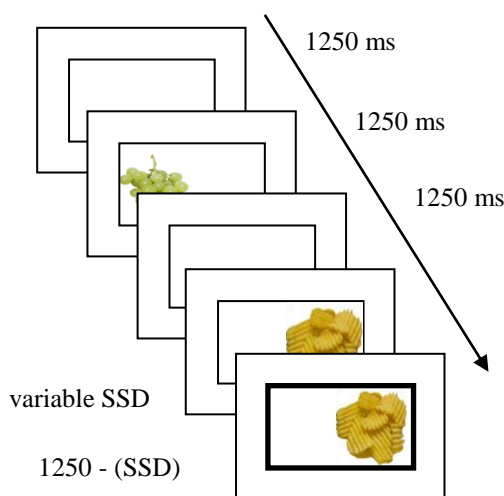
All training tasks were programmed in Matlab (Mathworks, Natick, MA) using Psychophysics Toolbox (www.psychtoolbox.org) and all stimuli were presented on a 19-inch flat-panel LCD monitor. The training tasks lasted approximately 15 minutes and consisted of eight blocks of 36 trials. Participants were given a 15 second break between each block. The blocks randomly presented nine images of unhealthy foods (three images each of chocolate, crisps and biscuits), nine images of healthy foods (three images each of fruit, rice cakes and salad vegetables) and 18 filler images

(clothes; three each of jeans, shirts, jumpers, socks, skirts and ties). One stimulus of each food type was a photographed image of the corresponding food item that was presented in the snack buffet. All images, including the snack food photographs, were close-up views of the food item against a white background; images were carefully selected on the basis that there were no additional ingredients or packaging, and they were matched for size and complexity (see Appendix 5).

For all trials the central rectangle appeared in the centre of the screen (fixation; 1250ms), followed by the presentation of a stimulus to either the left or right hand side (1250ms; see Figure 4.2). Stimuli were presented once per block in a random sequence and with equal probability to either location. For the no-signal trials participants were required to respond to the location of the stimulus as quickly and accurately as possible using their left and right index fingers ('C' or 'M' on a standard keyboard). The presentation of the signal and the relevant instructions for the signal trials are described below according to the training task.

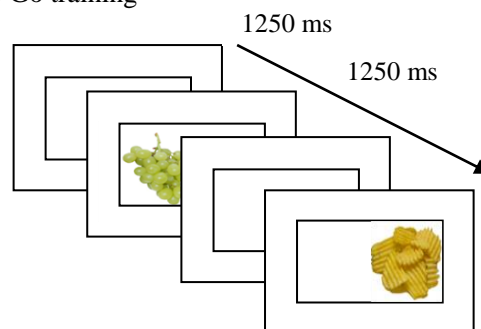
4.2.2.1.1. Stop-Signal Training – For the stop-signal training the majority of signals were mapped onto the unhealthy foods (8/9; 88.89% mapping), with a few occurring on the healthy (1/9; 11.11% mapping) and filler (1/18; 5.56% mapping) trials. For the presentation of the signal the central rectangle would turn bold after a variable delay (SSD, see section 3.2.2.1 for the simulated tracking procedure that was used to determine SSD length; see Figure 4.2a). Participants in the double-response group were instructed to make a secondary response when a signal was presented; following the response to the location of the stimulus they were required to make a thumb response on the space bar as quickly as possible. Participants in the stop group were instructed to withhold their response whenever a signal occurred. They were informed that the signal would sometimes appear quickly and sometimes after a longer delay, and that they should therefore find it easier to stop on some trials than others. They were instructed that they must not wait for a signal to occur.

a) Stop Signal Training



b) Go/No-Go training

i) Go training



ii) No-Go training

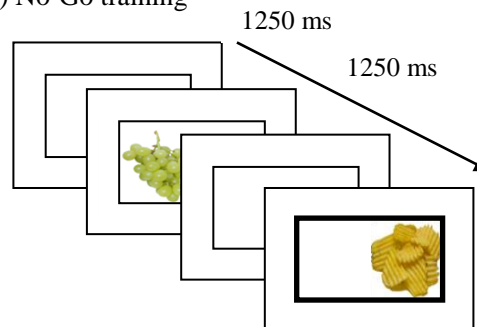


Figure 4.2. Display sequence for **a)** the stop-signal training tasks **b)** the go/no-go training tasks. For no-signal trials participants were presented with a fixation rectangle before seeing a stimulus appear on either the left or right hand side. Participants were instructed to respond as quickly and as accurately as possible to the stimulus location using the ‘C’ and ‘M’ keys. **a)** For the stop-signal training, signals were presented after a variable stop-signal delay (SSD) which was initially set to 250 ms and then adjusted according to a simulated tracking procedure (see section 3.2.2.1 for details). Participants in the stop group had to inhibit responses on these trials whereas participants in the double-response group had to make an additional response. **b)** For the GNG training, i) participants in the go group were presented only with the no-signal trials; ii) for the no-go group, stimuli and signals were presented simultaneously (0 ms SSD) and participants were required to refrain from responding on these trials.

4.2.2.1.2. Go/No-Go Training – In the GNG training, signals were only presented for the no-go group; all trials for the go group were no-signal trials, therefore they were required to make a location response on every trial (see Figure 4.2b.i). For the no-go group signals were consistently mapped onto the unhealthy foods (9/9; 100% mapping) with no signals occurring alongside the healthy food images (0/9; 0%

mapping). Filler images were inconsistently paired with a signal (9/18; 50% mapping) so that the overall rate of no-go signals was 50%. Signals appeared as a bold rectangle that replaced the fixation rectangle and lasted for the duration of the trial (1250ms; see Figure 4.2b.ii); this meant that there was no delay between the presentation of the stimulus and the signal. Participants in the no-go group were instructed to withhold their response when a signal was presented.

4.2.2.1.3. Observe Training – Participants in the observe group were presented with the same training stimuli as the go group. Images were presented to the left and right hand side within a central rectangle. Participants were informed that they were to watch the stimuli, and that they needed to pay some attention because they would be asked questions at the end of the session (these questions were presented in the form of a recognition task; see section 4.2.2.4. below). They were not required to make any responses to the stimuli.

4.2.2.2. Snack Buffet

Following training participants were taken to another testing room and were presented with a snack buffet with various unhealthy and healthy food items (unhealthy foods: chocolate (Cadbury milk chocolate ‘bitsa wispa’), crisps (Tesco ready salted crisps), biscuits (Fox’s mini malted milk biscuits) and cheese bites (Asda cheese bites, these are small savoury cheese flavoured snacks); healthy foods: grapes (green), rice cakes (mini wholemeal rice cakes), carrot sticks and mini breadsticks (Asda mini breadsticks); for weight and nutritional information see Appendix 6). All items were selected based on their calorie and fat content and whether they were believed to be sweet or savoury. The size of the item was also considered; foods with small individual pieces were used to ensure that our measure of consumption was as sensitive as possible. The weights were determined by average food intake from a pilot tasting session and approximate visual quantities. Participants were seated at a desk with eight bowls of food presented in front of them along with a jug of water (see Figure 4.3). The presentation of the food bowls was pseudo-randomised across participants to ensure that there was no bias in consumption based on the spatial proximity of the food item (see Appendix 7 for pseudorandom orders). Participants were asked to fill out a battery of non eating-

related personality questionnaires (see Questionnaires below) and were instructed to eat as much food as they liked but to ensure that they were not feeling hungry when the experimenter returned after 20 minutes. This meant that participants had free choice over which foods they selected and how much they consumed (compared to Veling *et al.*'s (2013a) study in which participants were required to choose eight snacks). Unknown to the participants, all food items were weighed before and after the snack buffet to measure calorie consumption.



Figure 4.3. Photograph of the snack buffet layout. Participants were presented with eight bowls of healthy and unhealthy foods (see Snack Buffet, section 4.2.2.2 for details), water, and a series of personality questionnaires (see Questionnaires, section 4.2.2.3. for details). The presentation of the food items was pseudo-randomised based on healthiness and colour to minimise the effect of spatial proximity and order on consumption (see Appendix 7 for details). Participants were left alone with the food and questionnaires for 20 minutes.

4.2.2.3. Questionnaires

During the snack buffet participants were provided with the same questionnaires as in Study 2 (The Big Five Inventory, the Brief Self Control Scale, the Emotion

Regulation Questionnaire, the UPPS impulsive behaviour scale, the Attentional Control Questionnaire and the Mood and Anxiety Symptom Questionnaire; see section 3.2.2.4 for full details). The purpose of these questionnaires was to keep the participant occupied for the duration of the snacking phase. After this time elapsed participants were taken back to the original testing room. They completed three hunger measures (three 100mm VAS measures to measure hunger, fullness and desire to eat, see section 3.2.3 for full details) along with a brief survey regarding the food items presented in the snack buffet. This questionnaire asked participants how often they normally consumed the food items (7 point Likert scale from 1 “Never” to 7 “Daily”), whether they consumed each of the items during the snack phase (this was to reduce the likelihood of participants believing that their food consumption was being measured), and if so how much they liked the taste of the item (10 point Likert scale from 1 “I didn’t like the taste at all” to 10 “I liked the taste very much”).

4.2.2.4. Recognition Task

The recognition task was presented to the observe group only to ensure that they did indeed observe the training task; the task consisted of one block of 72 trials. Each trial presented a central image that was either an image repeated from the training phase or a new image. All 36 images from the training task were used, of the remaining 36 trials: 12 were completely novel objects that were not representative of the images presented in the training phase (e.g. plants and household objects); 12 were relatively similar images of foods and clothes (for example a different type of fruit) and 12 were very similar objects of foods and clothes (for example a different image of an apple). The relatively and very similar items were included to ensure a certain degree of difficulty. Each stimulus was presented until a response was given or for a maximum of eight seconds. For each trial participants had to indicate whether or not they recognised the image from the training task (responding ‘Old’ or ‘New’ with the ‘J’ and ‘K’ buttons, respectively, on a standard keyboard). They were instructed to respond as quickly and accurately as possible; if they were unsure they were told to make their best guess. The task lasted approximately 2-5 minutes depending on performance speed and was programmed in Matlab (Mathworks, Natick, MA) using Psychophysics Toolbox (www.psychtoolbox.org); stimuli were presented on a 19-inch flat-panel LCD monitor.

4.2.3. Procedure

All participants were informed that they were taking part in a study investigating the effect of blood glucose on performance in cognitive tasks. This cover story was used to disguise the true aim of the study and to explain why participants were asked to refrain from eating prior to the study and to eat something during the study. They were informed that they were asked not to eat for three hours prior to the study to ensure that their glucose levels were relatively low when completing the task and that they would be given something to eat half way through the study to replenish their glucose levels before completing the task again. This allowed us to measure their performance at low and high blood glucose levels, respectively. The true purpose of asking participants to refrain from eating prior to the study was to control for levels of hunger and food motivation (Gibson & Desmond, 1999; Guerrieri *et al.*, 2009; Lawrence *et al.*, under review; Veling *et al.*, 2013a, 2013b). Testing therefore only took place between the hours of 12-7pm. After giving consent participants answered the initial hunger (VAS measures for hunger, fullness and desire to eat) and mood (PANAS) state questionnaires (see section 3.2.3 for full details). They then completed the training task before being taken to the snack buffet, where they also answered a battery of personality questionnaires. Afterwards the experimenter brought them back to the original testing room to answer the hunger scales and food survey (see Questionnaires above) and to complete the training task again. This task was identical to the initial training task with the exception that it only lasted ten minutes (4 blocks of 36 trials). The only purpose of this task was to make the cover story plausible. After finishing this task, participants were asked to complete two eating-related questionnaires (the DEBQ and G-FCQ-T; see section 3.2.2.4 for full details; these were included for the purpose of Study 1). They were then debriefed and probed for knowledge of the study's aims and stimulus mappings (see section 3.2.3 for full details), and their height and weight was recorded to calculate BMI (kg/m²).

4.2.4. Statistical Analyses

Although the broad goal of this study was to qualitatively compare the effectiveness of the stop-signal and go/no-go training tasks on food consumption, the specific aim

was to compare the effectiveness of each inhibition task relative to the control task within that training protocol – thus providing a partial replication of previous studies. For the analysis of calorie intake, these two training protocols were therefore initially analysed separately: the stop group was compared to the control group for that training task – the double-response group – and the no-go group was compared to the go group. The groups were then analysed together, including the additional observe group, to allow for a direct statistical comparison.

First, demographic, state and trait variables were analysed with nonparametric Kruskal-Wallis tests to ensure there were no statistically significant differences between training groups. Data from the training tasks, and recognition task for the observe group, were also analysed to ensure that participants were performing the task as expected and to guide participant exclusions. Exclusion criteria for the stop-signal task were identical to those used in Study 2 (see section 3.2.4). Three participants were excluded from the stop group: one due to a high percentage of errors on signal trials (failed inhibitions $>3SDs$ from the group mean) and two further exclusions were made due to prior participation in a similar study. Five exclusions were made in the double-response group: one participant's performance was $<85\%$ accurate on no-signal trials, one participant had already participated in a similar study, two participants were dieting at the time of the study and one participant correctly guessed the aim of the study during debrief. These exclusions meant a final sample size of 81 with 42 participants in the stop group and 39 participants in the double-response group. A sensitivity analysis (using G*Power; Faul *et al.*, 2007) for the main interaction between training condition and food type revealed that this sample had 80% power to detect a minimum effect size of $f=0.23$ for the main effect of training condition and $f=0.22$ for the interaction ($\alpha=0.05$, number of groups =2, number of repetitions =2, correlation among repeated measures =0.02, non-sphericity correction =1).

For the GNG training, exclusion criteria for no-signal trials were identical to those for the stop-signal training (see Chapter 3, section 3.2.4. for details). For the no-go group the percentage of erroneous responses on signal trials (commission errors) was also analysed and participants were excluded if this value exceeded $>3SDs$ from the

group mean. Two participants were excluded from the go group due to their performance on no-signal trials (<85% accuracy); no participants were excluded from the no-go group due to training performance, however, a further two participants, one from each group, were excluded due to previous eating disorders. This resulted in a final sample of 34 participants in the no-go group and 32 participants in go group. A sensitivity analysis (using G*Power; Faul *et al.*, 2007) for the main interaction between training condition and food type revealed that this sample had 80% power to detect a minimum effect size of $f=0.26$ for the main effect of training condition and 80% power to detect an effect size $f=0.23$ for the interaction ($\alpha=0.05$, number of groups =2, number of repetitions =2, correlation among repeated measures =0.13, non-sphericity correction =1).

For the observe group, percentage accuracy for the recognition task was analysed to ensure that participants paid attention to the task. Participants performed this task well; no exclusions were made according to the pre-set criterion of 70% accuracy. This value was set above chance but was not too high: because the stop-signal and GNG training tasks do not explicitly require attention to the stimuli (during debrief in Study 2, 38% of participants reported no awareness of the stimulus-signal associations) it was only necessary to ensure a moderate degree of attention. For the full analysis with all training groups there was a total sample size of 179 participants ($n=32$ for the observe group). A sensitivity analysis (using G*Power; Faul *et al.*, 2007) for the main interaction between training condition and food type revealed that this test had 80% power to detect a minimum effect size of $f=0.19$ for the main effect of training condition and 80% power to detect an effect size $f=0.18$ for the interaction ($\alpha=0.05$, number of groups =5, number of repetitions =2, correlation among repeated measures =0.02, non-sphericity correction =1).

For the food consumption data, outliers in calorie intake were first transformed to remove the potential influence of extreme values. Outliers were examined for each food separately and were considered as values >3SDs from the group mean. Outlier values were replaced with the nearest non-outlier value +1 (see section 3.2.4.; Tabachnick & Fidell, 2007). Secondly, to calculate calorie intake as a function of food type and food novelty, the mean calorie value for each food category was

calculated by dividing the total calories for that food category by the number of foods in that category. For example, for unhealthy old foods there were three different foods (chocolate, crisps and biscuits) but for unhealthy new foods there was only one food presented (cheese bites); calorie consumption for unhealthy old foods was therefore divided by three to calculate the mean intake for that category. Calorie intake was analysed with a mixed analysis of variance (ANOVA) with the between subjects factor of *training condition* and the within-subjects factors of *food type* (healthy and unhealthy) and *food novelty* (old and new). Due to the possibility of floor effects with the low calorie density in healthy foods, an additional analysis for consumption in grams was also performed with the between subjects factor of *training condition* and the within-subjects factor of *food novelty* (old and new).

Bayes factors for total food consumption were also calculated to allow for additional data collection and interpretation of null results. Following Dienes (2011, 2014), a half-normal distribution was used with a standard deviation of 49.19, which corresponded to the difference in intake between inhibition and control groups in previous studies (see Chapter 3 section 3.2.4 for full details). This difference value was the same value used in Study 2, however, in the present study Bayes factors must be interpreted with caution as they are likely to be overestimated. This difference score was based on previous studies which have typically included 1-3 foods in a bogus taste test (see Appendix 1), whereas in this study 8 foods were presented and participants were asked to have as much food as they liked as long as they were no longer hungry after 20 minutes. It is possible therefore that participants in the present study would have consumed more calories than participants in previous studies as increased food variety has been associated with increased food intake and obesity (Raynor & Epstein, 2001). Furthermore, there is evidence to suggest that increased food variety may interact with increased reward sensitivity (Guerrieri *et al.*, 2008a), although null results for an interaction between variety and increased impulsivity and poor inhibitory control have also been reported (Guerrieri *et al.*, 2007b, 2008a). However, with the additional personality questionnaires to occupy participants during the consumption phase in the present study, it is also possible that participants were more distracted from the food and therefore consumed fewer calories. As the extent to which these methodological changes

would interact with dietary restraint to influence food intake was unknown, Bayes factors were calculated in the same way as Study 2. For each comparison of interest a between-subjects t-test was performed to obtain the mean difference and standard error of the difference. These values, along with the expected difference (49.19), were entered into Dienes' online calculator (see footnote 6). Bayes factors >3 were interpreted as substantial evidence for the alternative hypothesis (i.e. inhibition training reduces food consumption compared to the control group) and Bayes factors <0.33 were interpreted as evidence for the null hypothesis (i.e. there is no effect of inhibition training on food intake).

All results are reported with unadjusted significance values; corrections for multiple comparisons were calculated for all within-test analyses and are only reported where these corrections changed the interpretation of an analysis from statistically significant to non-statistically significant. All statistical analyses were performed with SPSS.

4.3. Results

4.3.1. Group Differences

The training groups were well matched for age, BMI, initial RS score and hours since food consumption (all $\chi^2 < 4.49$, all $ps > 0.34$, all $\eta^2s < 0.03$; Table 4.1.). Due to the small number of males in each group a Chi square test was not performed, however, the groups were also matched for gender ratio with three males in each group. There were also no statistically significant differences for state measures of hunger and mood (all $\chi^2 < 3.47$, all $ps > 0.48$, all $\eta^2s < 0.02$) or for measures of food liking and frequency of consumption for the buffet foods (all $\chi^2 < 3.33$, all $ps > 0.51$, all $\eta^2s < 0.02$).

Table 4.1. Group characteristics, showing gender ratio and mean age, BMI, restraint score (RS), hours since last food consumption, hunger scores (VAS), positive affect score (PANAS) and negative affect score (PANAS), per training condition (SE within parentheses).

	Stop (n=42)	Double-response (n=39)	No-go (n=34)	Go (n=32)	Observe (n=32)	$\chi^2=$	$p=$
Gender (% female)	92.86	92.31	91.18	90.63	90.63		
Age	20.57 (0.47)	21.97 (1.01)	20.59 (0.43)	23.66 (1.35)	21.5 (0.89)	2.39	0.66
BMI	24.2 (0.68) ²	25.02 (0.81) ¹	23.49 (0.53) ¹	24.92 (0.68)	23.7 (0.68)	4.49	0.34
RS	18.73 (0.5)	18.05 (0.44)	18.76 (0.6)	17.88 (0.48)	18.63 (0.53)	2.22	0.7
Hours since food	5.21 (0.54) ⁴	5.65 (0.72)	5.13 (0.57)	6.3 (0.91)	5.73 (0.65)	1.79	0.77
Hunger	4.7 (0.31)	4.6 (0.35) ¹	4.99 (0.33)	4.98 (0.34) ¹	4.88 (0.32)	0.99	0.91
Fullness	1.74 (0.26)	1.84 (0.28) ¹	1.75 (0.27)	1.74 (0.33)	1.56 (0.27)	0.51	0.97
Desire to eat	5.29 (0.34)	5.01 (0.35)	5.55 (0.36)	5.32 (0.41)	5.52 (0.43)	1.97	0.74
Positive affect	23.66 (1.09) ¹	25.03 (1.07)	23.61 (1.04) ¹	25.78 (1.13)	25.75 (1.34)	3.47	0.48
Negative affect	13.15 (0.49) ¹	12.82 (0.46)	12.52 (0.5) ¹	13.09 (0.7)	13.41 (0.71)	1.01	0.91
Healthy food liking	6.64 (0.26) ⁹	6.73 (0.27) ¹⁰	6.98 (0.31) ⁸	6.22 (0.35) ⁸	6.97 (0.3)	3.33	0.51
Unhealthy food liking	7.19 (0.26) ⁹	7.16 (0.28) ¹¹	7.26 (0.28) ⁸	7.31 (0.29) ⁸	7.15 (0.26)	0.68	0.95
Healthy food consumption freq.	3.77 (0.22) ⁹	3.63 (0.19) ¹⁰	3.58 (0.17) ⁸	3.34 (0.17) ⁸	3.78 (0.18)	2.84	0.59
Unhealthy food consumption freq.	3.98 (0.17) ⁹	3.78 (0.22) ¹⁰	3.84 (0.18) ⁸	3.96 (0.21) ⁸	3.69 (0.21)	1.83	0.77

Note. Superscript denotes the number of participants missing for that variable. RS= Restraint Scale; BMI= body mass index

4.3.2. Training Data Analysis

The results for the training data are presented in Figure 4.4. The results revealed that on average participants in the stop group failed to inhibit their responses on 38.42% ($SE=2.23$) of signal trials. This is lower than in Study 2, most likely due to the removal of inter-block feedback, but shows evidence for competing stop and go processes (Logan *et al.*, 1997) despite the high percentage of mapping of signals onto unhealthy food stimuli (88.8%). Participants in the double-response group only responded incorrectly to signals on 4.38% ($SE=0.57$) of trials. For the no-go group, participants failed to inhibit their responses on 5.23% ($SE=0.63$) of trials; this was significantly lower than for the stop group ($U=2$, $p<0.001$, $r=0.85$). The immediate presentation of the signal and the consistent mapping in the GNG task ensures that the stop process is likely to end before the competing go process, therefore increasing the probability of a successful stop compared to the SST (Logan & Cowan, 1984).

For the GoRT there was evidence of proactive slowing (Verbruggen & Logan, 2009b) in the stop and no-go groups, compared to the double-response and go groups. A Kruskal-Wallis test revealed a significant difference between groups ($\chi^2(3)=74.88$, $p<0.001$, $\eta^2=0.51$). Post-hoc follow-up tests (after correcting for 6 comparisons: $\alpha=0.008$) revealed that the stop group ($M=587.51$, $SE=23.89$) had significantly longer GoRTs than both the double-response ($M=401.02$, $SE=9.13$; $U=155$, $p<0.001$, $r=0.7$) and go groups ($M=380.71$, $SE=12.507$; $U=108$, $p<0.001$, $r=0.72$). Similarly, the no-go group ($M=508.41$, $SE=12.33$) was significantly slower than both the double-response ($U=135$, $p<0.001$, $r=0.68$) and go groups ($U=103$, $p<0.001$, $r=0.7$). The differences between the two inhibition groups ($U=515$, $p=0.04$, $r=0.24$) and the two control groups ($U=458$, $p=0.06$, $r=0.23$) were both non-significant after correcting for multiple comparisons.

There was also a significant difference between groups for the percentage of incorrect responses on no-signal trials ($\chi^2(3)=11.73$, $p=0.008$, $\eta^2=0.08$). Follow-up tests (corrected $\alpha=0.008$) revealed that participants in the no-go group ($M=0.92$, $SE=0.24$) made significantly fewer errors than participants in both the stop ($M=2.82$,

$SE=0.56$; $U=451.5$, $p=0.005$, $r=0.32$) and go groups ($M=1.9$, $SE=0.37$; $U=311$, $p=0.002$, $r=0.37$). All other comparisons were non-significant after correcting for multiple comparisons (all $ps>0.02$, all $rs<0.27$). These results appear to be due to a minority of participants in the stop and go groups who responded incorrectly on more than 8% of trials.

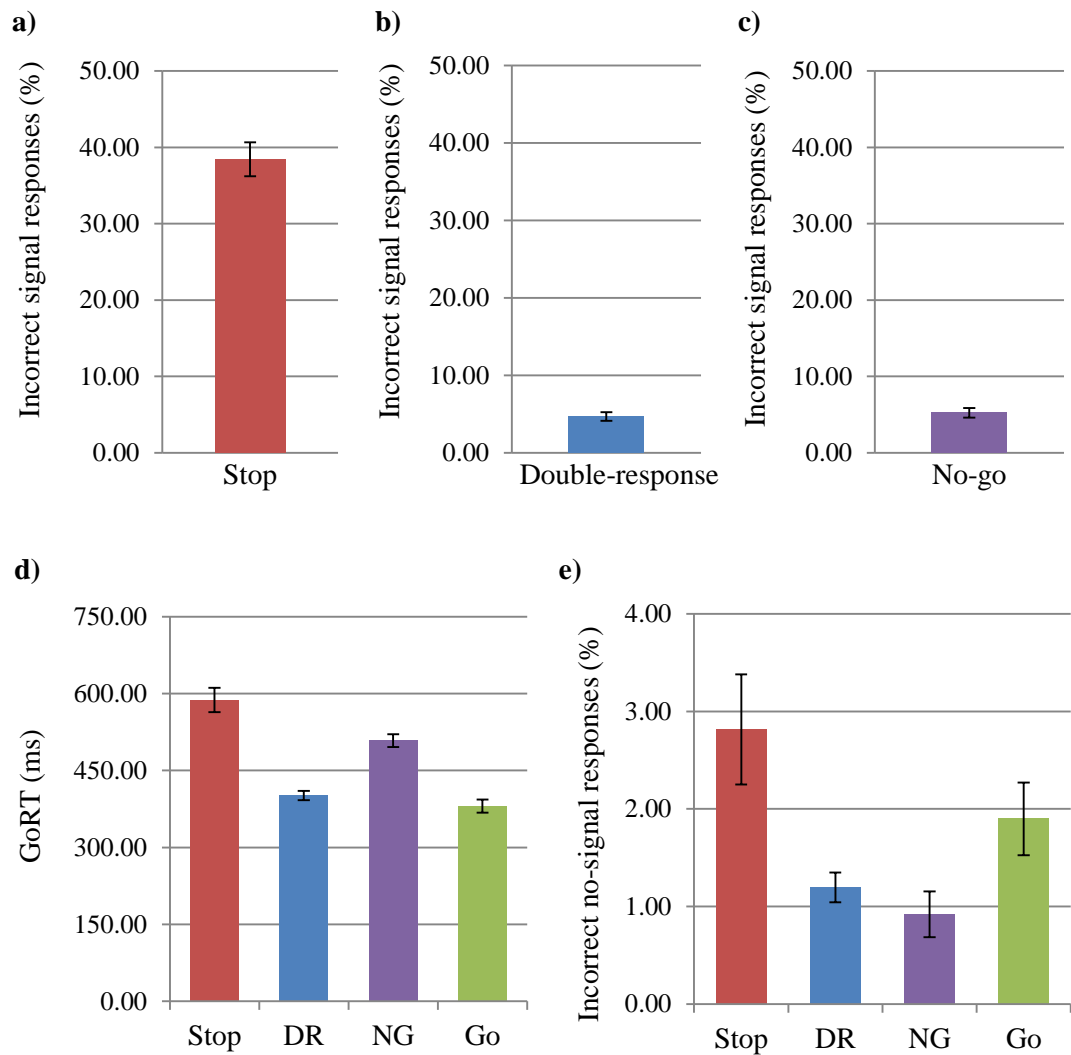


Figure 4.4. Training data for the stop, double-response (DR), no-go (NG) and go groups showing the mean percentage of erroneous signal responses for **a)** failed inhibition trials in the stop group, **b)** failed dual responses in the double-response group and **c)** failed inhibition trials in the no-go group, as well as the mean GoRT (**d)** and mean percentage of incorrect no-signal responses (**e)** for all groups. Note, there is no training data for the observe group as they were not required to make any responses during the training task. Error bars show $\pm 1SE$.

4.3.3. Recognition Task Analysis

All participants in the observe group performed this task well with a high level of accuracy ($M=91.41\%$; $SE=0.74$), indicating that they observed the training task with a sufficient degree of attention.

4.3.4. Consumption Data Analysis

4.3.4.1. Stop-Signal Training Results

Statistical analyses revealed a main effect of food type ($F(1,79)=90.5$, $p<0.001$, $\eta_p^2=0.53$, $f=1.06$) with participants consuming significantly more calories from the unhealthy foods ($M=77.79$, $SE=5.38$) than from the healthy foods ($M=24.03$, $SE=1.92$; see Figure 4.5a). There was also a significant main effect of food novelty ($F(1,79)=8.73$, $p=0.004$, $\eta_p^2=0.1$, $f=0.33$) with a higher calorie consumption of old ($M=58.86$, $SE=3.35$) compared to new foods ($M=42.96$, $SE=4.46$). This effect could be the result of increased preferences for these foods, increased variety or a familiarity effect. Analysis of the food liking scales suggests that this difference is likely to be due to an increased preference for old ($M=7.07$, $SE=0.15$) compared to new foods ($M=5.53$, $SE=0.29$; $t(52)=5.26$, $p<0.001$, $d_z=0.72$); however, as these scores were obtained following food consumption it is uncertain whether increased consumption was due to increased liking or vice versa. The interaction between food type and food novelty was not significant ($F(1,79)=1.83$, $p=0.18$, $\eta_p^2=0.02$, $f=0.14$). Importantly, there was a trend towards significance for the main effect of training condition ($F(1,79)=3.19$, $p=0.078$, $\eta_p^2=0.04$, $f=0.2$) with participants in the stop group consuming fewer calories ($M=45.75$, $SE=4.01$) than those in the double-response group ($M=56.07$, $SE=4.16$). However, training condition did not significantly interact with either food type ($F(1,79)=0.92$, $p=0.34$, $\eta_p^2=0.01$, $f=0.1$) or food novelty ($F(1,79)=0.01$, $p=0.94$, $\eta_p^2<0.001$, $f=0.01$). The three-way interaction between training condition, food type and food novelty was also non-significant ($F(1,79)=0.25$, $p=0.62$, $\eta_p^2=0.003$, $f=0.08$).

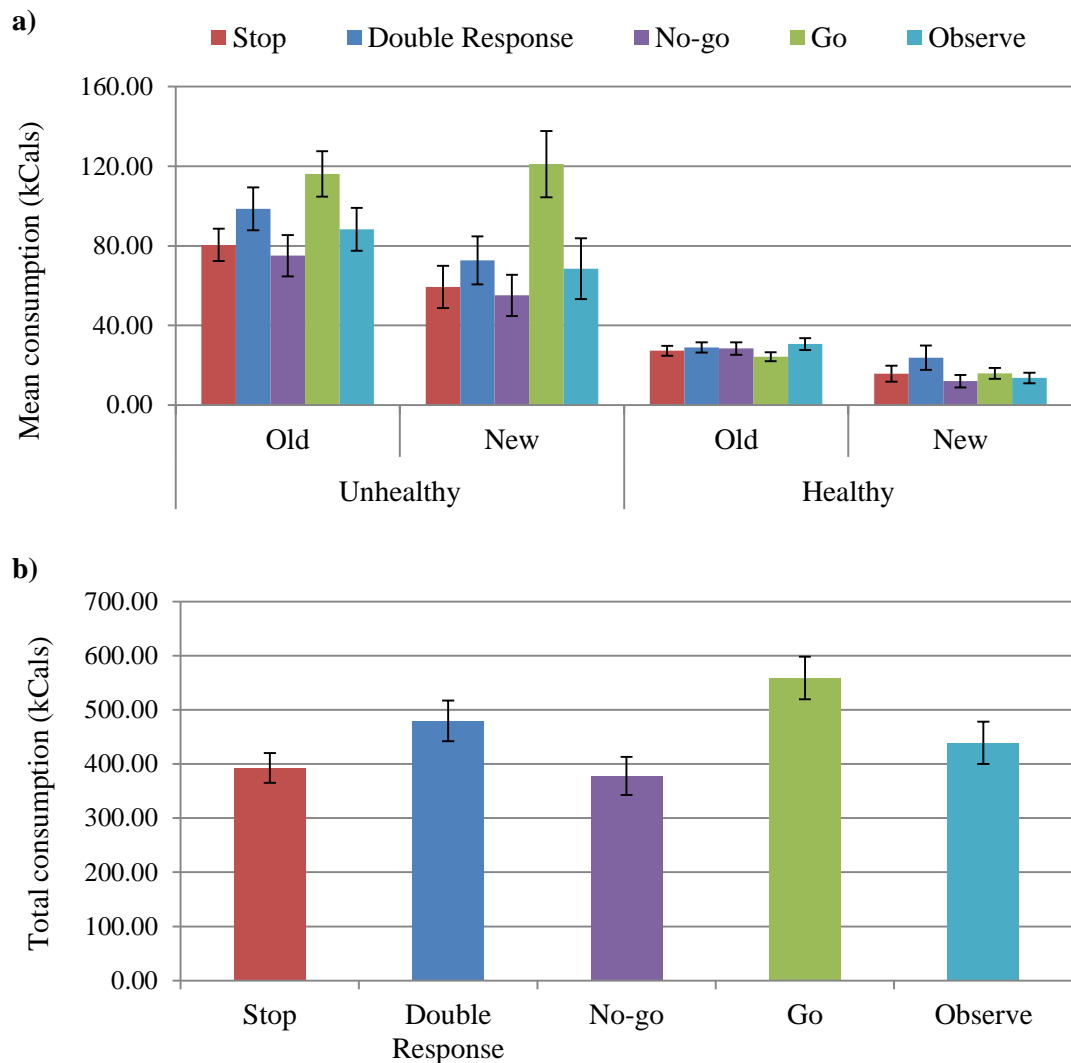


Figure 4.5. a) Mean calorie consumption (total calories / number of foods per category) as a function of training condition for the unhealthy and healthy foods that were presented in both the training and the snack buffet (old) and those that were presented in the snack buffet only (new). **b)** Total calorie intake as a function of training condition. Error bars show $\pm 1SE$.

These results show a trend towards a main effect of training condition but no significant stimulus-specific interactions, thus indicating that participants who received stop-signal training consumed fewer calories overall, compared to those who received double-response training. This was also confirmed with a Bayesian analysis on *total* calorie consumption (see Figure 4.5b; mean difference=87.102, SE of the difference=46.02); the results of which revealed substantial evidence for the experimental hypothesis that inhibition training reduces food consumption relative to

a control task ($B=3.25$; Dienes, 2011, 2014); however, as discussed earlier (section 4.2.4), this Bayes factor is likely to be an overestimate. This finding reflected an 18.16% difference in total calorie intake between the two groups. Analyses for unhealthy (23.55% difference) and healthy (18.22% difference) calorie intake separately were both inconclusive ($0.33 > B < 3$; unhealthy: mean difference = 70.24, SE of the difference = 44.46, $B=2.34$; healthy: mean difference = 17.13, SE of the difference = 12.32, $B=1.10$).

Furthermore, despite the lower calorie intake in the stop group compared to the double-response group, there were no statistically significant differences in post-intake measures of hunger (stop: $M=0.73$, $SE=0.18$; double-response: $M=0.5$, $SE=0.11$; $U=683$, $p=0.83$, $r=0.02$), fullness (stop: $M=5.69$, $SE=0.33$; double-response: $M=5.79$, $SE=0.28$; $U=682$, $p=0.82$, $r=0.03$) or desire to eat (stop: $M=1.72$, $SE=0.34$; double-response: $M=1.48$, $SE=0.3$; $U=643$, $p=0.66$, $r=0.05$). There was also no evidence to indicate that this was due to increased water consumption (measured in grams) in the stop ($M=112.55$, $SE=19.7$) compared to the double-response group ($M=125.6$, $SE=19.77$; $U=656$, $p=0.61$, $r=0.06$; all analyses for hunger scales and water intake were analysed using nonparametric Mann Whitney U tests as data for these variables was not normally distributed and could not be normalised with either a log or square root transformation)⁸.

For the analysis of healthy food consumption, in grams, a mixed 2x2 ANOVA revealed a significant main effect of food novelty showing that the mean consumption of old healthy foods ($M=47.24$, $SE=2.99$) was significantly greater than the consumption of the new healthy food ($M=4.44$, $SE=0.75$; $F(1,80)=190.09$, $p<0.001$, $\eta_p^2=0.7$, $f=1.53$). This finding may be due to the difference in weights between the old and new healthy foods; the old foods included both carrots and

⁸ Data were missing for six participants in the hunger and fullness analyses (4 stop, 2 double-response) and for seven participants in the desire to eat analysis (4 stop, 3 double-response). Six participants (4 stop, 2 double-response) were also missing for the analysis of water consumption as some participants either brought their own water to the study or asked to keep their water for the remainder of the study.

grapes which are considerably heavier than the breadsticks in the new food category. However, the liking scores for old healthy foods ($M=7.01$, $SE=0.2$) were also significantly greater than the liking scales for the novel healthy food ($M=3.02$, $SE=0.38$; $t(60)=9.06$, $p<0.001$, $d_z=1.16$) suggesting that preferences are also likely to play a role. It is also a possibility that this increased liking for old foods is an effect of training as participants were effectively trained to go towards these foods. Nevertheless, there was no statistically significant main effect of training condition ($F(1,80)=0.46$, $p=0.5$, $\eta_p^2=0.01$, $f=0.1$) and no significant interaction between training condition and food novelty ($F(1,80)=0.05$, $p=0.83$, $\eta_p^2=0.001$, $f=0.03$).

4.3.4.2. Go/No-go Training Results

To achieve normality in the consumption data all variables were transformed using a square root transformation. Statistical analyses were performed on the transformed data but all means and standard errors are presented for the non-transformed data for ease of interpretation. The results for the GNG training (Figure 4.5a) revealed a significant main effect of food type ($F(1,64)=218.26$, $p<0.001$, $\eta_p^2=0.77$, $f=1.83$) with a greater consumption of calories from unhealthy foods ($M=91.88$, $SE=6.02$) compared to healthy foods ($M=20.21$, $SE=1.42$). There was also a significant main effect of food novelty with a greater consumption of old ($M=61.0$, $SE=3.94$) compared to new foods ($M=51.08$, $SE=4.94$; $F(1,64)=18.32$, $p<0.001$, $\eta_p^2=0.22$, $f=0.53$). Again, this effect may be due to increased preferences, increased variety or a familiarity effect for the old foods. Analysis of the food liking scales revealed a statistical trend for an increased preference for old ($M=6.98$, $SE=0.16$) compared to new foods ($M=6.40$, $SE=0.31$; $t(46)=1.89$, $p=0.07$, $d_z=0.28$), suggesting that increased preferences may partially explain this difference.

Importantly, there was a significant main effect of training condition, with participants in the no-go group ($M=42.69$, $SE=4.69$) consuming significantly fewer calories than those in the go group ($M=69.41$, $SE=4.69$; $F(1,64)=16.76$, $p<0.001$, $\eta_p^2=0.21$, $f=0.52$). However, this effect was qualified by a significant interaction between training condition and food type ($F(1,64)=20.31$, $p<0.001$, $\eta_p^2=0.24$, $f=0.56$); pairwise comparisons revealed that participants in the no-go group consumed significantly fewer unhealthy calories ($M=65.12$, $SE=8.39$) than the go

group ($M=118.64$, $SE=8.65$; $F(1,64)=23.03$, $p<0.001$, $\eta_p^2=0.27$, $f=0.61$), however, there was no statistically significant difference in consumption for the healthy calories (no-go: $M=20.26$, $SE=1.98$; go: $M=20.17$, $SE=2.04$; $F(1,64)=0.96$, $p=0.33$, $\eta_p^2=0.2$, $f=0.5$). There was also a significant interaction between training condition and food novelty ($F(1,64)=4.08$, $p=0.048$, $\eta_p^2=0.06$, $f=0.25$). Participants in the no-go group consumed significantly more calories from the old foods ($M=51.76$, $SE=5.48$) compared to the new foods ($M=33.62$, $SE=6.89$; $p<0.001$) whereas the go group showed no statistically significant difference in intake between these foods (old: $M=70.26$, $SE=5.65$; new: $M=68.55$, $SE=7.1$; $p=0.12$). The interaction between food type and food novelty ($F(1,64)=1.83$, $p=0.18$, $\eta_p^2=0.03$, $f=0.18$) and the three-way interaction ($F(1,64)<0.001$, $p=0.99$, $\eta_p^2<0.001$, $f=0.001$) were both non-significant.

These results suggest that the effect of GNG training on food consumption may be specific to the foods that were associated with inhibition during training, in this case the unhealthy foods. This finding was confirmed with Bayesian analyses. Firstly, the difference for *total* calorie intake (Figure 4.5b) revealed strong evidence for the hypothesis that inhibition reduces food consumption compared to a control task (mean difference =180.86, SE of the difference =52.52, B=23.09). This reflected a 32.38% difference in total calorie intake between the two groups. Moreover, there was very strong evidence for this hypothesis for unhealthy calorie consumption with a 67.52% difference (mean difference =189.3, SE of the difference =49.17, B=57.4) and substantial evidence for the null hypothesis for healthy food consumption with the no-go group consuming 8.66% more healthy calories than the go group (mean difference =-8.44, SE of the difference =12.34, B=0.15). However, the results of the ANOVA showed no statistically significant interactions with food novelty, indicating that the effect of GNG training on reduced intake may generalise to other similarly unhealthy foods that are not necessarily paired with inhibition during training.

Furthermore, the significant difference in food intake was not accompanied by a difference on any of the post-intake hunger scales. There were no statistically significant differences between training groups for hunger (no-go: $M=0.78$,

$SE=0.16$; go: $M=0.55$, $SE=0.12$; $U=418.5$, $p=0.53$, $r=0.08$), fullness (no-go: $M=5.52$, $SE=0.29$; go: $M=6.05$, $SE=0.39$; $U=378.5$, $p=0.16$, $r=0.18$) or desire to eat (no-go: $M=1.86$, $SE=0.31$; go: $M=1.49$, $SE=0.26$; $U=434$, $p=0.53$, $r=0.08$). In addition, these results were not explained by a difference in water consumption between training groups (no-go: $M=151.18$, $SE=35.79$; go: $M=158.79$, $SE=33.66$; $U=465$, $p=0.85$, $r=0.02$; hunger scales and water consumption were analysed using Mann Whitney U tests as the data were not normally distributed and were not normalised with either a log or square root transformation)⁹.

For the analysis of healthy food consumption, in grams, all data was first transformed using a square root transformation for violation of normality. The results of the ANOVA showed a significant main effect of food novelty with the mean consumption of old healthy foods ($M=43.69$, $SE=3.07$) being significantly greater than the consumption of the new healthy food ($M=3.38$, $SE=0.51$; $F(1,64)=276.39$, $p<0.001$, $\eta_p^2=0.81$, $f=2.06$). Again, analysis of the food liking scales suggests that foods preferences may explain this difference; liking for old healthy foods ($M=6.83$, $SE=0.26$) was significantly greater than liking for the novel healthy food ($M=3.42$, $SE=0.44$; $t(49)=6.31$, $p<0.001$, $d_z=0.89$). The main effect of training condition ($F(1,64)=0.01$, $p=0.91$, $\eta_p^2<0.001$, $f=0.01$) and the interaction between training condition and stimulus type ($F(1,64)=2.7$, $p=0.11$, $\eta_p^2=0.04$, $f=0.2$) were both non-significant.

4.3.4.3. Comparison of all Groups, Including the Observe Group

As most variables were positively skewed statistical analyses were performed on square-root transformed data. All means and standard errors are presented for the non-transformed data for ease of interpretation. The results from the 5x2x2 ANOVA replicated the main effects of both food type (participants consumed significantly

⁹ Data were missing for five participants in the hunger analysis (1 no-go, 4 go) and for four participants in the fullness and desire to eat analyses (1 no-go, 3 go). Four participants (1 no-go, 3 go) were also missing for the analysis of water consumption; some participants brought their own water to the study and others kept hold of their water for the remainder of the study.

more calories from the unhealthy foods than the healthy foods; unhealthy: $M=83.56$, $SE=3.8$; healthy: $M=22.13$, $SE=1.08$; $F(1,174)=295.64$, $p<0.001$, $\eta_p^2=0.63$, $f=1.3$) and food novelty (significantly greater consumption for the old foods compared to the new foods; old: $M=59.85$, $SE=2.33$; new: $M=45.84$, $SE=3.09$; $F(1,174)=60.36$, $p<0.001$, $\eta_p^2=0.26$, $f=0.59$). Importantly, the ANOVA also demonstrated a significant main effect of training condition ($F(4,174)=4.39$, $p=0.002$, $\eta_p^2=0.09$, $f=0.3$). Bonferroni post hoc tests revealed that mean calorie intake in the go group was significantly greater than that for both the no-go ($M=42.69$, $SE=4.61$; $p=0.002$) and stop ($M=45.75$, $SE=4.14$; $p=0.01$) groups. There was also a statistical trend for greater consumption in the go group compared to the observe group ($M=50.31$, $SE=4.75$; $p=0.08$). All other comparisons, including the comparison between stop and double-response groups ($p=1.0$), were non-significant (all $ps>0.45$).

This main effect of training condition, however, was qualified by a significant interaction with food type ($F(4,174)=4.39$, $p=0.002$, $\eta_p^2=0.09$, $f=0.3$). Univariate tests showed that the effect of condition was significant for the consumption of unhealthy foods ($F(4,174)=5.71$, $p<0.001$, $\eta_p^2=0.12$, $f=0.37$) but not for the consumption of healthy foods ($F(4,174)=0.75$, $p=0.56$, $\eta_p^2=0.017$, $f=0.13$)¹⁰. Pairwise comparisons for unhealthy calorie intake showed that consumption in the go group was significantly greater than consumption for the no-go ($p<0.001$), stop ($p<0.001$), double-response ($p=0.005$) and observe ($p=0.001$) groups. All other comparisons, including the comparison between stop and double-response groups ($p=0.25$), were non-significant (all $ps>0.12$). The interactions between condition and food novelty ($F(4,174)=1.21$, $p=0.31$, $\eta_p^2=0.03$, $f=0.18$), food type and food novelty ($F(1,174)=0.74$, $p=0.39$, $\eta_p^2=0.004$, $f=0.06$) and the three-way interaction ($F(4,174)=0.43$, $p=0.79$, $\eta_p^2=0.01$, $f=0.1$) were all non-significant.

¹⁰ Again, an analysis for healthy consumption in grams only revealed a significant main effect of food novelty with increased consumption of old ($M=46.71$, $SE=1.98$) compared to new foods ($M=3.79$, $SE=0.41$; $F(1, 174)=759.79$, $p<0.001$, $\eta_p^2=0.81$, $f=2.06$). The main effect of condition ($F(4,174)=0.64$, $p=0.64$, $\eta_p^2=0.01$, $f=0.1$) and the interaction between condition and food novelty were both non-significant ($F(4,174)=1.24$, $p=0.3$, $\eta_p^2=0.03$, $f=0.18$).

Bayesian analyses provided some support for these findings. Bayes factors for the difference between the go and both the no-go ($B=23.09$) and stop groups (mean difference =166.21, SE of the difference =46.43; $B=40.48$) provided strong and very strong evidence for a statistical difference, respectively. As reported earlier, there was also substantial evidence for a difference in consumption between the stop and double-response groups ($B=3.25$); however, as previously discussed, this effect has to be interpreted with caution as it is likely to be inflated with this study design (see section 4.2.4). This is also true for the Bayes factors for the differences between the double-response and no-go groups (mean difference =101.75, SE of the difference =51.91; $B=3.28$) and between the go and observe groups (mean difference =119.37, SE of the difference =55.26; $B=3.87$) – both of which were only marginally greater than three. All other Bayes factors, including that for the difference between go and double-response groups (mean difference =79.11, SE of the difference =54.53; $B=1.98$), were inconclusive (all other $Bs < 1.59$). The Bayesian analyses therefore support the difference between the go group and inhibition groups but are largely inconclusive for the remaining comparisons.

4.3.5. Debrief Analysis

Participants in the stop, double-response and no-go groups were asked funnelled questions during the debrief to assess awareness of the stimulus mappings. Participants were categorised as ‘aware’ if they reported that the signals were mapped onto unhealthy foods, as ‘partially aware’ if they reported that the signals were mapped onto food in general and all other responses were categorised as ‘unaware’. In the stop training group 46% of participants were considered aware, 31% were partially aware and 23% were not aware. In the double-response group 23% were considered aware, 18% partially aware and 59% not aware and for the no-go group 37.5% were aware and 62.5% were not aware. A Chi square test revealed a statistically significant difference in these frequencies ($\chi^2(4)=12.16$, $p < 0.001$, $V=0.3$), reflecting the finding that more participants were aware of the association between unhealthy food and signals in the stop group compared to the double-response and no-go groups. Similarly, fewer participants were classified as not aware in the stop group compared to the double-response and no-go groups. These findings may reflect an increased cognitive load in the stop-signal training task and increased

attention towards the task. To see whether awareness had any influence on intake, a 3x2x2x2 mixed ANOVA (between subjects factors: *training condition*, stop, double-response and no-go; *awareness*, aware or not aware; within subjects factors: *food type*, unhealthy or healthy and *food novelty*, old or new) was performed. Due to the small number of participants who were categorised as partially aware the aware groups were collapsed to form one aware group. The results of this analysis revealed no significant main effect of awareness ($F(1,104)=0.81$, $p=0.37$, $\eta_p^2=0.008$, $f=0.09$) and no statistically significant interactions involving awareness (all $F_s < 1.47$; all $p_s > 0.25$; all $\eta_p^2_s < 0.03$).

Participants were also probed for awareness of the study's aims during the debrief. Only one participant in the double-response group correctly guessed the aim of the study and was excluded from all analyses. No other participants indicated that they were aware of the study's aims or that food consumption was being measured. The majority of participants (94%) reported not knowing the aim of the study or believed that the study was investigating the effect of blood glucose/hunger on performance. Three participants mentioned self-control or resisting temptation when asked about the study's aims and seven participants believed that the study was related to the effects of food on emotion or mood. When asked whether participants thought that the task had any influence on food intake (specifically, they were asked whether they thought the task had any influence on how they answered the questionnaires, the food they had or their performance on the second task), 16 participants (1 stop, 4 no-go, 1 double-response, 1 go, 9 observe) said that the task made them want unhealthy foods, eight participants (1 stop, 4 no-go, 3 observe) said that the task made them more conscious of or want healthy foods and ten participants (1 stop, 1 double-response, 2 go, 6 observe) said that the task made them hungry or think more about food. Three participants in the stop group also commented on the influence of the stop-signals/ stopping, reporting that it made them want to eat healthily (although they did not), made them think that unhealthy foods were 'bad' or indicated that they

were not allowed unhealthy foods¹¹. The majority of participants (79%) did not report any effect of training on food intake or hunger.

4.4. Discussion

The primary aim of this study was to investigate whether training inhibitory control can reduce food consumption in restrained eaters and to compare the effectiveness of the stop-signal and GNG training tasks. The results of the stop-signal training revealed a trend towards significance for the main effect of training condition with participants in the stop group consuming 18% fewer calories than those in the double-response group. However, there was no statistically significant interaction with food type or food novelty showing that participants in the stop group consumed fewer calories for all food types including novel and healthy foods. The transfer of training effects to novel and healthy foods and the greater involvement of top-down control in the SST may indicate that stop-signal training reduces food consumption by engaging global response inhibition mechanisms or increasing general self-control (Berkman *et al.*, 2009, 2012; Muraven & Baumeister, 2000; Thorell *et al.*, 2009; Verbruggen *et al.*, 2012; Verbruggen & Logan, 2008). These results are inconsistent with the null finding in Study 2 but are consistent with the findings of Lawrence *et al.* (under review) who showed that stop-signal training can effectively reduce food intake compared to a double-response control group. It is possible therefore that the null finding in Study 2 of this thesis was due to the intermediate implicit association test (IAT; Greenwald *et al.*, 1998), between the training task and consumption phase. However, Study 2 also failed to show an effect of stop-signal training on this intermediate task, which is inconsistent with previous findings demonstrating that inhibition training using the GNG task is associated with stimulus devaluation (Houben *et al.*, 2011a, 2012a; Veling *et al.*, 2013b). A second possible

¹¹ Removing these participants from the analysis reduced the mean consumption of the stop group. For the analysis of stop and double-response groups the main effect of condition remained a statistical trend with a slight increase in the effect size ($F(1,76)=3.82, p=0.054, \eta_p^2=0.05$). For the 5x2x2 ANOVA the difference in intake between the stop and double-response groups for the consumption of unhealthy foods remained non-significant ($p=0.19$).

explanation for the null results in Study 2 was that the effect of stop-signal training was smaller than that of no-go training – an explanation which is supported by the results of the present study.

The results from the GNG task revealed a significant main effect of training condition, with a large effect size, reflecting a 32% reduction in food intake in the no-go group compared to the go group. This result is consistent with previous findings showing an effect of food-related no-go training on reduced food consumption (Houben, 2011; Houben & Jansen, 2011; Veling *et al.*, 2011). However, this main effect was qualified by a significant interaction with food type, which showed that training resulted a significant difference in unhealthy, but not healthy food intake. For unhealthy food intake, participants in the no-go group consumed 67% fewer calories than those in the go group. This finding may lend support to the idea that the consistent unhealthy-no-go mapping on this task resulted in the engagement of automatic inhibition and implicit learning of stimulus-stop associations (Spierer *et al.*, 2013; Verbruggen *et al.*, in press; Verbruggen & Logan, 2008). Learning of these associations may have then caused a reduction in unhealthy food intake as these foods became devalued during training (McLaren & Verbruggen, submitted; Veling & Aarts, 2009; Veling *et al.*, 2008, 2013b; Verbruggen *et al.*, in press) and/or automatically retrieved the stop goal when encountered during the snack buffet (Chiu *et al.*, 2012; Lenartowicz *et al.*, 2011; Verbruggen & Logan, 2008). Visual inspection of the data and the non-significant interaction between training condition and food novelty, however, shows that participants in the no-go group also consumed fewer calories for the unhealthy new food that was presented in the snack buffet but not the training task. This transfer to the novel unhealthy food may reflect a generalisation of devaluation and inhibitory processes towards other, semantically similar, foods suggesting that these effects may have been based on concepts rather than specific exemplars. Taken in isolation, these findings would be encouraging from a clinical perspective as they demonstrate a substantial effect of no-go training on reduced unhealthy food consumption that also generalised to a novel unhealthy food. However, a final aim of this study was also to explore whether these effects were due to decreased consumption in the inhibition groups or increased consumption in the control groups. The inclusion of

an additional observe control group and statistical comparison for all training conditions provides some evidence for the latter.

As both the double-response and go tasks involved consistently responding to food it is possible that participants developed an approach bias towards these foods during training, which subsequently increased food intake. In the present study an additional control group was included who simply observed the go task prior to the snack buffet; they therefore received the same level of food exposure but were not required to make any responses. A comparison of all training groups, including the observe group, revealed a significant difference between training conditions for the consumption of unhealthy foods only. This result reflected increased consumption in the go group compared to all other groups, including the observe and double-response groups. Moreover, there were no other statistically significant differences between training groups. The finding that participants in the observe group differed significantly from the go group but not the no-go group suggests that effects of GNG training on food intake are driven by increased consumption in the go group and not by decreased consumption in the no-go group. Consumption in the observe group was intermediate between these two groups which may indicate that both processes occur together but statistical analyses indicate that the effect of go training is stronger than the effect of no-go training. This finding is consistent with other research showing that pairing food with a go or approach response can influence behaviour. For example, Kemps *et al.* (2013b) found that participants who consistently categorised chocolate with approach words, compared to avoid words, experienced a significant increase in chocolate cravings. Furthermore, Schonberg *et al.* (2014) demonstrated that food choices could be biased by pairing certain foods with responding during a GNG task.

Just as it has been argued that inhibition training may reduce food consumption by encouraging the development of stimulus-stop associations and activating an aversive centre (McLaren & Verbruggen, submitted; Verbruggen *et al.*, in press), it is also possible that go training may encourage the development of stimulus-go associations and activation of an appetitive centre. In addition, performing this task may have primed disinhibition generally: Guerrieri *et al.* (2012) found that

participants who performed a non-food-related SST in which the number of go trials increased across blocks consumed significantly more calories on a subsequent taste test than those in the inhibition and neutral-control conditions. However, the finding that food consumption was significantly greater in the go group compared to the double-response group appears at odds with these interpretations; as participants in the double-response group also made consistent go responses, and reinforced this action with a second response on a majority of unhealthy food trials, one would expect greater intake in the double-response group. This finding suggests that the additional elements of the double-response task, such as visual detection and action updating, may prime disinhibition to a lesser extent than the single-response go task – possibly by engaging neural networks that are also involved in the inhibition of responses (Verbruggen *et al.*, 2010). The suggestion that disinhibition is greater in the go task is also supported by the faster reaction times on no-signal trials in the go group compared to the double-response group, although this difference was not statistically significant. These results, however, are inconsistent with those of Lawrence *et al.* (under review) who found reduced calorie intake in the single-response ignore group, relative to the double-response group, despite faster reaction times in the ignore group. It is possible, therefore, that ignoring a signal may also prime inhibition to some extent.

In conclusion, the results of the present study cast some doubt on whether inhibition training can be used as an intervention to reduce food consumption. To date, the majority of studies investigating the potential of this training tool have used control conditions in which participants continually respond to images of palatable, unhealthy foods (see Appendices 1 and 2; Houben, 2011; Houben & Jansen, 2011; Lawrence *et al.*, under review; Veling *et al.*, 2011). Here, I provide evidence which indicates that the differences in post-training food consumption may be a result of increased consumption in the control groups rather than decreased consumption in the inhibition groups. In this study, intake was compared to an additional control group who received the same level of food exposure during training but did not make any responses towards food. Although consumption in this observe group was intermediate between the inhibition and response-control groups, the only statistically significant difference was for increased consumption in the go group.

Furthermore, this group appeared to be particularly disinhibited following training when compared to the double-response group. This study raises the question of which task can be considered to be the most appropriate control – what do we consider to be ‘normal’ food-related behaviour and where is the baseline? For example, it could be argued that we do not normally find ourselves having to view palatable foods for fifteen minutes without being able to touch them. As restrained eaters have been shown to demonstrate strong approach tendencies towards food (Veenstra & de Jong, 2010) it is possible that the observe task acted as another form of inhibition training and actually, the go task is more representative of how we typically interact with food. These results, therefore, do not necessarily negate the influence of inhibition training on reduced food intake.

Perhaps the most convincing evidence to date for the validity of inhibition training is evidence showing effects on actual weight loss (Lawrence *et al.*, in preparation; Veling *et al.*, 2014). Furthermore, both of these studies used a conservative control task in which participants inhibited responses towards non-food images. Assuming that exposure to food images in the experimental groups would increase hunger and food intake to a greater extent (Rogers & Hill, 1989) and that inhibiting responses towards food would be more effortful (Nederkoorn *et al.*, 2012), and consequently more ‘ego-depleting’ (Baumeister, 2003; Kahan *et al.*, 2003; Vohs & Heatherton, 2000), than the control task, it is likely that these effects underestimate the true effect of food-related inhibition training on food intake. The improved success of inhibition training in these studies may be due to the repeated training sessions that took place over a period of several weeks. These findings suggest that in future, researchers wishing to investigate the effectiveness of inhibition training on food consumption should carefully consider more appropriate control tasks and dependent variables as well as methods that may help to enhance training effects. In addition to repeated training sessions, future research could also consider personalised training stimuli, reward-based inhibition training (Kohls, Peltzer, Herpertz-Dahlmann & Konrad 2009; Sinopoli, Schachar & Dennis 2011) or even neuroenhancement. Indeed, two clinical trials (see Alonso-Alonso, 2013) and pilot testing undertaken as part of my doctoral research (see Appendix 11) have begun to investigate whether the application of prefrontal tDCS can be used to strengthen the effect of inhibition

training on food consumption and weight loss. Together, these studies should advance our understanding of whether food-related inhibition training can be used as an effective intervention, and if successful, they should also give insight into the underlying mechanisms and other potential avenues for improvement.

Chapter 5. Study 4

The effect of food-related go/no-go training on implicit and explicit attitudes towards palatable snacks: An internet-based study

5.1. Introduction

Recent studies have shown that inhibitory control training may reduce unhealthy food intake (Houben, 2011; Houben & Jansen, 2011; Lawrence *et al.*, under review, studies 1 & 2; Veling *et al.*, 2011; but see Study 3, Chapter 4) and contribute to weight loss (Lawrence *et al.*, in preparation; Veling *et al.*, 2014). These studies may have promising implications for reducing overeating and obesity; however, the mechanisms underlying these effects remain relatively unexplored. One possible mechanism which may explain these differences is stimulus devaluation. The Behaviour Stimulus Interaction theory (BSI; Veling *et al.*, 2008) argues that the conflict created from inhibiting a response towards a desired object is resolved through stimulus devaluation. By reducing the attractiveness of an object, our motivation towards it is also reduced – and thus, our ability to exert self-control increases (see also McLaren & Verbruggen, submitted). Veling *et al.* (2013b) have provided some evidence for the BSI theory with food-related inhibition training. In their study participants were given a go/no-go (GNG) task in which some foods were consistently paired with a go cue and always required a response, and others were consistently paired with a no-go cue, therefore requiring the inhibition of a response. These pairings were presented four, twelve or 24 times during the training task. After training participants rated each of the food items according to its attractiveness and palatability and were asked to select three snacks for consumption. Veling *et al.* found that for participants who took part in the study before lunch (i.e. with a high appetite), no-go foods were rated significantly less positively than go foods (as the two measures of attractiveness and palatability reacted similarly to inhibition training and were highly correlated with one another, a mean score of these two measures

was used). In addition, they found no effect of the number of pairings on reduced evaluations, suggesting that inhibiting a response towards a desired object can result in devaluation after only a small number of trials. Moreover, participants in the high appetite condition selected fewer no-go foods than go foods, and this effect was shown to be mediated by the evaluation of no-go foods.

Further evidence for the mediating role of stimulus devaluation comes from Houben *et al.* (2011a, 2012a) who explored the effect of inhibition training on alcohol consumption and implicit attitudes. Implicit attitudes were measured using the implicit association test (IAT; Greenwald *et al.*, 1998). As discussed in the literature review, the IAT is designed to measure the strength of implicit, or automatic, associations that an individual holds between two concepts. For example, in the beer IAT used by Houben *et al.*, participants had to categorise images of beer and water with both pleasant and unpleasant words. If an individual is faster to respond in blocks when beer is paired with pleasant words and water is paired with unpleasant words than vice versa, the individual is believed to hold a positive attitude towards beer – and the strength of this difference reflects the strength of the attitude. The main advantage of the IAT is that it is believed to measure attitudes without engaging conscious awareness, and is therefore thought to be more resistant to demand characteristics and self presentation compared to explicit attitude measures (Greenwald *et al.*, 1998). Houben *et al.* (2011a) found that a decrease in weekly alcohol intake following beer-related GNG training was accompanied by a significant increase in negative attitudes towards beer on the IAT. Furthermore, this finding was later replicated when Houben *et al.* (2012a) demonstrated that the effect of inhibition training on reduced alcohol intake was mediated by changes in IAT scores but not by changes in response inhibition or approach-avoidance tendencies (but see Bowley *et al.*, 2013).

Together these findings suggest that inhibition training may influence consumption behaviour by devaluing the trained stimuli both at an explicit and implicit level. However, in the second study presented in this thesis (see Chapter 3, Study 2), I found no evidence for an effect of chocolate-related stop-signal training on either positive or negative implicit attitudes towards chocolate. Assuming that these

findings do not reflect a statistical false negative, there are three possible explanations for these conflicting findings. Firstly, it is possible that these differences are due to the use of different dependent measures. In Study 2 implicit attitudes were measured with two unipolar, single-category versions of the IAT (SC-IAT; Karpinsky & Steinman, 2006) rather than the relative measure used by Houben *et al.* (2011a, 2012a). It is possible that the effect of inhibition training on implicit attitudes reported by Houben *et al.* was due to the devaluation of the no-go stimuli *relative* to the go stimuli. In their beer/no-go training task, images of water (2011a) or empty glasses (2012a) were consistently paired with a go response, which may have resulted in a positive approach association. The purpose of the unipolar SC-IAT in Study 2, however, was to measure both positive and negative implicit attitudes separately for the single target category of chocolate. Furthermore, with regards to measuring implicit food attitudes in restrained eaters, there is research to support the use of unipolar SC-IATs rather than the relative IAT. Restrained eaters are believed to hold an ambivalent attitude towards calorific foods with both strong positive and strong negative attitudes (Hoefling & Strack, 2008; Keller & van der Horst, 2013; Urland & Ito, 2005). When comparing these two tasks, Houben *et al.* (2010a) found no difference for high and low calorie foods between restrained and unrestrained eaters on the relative IAT – both groups showed a strong negative attitude towards high calorie foods. For the SC-IAT, conversely, participants demonstrated a strong positive attitude towards unhealthy snacks, and this finding was significantly stronger in those with high dietary restraint. Therefore, unipolar SC-IATs appear to be a more valid measure than the relative IAT for assessing implicit attitudes towards unhealthy foods in restrained eaters.

The second possible explanation for the difference between the findings of Study 2 and those of Houben *et al.* (2011a, 2012a) is the difference between training tasks. Houben *et al.* trained participants to inhibit their responses using the GNG task whereas I used the SST (see section 4.1.1. for a more detailed discussion of the differences between these two tasks). It is thought that the consistent associations in the GNG task may increase the likelihood of participants engaging automatic control and learning an association between the stimulus and stopping. Training on the SST, on the other hand, may be more reliant on top-down control and may result in

stimulus-*signal* rather than stimulus-stop associations (Shiffrin & Schneider, 1977; Spierer *et al.*, 2013; Verbruggen & Logan, 2008; Verbruggen *et al.*, in press). If stimulus devaluation is dependent on frequent and successful stopping and the learned association between a stimulus and inhibition (rather than a signal), it seems reasonable to suggest that stimuli are more likely to be devalued on the GNG task compared to the SST. Indeed, in Study 3 (Chapter 4) I found that the difference in food intake between the inhibition and control groups was greater for the GNG task compared to the SST; however, this effect appeared to be due to increased consumption in the go group rather than decreased consumption in the no-go group. This raises the possibility that GNG training may reveal an effect on implicit attitudes towards food on the SC-IAT, although significant findings may reflect increased evaluations in the go group rather than devaluation in the no-go group (as approach training may be associated with the activation of an appetitive centre; McLaren & Verbruggen, submitted). However, in Veling *et al.*'s (2013b) study, investigating the effect of inhibition training on food choice, they reported an effect of appetite on explicit evaluations of no-go but not go foods, suggesting that training resulted in the devaluation of no-go foods but did not affect the value of go foods (see also Fenske & Raymond, 2006; Veling *et al.*, 2008). The third possible explanation, however, is that food-related implicit attitudes may be particularly difficult to modify; food-related inhibition training may only devalue the stimulus at an explicit, and not an implicit, level.

In the present study I therefore investigated whether food-related GNG training had any effect on implicit and explicit attitudes towards palatable foods. Participants received a training task in which they either consistently responded to all foods (go group) or they responded only to the healthy foods and consistently inhibited their responses to the unhealthy snack foods (no-go group). This training was then followed by either two unipolar SC-IATs measuring positive and negative implicit attitudes towards snack foods (Study 4a), or an explicit evaluation task (Study 4a), in which participants rated the attractiveness, 'tastiness' and their desire to eat each of the foods. Consistent with previous studies in this thesis and other research showing an effect of inhibition training only for restrained eaters (Houben & Jansen, 2011; Lawrence *et al.*, under review; Veling *et al.*, 2011), participants were also

categorised according to their restraint status. In addition, I also investigated whether these effects were specific to the trained stimuli or whether they generalised to novel items presented in the attitude tasks. For the SC-IATs it was expected that restrained eaters would show weaker positive and/or stronger negative implicit attitudes towards snack foods in the no-go group compared to the go group. It was also expected that any effects may be limited to or stronger for the trained, compared to the novel stimuli. Similarly, for the explicit evaluation task it was expected that restrained eaters in the no-go group would evaluate the foods less positively than participants in the go group and that this effect would be greater for the trained stimuli. This study was conducted as an online study and therefore no measure of food intake was recorded.

Running psychology studies over the internet has several advantages compared to lab-based studies (Reips, 2002). One of the greatest advantages for the researcher is the potential for large sample sizes and increased statistical power (Musch & Reips, 2000). Furthermore, data collection is fast and inexpensive and online samples are believed to be more diverse, making it easier to generalise results to a wider population. Web-based research is also thought to have greater external validity as participants engage with experimental manipulations in a more naturalistic environment. Results are also thought to be more replicable with standardised experimental procedures including the removal of experimenter effects and reduction of demand characteristics (Klein *et al.*, 2012). However, there are also some disadvantages of web-based experiments. For example, the removal of experimenter interaction also means that participants may misunderstand instructions, although this can often be avoided with detailed instructions, practice blocks and immediate feedback. Another issue is the lack of experimental control with the possibility of increased variability in the data due to increased distracter effects and reduced motivation. Nevertheless, evidence suggests that web-based experiments tend to have good reliability and validity (Crump, McDonnell & Gureckis, 2013; Haworth *et al.*, 2007; Krantz & Dalal, 2000). In addition, with most studies it is important to show that effects generalise outside the lab to the ‘real world’ despite a reduction in experimental rigour. Other issues that need to be considered with online testing are multiple participations – although research indicates that this is rare (Krantz & Dalal,

2000; Musch & Reips, 2000) – and dropout. Dropout rates for online testing can be fairly high; Musch and Reips (2000) reported an average dropout rate of 34% with a range between 1– 87%. Providing participants with information about the nature of the study, the expected time commitment and financial incentives can all help to reduce dropout rates. Large internet studies with cognitive tasks and brain training have already been conducted with some success (Haworth *et al.*, 2007; Owen *et al.*, 2010; Wiers, Houben, Fadardi, van Beek, Rhemtulla & Cox, 2015), including a large-scale project for measuring implicit attitudes with the IAT (Project Implicit; <https://implicit.harvard.edu/implicit/>; e.g. Greenwald, Smith, Sriram, Bar-Anan & Nosek, 2009). Moreover, Veling *et al.* (2014) and Lawrence *et al.* (in preparation) have conducted online studies with food-related GNG training. Their results have shown that participants were able to perform the task correctly and also lost weight as a result of these web-based interventions (see also Jones, McGrath, Houben, Nederkoorn, Robinson & Field (2014) for a published study protocol for a web-based, inhibition training intervention for problematic alcohol use).

5.2. Method

5.2.1. Participants

Participants were recruited through pre-existing databases, electronic advertisements and an online experimental management system at Cardiff University and the study was also promoted via social media (specifically, Twitter and Facebook). Three hundred and twelve participants signed up to participate in the study; 153 fully completed Study 4a (three participants failed to finish the computer tasks and nine failed to complete the questionnaires) and 133 completed Study 4b (three participants failed to finish the computer tasks and 11 failed to complete the questionnaires). The total dropout rate was 8.33%. For the SC-IAT group (Study 4a), 69 participants were randomly assigned to the no-go training (61 females; age range 18-44; $M=20.81$; $SE=0.53$; three participants chose not to provide their demographic information) and 84 were randomly assigned to the go training (71 females; age range 18-48; $M=21.67$; $SE=0.62$; one participant did not report their age). For the stimulus evaluation task, 74 performed the no-go training (60 females; age range 18-59; $M=22.46$; $SE=1.06$) and 59 the go training (51 females; age range 18-68;

$M=21.6$; $SE=0.97$; two participants chose not to provide their age). Psychology undergraduate students participated in the study for course credit. For non-students a small financial incentive was given to reimburse participants and encourage study completion; for every participant we donated 50p to the mental health charity MIND and participants were also offered the chance to enter a prize draw for a £20 shopping voucher (one voucher was given for every 50 participants). The study was approved by the School of Psychology Research Ethics Committee, Cardiff University.

5.2.2. Experimental Distribution and Procedure

All tasks were programmed by the experimenter with Tatool (von Bastian, Locher, & Ruffin, 2013), an open-source, Java-based software designed for running cognitive training tasks locally and over the internet via Java WebStart. When Tatool is downloaded by the user, the programme runs on the local computer (termed ‘client-side’ processing) and not through the internet connection (termed ‘server-side’ processing), thus avoiding timing issues related to internet connectivity. Data is uploaded automatically to a local server where it can be downloaded by the experimenter.

The use of Tatool requires the researcher to programme in HyperText Markup Language (HTML), Extensible Markup Language (XML) and Java; Java is an object-oriented programming language that differs substantially from procedural-based languages such as psychtoolbox. Furthermore, the deployment of this internet-based study required the development of a web server for hosting Tatool modules and data storage as well as a website for the presentation of study information and experimental links. This website provided participants with a summary of the study procedure, ethical information and a step-by-step guide for how to run Tatool (with screen shots for different browsers to make the process as coherent as possible).

Participants were given the option of running either Study 4a or Study 4b, which unknown to the participants referred to the GNG training followed by the SC-IAT task and the GNG training followed by the evaluation task, respectively (see Figure 5.1). Allocation to go and no-go groups was randomised using Tatool. Participants

were instructed to run the task with minimal distractions, for example they were asked to sit in a quiet room where they were unlikely to be disturbed. They were told to run the study from either a laptop or computer as pilot testing revealed that the Tatoon software was not compatible with tablet or smart phone devices. Although this is not ideal for future distribution of the task as a potential clinical tool, this was useful to restrict responses to keyboard presses in accordance with previous studies presented in this thesis¹².

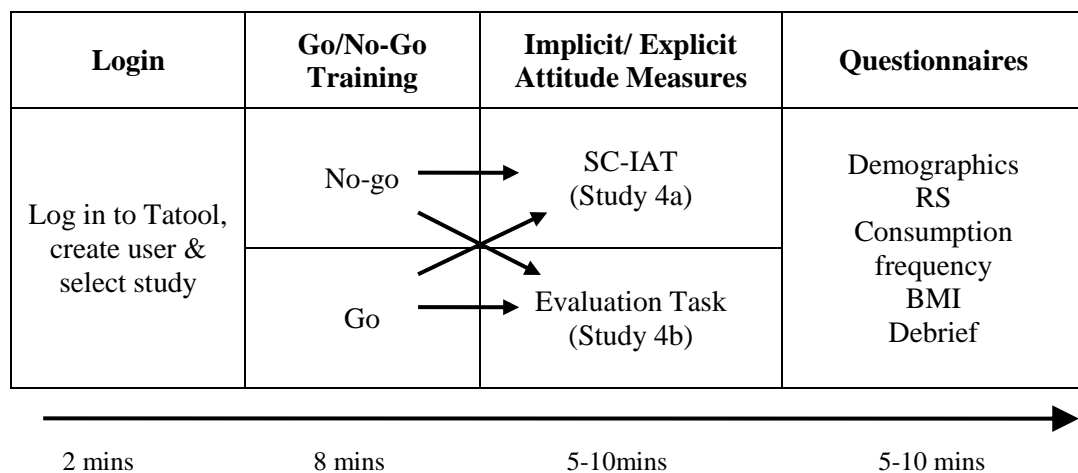


Figure 5.1. Schematic diagram of the study procedure. Participants logged into Tatoon and selected either Study 4a or Study 4b. They were then randomly allocated to either the no-go or go training task (see section 5.2.3.1). Following training they completed either the SC-IAT (see section 5.2.3.2) or evaluation task (see section 5.2.3.3); unknown to the participant this task was determined according to whether they selected Study 4a or Study 4b, respectively. At the end of the study participants were directed to an online survey where they optionally answered demographic questions, the Restraint Scale (RS; Herman & Polivy, 1980), frequency of consumption measures for each of the snack foods and reported their height and weight. They were also asked to guess the aim of the study (see section 5.2.3.4).

¹² Touch screen inhibition training has already been utilised by Veling *et al.* (2013a) and demonstrated statistically significant effects on food choices. This method has great ecological validity relating to how we naturally interact with food substances and requires further exploration, especially with effects on food consumption.

When Tatool was loaded, participants were presented with an electronic consent form and instructions for the training task. These instructions were presented in written format and also as a tutorial. The tutorial presented three trials with a central fixation rectangle (see the Go/No-Go Training section below) and the following messages presented inside (4000 ms): “Press ‘C’ when the stimulus appears on the left” – displayed on the left hand side; “Press ‘M’ when the stimulus appears on the right” – displayed on the right hand side, and the third trial was dependent on the training conditions: participants in the go group saw the message “Remember: respond as quickly as you can”; participants in the no-go group saw the message “Remember to stop your response when the rectangle flashes bold”. Participants then completed the GNG training practice and experimental blocks (see 5.2.3.1 below), followed by the second task which was either the SC-IATs (see 5.2.3.2 below) or the evaluation task (see 5.2.3.3 below). Following the computer tasks participants were given a link to an online survey. After answering the questionnaires (see 5.2.3.4 below) they were presented with a debrief page and were told to contact the lead researcher if they had any questions or wished to be entered into the prize draw.

5.2.3. Materials/ Measures

5.2.3.1. Go/No-Go Training

The GNG training task involved a practice block of 12 trials and four experimental blocks of 36 trials (144 experimental trials in total). The task lasted approximately eight minutes with a five second break between each block. The number of blocks was reduced compared to previous studies in this thesis to encourage participants to complete the study. With an online experiment it was important to keep the study time as short as possible to attract more participants and minimise dropout rates. As previous studies have shown effects of inhibition training on food consumption and evaluations with shorter training tasks (Veling *et al.*, 2011, 2013b), 144 trials was considered appropriate.

The stimuli for this task consisted of nine images of unhealthy snack foods (three each of crisps, chocolate and biscuits), nine images of healthy foods (three each of salad foods, fruit and rice/rye crackers), and 18 filler stimuli of clothing items (three

each of jeans, shirts, jumpers, socks, skirts and ties; see Appendix 8). The unhealthy snack foods were selected based on pilot data showing that these were the most commonly reported unhealthy snack foods in a UK population sample (Lawrence *et al.*, in preparation). All stimuli were selected carefully and were matched as closely as possible for visual complexity and size. All images were presented against a white background. For each trial a rectangle was presented in the centre of the screen for fixation (1250ms), a stimulus then appeared to the left or right of centre (1250ms). Participants were instructed to respond as quickly and as accurately as possible to the location of the stimulus using their left and right index fingers (pressing ‘C’ for left and ‘M’ for right). For participants in the no-go group a signal occurred on 50% of trials; the line of the central rectangle was presented in bold, indicating that the participant must withhold their response for that trial.

The trials for the practice block were fixed to ensure that the task was identical across all participants and involved the same responses (six images were presented to either location) and stimuli (three unhealthy foods, two healthy foods and seven filler stimuli). This also meant that the no-go group received the same number of signal trials, with signals mapped onto the same stimuli (one unhealthy food, two healthy foods and two filler stimuli). Participants were provided with feedback on each trial (700ms; for no-signal trials: ‘correct location’, ‘incorrect location’, ‘miss’; for signal trials (no-go group only): ‘correct stop’, ‘failed stop’). For the experimental trials, each stimulus type was presented once per block in a random sequence and with equal probability to the left or right hand-side. For the no-go group all of the unhealthy foods were presented alongside a signal (100% mapping), none of the healthy foods were mapped onto a signal (0% mapping) and half the filler stimuli were presented with a signal (50% mapping). Feedback was not provided in the experimental blocks.

5.2.3.2. Unipolar, Single-Category Implicit Association Test (Study 4a)

Two unipolar, Single-Category Implicit Association Tests (SC-IAT; Greenwald *et al.*, 1998; Houben *et al.*, 2010a; Karpinsky & Steinman, 2006) were used to measure positive and negative implicit attitudes towards palatable snack foods. The target category was ‘snack’ and involved two images of chocolate, two images of crisps

and two images of biscuits (see Appendix 9). For each of these food types one image was identical to that used in the training task (*image type* old) and the second image was a novel exemplar (*image type* new). For the positive SC-IAT the attribute categories were pleasant (words: delicious, delightful, great, heavenly, outstanding, tasty; label ‘pleasant’) and neutral (words: adequate, average, general, moderate, ordinary, undefined; label ‘neutral’). For the negative SC-IAT the attribute categories were unpleasant (words: awful, bad, disgusting, horrible, nasty, revolting; label ‘unpleasant’) and neutral. Words were selected and matched according to their concreteness, familiarity, imagability, number of syllables, verbal frequency (Brown verbal frequency) and written frequency (Thorndike-Lorge and Kucera-Frances written frequency measures; based on the MRC Psycholinguistic Database; see Appendix 4; footnote 4).

Both SC-IATs consisted of a practice block followed by two experimental blocks. In the practice block participants categorised the attribute categories (e.g. pleasant and neutral words for the positive SC-IAT). There were 24 trials with each word appearing twice in a randomised order. The experimental blocks consisted of one congruent block in which the snack foods were paired with either pleasant or unpleasant words (according to the positive and negative SC-IATs, respectively) and one incongruent block in which snack foods were paired with neutral words. To ensure an approximately equal number of key responses, a 5:2:5 ratio was used so that snack images were repeated five times (30 trials), attributes paired with these images were repeated twice (12 trials) and attributes not paired with the images were repeated five times (30 trials).

Participants were asked to categorise the words as quickly and as accurately as possible with the ‘C’ and ‘M’ buttons. Category labels were presented at the bottom of the screen, in the corner corresponding to the appropriate key press, throughout the block duration. All stimuli were presented in the centre of the screen for 1500ms and participants were provided with feedback in the centre of the screen for every trial (for a correct or incorrect categorisation a green tick or red cross appeared for 200ms, respectively; for a missed response the message “too slow!” appeared for 1500ms). The order of the positive and negative SC-IATs, the congruent and

incongruent blocks and the response mapping were counterbalanced across participants.

5.2.3.3. Explicit Stimulus Evaluation Task (Study 4b)

The evaluation task was designed to assess explicit ratings of the foods presented during training. Images of food were presented in the centre of the screen while participants answered questions relating to the attractiveness and tastiness of the item and their desire to eat the item (see Figure 5.2). Following the task instructions there was a short practice block with four trials including non-experimental foods (a sandwich, a seeded flapjack, a jacket potato with cheese and spring rolls). The experimental block randomly presented all 18 food images from the training task with nine additional novel unhealthy foods (five sweet foods: ice-cream, sponge pudding, doughnuts, cream cake, blueberry muffin; four savoury foods: chips, vegetable pizza, vegetable lasagne, savoury pie; see Appendix 10). Images were presented in the centre of the screen for 2000ms before the onset of the first question. Questions were presented at the bottom of the screen; the first two questions asked participants to rate the attractiveness and tastiness of the item, in a counterbalanced order (between-participants), using a nine-point scale (“How attractive do you rate this item to be?”, rated from 1 “not at all attractive” to 9 “very attractive”; “How tasty do you rate this item to be?”, rated from 1 “not at all tasty” to 9 “very tasty”). The third question asked participants to rate how strong their desire was to eat the item using a nine-point scale (How strong is your desire to eat this item right now?”, rated from 1 “no desire” to 9 “very strong desire”). This question was always asked last so that visualisations of eating the food item did not influence ratings of attractiveness or tastiness. Answers were always anchored on the neutral response (number 5) and participants were asked to make their response by decreasing or increasing this value using the ‘C’ and ‘M’ keys, respectively. There was no limit on the presentation time for each question; participants were instructed to consider each question carefully. The task was self-timed so that participants made a key response to move onto the next question or stimulus.

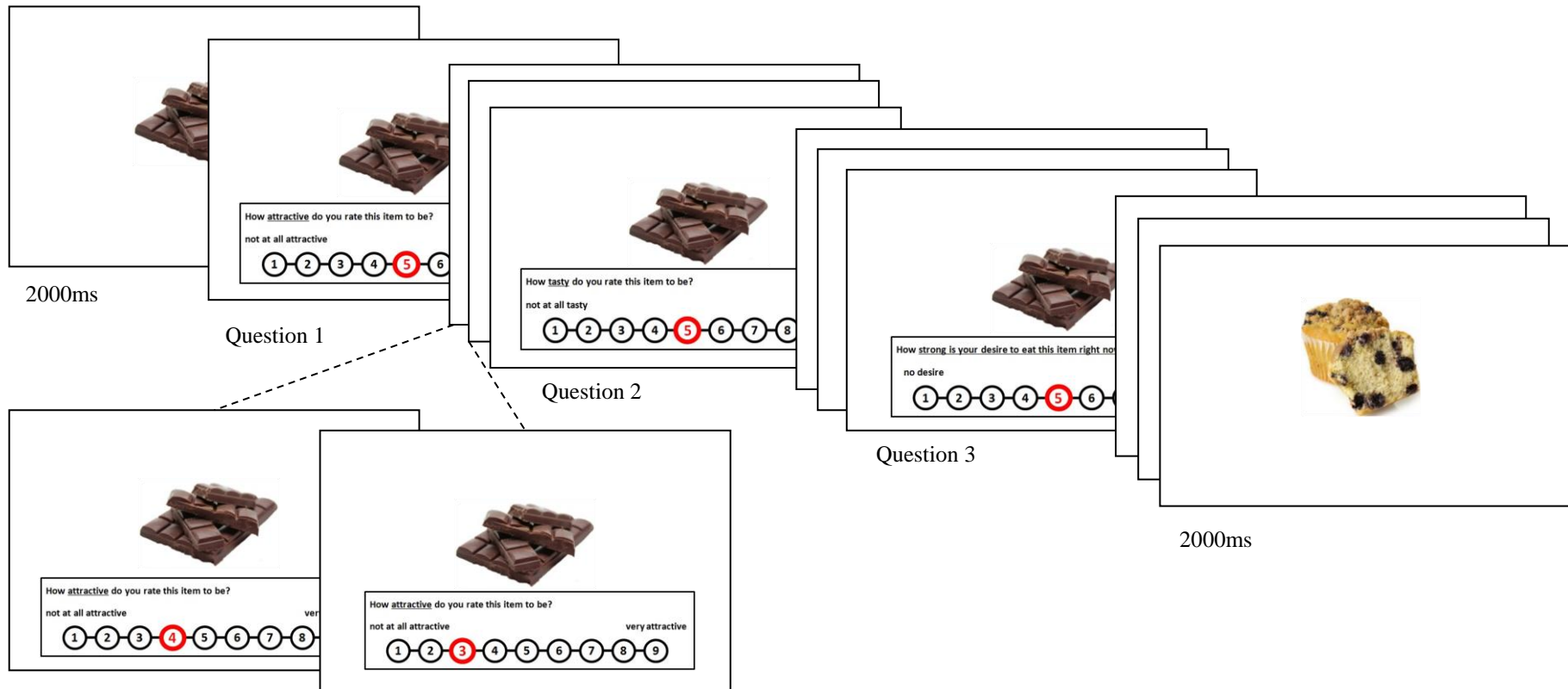


Figure 5.2. Schematic diagram of the evaluation task. Participants are presented with a food item in the centre of the screen (2000ms) and are then asked to rate the attractiveness and tastiness of the food (in a counterbalanced order) before being asked to indicate the strength of their desire to eat the food item. The questions appear at the bottom of the screen with a nine-point scale; participants must press the ‘C’ and ‘M’ buttons to decrease or increase the rating, respectively. The diagram shows the display sequence for a participant responding twice with the ‘C’ button to decrease the attractiveness rating of the food item from five to three. Participants then press a key to confirm their rating and move on to the next question or stimulus.

5.2.3.4. Questionnaires

Following both tasks participants followed a link to an online survey. They were asked to report their age and gender (optionally) and were presented with the Restraint Scale (RS; Herman & Polivy, 1980; see 2.2.2.1 for full details). As frequency of consumption has previously been shown to moderate the effect of inhibition training on food choice (Veling *et al.*, 2013a), participants were asked to indicate their consumption frequency, on a seven-point scale (from ‘Never’ to ‘Daily’), for chocolate, crisps and biscuits. They were then asked to report their height and weight (to calculate BMI; kg/m²) and were asked to guess the aim of the study.

5.2.4. Statistical Analysis

For consistency between this study and others presented in this thesis, all participants were categorised as either restrained or unrestrained according to the Restraint Scale (Herman & Polivy, 1980) and analyses were conducted with restraint status as a between-subjects factor. In accordance with previous research, participants with a score of 15 or more were considered restrained and those scoring less than 15 were considered unrestrained (e.g. Houben & Jansen, 2011; Polivy & Herman, 1999; Roefs *et al.*, 2005)¹³.

Group differences were analysed separately for each study (Study 4a: SC-IAT; Study 4b: explicit stimulus evaluation task) to ensure that there were no statistically significant differences for gender distribution, age, BMI or frequency of snack consumption. As the majority of continuous variables were not normally distributed, and could not be normalised with either a square root or log transformation, nonparametric Kruskal-Wallis tests were performed with follow-up Mann Whitney U tests.

¹³ Moderated regression analyses (using the modprobe SPSS macro; Hayes & Matthes, 2009) revealed that RS scores did not significantly interact with condition to predict any of the SC-IAT scores (all $t_s < 1.17$, all $p_s > 0.24$, all $\Delta R^2 < 0.01$) or any of the explicit evaluation ratings (all $t_s < 1.06$, all $p_s > 0.29$, all $\Delta R^2 < 0.05$) and therefore these analyses were not explored further.

Training data were analysed to ensure that participants performed the tasks as expected and to guide exclusions. Participants were excluded if their performance on no-signal trials was below 85% (including incorrect location responses and missed trials) or their reaction time on no-signal trials (goRT) was $>3SDs$ from the group mean for that training condition. Participants who performed the no-go training task were also excluded if their percentage of erroneous responses on signal trials (i.e. their commission error rate) was $>3SDs$ from the group mean. For Study 4a nine participants were excluded from the no-go group based on their training data: six participants were excluded based on their commission error rate (these participants had 80-100% errors indicating that they did not follow the task instructions), two were excluded due to their performance accuracy on no-signal trials and one due to their goRT. Two participants were excluded from the go group: one for their performance accuracy on no-signal trials and one due to their goRT. For Study 4b only one participant from the no-go group was excluded for a high commission error rate (45.83%).

In Study 4a implicit positive and negative attitudes towards snack foods were explored with two, unipolar SC-IATs. Seventeen participants (7 no-go and 10 go) were excluded from analysis based on $>20\%$ error rate for (Karpinsky & Steinman, 2006). IAT bias scores were calculated using a scoring algorithm modelled on the *D*-score algorithm for the IAT (Greenwald, Nosek & Banaji, 2003). This method was identical to that used in Study 2 (see section 3.2.4. for full details). One sample *t*-tests were first performed for each bias score to see whether attitude scores significantly differed from zero. A mixed $2 \times 2 \times 2$ ANOVA with between-subjects variables *training condition* (no-go or go) and *restraint status* (restrained or unrestrained) and the within-subjects variable *SC-IAT* (positive and negative) was then performed on bias scores. To investigate whether any effects of training were stimulus-specific, bias scores for snack food images were analysed with a mixed $2 \times 2 \times 2 \times 2$ ANOVA including the additional within-subjects variable of *image type* (old or new). After two additional exclusions (one participant in the no-go group correctly guessed the aim of the study and another participant in the no-go group was excluded for timing issues in their data) there was a final sample of 123 participants

in Study 4a (74 no-go and 89 go). A sensitivity analysis (using G*Power; Faul *et al.*, 2007) for the main interaction between training condition, restraint status and SC-IAT revealed that this sample had 80% power to detect a minimum effect size of $f=0.22$ ($\alpha=0.05$, number of groups =4, number of repetitions =2, correlation among repeated measures =-0.029, non-sphericity correction =1).

For Study 4b explicit ratings for attractiveness, tastiness and desire to eat were analysed separately¹⁴ with three mixed 2x2x3 ANOVAs with *condition* (no-go or go) and *restraint status* (restrained or unrestrained) as between-subject variables and *food type* (unhealthy-old, healthy-old and healthy-new) as the within-subjects variable. After four additional exclusions were made (four participants in the no-go group guessed the aim of the study) there was a total sample of 128 participants in Study 4b (69 no-go and 59 go). A sensitivity analysis (using G*Power; Faul *et al.*, 2007) for the main interaction between training condition, restraint status and food type revealed that this sample had 80% power (with $\alpha=0.05$, number of groups =4, number of repetitions =3) to detect a minimum effect size of $f=0.18$ for attractiveness (most conservative estimate with correlation among repeated measures =0.21, non-sphericity correction =0.88), $f=0.19$ for tastiness (most conservative estimate with correlation among repeated measures =0.26, non-sphericity correction =0.80) and $f=0.19$ for desire to eat (most conservative estimate with correlation among repeated measures =0.22, non-sphericity correction =0.72).

All results are reported with unadjusted significance values; corrections for multiple comparisons were calculated for all within-test analyses and are only reported where these corrections changed the interpretation of an analysis from statistically significant to non-statistically significant. All statistical analyses were performed with SPSS.

¹⁴ Measures were significantly and positively correlated with one another, however, these measures were analysed separately as all $r_s < 0.78$.

5.3. Results

5.3.1. Study 4a: Implicit Evaluation Results

5.3.1.1. Group Differences

Descriptive values for RS score, age, BMI and frequency of snack consumption (crisps, biscuits, chocolate) are presented in Table 5.1. A Kruskal-Wallis test revealed a significant effect of restraint across the four groups ($\chi^2(3)=90.44$, $p<0.001$, $\eta^2=0.74$). Importantly, follow-up Mann-Whitney U tests showed that there was no statistically significant differences between restrained eaters in the no-go vs. go groups ($U=554$, $p=0.67$, $r=0.05$) and no statistically significant difference between unrestrained eaters in the no-go vs. go groups ($U=259$, $p=0.88$, $r=0.02$). All tests between restrained and unrestrained eaters were statistically significant after correcting for multiple comparisons ($\alpha/6=0.008$; all $U_s<0.001$, all $p_s<0.001$, all $r_s>0.75$). Gender distribution across groups was fairly even; the percentage of females in restrained groups was greater than that for unrestrained groups, which is consistent with findings showing that females score higher on the RS than males (see Study 1; e.g. Wardle, 1986), however, within each restraint group gender was evenly distributed between training conditions (due to the small number of males in each group a Chi square analysis was not conducted). Groups were also well matched for age, BMI and frequency of snack consumption; after correcting for multiple comparisons there were no statistically significant differences between groups ($\alpha/3=0.017$; all $\chi^2<6.06$, all $p_s >0.11$, all $\eta^2<0.05$).

Table 5.1. Group characteristics for mean RS score, gender distribution, and mean age, BMI and frequency of snack consumption (SE within parentheses).

	No-go		Go	
	Unrestrained (<i>n</i> =13)	Restrained (<i>n</i> =38)	Unrestrained (<i>n</i> =41)	Restrained (<i>n</i> =31)
RS	9.84 (0.88)	19.89 (0.6)	9.71 (0.47)	19.65 (0.73)
Gender (% female)	69.2	94.7	75.6	90.3
Age	20.77 (0.84)	21.16 (0.89) ¹	21.76 (0.86)	22.2 (1.19) ¹
BMI	22.81 (1.26)	24.63 (0.78) ²	22.2 (0.46)	23.83 (0.92) ¹
Snack consumption freq.	4.31 (0.41)	4.94 (0.18)	4.9 (0.22)	4.44 (0.22)

Note. Superscript denotes the number of participants missing for that variable. RS= Restraint Scale; BMI= body mass index

5.3.1.2. Training Data Analysis

Training data is presented in Figure 5.3. For the no-go training task, the overall commission error rate (incorrect responses on signal trials; Figure 5.3a) was low for both restrained and unrestrained eaters (restrained: $M=6.83$, $SE=1.07$; unrestrained: $M=5.88$, $SE=2.56$; $U=194.5$, $p=0.25$, $r=0.16$). For GoRTs there was evidence of proactive slowing with participants in the no-go groups ($M=524.42$, $SE=9.21$) responding significantly slower than participants in the go groups ($M=371.53$, $SE=6.82$; $F(1,119)=177.89$, $p<0.001$, $\eta_p^2=0.6$, $f=1.22$; Verbruggen & Logan, 2009b; Figure 5.3b). Both the main effect of restraint status ($F(1,119)=0.24$, $p=0.62$, $\eta_p^2=0.002$, $f=0.04$) and the interaction were non-significant ($F(1,119)=0.01$, $p=0.93$, $\eta_p^2<0.001$, $f=0.008$). The percentage of errors on no-signal trials was low across all groups (see Figure 5.3c). There was no significant main effect of condition ($F(1,119)=0.98$, $p=0.32$, $\eta_p^2=0.008$, $f=0.09$) or restraint status ($F(1,119)=0.16$, $p=0.69$, $\eta_p^2=0.001$, $f=0.03$) and no statistically significant interaction between the two ($F(1,119)=0.53$, $p=0.47$, $\eta_p^2=0.004$, $f=0.06$).

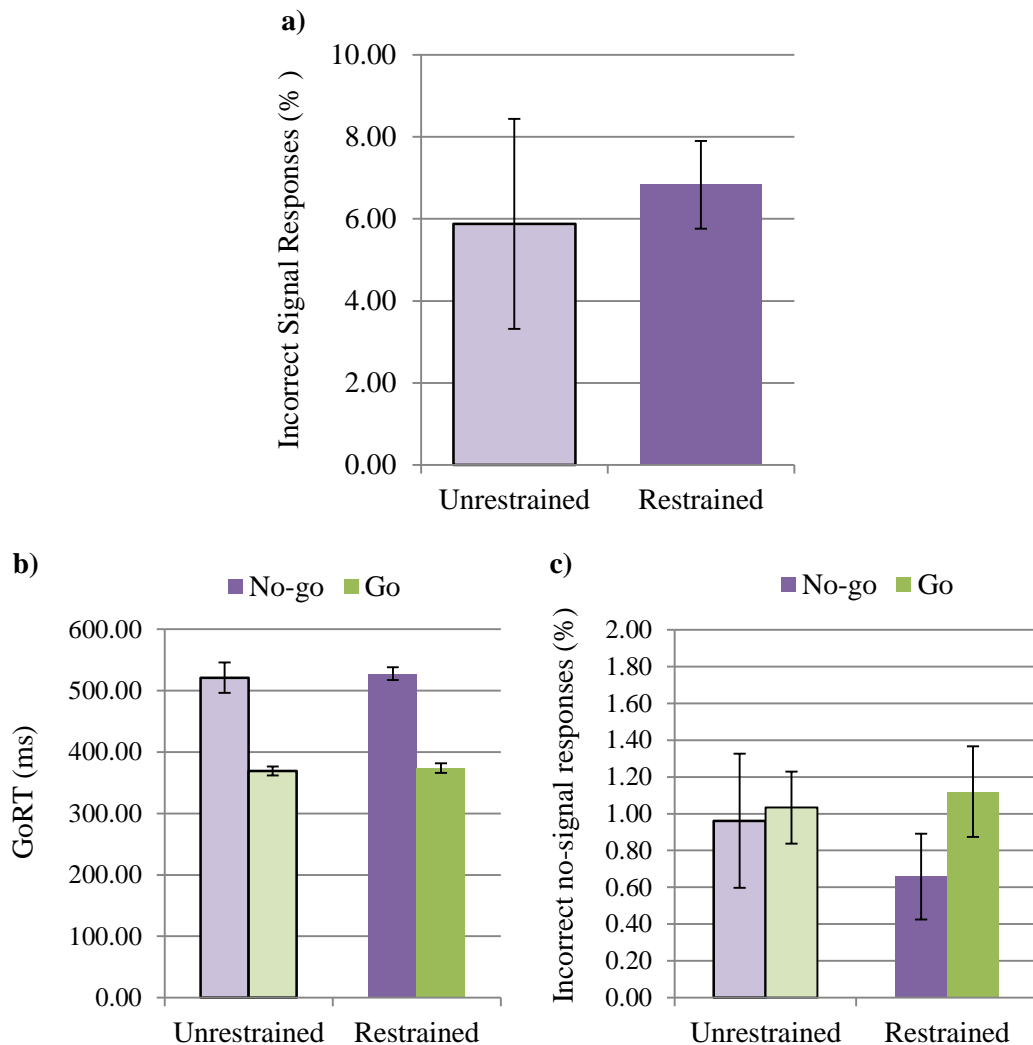


Figure 5.3. Training data according to restraint status showing: **a)** mean percentage of incorrect responses on a signal trial (commission errors) for the no-go training condition; **b)** mean reaction time for no-signal trials (GoRT); **c)** mean percentage of incorrect responses on no-signal trials. Error bars show $\pm 1SE$.

5.3.1.3. Unipolar, SC-IAT Data Analysis

Before correcting for multiple comparisons, one-sample t-tests revealed that all scores for the positive/pleasant SC-IAT were significantly above zero (all $t_s > 2.28$, all $p_s < 0.03$, all $d_z s > 0.35$; see Figure 5.4), indicating that participants showed a positive bias for snack foods. For the negative/unpleasant SC-IAT only the score for restrained eaters in the no-go group showed a statistical trend towards significance ($t(37) = 1.81$, $p = 0.08$, $d_z = 0.29$; for all other tests: all $t_s < 0.95$, all $p_s > 0.35$, all $d_z s < 0.17$); this value was positive indicating that these participants also showed a

negative attitude towards snack foods. However, after correcting for multiple comparisons ($\alpha/8=0.006$), the only remaining statistically significant test was on the positive SC-IAT for restrained eaters in the no-go group ($t(38)=4.92, p<0.001, dz=0.79$). Results from the ANOVA revealed a statistically significant main effect of SC-IAT ($F(1,119)=8.34, p=0.005, \eta_p^2=0.07, f=0.27$) with stronger bias scores for the positive ($M=0.19, SE=0.03$), compared to the negative SC-IAT ($M=0.05, SE=0.03$). These results are similar to those of Study 2 (see Chapter 3) and suggest that participants hold stronger associations between snack foods (chocolate only in Study 2) and pleasant words than snack foods and unpleasant words. There was also a trend towards significance for the main effect of training condition ($F(1,119)=3.04, p=0.08, \eta_p^2=0.03, f=0.18$) indicating stronger bias scores in the no-go group ($M=0.16, SE=0.04$) compared to the go group ($M=0.08, SE=0.03$). Importantly, there was no statistically significant interaction between training condition and SC-IAT ($F(1,119)=0.92, p=0.34, \eta_p^2=0.008, f=0.09$) and no significant three-way interaction with restraint status ($F(1,119)=0.03, p=0.86, \eta_p^2<0.001, f=0.03$). The main effect of restraint status and other two-way interactions involving restraint status were all non-significant (all $F_s<1.78$, all $p_s>0.19$, all $\eta_p^2_s<0.02$, all $f_s<0.14$).

Similar results were also found when examining the effect of training on implicit attitudes towards snack food images that were either presented during training (old) or novel (new). There was a significant main effect of SC-IAT ($F(1,119)=7.21, p=0.008, \eta_p^2=0.06, f=0.25$), showing a stronger bias on the positive ($M=0.27, SE=0.04$) compared to the negative SC-IAT ($M=0.11, SE=0.05$), and a significant main effect of training condition ($F(1,119)=4.64, p=0.03, \eta_p^2=0.04, f=0.2$), with participants in the no-go groups ($M=0.26, SE=0.05$) showing a stronger bias overall than participants in the go groups ($M=0.12, SE=0.04$). However, all other main effects and interactions were non-significant including the interactions between condition and SC-IAT ($F(1,119)=0.95, p=0.33, \eta_p^2=0.008, f=0.09$), condition, restraint status and SC-IAT ($F(1,119)=1.08, p=0.3, \eta_p^2=0.009, f=0.1$) and between condition, restraint status, SC-IAT and stimulus type ($F(1,119)=0.12, p=0.73, \eta_p^2=0.001, f=0.03$; all other $F_s<2.65, p_s>0.11, \eta_p^2_s<0.02, f_s<0.14$). After correcting for multiple comparisons ($\alpha/16=0.003$), one-sample t-tests revealed that the only significant bias scores were for restrained no-go participants for both positive-old

and positive-new scores and for unrestrained no-go participants for positive-new scores.

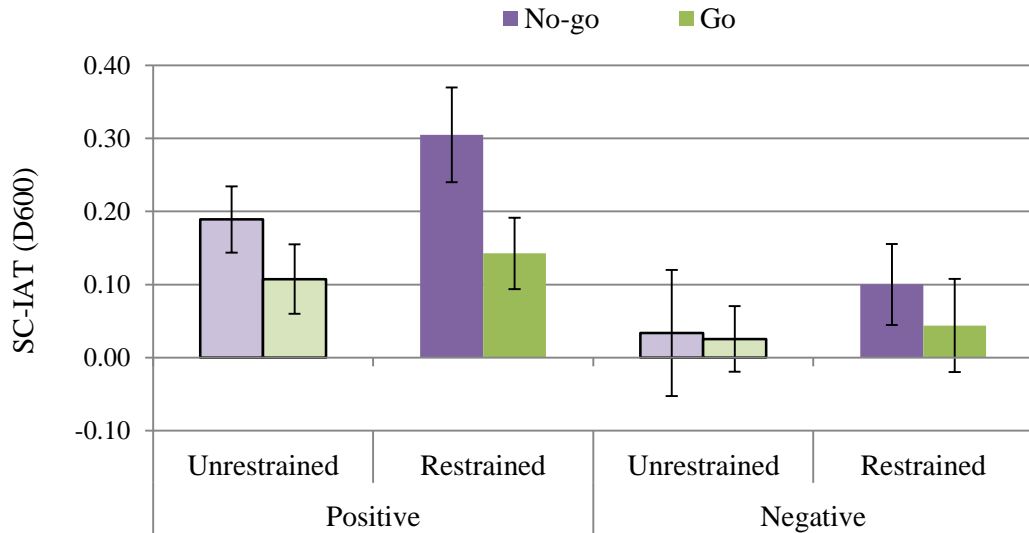


Figure 5.4. Mean bias scores for the positive and negative SC-IATs (D600 scoring algorithm) according to training condition and restraint status. Positive values indicate a faster association for congruent trials (snack foods and pleasant words on the positive SC-IAT and snack foods and unpleasant words on the negative SC-IAT) whereas negative values indicate a faster association in the incongruent trials (snack foods and neutral words). Error bars show $\pm 1SE$.

5.3.2. Study 4b: Explicit Evaluation Results

5.3.2.1. Group Differences

Table 5.2 presents the descriptive values for RS score, age, BMI and frequency of snack consumption. There was a significant effect of restraint across the four groups ($\chi^2(3)=95.09, p<0.001, \eta^2=0.75$). Follow-up Mann-Whitney U tests showed that there was no statistically significant difference between restrained eaters ($U=526.5, p=0.35, r=0.11$) and no statistically significant difference between unrestrained eaters in the no-go and go groups ($U=385.5, p=0.59, r=0.07$). All tests between restrained and unrestrained eaters were statistically significant after correcting for multiple comparisons ($\alpha/6=0.008$; all $U_s<0.001$, all $p_s<0.001$, all $r_s>0.85$). Gender was fairly evenly distributed; the percentage of females in restrained groups was

greater than that for unrestrained groups (again, this is consistent with previous findings showing that females are more restrained than males; see Study 1; e.g. Wardle, 1986), however, within each restraint group gender was evenly distributed between training conditions (due to the small number of males in each group a Chi square analysis was not conducted). Groups were also well matched for age and BMI (after correcting for multiple comparisons ($\alpha/3=0.017$) both were non-significant: age: $\chi^2(3)=0.29, p=0.96, \eta^2=0.002$; BMI: $\chi^2(3)=9.53, p=0.023, \eta^2=0.08$). There was a significant difference between groups for snack consumption ($\chi^2(3)=13.65, p=0.003, \eta^2=0.11$) reflecting a greater frequency of consumption in the unrestrained eaters in the no-go group (after correcting for multiple comparisons ($\alpha/6=0.008$) this group significantly differed from both the restrained no-go group ($U=354, p=0.005, r=0.34$) and the restrained go group ($U=224, p<0.001, r=0.45$); all other comparisons were non-significant.

Table 5.2. Group characteristics for mean RS score, gender distribution, and mean age, BMI and frequency of snack consumption (SE within parentheses).

	No-go		Go	
	Unrestrained (<i>n</i> =30)	Restrained (<i>n</i> =39)	Unrestrained (<i>n</i> =28)	Restrained (<i>n</i> =31)
RS	10.13 (0.56)	18.51 (0.49)	9.64 (0.62)	19 (0.47)
Gender (% female)	66.7	89.7	78.6	93.5
Age	24.47 (2.23)	21.41 (1.01)	21.71 (1.78)	21.48 (0.88) ²
BMI	22.65 (0.68) ¹	22.42 (0.52) ¹	21.67 (0.62) ¹	24.45 (0.77) ²
Snack consumption freq.	5.31 (0.18)	4.47 (0.2)	4.75 (0.23)	4.28 (0.2)

Note. Superscript denotes the number of participants missing for that variable. RS= Restraint Scale; BMI= body mass index

5.3.2.2. Training Data Analysis

Training data is presented in Figure 5.5. The overall commission error rate was low for both restrained ($M=5.02, SE=0.76$) and unrestrained eaters ($M=6.85, SE=0.87; U=454, p=0.11, r=0.19$). For the GoRT there was again evidence of proactive

slowing in the no-go groups ($M=531.24$, $SE=8.34$) compared to the go groups ($M=381.39$, $SE=8.95$; $F(1,124)=150.04$, $p<0.001$, $\eta_p^2=0.55$, $f=1.11$; Verbruggen & Logan, 2009b). There was also a trend towards significance for the main effect of restraint with restrained eaters ($M=445.0$, $SE=8.26$) responding faster on no-signal trials than unrestrained eaters ($M=467.62$, $SE=9.02$; $F(1,124)=3.42$, $p=0.07$, $\eta_p^2=0.03$, $f=0.18$). This finding may reflect a stronger attentional or approach bias towards food in restrained eaters (Francis *et al.*, 1997; Hollitt *et al.*, 2010; Papies *et al.*, 2008; Stewart & Samoluk, 1997; Veenstra & de Jong, 2010). The interaction between condition and restraint status was not statistically significant ($F(1,124)=0.42$, $p=0.52$, $\eta_p^2=0.003$, $f=0.05$). For the percentage of errors on no-signal trials there was a main effect of condition with more errors in the go group ($M=1.07$, $SE=0.21$) compared to the no-go group ($M=0.49$, $SE=0.2$; $F(1,124)=4.02$, $p=0.047$, $\eta_p^2=0.03$, $f=0.18$). This effect could reflect a speed-accuracy tradeoff in the go group although it appears to be driven by one participant whose error rate fell just below the 15% exclusion criterion (14.58%). Both the main effect of restraint status ($F(1,124)=1.3$, $p=0.26$, $\eta_p^2=0.01$, $f=0.1$) and the interaction ($F(1,124)=0.49$, $p=0.49$, $\eta_p^2=0.004$, $f=0.06$) were non-significant.

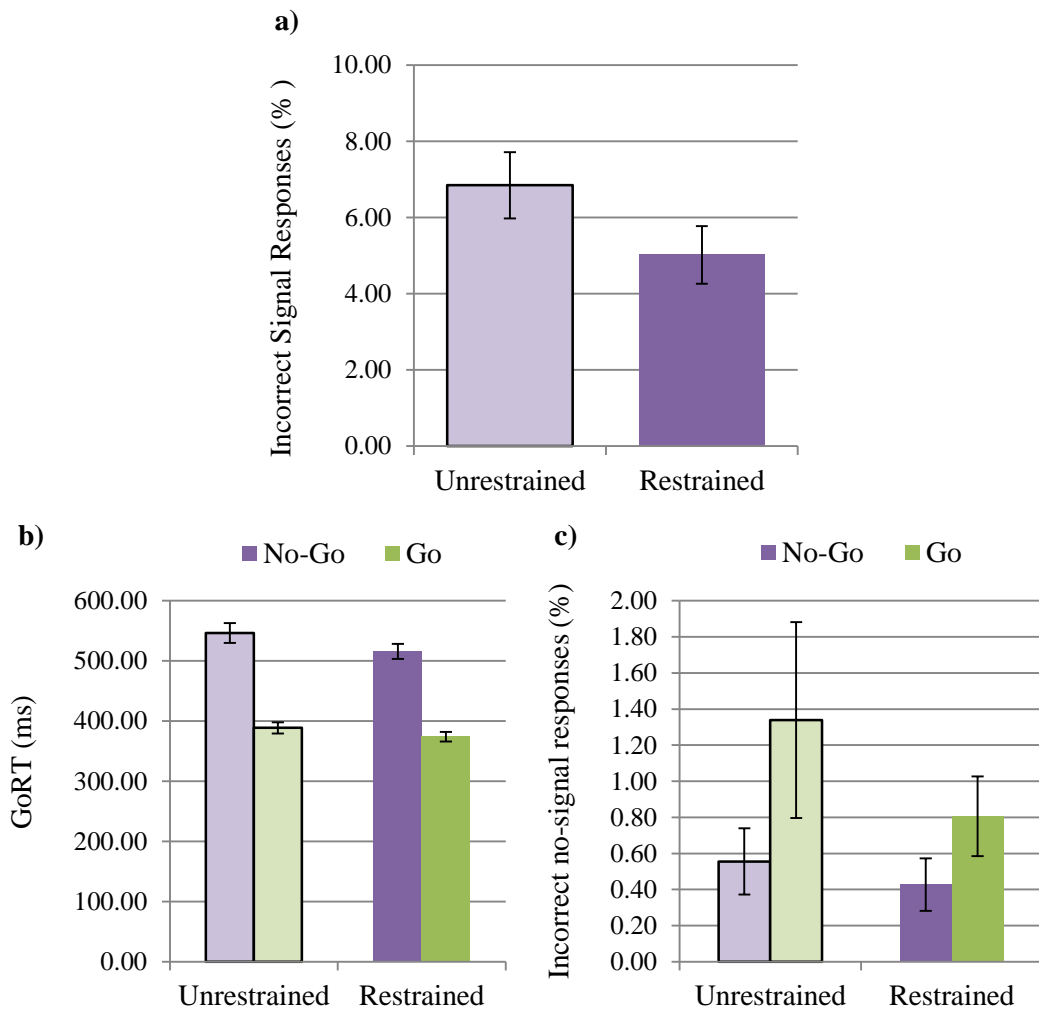


Figure 5.5. Training data according to restraint status showing: **a)** mean percentage of incorrect responses on a signal trial (commission errors) for the no-go training condition; **b)** mean reaction time for no-signal trials (GoRT); **c)** mean percentage of incorrect responses on no-signal trials. Error bars show $\pm 1SE$.

5.3.2.3. Explicit Stimulus Evaluation Task Analysis

For ratings of attractiveness (Figure 5.6a.) there was a significant main effect of food ($F(1.76, 217.91)=50.63, p<0.001, \eta_p^2=0.29, f=0.64$; with Huynh-Feldt correction for non-sphericity (Mauchley's test: $\chi^2(2)=24.42, p<0.001$); both unhealthy-old ($M=5.52, SE=0.12$) and unhealthy-new ($M=6.06, SE=0.11$) foods were rated as significantly more attractive than healthy foods ($M=4.91, SE=0.09$) and unhealthy-new foods were rated as more attractive than unhealthy-old foods (all $ps<0.001$). There was also a trend towards a main effect of training condition ($F(1,124)=2.82, p=0.095, \eta_p^2=0.02, f=0.14$), with overall lower ratings in the no-go group ($M=5.36$,

$SE=0.11$) compared to the go group ($M=5.64$, $SE=0.12$), and a statistical trend for the three-way interaction between training condition, food and restraint status ($F(1.76, 217.91)=2.36$, $p=0.1$, $\eta_p^2=0.02$, $f=0.14$). Univariate tests showed statistical trends for lower attractiveness ratings in no-go restrained, compared to go restrained, participants for both unhealthy-old (no-go restrained: $M=5.17$, $SE=0.22$; go restrained: $M=5.81$, $SE=0.25$; $F(1, 124)=3.76$, $p=0.06$, $\eta_p^2=0.03$, $f=0.18$) and healthy foods (no-go restrained: $M=4.82$, $SE=0.16$; go restrained: $M=5.25$, $SE=0.18$; $F(1,124)=3.5$, $p=0.06$, $\eta_p^2=0.03$, $f=0.18$). All other univariate tests were non-significant (all $F_s < 1.21$, all $p_s > 0.27$, all $\eta_p^2_s < 0.01$). The main effect of restraint ($F(1,124)=0.05$, $p=0.82$, $\eta_p^2 < 0.001$, $f=0.03$) and the two-way interactions between restraint and both condition ($F(1,124)=0.64$, $p=0.42$, $\eta_p^2=0.005$, $f=0.07$) and food ($F(1.76, 217.91)=1.18$, $p=0.31$, $\eta_p^2=0.009$, $f=0.1$), along with the interaction between condition and food ($F(1.76, 217.91)=0.19$, $p=0.8$, $\eta_p^2=0.001$, $f=0.03$), were all non-significant.

For ratings of tastiness (Figure 5.6b.) there was a significant main effect of food type ($F(1.6,198.22)=123.16$, $p < 0.001$, $\eta_p^2=0.5$, $f=0.23$; with Huynh-Feldt correction for non-sphericity (Mauchley's test: $\chi^2(2)=42.92$, $p < 0.001$); unhealthy-old ($M=6.38$, $SE=0.11$) and unhealthy-new ($M=6.64$, $SE=0.1$) foods were rated as more tasty than healthy foods ($M=5.00$, $SE=0.09$) and unhealthy-new foods were also rated as more tasty than unhealthy-old foods (all $p_s < 0.001$). The main effects of condition ($F(1,124)=2.14$, $p=0.15$, $\eta_p^2=0.02$, $f=0.14$) and restraint ($F(1,124)=0.53$, $p=0.47$, $\eta_p^2=0.004$, $f=0.06$) and all interactions (all $F_s < 0.91$, all $p_s > 0.39$, all $\eta_p^2_s < 0.007$) were non-significant.

For ratings of desire to eat (Figure 5.6c.) there was a significant main effect of food type ($F(1.43,177.68)=33.68$, $p < 0.001$, $\eta_p^2=0.21$, $f=0.15$; with Greenhouse-Geisser correction for non-sphericity (Mauchley's test: $\chi^2(2)=61.96$, $p < 0.001$); participants rated their desire for unhealthy-old ($M=5.19$, $SE=0.16$) and unhealthy-new ($M=5.33$, $SE=0.16$) foods as significantly greater than their desire for healthy foods ($M=4.17$, $SE=0.12$; both $p_s < 0.001$). The difference between unhealthy-old and unhealthy-new foods was not statistically significant ($p=0.12$). Main effects of condition ($F(1,124)=2.04$, $p=0.16$, $\eta_p^2=0.02$, $f=0.14$) and restraint status ($F(1,124)=0.01$,

$p=0.94$, $\eta_p^2<0.001$, $f=0.03$) and all interactions (all $F_s<1.91$, all $p_s>0.17$, all $\eta_p^2_s<0.02$) were non-significant.

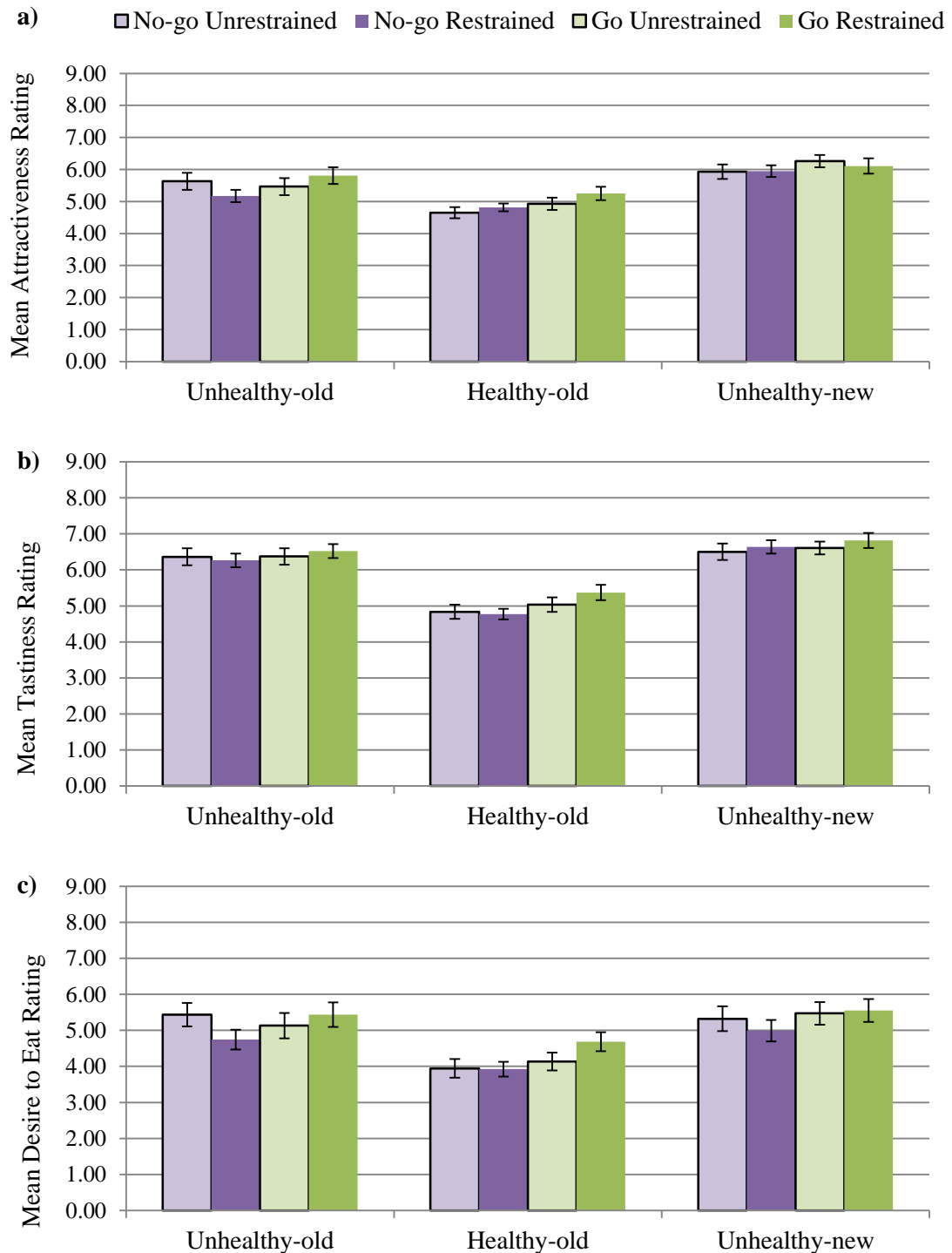


Figure 5.6. Mean ratings for **a)** attractiveness, **b)** tastiness and **c)** desire to eat the food according to training condition and restraint status. Error bars show $\pm 1SE$.

5.4. Discussion

Previous studies have shown that effects of inhibition training on behaviour may be due to the devaluation of inhibited stimuli (Houben *et al.*, 2011a, 2012a; Veling *et al.*, 2013b; Wessel *et al.* in press). However, in Study 2 (Chapter 3), I found no evidence for an effect of chocolate-related stop-signal training on either positive or negative implicit attitudes towards chocolate. This result was inconsistent with previous findings and may have been due to differences in either the training task or the dependent attitude measures. The aim of the present study, therefore, was to investigate whether food-related GNG training had any effect on either implicit or explicit attitudes towards food.

The results of Study 4a indicate that participants showed a stronger positive bias towards snack foods on the positive/pleasant SC-IAT compared to the negative/unpleasant SC-IAT. This result supports previous findings showing that both restrained and unrestrained eaters show an implicit preference for snack foods on the SC-IAT (Houben *et al.*, 2010a; see also Study 2, Chapter 3). There was also evidence to suggest that participants in the no-go group showed a stronger bias on both SC-IATs compared to the go group, however, there were no significant interactions between training condition and SC-IAT (either with or without restraint status). Results from one-sample t-tests indicated that, contrary to expectation, participants in the no-go group (particularly restrained eaters) showed stronger positive attitudes towards snack foods than participants in the go group. Although no-go participants also showed stronger negative attitudes, these bias scores did not reach statistical significance. These results are similar to those reported in Study 2 (Chapter 3) which also found no significant interaction between training condition and SC-IAT scores using a modified stop-signal training paradigm. Together these studies suggest that inhibition training does not result in either reduced positive or increased negative attitudes towards inhibited stimuli. Conversely, both studies found evidence suggesting that inhibition training may increase positive attitudes relative to the control group.

One possible explanation for this finding is that during training participants in the no-go group became ‘ego-depleted’ (Baumeister *et al.*, 1998; Hagger *et al.*, 2009; Muraven & Baumeister, 2000; Muraven *et al.*, 1998). Depleting self-control resources on the first task could have meant that participants were less able, or less motivated (Inzlicht & Schmeichel, 2012; Inzlicht *et al.*, 2014), to engage self-control on the second task, and therefore positive biases towards snack foods were more apparent. Although the IAT is believed to be less susceptible to influences of social desirability and self presentation, compared to explicit measures, there is also evidence to show that participants are able to intentionally modify their bias scores (Fiedler & Bluemke, 2005; Steffens, 2004). This explanation could also account for larger effects in restrained eaters as their strong motivations for unhealthy foods (e.g. Fedoroff *et al.*, 1997, 2003; Hollitt *et al.*, 2010; Veenstra & de Jong, 2010) would not only result in a greater positive bias towards these foods (Hoefling & Strack, 2008; Houben *et al.*, 2010a, 2012c), but could also make inhibiting responses towards them more effortful (see Lattimore & Maxwell, 2004; Nederkoorn *et al.*, 2004; Ward & Mann, 2000). These results are inconsistent with those of Houben *et al.* (2011a, 2012a) who found that training participants to inhibit their responses to images of beer on a GNG task resulted in significantly more negative implicit attitudes towards beer. To date, no effects of food-related inhibition training on implicit attitudes towards food have been reported; it is possible therefore that implicit food-related attitudes are particularly difficult to modify through training.

Study 4b explored whether GNG training resulted in changes in explicit attitudes; the results of which revealed limited evidence for an effect of inhibition training on explicit food ratings. Ratings for the attractiveness of foods revealed a trend towards a main effect of training condition in the expected direction; compared to participants in the go group, participants in the no-go group rated foods as generally less attractive. There was also a trend for a three-way interaction between condition, restraint status and food type. Follow-up analyses revealed that restrained eaters in the no-go group rated both the unhealthy-old and healthy-old foods, which were presented during training, as less attractive than restrained eaters in the go group. These results suggest that inhibition training may cause restrained eaters to perceive foods as less attractive. However, the finding that this effect transferred to the

healthy foods that were never presented alongside inhibition signals in the training task cannot be explained by theories of inhibition-induced devaluation (McLaren & Verbruggen, submitted; Veling *et al.*, 2008). Rather, this finding suggests that this effect may be due to increased ratings of attractiveness in restrained go participants who consistently responded to both unhealthy-old and healthy-old foods, but not to unhealthy-new foods. This suggestion is also consistent with the results of Study 3, presented in this thesis (Chapter 4), in which I found evidence to show that effects of GNG training on behaviour may be due to increased disinhibition in the go group rather than increased inhibitory control in the no-go group. Other studies, however, have found that evaluations of go stimuli do not increase (Fenske & Raymond, 2006; Veling *et al.*, 2008; Wessel *et al.*, in press). Nevertheless, ratings for the perceived tastiness of foods and the desire to eat those foods revealed no significant main effects of training condition or any significant interactions. These results are therefore inconsistent with those of Veling *et al.* (2013b) and suggest that inhibition training need not result in the devaluation of trained stimuli. However, Veling *et al.* only found a significant effect of inhibition training on explicit attitudes for individuals who participated in their study before lunch and were believed to be more sensitive to the incentive value of food. Consistent with previous studies in this thesis, the current study focused on dietary restraint as a potential moderator as restrained eaters have been shown to demonstrate a strong motivation towards food (e.g. Fedoroff *et al.*, 1997, 2003; Hollitt *et al.*, 2010; Veenstra & de Jong, 2010) and have previously been responsive to the effects of inhibition training on food consumption (Houben & Jansen, 2011; Lawrence *et al.*, under review; Veling *et al.*, 2011). It is possible, however, that restrained eaters may also need to be in a high appetitive state for inhibition training to effectively influence either food attitudes or consumption – studies that have investigated the effects of inhibition training on food intake in restrained eaters have asked participants not to eat for several hours prior to the study (Houben & Jansen, 2011; Lawrence *et al.*, under review). This was a limitation of the present study as participants could have been asked to report the number of hours before they last consumed a meal during the questionnaires. This potential moderator could have been explored to see whether previous results were replicable (Veling *et al.*, 2013b).

Nevertheless, the current study found little evidence to support the hypothesis that food-related GNG training influences either explicit or implicit attitudes towards food. Moreover, the evidence that did suggest an effect of inhibition training on implicit attitudes was in the opposite direction to that expected; it is possible that food-related no-go training increases positive implicit attitudes towards food – which may reflect an effect of ego-depletion during training (Baumeister *et al.*, 1998; Hagger *et al.*, 2009; Muraven & Baumeister, 2000; Muraven *et al.*, 1998). In addition, a difference in the attractiveness rating of foods following training may reflect increased scores in the go group rather than decreased scores in the no-go group. This latter finding is consistent with the results of Study 3 (Chapter 4) showing an effect of go training on increased food consumption; however, it seems unlikely that a small increase in the perceived attractiveness of unhealthy go foods could explain the large (67%) increase in consumption for these foods in Study 3. The training parameters in these two studies were very similar with the exception that the number of training blocks was halved in the current study. This modification was made to encourage participation and reduce dropout rates, however, it is possible that extended periods of training may result in larger effects (but see Veling *et al.*, 2013b). It is also possible that the lack of experimental control in the present study may partially explain the null findings. Although participants were asked to complete the study with minimal distractions there was no way to control for this and various extraneous variables may have added noise to the data. As discussed in the introduction, the lack of experimental rigour in web-based studies is one of their advantages; for many studies it is important to see whether effects are replicable in the ‘real world’ when participants can interact with the experimental manipulations in a more naturalistic way. This is especially true for the current research as potential interventions must show effects outside of the lab to be considered effective. Recent studies have shown that several sessions of food-related inhibition training in the home can increase weight loss – suggesting that this may be an effective intervention tool (Lawrence *et al.*, in preparation; Veling *et al.*, 2014). However, the results of the current study suggest that changes in the evaluative properties of these foods are unlikely to underlie these effects. To fully understand how inhibition training may influence behaviour future studies should explore other potential mechanisms such as changes in attentional and approach biases or changes in inhibitory control (e.g.

Bowley *et al.*, 2013; Kakoschke *et al.*, 2014). These studies have already been programmed as online studies in our own lab and will be investigated in the future.

Chapter 6. General Discussion

The primary aim of this thesis was to investigate whether training response inhibition can be used as an effective intervention for reducing food consumption. Specifically, restrained eaters received either food-related inhibition or control training and their subsequent food intake was measured in the lab using a bogus taste test or snack buffet. A secondary aim was to investigate the potential mechanisms that may underlie these effects; post-training measures of food-related implicit and explicit attitudes were recorded to see whether stimulus devaluation occurred as a result of inhibition training. Furthermore, I explored the role of stimulus-specific associations, sample characteristics and different training paradigms. This chapter presents a summary of findings from these studies and discusses how they relate to previous research and advance our understanding in this field. Methodological limitations and future directions are also presented.

6.1. Summary and Discussion of Findings

Obesity rates have soared over the last few decades creating a global epidemic with gross implications for personal and economic health (e.g. Bray, 2004; Fry & Finley, 2005; Mokdad *et al.*, 2003). Furthermore, obesity rates continue to increase, especially amongst children and adolescents, despite government interventions (Branca, Nikogosian, & Lobstein, 2007). Traditional weight loss methods that focus on healthy eating and increased physical exercise load heavily on self-regulatory resources and are often associated with high dropout rates and eventual weight gain (Bacon & Aphramor, 2011; Lowe *et al.*, 2006; Mann *et al.*, 2007; Pietiläinen *et al.*, 2012). Considering the large body of evidence that indicates a link between poor self-control and overeating/ obesity (Allan *et al.*, 2010; Batterink *et al.*, 2010; Cohen *et al.*, 2011; Guerrieri *et al.*, 2008a; Nederkoorn *et al.*, 2006c), these results do not seem surprising. Moreover, these methods appear to treat the outcome of overeating rather than the underlying cause. One approach which may be more successful, is to target self-control directly. Response inhibition training is one such approach that

has already been trialled with some success. Recent research has shown that training individuals to inhibit their responses to images of palatable foods may reduce both the selection and consumption of these foods (Houben, 2011; Houben & Jansen, 2011; Lawrence *et al.*, under review; van Koningsbruggen *et al.*, 2013a; Veling *et al.*, 2011, 2013a, 2013b). This training technique appears to be particularly effective for those who score high on measures of restrained eating, with some studies only finding significant effects for this population (Houben & Jansen, 2011; Lawrence *et al.*, under review, Study 2; Veling *et al.*, 2011; but see Lawrence *et al.*, under review, Study 1). The research in this thesis therefore focussed on whether inhibition training could reduce food consumption amongst those with high dietary restraint.

Paradoxically, restrained eating, according to the Restraint Scale (RS; Herman & Polivy, 1980), has been associated with an increased motivation towards food and dietary disinhibition (Fedoroff *et al.*, 1997, 2003; Houben *et al.*, 2010a, 2012c). Consistent with this idea, Houben and Jansen (2011) found that chocolate consumption on a bogus taste test increased as a function of restraint following a control task; conversely, this relationship was reversed when participants performed an inhibition training task showing that food-related no-go training may help restrained eaters to control their food intake. A similar result was reported by Veling *et al.* (2011) who found a significant effect of food-related no-go training on ad-libitum food consumption in the home environment only for individuals who scored high on the concern for dieting subscale of the RS (RSCD). It was thought that restrained eaters benefited the most from inhibition training due to their strong impulses towards food (Veenstra & de Jong, 2010) and poor control over these impulses (Nederkoorn *et al.*, 2004). However, Lawrence *et al.* (under review; Study 2) also found that scores on the restrained eating scale of the DEBQ (DEBQRE; Van Strien *et al.*, 1986a) moderated the effect of food-related stop-signal training on food consumption. As discussed in Chapters 1 and 2 (see sections 1.4.3., 2.1.), although these different restraint measures are highly related with one another (Allison *et al.*, 1992; Laessle *et al.*, 1989; Wardle, 1986), unlike the RS, the DEBQRE has been shown to correlate negatively with calorie intake and is thought to reflect successful dietary restriction (Brogan & Hevey, 2013; Laessle *et al.*, 1989; Wardle & Beales, 1987).

It was due to the above findings, demonstrating a significant effect of inhibition training for restrained eaters only, and evidence suggesting that the RS is more strongly associated with dietary disinhibition than other restraint measures, that I prescreened all participants for the lab studies in this thesis using the RS. In Study 1 I found evidence to validate this approach. In this study I explored the similarities and differences between three measures of restrained eating – the RS, RSCD and DEBQRE – with regards to measures of disinhibited eating. The results supported previous findings demonstrating that although these measures were positively correlated with one another, there were also significant differences between them. Importantly, I found that the RS was most strongly associated with increased food craving and external eating compared to the DEBQRE. Furthermore, in a sample of highly restrained eaters (15+ on the RS), the full RS, but not the RSCD, was positively correlated with BMI which may indicate that the full scale is more sensitive to those who struggle the most with their weight. These results support the use of the RS as a screening tool, and have implications for both the interpretation of previous findings and for the planning of future research in this field.

In Study 2 I recruited individuals who were classified as restrained eaters according to the RS (15+). The aim of study 2 was to investigate whether stop-signal training could be used to reduce food consumption and to see whether an effect was mediated by changes in implicit attitudes. High chocolate cravers were preselected as previous research has argued that inhibition training may be most effective for those with strong desires for particular foods (Houben & Jansen, 2011). Restrained chocolate cravers were therefore believed to be a population who were most likely to benefit from inhibition training. Participants were trained to either inhibit their responses (stop group) or make an additional response (double-response group) towards images of chocolate and crisps on a majority and minority of trials, respectively, using a modified SST. Positive and negative implicit attitudes towards chocolate and chocolate consumption were then measured using two unipolar, single-category implicit association tests (SC-IATs; Greenwald *et al.*, 1998; Karpinsky & Steinman, 2006) and a bogus taste test, respectively. Contrary to my hypothesis I found no significant difference in attitudes or intake between the two training groups.

Furthermore, a Bayesian inferential analysis found substantial evidence for the null hypothesis as participants in the stop group consumed more calories than those in the double-response control group. These results were inconsistent with previous studies. In particular, they failed to replicate the findings of Houben and Jansen (2011) who showed an effect of chocolate-related inhibition training on reduced chocolate consumption – also in restrained chocolate cravers. They also failed to replicate the findings of Lawrence *et al.*'s (under review) second study in which a very similar design and methodology were used. One major difference between Study 2 and the aforementioned studies, which may explain these inconsistencies, was the inclusion of an intermediate SC-IAT between training and consumption in Study 2.

Previous research has argued that stimulus devaluation may underlie the effects of inhibition training on behaviour (McLaren & Verbruggen, submitted; Veling *et al.*, 2008; Veling *et al.*, 2013b; Verbruggen *et al.*, in press; Wessel *et al.*, in press). The SC-IAT was therefore included in Study 2 to measure implicit attitudes towards chocolate as a potential mediator of any training-related effects on food intake. It is possible, however, that presenting participants with a task in which they had to respond quickly to images of chocolate, and pair them with pleasant and unpleasant words, may have disrupted any effect of inhibition training on food intake. Although previous studies of food-related inhibition training have not included any intermediate tasks, the procedure in Study 2 was based on two similar studies investigating the effects of alcohol-related inhibition training on attitudes and consumption (Houben *et al.*, 2011a, 2012a). Houben *et al.* (2011a) found that following beer-related inhibition training, participants demonstrated significantly more negative attitudes towards beer and showed a statistical trend for reduced beer consumption in a subsequent taste test. Participants in the inhibition group also showed a significant reduction in self-reported weekly alcohol intake – a finding that was later replicated despite three intermediate tasks measuring attitudes, approach tendencies and response inhibition (Houben *et al.*, 2012a). Houben *et al.* (2012a) also demonstrated that the effects of inhibition training on intake were mediated by changes in implicit attitudes. As Study 2 also failed to demonstrate an effect of training on either positive or negative attitudes towards chocolate, this raised the

possibility that the training procedure in Study 2 was not sufficient to produce changes in either attitudes or behaviour.

As mentioned earlier, Study 2 used a modified SST and the training protocol was very similar to that used in Lawrence *et al.* (under review, Study 2; see Appendix 2). One methodological change that may explain the difference in findings was the inclusion of feedback in Study 2. This feedback was included to ensure maximal competition between the stop and go processes, and required participants to engage top-down control throughout training (Logan *et al.*, 1997). Originally it was thought that training top-down control would be most effective as results would be more likely to generalise to other foods (Spierer *et al.*, 2013). For example, Verbruggen *et al.* (2012) trained participants on a standard version of the SST with inter-block feedback and found that compared to participants in the control group, participants in the inhibition group were more risk averse on an unrelated gambling task two hours later. However, subsequent research investigating the effectiveness of food-related inhibition training suggests that stimulus-specific associations between the target food and the act of stopping appear to play an important role (Guerrieri *et al.*, 2012; Lawrence *et al.*, under review, Study 3; and this may also be true for alcohol-related inhibition training; see Jones & Field, 2013). Moreover, the more consistent these associations are, the more likely the participant is to learn this association and engage an automatic, bottom-up, form of inhibitory control (Shiffrin & Schneider, 1977; Spierer *et al.*, 2013; Verbruggen & Logan, 2008). When these associations are less consistent, for example when participants fail to inhibit their responses on a more demanding task, the participant may learn a stimulus-*signal* rather than a stimulus-stop association (Verbruggen *et al.*, in press). Indeed, analysis of the training data revealed that participants correctly inhibited their responses more often in Lawrence *et al.*'s study (66%) compared to Study 2 (55%). This may also explain the difference in findings between Study 2 and Houben *et al.*'s (2011a, 2012a) studies for the effects of inhibition training on alcohol-related attitudes and consumption. In both of these studies Houben *et al.* trained participants using a GNG task. As discussed in Chapters 3 and 4, these GNG tasks involved consistent mapping (i.e. 100%) between the stimulus and the no-go signal. In addition, the GNG task presents the signal more frequently (typically 50% of trials, compared to

25-33% on the SST) and at the same time as the stimulus, increasing the likelihood of a participant successfully withholding their response. Although Houben *et al.* did not publish their training data, it is believed that participants are more likely to learn stimulus-stop associations and engage automatic inhibition on this task (Verbruggen & Logan, 2008). This would suggest that a GNG training paradigm could be more effective than a stop-signal paradigm for reducing food consumption and devaluing the trained stimuli. Due to the above concerns, that an intermediate task may interfere with effects of training on consumption, these questions were investigated separately in studies 3 and 4.

In Study 3 restrained eaters were allocated to either the SST or the GNG task and were then randomly divided into the inhibition and control groups. Following training, the consumption of unhealthy and healthy foods was measured in a snack buffet which also included one novel unhealthy and one novel healthy food. The results of the SST revealed a trend towards significance with participants in the stop group consuming 18% fewer calories than those in the double-response group. With the removal of inter-block feedback participants in the stop group correctly inhibited their responses on a greater number of trials (62%) compared to those in Study 2. However, a non-significant interaction term and visual inspection of the data showed that this effect was not specific to the trained unhealthy foods. Participants in the stop group appeared to consume fewer calories of the healthy foods that were infrequently presented alongside a stop signal and of the novel foods that were only presented during the consumption phase. These results are inconsistent with the theory that the effect of stop training on food consumption is reliant on stimulus-specific associations between the stimulus and stopping (Lawrence *et al.*, under review, Study 2). Rather, they suggest that the increased frequency of stopping on this task may have engaged a more general self-control mechanism (Berkman *et al.*, 2009, 2012). The results for the GNG task, on the other hand, did provide some evidence to suggest that training consistent stimulus-stop associations is more effective as an intervention tool for reducing food consumption. The difference in intake for this task was specific to the unhealthy foods with no-go participants consuming 67% fewer unhealthy calories than those in the go group. With the no-go group correctly withholding their response on 95% of signal trials it is possible that

this result was due to a learned stimulus-stop association – although, this effect also transferred to the novel unhealthy food. This latter finding suggests that stimulus-stop associations may generalise to other semantically similar foods.

At this point, these results are consistent with previous research showing an effect of food-related inhibition training on reduced food consumption in restrained eaters (Houben & Jansen, 2011; Lawrence *et al.*, under review, Study 2; Veling *et al.*, 2011). The greater difference in intake following GNG training may also explain why findings appear to be more consistent with this training task compared to the SST (Study 2; see Appendix 1) and why effects on implicit attitudes have been observed with GNG (Houben *et al.*, 2011a, 2012a) but not stop-signal (Study 2) training. However, the inclusion of an additional control group in Study 3, and comparison of all training groups provides a very different interpretation of these conclusions. Recent studies showing that pairing food stimuli with an approach or go response may increase craving for or selection of these foods (Kemps *et al.*, 2013b; Schonberg *et al.*, 2014) have cast doubt on whether effects of inhibition training on food consumption may actually be due to increased consumption in the ‘control’ groups. In these groups participants often perform a task in which they repeatedly respond to images of palatable, unhealthy foods. As discussed in the literature review (see section 1.3.2.), just as we may possess an inherent association between inhibition and aversion, we may also possess a hard-wired approach-good association (McLaren & Verbruggen, submitted; Guitart-Masip *et al.*, 2012). It is possible, therefore, that participants in these groups may consume more calories as a result of a trained approach response.

The results of Study 3 support this interpretation: participants in the go group were found to consume significantly more calories than participants in all other groups, including a group who simply observed the training task. The observe group therefore made no approach responses, nor did they inhibit any responses. Moreover, there were no other significant differences between groups. These results suggest that go training increased calorie consumption, whereas training participants to inhibit their responses (or make double-responses) towards food had no significant effect on food intake compared to a passive observe group. It is possible that

increased consumption in the go group was a result of priming general disinhibition (Guerrieri *et al.*, 2009, 2012) – the finding that this effect transferred to the novel unhealthy food supports this theory. However, there was no difference in intake for healthy foods suggesting that go training may interact with an initial desire for the food. Restrained eaters have previously been shown to demonstrate strong implicit preferences for snack foods (Houben *et al.*, 2010a, 2012c) and go training may act to enhance this preference. This may also explain why restrained eaters are more likely to show a difference in intake following GNG training compared to unrestrained eaters (Houben & Jansen, 2011; Veling *et al.*, 2011; see also Guerrieri *et al.*, 2009).

In Study 4 I investigated whether food-related GNG training had any effect on either implicit or explicit attitudes using an online study. Following training, participants in Study 4a were presented with two unipolar, SC-IATs (Greenwald *et al.*, 1998; Karpinsky & Steinman, 2006) to measure positive and negative attitudes towards snack foods. In Study 4b participants explicitly rated food stimuli according to their attractiveness, tastiness and their desire to eat the foods. Although the aim of this study was to replicate previous findings showing an effect of no-go training on stimulus devaluation (Houben *et al.*, 2011a, 2012a; Veling *et al.*, 2013b), relative to a control group, the results of Study 3 indicate that a result in this direction may reflect increased evaluations in the go group. Such a result would suggest that the effect of go training on increased food intake could be partly due to a hard-wired approach-good association and the activation of an appetitive centre (McLaren & Verbruggen, submitted; Guitart-Masip *et al.*, 2012). The results of Study 4, however, revealed very little evidence to suggest that GNG training affects the evaluative properties of trained stimuli. For the implicit measures of positive and negative attitudes, there was no significant interaction with training condition. Contrary to expectation, there was some evidence to suggest that no-go training may increase positive attitudes towards food, particularly in restrained eaters. This finding may reflect an effect of ‘ego-depletion’ whereby participants’ self-control resources were depleted during training, leading participants to become more disinhibited when they had to repeatedly pair images of snack food with pleasant words during the attitude task (Baumeister, 2003; Kahan *et al.*, 2003; Vohs & Heatherton, 2000). It is possible that this finding in Study 4 but not in Study 2 could be due to a greater ego-depletion

effect in Study 4 with the increased number of successful inhibition trials – although, the reverse could be expected if inhibition on the SST is more effortful (Christiansen *et al.*, 2012). Another possibility is that positive implicit attitudes towards chocolate were already at ceiling in Study 2 within a sample of trait chocolate cravers. For explicit ratings of attractiveness in Study 4b there was a statistical trend in the expected direction with lower ratings for trained stimuli from the restrained no-go participants compared to the restrained go participants. However, there were no significant effects of training on ratings of either the tastiness or desire to eat the foods. It seems unlikely therefore that a marginal difference in attractiveness ratings could explain the large effect of go training on increased food intake in Study 3. This finding would need to be replicated with pre- and post- training ratings and measures of food consumption to determine any mediating effects.

To summarise, the research presented in this thesis found very little evidence to suggest that inhibition training causes stimulus devaluation. Moreover, there was limited evidence to support the effectiveness of response inhibition training on reducing food consumption in restrained eaters. The main finding of this research was that food-related inhibition training had no statistically significant effect on food intake compared to a passive observe group, whereas go training was found to increase consumption compared to this group. These findings are consistent with dual process models which argue that behaviour is determined by the interaction of an impulsive system and a self-control (or reflective) system (Hofmann *et al.*, 2008; Metcalfe & Mischel, 1999; Smith & DeCoster, 2000; Shiffrin & Schneider, 1977; Strack & Deutsch, 2004). These models propose that whereas the self-control system is slow, deliberative and dependent on high cognitive capacity, the impulsive system is fast, automatic and always engaged in processing. Impulsive behavioural associations are therefore likely to be learned and retrieved more readily than associations that are reliant on the self-control system. Although increasing consumption by activating the impulsive system could have the potential to be beneficial, behaviour according to this system is largely driven by hedonic motivations – which can explain why the effect of go training on increased consumption did not transfer to the healthy foods. These results have theoretical and practical implications as they suggest that control tasks in which participants

continuously respond to images of desirable food (or alcohol) may be driving, or partly contributing to, effects of training on intake. However, this does not necessarily negate previous findings. For example, although Houben and Jansen (2011) and Veling *et al.* (2011) found a positive association between restraint and intake in the go groups, these relationships were not statistically significant. For the no-go groups, on the other hand, the relationships were negative and significant (Veling *et al.*, 2011) or marginally significant (Houben & Jansen, 2011).

These findings suggest that inhibition and control training may engage both the self-control and impulsive systems, respectively, and results will therefore depend on which system is more strongly activated according to the experimental conditions. In Study 3, the inhibition groups did consume fewer calories than the observe group, although, the effect of go training on increased intake was much greater. One possibility is that the complexity of the design in Study 3 diluted any effects of inhibition training as participants inhibited responses to a number of different unhealthy foods; both Houben and Jansen (2011) and Veling *et al.* (2011) included just one food type for training and consumption. Another possibility is that go training in Study 3 had a particularly large effect on increased impulsivity – again this may be due to the increased variety of foods presented in both the training task and/or the snack buffet. The null findings in Study 4 for the effect of go training on food-related attitudes may provide some evidence for the latter. Furthermore, Houben (2011) found that when participants with high inhibitory control abilities were presented with three different foods during a taste test, there was a statistical trend for the increased consumption of foods that were consistently associated with a go response. Similarly, Guerrieri *et al.* (2012) presented participants with four different foods for tasting and only found an effect of impulsivity training, and not inhibition training (using a standard SST), on food intake.

The possibility that inhibition training may only prove effective when a limited variety of foods are available, means that such an intervention is unlikely to be successful when we consider our ‘obesogenic’ environment (Levitsky, 2005; Raynor & Epstein, 2001). However, recent studies have shown that repeated sessions of food-related inhibition training can increase weight loss when compared to

individuals who receive inhibition training with non-food stimuli (Lawrence *et al.*, in preparation; Veling *et al.*, 2014). These studies demonstrate that inhibition training can be a useful intervention in the ‘real-world’ and remains a worthy avenue of investigation. To understand the potential efficacy of this intervention, future studies must also begin to consider the conditions in which an intervention can be declared successful. The World Medical Association recommends that new methods should be tested against the best available treatment (see Castro, 2007). Future research should therefore begin to consider comparing response inhibition training with other potential cognitive and behavioural interventions to truly consider the potential success of such methods. Before these steps are taken, however, there are some smaller limitations of this research that must first be addressed. These limitations are discussed below, and although they mainly relate to the studies in this thesis, many also apply to the wider research field.

6.2. Limitations and Directions for Further Research

6.2.1. Sample Selection and Generalisability

6.2.1.1. Psychology Undergraduates

The majority of participants for this research were recruited using advertisements at Cardiff University. Although efforts were made to recruit participants through external sources (such as council sports facilities and a local newspaper advertisement) uptake from these methods was low. This is a limitation because it has implications for the generalisability of findings to the wider population. Many psychology studies involve samples that consist entirely of psychology undergraduates as these individuals take part in studies as a course requirement. Although University students from other Schools, as well as staff, were recruited by placing adverts in other departments, halls of residence, and on a general electronic noticeboard, psychology students were also recruited for course credit for these studies. The overreliance on psychology students has previously been criticised as results are only representative of individuals who are from Western-educated, industrialised, rich and democratic societies (Henrich, Heine & Norenzayan, 2010). Moreover, compared to paid participants, the motivation of students has been

questioned, particularly for those who participate late in the semester (Nicholls, Loveless, Thomas, Loetscher & Churches, 2014). With an increased knowledge of psychological research methods, it is also more likely that psychology students are suspicious of possible deception or study hypotheses and modify their behaviour accordingly (or discordantly). This may have been particularly problematic for the present studies which used measures of implicit attitudes and covert measures of food consumption. Although only a few participants correctly guessed the aim of the research, it is plausible that others were also aware but chose not to disclose it (Nichols & Manner, 2008; see below for a more detailed discussion of demand characteristics and expectancy effects).

6.2.1.2. Self-Selection

A second issue with recruitment and generalisability concerns the way in which studies were advertised – as advertisements were for food-related research, it is quite likely that the self-selected samples were mainly individuals with a high interest in food. Although this limitation again has the potential to reduce the generalisability of these findings to a wider population, these studies required the recruitment of individuals with high dietary restraint – a trait which is associated with high levels of motivation towards food (Fedoroff *et al.*, 1997, 2003; Veenstra & de Jong, 2010). It is likely, therefore, that these adverts were appropriate for the target sample. For example, approximately 80% of respondents were female, which is consistent with findings showing that women are more preoccupied with food than men (Tapper & Pothos, 2010). Moreover, this is also advantageous in this context as women score significantly higher on measures of dietary restraint than men (see Study 1; Allison *et al.*, 1992; Burton *et al.*, 2007; Drewnowski *et al.*, 1982; Rand & Kuldau, 1991; Wardle, 1986). Indeed, previous studies investigating the effect of inhibition training on food consumption have restricted their samples to just females (Houben, 2011; Houben & Jansen, 2011); males were included in the present studies as there is no theoretical reason for assuming that inhibition training should interact with gender to predict food intake (males have also been included in other food-related inhibition training studies; Lawrence *et al.*, under review; Veling *et al.*, 2011).

6.2.1.3. Menstrual Cycle and Food Cravings

One gender difference that has been overlooked, both here and also in other studies of inhibition training, was the role of the menstrual cycle (and pregnancy) on food cravings in women (Davidsen, Vistisen & Astrup, 2007; Rogers & Smit, 2000). Food cravings have been associated with increased food intake, binge eating and a higher BMI (Burton *et al.*, 2007; Dalton *et al.*, 2013; Hill *et al.*, 1991; Lafay *et al.*, 2001; White *et al.*, 2002) and efforts should therefore be made to control for this factor. For example, some studies have only tested female participants in a particular phase of their menstrual cycle (Barth *et al.*, 2011; Goldman *et al.*, 2011; Uher *et al.*, 2005). Although this was not controlled for in the present studies, a general measure of desire to eat was recorded at the beginning of each lab study; on average participants reported a moderate degree of food desire (50-60 on a 100mm VAS) and importantly there were no statistically significant differences for this measure between the experimental and control groups for any studies. Nevertheless, this measure may not accurately reflect *specific* food cravings. For future studies, researchers may wish to explore whether specific and intense food cravings, or the menstrual phase, interact with the effect of inhibition training on food consumption, especially for particular foods. For example, effects may be greater when women are in the luteal phase and food cravings are most frequent (Davidsen *et al.*, 2007).

6.2.1.4. Restrained Eaters

Although the above issues have been raised due to potential issues with generalisability, the most limiting factor in this regard is the recruitment of restrained eaters. Restrained eaters were preselected for participation, *a priori*, as previous findings have demonstrated a significant effect of inhibition training on reduced food consumption only for these individuals (Houben & Jansen, 2011; Lawrence *et al.*, under review, Study 2; Veling *et al.*, 2011). Counterintuitively, it was believed that this was because restrained eating is strongly associated with a high motivation towards food and dietary disinhibition. It may have been more parsimonious, therefore, to recruit participants based on a more direct measure of disinhibited eating such as the disinhibition scale of the Three Factor Eating Questionnaire (TFEQ; Stunkard & Messick, 1985) rather than using restraint scores. It is possible, however, that to benefit from inhibitory control training, individuals must also

possess a desire to cut down their food intake. To fully understand the moderating roles of these traits, and to appreciate who can benefit the most from inhibition training, researchers should explore these different measures and how they influence training-related outcomes in more detail.

A further issue with the eligibility criteria for these studies, and others, is the recruitment of restrained eaters who were not actively dieting and who were able to refrain from eating for a number of hours prior to testing. Firstly, as the definition of restrained eating involves the chronic limitation of food intake in order to lose weight (Herman & Polivy, 1980), finding restrained eaters who are not dieting is fairly difficult and restrictive. In accordance with previous research, a distinction was made in these studies between dieting to lose weight and being mindful of food intake so as not to gain weight (Massey & Hill, 2012). It was hoped that this would apply to most restrained eaters due to the chronic nature of food avoidance rather than transient periods of calorie restriction. However, it is possible that this requirement led to the exclusion of participants whose restrained eating was most problematic. Secondly, this may also be true for the requirement of not eating for three hours; individuals who are able to go without eating or snacking for more than three hours may be considered to have a relatively good degree of control over their food intake. This raises questions regarding the ‘success’ of inhibition training in reducing food consumption in these individuals, especially as the average BMI of participants in the present studies was in the healthy range.

6.2.1.5. Alternative Samples

To really be considered *successful*, interventions aimed at reducing food intake should demonstrate significant, robust and replicable effects for those who suffer from weight-related health risks. Obesity is fast becoming the leading cause of preventable death (Haslam & James, 2005; Jia & Lubetkin, 2010) and is associated with an array of serious health maladies with huge economic costs (Bray, 2004; Carpenter *et al.*, 2000; Fry & Finley, 2005; Van Gaal *et al.*, 2006). It is important, therefore, to see whether inhibition training is effective for reducing food intake in these individuals. One approach would be to focus on BMI determined obesity (BMI of 30+); indeed, Veling *et al.* (2014) have shown that inhibition training may aid

weight loss most effectively for those with a high initial BMI. An important issue with BMI, however, is that it does not take body composition into account (i.e. there is no distinction between fat, bone and muscle), which can lead to misclassifications with both false positives and false negatives (Burkhauser & Cawley, 2008; Rothman, 2008; Smalley, Knerr, Kendrick, Colliver & Owen, 1990). Some researchers have argued that other measures, such as waist circumference, are more strongly associated with obesity-related health risks and recommend their use for classification (Janssen, Katzmarzyk & Ross, 2004) – and this is certainly an easy and inexpensive measure for researchers to obtain. Conversely, increased risk of hypertension and cardiovascular disease has also been reported in individuals with normal-weight obesity – a term which refers to having a normal BMI but excess body fat (Romero-Corral *et al.*, 2010; Shea, King, Gulliver & Sun, 2012). This typically involves excess fat tissue in and around the organs and measurement of this ectopic fat requires the use of specialised equipment such as full body scanners or bioelectrical impedance analysers (see Thomas, Frost, Taylor-Robinson & Bell, 2012). When considering potential target populations and the implications and generalisability of findings, researchers should be aware of these different measures and the associated limitations.

In addition to investigating the effectiveness of response inhibition training in clinically obese populations, another important population to consider is those with binge-eating disorder (BED). As discussed in the literature review (see 1.2.1.), BED is characterised by frequent episodes of binge eating in which the individual loses control over their food intake (DSM-V; APA, 2013). Unlike bulimia nervosa, individuals with BED do not engage in compensatory behaviours and therefore BED is also associated with overweight/ obesity (DSM-V; APA, 2013). Moreover, those with BED have been shown to be more impulsive and have less efficient inhibitory control compared to non-BED obese individuals (Galanti *et al.*, 2007; Nasser, Gluck, & Geliebter, 2004; Schag *et al.*, 2013; Svaldi, Naumann, Trentowska, & Schmitz, 2014). It seems reasonable, therefore, to suggest that these individuals may benefit the most from inhibition training – a possibility which is yet to be explored. Similarly, future research should also consider using the Yale Food Addiction Scale (YFAS; Gearhardt *et al.*, 2009b) as a potential moderator for the effect of inhibitory

control training on reduced food consumption. Although I have argued here (see 1.2.6.) that the term ‘food addiction’ should be used with caution, scores on the YFAS may still prove informative for identifying those who experience a more compulsive relationship with food. For example, Gearhardt *et al.* (2009b; 2012) have found that the YFAS is able to predict binge-eating more adequately than other measures of eating pathology. With valid use of this scale reported in children (Gearhardt *et al.*, 2013), early detection and intervention may also be possible as a prevention for the onset of obesity. The effect of response inhibition training in children is another worthy avenue of investigation.

6.2.2. Training Protocols

6.2.2.1. Task Parameters

In the present series of studies participants were trained to inhibit their motor responses towards images of food using either a stop-signal or GNG task. Although efforts were made to keep these tasks as consistent as possible across studies, there are still variations according to different task parameters, including the images presented, the number of blocks, number of trials, the number of images within each stimulus category, the number of signals as well as differences between the ratios of these variables (see Appendix 2). Moreover, these variables also differ to previous studies making comparisons between them difficult. Generally, however, results across studies are fairly consistent and there is some indication that increasing the number of stimulus-stop pairings has little effect on behaviour (Veling *et al.*, 2013b). Nevertheless, exploring the effects of these variables on outcomes should help training procedures to become more standardised and also more effective. Another way in which training tasks can become more standardised and controlled is with increased consistency across stimuli. This should now be more feasible with the recent release of a new database of food images for experimental research (Blechert, Meule, Busch & Ohla, 2014). These images have been rated by nearly two thousand participants according to several variables including valence, arousal, complexity and palatability and all data on macronutrient values and image properties are provided. This database should not only make it easier to control for stimuli characteristics but should also make it possible for these tasks to become

personalised. Presumably, if participants could select their own images based on the foods that they fail to resist the most, this should improve the effects of training.

6.2.2.2. Control Tasks

Another, perhaps more important, consideration is the variation between control tasks across studies. As discussed earlier (section 6.1), the significant difference in intake for GNG training in Study 3 was due to a significant increase in consumption in the go group, rather than decreased consumption in the no-go group. This result suggested that training participants to continuously respond to food stimuli may increase food-related disinhibition, especially when later presented with a variety of unhealthy foods for consumption. However, consumption in the go group was also significantly greater than consumption in the double-response group, who also consistently responded, and frequently made an additional response, to all unhealthy foods. This finding indicates that the additional processes in the double-response task (Tabu *et al.*, 2011; Verbruggen *et al.*, 2010; Verbruggen *et al.*, 2012) may have primed disinhibition to a lesser extent than the single response task (but see Lawrence *et al.*, under review, Study 2). Control tasks in other studies using GNG training have involved both a consistent go response (van Koningsbruggen *et al.*, 2013a; Veling *et al.*, 2011) and a task in which participants have to inconsistently respond and withhold responses to images of food (Houben & Jansen, 2011; Veling *et al.*, 2013a). Although tasks in which participants inconsistently respond to food images appear more conservative due to the inclusion of inhibition trials, associative uncertainty has also been shown to increase motivation and responding, which could explain increased intake in these groups (Anselme *et al.*, 2013; Collins & Pearce, 1985; Collins *et al.*, 1983; Pearce & Hall, 1980).

There is, however, evidence to indicate that the effect of inhibitory control training on behaviour is due to the inhibition of responses and not the control tasks. Firstly, Veling *et al.* (2013b) demonstrated in a mediation analysis that withholding responses to food stimuli on a GNG task resulted in the devaluation and reduced selection of these ‘no-go’ foods; the evaluation of ‘go’ foods, on the other hand, had no effect on food choice. Similarly, Wessel *et al.* (in press) found that stimuli associated with response inhibition on the SST were subsequently devalued in an

auction task, whereas a double-response task had no effect on this measure. Jones *et al.* (2011) also found that performance on an alcohol-related SST was significantly correlated with alcohol consumption for the group who were instructed to focus on successful stopping but not for the group who focussed on rapid responses. Moreover, recent studies have shown an effect of response inhibition training on weight loss, therefore demonstrating the efficacy of this training procedure as a potential clinical intervention (Lawrence *et al.*, in preparation; Veling *et al.*, 2014). Furthermore, both of these studies used a control group who inhibited their responses towards non-food stimuli which may be the most appropriate and conservative control group for this field of research. This method also reduces the likelihood that results are due to increased effort in the inhibition groups, although it is possible that withholding a response from food images is more effortful compared to non-food images (Nederkoorn *et al.*, 2012). Nevertheless, according to the ego-depletion model, exerting an increased amount of effort on one cognitive task should result in reduced self-regulation; this influence would therefore be expected to have an opposing effect to inhibition training by increasing food consumption (Baumeister, 2003; Christiansen *et al.*, 2012; Kahan *et al.*, 2003; Vohs & Heatherton; 2000).

6.2.3. Measuring Food Consumption

6.2.3.1. The Cover Story

The primary dependent variable in these studies was food consumption. This was measured by presenting participants with bowls of food along with a cover story explaining why it was necessary for them to eat something during the study. In Study 2 this cover story involved a bogus taste test. This is a standard procedure in experimental food research (e.g. Guerrieri *et al.*, 2007a, 2007b, 2008a, 2009, 2012; Houben, 2011; Houben *et al.*, 2012b; Houben & Jansen, 2011; Nederkoorn *et al.*, 2009a) and was considered appropriate for the purpose of the current studies as restrained eaters were required to eat some of the food and break their self-imposed calorie restrictions. Previous testing in our lab revealed that some participants chose not to consume any food when it was provided as ‘refreshments’ – this finding is consistent with the idea that dietary restraint is only associated with disinhibited eating when self-control is disrupted (Herman & Mack, 1975; Hofmann *et al.*, 2007;

Ruderman, 1986; Lawrence *et al.*, under review). Furthermore, Robinson *et al.* (2014) found that more than 90% of participants in their study believed the taste test cover story. This was supported with the results for food intake, which showed that participants in the taste test group consumed more calories than those in a monitored condition, who were explicitly told that their food intake was being monitored, but did not consume any fewer calories than those in an unmonitored condition, who were instructed to throw their food away following consumption (although 40% of participants in this group actually failed to do so). These results suggest that the taste test cover story is effective for disguising the covert measurement of food intake.

In Study 3 a slightly different approach was used. As participants were presented with eight bowls of unhealthy and healthy foods, and I was interested in their voluntary choices between them, participants were asked to have as much food as they liked but to ensure that they were no longer hungry after twenty minutes. During recruitment participants were informed that the purpose of the study was to measure cognitive performance according to different levels of blood glucose; this aim explained why they were asked not to eat for three hours prior to testing and why they were required to eat during the study. To make this cover story more plausible they were also asked to repeat the training task after the consumption phase and were explicitly asked which foods they consumed. Only one participant in this study reported a suspicion that food intake was being measured during the debrief and only a few participants reported noticing that the foods in the snack phase were identical to those in the training task. These cover stories are an important feature for any study wishing to covertly measure food intake. What we eat in social situations, and how much, has been shown to be largely determined by impression-management; an awareness that food intake is being monitored therefore has the potential to cause floor effects as most people tend to consume less when they believe their intake is being measured (Robinson *et al.*, 2014; Roth *et al.*, 2001; Vartanian, Herman & Polivy, 2007).

6.2.3.2. Ecological Validity

Although there was no evidence in the current studies to indicate that participants were aware that their food intake was being measured (either in the debrief phase or

a low level of food intake; average calorie consumption was ~400 kCals), the measurement of food intake in the lab environment has been criticised for a lack of ecological validity (de Castro, 2000; Meiselman, 1992; also see Kissileff, 1992; Mela, Rogers, Shepherd & MacFie, 1992). It has been argued that both the environment itself and the foods provided in this context are unnatural and do not reflect normal eating behaviour. For example, de Castro (2000) discusses the environmental, social and psychological variables that influence intake in the real-world but are often completely absent in a controlled lab environment. If measuring food intake in the lab is not reflective of typical eating behaviour, this raises concerns regarding the validity and generalisability of findings with this type of research. For example, people have been shown to compensate for increased calorie intake over a period of days (de Castro, 1998, 2000). It is also possible therefore that although participants in the inhibition groups may consume fewer calories in the lab, they may compensate, or even overcompensate, for this decrease later on (see Kemps *et al.*, 2013a). This inevitably leads to questions concerning the longevity and context-specific nature of these effects; this could be investigated by supplementing laboratory measures of food intake with more natural measures, such as self-reported diet diaries – although, these methods are not without their weaknesses (de Castro, 2000; Meiselman, 1992; Thompson & Byers, 1994). Research showing an effect of inhibitory control training on reduced BMI has helped to alleviate these concerns (Lawrence *et al.*, in preparation; Veling *et al.*, 2014). These studies have shown that repeated sessions of inhibition training in the home environment can aid weight loss over a period of weeks; however, as weight loss was measured at the end of the training programme, the longevity of these effects remains unclear (see Verbruggen, Adams, van 't Wout, Stevens, McLaren & Chambers, 2013).

6.2.4. Demand Characteristics and Placebo Effects

A major concern for studies showing an effect of inhibition training on decreased food consumption is the possibility that results are due to demand characteristics. Previous studies have shown a significant effect of training on food intake only for participants who scored highly on measures of dietary restraint (Houben & Jansen, 2011; Lawrence *et al.*, under review, Study 2; Veling *et al.*, 2011), suggesting that

either participants did not necessarily act in accordance with demand characteristics, or that somehow dietary restraint interacted with such compliance. It is possible, therefore, that participants in the inhibition groups consumed fewer calories not as a direct effect of inhibition training per se, but because they were either consciously or subconsciously aware of the study aims and what was expected of them (Klein *et al.*, 2012).

A second, related, issue is the problem of placebo effects (Boot, Simons, Stothart & Stutts, 2013). With studies in which an improvement is expected, due to an intervention of some kind, placebo effects refer to the possibility that such improvements are due to receipt of the intervention but not necessarily the ‘inherent powers of that substance or procedure’ (pg. 326; Stewart-Williams & Podd, 2004). In other words, significant effects can be mediated by either explicit expectancies of those effects or classically conditioned responses to the substance or procedure (Stewart-Williams & Podd, 2004). Boot *et al.* (2013) argue that although active control groups in psychological research are superior to passive or ‘no-contact’ groups, the problem of placebo effects cannot be discounted unless both groups are matched in terms of the same expectation of improvements. They provide an example in the video-game-training literature in which cognitive performance is examined following either a cognitively demanding game or an active control game which is less effortful. Unsurprisingly, participants believed that the demanding game would have a greater effect on performance compared to the less effortful task. It is plausible, therefore, that the effects of inhibition training on food consumption, choices and weight loss may be, in part, due to differences in expected outcomes.

Boot *et al.* (2013) recommend explicitly questioning awareness to assess expectancy effects; however, in the present studies it was believed that this approach could cause issues for the recruitment of naïve participants in the future. To covertly measure awareness participants in the lab studies were asked whether they thought that the training procedure had any effect on their questionnaire responses, food intake or their performance in the other cognitive tasks that they completed. With the exception of one participant who correctly guessed the aim of the study, no other participants reported an expected effect on reduced food intake; rather, participants

in both groups mentioned that they thought the task may have made them hungrier or made them desire specific foods. However, research has shown that these suspicion probes may be ineffective for detecting expectancy effects; Nichols and Manner (2008) found that when participants were told the experimental aims by a confederate prior to the study (the participant was informed that the researcher believed people selected objects more often when they were presented on the left-hand side than on the right-hand side when the reverse is believed to be true), not a single participant admitted to this knowledge in a post-hoc review, despite participants conforming to the study's aims. The null result in Study 2 and modest difference for the SST in Study 3 provide some indication that participants in these studies were not simply conforming to experimental expectations. The second recommendation from Boot *et al.* is to employ better active control tasks; as discussed earlier, control tasks which also require the inhibition of prepotent responses have already been used with some success (Lawrence *et al.*, in preparation; Veling *et al.*, 2014). It is possible, however, that participants who inhibit their responses to images of food with expect a greater effect on weight loss compared to those who inhibit responses to non-food images.

6.2.5. Summary

In summary, several limitations have been outlined above which apply to the current series of studies, and also, in some cases, to other published studies. These include issues with sample selections, training procedures, dependent variables and the possibility of placebo effects. Possible solutions and directions for future research were also discussed. Firstly, assuming that the aim of studies training food-related response inhibition is to develop an intervention that can improve diet- and health-related outcomes, consideration of an appropriate target sample is essential. Future studies should therefore focus on individuals with eating-related health issues, such as those who are clinically obese (although definitions of obesity can be improved by recognising measures beyond BMI) or those with compulsive eating tendencies. However, before this field of research moves into the clinical domain, researchers should further explore the effects of different experimental parameters with the aim of developing the most effective training techniques. This may involve repeated

sessions, personalisation of training stimuli or even reward-based inhibition training (Kohls *et al.*, 2009; Sinopoli *et al.*, 2011). The addition of prefrontal brain stimulation is also being investigated to see whether such techniques can be used to augment learning effects (see Alonso-Alonso, 2013; Appendix 11). Future research should also involve long-term follow-up measures with multiple dependent variables that have greater external validity with regards to health implications (e.g. % body fat or blood pressure). If, and when, these effects are found and replicated, response inhibition training may be considered a valid intervention tool for improving diet-related health.

6.3. Conclusion

The aim of this research was to explore whether training inhibitory control could reduce food consumption and to investigate the mechanisms that may underlie such an effect. The research failed to replicate previous findings showing effects of food-related inhibition training on reduced food intake and stimulus devaluation. Possible explanations and future directions have been discussed. Importantly, this research shows that careful consideration of control conditions is essential for future studies to determine the validity of any effects. Moreover, conclusions regarding the success of inhibition training as a potential clinical intervention also depend on studies with alternative samples, improved dependent measures and comparisons with other established interventions.

References

- Ahern, A. L., Field, M., Yokum, S., Bohon, C., & Stice, E. (2010). Relation of dietary restraint scores to cognitive biases and reward sensitivity. *Appetite*, *55*, 61-68. doi:10.1016/j.appet.2010.04.001
- Allan, J. L., Johnston, M., & Campbell, N. (2010). Unintentional eating. What determines goal-incongruent chocolate consumption? *Appetite*, *54*, 422-425. doi:10.1016/j.appet.2010.01.009
- Allan, J. L., Johnston, M., & Campbell, N. (2011). Missed by an inch or a mile? Predicting the size of intention-behaviour gap from measures of executive control. *Psychology & Health*, *26*, 635-650. doi:10.1080/08870441003681307
- Allison, D. B., Kalinsky, L. B., & Gorman, B. S. (1992). A comparison of the psychometric properties of three measures of dietary restraint. *Psychological Assessment*, *4*, 391-398. doi: 10.1037/1040-3590.4.3.391
- Allom, V., & Mullan, B. (2014). Individual differences in executive function predict distinct eating behaviours. *Appetite*, *80*, 123-30. doi:10.1016/j.appet.2014.05.007
- Alonso-Alonso, M. (2013). Translating tDCS into the field of obesity: mechanism-driven approaches. *Frontiers in Human Neuroscience*, *512*, doi: 10.3389/fnhum.2013.00512
- American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders, 5th edn. American Psychiatric Association: Washington, DC, 2013.
- Amiaz, R., Levy, D., Vainiger, D., Grunhaus, L., & Zangen, A. (2009). Repeated high-frequency transcranial magnetic stimulation over the dorsolateral prefrontal cortex reduces cigarette craving and consumption. *Addiction*, *104*, 653-660. doi:10.1111/j.1360-0443.2008.02448.x
- 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*, 401-408.
- Anselme, P., Robinson, M. J. F., & Berridge, K. C. (2013). Reward uncertainty enhances incentive salience attribution as sign-tracking. *Behavioural Brain Research*, *238*, 53-61.
- Antal, A., Terney, D., Poreisz, C., & Paulus, W. (2007). Towards unravelling task-related modulations of neuroplastic changes induced in the human motor cortex. *The European Journal of Neuroscience*, *26*, 2687-2691. doi:10.1111/j.1460-9568.2007.05896.x
- Appelans, B. M. (2009). Neurobehavioral inhibition of reward-driven feeding: Implications for dieting and obesity. *Obesity*, *17*, 640-647. doi:10.1038/oby.2008.638
- Appelans, B. M., Woolf, K., Pagoto, S. L., Schneider, K. L., Whited, M. C., & Liebman, R. (2011). Inhibiting food reward: delay discounting, food reward sensitivity, and palatable food intake in overweight and obese women. *Obesity*, *19*, 2175-2182. doi:10.1038/oby.2011.57
- Aron, A. R., Fletcher, P. C., Bullmore, E. T., Sahakian, B. J., & Robbins, T. W. (2003). Stop-signal inhibition disrupted by damage to right inferior frontal gyrus in humans. *Nature Neuroscience*, *6*, 115-116. doi:10.1038/nn1003
- Aron, A. R., Robbins, T. W., & Poldrack, R. A. (2004). Inhibition and the right inferior frontal cortex. *Trends in Cognitive Sciences*, *8*, 170-177.
- Aron, A. R., Robbins, T. W., & Poldrack, R. A. (2014). Inhibition and the right inferior frontal cortex: one decade on. *Trends in Cognitive Sciences*, *18*, 177-185. doi:10.1016/j.tics.2013.12.003
- 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*, 1875-1882.

- Avena, N. M., Bocarsly, M. E., Rada, P., Kim, A., & Hoebel, B. G. (2008b). After daily bingeing on a sucrose solution, food deprivation induces anxiety and accumbens dopamine/acetylcholine imbalance. *Physiology & Behavior*, *94*, 309–315. doi:10.1016/j.physbeh.2008.01.008
- Avena, N. M., Gearhardt, A. N., Gold, M. S., Wang, G.-J., & Potenza, M. N. (2012). Tossing the baby out with the bathwater after a brief rinse? The potential downside of dismissing food addiction based on limited data. *Nature Reviews. Neuroscience*, *13*, 514; doi:10.1038/nrn3212-c1
- Avena, N. M., Rada, P., & Hoebel, B. G. (2008a). Evidence for sugar addiction: Behavioral and neurochemical effects of intermittent, excessive sugar intake. *Neuroscience and Biobehavioral Reviews*, *32*, 20–39.
- Bacon, L., & Aphramor, L. (2011). Weight science: evaluating the evidence for a paradigm shift. *Nutrition Journal*, *10*, 9. doi:10.1186/1475-2891-10-9
- Badry, R., Mima, T., Aso, T., Nakatsuka, M., Abe, M., Fathi, D., ... Fukuyama, H. (2009). Suppression of human cortico-motoneuronal excitability during the Stop-signal task. *Clinical Neurophysiology*, *120*, 1717–1723. doi:10.1016/j.clinph.2009.06.027
- Baptista, T., Parada, M., & Hernandez, L. (1987). Long term administration of some antipsychotic drugs increases body weight and feeding in rats. Are D2 dopamine receptors involved? *Pharmacology, Biochemistry, and Behavior*, *27*, 399–405.
- Bardo, M. Y., Fishbein, D. H., & Milich, R. (2011). Inhibitory control and drug abuse prevention: From research to translation. New York, NY: Springer.
- Barone, J. J., & Roberts, H. R. (1996). Caffeine Consumption. *Food and Chemical Toxicology*, *34*, 119–129.
- Barr, M. S., Fitzgerald, P. B., Farzan, F., George, T. P., & Daskalakis, Z. J. (2008). Transcranial magnetic stimulation to understand the pathophysiology and treatment of substance use disorders. *Current Drug Abuse Reviews*, *1*, 328–339.
- Barry, D., Clarke, M., & Petry, N. M. (2009). Obesity and its relationship to addictions: Is overeating a form of addictive behavior? *The American Journal on Addictions*, *18*, 439–451. doi:10.3109/10550490903205579
- Barth, K. S., Rydin-Gray, S., Kose, S., Borckardt, J. J., O’Neil, P. M., Shaw, D., Modan, A., Budak, A., & George, M. S. (2011). Food cravings and the effects of left prefrontal repetitive transcranial magnetic stimulation using an improved sham condition. *Frontiers in Psychiatry*, *2*, 9. doi:10.3389/fpsy.2011.00009
- Batterink, L., Yokum, S., & Stice, E. (2010). Body mass correlates inversely with inhibitory control in response to food among adolescent girls: An fMRI study. *NeuroImage*, *52*, 1696–1703. doi:10.1016/j.neuroimage.2010.05.059
- Baumeister, R. F. (2003). Ego depletion and self-regulation failure: A resource model of self-control. *Alcoholism, Clinical and Experimental Research*, *27*, 281–4. doi:10.1097/01.ALC.0000060879.61384.A4
- Baumeister, R. F., Bratslavsky, E., Muraven, M., & Tice, D. M. (1998). Ego depletion: Is the active self a limited resource? *Journal of Personality and Social Psychology*, *74*, 1252–1265.
- Baumeister, R.F., Gailliot, M.T., DeWall, C.N., & Oaten, M. (2006). Self-regulation and personality: How interventions increase regulatory success, and how depletion moderates the effects of traits on behavior. *Journal of Personality*, *74*, 1773–1801.
- Bechara, A. (2005). Decision making, impulse control and loss of willpower to resist drugs: A neurocognitive perspective. *Nature Neuroscience*, *8*, 1458–1463. doi:10.1038/nn1584

- Bechara, A., & Martin, E. M. (2004). Impaired decision making related to working memory deficits in individuals with substance addictions. *Neuropsychology*, *18*, 152–162. doi:10.1037/0894-4105.18.1.152
- Bechara, A., Dolan, S., Denburg, N., Hindes, A., Anderson, S. W., & Nathan, P. E. (2001). Decision-making deficits, linked to a dysfunctional ventromedial prefrontal cortex, revealed in alcohol and stimulant abusers. *Neuropsychologia*, *39*, 376–89.
- Beeli, G., Casutt, G., Baumgartner, T., & Jäncke, L. (2008). Modulating presence and impulsiveness by external stimulation of the brain. *Behavioral and Brain Functions*, *4*, 33. doi:10.1186/1744-9081-4-33
- Behan, B., Stone, A., & Garavan, H. (2014). Right prefrontal and ventral striatum interactions underlying impulsive choice and impulsive responding. *Human Brain Mapping*, doi:10.1002/hbm.22621
- Bellisle, F., Clément, K., Le Barzic, M., Le Gall, A., Guy-Grand, B., & Basdevant, A. (2004). The Eating Inventory and body adiposity from leanness to massive obesity: A study of 2509 adults. *Obesity Research*, *12*, 2023-2030.
- Bello, N. T., Lucas, L. R., & Hajnal, A. (2002). Repeated sucrose access influences dopamine D2 receptor density in the striatum. *Neuroreport*, *13*, 1575–1578.
- Benton, D., Greenfield, K., & Morgan, M. (1998). The development of the attitudes to chocolate questionnaire. *Personality and Individual Differences*, *24*, 513-520.
- Bergen, A. E., Newby-Clarke, I. R., & Brown, A. (2012). Low trait self-control in problem gamblers: Evidence from self-report and behavioral measures. *Journal of Gambling Studies*, *28*, 637-648.
- Berkman, E. T., Burklund, L., & Lieberman, M. D. (2009). Inhibitory spillover: Intentional motor inhibition produces incidental limbic inhibition via right inferior frontal cortex. *NeuroImage*, *47*, 705–712. doi:10.1016/j.neuroimage.2009.04.084
- Berkman, E. T., Falk, E. B., & Lieberman, M. D. (2011). In the trenches of real-world self-control: Neural correlates of breaking the link between craving and smoking. *Psychological Science*, *22*, 498–506.
- Berkman, E. T., Graham, A. M., & Fisher, P. A. (2012). Training self-control: A domain-general translational neuroscience approach. *Child Development Perspectives*, *6*, 374–384. doi:10.1111/j.1750-8606.2012.00248.x
- Berkman, E. T., Kahn, L. E., & Merchant, J. S. (2014). Training-induced changes in inhibitory control network activity. *The Journal of Neuroscience*, *34*, 149–157. doi:10.1523/JNEUROSCI.3564-13.2014
- Berridge, K. C. (2009). “Liking” and “wanting” food rewards: Brain substrates and roles in eating disorders. *Physiology & Behavior*, *97*, 537–550. doi:10.1016/j.physbeh.2009.02.044
- Berridge, K. C., Ho, C.-Y., Richard, J. M., & DiFeliceantonio, A. G. (2010). The tempted brain eats: Pleasure and desire circuits in obesity and eating disorders. *Brain Research*, *1350*, 43–64. doi:10.1016/j.brainres.2010.04.003
- Bestman, S., de Berker, A. O., & Bonaiuto, J. (under review). Are there true behavioural improvements with non-invasive brain neurostimulation?
- Billieux, J., Gay, P., Rochat, L., Khazaal, Y., Zullino, D., & Van der Linden, M. (2010). Lack of inhibitory control predicts cigarette smoking dependence: evidence from a non-deprived sample of light to moderate smokers. *Drug and Alcohol Dependence*, *112*, 164–167. doi:10.1016/j.drugalcdep.2010.06.006
- Blanchard, F. A., & Frost, R. (1983). Two factors of restraint: Concern for dieting and weight fluctuation. *Behavior Research and Therapy*, *21*, 259–267.

- 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*, 617. doi:10.3389/fpsyg.2014.00617
- Blum, K., Braverman, E. R., Holder, J. M., Lubar, J. F., Monastera, V. J., Miller, D., Lubar, J. O., Chen, T. J. H., & Comings, D. E. (2000). Reward deficiency syndrome: A biogenetic model for the diagnosis and treatment of impulsive, addictive and compulsive behaviors. *Journal of Psychoactive Drugs, 32*(sup1), 1–112. doi:10.1080/02791072.2000.10736099
- Blum, K., Braverman, E. R., Wood, R. C., Gill, J., Li, C., Chen, T. J. H., Taub, M., Montgomery, A. R., Sheridan, P. J., & Cull, J. G. (1996). Increased prevalence of the TaqI A1 allele of the dopamine receptor gene (DRD2) in obesity with comorbid substance use disorder: A preliminary report. *Pharmacogenetics, 6*, 297–305.
- Blumenthal, D. M., & Gold, M. S. (2010). Neurobiology of food addiction. *Current Opinion in Clinical Nutrition and Metabolic Care, 13*, 359–365. doi:10.1097/MCO.0b013e32833ad4d4
- Blundell, J. E., Burley, V. J., Cotton, J. R., & Lawton, C. L. (1993). Dietary fat and the control of energy intake: evaluating the effects of fat on meal size and postmeal satiety. *American Journal of Clinical Nutrition, 57*, S772-S778.
- Boggio, P. S., Liguori, P., Sultani, N., Rezende, L., Fecteau, S., & Fregni, F. (2009). Cumulative priming effects of cortical stimulation on smoking cue-induced craving. *Neuroscience Letters, 463*, 82–86. doi:10.1016/j.neulet.2009.07.041
- Boggio, P. S., Sultani, N., Fecteau, S., Merabet, L., Mecca, T., Pascual-Leone, A., Basaglia, A., & Fregni, F. (2008). Prefrontal cortex modulation using transcranial DC stimulation reduces alcohol craving: A double-blind, sham-controlled study. *Drug and Alcohol Dependence, 92*, 55–60. doi:10.1016/j.drugalcdep.2007.06.011
- Boggio, P. S., Zaghi, S., Villani, A. B., Fecteau, S., Pascual-leone, A., & Fregni, F. (2010). Modulation of risk-taking in marijuana users by transcranial direct current stimulation (tDCS) of the dorsolateral prefrontal cortex (DLPFC). *Drug and Alcohol Dependence, 112*, 220–225. doi:10.1016/j.drugalcdep.2010.06.019
- Boon, B., Stroebe, W., Schut, H., & Ijntema, R. (2002). Ironic processes in the eating behaviour of restrained eaters. *British Journal of Health Psychology, 7*, 1–10.
- Boot, W. R., Simons, D. J., Stothart, C., & Stutts, C. (2013). The pervasive problem with placebos in psychology: Why active control groups are not sufficient to rule out placebo effects. *Perspectives on Psychological Science, 8*, 445–454. doi:10.1177/1745691613491271
- Boschi, V., Iorio, D., Margiotta, N., D’Orsi, P. & Falconi, C. (2001). The Three-Factor Eating Questionnaire in the evaluation of eating behaviour in subjects seeking participation in a dietotherapy programme. *Annals of Nutrition and Metabolism, 45*, 72-77.
- Bottlender, M., & Soyka, M. (2004). Impact of craving on alcohol relapse during, and 12 months following, outpatient treatment. *Alcohol and Alcoholism, 39*, 357–361. doi:10.1093/alcalc/agh073
- Bowirrat, A., & Oscar-Berman, M. (2005). Relationship between dopaminergic neurotransmission, alcoholism, and reward deficiency syndrome. *American Journal of Medical Genetics, 132B*, 29–37. doi:10.1002/ajmg.b.30080
- Bowley, C., Faricy, C., Hegarty, B., J Johnstone, S., L Smith, J., J Kelly, P., & A Rushby, J. (2013). The effects of inhibitory control training on alcohol consumption, implicit alcohol-related cognitions and brain electrical activity. *International Journal of Psychophysiology, 89*, 342–8. doi:10.1016/j.ijpsycho.2013.04.011

- Bradley, B. P., Field, M., Mogg, K., & De Houwer, J. (2004). Attentional and evaluative biases for smoking cues in nicotine dependence: Component processes of biases in visual orienting. *Behavioural Pharmacology*, *15*, 29–36.
- 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*, 66-72.
- Braet, C., Claus, L., Verbeken, S., & Van Vlierberghe, L. (2007). Impulsivity in overweight children. *European Child & Adolescent Psychiatry*, *16*, 473–483. doi:10.1007/s00787-007-0623-2
- Branca, F., Nikogosian, H., & Lobstein, T., editors. (2007). The challenge of obesity in the WHO European region and the strategies for response. WHO.
- Bray, G. A. (2004). Medical consequences of obesity. *The Journal of Clinical Endocrinology and Metabolism*, *89*, 2583–2589. doi:10.1210/jc.2004-0535
- Brignell, C., Griffiths, T., Bradley, B. P., & Mogg, K. (2009). Attentional and approach biases for pictorial food cues. Influence of external eating. *Appetite*, *52*, 299–306. doi:10.1016/j.appet.2008.10.007
- Brody, A. L., Mandelkern, M. A., Olmsted, R. E., Jou, J., Tinson, E., Allen, V., Scheibal, D., London, E. D., Monterosso, J. R., Tiffany, S. T., Korb, A., Gan, J. J., & Cohen, M. S. (2007). Neural substrates of resisting craving during cigarette cue exposure. *Biological Psychiatry*, *62*, 642–651.
- Brogan, A., & Hevey, D. (2013). Eating styles in the morbidly obese: restraint eating, but not emotional and external eating, predicts dietary behaviour. *Psychology & Health*, *28*, 714–25. doi:10.1080/08870446.2012.760033
- 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*, 37–51. doi:10.1016/j.cpr.2010.09.006
- Bruinsma, K., & Taren, D. (1999). Chocolate: Food or drug? *Journal of the American Dietetic Association*, *99*, 1249–1256.
- Brunner Huber, L. R. (2007). Validity of self-reported height and weight in women of reproductive age. *Maternal and Child Health Journal*, *11*, 137–44. doi:10.1007/s10995-006-0157-0
- Brunstrom, J. M., Yates, H. M., & Witcomb, G. L. (2004). Dietary restraint and heightened reactivity to food. *Physiology and Behavior*, *81*, 85–90.
- Bryant, E. J., King, N. A., & Blundell, J. E. (2008). Disinhibition: its effects on appetite and weight regulation. *Obesity reviews: An official journal of the International Association for the Study of Obesity*, *9*, 409–19.
- Burger, K. S., & Stice, E. (2011). Variability in reward responsivity and obesity: evidence from brain imaging studies. *Current Drug Abuse Reviews*, *4*, 182–189.
- Burton, P., Smit, H. J., & Lightowler, H. J. (2007). The influence of restrained and external eating patterns on overeating. *Appetite*, *49*, 191–7. doi:10.1016/j.appet.2007.01.007
- Burkhauser, R. V., & Cawley, J. (2008). Beyond BMI: The value of more accurate measures of fatness and obesity in social science research. *Journal of Health Economics*, *27*, 519–529. doi:10.1016/j.jhealeco.2007.05.005
- Butler, G. K. L., & Montgomery, M. J. (2004). Impulsivity, risk taking and recreational “ecstasy” (MDMA) use. *Drug and Alcohol Dependence*, *76*, 55–62. doi:10.1016/j.drugalcdep.2004.04.003
- Cacioppo, J.T., Priester, J.R., & Berntson, G. G. (1993). Rudimentary determinants of attitudes: II. Arm flexion and extension have differential effects on attitudes. *Journal of Personality and Social Psychology*, *65*, 5-17. doi: 10.1037/0022-3514.65.1.5

- Cai, W., Oldenkamp, C. L., & Aron, A. R. (2012). Stopping speech suppresses the task-irrelevant hand. *Brain and Language*, *120*, 412–415. doi:10.1016/j.bandl.2011.11.006
- Caine, S. B., & Koob, G. F. (1994). Effects of mesolimbic dopamine depletion on responding maintained by cocaine and food. *Journal of the Experimental Analysis of Behavior*, *2*, 213–221.
- Camprodon, J. A., Martínez-Raga, J., Alonso-Alonso, M., Shih, M.-C., & Pascual-Leone, A. (2007). One session of high frequency repetitive transcranial magnetic stimulation (rTMS) to the right prefrontal cortex transiently reduces cocaine craving. *Drug and Alcohol Dependence*, *86*, 91–94. doi:10.1016/j.drugalcdep.2006.06.002
- Carelli, R. M., James, S. G., & Crumling, A. J. (2000). Evidence that separate neural circuits in the nucleus accumbens encode cocaine versus “natural” (water and food) reward. *The Journal of Neuroscience*, *20*, 4255–4266.
- Carey, K. B., Scott-Sheldon, L. A. J., Carey, M. P., & DeMartini, K. S. (2007). Individual level interventions to reduce college student drinking: A meta-analytic review. *Addictive Behaviors*, *32*, 2469–2494.
- Carnell, S., Gibson, C., Benson, L., Ochner, C. N., & Geliebter, A. (2012). Neuroimaging and obesity: Current knowledge and future directions. *Obesity Reviews*, *13*, 43–56. doi:10.1111/j.1467-789X.2011.00927.x.
- Carpenter, K. M., Hasin, D. S., Allison, D. B., & Faith, M. S. (2000). Relationships between obesity and DSM-IV major depressive disorder, suicide ideation, and suicide attempts: results from a general population study. *American Journal of Public Health*, *90*, 251–257.
- Carroll, K. M., & Rounsaville, B. J. (1993). History and significance of childhood attention deficit disorder in treatment-seeking cocaine abusers. *Comprehensive Psychiatry*, *34*, 75–82.
- Carter, B. L., & Tiffany, S. T. (1999). Meta-analysis of cue-reactivity in addiction research. *Addiction*, *94*, 327–340.
- Casey, B. J., Trainor, R. J., Orendi, J. L., Schubert, a B., Nystrom, L. E., Giedd, J. N., ... Rapoport, J. L. (1997). A developmental functional MRI study of prefrontal activation during performance of a Go-No-Go task. *Journal of Cognitive Neuroscience*, *9*, 835–847. doi:10.1162/jocn.1997.9.6.835
- Cassin, S. E., & von Ranson, K. M. (2007). Is binge eating experienced as an addiction? *Appetite*, *49*, 687–490. doi:10.1016/j.appet.2007.06.012
- 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*, 1063–1073. doi:10.1038/ijo.2009.138
- Castro, M. (2007). Placebo versus best-available-therapy control group in clinical trials for pharmacologic therapies: Which is better? *Proceedings of the American Thoracic Society*, *4*, 570–573. doi:10.1513/pats.200706-073JK
- 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*, 151–173.
- Chalmers, D. K., Bowyer, C. A., & Olenick, N. L. (1990). Problem drinking and obesity: A comparison in personality patterns and life-style. *International Journal of the Addictions*, *25*, 803–817.
- Chambers, C. D., Bellgrove, M. A., Stokes, M. G., Henderson, T. R., Garavan, H., Robertson, I. H., ... Mattingley, J. B. (2006). Executive “brake failure” following deactivation of

- human frontal lobe. *Journal of Cognitive Neuroscience*, *18*, 444–455.
doi:10.1162/089892906775990606
- Chambers, C. D., Bellgrove, M. A., Gould, I. C., English, T., Garavan, H., McNaught, E., ... Mattingley, J. B. (2007). Dissociable mechanisms of cognitive control in prefrontal and premotor cortex. *Journal of Neurophysiology*, *98*, 3638–47. doi:10.1152/jn.00685.2007
- Chen, E. Y., & Brown, M. (2005). Obesity stigma in sexual relationships. *Obesity Research*, *13*, 1393–1397. doi:10.1038/oby.2005.168
- Chen, M., & Bargh, J. A. (1999). Consequences of automatic evaluation: Immediate behavioral predispositions to approach or avoid the stimulus. *Personality and Social Psychology Bulletin*, *25*, 215–224. doi:10.1177/0146167299025002007
- Chevrier, A.D., Noseworthy, M. D., Schachar, R. (2007). Dissociation of response inhibition and performance monitoring in the stop signal task using event-related fMRI. *Human Brain Mapping*, *28*, 1347–1358.
- Chikazoe, J., Konishi, S., Asari, T., Jimura, K., & Miyashita, Y. (2007). Activation of right inferior frontal gyrus during response inhibition across response modalities. *Journal of Cognitive Neuroscience*, *19*, 69–80. doi:10.1162/jocn.2007.19.1.69
- Chiu, Y., Cools, R., & Aron, A. R. (2014). Opposing Effects of Appetitive and Aversive Cues on Go/No-go Behavior and Motor Excitability. *Journal of Cognitive Neuroscience*. doi:10.1162/jocn_a_00585
- Chiu, Y.-C., Aron, A. R., & Verbruggen, F. (2012). Response suppression by automatic retrieval of stimulus-stop association: Evidence from transcranial magnetic stimulation. *Journal of Cognitive Neuroscience*, *24*, 1908–1918. doi:10.1162/jocn_a_00247
- Christensen, H., Low, L. F., & Anstey, K. J. (2006). Prevalence, risk factors and treatment for substance abuse in older adults. *Current Opinion in Psychiatry*, *19*, 587-592.
- Christiansen, P., Cole, J. C., & Field, M. (2012). Ego depletion increases ad-lib alcohol consumption: Investigating cognitive mediators and moderators. *Experimental and Clinical Psychopharmacology*, *20*, 118-128.
- Churchill, S., & Jessop, D. C. (2011). Reflective and non-reflective antecedents of health-related behaviour: exploring the relative contributions of impulsivity and implicit self-control to the prediction of dietary behaviour. *British Journal of Health Psychology*, *16*(Pt 2), 257–272. doi:10.1348/135910710X498688
- Cincotta, A. H., & Meier, A. H. (1996). Bromocriptine (Ergoset) reduces body weight and improves glucose tolerance in obese subjects. *Diabetes Care*, *19*, 667-670.
- Clark, S. M., & Saules, K. K. (2013). Validation of the Yale Food Addiction Scale among a weight-loss surgery population. *Eating Behaviors*, *14*, 216–219. doi:10.1016/j.eatbeh.2013.01.002
- Claudino, A. M., Van den Eynde, F., Stahl, D., Dew, T., Andiappan, M., Kalthoff, J., Schmidt, U., & Campbell, I. C. (2011). Repetitive transcranial magnetic stimulation reduces cortisol concentrations in bulimic disorders. *Psychological Medicine*, *41*, 1329–1336. doi:10.1017/S0033291710001881
- Clifton, P. G., Rusk, I. N., & Cooper, S. J. (1991). Effects of dopamine D1 and dopamine D2 antagonists on the free feeding and drinking patterns of rats. *Behavioral Neuroscience*, *105*, 272–281.
- Clure, C., Brady, K. T., Saladin, M. E., Johnson, D., Waid, R., & Rittenbury, M. (1999). Attention-deficit/hyperactivity disorder and substance use: Symptom pattern and drug choice. *American Journal of Drug and Alcohol Abuse*, *25*, 441-448.
- Cocores, J. A., & Gold, M. S. (2009). The Salted Food Addiction Hypothesis may explain overeating and the obesity epidemic. *Medical Hypotheses*, *73*, 892–899. doi:10.1016/j.mehy.2009.06.049

- Coffey, S. F., Gudleski, G. D., Saladin, M. E., & Brady, K. T. (2003). Impulsivity and rapid discounting of delayed hypothetical rewards in cocaine-dependent individuals. *Experimental and Clinical Psychopharmacology*, *11*, 18–25. doi:10.1037/1064-1297.11.1.18
- Cohen, J. I., Yates, K. F., Duong, M., & Convit, A. (2011). Obesity, orbitofrontal structure and function are associated with food choice: A cross-sectional study. *BMJ Open*, *1*, e000175. doi:10.1136/bmjopen-2011-000175
- Colantuoni, C., Rada, P., McCarthy, J., Patten, C., Avena, N. M., Chadeayne, A., & Hoebel, B. G. (2002). Evidence that intermittent, excessive sugar intake causes endogenous opioid dependence. *Obesity Research*, *10*, 478–488. doi:10.1038/oby.2002.66
- Colantuoni, C., Schwenker, J., McCarthy, J., Rada, P., Ladenheim, B., Cadet, J. L., Schwartz, G. J., & Hoebel, B. G. (2001). Excessive sugar intake alters binding to dopamine and mu-opioid receptors in the brain. *Neuroreport*, *12*, 3549–3552.
- Collins, A., & Mullan, B. (2011). An extension of the theory of planned behavior to predict immediate hedonic behaviors and distal benefit behaviors. *Food Quality and Preference*, *22*, 638–646. doi:10.1016/j.foodqual.2011.03.011
- Collins, L., & Pearce, J. M. (1985). Predictive accuracy and the effects of partial reinforcement on serial autoshaping. *Journal of Experimental Psychology: Animal Behavior Processes*, *11*, 548–564.
- Collins, L., Young, D. B., Davies, K., & Pearce, J. M. (1983). The influence of partial reinforcement on serial autoshaping with pigeons. *Quarterly Journal of Experimental Psychology*, *35B*, 275–90.
- Comings, D. E., Muhleman, D., Ahn, C., Gysin, R., & Flanagan, S. D. (1994). The dopamine D2 receptor gene: A genetic risk factor in substance abuse. *Drug and Alcohol Dependence*, *34*, 175–180.
- Connor, M., & Higgins, A. R. (2010). Long-term effects of implementation intentions on prevention of smoking uptake among adolescents: A cluster randomized controlled trial. *Health Psychology*, *29*, 529–538.
- Contento, I. R., Zybert, P., & Williams, S. S. (2005). Relationship of cognitive restraint of eating and disinhibition to the quality of food choices of Latina women and their young children. *Preventive medicine*, *40*, 326–336.
- Cornell, C. E., Rodin, J., & Weingarten, H. (1989). Stimulus-induced eating when satiated. *Physiology & Behavior*, *45*, 695–704.
- Corsica, J. A., & Pelchat, M. L. (2010). Food addiction: true or false? *Current Opinion in Gastroenterology*, *26*, 165–169. doi:10.1097/MOG.0b013e328336528d
- Corsica, J. A., & Spring, B. J. (2008). Carbohydrate craving: a double-blind, placebo-controlled test of the self-medication hypothesis. *Eating Behaviors*, *9*, 447–454. doi:10.1016/j.eatbeh.2008.07.004
- Cousijn, J., Goudriaan, A. E., & Wiers, R. W. (2011). Reaching out towards cannabis: Approach-bias in heavy cannabis users predicts changes in cannabis use. *Addiction*, *106*, 1667–1674.
- Crescioni, A. W., Ehrlinger, J., Alquist, J. L., Conlon, K. E., Baumeister, R. F., Schatschneider, C., & Dutton, G. R. (2011). High trait self-control predicts positive health behaviors and success in weight loss. *Journal of Health Psychology*, *16*, 750–759. doi:10.1177/1359105310390247
- Criaud, M., & Boulinguez, P. (2013). Have we been asking the right questions when assessing response inhibition in go/no-go tasks with fMRI? A meta-analysis and critical review. *Neuroscience and Biobehavioral Reviews*, *37*, 11–23. doi:10.1016/j.neubiorev.2012.11.003

- Crump, M. J. C., McDonnell, J. V., & Gureckis, T. M. (2013). Evaluating Amazon's Mechanical Turk as a tool for experimental behavioral research. *PloS One*, 8, e57410. doi:10.1371/journal.pone.0057410
- Cummins, S., & Macintyre, S. (2006). Food environments and obesity - neighbourhood or nation? *International Journal of Epidemiology*, 35, 100-104.
- Curtis, C., & Davis, C. (2014). A qualitative study of binge eating and obesity from an addiction perspective. *Eating Disorders*, 22, 19-32. doi:10.1080/10640266.2014.857515
- Dalton, M., Blundell, J., & Finlayson, G. S. (2013). Examination of food reward and energy intake under laboratory and free-living conditions in a trait binge eating subtype of obesity. *Frontiers in Psychology*, 4, 757. doi:10.3389/fpsyg.2013.00757
- Dambacher, F., Sack, A. T., Lobbestael, J., Arntz, A., Brugmann, S., & Schuhmann, T. (2014). The role of right prefrontal and medial cortex in response inhibition: Interfering with action restraint and action cancellation using transcranial magnetic brain stimulation. *Journal of Cognitive Neuroscience*, 26, 1775-1784. doi:10.1162/jocn
- Dansinger, M. L., Gleason, J. A., Griffith, J. L., Selker, H. P., & Schaefer, E. J. (2005). Comparison of the Atkins, Ornish, Weight Watchers, and Zone diets for weight loss and heart disease risk reduction: a Randomized trial. *JAMA : The Journal of the American Medical Association*, 293, 43-53. doi:10.1001/jama.293.1.43
- Davidson, L., Vistisen, B., & Astrup, A. (2007). Impact of the menstrual cycle on determinants of energy balance: A putative role in weight loss attempts. *International Journal of Obesity*, 31, 1777-1785. doi:10.1038/sj.ijo.0803699
- Davidson, D., Tiffany, S. T., Johnston, W., Flury, L., & Li, T.-K. (2003). Using the cue-availability paradigm to assess cue reactivity. *Alcoholism, Clinical and Experimental Research*, 27, 1251-1256. doi:10.1097/01.ALC.0000080666.89573.73
- Davis, C. A., Levitan, R. D., Reid, C., Carter, J. C., Kaplan, A. S., Patte, K. A., King, N., Curtis, C., & Kennedy, J. L. (2009). Dopamine for "wanting" and opioids for "liking": a comparison of obese adults with and without binge eating. *Obesity*, 17, 1220-1225. doi:10.1038/oby.2009.52
- Davis, C., & Carter, J. C. (2009). Compulsive overeating as an addiction disorder. A review of theory and evidence. *Appetite*, 53, 1-8. doi:10.1016/j.appet.2009.05.018
- Davis, C., Curtis, C., Levitan, R. D., Carter, J. C., Kaplan, A. S., & Kennedy, J. L. (2011). Evidence that "food addiction" is a valid phenotype of obesity. *Appetite*, 57, 711-717. doi:10.1016/j.appet.2011.08.017
- Davis, C., Levitan, R. D., Carter, J., Kaplan, A. S., Reid, C., Curtis, C., Patte, K., & Kennedy, J. L. (2008a). Personality and eating behaviors: A case-control study of binge eating disorder. *The International Journal of Eating Disorders*, 41, 243-250. doi:10.1002/eat.20499
- Davis, C., Levitan, R. D., Kaplan, A. S., Carter, J., Reid, C., Curtis, C., Patte, K., Hwang, R., & Kennedy, J. L. (2008b). Reward sensitivity and the D2 dopamine receptor gene: A case-control study of binge eating disorder. *Progress in Neuro-Psychopharmacology & Biological Psychiatry*, 32, 620-628. doi:10.1016/j.pnpbp.2007.09.024
- Davis, C., Levitan, R. D., Muglia, P., Bewell, C., & Kennedy, J. L. (2004b). Decision-making deficits and overeating: A risk model for obesity. *Obesity Research*, 12, 929-935. doi:10.1038/oby.2004.113
- Davis, C., Loxton, N. J., Levitan, R. D., Kaplan, A. S., Carter, J. C., & Kennedy, J. L. (2013). "Food addiction" and its association with a dopaminergic multilocus genetic profile. *Physiology & Behavior*, 118, 63-69. doi:10.1016/j.physbeh.2013.05.014

- Davis, C., Patte, K., Curtis, C., & Reid, C. (2010). Immediate pleasures and future consequences. A neuropsychological study of binge eating and obesity. *Appetite*, *54*, 208–213. doi:10.1016/j.appet.2009.11.002
- Davis, C., Patte, K., Levitan, R., Reid, C., Tweed, S., & Curtis, C. (2007). From motivation to behaviour: A model of reward sensitivity, overeating, and food preferences in the risk profile for obesity. *Appetite*, *48*, 12–19. doi:10.1016/j.appet.2006.05.016
- Davis, C., Strachan, S., & Berkson, M. (2004b). Sensitivity to reward: implications for overeating and overweight. *Appetite*, *42*, 131–138. doi:10.1016/j.appet.2003.07.004
- Dawe, S., & Loxton, N. J. (2004). The role of impulsivity in the development of substance use and eating disorders. *Neuroscience and Biobehavioral Reviews*, *28*, 343–351. doi:10.1016/j.neubiorev.2004.03.007
- de Boer, B. J., van Hooft, E. A. J., & Bakker, A. B. (2011). Stop and start control: A distinction within self-control. *European Journal of Personality*, *25*, 349–362.
- de Castro, J. M. (1998). Prior day's intake has macronutrient-specific delayed negative feedback effects on the spontaneous food intake of free-living humans. *Human Nutrition and Metabolism*, *128*, 61–67.
- de Castro, J. M. (2000). Eating behavior: Lessons from the real world of humans. *Nutrition*, *16*, 800–13.
- de Lauzon-Guillain, B. De, Basdevant, A., Romon, M., Karlsson, J., Borys, J., & Charles, M. A. (2006). Is restrained eating a risk factor for weight gain in a general population? *The American Journal of Clinical Nutrition*, *83*, 132–138.
- De Ridder, D. T. D., de Boer, B. J., Lugtig, P., Bakker, A. B., & van Hooft, E. A. J. (2011). Not doing bad things is not equivalent to doing the right thing: Distinguishing between inhibitory and initiatory self-control. *Personality and Individual Differences*, *50*, 1006–1011. doi:10.1016/j.paid.2011.01.015
- de Wit, H. (2008). Impulsivity as a determinant and consequence of drug use: A review of underlying processes. *Addiction Biology*, *14*, 22–31. doi:10.1111/j.1369-1600.2008.00129.x
- de Zubicaray, G. I., Andrew, C., Zelaya, F. O., Williams, S. C., & Dumanoir, C. (2000). Motor response suppression and the prepotent tendency to respond: A parametric fMRI study. *Neuropsychologia*, *38*, 1280–91.
- Dehghani Arani, F., Rostami, R., & Nadali, H. (2013). Neurofeedback training for opiate addiction: improvement of mental health and craving. *Applied Psychophysiology and Biofeedback*, *38*, 133–141. doi:10.1007/s10484-013-9218-5
- Dehghani Arani, F., Rostami, R., & Nostratabadi, M. (2010). Effectiveness of neurofeedback training as a treatment for opioid-dependent patients. *Clinical EEG and Neuroscience*, *41*, 170–177. doi:10.1177/155005941004100313
- Del Parigi, A., Chen, K., Salbe, A. D., Hill, J. O., Wing, R. R., Reiman, E. M., & Tataranni, P. A. (2007). Successful dieters have increased neural activity in cortical areas involved in the control of behavior. *International Journal of Obesity*, *31*, 440–8. doi:10.1038/sj.ijo.0803431
- Del Parigi, A., Chen, K., Salbe, A. D., Reiman, E. M., & Tataranni, P. A. (2003). Are we addicted to food? *Obesity Research*, *11*, 493–495. doi:10.1038/oby.2003.68
- Del Parigi, A., Pannacciulli, N., Le, D. N., & Tataranni, P. A. (2005). In pursuit of neural risk factors for weight gain in humans. *Neurobiology of Aging*, *26S*, S50–5. doi:10.1016/j.neurobiolaging.2005.09.008
- Denson, T. F., Capper, M. M., Oaten, M., Friese, M., & Schofield, T. P. (2011a). Self-control training decreases aggression in response to provocation in aggressive individuals. *Journal of Research in Personality*, *45*, 252–256.

- Denson, T. F., Pedersen, W. C., Friese, M., Hahm, A., & Roberts, L. (2011b). Understanding impulsive aggression: Angry rumination and reduced self-control capacity are mechanisms underlying the provocation-aggression relationship. *Personality & Social Psychology Bulletin*, *37*, 850–62.
- Derryberry, D., & Reed, M. A. (2002). Anxiety-related attentional biases and their regulation by attentional control. *Journal of Abnormal Psychology*, *111*, 225–236. doi:10.1037//0021-843X.111.2.225
- Deutsch, R., & Strack, F. (2006). Reflective and impulsive determinants of addictive behavior. In R. W. Wiers & A. W. Stacy (Eds.), *Handbook of implicit cognition and addiction* (pp. 45-57). Thousand Oaks, CA: Sage.
- DeWall, C. N., Baumeister, R. F., Stillman, T. F., & Gailliot, M. T. (2007). Violence restrained: Effects of self-regulation and its depletion on aggression. *Journal of Experimental Social Psychology*, *43*, 62–76.
- Dewiputri, W. I., & Auer, T. (2013). Functional magnetic resonance imaging (fMRI) neurofeedback: Implementations and applications. *The Malaysian Journal of Medical Sciences*, *20*, 5–15.
- Diana, M. (2011). The dopamine hypothesis of drug addiction and its potential therapeutic value. *Frontiers in Psychiatry*, *2*, 64. doi:10.3389/fpsy.2011.00064
- Dienes, Z. (2011). Bayesian versus orthodox statistics: Which side are you on? *Perspectives on Psychological Science*, *6*, 274–290. doi:10.1177/1745691611406920
- Dienes, Z. (2014). Using Bayes to get the most out of non-significant results. *Frontiers in Psychology*, *5*, 1–17. doi:10.3389/fpsyg.2014.00781
- DiLeone, R. J., Taylor, J. R., & Picciotto, M. R. (2012). The drive to eat: comparisons and distinctions between mechanisms of food reward and drug addiction. *Nature Neuroscience*, *15*, 1330–1335. doi:10.1038/nn.3202
- Dillon, D. G., & Pizzagalli, D. A. (2007). Inhibition of action, thought, and emotion: A selective neurobiological review. *Applied and Preventative Psychology*, *12*, 99–114. doi: 10.1016/j.appsy.2007.09.004
- Dinn, W. M., Ayicegi, A., Harris, C. L. (2004). Cigarette smoking in a student sample: neurocognitive and clinical correlates. *Addictive Behaviors*, *29*, 107–126.
- Dishman, R. K. (1991). Increasing and maintaining exercise and physical activity. *Behavior Therapy*, *22*, 345–378.
- Dissabandara, L. O., Loxton, N. J., Dias, S. R., Dodd, P. R., Daghlish, M., & Stadlin, A. (2014). Dependent heroin use and associated risky behaviour: the role of rash impulsiveness and reward sensitivity. *Addictive Behaviors*, *39*, 71–76. doi:10.1016/j.addbeh.2013.06.009
- Ditye, T., Jacobson, L., Walsh, V., & Lavidor, M. (2012). Modulating behavioral inhibition by tDCS combined with cognitive training. *Experimental Brain Research*, *219*, 363–8.
- Doherty, K., Kinnunen, T., Militello, F. S., & Garvey, A. J. (1995). Urges to smoke during the first month of abstinence: relationship to relapse and predictors. *Psychopharmacology*, *119*, 171–178.
- Drewnowski, A. (1989). Sensory preferences for fat and sugar in adolescence and adult life. *Annals of the New York Academy of Sciences*, *561*, 243–250.
- Drewnowski, A., Brunzell, J. D., Sande, K., Iverius, P. H., & Greenwood, M. R. C. (1985). Sweet tooth reconsidered: taste responsiveness in human obesity. *Physiology & Behavior*, *35*, 617-622.
- Drewnowski, A., Kurth, C., Holden-Wiltse, J., & Saari, J. (1992). Food preferences in human obesity – carbohydrates versus fats. *Appetite*, *18*, 207-221.

- Drewnowski, A., Riskey, D., & Desor, J. A. (1982). Feeling fat yet unconcerned: Self-reported overweight and the restraint scale. *Appetite, 3*, 273-279.
- Dykes, J., Brunner, E. J., Martikainen, P. T., & Wardle, J. (2004). Socioeconomic gradient in body size and obesity among women: The role of dietary restraint, disinhibition and hunger in the Whitehall II study. *International Journal of Obesity and Related Metabolic Disorders, 28*, 262–268.
- Dubljević, V., Saigle, V., & Racine, E. (2014). The rising tide of tDCS in the media and academic literature. *Neuron, 82*, 731–736.
<http://dx.doi.org/10.1016/j.neuron.2014.05.003>
- Eagle, D. M., Bari, A., & Robbins, T. W. (2008). The neuropsychopharmacology of action inhibition: Cross-species translation of the stop-signal and go/no-go tasks. *Psychopharmacology, 199*, 439–56. doi:10.1007/s00213-008-1127-6
- Earnshaw, V., Smith, L., & Copenhaver, M. (2013). Drug addiction stigma in the context of methadone maintenance therapy: an investigation into understudied sources of stigma. *International Journal of Mental Health and Addiction, 11*, 110–122.
 doi:doi:10.1007/s11469-012-9402-5
- Ehrman, R. N., Robbins, S. J., Bromwell, M. A., Lankford, M. E., Monterosso, J. R., & O'Brien, C. P. (2002). Comparing attentional bias to smoking cues in current smokers, former smokers, and non-smokers using a dot-probe task. *Drug and Alcohol Dependence, 67*, 185–191.
- Eichen, D. M., Lent, M. R., Goldbacher, E., & Foster, G. D. (2013). Exploration of “food addiction” in overweight and obese treatment-seeking adults. *Appetite, 67*, 22–24.
 doi:10.1016/j.appet.2013.03.008
- Eichhammer, P., Johann, M., Kharraz, A., Binder, H., Pittrow, D., Wodarz, N., & Hajak, G. (2003). High-frequency repetitive transcranial magnetic stimulation decreases cigarette smoking. *The Journal of Clinical Psychiatry, 64*, 951–953.
- Eisenberg, D. T. A., Mackillop, J., Modi, M., Beauchemin, J., Dang, D., Lisman, S. A., Lum, J. K., & Wilson, D. S. (2007). Examining impulsivity as an endophenotype using a behavioral approach: A DRD2 TaqI A and DRD4 48-bp VNTR association study. *Behavioral and Brain Functions, 3*, 2. doi:10.1186/1744-9081-3-2
- Elias, M. F., Elias, P. K., Sullivan, L. M., Wolf, P. A., & D'Agostino, R. B. (2005). Obesity, diabetes and cognitive deficit: The Framingham Heart Study. *Neurobiology of Aging, 26S*, S11–16. doi:10.1016/j.neurobiolaging.2005.08.019
- Engle, R. W. (2002). Working memory capacity as executive attention. *Current Directions in Psychological Science, 11*, 19–23.
- Erika-Florence, M., Leech, R., & Hampshire, A. (2014). A functional network perspective on response inhibition and attentional control. *Nature Communications, 5*, 4073.
 doi:10.1038/ncomms5073
- Evans, J. S. B. T., & Coventry, K. (2006). A dual process approach to behavioural addiction: The case of gambling. In R. W. Wiers & A. W. Stacy (Eds.), *Handbook of Implicit Cognition and Addiction* (pp. 29–43). Thousand Oaks, CA: Sage.
- Faber, R. J., & Vohs, K. D. (2004). To buy or not to buy?: Self-control and self-regulatory failure in purchase behavior. In R. F. Baumeister & K. D. Vohs (Eds.), *Handbook of Self-Regulation: Research, Theory and Applications* (pp. 509-524). New York, NY: Guilford Press.
- Fadardi, J. S., & Cox, W. M. (2009). Reversing the sequence: Reducing alcohol consumption by overcoming alcohol attentional bias. *Drug and Alcohol Dependence, 101*, 137–145.
 doi:10.1016/j.drugalcdep.2008.11.015

- Faul, F., Erdfelder, E., Lang, A.-G., & Buchner, A. (2007). G*Power 3: A flexible statistical power analysis program for the social, behavioral, and biomedical sciences. *Behavior Research Methods*, *39*, 175–91.
- Fedoroff, I. C., Polivy, J., & Herman, C. P. (1997). The effect of pre-exposure to food cues on the eating behavior of restrained and unrestrained eaters. *Appetite*, *28*, 33–47.
- Fedoroff, I., Polivy, J., & Herman, C. P. (2003). The specificity of restrained versus unrestrained eaters' responses to food cues: general desire to eat, or craving for the cued food?. *Appetite*, *41*, 7–13.
- Fehr, C., Yakushev, I., Hohmann, N., Buchholz, H.-G., Landvogt, C., Deckers, H., Eberhardt, A., Kläger, M., Smolka, M. N., Scheurich, A., Dielentheis, T., Schmidt, L. G., Rösch, F. R., Bartenstein, P., Gründer, G., & Schreckenberger, M. (2008). Association of low striatal dopamine D2 receptor availability with nicotine dependence similar to that seen with other drugs of abuse. *The American Journal of Psychiatry*, *165*, 507–514. doi:10.1176/appi.ajp.2007.07020352
- Feil, J., & Zangen, A. (2010). Brain stimulation in the study and treatment of addiction. *Neuroscience and Biobehavioral Reviews*, *34*, 559–574. doi:10.1016/j.neubiorev.2009.11.006
- Fenske, M. J., & Raymond, J. E. (2006). Affective influences of selective attention. *Current Directions in Psychological Science*, *15*, 312–316. doi:10.1111/j.1467-8721.2006.00459.x
- Fernández-Serrano, M. J., Pérez-García, M., & Verdejo-García, A. (2011). What are the specific vs. generalized effects of drugs of abuse on neuropsychological performance? *Neuroscience and Biobehavioral Reviews*, *35*, 377–406.
- Fernie, G., Cole, J. C., Goudie, A. J., & Field, M. (2010). Risk-taking but not response inhibition or delay discounting predict alcohol consumption in social drinkers. *Drug and Alcohol Dependence*, *112*, 54–61. doi:10.1016/j.drugalcdep.2010.05.011
- Ferrey, A. E., Frischen, A., & Fenske, M. J. (2012). Hot or not: response inhibition reduces the hedonic value and motivational incentive of sexual stimuli. *Frontiers in Psychology*, *3*, 575. doi:10.3389/fpsyg.2012.00575
- Fiedler, K., & Bluemke, M. (2005). Faking the IAT: Aided and unaided response control on the Implicit Association Tests. *Basic and Applied Social Psychology*, *27*, 307–316. doi:10.1207/s15324834basps2704_3
- Field, M., & Cox, W. M. (2008). Attentional bias in addictive behaviors: a review of its development, causes, and consequences. *Drug and Alcohol Dependence*, *97*, 1–20. doi:10.1016/j.drugalcdep.2008.03.030
- Field, M., & Eastwood, B. (2005). Experimental manipulation of attentional bias increases the motivation to drink alcohol. *Psychopharmacology*, *183*, 350–357.
- 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*, 593–608
- Field, M., Duka, T., Tyler, E., & Schoenmakers, T. (2009). Attentional bias modification in tobacco smokers. *Nicotine & Tobacco Research*, *11*, 812–22. doi:10.1093/ntr/ntp067
- Field, M., Eastwood, B., Bradley, B. P., Mogg, K. (2006). Selective processing of cannabis cues in regular cannabis users. *Drug and Alcohol Dependence*, *85*, 75–82.
- Field, M., Kiernan, A., Eastwood, B., & Child, R. (2008). Rapid approach responses to alcohol cues in heavy drinkers. *Journal of Behavior Therapy and Experimental Psychiatry*, *39*, 209–218.

- Field, M., Mogg, K., Zetteler, J., & Bradley, B. P. (2004). Attentional biases for alcohol cues in heavy and light social drinkers: The roles of initial orienting and maintained attention. *Psychopharmacology*, *176*, 88–93.
- Fillmore, M. T., & Rush, C. R. (2002). Impaired inhibitory control of behavior in chronic cocaine users. *Drug and Alcohol Dependence*, *66*, 265–273.
- Finkel, E. J., DeWall, C. N., Slotter, E. B., Oaten, M., & Foshee, V. A. (2009). Self-regulatory failure and intimate partner violence perpetration. *Journal of Personality and Social Psychology*, *97*, 483–499.
- Fishbach, A., & Shah, J. Y. (2006). Self-control in action: Implicit dispositions toward goals and away from temptations. *Journal of Personality and Social Psychology*, *90*, 820–832.
- Fitzgerald, P. B., Fountain, S., & Daskalakis, Z. J. (2006). A comprehensive review of the effects of rTMS on motor cortical excitability and inhibition. *Clinical Neurophysiology*, *117*, 2584–2596. doi:10.1016/j.clinph.2006.06.712
- Flint, A. J., Gearhardt, A. N., Corbin, W. R., Brownell, K. D., Field, A. E., & Rimm, E. B. (2014). Food-addiction scale measurement in 2 cohorts of middle-aged and older women. *American Journal of Clinical Nutrition*, *99*, 578–586. doi:10.3945/ajcn.113.068965.578
- Flint, A., Raben, A., Blundell, J. E., & Astrup, A. (2000). Reproducibility, power and validity of visual analogue scales in assessment of appetite sensations in single test meal studies. *International Journal of Obesity and Related Metabolic Disorders*, *24*, 38–48.
- Francis, J. A., Stewart, S. H., & Hounsell, S. (1997). Dietary restraint and the selective processing of forbidden and nonforbidden food words. *Cognitive Therapy and Research*, *21*, 633–646.
- Francis, L. A., & Susman, E. J. (2009). Self-regulation and rapid weight gain in children from age 3 to 12 years. *Archives of Pediatrics & Adolescent Medicine*, *163*, 297–302.
- Frank, S., Kullmann, S., & Veit, R. (2013). Food related processes in the insular cortex. *Frontiers in Human Neuroscience*, *7*, 499. doi:10.3389/fnhum.2013.00499
- Frank, S., Lee, S., Preissl, H., Schultes, B., Birbaumer, N., & Veit, R. (2012). The obese brain athlete: Self-regulation of the anterior insula in adiposity. *PloS One*, *7*, e42570. doi:10.1371/journal.pone.0042570
- Franken, I. H. A. (2003). Drug craving and addiction: integrating psychological and neuropsychopharmacological approaches. *Progress in Neuro-Psychopharmacology & Biological Psychiatry*, *27*, 563–579. doi:10.1016/S0278-5846(03)00081-2
- Franken, I. H. A., & Muris, P. (2005). Individual differences in reward sensitivity are related to food craving and relative body weight in healthy women. *Appetite*, *45*, 198–201. doi:10.1016/j.appet.2005.04.004
- Frascella, J., Potenza, M. N., Brown, L. L., & Childress, R. (2010). Carving addiction at a new joint? Shared brain vulnerabilities open the way for non-substance addictions. *Annals of the New York Academy of Sciences*, *1187*, 294–315. doi:10.1111/j.1749-6632.2009.05420.x.
- Fregni, F., Orsati, F., Pedrosa, W., Fecteau, S., Tome, F. a M., Nitsche, M. A., Mecca, T., Macedo, E. C., Pascual-Leone, A., & Boggio, P. S. (2008). Transcranial direct current stimulation of the prefrontal cortex modulates the desire for specific foods. *Appetite*, *51*, 34–41. doi:10.1016/j.appet.2007.09.016
- French, S. A., Jeffery, R. W., Sherwood, N. E., & Neumark-Sztainer, D. (1999). Prevalence and correlates of binge eating in a nonclinical sample of women enrolled in a weight gain prevention program. *International Journal of Obesity*, *23*, 576–585.

- French, S. A., Story, M., & Jeffery, R. W. (2001). Environmental influences on eating and physical activity. *Annual Review of Public Health, 22*, 309-335.
- Friese, M., & Hofmann, W. (2009). Control me or I will control you: Impulses, trait self-control, and the guidance of behavior. *Journal of Research in Personality, 43*, 795–805. doi:10.1016/j.jrp.2009.07.004
- Friese, M., Hofmann, W., & Wänke, M. (2008). When impulses take over: Moderated predictive validity of explicit and implicit attitude measures in predicting food choice and consumption behaviour. *The British Journal of Social Psychology, 47*, 397–419. doi:10.1348/014466607X241540
- Friese, M., Hofmann, W., & Wiers, R. W. (2011). On taming horses and strengthening riders: Recent developments in research on interventions to improve self-control in health behaviors. *Self and Identity, 10*, 336–351.
- Frischen, A., Ferrey, A. E., Burt, D. H. R., Pistchik, M., & Fenske, M. J. (2012). The affective consequences of cognitive inhibition: devaluation or neutralization? *Journal of Experimental Psychology. Human Perception and Performance, 38*, 169–79. doi:10.1037/a0025981
- Fry, J., & Finley, W. (2005). The prevalence and costs of obesity in the EU. *Proceedings of the Nutrition Society, 64*, 359–362. doi:10.1079/PNS2005443
- Gailliot, M. T., Plant, E. A., Butz, D. A., & Baumeister, R. F. (2007). Increasing self-regulatory strength can reduce the depleting effect of suppressing stereotypes. *Personality and Social Psychology Bulletin, 33*, 281-294.
- Galanti, K., Gluck, M. E., & Geliebter, A. (2007). Test meal intake in obese binge eaters in relation to impulsivity and compulsivity. *International Journal of Eating Disorders, 40*, 727–732. doi:10.1002/eat
- Galic, M. A., & Persinger, M. A. (2002). Voluminous sucrose consumption in female rats: Increased “nippiness” during periods of sucrose removal and possible oestrus periodicity. *Psychological Reports, 90*, 58–60.
- Gandiga, P. C., Hummel, F. C., & Cohen, L. G. (2006). Transcranial DC stimulation (tDCS): a tool for double-blind sham-controlled clinical studies in brain stimulation. *Clinical Neurophysiology, 117*, 845–850. doi:10.1016/j.clinph.2005.12.003
- Garavan, H., Hester, R., Murphy, K., Fassbender, C., & Kelly, C. (2006). Individual differences in the functional neuroanatomy of inhibitory control. *Brain Research, 1105*, 130–142. doi:10.1016/j.brainres.2006.03.029
- Garavan, H., Ross, T. J., & Stein, E. A. (1999). Right hemispheric dominance of inhibitory control: an event-related functional MRI study. *Proceedings of the National Academy of Sciences of the United States of America, 96*, 8301–6.
- Gasquoine, P. G. (2013). Localization of function in anterior cingulate cortex: from psychosurgery to functional neuroimaging. *Neuroscience and Biobehavioral Reviews, 37*, 340–348. doi:10.1016/j.neubiorev.2013.01.002
- Gautier, J. F., Chen, K., Salbe, A. D., Bandy, D., Pratley, R. E., Heiman, M., Ravussin, E., Reiman, E. M., & Tataranni, P. A. (2000). Differential brain responses to satiation in obese and lean men. *Diabetes, 49*, 838–846. doi: 10.2337/diabetes.49.5.838
- Gearhardt, A. N., Corbin, W. R., & Brownell, K. D. (2009a). Food addiction: An examination of the diagnostic criteria for dependence. *Journal of Addiction Medicine, 3*, 1–8.
- Gearhardt, A. N., Corbin, W. R., & Brownell, K. D. (2009b). Preliminary validation of the Yale Food Addiction Scale. *Appetite, 52*, 430–436. doi:10.1016/j.appet.2008.12.003
- Gearhardt, A. N., Davis, C., Kushner, R., & Brownell, K. D. (2011b). The addiction potential of hyperpalatable foods. *Current Drug Abuse Reviews, 4*, 140–145.

- Gearhardt, A. N., Grilo, C. M., DiLeone, R. J., Brownell, K. D., & Potenza, M. N. (2011c). Can food be addictive? Public health and policy implications. *Addiction, 106*, 1208–1212. doi:10.1111/j.1360-0443.2010.03301.x
- Gearhardt, A. N., Roberto, C. A., Seamans, M. J., Corbin, W. R., & Brownell, K. D. (2013). Preliminary validation of the Yale Food Addiction Scale for children. *Eating Behaviors, 14*, 508–512. doi:10.1016/j.eatbeh.2013.07.002
- Gearhardt, A. N., White, M. A., Masheb, R. M., Morgan, P. T., Crosby, R. D., & Grilo, C. M. (2012). An examination of the food addiction construct in obese patients with binge eating disorder. *The International Journal of Eating Disorders, 45*, 657–663. doi:10.1002/eat.20957
- Gearhardt, A. N., Yokum, S., Orr, P. T., Stice, E., Corbin, W. R., & Brownell, K. D. (2011a). Neural correlates of food addiction. *Archives of General Psychiatry, 68*, 808–816. doi:10.1001/archgenpsychiatry.2011.32
- Geiger, B. M., Haburcak, M., Avena, N. M., Moyer, M. C., Hoebel, B. G., & Pothos, E. N. (2009). Deficits of mesolimbic dopamine neurotransmission in rat dietary obesity. *Neuroscience, 159*, 1193–1199. doi:10.1016/j.neuroscience.2009.02.007
- Gendall, K. A., Sullivan, P. F., Joyce, P. R., Fear, J. L., & Bulik, C. M. (1997). Psychopathology and personality of young women who experience food cravings. *Addictive Behaviors, 22*, 545–555.
- Gerrits, J. H., O'Hara, R. E., Piko, B. F., Gibbons, F. X., de Ridder, D. T. D., Keresztes, N., Kamble, S. V., & de Wit, J. B. F. (2010). Self-control, diet concerns and eater prototypes influence fatty foods consumption of adolescents in three countries. *Health Education Research, 25*, 1031–1041. doi:10.1093/her/cyq055
- Ghahremani, D. G., Lee, B., Robertson, C. L., Tabibnia, G., Morgan, A. T., De Shetler, N., Brown, A. K., Monterosso, J. R., Aron, A. A., Mandelkern, M. A., Poldrack, R. A., & London, E. D. (2012). Striatal dopamine D₂/D₃ receptors mediate response inhibition and related activity in frontostriatal neural circuitry in humans. *The Journal of Neuroscience, 32*, 7316–7324. doi:10.1523/JNEUROSCI.4284-11.2012
- Giancola, P. R., & Tarter, R. E. (1999). Executive cognitive functioning and risk for substance abuse. *Psychological Science, 10*, 203–205.
- Gibson, C. D., Karmally, W., McMahan, D. J., Wardlaw, S. L., & Korner, J. (2012). Randomized pilot study of cabergoline, a dopamine receptor agonist: Effects on body weight and glucose tolerance in obese adults. *Diabetes, Obesity and Metabolism, 14*, 335–340. doi:10.1111/j.1463-1326.2011.01534.x.
- Gibson, E. L., & Desmond, E. (1999). Chocolate craving and hunger state: implications for the acquisition and expression of appetite and food choice. *Appetite, 32*, 219–40. doi:10.1006/appe.1998.0207
- Giesen, C., & Rothermund, K. (2013). You better stop! Binding “stop” tags to irrelevant stimulus features. *Quarterly Journal of Experimental Psychology, 67*, 809–32. doi:10.1080/17470218.2013.834372
- Glass, J. M., Buu, A., Adams, K. M., Nigg, J. T., Puttler, L. I., Jester, J. M., & Zucker, R. A. (2009). Effects of alcoholism severity and smoking on executive neurocognitive function. *Addiction, 104*, 38–48. doi:10.1111/j.1360-0443.2008.02415.x
- Gold, M. S., Frost-Pineda, K., & Jacobs, W. S. (2003). Overeating, binge eating, and eating disorders as addictions. *Psychiatric Annals, 33*, 117–122.
- Gold, M. S., Graham, N. A., Cocores, J. A., & Nixon, S. J. (2009). Food addiction? *Journal of Addiction Medicine, 3*, 42–45. doi:10.1097/ADM.0b013e318199cd20
- Golding, J. F., Harpur, T., & Brent-Smith, H. (1983). Personality, drinking and drug-taking cigarette smoking correlates of. *Personality and Individual Differences, 4*, 703–706.

- Goldman, R. L., Borckardt, J. J., Frohman, H. A., O'Neil, P. M., Madan, A., Campbell, L. K., Budak, A., & George, M. S. (2011). Prefrontal cortex transcranial direct current stimulation (tDCS) temporarily reduces food cravings and increases the self-reported ability to resist food in adults with frequent food craving. *Appetite*, *56*, 741–746. doi:10.1016/j.appet.2011.02.013
- Goldstein, R. Z., & Volkow, N. D. (2002). Drug addiction and its underlying neurobiological basis: neuroimaging evidence for the involvement of the frontal cortex. *American Journal of Psychiatry*, *159*, 1642–1652.
- Goldstein, R. Z., & Volkow, N. D. (2011). Dysfunction of the prefrontal cortex in addiction: neuroimaging findings and clinical implications. *Nature Reviews. Neuroscience*, *12*, 652–69. doi:10.1038/nrn3119
- Goldstein, S. P., Forman, E. M., Meiran, N., Herbert, J. D., Juarascio, A. S., & Butryn, M. L. (2014). The discrepancy between implicit and explicit attitudes in predicting disinhibited eating. *Eating Behaviors*, *15*, 164–170. doi:10.1016/j.eatbeh.2013.10.021
- Gollwitzer, P.M., & Sheeran, P. (2006). Implementation intentions and goal achievement: A meta-analysis of effects and processes. *Advances in Experimental Social Psychology*, *38*, 69-119.
- Goudie, A. J., Cooper, G. D., & Halford, J. C. G. (2005). Antipsychotic-induced weight gain. *Diabetes, Obesity and Metabolism*, *7*, 478–487. doi: 10.1111/j.1463-1326.2004.00413.x
- Grau, E., & Ortet, G. (1999). Personality traits and alcohol consumption in a sample of non-alcoholic women. *Personality and Individual Differences*, *27*, 1057–1066. doi:10.1016/S0191-8869(99)00047-1
- Greenwald, A. G., McGhee, D. E., & Schwartz, J. L. K. (1998). Measuring individual differences in implicit cognition: The implicit association test. *Journal of Personality and Social Psychology*, *74*, 1464-1480.
- Greenwald, A. G., Nosek, B. A., & Banaji, M. R. (2003). Understanding and using the implicit association test: I. An improved scoring algorithm. *Attitudes and Social Cognition*, *85*, 197-216.
- Greenwald, A. G., Smith, C. T., Sriram, N., Bar-Anan, Y., & Nosek, B. A. (2009). Implicit race attitudes predicted vote in the 2008 U.S. presidential election. *Analyses of Social Issues and Public Policy*, *9*, 241–253. doi:10.1111/j.1530-2415.2009.01195.x
- Griffin, K. W., Scheier, L. M., Acevedo, B., Grenard, J. L., & Botvin, G. J. (2012). Long-term effects of self-control on alcohol use and sexual behavior among urban minority young women. *International Journal of Environmental Research and Public Health*, *9*, 1–23.
- Griffiths, L. J., Wolke, D., Page, A. S., & Horwood, J. P. (2006). Obesity and bullying: different effects for boys and girls. *Archives of Disease in Childhood*, *91*, 121–125. doi:10.1136/adc.2005.072314
- Gross, J. J., & John, O. P. (2003). Individual differences in two emotion regulation processes: Implications for affect, relationships, and well-being. *Journal of Personality and Social Psychology*, *85*, 348-362.
- Guerrieri, R., Nederkoorn, C., & Jansen, A. (2007b). How impulsiveness and variety influence food intake in a sample of healthy women. *Appetite*, *48*, 119–122. doi:10.1016/j.appet.2006.06.004
- Guerrieri, R., Nederkoorn, C., & Jansen, A. (2008a). The interaction between impulsivity and a varied food environment: its influence on food intake and overweight. *International Journal of Obesity*, *32*, 708–714. doi:10.1038/sj.ijo.0803770

- Guerrieri, R., Nederkoorn, C., & Jansen, A. (2008b). The effect of an impulsive personality on overeating and obesity: Current state of affairs. *Psychological Topics, 17*, 265–286.
- Guerrieri, R., Nederkoorn, C., & Jansen, A. (2012). Disinhibition is easier learned than inhibition. The effects of (dis)inhibition training on food intake. *Appetite, 59*, 96–99.
- Guerrieri, R., Nederkoorn, C., Schrooten, M., Martijn, C., & Jansen, A. (2009). Inducing impulsivity leads high and low restrained eaters into overeating, whereas current dieters stick to their diet. *Appetite, 53*, 93–100.
- Guerrieri, R., Nederkoorn, C., Stankiewicz, K., Alberts, H., Geschwind, N., Martijn, C., & Jansen, A. (2007a). The influence of trait and induced state impulsivity on food intake in normal-weight healthy women. *Appetite, 49*, 66–73. doi:10.1016/j.appet.2006.11.008
- Guitart-Masip, M., Huys, Q. J. M., Fuentemilla, L., Dayan, P., Duzel, E., & Dolan, R. J. (2012). Go and no-go learning in reward and punishment: Interactions between affect and effect. *NeuroImage, 62*, 154–66. doi:10.1016/j.neuroimage.2012.04.024
- Gullo, M. J., & Dawe, S. (2008). Impulsivity and adolescent substance use: Rashly dismissed as “all-bad”? *Neuroscience and Biobehavioral Reviews, 32*, 1507–1518. doi:10.1016/j.neubiorev.2008.06.003
- Gunstad, J., Paul, R. H., Cohen, R. A., Tate, D. F., Spitznagel, M. B., & Gordon, E. (2007). Elevated body mass index is associated with executive dysfunction in otherwise healthy adults. *Comprehensive Psychiatry, 48*, 57–61. doi:10.1016/j.comppsy.2006.05.001
- Hagger, M. S., & Chatzisarantis, N. L. D. (2013). The strength model of self-control: Recent advances and implications for public health. In P. A. Hall (Ed.), *Social neuroscience and public health: Foundations for the science of chronic disease prevention*. New York: Springer New York.
- Hagger, M. S., Wood, C. W., Stiff, C., & Chatzisarantis, N. L. D. (2009). The strength model of self-regulation failure and health-related behaviour. *Health Psychology Review, 3*, 208–238.
- Hagger, M. S., Wood, C. W., Stiff, C., & Chatzisarantis, N. L. D. (2010). Ego depletion and the strength model of self-control: A meta-analysis. *Psychological Bulletin, 136*, 495–525.
- Hajnal, A., Smith, G. P., & Norgren, R. (2004). Oral sucrose stimulation increases accumbens dopamine in the rat. *American Journal of Physiology. Regulatory, Integrative and Comparative Physiology, 286*, R31–37. doi:10.1152/ajpregu.00282.2003
- Hall, P. A. (2012). Executive control resources and frequency of fatty food consumption: findings from an age-stratified community sample. *Health Psychology, 31*, 235–41. doi:10.1037/a0025407
- Hall, P. A., & Fong, G. T. (2007). Temporal self-regulation theory: A model for individual health behavior. *Health Psychology Review, 1*, 6–52. doi:10.1080/17437190701492437
- Hall, P. A., Fong, G. T., Epp, L. J., & Elias, L. J. (2008). Executive function moderates the intention-behavior link for physical activity and dietary behavior. *Psychology & Health, 23*, 309–326. doi:10.1080/14768320701212099
- Hampshire, A., Chamberlain, S. R., Monti, M. M., Duncan, J., & Owen, A. M. (2010). The role of the right inferior frontal gyrus: inhibition and attentional control. *NeuroImage, 50*, 1313–9. doi:10.1016/j.neuroimage.2009.12.109
- Han, D. H., Yoon, S. J., Sung, Y. H., Lee, Y. S., Kee, B. S., Lyoo, I. K., Renshaw, P. F., & Cho, S. C. (2008). A preliminary study: Novelty seeking, frontal executive function, and dopamine receptor (D2) TaqI A gene polymorphism in patients with methamphetamine dependence. *Comprehensive Psychiatry, 49*, 387–392. doi:10.1016/j.comppsy.2008.01.008

- Hanlon, C. A., Hartwell, K. J., Canterberry, M., Li, X., Owens, M., LeMatty, T., Prisciandaro, J. J., Borckardt, J., Brady, K. T., & George, M. S. (2013). Reduction of cue-induced craving through realtime neurofeedback in nicotine users: The role of region of interest selection and multiple visits. *Psychiatry Research, 213*, 79–81. doi:10.1016/j.psychresns.2013.03.003
- Hardman, C. A., Herbert, V. M. B., Brunstrom, J. M., Munafò, M. R., & Rogers, P. J. (2012). Dopamine and food reward: Effects of acute tyrosine/phenylalanine depletion on appetite. *Physiology & Behavior, 105*, 1202–1207. doi:10.1016/j.physbeh.2011.12.022
- 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. doi:10.1016/j.appet.2013.08.021
- Hardman, C. A., Scott, J., Field, M., & Jones, A. (2014). To eat or not to eat. The effects of expectancy on reactivity to food cues. *Appetite, 76*, 153–60. doi:10.1016/j.appet.2014.02.005
- Hare, T. A., Malmaud, J., & Rangel, A. (2011). Focusing attention on the health aspects of foods changes value signals in vmPFC and improves dietary choice. *The Journal of Neuroscience, 31*, 11077–87. doi:10.1523/JNEUROSCI.6383-10.2011
- Hare, T. A., Camerer, C. F., & Rangel, A. (2009). Self-control in decision-making involves modulation of the vmPFC valuation system. *Science, 324*, 646–8. doi:10.1126/science.1168450
- Harrison, D. M. (2008). Oral sucrose for pain management in infants: Myths and misconceptions. *Journal of Neonatal Nursing, 14*, 39–46. doi:10.1016/j.jnn.2007.12.002
- Hartwell, K. J., Prisciandaro, J. J., Borckardt, J., Li, X., George, M. S., & Brady, K. T. (2013). Real-time fMRI in the treatment of nicotine dependence: A conceptual review and pilot studies. *Psychology of Addictive Behaviors, 27*, 501–509. doi:10.1037/a0028215
- Haslam, D. W., & James, W. P. T. (2005). Obesity. *Lancet, 366*, 1197–209. doi:10.1016/S0140-6736(05)67483-1
- Havermans, R. C., Giesen, J. C. A. H., Houben, K., & Jansen, A. (2011). Weight, gender, and snack appeal. *Eating Behaviors, 12*, 126–130. doi:10.1016/j.eatbeh.2011.01.010
- Haworth, C. M. A., Harlaar, N., Kovas, Y., Davis, O. S. P., Oliver, B. R., Hayiou-Thomas, M. E., ... Plomin, R. (2007). Internet cognitive testing of large samples needed in genetic research. *Twin Research and Human Genetics, 10*, 554–63. doi:10.1375/twin.10.4.554
- Hayden-Wade, H. A., Stein, R. I., Ghaderi, A., Saelens, B. E., Zabinski, M. F., & Wilfley, D. E. (2005). Prevalence, characteristics, and correlates of teasing experiences among overweight children vs. non-overweight peers. *Obesity Research, 13*, 1381–1392.
- Hayes, A. F., & Matthes, J. (2009). Computational procedures for probing interactions in OLS and logistic regression: SPSS and SAS implementations. *Behavior Research Methods, 41*, 924–936.
- Hays, N. P., & Roberts, S. B. (2008). Aspects of eating behaviors “disinhibition” and “restraint” are related to weight gain and BMI in women. *Obesity, 16*, 52–58.
- Hays, N. P., Bathalon, G. P., McCrory, M. A., Roubenoff, R., Lipman, R., & Roberts, S. B. (2002). Eating behavior correlates of adult weight gain and obesity in healthy women aged 55–65 y. *The American Journal of Clinical Nutrition, 75*, 476–83.
- Heatherton, T. F., Herman, C. P., Polivy, J., King, G. A., & McGree, S. T. (1988). The (mis) measurement of restraint: An analysis of conceptual and psychometric issues. *Journal of Abnormal Psychology, 97*, 19–28.

- Hedley, A. A., Ogden, C. L., Johnson, C. L., Carroll, M. D., Curtin, L. R. & Flegal, K. M. (2004). Prevalence of overweight and obesity among US children, adolescents, and adults, 1999-2002. *The Journal of the American Medical Association*, *291*, 2847-2850.
- Heinz, A., Siessmeier, T., Wrase, J., Hermann, D., Klein, S., Grüsser-Sinopoli, S. M., Flor, H., Braus, D. F., Buchholz, H. G., Gründer, G., Schreckenberger, M., Smolka, M. N., Rösch, F., Mann, K., & Bartenstein, P. (2004). Correlation between dopamine D2 receptors in the ventral striatum and central processing of alcohol cues and craving. *The American Journal of Psychiatry*, *161*, 1783–1789. doi:10.1176/appi.ajp.161.10.1783
- Henrich, J., Heine, S. J., & Norenzayan, A. (2010). The weirdest people in the world? *The Behavioral and Brain Sciences*, *33*, 61–83. doi:10.1017/S0140525X0999152X
- Herman, C. P., & Mack, D. (1975). Restrained and unrestrained eating. *Journal of Personality*, *43*, 647-660.
- Herman, C. P., & Polivy, J. (1975). Anxiety, restraint, and eating behavior. *Journal of Abnormal Psychology*, *84*, 666–672.
- Herman, C. P., & Polivy, J. P. (1980). Restrained eating. In A. J. Stunkard (Ed.), *Obesity* (pp. 208–225). Philadelphia: Saunders.
- Herman, C.P., & Polivy, J. (2004). The self-regulation of eating. In R. F. Baumeister & K. D. Vohs (Eds.), *The handbook of self-regulation: Research, theory, and applications* (pp. 492–508). New York: Guilford.
- Hernandez, L., & Hoebel, B. G. (1988). Food reward and cocaine increase extracellular dopamine in the nucleus accumbens as measured by microdialysis. *Life Sciences*, *42*, 1705–1712.
- Herremans, S. C., & Baeken, C. (2012). The current perspective of neuromodulation techniques in the treatment of alcohol addiction: A systematic review. *Psychiatria Danubina*, *24 Suppl 1*, S14–20.
- Herremans, S. C., Baeken, C., Vanderbruggen, N., Vanderhasselt, M. A., Zeeuws, D., Santermans, L., & De Raedt, R. (2012). No influence of one right-sided prefrontal HF-rTMS session on alcohol craving in recently detoxified alcohol-dependent patients: results of a naturalistic study. *Drug and Alcohol Dependence*, *120*, 209–213. doi:10.1016/j.drugalcdep.2011.07.021
- Hester, R., & Garavan, H. (2004). Executive dysfunction in cocaine addiction: Evidence for discordant frontal, cingulate, and cerebellar activity. *The Journal of Neuroscience*, *24*, 11017–22. doi:10.1523/JNEUROSCI.3321-04.2004
- Hester, R., Bell, R. P., Foxe, J. J., & Garavan, H. (2013). The influence of monetary punishment on cognitive control in abstinent cocaine-users. *Drug and Alcohol Dependence*, *133*, 86–93. doi:10.1016/j.drugalcdep.2013.05.027
- Hetherington, M. M., & Macdiarmid, J. I. (1993). “Chocolate addiction”: A preliminary study of its description and its relationship to problem eating. *Appetite*, *21*, 233-246.
- Hetherington, M. M., & Macdiarmid, J.I. (1995). Pleasure and excess: Liking for and overconsumption of chocolate. *Physiology and Behavior*, *57*, 27-35.
- Hill, A. J. (2004). Does dieting make you fat? *British Journal of Nutrition*, *92*, S1- S15. doi:10.1079/BJN20041135
- Hill, A. J., & Heaton-Brown, L. (1994). The experience of food craving: A prospective investigation in healthy women. *Journal of Psychosomatic Research*, *38*, 801–814.
- Hill, A. J., Weaver, C. F., & Blundell, J. E. (1991). Food craving, dietary restraint and mood. *Appetite*, *17*, 187–197.
- Hill, J. O., & Peters, J. C. (1998). Environmental contributions to the obesity epidemic. *Science*, *280*, 1371-1374

- Hirshi, T. (2004). Self-control and crime. In R. F. Baumeister & K. D. Vohs (Eds.), *Handbook of Self-Regulation: Research, Theory and Applications* (pp. 537-552). New York, NY: Guildford Press.
- Hoefling, A., & Strack, F. (2008). The tempting effect of forbidden foods. High calorie content evokes conflicting implicit and explicit evaluations in restrained eaters. *Appetite, 51*, 681–689.
- Hofmann, W., Friese, M., & Roefs, A. (2009a). Three ways to resist temptation: The independent contributions of executive attention, inhibitory control, and affect regulation to the impulse control of eating behavior. *Journal of Experimental Social Psychology, 45*, 431–435. doi:10.1016/j.jesp.2008.09.013
- Hofmann, W., Friese, M., & Strack, F. (2009b). Impulse and self-control from a dual-systems perspective. *Perspectives on Psychological Science, 4*, 162–176.
- Hofmann, W., Friese, M., & Wiers, R. W. (2008). Impulsive versus reflective influences on health behavior: A theoretical framework and empirical review. *Health Psychology Review, 2*, 111–137.
- Hofmann, W., Rauch, W., & Gawronski, B. (2007). And deplete us not into temptation: Automatic attitudes, dietary restraint, and self-regulatory resources as determinants of eating behavior. *Journal of Experimental Social Psychology, 43*, 497–504.
- Hofmann, W., Schmeichel, B. J., & Baddeley, A. D. (2012). Executive functions and self-regulation. *Trends in Cognitive Sciences, 16*, 174–80.
- Hogarth, L., Dickinson, A., Hutton, S. B., Bamborough, H., & Duka, T. (2006). Contingency knowledge is necessary for learned motivated behaviour in humans: relevance for addictive behaviour. *Addiction, 101*, 1153–66. doi:10.1111/j.1360-0443.2006.01459.x
- Hollitt, S., Kemps, E., Tiggemann, M., Smeets, E., & Mills, J. S. (2010). Components of attentional bias for food cues among restrained eaters. *Appetite, 54*, 309–313. doi:10.1016/j.appet.2009.12.005
- Hollmann, M., Hellrung, L., Pleger, B., Schlögl, H., Kabisch, S., Stumvoll, M., Villringer, A., & Horstmann, A. (2012). Neural correlates of the volitional regulation of the desire for food. *International Journal of Obesity, 36*, 648–655. doi:10.1038/ijo.2011.125
- Horrell, T., El-Baz, A., Baruth, J., Tasman, A., Sokhadze, G., Stewart, C., & Sokhadze, E. (2010). Neurofeedback effects on evoked and induced EEG gamma band reactivity to drug-related cues in cocaine addiction. *Journal of Neurotherapy, 14*, 195–216. doi:10.1080/10874208.2010.501498.
- 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*, 424–427. doi:10.1016/j.appet.2011.01.019
- Houben, K. (2011). Overcoming the urge to splurge: Influencing eating behavior by manipulating inhibitory control. *Journal of Behavior Therapy and Experimental Psychiatry, 42*, 384-388.
- Houben, K., & Jansen, A. (2011). Training inhibitory control. A recipe for resisting sweet temptations. *Appetite, 56*, 345-349.
- Houben, K., & Wiers, R. W. (2009). Response inhibition moderates the relationship between implicit associations and drinking behavior. *Alcoholism: Clinical and Experimental Research, 33*, 626-633.
- Houben, K., Havermans, R. C., Nederkoorn, C., & Jansen, A. (2012a). Beer à no-go: Learning to stop responding to alcohol cues reduces alcohol intake via reduced affective associations rather than increased response inhibition. *Addiction, 107*, 1280-1287.

- Houben, K., Nederkoorn, C., & Jansen, A. (2012b). Too tempting to resist? Past success at weight control rather than dietary restraint determines exposure-induced disinhibited eating. *Appetite*, *59*, 550–5. doi:10.1016/j.appet.2012.07.004
- Houben, K., Nederkoorn, C., Wiers, R. W., & Jansen, A. (2011a). Resisting temptation: Decreasing alcohol-related affect and drinking behaviour by training response inhibition. *Drug and Alcohol Dependence*, *116*, 132-136.
- Houben, K., Roefs, A., & Jansen, A. (2010a). Guilty pleasures: Implicit preferences for low and high calorie food in restrained eating. *Appetite*, *55*, 18-24.
- Houben, K., Roefs, A., & Jansen, A. (2012c). Eating behaviors guilty pleasures II : Restrained eaters' implicit preferences for high, moderate and low-caloric food. *Eating Behaviors*, *13*, 275–277.
- Houben, K., Wiers, R. W., & Jansen, A. (2011b). Getting a grip on drinking behavior: training working memory to reduce alcohol abuse. *Psychological Science*, *22*, 968-975.
- Hull, J. G., & Slone, L. B. (2004). Alcohol and self-regulation. In R. F. Baumeister & K. D. Vohs (Eds.), *Handbook of Self-Regulation: Research, Theory and Applications* (pp. 466-491). New York, NY: Guilford Press.
- Hung, I. W., & Labroo, A. A. (2011). From firm muscles to firm willpower: Understanding the role of embodied cognition in self-regulation. *Journal of Consumer Research*, *37*, 1046–1064. doi:10.1086/657240
- Iacono, W. G., Malone, S. M., & McGue, M. (2008). Behavioral disinhibition and the development of early-onset addiction: common and specific influences. *Annual Review of Clinical Psychology*, *4*, 325–348. doi:10.1146/annurev.clinpsy.4.022007.141157
- Ifland, J. R., Preuss, H. G., Marcus, M. T., Rourke, K. M., Taylor, W. C., Burau, K., Jacobs, W. S., Kadish, W., & Manso, G. (2009). Refined food addiction: A classic substance use disorder. *Medical Hypotheses*, *72*, 518–526. doi:10.1016/j.mehy.2008.11.035
- Inzlicht, M., & Schmeichel, B. J. (2012). What is ego depletion? Toward a mechanistic revision of the resource model of self-control. *Perspectives on Psychological Science*, *7*, 450–463. doi:10.1177/1745691612454134
- Inzlicht, M., Schmeichel, B. J., & Macrae, C. N. (2014). Why self-control seems (but may not be) limited. *Trends in Cognitive Sciences*, *18*, 127–33. doi:10.1016/j.tics.2013.12.009
- Irvin, J. E., Bowers, C. A., Dunn, M. E., & Wang, M. C. (1999). Efficacy of relapse prevention: a meta-analytic review. *Journal of consulting and clinical Psychology*, *67*, 563-570.
- Jacobson, L., Javitt, D. C., & Lavidor, M. (2011). Activation of inhibition: Diminishing impulsive behavior by direct current stimulation over the inferior frontal gyrus. *Journal of Cognitive Neuroscience*, *23*, 3380–7. doi:10.1162/jocn_a_00020
- Jansen, A. (1998). A learning model of binge eating: Cue reactivity and cue exposure. *Behaviour Research and Therapy*, *36*, 257–272.
- Jansen, A., & van den Hout, M. (1991). On being led into temptation: "counterregulation" of dieters after smelling a "preload". *Addictive Behaviors*, *16*, 247-253.
- Jansen, A., Klaver, J., Merckelbach, H., & van den Hout, M. (1989). Restrained eaters are rapidly habituating sensation seekers. *Behaviour Research and Therapy*, *27*, 247-252.
- Jansen, A., Nederkoorn, C., van Baak, L., Keirse, C., Guerrieri, R., & Havermans, R. (2009). High-restrained eaters only overeat when they are also impulsive. *Behaviour Research and Therapy*, *47*, 105-110.
- Jansen, J. M., Daams, J. G., Koeter, M. W. J., Veltman, D. J., van den Brink, W., & Goudriaan, A. E. (2013). Effects of non-invasive neurostimulation on craving: A meta-analysis. *Neuroscience and Biobehavioral Reviews*, *37*, 2472–2480. doi:10.1016/j.neubiorev.2013.07.009

- Janssen, I., Katzmarzyk, P. T., & Ross, R. (2004). Waist circumference and not body mass index explains obesity-related health risk. *The American Journal of Clinical Nutrition*, *79*, 379–84.
- Jasinska, A. J., Yasuda, M., Burant, C. F., Gregor, N., Khatri, S., Sweet, M., & Falk, E. B. (2012). Impulsivity and inhibitory control deficits are associated with unhealthy eating in young adults. *Appetite*, *59*, 738–747. doi:10.1016/j.appet.2012.08.001
- Jeffery, R. W., & Utter, J. (2003). The changing environment and population obesity in the United States. *Obesity Research*, *11*, 12S–22S.
- Jeffery, R.W., Drewnowski, A., Epstein, L. H., Stunkard, A. J., Wilson, G. T., Wing, R. R., & Hill, D. R. (2000). Long term maintenance of weight loss: Current status. *Health Psychology*, *19*, S5-S16.
- Jentsch, J. D., & Pennington, Z. T. (2014). Reward, interrupted: Inhibitory control and its relevance to addictions. *Neuropharmacology*, *76 Pt B*, 479–486. doi:10.1016/j.neuropharm.2013.05.022
- Jia, H., & Lubetkin, E. I. (2010). Trends in quality-adjusted life-years lost contributed by smoking and obesity. *American Journal of Preventive Medicine*, *38*, 138–144. doi:10.1016/j.amepre.2009.09.043
- John, O. P., Naumann, L. P., & Soto, C. J. (2008). Paradigm shift to the integrative big-five trait taxonomy: history, measurement, and conceptual issues. In O. P. John, R. W. Robins, & L. A. Pervin (Eds.), *Handbook of personality: Theory and research* (pp. 114–158). New York, NY: Guilford Press.
- Johnson, F., Pratt, M., & Wardle, J. (2012). Dietary restraint and self-regulation in eating behavior. *International Journal of Obesity*, *36*, 665–674. doi:10.1038/ijo.2011.156
- Johnson, P. M., & Kenny, P. J. (2010). Dopamine D2 receptors in addiction-like reward dysfunction and compulsive eating in obese rats. *Nature Neuroscience*, *13*, 635–641. doi:10.1038/nn.2519
- Jones, A., & Field, M. (2013). The effects of cue-specific inhibition training on alcohol consumption in heavy social drinkers. *Experimental and Clinical Psychopharmacology*, *21*, 8–16. doi:10.1037/a0030683
- Jones, A., Christiansen, P., Nederkoorn, C., Houben, K., & Field, M. (2013). Fluctuating disinhibition: implications for the understanding and treatment of alcohol and other substance use disorders. *Frontiers in Psychiatry*, *4*, 140. doi:10.3389/fpsy.2013.00140
- Jones, A., Guerrieri, R., Fernie, G., Cole, J., Goudie, A., & Field, M. (2011). The effects of priming restrained versus disinhibited behaviour on alcohol-seeking in social drinkers. *Drug and Alcohol Dependence*, *113*, 55–61. doi:10.1016/j.drugalcdep.2010.07.006
- Jones, A., McGrath, E., Houben, K., Nederkoorn, C., Robinson, E., & Field, M. (2014). A comparison of three types of web-based inhibition training for the reduction of alcohol consumption in problem drinkers: study protocol. *BMC Public Health*, *14*, 796. doi:10.1186/1471-2458-14-796
- Jönsson, E. G., Nöthen, M. M., Grünhage, F., Farde, L., Nakashima, Y., Propping, P., & Sedvall, G. C. (1999). Polymorphisms in the dopamine D2 receptor gene and their relationships to striatal dopamine receptor density of healthy volunteers. *Molecular Psychiatry*, *4*, 290–296.
- Jorm, A. F., Christensen, H., Henderson, A. S., Jacomb, P. A., Korten, A. E., & Rodgers, B. (1999). Using the BIS / BAS scales to measure behavioural inhibition and behavioural activation: Factor structure, validity and norms in a large community sample. *Personality and Individual Differences*, *26*, 49–58.

- Kahan, D., Polivy, J., & Herman, C. P. (2003). Conformity and dietary disinhibition: a test of the ego-strength model of self-regulation. *The International Journal of Eating Disorders, 33*, 165–71. doi:10.1002/eat.10132
- Kahn, S. E., Hull, R. L., & Utzschneider, K. M. (2006). Mechanisms linking obesity to insulin resistance and type 2 diabetes. *Nature, 444*, 840–846. doi:10.1038/nature05482
- Kaiser, H. F. (1974). An index of factorial simplicity. *Psychometrika, 39*, 31–36.
- Kakoschke, N., Kemps, E., & Tiggemann, M. (2014). Attentional bias modification encourages healthy eating. *Eating Behaviors, 15*, 120–124. doi:10.1016/j.eatbeh.2013.11.001
- Kambouropoulos, N., & Staiger, P. K. (2001). The influence of sensitivity to reward on reactivity to alcohol-related cues. *Addiction, 96*, 1175–1185. doi:10.1080/09652140120060761
- Kane, T. A., Loxton, N. J., Staiger, P. K., & Dawe, S. (2004). Does the tendency to act impulsively underlie binge eating and alcohol use problems? An empirical investigation. *Personality and Individual Differences, 36*, 83–94. doi:10.1016/S0191-8869(03)00070-9
- Karhunen, L. J., Lappalainen, R. I., Tammela, L., Turpeinen, a K., & Uusitupa, M. I. (1997). Subjective and physiological cephalic phase responses to food in obese binge-eating women. *The International Journal of Eating Disorders, 21*, 321–328.
- Karpinsky, A., & Steinman, R. B. (2006). The single category implicit association test as a measure of implicit social cognition. *Journal of Personality and Social Psychology, 91*, 16-32.
- Keller, C., & van der Horst, K. (2013). Dietary restraint, ambivalence toward eating, and the valence and content of spontaneous associations with eating. *Appetite, 62*, 150–9. doi:10.1016/j.appet.2012.11.012
- Kemps, E., & Tiggemann, M. (2009). Attentional bias for craving-related (chocolate) food cues. *Experimental and Clinical Psychopharmacology, 17*, 425–433. doi:10.1037/a0017796
- Kemps, E., Tiggemann, M., Martin, R., & Elliott, M. (2013b). Implicit approach-avoidance associations for craved food cues. *Journal of Experimental Psychology. Applied, 19*, 30–38. doi:10.1037/a0031626
- Kemps, E., Tiggemann, M., Orr, J., & Grear, J. (2013a). Attentional retraining can reduce chocolate consumption. *Journal of Experimental Psychology. Applied, 20*, 94–102. doi:10.1037/xap0000005
- Killen, J. D., & Fortmann, S. P. (1997). Craving is associated with smoking relapse: findings from three prospective studies. *Experimental and Clinical Psychopharmacology, 5*, 137–142.
- King, J. M. (1978). Patterns of sugar consumption in early infancy. *Community Dentistry and Oral Epidemiology, 6*, 47–52.
- King, K. M., Fleming, C. B., Monahan, K. C., & Catalano, R. F. (2011). Changes in self-control problems and attention problems during middle school predict alcohol, tobacco, and marijuana use during high school. *Psychology of Addictive Behaviors, 25*, 69–79.
- Kiss, M., Raymond, J. E., Westoby, N., Nobre, A. C., & Eimer, M. (2008). Response inhibition is linked to emotional devaluation: Behavioural and electrophysiological evidence. *Frontiers in Human Neuroscience, 2*, 13. doi:10.3389/neuro.09.013.2008
- Kissileff, H. R. (1992). Where should human eating be studied and what should be measured? *Appetite, 19*, 61–68.
- Klajner, F., Herman, C. P., Polivy, J., & Chhabra, R. (1981). Human obesity, dieting, and anticipatory salivation to food. *Physiology & Behavior, 27*, 195–198.

- Klein, O., Doyen, S., Leys, C., Magalhaes de Saldanha da Gama, P. A., Miller, S., Questienne, L., & Cleeremans, A. (2012). Low hopes, high expectations: Expectancy effects and the replicability of behavioral experiments. *Perspectives on Psychological Science*, *7*, 572–584. doi:10.1177/1745691612463704
- Klein, T. A., Neumann, J., Reuter, M., Hennig, J., von Cramon, D. Y., & Ullsperger, M. (2007). Genetically determined differences in learning from errors. *Science*, *318*, 1642–1645. doi:10.1126/science.1145044
- Klingberg, T. (2010). Training and plasticity of working memory. *Trends in Cognitive Sciences*, *14*, 317–324.
- Klingberg, T., Forssberg, H., & Westerberg, H. (2002). Training of working memory in children with ADHD. *Journal of Clinical and Experimental Neuropsychology*, *24*, 781–791.
- Knight, L. J., & Boland, F. J. (1989). Restrained eating: An experimental disentanglement of the disinhibiting variables of perceived calories and food type. *Journal of Abnormal Psychology*, *98*, 412–20.
- Knoch, D., Gianotti, L. R. R., Pascual-leone, A., Treyer, V., Regard, M., Hohmann, M., & Brugger, P. (2006). Disruption of right prefrontal cortex by low-frequency repetitive transcranial magnetic stimulation induces risk-taking behavior. *The Journal of Neuroscience*, *26*, 6469–6472. doi:10.1523/JNEUROSCI.0804-06.2006
- Kohls, G., Peltzer, J., Herpertz-Dahlmann, B., & Konrad, K. (2009). Differential effects of social and non-social reward on response inhibition in children and adolescents. *Developmental Science*, *12*, 614–25. doi:10.1111/j.1467-7687.2009.00816.x
- Koob, G. F., Sanna, P. P., & Bloom, F. E. (1998). Neuroscience of addiction. *Neuron*, *21*, 467–476.
- Krahn, D. D., Kurth, C. L., Gomberg, E., & Drenowski, A. (2005). Pathological dieting and alcohol use in college women – a continuum of behaviors. *Eating Behaviors*, *6*, 43–52.
- Krantz, J. H., & Dalal, R. (2000). Validity of Web-based psychological research. In M. H. Birnbaum (Ed.), *Psychological Experiments on the Internet* (pp. 35–60). San Diego, CA: Academic Press
- Krieglmeyer, R., Deutsch, R., Houwer, J. De, & De Raedt, R. (2010). Being Moved: Valence activates approach-avoidance behavior independently of evaluation and approach-avoidance intentions. *Psychological Science*, *21*, 607–6013. doi:10.1177/0956797610365131
- Krishnan-Sarin, S., Reynolds, B., Duhig, A. M., Smith, A., Liss, T., McFetridge, A., Cavallo, D. A., Carroll, K. M., & Potenza, M. N. (2007). Behavioral impulsivity predicts treatment outcome in a smoking cessation program for adolescents. *Drug and Alcohol Dependence*, *88*, 79–82.
- Kuczmarski, M. F., Kuczmarski, R. J., & Najjar, M. (2001). Effects of age on validity of self-reported height, weight, and body mass index: Findings from the Third National Health and Nutrition Examination Survey, 1988-1994. *Journal of the American Dietetic Association*, *101*, 28-34.
- Kuijper, R., de Ridder, D., Ouweland, C., Houx, B., & van Den Bos, R. (2008). Dieting as a case of behavioural decision making. Does self-control matter? *Appetite*, *51*, 506–511. doi:10.1016/j.appet.2008.03.014
- Laessle, R. G., Tuschl, R. J., Kotthaus, B. C., & Pirke, K. M. (1989). A comparison of the validity of three scales for the assessment of dietary restraint. *Journal of Abnormal Psychology*, *98*, 504–7.
- Lafay, L., Thomas, F., Mennen, L., Charles, M. A., Eschwege, E., Borys, J. M., & Basdevant, A. (2001). Gender differences in the relation between food cravings and mood in an

- adult community: Results from the fleurbaix laventie ville santé study. *The International Journal of Eating Disorders*, 29, 195–204.
- Latner, J. D., Puhl, R. M., Murakami, J. M., & O'Brien, K. S. (2014). Food addiction as a causal model of obesity. Effects on stigma, blame, and perceived psychopathology. *Appetite*, 77, 77–82. doi:10.1016/j.appet.2014.03.004
- Lattimore, P., & Maxwell, L. (2004). Cognitive load, stress, and disinhibited eating. *Eating Behaviors*, 5, 315–324. doi:10.1016/j.eatbeh.2004.04.009
- Lawrence, A. J., Luty, J., Bogdan, N. A., Sahakian, B. J., & Clark, L. (2009). Impulsivity and response inhibition in alcohol dependence and problem gambling. *Psychopharmacology*, 207, 163–172. doi:10.1007/s00213-009-1645-x
- Lawrence, N. S., Hinton, E. C., Parkinson, J. A., & Lawrence, A. D. (2012). Nucleus accumbens response to food cues predicts subsequent snack consumption in women and increased body mass index in those with reduced self-control. *NeuroImage*, 63, 415–22. doi:10.1016/j.neuroimage.2012.06.070
- Lawrence, N. S., Verbruggen, F., Morrison, S., Adams, R. C., & Chambers, C. D. (under review). Stopping to food pictures reduces food intake: Effects of cue specificity, control conditions and individual differences.
- Lawrence, N. S., O'Sullivan, J., Parslow, D., Javaid, M., Adams, R. C., Chambers, C. D. & Verbruggen, F. (in preparation). Training response inhibition to food is associated with weight loss and reduced calorie intake.
- Le, D. S. N. T., Pannacciulli, N., Chen, K., Del Parigi, A., Salbe, A. D., Reiman, E. M., & Krakoff, J. (2006). Less activation of the left dorsolateral prefrontal cortex in response to a meal: A feature of obesity. *The American Journal of Clinical Nutrition*, 84, 725–731.
- Le, D. S. N. T., Pannacciulli, N., Chen, K., Salbe, A. D., Del Parigi, A., Hill, J. O., Wing, R. R., Reiman, E. M., & Krakoff, J. (2007). Less activation in the left dorsolateral prefrontal cortex in the reanalysis of the response to a meal in obese than in lean women and its association with successful weight loss. *The American Journal of Clinical Nutrition*, 86, 573–579.
- Lenartowicz, A., Verbruggen, F., Logan, G. D., & Poldrack, R. A. (2011). Inhibition-related activation in the right inferior frontal gyrus in the absence of inhibitory cues. *Journal of Cognitive Neuroscience*, 23, 3388–99. doi:10.1162/jocn_a_00031
- Leung, H.-C., & Cai, W. (2007). Common and differential ventrolateral prefrontal activity during inhibition of hand and eye movements. *The Journal of Neuroscience*, 27, 9893–900. doi:10.1523/JNEUROSCI.2837-07.2007
- Levitsky, D. A. (2005). The non-regulation of food intake in humans: Hope for reversing the epidemic of obesity. *Physiology & Behavior*, 86, 623–632. doi:10.1016/j.physbeh.2005.08.053
- Li, C. R., Huang, C., Constable, R. T., & Sinha, R. (2006). Imaging response inhibition in a stop-signal task: neural correlates independent of signal monitoring and post-response processing. *The Journal of Neuroscience*, 26, 186–92. doi:10.1523/JNEUROSCI.3741-05.2006
- Li, X., Hartwell, K. J., Borckardt, J., Prisciandaro, J. J., Saladin, M. E., Morgan, P. S., Johnson, K. A., LeMatty, T., Brady, K. T., & George, M. S. (2012). Volitional reduction of anterior cingulate cortex activity produces decreased cue craving in smoking cessation: A preliminary real-time fMRI study. *Addiction Biology*, 18, 739–748. doi:10.1111/j.1369-1600.2012.00449.x
- Liddle, P. F., Kiehl, K. A., & Smith, A. M. (2001). Event-related fMRI study of response inhibition. *Human Brain Mapping*, 12, 100–109.

- Lieberman, H. R., Wurtman, J. J., & Chew, B. (1986). Changes in mood after carbohydrate consumption among obese individuals. *The American Journal of Clinical Nutrition*, *44*, 772–778.
- Liebetanz, D., Nitsche, M. A, Tergau, F., & Paulus, W. (2002). Pharmacological approach to the mechanisms of transcranial DC-stimulation-induced after-effects of human motor cortex excitability. *Brain*, *125*, 2238–47.
- Lillis, J., Levin, M. E., & Trafton, J. A. (2012). Addictive Behaviors Elevated BMI and illicit drug use are associated with decreased ability to inhibit prepotent behaviors. *Addictive Behaviors*, *37*, 544–547. doi:10.1016/j.addbeh.2011.11.033
- Litt, M. D., Cooney, N. L., & Morse, P. (2000). Reactivity to alcohol-related stimuli in the laboratory and in the field: Predictors of craving in treated alcoholics. *Addiction*, *95*, 889–900.
- Lobstein, T. J., James, W. P. T., & Cole, T. J. (2003). Increasing levels of excess weight among children in England. *International Journal of Obesity*, *27*, 1136–1138. doi:10.1038/sj.ijo.0802324
- Lobstein, T., & Frelut, M. -L. (2003). Prevalence of overweight among children in Europe. *Obesity Reviews*, *4*, 195-200.
- Logan, G. D. (1985). Executive control of thought and action. *Acta Psychologica*, *60*, 193-210.
- Logan, G. D., & Cowan, W. B. (1984). On the ability to inhibit thought and action: A theory of an act of control. *Psychological Review*, *91*, 295-327.
- Logan, G. D., Schachar, R. J., & Tannock, R. (1997). Impulsivity and inhibitory control. *Psychological Science*, *8*, 60-64.
- Loo, C. K., McFarquhar, T. F., & Mitchell, P. B. (2008). A review of the safety of repetitive transcranial magnetic stimulation as a clinical treatment for depression. *The International Journal of Neuropsychopharmacology*, *11*, 131–147. doi:10.1017/S1461145707007717
- Lopez, R. B., Hofmann, W., Wagner, D. D., Kelley, W. M., & Heatherton, T. F. (2014). Neural predictors of giving in to temptation in daily life. *Psychological Science*. doi:10.1177/0956797614531492
- Lopresti, A. L., & Drummond, P. D. (2013). Obesity and psychiatric disorders: commonalities in dysregulated biological pathways and their implications for treatment. *Progress in Neuro-Psychopharmacology & Biological Psychiatry*, *45*, 92–99. doi:10.1016/j.pnpbp.2013.05.005
- Lotz, A., Uengoer, M., Koenig, S., Pearce, J. M., & Lachnit, H. (2012). An exploration of the feature-positive effect in adult humans. *Learning & Behavior*, *40*, 222–30. doi:10.3758/s13420-011-0057-z
- Lowe, M. R. (1993). The effects of dieting on eating behavior: A three-factor model. *Psychological Bulletin*, *114*, 100–121.
- Lowe, M. R., Annunziato, R. A., Markowitz, J. T., Didie, E., Bellace, D. L., Riddell, L., Maille, C., McKinney, S., & Stice, E. (2006). Multiple types of dieting prospectively predict weight gain during the freshman year of college. *Appetite*, *47*, 83–90. doi:10.1016/j.appet.2006.03.160
- Lowe, R., & Kleifield, E. I. (1988). Cognitive restraint, weight suppression, and the regulation of eating. *Appetite*, *10*, 159-168.
- Loxton, N. J., & Dawe, S. (2006). Reward and punishment sensitivity in dysfunctional eating and hazardous drinking women: Associations with family risk. *Appetite*, *47*, 361–371. doi:10.1016/j.appet.2006.05.014

- Lubman, D. I., Peters, L. A., Mogg, K., Bradley, B. P., & Deakin, J. F. W. (2000). Attentional bias for drug cues in opiate dependence. *Psychological Medicine*, *30*, 169-175.
- Ludwig, D. S., Peterson, K. E., & Gortmaker, S. L. (2001). Relation between consumption of sugar-sweetened drinks and childhood obesity: A prospective, observational analysis. *Lancet*, *357*, 505–508. doi:10.1016/S0140-6736(00)04041-1
- Luijten, M., Littel, M., & Franken, I. H. A. (2011). Deficits in inhibitory control in smokers during a Go/NoGo task: an investigation using event-related brain potentials. *PloS One*, *6*, e18898. doi:10.1371/journal.pone.0018898
- Luppino, F. S., de Wit, L. M., Bouvy, P. F., Stijnen, T., Cuijpers, P., Penninx, B. W. J. H., & Zitman, F. G. (2010). Overweight, obesity, and depression: A systematic review and meta-analysis of longitudinal studies. *Archives of General Psychiatry*, *67*, 220–229.
- Maayan, L., Hoogendoorn, C., Sweat, V., & Convit, A. (2011). Disinhibited eating in obese adolescents is associated with orbitofrontal volume reductions and executive dysfunction. *Obesity*, *19*, 1382–1387. doi:10.1038/oby.2011.15
- Macdiarmid, J. I., Loe, J., Kyle, J., & McNeill, G. (2013). “It was an education in portion size”. Experience of eating a healthy diet and barriers to long term dietary change. *Appetite*, *71*, 411–419. doi:10.1016/j.appet.2013.09.012
- MacDonald, A. W., Cohen, J. D., Stenger, V. A., & Carter, C. S. (2000). Dissociating the Role of the Dorsolateral Prefrontal and Anterior Cingulate Cortex in Cognitive Control. *Science*, *288*, 1835–1838. doi:10.1126/science.288.5472.1835
- Mahler, S. V., & de Wit, H. (2010). Cue-reactors: Individual differences in cue-induced craving after food or smoking abstinence. *PloS One*, *5*, e15475. doi:10.1371/journal.pone.0015475
- Maizey, L., Allen, C. P. G., Dervinis, M., Verbruggen, F., Varnava, A., Kozlov, M., Adams, R. C., Stokes, M., Klemen, J., Bungert, A., Hounsell, C. A., & Chambers, C. D. (2013). Comparative incidence rates of mild adverse effects to transcranial magnetic stimulation. *Clinical Neurophysiology*, *124*, 536–544. doi:10.1016/j.clinph.2012.07.024
- Majid, D. S. A., Cai, W., George, J. S., Verbruggen, F., & Aron, A. R. (2012). Transcranial magnetic stimulation reveals dissociable mechanisms for global versus selective corticomotor suppression underlying the stopping of action. *Cerebral Cortex*, *22*, 363–71. doi:10.1093/cercor/bhr112
- Malik, V. S., Schulze, M. B., & Hu, F. B. (2006). Intake of sugar-sweetened beverages and weight gain: A systematic review. *American Journal of Clinical Nutrition*, *84*, 274–288.
- Mann, T., Tomiyama, A. J., Westling, E., Lew, A.-M., Samuels, B., & Chatman, J. (2007). Medicare’s search for effective obesity treatments: Diets are not the answer. *The American Psychologist*, *62*, 220–233. doi:10.1037/0003-066X.62.3.220
- Martel, P., & Fantino, M. (1996a). Influence of the amount of food ingested on mesolimbic dopaminergic system activity: A microdialysis study. *Pharmacology, Biochemistry, and Behavior*, *55*, 297–302.
- Martel, P., & Fantino, M. (1996b). Mesolimbic dopaminergic system activity as a function of food reward: A microdialysis study. *Pharmacology, Biochemistry, and Behavior*, *53*, 221–226.
- Martin, L. E., Holsen, L. M., Chambers, R. J., Bruce, A. S., Brooks, W. M., Zarcone, J. R., Butler, M. G., & Savage, C. R. (2010). Neural mechanisms associated with food motivation in obese and healthy weight adults. *Obesity*, *18*, 254–260. doi:10.1038/oby.2009.220
- Massey, A., & Hill, A. J. (2012). Dieting and food craving. A descriptive, quasi-prospective study. *Appetite*, *58*, 781–785. doi:10.1016/j.appet.2012.01.020

- McCaffery, J. M., Haley, A. P., Sweet, L. H., Phelan, S., Raynor, H. A., Del Parigi, A., Cohen, R., & Wing, R. R. (2009). Differential functional magnetic resonance imaging response to food pictures in successful weight-loss maintainers relative to normal-weight and obese controls. *American Journal of Clinical Nutrition*, *90*, 928–934. doi:10.3945/ajcn.2009.27924.928
- McCrary, M. A., Fuss, P. J., McCallum, J. E., Yao, M., Vinken, A. G., Hays, N. P., & Roberts, S. B. (1999). Dietary variety within food groups: association with energy intake and body fatness in men and women. *The American Journal of Clinical Nutrition*, *69*, 440–447.
- McLaren, I.P.L., & Verbruggen, F. (2014). Association and inhibition. Submitted for publication.
- Meiselman, H. L. (1992). Methodology and theory in human eating research. *Appetite*, *19*, 49–55.
- Mela, D. J. (2001). Determinants of food choice: Relationships with obesity and weight control. *Obesity Research*, *9*, 249S–255S
- Mela, D. J., Rogers, P. J., Shepherd, R., & Macfie, H. J. H. (1992). Real people, real foods, real eating situations: Problems and real advantages. *Appetite*, *19*, 69–73.
- Mela, D. J., & Sacchetti, D. A. (1991). Sensory preferences for fats: relationships with diet and body composition. *The American Journal of Clinical Nutrition*, *53*, 908–915.
- Menon, V., Adleman, N. E., White, C. D., Glover, G. H., & Reiss, A. L. (2001). Error-related brain activation during a Go/NoGo response inhibition task. *Human Brain Mapping*, *12*, 131–43.
- Merlo, L. J., Klingman, C., Malasanos, T. H., & Silverstein, J. H. (2009). Exploration of food addiction in pediatric patients: A preliminary investigation. *Journal of Addiction Medicine*, *3*, 26–32. doi:10.1097/ADM.0b013e31819638b0.
- Metcalf, J., & Mischel, W. (1999). A hot/cool system analysis of delay of gratification: Dynamics of willpower. *Psychological Review*, *106*, 3–19.
- Meule, A. (2013). Impulsivity and overeating: A closer look at the subscales of the Barratt Impulsiveness Scale. *Frontiers in Psychology*, *4*, 177. doi:10.3389/fpsyg.2013.00177
- Meule, A. (2014). Are certain foods addictive? *Frontiers in Psychiatry*, *5*, 38. doi:10.3389/fpsyg.2014.00038
- Meule, A., & Kübler, A. (2012). Food cravings in food addiction: The distinct role of positive reinforcement. *Eating Behaviors*, *13*, 252–255. doi:10.1016/j.eatbeh.2012.02.001
- Meule, A., Heckel, D., & Kübler, A. (2012a). Factor structure and item analysis of the Yale Food Addiction Scale in obese candidates for bariatric surgery. *European Eating Disorders Review*, *20*, 419–422. doi:10.1002/erv.2189
- Meule, A., Lukito, S., Vögele, C., & Kübler, A. (2011a). Enhanced behavioral inhibition in restrained eaters. *Eating Behaviors*, *12*, 152–5. doi:10.1016/j.eatbeh.2011.01.006
- Meule, A., Lutz, A. P. C., Krawietz, V., Stützer, J., Vögele, C., & Kübler, A. (2014a). Food-cue affected motor response inhibition and self-reported dieting success: A pictorial affective shifting task. *Frontiers in Psychology*, *5*, 216. doi:10.3389/fpsyg.2014.00216
- Meule, A., Lutz, A. P. C., Vögele, C., & Kübler, A. (2014b). Impulsive reactions to food-cues predict subsequent food craving. *Eating Behaviors*, *15*, 99–105. doi:10.1016/j.eatbeh.2013.10.023
- Meule, A., Lutz, A., Vögele, C., & Kübler, A. (2012b). Food cravings discriminate differentially between successful and unsuccessful dieters and non-dieters. Validation of the Food Cravings Questionnaires in German. *Appetite*, *58*, 88–97. doi:10.1016/j.appet.2011.09.010

- Meule, A., Westenhöfer, J., & Kübler, A. (2011b). Food cravings mediate the relationship between rigid, but not flexible control of eating behavior and dieting success. *Appetite*, *57*, 582–584. doi:10.1016/j.appet.2011.07.013
- Michener, W., & Rozin, P. (1994). Pharmacological versus sensory factors in the satiation of chocolate craving. *Physiology & Behavior*, *56*, 419–422.
- Miller, M. A., & Fillmore, M. T. (2010). The effect of image complexity on attentional bias towards alcohol-related images in adult drinkers. *Addiction*, *105*, 883–890. doi:10.1111/j.1360-0443.2009.02860.x
- Miranda, R., Rohsenow, D. J., Monti, P. M., Tidey, J., & Ray, L. (2008). Effects of repeated days of smoking cue exposure on urge to smoke and physiological reactivity. *Addictive Behaviors*, *33*, 347–353. doi:10.1016/j.addbeh.2007.09.011.
- Mishra, B. R., Nizamie, S. H., Das, B., & Praharaaj, S. K. (2010). Efficacy of repetitive transcranial magnetic stimulation in alcohol dependence: A sham-controlled study. *Addiction*, *105*, 49–55. doi:10.1111/j.1360-0443.2009.02777.x
- Miyake, A., Friedman, N. P., Emerson, M. J., Witzki, A. H., & Howerter, A. (2000). The unity and diversity of executive functions and their contributions to complex “frontal lobe” tasks: A latent variable analysis. *Cognitive Psychology*, *41*, 49–100.
- Mogg, K., Bradley, B. P., Field, M., & De Houwer, J. (2003). Eye movements to smoking-related pictures in smokers: Relationship between attentional biases and implicit and explicit measures of stimulus valence. *Addiction*, *98*, 825–836.
- Mogg, K., Bradley, B. P., O’Neill, B., Bani, M., Merlo-Pich, E., Koch, A., Bullmore, E. T., & Nathan, P. J. (2012). Effect of dopamine D₃ receptor antagonism on approach responses to food cues in overweight and obese individuals. *Behavioural Pharmacology*, *23*, 603–608. doi:10.1097/FBP.0b013e3283566a4a
- Mokdad, A. H., Ford, E. S., Bowman, B. A., Dietz, W. H., Vinicor, F., Bales, V. S., & Marks, J. S. (2003). Prevalence of obesity, diabetes, and obesity-related health risk factors, 2001. *The Journal of the American Medical Association*, *289*, 76–79.
- Molina, B. S. G., & Pelham, W. E. (2003). Childhood predictors of adolescent substance use in a longitudinal study of children with ADHD. *Journal of Abnormal Psychology*, *112*, 497–507.
- Montag, C., Weber, B., Jentgens, E., Elger, C., & Reuter, M. (2010). An epistasis effect of functional variants on the BDNF and DRD2 genes modulates gray matter volume of the anterior cingulate cortex in healthy humans. *Neuropsychologia*, *48*, 1016–1021. doi:10.1016/j.neuropsychologia.2009.11.027
- Montenegro, R. A., Okano, A. H., Cunha, F. A., Gurgel, J. L., Fontes, E. B., & Farinatti, P. T. V. (2012). Prefrontal cortex transcranial direct current stimulation associated with aerobic exercise change aspects of appetite sensation in overweight adults. *Appetite*, *58*, 333–338. doi:10.1016/j.appet.2011.11.008
- Monterosso, J. R., Aron, A. R., Cordova, X., Xu, J., & London, E. D. (2005). Deficits in response inhibition associated with chronic methamphetamine abuse. *Drug and Alcohol Dependence*, *79*, 273–277. doi:10.1016/j.drugalcdep.2005.02.002
- Mostofsky, S. H., & Simmonds, D. J. (2008). Response inhibition and response selection: Two sides of the same coin. *Journal of Cognitive Neuroscience*, *20*, 751–61. doi:10.1162/jocn.2008.20500
- Mostofsky, S. H., Schafer, J. G. B., Abrams, M. T., Goldberg, M. C., Flower, A. A., Boyce, A., ... Pekar, J. J. (2003). fMRI evidence that the neural basis of response inhibition is task-dependent. *Brain Research. Cognitive Brain Research*, *17*, 419–30.

- Mumford, G. K., Evans, S. M., Kaminski, B. J., Preston, K. L., Sannerud, C. A., Silverman, K., & Griffiths, R. R. (1994). Discriminative stimulus and subjective effects of theobromine and caffeine in humans, *Psychopharmacology*, *115*, 1–8.
- Muraven, M. (2010). Building self-control strength: Practicing self-control leads to improved self-control performance. *Journal of Experimental Social Psychology*, *46*, 465–468.
- Muraven, M., & Baumeister, R. F. (2000). Self-regulation and depletion of limited resources: Does self-control resemble a muscle? *Psychological Bulletin*, *126*, 247–259.
- Muraven, M., Baumeister, R. F., & Tice, D. M. (1999). Longitudinal improvement of self-regulation through practice: Building self-control strength through repeated exercise. *The Journal of Social Psychology*, *139*, 446–457.
- Muraven, M., Tice, D.M., & Baumeister, R.F. (1998). Self-control as a limited resource: Regulatory depletion patterns. *Journal of Personality and Social Psychology*, *74*, 774–789.
- Murphy, C. M., Stojek, M. K., & Mackillop, J. (2014). Interrelationships among impulsive personality traits, food addiction, and body mass index. *Appetite*, *73*, 45–50. doi:10.1016/j.appet.2013.10.008
- Murphy, P., & Garavan, H. (2011). Cognitive predictors of problem drinking and AUDIT scores among college students. *Drug and Alcohol Dependence*, *115*, 94–100. doi:10.1016/j.drugalcdep.2010.10.011
- Musch, J., & Reips, U-D. (2000). A Brief History of Web Experimenting, In M. H. Birnbaum (Ed.), *Psychological Experiments on the Internet* (pp. 61-88). SanDiego, CA: Academic Press.
- Mussell, M. P., Mitchell, J. E., deZwaan, M., Crosby, R. D., Seim, H. C., & Crow, S. J. (1996). Clinical characteristics associated with binge eating in obese females: A descriptive study. *International Journal of Obesity*, *20*, 324–331.
- Nakata, H., Inui, K., Wasaka, T., Tamura, Y., Akatsuka, K., Kida, T., & Kakigi, R. (2006). Higher anticipated force required a stronger inhibitory process in go/nogo tasks. *Clinical Neurophysiology*, *117*, 1669–76. doi:10.1016/j.clinph.2006.03.032
- Nardone, R., Bergmann, J., Christova, M., Lochner, P., Tezzon, F., Golaszewski, S., Trinkka, E., & Brigo, F. (2012). Non-invasive brain stimulation in the functional evaluation of alcohol effects and in the treatment of alcohol craving: A review. *Neuroscience Research*, *74*, 169–176. doi:10.1016/j.neures.2012.08.003
- Nasser, J. A., Gluck, M. E., & Geliebter, A. (2004). Impulsivity and test meal intake in obese binge eating women. *Appetite*, *43*, 303–307. doi:10.1016/j.appet.2004.04.006
- Nederkoorn, C., Braet, C., Van Eijs, Y., Tanghe, A., & Jansen, A. (2006a). Why obese children cannot resist food: The role of impulsivity. *Eating behaviors*, *7*, 315–322. doi:10.1016/j.eatbeh.2005.11.005
- Nederkoorn, C., Coelho, J. S., Guerrieri, R., Houben, J., & Jansen, A. (2012). Specificity of the failure to inhibit responses in overweight children. *Appetite*, *59*, 409–413.
- Nederkoorn, C., Guerrieri, R., Havermans, R. C., Roefs, A., & Jansen, A. (2009a). The interactive effect of hunger and impulsivity on food intake and purchase in a virtual supermarket. *International Journal of Obesity*, *33*, 905–912.
- Nederkoorn, C., Houben, K., Hofmann, W., Roefs, A., & Jansen, A. (2010). Control yourself or just eat what you like? Weight gain over a year is predicted by an interactive effect of response inhibition and implicit preference for snack foods. *Health Psychology*, *29*, 389–393. doi:10.1037/a0019921
- Nederkoorn, C., Jansen, E., Mulkens, S., & Jansen, A. (2006b). Impulsivity predicts treatment outcome in obese children. *Behaviour Research and Therapy*, *45*, 1071–1075. doi:10.1016/j.brat.2006.05.009

- Nederkoorn, C., Smulders, F. T. Y., Havermans, R. C., Roefs, A., & Jansen, A. (2006c). Impulsivity in obese women. *Appetite*, *47*, 253–256. doi:10.1016/j.appet.2006.05.008
- Nederkoorn, C., Smulders, F. T., & Jansen, A. (2000). Cephalic phase responses, craving and food intake in normal subjects. *Appetite*, *35*, 45–55. doi:10.1006/appe.2000.0328
- Nederkoorn, C., Van Eijs, Y., & Jansen, A. (2004). Restrained eaters act on impulse. *Personality and Individual Differences*, *37*, 1651–1658.
- Neumark-Sztainer, D., Falkner, N., Story, M., Perry, C., Hannan, P. J., & Mulert, S. (2002). Weight-teasing among adolescents: correlations with weight status and disordered eating behaviors. *International Journal of Obesity and Related Metabolic Disorders*, *26*, 123–131. doi:10.1038/sj.ijo.0801853
- Ng, L., & Davis, C. (2013). Cravings and food consumption in Binge Eating Disorder. *Eating Behaviors*, *14*, 472–475. doi:10.1016/j.eatbeh.2013.08.011
- Nichols, A. L., & Maner, J. K. (2008). The good-subject effect: Investigating participant demand characteristics. *The Journal of General Psychology*, *135*, 151–65. doi:10.3200/GENP.135.2.151-166
- Nicholls, W., & Hulbert-Williams, L. (2013). British English translation of the Food Craving Inventory (FCI-UK). *Appetite*, *67*, 37–43. doi:10.1016/j.appet.2013.03.010
- Nicholls, M. E. R., Loveless, K. M., Thomas, N. A., Loetscher, T., & Churches, O. (2014). Some participants may be better than others: Sustained attention and motivation are higher early in semester. *The Quarterly Journal of Experimental Psychology*, 1–9. doi:10.1080/17470218.2014.925481
- Niedhammer, I., Bugel, I., Bonenfant, S., Goldberg, M., & Leclerc, A. (2000). Validity of self-reported weight and height in the French GAZEL cohort. *International Journal of Obesity and Related Metabolic Disorders*, *24*, 1111–8.
- Nielsen, S. J., & Popkin, B. M. (2003). Patterns and trends in food portion sizes, 1977–1998. *JAMA : The Journal of the American Medical Association*, *289*, 450–453.
- Nielsen, S. J., & Popkin, B. M. (2004). Changes in beverage intake between 1977 and 2001. *American Journal of Preventive Medicine*, *27*, 205–210. doi:10.1016/j.amepre.2004.05.005
- Nigg, J. T., Wong, M. M., Martel, M. M., Jester, J. M., Puttler, L. I., Glass, J. M., Adams, K. M., & Fitzgerald, H. E., & Zucker, R. A. (2006). Poor response inhibition as a predictor of problem drinking and illicit drug use in adolescents at risk for alcoholism and other substance use disorders. *Journal of the American Academy of Child and Adolescent Psychiatry*, *45*, 468–475. doi:10.1097/01.chi.0000199028.76452.a9
- Nijs, I. M. T., Franken, I. H. A., & Muris, P. (2007). The modified Trait and State Food-Cravings Questionnaires: Development and validation of a general index of food craving. *Appetite*, *49*, 38–46. doi:10.1016/j.appet.2006.11.001
- 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*, 243–254. doi:10.1016/j.appet.2009.11.004
- Nitsche, M. A., & Paulus, W. (2000). Excitability changes induced in the human motor cortex by weak transcranial direct current stimulation. *The Journal of Physiology*, *527*, 633–9.
- Nitsche, M. A., Fricke, K., Henschke, U., Schlitterlau, A., Liebetanz, D., Lang, N., ... Paulus, W. (2003b). Pharmacological modulation of cortical excitability shifts induced by transcranial direct current stimulation in humans. *The Journal of Physiology*, *553*, 293–301. doi:10.1113/jphysiol.2003.049916
- Nitsche, M. A., Schauenburg, A., Lang, N., Liebetanz, D., Exner, C., Paulus, W., & Tergau, F. (2003a). Facilitation of implicit motor learning by weak transcranial direct current

- stimulation of the primary motor cortex in the human. *Journal of Cognitive Neuroscience*, *15*, 619–26. doi:10.1162/089892903321662994
- Nitsche, M. A., Seeber, A., Frommann, K., Klein, C. C., Rochford, C., Nitsche, M. S., ... Tergau, F. (2005). Modulating parameters of excitability during and after transcranial direct current stimulation of the human motor cortex. *The Journal of Physiology*, *568*, 291–303. doi:10.1113/jphysiol.2005.092429
- Nitsche, M. A., & Paulus, W. (2001). Sustained excitability elevations induced by transcranial DC motor cortex stimulation in humans. *Neurology*, *57*, 1899–1901.
- Nitsche, M. A., Cohen, L. G., Wassermann, E. M., Priori, A., Lang, N., Antal, A., Paulus, W., Hummel, F., Boggio, P. S., Fregni, F., & Pascual-Leone, A. (2008). Transcranial direct current stimulation: State of the art 2008. *Brain Stimulation*, *1*, 206–223. doi:10.1016/j.brs.2008.06.004
- Noble, E. P. (2000). Addiction and its reward process through polymorphisms of the D2 dopamine receptor gene: a review. *European Psychiatry*, *15*, 79–89.
- Noël, X., Bechara, A., Dan, B., Hanak, C., & Verbanck, P. (2007). Response inhibition deficit is involved in poor decision making under risk in nonamnesic individuals with alcoholism. *Neuropsychology*, *21*, 778–786. doi:10.1037/0894-4105.21.6.778
- Noël, X., Van der Linden, M., Brevers, D., Campanella, S., Verbanck, P., Hanak, C., Kornreich, C., & Verbruggen, F. (2013). Separating intentional inhibition of prepotent responses and resistance to proactive interference in alcohol-dependent individuals. *Drug and Alcohol Dependence*, *128*, 200–205. doi:10.1016/j.drugalcdep.2012.08.021
- Nyholm, M., Gullberg, B., Merlo, J., Lundqvist-Persson, C., Råstam, L., & Lindblad, U. (2007). The validity of obesity based on self-reported weight and height: Implications for population studies. *Obesity*, *15*, 197–208. doi:10.1038/oby.2007.536
- Oaten, M., & Cheng, K. (2006a). Longitudinal gains in self-regulation from regular physical exercise. *British Journal of Health Psychology*, *11*, 717–733.
- Oaten, M., & Cheng, K. (2006b). Improved self-control: The benefits of a regular program of academic study. *Basic and Applied Social Psychology*, *28*, 1–16.
- Oaten, M., & Cheng, K. (2007). Improvements in self-control from financial monitoring. *Journal of Economic Psychology*, *28*, 487–501.
- Ochner, C. N., Kwok, Y., Conceicao, E., Pantazatos, S. P., Puma, L. M., Carnell, S., ... Geliebter, A. (2011). Selective reduction in neural responses to high calorie foods following gastric bypass surgery. *Annals of Surgery*, *253*, 502–507.
- Ogden, C. L., Carroll, M. D., Curtin, L. R., McDowell, M. A., Tabak, C. J., & Flegal, K. M. (2006). Prevalence of overweight and obesity in the United States, 1999–2004. *Journal of the American Medical Association*, *295*, 1549–1555.
- Oosterlaan, J., Logan, G. D., & Sergeant, J. A. (1998). Response inhibition in AD/HD, CD, comorbid AD/HD + CD, anxious, and control children: A meta-analysis of studies with the stop task. *Journal of Child Psychology and Psychiatry, and Allied Disciplines*, *39*, 411–25.
- Orford, J. (2001). Addiction as excessive appetite. *Addiction*, *96*, 15–31. doi:10.1080/09652140020016932
- Owen, A. M., Hampshire, A., Grahn, J. A., Stenton, R., Dajani, S., Burns, A. S., ... Ballard, C. G. (2010). Putting brain training to the test. *Nature*, *465*, 775–8. doi:10.1038/nature09042
- Paliwal, P., Hyman, S. M., & Sinha, R. (2008). Craving predicts time to cocaine relapse: Further validation of the Now and Brief versions of the cocaine craving questionnaire. *Drug and Alcohol Dependence*, *93*, 252–259. doi:10.1016/j.drugalcdep.2007.10.002

- Pannacciulli, N., Del Parigi, A., Chen, K., Le, D. S. N. T., Reiman, E. M., & Tataranni, P. A. (2006). Brain abnormalities in human obesity: A voxel-based morphometric study. *NeuroImage*, *31*, 1419–1425. doi:10.1016/j.neuroimage.2006.01.047
- Papachristou, H., Nederkoorn, C., Havermans, R., van der Horst, M., & Jansen, A. (2012). Can't stop the craving: The effect of impulsivity on cue-elicited craving for alcohol in heavy and light social drinkers. *Psychopharmacology*, *219*, 511–518. doi:10.1007/s00213-011-2240-5
- Papies, E. K., & Hamstra, P. (2010). Goal priming and eating behavior: Enhancing self-regulation by environmental cues. *Health Psychology*, *29*, 384–388.
- Papies, 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*, 1283–1292.
- Papies, E. K., Stroebe, W., & Aarts, H. (2009). Who likes it more? Restrained eaters' implicit attitudes towards food. *Appetite*, *53*, 279–287.
- Parvaz, M. A., Alia-Klein, N., Woicik, P. A., Volkow, N. D., & Goldstein, R. Z. (2011). Neuroimaging for drug addiction and related behaviors. *Reviews in the Neurosciences*, *22*, 609–624. doi:10.1515/RNS.2011.055.
- Pascual-Leone, A., Houser, C. M., Reese, K., Shotland, L. I., Grafman, J., Sato, S., Valls-Solé, J., Brasil-Neto, J. P., Wasserman, E. M., & Cohen, L. G. (1993). Safety of rapid-rate transcranial magnetic stimulation in normal volunteers. *Electroencephalography and Clinical Neurophysiology*, *89*, 120–130.
- Pauli-Pott, U., Albayrak, Ö., Hebebrand, J., & Pott, W. (2010). Association between inhibitory control capacity and body weight in overweight and obese children and adolescents: Dependence on age and inhibitory control component. *Child Neuropsychology*, *37*–41.
- Pearce, J. M., & Hall, G. (1980). A model for Pavlovian learning: Variations in the effectiveness of conditioned but not of unconditioned stimuli. *Psychological Review*, *87*, 532–52.
- Pearce, M. J., Boergers, J., & Prinstein, M. J. (2002). Adolescent obesity, overt and relational peer victimization, and romantic relationships. *Obesity Research*, *10*, 386–393. doi:10.1038/oby.2002.53
- Pelchat, M. L. (2009). Food addiction in humans. *The Journal of Nutrition*, *139*, 620–622. doi:10.3945/jn.108.097816.1
- Peoples, L. L. (2002). Will, anterior cingulate cortex, and addiction. *Science*, *296*, 1623–1624. doi:10.1126/science.1072997
- Pessoa, L., Padmala, S., Kenzer, A., & Bauer, A. (2012). Interactions between cognition and emotion during response inhibition. *Emotion*, *12*, 192–197. doi:10.1037/a0024109.
- Phaf, R. H., & Rotteveel, M. (2012). Affective monitoring: a generic mechanism for affect elicitation. *Frontiers in Psychology*, *3*, 47. doi:10.3389/fpsyg.2012.00047
- Pierce, R. C., & Kumaresan, V. (2006). The mesolimbic dopamine system: The final common pathway for the reinforcing effect of drugs of abuse? *Neuroscience and Biobehavioral Reviews*, *30*, 215–238. doi:10.1016/j.neubiorev.2005.04.016
- Pietiläinen, K. H., Saarni, S. E., Kaprio, J., & Rissanen, A. (2012). Does dieting make you fat? A twin study. *International Journal of Obesity*, *36*, 456–464. doi:10.1038/ijo.2011.160
- Pijl, H., Ohashi, S., Matsuda, M., Miyazaki, Y., Mahankali, A., Kumar, V., Pipek, R., Iozzo, P., Lancaster, J. L., Cincotta, A. H., & DeFronzo, R. A. (2000). Bromocriptine: A novel approach to the treatment of type 2 diabetes. *Diabetes Care*, *23*, 1154–1161.

- Politi, E., Fauci, E., Santoro, A., & Smeraldi, E. (2008). Daily sessions of transcranial magnetic stimulation to the left prefrontal cortex gradually reduce cocaine craving. *The American Journal on Addictions, 17*, 345–346. doi:10.1080/10550490802139283
- Polivy, J., & Herman, C. P. (1985). Dieting and bingeing a causal analysis. *American Psychologist, 40*, 193–201.
- Polivy, J., & Herman, P. (1999). Distress and eating: Why do dieters overeat? *International Journal of Eating Disorders, 26*, 153–164. doi: 10.1002/(SICI)1098-108X
- Polivy, J., Coleman, J., & Herman, C. P. (2005). The effect of deprivation on food cravings and eating behavior in restrained and unrestrained eaters. *The International Journal of Eating Disorders, 38*, 301–9. doi:10.1002/eat.20195
- Poreisz, C., Boros, K., Antal, A., & Paulus, W. (2007). Safety aspects of transcranial direct current stimulation concerning healthy subjects and patients. *Brain Research Bulletin, 72*, 208–214. doi:10.1016/j.brainresbull.2007.01.004
- Pretlow, R. A. (2011). Addiction to highly pleasurable food as a cause of the childhood obesity epidemic: A qualitative Internet study. *Eating Disorders, 19*, 295–307. doi:10.1080/10640266.2011.584803
- Price, T. F., Peterson, C. K., & Harmon-Jones, E. (2012). The emotive neuroscience of embodiment. *Motivation and Emotion, 36*, 27–37. doi:10.1007/s11031-011-9258-1
- Priori, A. (2003). Brain polarization in humans: a reappraisal of an old tool for prolonged non-invasive modulation of brain excitability. *Clinical Neurophysiology, 114*, 589–595. doi:10.1016/S1388-2457(02)00437-6
- Provencher, V., Drapeau, V., Tremblay, A., Després, J. –P., & Lemieux, S. (2003). Eating behaviors and indexes of body composition in men and women from the Québec family study. *Obesity Research, 11*, 783-792.
- Puhl, R. M., & Heuer, C. A. (2009). The stigma of obesity: A review and update. *Obesity, 17*, 941–964. doi:10.1038/oby.2008.636
- Puhl, R. M., & Latner, J. D. (2007). Stigma, obesity, and the health of the nation's children. *Psychological Bulletin, 133*, 557–580. doi:10.1037/0033-2909.133.4.557
- Puhl, R., & Brownell, K. D. (2001). Bias, discrimination, and obesity. *Obesity Research, 9*, 788–805. doi:10.1038/oby.2001.108
- Purpura, D. P., & McMurtry, J. G. (1965). Intracellular activities and evoked potential changes during of motor cortex. *Journal of Neurophysiology, 28*, 166–185.
- Quinn, P. D., & Fromme, K. (2010). Self-regulation as a protective factor against risky drinking and sexual behavior. *Psychology of Addictive Behaviours, 24*, 376–85.
- Rada, P., Avena, N. M., & Hoebel, B. G. (2005). Daily bingeing on sugar repeatedly releases dopamine in the accumbens shell. *Neuroscience, 134*, 737–744. doi:10.1016/j.neuroscience.2005.04.043
- Radhakishun, F. S., van Ree, J. M., & Westerink, B. H. (1988). Scheduled eating increases dopamine release in the nucleus accumbens of food-deprived rats as assessed with on-line brain dialysis. *Neuroscience Letters, 85*, 351–356.
- Rand, C. S. W. & Kuldau, J. M. (1991) Restrained eating (weight concerns) in the general population and among students. *International Journal of Eating Disorders, 10*, 699-708.
- Raynor, H. A., & Epstein, L. H. (2001). Dietary variety, energy regulation, and obesity. *Psychological Bulletin, 127*, 325-341.
- Reimann, M., Feye, W., Malter, A. J., Ackerman, J. M., Castaño, R., Garg, N., ... Zhong, C.-B. (2012). Embodiment in judgment and choice. *Journal of Neuroscience, Psychology, and Economics, 5*, 104–123. doi:10.1037/a0026855

- Reinholz, J., Skopp, O., Breitenstein, C., Bohr, I., Winterhoff, H., & Knecht, S. (2008). Compensatory weight gain due to dopaminergic hypofunction: New evidence and own incidental observations. *Nutrition & Metabolism*, *5*, 35. doi:10.1186/1743-7075-5-35
- Reips, U.-D. (2002). Standards for Internet-based experimenting. *Experimental Psychology*, *49*, 243–256. doi:10.1027//1618-3169.49.4.243
- Reynolds, B., Patak, M., Shroff, P., Penfold, R., Melanko, S., & Duhig, A. (2007). Laboratory and self-report assessments of impulsive behavior in adolescent daily smokers and nonsmokers. *Experimental and Clinical Psychopharmacology*, *15*, 264–271.
- Ridderinkhof, K. R., van den Wildenberg, W. P. M., Segalowitz, S. J., & Carter, C. S. (2004). Neurocognitive mechanisms of cognitive control: The role of prefrontal cortex in action selection, response inhibition, performance monitoring, and reward-based learning. *Brain and Cognition*, *56*, 129–40. doi:10.1016/j.bandc.2004.09.016
- Riggs, N. R., Spruijt-Metz, D., Chou, C.-P., & Pentz, M. A. (2012). Relationships between executive cognitive function and lifetime substance use and obesity-related behaviors in fourth grade youth. *Child Neuropsychology*, *18*, 1-11.
- 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. doi: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. Brain Research Reviews*, *18*, 247–291. Retrieved from <http://www.ncbi.nlm.nih.gov/pubmed/8401595>
- Robinson, T. E., & Berridge, K. C. (2001). Incentive-sensitization and addiction. *Addiction*, *96*, 103–114. doi:10.1080/09652140020016996
- Robinson, T. E., & Berridge, K. C. (2003). Addiction. *Annual Review of Psychology*, *54*, 25-53.
- Roefs, A., Herman, C. P., Macleod, C. M., Smulders, F. T. Y., & Jansen, A. (2005). At first sight: How do restrained eaters evaluate high-fat palatable foods? *Appetite*, *44*, 103–114. doi:10.1016/j.appet.2004.08.001
- Rogers, P. J., & Hill, J. (1989). Breakdown of dietary restraint following mere exposure to food stimuli: Interrelationships between restraint, hunger, salivation, and food intake. *Addictive Behaviors*, *14*, 387–397.
- Rogers, P. J., & Smit, H. J. (2000). Food craving and food “addiction”: A critical review of the evidence from a biopsychosocial perspective. *Pharmacology, Biochemistry, and Behavior*, *66*, 3–14.
- Romero-Corral, A., Somers, V. K., Sierra-Johnson, J., Korenfeld, Y., Boarin, S., Korinek, J., ... Lopez-Jimenez, F. (2010). Normal weight obesity: A risk factor for cardiometabolic dysregulation and cardiovascular mortality. *European Heart Journal*, *31*, 737–46. doi:10.1093/eurheartj/ehp487
- Rossow, I., Kjaernes, U., & Holst, D. (1990). Patterns of sugar consumption in early childhood. *Community Dentistry and Oral Epidemiology*, *18*, 12-16.
- Rosval, L., Steiger, H., Bruce, K., Israël, M., Richardson, J., & Aubut, M. (2006). Impulsivity in women with eating disorders: Problem of response inhibition, planning, or attention? *International Journal of Eating Disorders*, *39*, 590-593. doi:10.1002/eat
- Rotenberg, K. J., & Flood, D. (2000). Dietary restraint, attributional styles for eating, and preloading effects. *Eating Behaviors*, *1*, 63-78.
- Rotenberg, K. J., Lancaster, C., Marsden, J., Pryce, S., Williams, J., & Lattimore, P. (2005). Effects of priming thoughts about control on anxiety and food intake as moderated by dietary restraint. *Appetite*, *44*, 235–241. doi:10.1016/j.appet.2004.09.001

- Roth, D. A., Herman, C. P., Polivy, J., & Pliner, P. (2001). Self-presentational conflict in social eating situations: a normative perspective. *Appetite*, *36*, 165–71. doi:10.1006/appe.2000.0388
- Rothmund, Y., Preuschhof, C., Bohner, G., Bauknecht, H.-C., Klingebiel, R., Flor, H., & Klapp, B. F. (2007). Differential activation of the dorsal striatum by high-calorie visual food stimuli in obese individuals. *NeuroImage*, *37*, 410–421. doi:10.1016/j.neuroimage.2007.05.008
- Rothman, K. J. (2008). BMI-related errors in the measurement of obesity. *International Journal of Obesity*, *32 Suppl 3*, S56–9. doi:10.1038/ijo.2008.87
- Rozin, P., Levine, E., & Stoess, C. (1991). Chocolate craving and liking. *Appetite*, *17*, 199–212.
- Rubia, K., Russell, T., Overmeyer, S., Brammer, M. J., Bullmore, E. T., Sharma, T., ... Taylor, E. (2001). Mapping motor inhibition: conjunctive brain activations across different versions of go/no-go and stop tasks. *NeuroImage*, *13*, 250–61. doi:10.1006/nimg.2000.0685
- Rubia, K., Smith, A. B., Brammer, M. J., & Taylor, E. (2003). Right inferior prefrontal cortex mediates response inhibition while mesial prefrontal cortex is responsible for error detection. *Neuroimage*, *20*, 351–358.
- Ruderman, A. J. (1983). The restraint scale: A psychometric investigation. *Behaviour Research and Therapy*, *21*, 253–258.
- Ruderman, A. J. (1986). Dietary restraint: A theoretical and empirical review. *Psychological Bulletin*, *99*, 247–262.
- Ruderman, A. J. (1985). Restraint, obesity and bulimia. *Behaviour Research and Therapy*, *2*, 151–156.
- Russell-Mayhew, S., von Ranson, K. M., & Masson, P. C. (2010). How does overeaters anonymous help its members? A qualitative analysis. *European Eating Disorders Review*, *18*, 33–42. doi:10.1002/erv.966
- Rydén, A., Sullivan, M., Torgerson, J. S., Karlsson, J., Lindroos, A.-K., & Taft, C. (2003). Severe obesity and personality: A comparative controlled study of personality traits. *International Journal of Obesity*, *27*, 1534–1540. doi:10.1038/sj.ijo.0802460
- Salo, R., Nordahl, T. E., Possin, K., Leamon, M., Gibson, D. R., Galloway, G. P., ... Sullivan, E. V. (2002). Preliminary evidence of reduced cognitive inhibition in methamphetamine-dependent individuals. *Psychiatry Research*, *111*, 65–74.
- Schachar, R., Logan, G. D., Robaey, P., Chen, S., Ickowicz, A., & Barr, C. (2007). Restraint and cancellation: multiple inhibition deficits in attention deficit hyperactivity disorder. *Journal of Abnormal Child Psychology*, *35*, 229–38. doi:10.1007/s10802-006-9075-2
- Schachter, S. (1971). Some extraordinary facts about obese humans and rats. *American Psychologist*, *26*, 129–144.
- Schag, K., Teufel, M., Junne, F., Preissl, H., Hautzinger, M., Zipfel, S., & Giel, K. E. (2013). Impulsivity in binge eating disorder: food cues elicit increased reward responses and disinhibition. *PloS One*, *8*, e76542. doi:10.1371/journal.pone.0076542
- Schoenmakers, T., Wiers, R. W., Jones, B. T., Bruce, G., & Jansen, A. T. M. (2007). Attentional retraining decreases attentional bias in heavy drinkers without generalization. *Addiction*, *102*, 399–405.
- 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*, 30–36.

- Schonberg, T., Bakkour, A., Hover, A. M., Mumford, J. A., Nagar, L., Perez, J., & Poldrack, R. A. (2014). Changing value through cued approach: An automatic mechanism of behavior change. *Nature Neuroscience*, *17*, 625–30. doi:10.1038/nn.3673
- Schur, E. A., Heckbert, S. R., & Goldberg, J. H. (2010). The association of restrained eating with weight change over time in a community-based sample of twins. *Obesity*, *18*, 1146-1152.
- Schwimmer, J. B., Burwinkle, T. M., & Varni, J. W. (2003). Health-related quality of life of severely obese children and adolescents. *The Journal of the American Medical Association*, *289*, 1813–1819. doi:10.1001/jama.289.14.1813
- Scislowski, P. W., Tozzo, E., Zhang, Y., Phaneuf, S., Prevelige, R., & Cincotta, A. H. (1999). Biochemical mechanisms responsible for the attenuation of diabetic and obese conditions in ob/ob mice treated with dopaminergic agonists. *International Journal of Obesity and Related Metabolic Disorders*, *23*, 425–431.
- Scott, W. C., Kaiser, D., Othmer, S., & Sideroff, S. I. (2005). Effects of an EEG biofeedback protocol on a mixed substance abusing population. *The American Journal of Drug and Alcohol Abuse*, *31*, 455–469. doi:10.1081/ADA-200056807
- Sharp, D. J., Bonnelle, V., De Boissezon, X., Beckmann, C. F., James, S. G., Patel, M. C., & Mehta, M. A. (2010). Distinct frontal systems for response inhibition, attentional capture, and error processing. *Proceedings of the National Academy of Sciences of the United States of America*, *107*, 6106–11. doi:10.1073/pnas.1000175107
- Shea, J. L., King, M. T. C., Yi, Y., Gulliver, W., & Sun, G. (2012). Body fat percentage is associated with cardiometabolic dysregulation in BMI-defined normal weight subjects. *Nutrition, Metabolism, and Cardiovascular Diseases*, *22*, 741–7. doi:10.1016/j.numecd.2010.11.009
- Shiffrin, R. M., & Schneider, W. (1977). Controlled and automatic human information processing: II. Perceptual learning, automatic attending, and a general theory. *Psychological Review*, *84*, 127-190.
- Shimizu, M., & Wansink, B. (2011). Watching food-related television increases caloric intake in restrained eaters. *Appetite*, *57*, 661–664.
- Simmonds, D. J., Pekar, J. J., & Mostofsky, S. H. (2008). Meta-analysis of Go/No-go tasks demonstrating that fMRI activation associated with response inhibition is task-dependent. *Neuropsychologia*, *46*, 224–32. doi:10.1016/j.neuropsychologia.2007.07.015
- Sinha, R., Fuse, T., Aubin, L.-R., & O'Malley, S. S. (2000). Psychological stress, drug-related cues and cocaine craving. *Psychopharmacology*, *152*, 140–148. doi:10.1007/s002130000499
- Sinopoli, K. J., Schachar, R., & Dennis, M. (2011). Reward improves cancellation and restraint inhibition across childhood and adolescence. *Developmental Psychology*, *47*, 1479–89. doi:10.1037/a0024440
- Slater, R., Cornelissen, L., Fabrizi, L., Patten, D., Yoxen, J., Worley, A., Boyd, S., Meek, J., & Fitzgerald, M. (2010). Oral sucrose as an analgesic drug for procedural pain in newborn infants: a randomised controlled trial. *Lancet*, *376*, 1225–1232. doi:10.1016/S0140-6736(10)61303-7
- Small, D. M., Jones-Gotman, M., & Dagher, A. (2003). Feeding-induced dopamine release in dorsal striatum correlates with meal pleasantness ratings in healthy human volunteers. *NeuroImage*, *19*, 1709–1715. doi:10.1016/S1053-8119(03)00253-2
- Smalley, K. J., Knerr, A. N., Kendrick, V., Colliver, J. A., & Owen, O. E. (1990). Reassessment of body mass indices. *American Journal of Clinical Nutrition*, *52*, 405–408.

- Smeets, E., Roefs, A., & Jansen, A. (2009). Experimentally induced chocolate craving leads to an attentional bias in increased distraction but not in speeded detection. *Appetite*, *53*, 370–375. doi:10.1016/j.appet.2009.07.020
- Smith, D. G., & Robbins, T. W. (2013). The neurobiological underpinnings of obesity and binge eating: a rationale for adopting the food addiction model. *Biological Psychiatry*, *73*, 804–810. doi:10.1016/j.biopsych.2012.08.026
- Smith, E. C., & DeCoster, J. (2000). Dual-process models in social and cognitive psychology: Conceptual integration and links to underlying memory systems. *Personality and Social Psychology Review*, *4*, 108–131.
- Snoek, H. M., van Strien, T., Janssens, J. M. A. M., & Engels, R. C. M. E. (2008). Restrained eating and BMI: a longitudinal study among adolescents. *Health Psychology*, *27*, 753–9. doi:10.1037/0278-6133.27.6.753
- Sobik, L., Hutchison, K., & Craighead, L. (2005). Cue-elicited craving for food: A fresh approach to the study of binge eating. *Appetite*, *44*, 253–261. doi:10.1016/j.appet.2004.12.001
- Spencer, E. A., Appleby, P. N., Davey, G. K., & Key, T. J. (2002). Validity of self-reported height and weight in 4808 EPIC-Oxford participants. *Public Health Nutrition*, *5*, 561–5. doi:10.1079/PHN2001322
- Spencer, J. A., & Fremouw, W. J. (1979). Binge eating as a function of restraint and weight classification. *Journal of Abnormal Psychology*, *88*, 262–267.
- Spierer, L., Chavan, C. F., & Manuel, A. L. (2013). Training-induced behavioral and brain plasticity in inhibitory control. *Frontiers in Human Neuroscience*, *7*, 427. doi:10.3389/fnhum.2013.00427
- Spillane, N. S., Smith, G. T., & Kahler, C. W. (2010). Impulsivity-like traits and smoking behavior in college students. *Addictive Behaviors*, *35*, 700–705. doi:10.1016/j.addbeh.2010.03.008
- Spinella, M. (2002). Correlations between orbitofrontal dysfunction and tobacco smoking. *Addiction Biology*, *7*, 381–384. doi:10.1080/1355621021000005964
- Spitz, M. R., Detry, M. A., Pillow, P., Hu, Y., Amos, C. I., Hong, W. K., & Wu, X. (2000). Variant alleles of the D2 dopamine receptor gene and obesity. *Nutrition Research*, *20*, 371–380.
- Spring, B., Schneider, K., Smith, M., Kendzor, D., Hedeker, D., & Pagoto, S. (2010). Abuse potential of carbohydrates for overweight carbohydrate cravers. *Psychopharmacology*, *197*, 637–647. doi:10.1007/s00213-008-1085-z.
- Spunt, R. P., Lieberman, M. D., Cohen, J. R., & Eisenberger, N. I. (2012). The phenomenology of error processing: the dorsal ACC response to stop-signal errors tracks reports of negative affect. *Journal of Cognitive Neuroscience*, *24*, 1753–65. doi:10.1162/jocn_a_00242
- Steel, P. (2007). The nature of procrastination: A meta-analytic and theoretical review of quintessential self-regulatory failure. *Psychological Bulletin*, *133*, 65–94.
- Steele, K. E., Prokopowicz, G. P., Schweitzer, M. A., Magunson, T. H., Lidor, A. O., Kuwabawa, H., Kuma, A., Brasic, J., & Wong, D. F. (2010). Alterations of central dopamine receptors before and after gastric bypass surgery. *Obesity Surgery*, *20*, 369–374. doi:10.1007/s11695-009-0015-4
- Steffens, M. C. (2004). Is the Implicit Association Test immune to faking? *Experimental Psychology*, *51*, 165–179. doi:10.1027/1618-3169.51.3.165
- Stevens, T., Brevers, D., Chambers, C. D., Lavric, A., McLaren, I. P. L., Mertens, M., Noël, X., & Verbruggen, F. (under review). How does response inhibition influence decision-making when gambling?

- Stewart, S. H., & Samoluk, S. B. (1997). Effects of short-term food deprivation and chronic dietary restraint on the selective processing of appetitive-related cues. *International Journal of Eating Disorders, 21*, 129–135.
- Stewart, S. H., Angelopoulos, M., Baker, J. M., & Boland, F. J. (2000). Relations between dietary restraint and patterns of alcohol use in young adult women. *Psychology of Addictive Behaviors, 14*, 77–82.
- Stewart-Williams, S., & Podd, J. (2004). The placebo effect: Dissolving the expectancy versus conditioning debate. *Psychological Bulletin, 130*, 324–40. doi:10.1037/0033-2909.130.2.324
- Stice, E., Figlewicz, D. P., Gosnell, B. A., Levine, A. S., & Pratt, W. E. (2013). The contribution of brain reward circuits to the obesity epidemic. *Neuroscience and Biobehavioral Reviews, 37*, 2047–2058. doi:10.1016/j.neubiorev.2012.12.001
- Stice, E., Ozer, S., & Kees, M. (1997). Relation of dietary restraint to bulimic symptomatology: The effects of the criterion confounding of the Restraint Scale. *Behavior Research and Therapy, 35*, 145–152.
- Stice, E., Spoor, S., Bohon, C., & Small, D. M. (2008). Relation between obesity and blunted striatal response to food is moderated by TaqIA A1 allele. *Science, 322*, 449–452. doi:10.1126/science.1161550
- Stice, E., Spoor, S., Bohon, C., Veldhuizen, M. G., & Small, D. M. (2008). Relation of reward from food intake and anticipated food intake to obesity: A functional magnetic resonance imaging study. *Journal of Abnormal Psychology, 117*, 924–935. doi:10.1037/a0013600
- Stoeckel, L. E., Weller, R. E., Cook, E. W., Twieg, D. B., Knowlton, R. C., & Cox, J. E. (2008). Widespread reward-system activation in obese women in response to pictures of high-calorie foods. *NeuroImage, 41*, 636–647. doi:10.1016/j.neuroimage.2008.02.031
- Strack, F., & Deutsch, R. (2004). Reflective and impulsive determinants of social behavior. *Personality and Social Psychology Review, 8*, 220–247.
- Strafella, A. P., Paus, T., Barrett, J., & Dagher, A. (2001). Repetitive transcranial magnetic stimulation of the human prefrontal cortex induces dopamine release in the caudate nucleus. *The Journal of Neuroscience, 21*, RC157.
- Streeter, C. C., Terhune, D. B., Whitfield, T. H., Gruber, S., Sarid-Segal, O., Silveri, M. M., Tzilos, G., Afshar, M., Rouse, E. D., Tian, H., Renshaw, P. F., Ciraulo, D. A., & Yurgelun-Todd, D. A. (2008). Performance on the Stroop predicts treatment compliance in cocaine-dependent individuals. *Neuropsychopharmacology, 33*, 827–836. doi:10.1038/sj.npp.1301465
- Stroebe, W., Mensink, W., Aarts, H., Schut, H., & Kruglanski, A. W. (2008). Why dieters fail: Testing the goal conflict model of eating. *Journal of Experimental Social Psychology, 44*, 26–36.
- Stroebe, W., van Koningsbruggen, G. M., Papies, E. K., & Aarts, H. (2013). Why most dieters fail but some succeed: A goal conflict model of eating behavior. *Psychological Review, 120*, 110–138.
- Stroop, J. R. (1935). Studies of interference in serial verbal reaction. *Journal of Experimental Psychology, 18*, 643–661
- Stunkard, A. J., & Messick, S. (1985). The three-factor eating questionnaire to measure dietary restraint, disinhibition and hunger. *Journal of Psychosomatic Research, 29*, 71–83.
- Styn, M. A., Bovbjerg, D. H., Lipsky, S., & Erblich, J. (2013). Cue-induced cigarette and food craving: A common effect? *Addictive Behaviors, 38*, 1840–1843. doi:10.1016/j.addbeh.2012.09.010.

- Svaldi, J., Naumann, E., Trentowska, M., & Schmitz, F. (2014). General and food-specific inhibitory deficits in binge eating disorder. *The International Journal of Eating Disorders, 47*, 534–42. doi:10.1002/eat.22260
- Swick, D., Ashley, V., & Turken, A. U. (2008). Left inferior frontal gyrus is critical for response inhibition. *BMC Neuroscience, 9*, 102. doi:10.1186/1471-2202-9-102
- Swick, D., Ashley, V., & Turken, U. (2011). Are the neural correlates of stopping and not going identical? Quantitative meta-analysis of two response inhibition tasks. *NeuroImage, 56*, 1655–65. doi:10.1016/j.neuroimage.2011.02.070
- Tabachnick, B. G., & Fidell, L. S. (2007). Using multivariate statistics (5th ed.). Boston: Allyn and Bacon.
- Tabibnia, G., Monterosso, J. R., Baicy, K., Aron, A. R., Poldrack, R. A, Chakrapani, S., ... London, E. D. (2011). Different forms of self-control share a neurocognitive substrate. *The Journal of Neuroscience, 31*, 4805–10. doi:10.1523/JNEUROSCI.2859-10.2011
- Tabu, H., Mima, T., Aso, T., Takahashi, R., & Fukuyama, H. (2011). Functional relevance of pre-supplementary motor areas for the choice to stop during Stop signal task. *Neuroscience Research, 70*, 277–84. doi:10.1016/j.neures.2011.03.007
- Taki, Y., Kinomura, S., Sato, K., Inoue, K., Goto, R., Okada, K., Uchida, S., Kawashima, R., & Fukuda, H. (2008). Relationship between body mass index and gray matter volume in 1,428 healthy individuals. *Obesity, 16*, 119–124. doi:10.1038/oby.2007.4
- Tangney, J. P., Baumeister, R. F., & Boone, A. L. (2004). High self-control predicts good adjustment, less pathology, better grades, and interpersonal success. *Journal of Personality, 72*, 271–324.
- Tapper, K., & Pothos, E. M. (2010). Development and validation of a Food Preoccupation Questionnaire. *Eating Behaviors, 11*, 45–53. doi:10.1016/j.eatbeh.2009.09.003
- Tepper, B. J. (1992). Dietary restraint and responsiveness to sensory-based food cues as measured by cephalic phase salivation and sensory specific satiety. *Physiology and Behavior, 52*, 305–311.
- Tetley, A., Brunstrom, J., & Griffiths, P. (2009). Individual differences in food-cue reactivity. The role of BMI and everyday portion-size selections. *Appetite, 52*, 614–20. doi:10.1016/j.appet.2009.02.005
- Thanos, P. K., Volkow, N. D., Freimuth, P., Umegaki, H., Ikari, H., Roth, G., Ingram, D. K., & Hitzemann, R. (2001). Overexpression of dopamine D2 receptors reduces alcohol. *Journal of Neurochemistry, 78*, 1094–1103.
- Thomas, E. L., Frost, G., Taylor-Robinson, S. D., & Bell, J. D. (2012). Excess body fat in obese and normal-weight subjects. *Nutrition Research Reviews, 25*, 150–61. doi:10.1017/S0954422412000054
- Thompson, F. E., & Byers, T. (1994). Dietary assessment resource manual. *The Journal of Nutrition, 124*, (11 Suppl), 2245S–2317S.
- Thorell, L. B., Lindqvist, S., Bergman Nutley, S., Bohlin, G., & Klingberg, T. (2009). Training and transfer effects of executive functions in preschool children. *Developmental Science, 12*, 106–13. doi:10.1111/j.1467-7687.2008.00745.x
- Tice, D. M., Bratslavsky, E., & Baumeister, R. F. (2001). Emotional distress regulation takes precedence over impulse control: if you feel bad, do it! *Journal of Personality and Social Psychology, 80*, 53–67.
- Tomkins, D. M., & Sellers, E. M. (2001). Addiction and the brain: The role of neurotransmitters in the cause and treatment of drug dependence. *Canadian Medical Association Journal, 164*, 817–821.

- Toumbourou, J. W., Stockwell, T., Neighbors, C., Marlatt, G. A., Sturge, J., & Rehm, J. (2007). Interventions to reduce harm associated with adolescent substance use. *Lancet*, *369*, 1391-1401.
- Townshend, T., & Duka, J. M. (2001). Attentional bias associated with alcohol cues: Differences between heavy and occasional social drinkers. *Psychopharmacology*, *157*, 67-74. doi:10.1007/s002130100764
- Uher, R., Yoganathan, D., Mogg, A., Eranti, S. V., Treasure, J., Campbell, I. C., McLoughlin, D. M., & Schmidt, U. (2005). Effect of left prefrontal repetitive transcranial magnetic stimulation on food craving. *Biological Psychiatry*, *58*, 840-842. doi:10.1016/j.biopsych.2005.05.043
- Urland, G. R., & Ito, T. A. (2005). Have your cake and hate it, too: Ambivalent food attitudes are associated with dietary restraint. *Basic and Applied Social Psychology*, *27*, 343-360. doi:10.1207/s15324834basps2704_8
- Van den Eynde, F., Claudino, A. M., Mogg, A., Horrell, L., Stahl, D., Ribeiro, W., Uher, R., Campbell, I., & Schmidt, U. (2010). Repetitive transcranial magnetic stimulation reduces cue-induced food craving in bulimic disorders. *Biological Psychiatry*, *67*, 793-795. doi:10.1016/j.biopsych.2009.11.023
- Van Gaal, L. F., Mertens, I. L., & De Block, C. E. (2006). Mechanisms linking obesity with cardiovascular disease. *Nature*, *444*, 875-880. doi:10.1038/nature05487
- Van Gucht, D., Vansteenwegen, D., Van den Bergh, O., & Beckers, T. (2008). Conditioned craving cues elicit an automatic approach tendency. *Behaviour Research and Therapy*, *46*, 1160-1169. doi:10.1016/j.brat.2008.05.010
- van Koningsbruggen, G. M., Stroebe, W., & Aarts, H. (2013b). Successful restrained eating and trait impulsiveness. *Appetite*, *60*, 81-84
- Van Koningsbruggen, G. M., Veling, H., Stroebe, W., & Aarts, H. (2013a). Comparing two psychological interventions in reducing impulsive processes of eating behaviour: Effects on self-selected portion size. *British Journal of Health Psychology*, 1-16. doi:10.1111/bjhp.12075
- van Strien, T., Breteler, M. H. M., & Ouwens, M. A. (2002). Restraint Scale, its sub-scales concern for dieting and weight fluctuation. *Personality and Individual Differences*, *33*, 791-802.
- van Strien, T., Frijters, J. E. R., Bergers, G. P. A., & Defares, P. B. (1986a). The Dutch Eating Behavior Questionnaire (DEBQ) for assessment of restrained, emotional and external eating behavior. *International Journal of Eating Disorders*, *5*, 295-315.
- van Strien, T., Frijters, J. E. R., Van Staveren, W. A., Defares, P. B., & Deurenberg, P. (1986b). The predictive validity of the Dutch restrained eating scale. *International Journal of Eating Disorders*, *5*, 747-755.
- Vartanian, L. R., Herman, C. P., & Polivy, J. (2007). Consumption stereotypes and impression management: How you are what you eat. *Appetite*, *48*, 265-77. doi:10.1016/j.appet.2006.10.008
- Veenstra, E. M., & De Jong, P. J. (2010). Restrained eaters show enhanced automatic approach tendencies towards food. *Appetite*, *55*, 30-36. doi:10.1016/j.appet.2010.03.007
- Veling, H., & Aarts, H. (2009). Putting behavior on hold decreases reward value of need-instrumental objects outside of awareness. *Journal of Experimental Social Psychology*, *45*, 1020-1023
- Veling, H., Aarts, H., & Papies, E. K. (2011). Using stop signals to inhibit chronic dieters' responses toward palatable foods. *Behaviour Research and Therapy*, *49*, 771-780.

- Veling, H., Aarts, H., & Stroebe, W. (2013a). Using stop signals to reduce impulsive choices for palatable unhealthy foods. *British Journal of Health Psychology*, *18*, 354–368.
- Veling, H., Aarts, H., & Stroebe, W. (2013b). Stop signals decrease choices for palatable foods through decreased food evaluation. *Frontiers in Psychology*, *4*, 875. doi:10.3389/fpsyg.2013.00875
- Veling, H., Holland, R. W., & van Knippenberg, A. (2008). When approach motivation and behavioral inhibition collide: Behavior regulation through stimulus devaluation. *Journal of Experimental Social Psychology*, *44*, 1013-1019.
- Veling, H., van Koningsbruggen, G. M., Aarts, H., & Stroebe, W. (2014). Targeting impulsive processes of eating behavior via the internet. Effects on body weight. *Appetite*, *78*, 102–9. doi:10.1016/j.appet.2014.03.014
- Verbruggen, F., & Logan, G. D. (2009a). Models of response inhibition in the stop-signal and stop-change paradigms. *Neuroscience and Biobehavioural Reviews*, *33*, 647-661. doi:10.1016/j.neubiorev.2008.08.014.
- Verbruggen, F., & Logan, G. D. (2009b). Proactive adjustments of response strategies in the stop-signal paradigm. *Journal of Experimental Psychology. Human Perception and Performance*, *35*, 835–54. doi:10.1037/a0012726
- Verbruggen, F., & Logan, G. S. (2008). Automatic and controlled response inhibition: Associative learning in the go/no-go and stop-signal paradigms. *Journal of Experimental Psychology: General*, *137*, 649-672.
- Verbruggen, F., Adams, R. C., van 't Wout, F., Stevens, T., McLaren, I. P. L., & Chambers, C. D. (2013). Are the effects of response inhibition on gambling long-lasting? *PLOS One*, *7*, e70155. doi:10.1371/journal.pone.0070155
- Verbruggen, F., Adams, R., & Chambers, C. D. (2012). Proactive motor control reduces monetary risk taking in gambling. *Psychological Science*, *23*, 805-815.
- Verbruggen, F., Aron, A. R., Stevens, M. A., & Chambers, C. D. (2010). Theta burst stimulation dissociates attention and action updating in human inferior frontal cortex. *Proceedings of the National Academy of Sciences of the United States of America*, *107*, 13966–13971. doi:10.1073/pnas.1001957107/-/DCSupplemental.www.pnas.org/cgi/doi/10.1073/pnas.1001957107
- Verbruggen, F., Best, M., Bowditch, W. A., Stevens, T., & McLaren, I. P. L. (in press). The inhibitory control reflex. *Neuropsychologia*, doi:10.1016/j.neuropsychologia.2014.08.014
- Verdejo-García, A. J., Perales, J. C., & Pérez-García, M. (2007). Cognitive impulsivity in cocaine and heroin polysubstance abusers. *Addictive Behaviors*, *32*, 950–966. doi:10.1016/j.addbeh.2006.06.032
- Verdejo-García, A., Lawrence, A. J., & Clark, L. (2008). Impulsivity as a vulnerability marker for substance-use disorders: Review of findings from high-risk research, problem gamblers and genetic association studies. *Neuroscience and Biobehavioral Reviews*, *32*, 777–810. doi:10.1016/j.neubiorev.2007.11.003
- Viswesvaran, C., & Schmidt, F. L. (1992). A meta-analytic comparison of the effectiveness of smoking cessation methods. *Journal of Applied Psychology*, *77*, 554–561.
- Vitaro, F., Ferland, F., Jacques, C., & Ladouceur, R. (1998). Gambling, substance use, and impulsivity during adolescence. *Psychology of Addictive Behaviors*, *12*, 185-194.
- Vohs, K. D., & Heatherton, T. F. (2000). Self-regulatory failure. *Psychological Science*, *11*, 249–254.
- Volkow, N. D., & Wise, R. A. (2005). How can drug addiction help us understand obesity? *Nature Neuroscience*, *8*, 555–560. doi:10.1038/nn1452

- Volkow, N. D., Chang, L., Wang, G. J., Fowler, J. S., Ding, Y. S., Sedler, M., Logan, J., Franceschi, D., Gatley, J., Hitzemann, R., Gifford, A., Wong, C., & Pappas, N. (2001). Low level of brain dopamine D2 receptors in methamphetamine abusers: Association with metabolism in the orbitofrontal cortex. *The American Journal of Psychiatry*, *158*, 2015–2021.
- Volkow, N. D., Fowler, J. S., & Wang, G. (2003b). The addicted human brain: insights from imaging studies. *The Journal of Clinical Investigation*, *111*, 1444–1451. doi:10.1172/JCI200318533.
- Volkow, N. D., Fowler, J. S., Wang, G. J., Hitzemann, R., Logan, J., Schlyer, D. J., Dewey, S. L., & Wolf, A. P. (1993). Decreased dopamine D2 receptor availability is associated with reduced frontal metabolism in cocaine abusers. *Synapse*, *14*, 169–177. doi:10.1002/syn.890140210
- Volkow, N. D., Wang, G., & Baler, R. D. (2011a). Reward, dopamine and the control of food intake: Implications for obesity. *Trends in Cognitive Sciences*, *15*, 37–46. doi:10.1016/j.tics.2010.11.001
- Volkow, N. D., Wang, G.-J., Begleiter, H., Porjesz, B., Fowler, J. S., Telang, F., Wong, C., Ma, Y., Logan, J., Goldstein, R., Alexoff, D., & Thanos, P. K. (2006). High levels of dopamine D2 receptors in unaffected members of alcoholic families. *Archives of General Psychiatry*, *63*, 999–1008.
- Volkow, N. D., Wang, G.-J., Fowler, J. S., & Telang, F. (2008a). Overlapping neuronal circuits in addiction and obesity: Evidence of systems pathology. *Philosophical Transactions of the Royal Society of London. Series B, Biological Sciences*, *363*, 3191–3200. doi:10.1098/rstb.2008.0107
- Volkow, N. D., Wang, G.-J., Fowler, J. S., Logan, J., Hitzemann, R., Ding, Y.-S., Pappas, N., Shea, C., & Piscani, K. (1996). Decreases in dopamine receptors but not in dopamine transporters in alcoholics. *Alcoholism, Clinical and Experimental Research*, *20*, 1594–1598.
- Volkow, N. D., Wang, G.-J., Fowler, J. S., Logan, J., Jayne, M., Franceschi, D., Wong, C., Gatley, S. J., Gifford, A. N., Ding, Y.-S., & Pappas, N. (2002a). “Nonhedonic” food motivation in humans involves dopamine in the dorsal striatum and methylphenidate amplifies this effect. *Synapse*, *44*, 175–180. doi:10.1002/syn.10075
- Volkow, N. D., Wang, G.-J., Fowler, J. S., Tomasi, D., Telang, F., & Baler, R. (2010b). Addiction: Decreased reward sensitivity and increased expectation sensitivity conspire to overwhelm the brain’s control circuit. *BioEssays : News and Reviews in Molecular, Cellular and Developmental Biology*, *32*, 748–755. doi:10.1002/bies.201000042
- Volkow, N. D., Wang, G.-J., Maynard, L., Jayne, M., Fowler, J. S., Zhu, W., Logan, J., Gatley, S. J., Ding, Y.-S., Wong, C., & Pappas, N. (2003a). Brain dopamine is associated with eating behaviors in humans. *The International Journal of Eating Disorders*, *33*, 136–142. doi:10.1002/eat.10118
- Volkow, N. D., Wang, G.-J., Telang, F., Fowler, J. S., Goldstein, R. Z., Alia-Klein, N., Logan, J., Wong, C., Thanos, P. K., Ma, Y., & Pradhan, K. (2009a). Inverse association between BMI and prefrontal metabolic activity in healthy adults. *Obesity*, *17*, 60–65. doi:10.1038/oby.2008.469
- Volkow, N. D., Wang, G.-J., Telang, F., Fowler, J. S., Thanos, P. K., Logan, J., Alexoff, D., Ding, Y.-S., Wong, C., Ma, Y., & Pradhan, K. (2008b). Low dopamine striatal D2 receptors are associated with prefrontal metabolism in obese subjects: Possible contributing factors. *NeuroImage*, *42*, 1537–1543. doi:10.1016/j.neuroimage.2008.06.002.

- Volkow, N. D., Wang, G.-J., Tomasi, D., & Baler, R. D. (2013). The addictive dimensionality of obesity. *Biological Psychiatry*, *73*, 811–818. doi:10.1016/j.biopsych.2012.12.020
- von Bastian, C. C., Locher, A., & Rufin, M. (2013). Tootool: A Java-based open-source programming framework for psychological studies. *Behavior Research Methods*, *45*, 108–115. doi: 10.3758/s13428-012-0224-y
- Wager, T. D., Sylvester, C.-Y. C., Lacey, S. C., Nee, D. E., Franklin, M., & Jonides, J. (2005). Common and unique components of response inhibition revealed by fMRI. *NeuroImage*, *27*, 323–240. doi:10.1016/j.neuroimage.2005.01.054
- Walther, K., Birdsill, A. C., Glisky, E. L., & Ryan, L. (2010). Structural brain differences and cognitive functioning related to body mass index in older females. *Human Brain Mapping*, *31*, 1052–1064. doi:10.1002/hbm.20916
- Wang, G. J., Volkow, N. D., Logan, J., Pappas, N. R., Wong, C. T., Zhu, W., Netusil, N., & Fowler, J. S. (2001). Brain dopamine and obesity. *Lancet*, *357*, 354–357.
- Wang, G., Volkow, N. D., Thanos, P. K., & Fowler, J. S. (2004). Similarity between obesity and drug addiction as assessed by neurofunctional imaging: A concept review. *Journal of Addictive Diseases*, *23*, 39–53.
- Wang, G.-J., Volkow, N. D., Felder, C., Fowler, J. S., Levy, A. V, Pappas, N. R., Wong, C., Zhu, W., & Netusil, N. (2002). Enhanced resting activity of the oral somatosensory cortex in obese subjects. *Neuroreport*, *13*, 1151–1155.
- Wang, Y., & Beydoun, M. A. (2007). The obesity epidemic in the United States — gender, age, socioeconomic, racial/ethnic, and geographic characteristics: A systematic review and meta-regression analysis. *Epidemiologic Reviews*, *29*, 6–28. doi:10.1093/epirev/mxm007
- Ward, A., & Mann, T. (2000). Don't mind if I do: Disinhibited eating under cognitive load. *Journal of Personality and Social Psychology*, *78*, 753–763.
- Wardle, J. (1986). The assessment of restrained eating. *Behaviour Research and Therapy*, *24*, 13–215.
- Wardle, J., & Beales, S. (1988). Control and loss of control over eating: an experimental investigation. *Journal of Abnormal Psychology*, *97*, 35–40.
- Wardle, J., & Beales, S. (1987). Restraint and food intake: An experimental study of eating patterns in the laboratory and normal life. *Behaviour Research and Therapy*, *25*, 179–185.
- Watanabe, J., Sugiura, M., Sato, K., Sato, Y., Maeda, Y., Matsue, Y., ... Kawashima, R. (2002). The human prefrontal and parietal association cortices are involved in no-go performances: An event-related fMRI study. *NeuroImage*, *17*, 1207–1216. doi:10.1006/nimg.2002.1198
- Watson, D., Clark, L. A., & Tellegen, A. (1988). Development and validation of brief measures of positive and negative affect: The PANAS scales. *Journal of Personality and Social Psychology*, *54*, 1063–70.
- Watson, D., Clark, L. A., Weber, K., Assenheimer, J. S., Strauss, M. E., & McCormick, R. A. (1995). Testing a tripartite model: II. Exploring the symptom structure of anxiety and depression in student, adult, and patient samples. *Journal of Abnormal Psychology*, *104*, 15–25.
- Watson, P., de Wit, S., Hommel, B., & Wiers, R. W. (2012). Motivational mechanisms and outcome expectancies underlying the approach bias toward addictive substances. *Frontiers in Psychology*, *3*, 440. doi:10.3389/fpsyg.2012.00440
- Weingarten, H. P., & Elston, D. (1990). The phenomenology of food cravings. *Appetite*, *15*, 231–246.

- Weingarten, H. P., & Elston, D. (1991). Food cravings in a college population. *Appetite, 17*, 167-175.
- Weller, R. E., Cook, E. W., III, Avsar, K. B., & Cox, J. E. (2008). Obese women show greater delay discounting than healthy-weight women. *Appetite, 51*, 563–569. doi:10.1016/j.appet.2008.04.010
- 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*, 136–143. doi:10.1016/j.jbtep.2013.09.009
- Werthmann, J., Roefs, A., Nederkoorn, C., & Jansen, A. (2013b). Desire lies in the eyes: attention bias for chocolate is related to craving and self-endorsed eating permission. *Appetite, 70*, 81–89. doi:10.1016/j.appet.2013.06.087
- 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*, 561–569. doi:10.1037/a0024291
- Wessel, J. R., O’Doherty, J. P., Berkebile, M. M., Linderman, D., Aron, A. R. (in press). Stimulus devaluation induced by stopping action. *Journal of Experimental Psychology: General*.
- Wessel, J. R., Reynoso, H. S., & Aron, A. R. (2013). Saccade suppression exerts global effects on the motor system. *Journal of Neurophysiology, 110*, 883–90. doi:10.1152/jn.00229.2013
- Westenhoefer, J., Broeckmann, P., Münch, A-K., & Pudel, V. (1994). Cognitive control of eating behaviour and the disinhibition effect. *Appetite, 23*, 27-41.
- Weygandt, M., Mai, K., Dommès, E., Leupelt, V., Hackmack, K., Kahnt, T., Rothmund, Y., Spranger, J., & Haynes, J.-D. (2013). The role of neural impulse control mechanisms for dietary success in obesity. *NeuroImage, 83*, 669–678. doi:10.1016/j.neuroimage.2013.07.028
- White, M. A., Whisenhunt, B. L., Williamson, D. A., Greenway, F. L., & Netemeyer, R. G. (2002). Development and validation of the food-craving inventory. *Obesity Research, 10*, 107–114. doi:10.1038/oby.2002.17
- Whiteside, S. P., & Lynam, D. R. (2001). The Five Factor Model and impulsivity: Using a structural model of personality to understand impulsivity. *Personality and Individual Differences, 30*, 669–689. doi:10.1016/S0191-8869(00)00064-7
- WHO. (2003). Obesity and overweight. World Health Organization.
- Wideman, C. H., Nadzam, G. R., & Murphy, H. M. (2005). Implications of an animal model of sugar addiction, withdrawal and relapse for human health. *Nutritional Neuroscience, 8*, 269–276.
- Wiederman, M. W. (2004). Self-control and sexual behavior. In R. F. Baumeister & K. D. Vohs (Eds.), *Handbook of Self-Regulation: Research, Theory and Applications* (pp. 525-536). New York, NY: Guilford Press.
- Wiers, R. W., & Stacy, A. W. (2006). Implicit cognition and addiction. *Current Directions in Psychological Science, 15*, 292–296.
- Wiers, R. W., Ames, S. L., Hofmann, W., Krank, M., & Stacy, A. W. (2010a). Impulsivity, impulsive and reflective processes and the development of alcohol use and misuse in adolescents and young adults. *Frontiers in psychology, 1*, 1–12.
- Wiers, R. W., Bartholow, B. D., van den Wildenberg, E., Thush, C., Engels, R. C., Sher, K. J., Grenard, J., Ames, S. L., & Stacy, A. W. (2007). Automatic and controlled processes and the development of addictive behaviors in adolescents: A review and a model. *Pharmacology Biochemistry and Behavior, 86*, 263–283.

- Wiers, R. W., Eberl, C., Rinck, M., Becker, E., & Lindenmeyer, J. (2011). Re-training automatic action tendencies changes alcoholic patients' approach bias for alcohol and improves treatment outcome. *Psychological Science*, *22*, 490–497.
- Wiers, R. W., Galdwin, T. E., Hofmann, W., Salemink, E., & Ridderinkhof, R. (2013). Cognitive bias modification and cognitive control training in addiction and related psychopathology: Mechanisms, clinical perspectives, and ways forward. *Clinical Psychological Science*, *1*, 192–212.
- Wiers, R. W., Houben, K., Fadardi, J. S., van Beek, P., Rhemtulla, M. T., & Cox, W. M. (2015). Alcohol cognitive bias modification training for problem drinkers over the web. *Addictive Behaviors*, *40*, 21–26. doi:10.1016/j.addbeh.2014.08.010
- Wiers, R. W., Rinck, M., Kordts, R., Houben, K., & Strack, F. (2010b). Retraining automatic action-tendencies to approach alcohol in hazardous drinkers. *Addiction*, *105*, 279–287.
- Williams, J., Wake, M., Hesketh, K., Maher, E., & Waters, E. (2005). Health-related quality of life of overweight and obese children. *The Journal of the American Medical Association*, *293*, 70–76. doi:10.1001/jama.293.1.70
- Wills, T. A., Walker, C., Mendoza, D., & Ainette, M. G. (2006). Behavioral and emotional self-control: Relations to substance use in samples of middle and high school students. *Psychology of Addictive Behaviors*, *20*, 265–78.
- Wills, T. A., & Stoolmiller, M. (2002). The role of self-control in early escalation of substance use: A time-varying analysis. *Journal of Consulting and Clinical Psychology*, *70*, 986–997.
- Wills, T. A., Ainette, M. G., Stoolmiller, M., Gibbons, F. X., & Shinar, O. (2008). Good self-control as a buffering agent for adolescent substance use: An investigation in early adolescence with time-varying covariates. *Psychology of Addictive Behaviors*, *22*, 459–471. doi:10.1037/a0012965
- Wills, T. A., Isasi, C. R., Mendoza, D., & Ainette, M. G. (2007). Self-control constructs related to measures of dietary intake and physical activity in adolescents. *Journal of Adolescent Health*, *41*, 551–558. doi:10.1016/j.jadohealth.2007.06.013
- Wilson, G. T. (1991). The addiction model of eating disorders: A critical analysis. *Advances in Behaviour Research and Therapy*, *13*, 27–72.
- Wilson, G. T. (2010). Eating disorders, obesity and addiction. *European Eating Disorders Review*, *18*, 341–351. doi:10.1002/erv.1048
- Wirt, T., Hundsdörfer, V., Schreiber, A., Keszyüs, D., & Steinacker, J. M. (2014). Associations between inhibitory control and body weight in German primary school children. *Eating Behaviors*, *15*, 9–12. doi:10.1016/j.eatbeh.2013.10.015
- Woltering, S., Liu, Z., Rokeach, A., & Tannock, R. (2013). Neurophysiological differences in inhibitory control between adults with ADHD and their peers. *Neuropsychologia*, *51*, 1888–95. doi:10.1016/j.neuropsychologia.2013.06.023
- Xue, G., Aron, A. R., & Poldrack, R. A. (2008). Common neural substrates for inhibition of spoken and manual responses. *Cerebral Cortex*, *18*, 1923–32. doi:10.1093/cercor/bhm220
- Yeomans, M. R. (2000). Rating changes over the course of meals: What do they tell us about motivation to eat? *Neuroscience and Biobehavioral Reviews*, *24*, 249–59.
- Yokum, S., & Stice, E. (2013). Cognitive regulation of food craving: effects of three cognitive reappraisal strategies on neural response to palatable foods. *International Journal of Obesity*, *37*, 1565–70. doi:10.1038/ijo.2013.39
- Yokum, S., Ng, J., & Stice, E. (2012). Relation of regional gray and white matter volumes to current BMI and future increases in BMI: A prospective MRI study. *International Journal of Obesity*, *36*, 656–664. doi:10.1038/ijo.2011.175

- Yoshida, M., Yokoo, H., Mizoguchi, K., Kawahara, H., Tsuda, A., Nishikawa, T., & Tanaka, M. (1992). Eating and drinking cause increased dopamine release in the nucleus accumbens and ventral tegmental area in the rat: Measurement by in vivo microdialysis. *Neuroscience Letters*, *139*, 73–76.
- Zhang, Y., von Deneen, K. M., Tian, J., Gold, M. S., & Liu, Y. (2011). Food addiction and neuroimaging. *Current Pharmaceutical Design*, *17*, 1149–1157.
- Zheng, D., Oka, T., Bokura, H., & Yamaguchi, S. (2008). The key locus of common response inhibition network for no-go and stop signals. *Journal of Cognitive Neuroscience*, *20*, 1434–1442. doi:10.1162/jocn.2008.20100
- Ziauddeen, H., & Fletcher, P. C. (2013). Is food addiction a valid and useful concept? *Obesity Reviews*, *14*, 19–28. doi:10.1111/j.1467-789X.2012.01046.x
- Ziauddeen, H., Farooqi, I. S., & Fletcher, P. C. (2012a). Food addiction: Is there a baby in the bathwater? *Nature Reviews Neuroscience*, *13*, 514. doi:10.1038/nrn3212-c2
- Ziauddeen, H., Farooqi, I. S., & Fletcher, P. C. (2012b). Obesity and the brain: How convincing is the addiction model? *Nature Reviews. Neuroscience*, *13*, 279–286. doi:10.1038/nrn3212

Appendices

Appendix 1. Summary of methods and results for published studies investigating the effect of inhibition training on food intake, food choice and weight change.	A3
Appendix 2. Training parameters for studies in this thesis and other published studies investigating the effect of food-related inhibition training on food intake, food choice and weight change.	A10
Appendix 3: Stimuli used in the stop-signal training task in Study 2.	A14
Appendix 4: Words used in the unipolar, SC-IATs, Studies 2 and 4.	A16
Appendix 5: Stimuli used in the stop-signal and go/no-go training tasks in Study 3.	A17
Appendix 6: Nutritional information and weights for the unhealthy and healthy foods presented in the snack phase in Study 3.	A19
Appendix 7: Pseudo-random orders for the presentation of unhealthy and healthy foods in the snack buffet for Study 3.	A20
Appendix 8: Food stimuli used in the go/no-go training tasks in Study 4.	A21
Appendix 9: Snack food stimuli used in the SC-IAT in Study 4.	A22
Appendix 10: ‘New’ snack food stimuli used in the Evaluation Task in Study 4.	A23
Appendix 11. Study A1: Pilot study investigating the effect of combining inhibitory control training with prefrontal brain stimulation to reduce food consumption	A24
Appendix 12: Safety Screening Questionnaire for TMS and tDCS contraindications.	A51
Appendix 13: Food stimuli used in the go/no-go training tasks in Study A1 (Appendix 11).	A52
Appendix 14: Nutritional information and weights for the unhealthy and healthy foods presented per food selection in the snack buffet in Study A1 (Appendix 11).	A53
Appendix 15: Pseudo-random orders for the presentation of unhealthy and healthy foods in the snack buffet for Study A1 (Appendix 11).	A54
Appendix 16: Correlation matrices for the difference in pre- and post- tDCS/training	A56

measures of state hunger (hunger, fullness and desire to eat; VAS) and mood (positive and negative affect; PANAS) and calorie consumption according to food type, and for total calorie consumption, Study A1 (Appendix 11).

Appendix 17: Correlation matrices for the difference in pre- and post- tDCS/training measures of state food craving (G-FCQ-S) and calorie consumption according to food type, and for total calorie consumption, Study A1 (Appendix 11). A57

Appendix 1. Summary of methods and results for published studies investigating the effect of inhibition training on food intake, food choice and weight change.

Study	Participants and Methods	Main Findings
Guerrieri <i>et al.</i> (2012)	<p>Sample: Healthy-weight female undergraduate students. Participants were asked not to eat for 2 hours before the study.</p> <p>Training: Participants in the inhibition and impulsivity groups performed a standard SST (non-food-related) in which the proportion of stop and go trials were increased, respectively. Stop trials increased by 5% in each block from 25-50% in the inhibition group and go trials increased from 75-100% in the impulsivity group. A neutral control group performed a critical reading task for two stories that were not food-related.</p> <p>DV: Participants took part in a bogus taste test for ten minutes. They were presented with four bowls of mini chocolate chip cookies, wine gums, crisps and saltines (salted crackers).</p>	<p>Calorie intake was significantly greater in the impulsivity group compared to both the inhibition and neutral control groups. There was no statistically significant difference between the inhibition and control groups.</p>
Houben (2011)	<p>Sample: Female undergraduate students who liked nuts, crisps and M&Ms to a similar extent. Participants were asked not to eat for 2 hours before the study.</p> <p>Training: Participants performed a SST with images of nuts, crisps, M&Ms and filler images of chairs (see Appendix 2 for full task details). Participants were required to categorise the images as quickly as possible as food or non-food and were instructed to inhibit their response when an auditory stop-signal was presented (on 25% of trials). The</p>	<p>There was a significant interaction between the manipulation and baseline inhibitory control ability (SSRT measured using a standard SST, i.e. a non-food-related SST). Intake of the control food was significantly greater for those with low, compared to high, inhibitory control.</p>

Study	Participants and Methods	Main Findings
Houben & Jansen (2011)	<p>signal was presented after a variable delay using set SSDs. For each participant one type of food was consistently associated with responding (go food), one food was consistently associated with stopping (stop food) and one food was presented evenly with go and stop trials (50:50 mapping; control food).</p> <p>DV: Following training participants completed a bogus taste test for 10 minutes with nuts, crisps and M&Ms.</p> <p>Sample: Female undergraduate students who were high trait chocolate cravers (ACQ 10+). Participants were asked not to eat for 2 hours before the study.</p> <p>Training: Participants performed a GNG task with images of chocolate, empty plates and filler images (see Appendix 2 for full task details). For the control group all images were presented once with a go signal and once with a no-go signal. For the no-go group all chocolate images were paired with a no-go signal, all empty plates were paired with a go signal and for the filler trials half were go trials and half were no-go trials. In the go group all chocolate images were paired with a go signal, all empty plates with a no-go signal and filler images were split 50:50 for go and no-go signals.</p> <p>DV: Following training participants completed a bogus taste test for 10 minutes with milk, dark and extra dark chocolate pellets.</p>	<p>For those with low inhibitory control stop training reduced consumption relative to the control group; for those with high inhibitory control there was a trend showing that go training increased consumption compared to the control group ($p=0.06$, $\eta^2_p=0.12$).</p> <p>The no-go group consumed significantly fewer calories than the control group, however, the go group did not significantly differ from either the control or no-go groups. After controlling for dietary restraint (RS) simple slopes analyses showed that chocolate consumption increased as a function of restraint in the control condition, this effect was in the same direction for the go group but was not significant and consumption decreased as a function of restraint in the no-go group (statistical trend $p=0.06$, $\beta=-0.36$).</p>

Study	Participants and Methods	Main Findings
Lawrence <i>et al.</i> (under review)	Sample: Mixed gender sample of staff and students who were asked not to eat for 3 hours before the study.	The stop group consumed significantly fewer calories than the double-response group. This effect was not moderated by dietary restraint (using the DEBQRE).
Study 1	<p>Training: Training involved a SST with images of food (50% of which were crisps; see Appendix 2 for full task details) and filler images of household items. Participants responded to the location of the image and a visual signal was presented on 33% of trials after a variable delay (SSD was set according to simulated tracking procedure). The majority of signals were mapped onto the food images. Participants in the stop group had to inhibit their responses when a signal was presented and participants in the double-response group made an additional response.</p> <p>DV: Participants were provided with a bowl of crisps as ‘refreshments’ while they completed a battery of personality questionnaires.</p>	
Lawrence <i>et al.</i> (under review)	Sample: Mixed gender sample of staff and students who were asked not to eat for 3 hours before the study.	There was no main effect of training group and no significant interaction with food type (signal or non-signal food). A direct comparison of stop and double-response groups also failed to replicate the findings of Study 1 (above). There was, however, a significant interaction with restraint scores (DEBQRE): there was no significant effect of training at low levels of
Study 2	<p>Training: Participants performed a SST with images of chocolate, crisps, other foods and filler images (see Appendix 2 for full task details). Signals were presented on 25% of trials and the majority of signals were mapped onto either the chocolate or crisps (counterbalanced across participants) so that there was one ‘signal food’ and one ‘non-signal food’. In addition to the stop and double-response groups, described above, there</p>	

Study	Participants and Methods	Main Findings
	<p>was a third group who ignored the signals and made a location response on every trial.</p> <p>DV: A bogus taste-test was presented after training with chocolate and crisps. Participants were left with the food for 20 minutes.</p>	<p>restraint but at high levels there was a significant decrease in intake of the signal food in the stop group relative to the double-response group. There was also a statistical trend for a decrease in the stop group relative to the ignore group ($p=0.098$, $d=0.35$).</p>
<p>Lawrence <i>et al.</i> (under review)</p>	<p>Sample: Mixed gender sample of staff and students who were asked not to eat for 3 hours before the study.</p>	<p>There was no significant main effect of training condition on food intake and no statistically significant moderating effect of dietary restraint (DEBQRE).</p>
<p>Study 3</p>	<p>Training: The training task was the same as that in Study 2 (see above) with the exception that all images were non-food images. There was a general stop group, for whom there were no specific stimulus-stop associations (for this group SSD was set according to a standard tracking procedure), and a stop group and double-response group – in these groups signals were mapped onto one stimulus type (e.g. pens).</p> <p>DV: Following training participants were presented with a bogus taste test, with crisps and chocolate, and were left alone for 20 minutes.</p>	

Study	Participants and Methods	Main Findings
van Koningsbruggen et al. (2013a)	<p>Sample: Mostly undergraduate students, mixed gender.</p> <p>Training: Participants were given a GNG task with images of sweets and everyday objects (see Appendix 2 for full task details). Participants responded to go cues and in the no-go group participants were instructed to withhold their response when a no-go signal was presented. In this group all sweet images were presented alongside a no-go signal. Participants also formed implementation intentions that were either diet-related or not.</p> <p>DV: Following the training task participants were asked to serve themselves sweets for a later taste test either manually (Study 1) or using a computerised dispenser (Study 2).</p>	<p>There was a significant effect of both interventions, in both studies; participants who were in the no-go training and diet-related implementation intention groups served themselves significantly fewer sweets than participants in the go, non-diet related implementation intention group (double control group). There was no additional reduction in consumption for combining the two interventions (the no-go, diet-related implementation intention group).</p>
Veling et al. (2011) Study 2	<p>Sample: Mixed gender, undergraduate students.</p> <p>Training: Training involved a GNG task with images of sweets and filler items (see Appendix 2 for full task details). In the no-go group sweets were always presented alongside a no-go signal, in the go group participants responded on every trial.</p> <p>DV: Participants were given a bag of sweets and were asked to taste and rate one sweet in the lab. They then took the bag home with them and could eat as many sweets as they liked. Participants were asked to bring the sweets back in with them the following day for the second part of the study.</p>	<p>There was no main effect of training condition on consumption but there was a significant interaction with restraint score (RSCD). For the low restraint participants there was no effect of training but for the high restraint participants those in the no-go group consumed significantly fewer sweets than those in the go group.</p>

Study	Participants and Methods	Main Findings
Velting <i>et al.</i> (2013a)	<p>Sample: Mixed gender sample who participated either before or after their lunch.</p> <p>Training: Participants were presented with a GNG task with images of palatable foods and filler stimuli (see Appendix 2 for full task details). For the no-go group foods were always paired with a no-go signal; for the go group foods were always paired with a response.</p> <p>DV: Participants were asked to select eight snack foods that they would like to consume from an array of sixteen healthy and unhealthy snacks.</p>	<p>For participants with a high appetite (participated before lunch; Study 1) and for those who consumed the foods frequently (Study 2), participants in the no-go group chose the foods presented during training less often than participants in the go group.</p>
Velting <i>et al.</i> (2013b)	<p>Sample: Mixed gender sample who participated either before or after their lunch.</p> <p>Training: Training involved a GNG task in which images of food were either paired with a no-go signal, 4, 12 or 24 times, or they were paired with a go signal, 4, 12 or 24 times (see Appendix 2 for full task details).</p> <p>DV: Participants rated the attractiveness of the foods before selecting 3 of 7 palatable foods. Following food choices they rated the palatability and frequency of consumption for each food.</p>	<p>For high appetite participants (participated before lunch), no-go foods were evaluated less positively than go foods (but both evaluations were non-significant compared to a novel, baseline, food). High appetite participants also selected fewer no-go than go foods. A mediation analysis revealed that the evaluation of no-go foods had a direct effect on food choice and mediated the effect of appetite on food choice. The evaluation of go foods, conversely, did not have a direct effect on food choice and did not mediate the effect of appetite on choice.</p>

Study	Participants and Methods	Main Findings
Velting <i>et al.</i> (2014)	<p>Sample: Mainly student sample with a secondary education and BMI<35.</p> <p>Training: Training was an online GNG task. For the food GNG group images of food and drink were always paired with a no-go signal and filler images were always paired with a go signal. In the control no-go group there were no food images; half the filler trials were presented with a go signal and half with a no-go signal (see Appendix 2 for full task details). Participants repeated the training program once a week, for four weeks</p> <p>Participants also formed with diet-related or non-diet-related implementation intentions.</p> <p>DV: Participants were weighed at the beginning and end of the study to determine weight loss.</p>	<p>Both diet-related implementation intentions and food GNG training facilitated weight loss compared to the double-control group who performed the control GNG task and non-diet-related implementation intentions. Dieting implementation intentions appeared to be particularly effective for those with a strong dieting goal whereas food GNG training was particularly effective for those with a high BMI.</p>

Appendix 2. Training parameters for studies in this thesis and other published studies investigating the effect of food-related inhibition training on food intake, food choice and weight change.

Study	Num. of blocks	Num. of trials per block	Total num. of trials	Stimulus type, number of stimuli per block and number of associated inhibition-signals per block			Overall % of inhibition trials	Control group
Study 2: SST (this thesis)	10	48	480	Chocolate 8 (16.7%) 7 (87.5%)	Crisps 8 (16.7%) 1 (12.5%)	Fillers (household items) 32 (66.6%) 4 (12.5%)	25%	Double-response to signals
Study 3: SST (this thesis)	8	36	288	Unhealthy foods 9 (25%) 8 (88.9%)	Healthy foods 9 (25%) 1 (11.1%)	Fillers (clothes) 18 (50%) 1 (5.56%)	27.8%	Double-response to signals
Study3: GNG (this thesis)	8	36	288	Unhealthy foods 9 (25%) 9 (100%)	Healthy foods 9 (25%) 0 (0%)	Fillers (clothes) 18 (50%) 9 (50%)	50%	Single response on every trial
Study 4: GNG (this thesis)	4	36	144	Unhealthy foods 9 (25%) 9 (100%)	Healthy foods 9 (25%) 0 (0%)	Fillers (clothes) 18 (50%) 9 (50%)	50%	Single response on every trial

Study	Num. of blocks	Num. of trials per block	Total num. of trials	Stimulus type, number of stimuli per block and number of associated inhibition-signals per block				Overall % of inhibition trials	Control group
Study A1: GNG (this thesis, Appendix 13)	8	40	320	Unhealthy foods	Healthy foods	Fillers (clothes)		50%	Single response on every trial
				10 (25%)	10 (25%)	20 (50%)			
				10 (100%)	0 (0%)	10 (50%)			
Houben & Jansen (2011): GNG	2	160	320	Chocolate	Empty plates	Fillers (other snack foods)		50%	Go to food / stop to plates / inconsistent responding group
				4 (25%)	4 (25%)	8 (50%)			
				4 (100%)	0 (0%)	4 (50%)			
Houben (2011): SST	6	48	288	Chocolate	Crisps	Nuts	Fillers (chairs)	50%	Within-subjects design
				8 (16.7%)	8 (16.7%)	8 (16.7%)	24 (50%)		
				1 food paired with 8 signals (100%), 1 food paired with no signals (0%) and 1 food paired with 4 signals (50%)					
Lawrence <i>et al.</i> (under review) Study 1: SST	10	48	480	Crisps	Other foods	Fillers (household objects/ clothes)		33.3%	Double-response to signals
				8 (16.7%)	8 (16.7%)	32 (66.7%)			
				7 (87.5%)	7 (87.5%)	2 (6.25%)			

Study	Num. of blocks	Num. of trials per block	Total num. of trials	Stimulus type, number of stimuli per block and number of associated inhibition-signals per block				Overall % of inhibition trials	Control group
Lawrence <i>et al.</i> (under review) Study 2: SST	8	64	512	Signal food (chocolate or crisps) 8 (12.5%) 7 (87.5%)	No-signal food (chocolate or crisps) 8 (12.5%) 1 (12.5%)	Other foods 16 (25%) 8 (50%)	Fillers (household objects/ clothes) 32 (50%) 0 (0%)	25%	Double-response to signals and group who ignored signals
van Koningsbruggen <i>et al.</i> (2013a)	12	6	72	Sweets 1 (16.7%) 1 (100%)	Fillers (common objects) 5 (83.3%) 2 (40%)			50%	Single response on every trial
Veling <i>et al.</i> (2011) Study 2	12	6	72	Sweets 1 (16.7%) 1 (100%)	Fillers (common objects and art) 5 (83.3%) 2 (40%)			50%	Single response on every trial
Veling <i>et al.</i> (2013a)	8	12	96	Unhealthy foods 4 (33.3%) 4 (100%)	Fillers (common objects) 8 (66.7%) 2 (25%)			50%	Respond to all foods/ inhibit on 75% filler images

Study	Num. of blocks	Num. of trials per block	Total num. of trials	Stimulus type, number of stimuli per block and number of associated inhibition-signals per block		Overall % of inhibition trials	Control group
Veling <i>et al.</i> (2013b)	1	80	80	Palatable foods 1 image each paired with 4, 12 and 24 no-go signals pairings; 1 image each paired with 4, 12 and 24 responses			Within-subjects design
Veling <i>et al.</i> (2014)			200	Food and drink 100 (50%) 100 (100%)	Filler (common objects) 100 (50%) 0 (0%)		50% inhibition to non-food images

Appendix 3: Stimuli used in the stop-signal training task in Study 2.

Chocolate
Images



Crisp
Images



Filler
Images



Appendix 4: Words used in the unipolar, SC-IATs, Studies 2 and 4.

Pleasant words	Unpleasant words	Neutral words
tasty	revolting	average
delicious	bad	moderate
great	nasty	adequate
heavenly	awful	general
outstanding	disgusting	undefined

Mean values	Pleasant	Unpleasant	Neutral	<i>p</i>
Length	8	6.67	7.83	0.53
Concreteness	311	308	408	
Familiarity	588	549.5	547.25	0.83
Imageability	390	387.5	331.75	0.74
Number of syllables	2.5	2.33	3.17	0.14
Brown verbal frequency	35.6	17.8	26	0.75
Kucera-Frances written frequency	123.83	36.2	157.4	0.62
Thorndike-Lorge written frequency	720.33	328.8	422.75	0.81

Appendix 5: Stimuli used in the stop-signal and go/no-go training tasks in Study 3.

Note. Foods presented with an asterisk were the foods presented in the snack buffet.

Unhealthy Foods

*



*



*



Healthy Foods

*



*



*



Filler Images



Appendix 6: Nutritional information and weights for the unhealthy and healthy foods presented in the snack phase in Study 3.

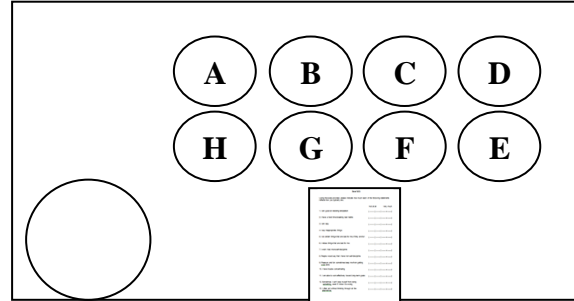
		Weight provided (g)	kCals per 100g	Fat per 100g
Unhealthy foods	Chocolate	~269	554	34.2
	- Cadbury 'Bitsa Wispa'			
	Biscuits (mini)	~158	484	21.4
	- Fox's mini malted milk biscuits			
	Crisps	~76	550	36.3
	- Tesco's ready salted crisps			
Healthy foods	Grapes	~387	70	0.1
	- green grapes			
	Carrot batons	~279	42	0.3
	- pre-cut carrots			
	Rice cakes (mini)	~57	388	3
	- Boots' organic plain rice cakes			
Novel unhealthy food	Cheese Bites	~172	536	29.2
	- ASDA's cheese bites			
Novel healthy food	Breadsticks (mini)	~110	413	7.4
	- ASDA's mini breadsticks			

Appendix 7: Pseudo-random orders for the presentation of unhealthy and healthy foods in the snack buffet for Study 3.

Note. For clarity the orders have been presented here in numerical order – these were randomised for presentation in the study.

Food codes

- 1 Chocolate
- 2 Carrots
- 3 Cheese bites
- 4 Bread sticks (mini)
- 5 Biscuits (mini)
- 6 Grapes
- 7 Crisps
- 8 Rice cakes (mini)



Schematic diagram of the buffet layout with 8 food bowls (A-H), questionnaires and a serving plate

A	B	C	D	E	F	G	H
1	2	3	7	4	6	8	5
1	2	4	8	3	5	6	7
1	2	6	7	3	5	4	8
1	2	7	8	6	5	3	4
1	3	7	5	4	6	8	2
1	6	4	2	7	8	3	5
1	7	2	5	4	6	8	3
1	7	6	3	5	8	4	2
1	8	5	4	6	3	2	7
2	1	5	4	3	7	8	6
2	3	6	1	5	4	7	8
2	3	6	5	8	7	1	4
2	4	5	6	8	1	7	3
2	4	6	1	5	3	8	7
2	5	6	4	8	3	1	7
2	6	8	5	1	4	7	3
2	8	3	6	7	4	5	1
3	4	2	6	7	8	5	1
3	5	7	2	4	8	6	1
3	6	4	7	2	1	5	8
3	8	1	6	7	5	4	2
3	8	2	6	4	5	1	7
3	8	2	6	5	7	4	1
4	3	1	7	2	5	8	6
4	3	5	7	1	8	6	2
4	3	7	8	6	5	2	1
5	2	1	7	3	8	4	6
5	2	7	1	3	6	8	4
5	3	2	6	7	8	4	1
5	3	4	1	6	2	8	7

A	B	C	D	E	F	G	H
5	3	6	1	2	8	7	4
5	3	8	4	2	1	6	7
5	4	2	3	1	8	7	6
5	6	3	1	2	4	8	7
5	6	4	3	1	8	7	2
5	6	8	2	7	3	4	1
5	7	3	2	6	8	4	1
5	8	6	7	3	2	1	4
6	1	3	2	8	4	7	5
6	2	1	4	3	7	5	8
6	2	3	4	7	8	1	5
6	3	5	8	1	4	7	2
6	3	7	5	2	8	1	4
6	5	2	4	8	3	1	7
6	5	8	3	7	2	1	4
6	7	1	4	8	5	3	2
6	8	7	4	1	3	5	2
7	1	2	5	8	6	3	4
7	1	5	2	3	4	8	6
7	1	6	8	4	5	3	2
7	2	1	8	6	5	4	3
7	3	4	2	1	5	8	6
7	4	1	5	8	3	6	2
7	6	1	4	3	5	2	8
7	6	4	5	3	1	8	2
8	1	2	7	3	5	4	6
8	2	4	5	6	7	3	1
8	2	7	1	3	5	4	6
8	3	7	1	4	2	6	5
8	4	3	5	1	6	2	7

Appendix 8: Food stimuli used in the go/no-go training tasks in Study 4.

Note. Filler stimuli in this task were identical to those used in Study 3.

Foods presented with an asterisk were also presented in the SC-IAT as ‘Old’ foods; see Appendix 11.

All food stimuli were also presented in the evaluation task. The evaluation task also included novel images – these images are presented in Appendix 12.

Unhealthy Foods

*



*



*



Healthy Foods



Appendix 9: Snack food stimuli used in the SC-IAT in Study 4.

'Old' Foods	'Novel' Foods
 A pile of broken, dark chocolate bars, showing the segmented texture of the chocolate.	 Two dark chocolate bars, one partially broken, showing the segmented texture.
 A stack of rectangular, golden-brown biscuits with a slightly textured surface.	 A stack of round, golden-brown biscuits with a slightly textured surface.
 A pile of golden-brown, ruffled potato chips.	 A pile of golden-brown, flat potato chips.

Appendix 10: ‘New’ snack food stimuli used in the Evaluation Task in Study 4.

Note. ‘Old’ snack food stimuli were identical to those presented in the training task; see Appendix 10.

Practice Images



Sweet ‘New’
Images



Savoury ‘New’
Images



Appendix 11. Study A1: Pilot study investigating the effect of combining inhibitory control training with prefrontal brain stimulation to reduce food consumption

The aim of this pilot study was to investigate the effect of prefrontal transcranial direct current stimulation (tDCS) combined with inhibition training on food consumption. Eight participants were included in the study, all of whom performed a food-related no-go training task while simultaneously receiving either active or sham tDCS to the dorsolateral prefrontal cortex (DLPFC), across two counterbalanced sessions. In the full experimental design the aim is to include both the inhibition and control training groups, thus enabling us to investigate whether combining tDCS and inhibition training has an additive effect on reducing consumption; furthermore, this design also allows for a replication of the effects found in Study 3 (see Chapter 4) in the sham condition. The pilot study presented in this chapter only included participants in the inhibition group to firstly ascertain whether tDCS can be used to augment the effect of inhibition training before adding a control group. Moreover, due to the within-subjects element of this design (sham and active tDCS), it was important to establish whether there were any effects of repeated sessions which may overshadow effects of tDCS. A within-subjects design was implemented to maximise statistical power. Although the aim is to determine sample size according to Bayes factors (which allows for a flexible stopping rule on data collection without correcting for the elevation of Type I error; Dienes, 2011, 2014; see section 3.2.4), power analyses (using G*Power; Faul *et al.*, 2007) were also performed to calculate a maximum sample size (the full study design is currently being prepared for pre-registration on the open-science framework). To achieve 90% power to detect a medium effect size (Cohen's $d_z=0.5$) using a two-tailed paired t-test (a comparison between sham and active tDCS conditions for the effect on overall food consumption; $\alpha=0.05$), a sample size of 44 is required for a within-subjects design whereas 172 participants would be necessary for a between-subjects design. Considering resources, it was felt that a within-subjects design for this study was more appropriate; however, the results from this pilot study indicate that repeated sessions may be problematic for this experimental design as it stands. These issues and possible solutions are discussed in the results and discussion.

A11.1. Introduction

In this study I investigated whether the application of transcranial direct current stimulation (tDCS) to the dorsolateral prefrontal cortex (DLPFC) could augment the effect of inhibitory control training on food consumption. Previous research has shown that one of the underlying mechanisms that may explain the effect of inhibition training on reduced food consumption is the devaluation of foods that are paired with inhibition (Veling *et al.*, 2013b). It is possible, therefore, that inhibition training may result in increased activity within areas of the prefrontal cortex that modulate value signals, such as the DLPFC (Hare *et al.*, 2009, 2011; Miller & Cohen, 2001; Weygandt *et al.*, 2013). For example, Hare *et al.* (2009) showed that increased activity in the DLPFC was associated with successful self-control when making food choices and was also found to down-regulate the goal value of unhealthy palatable foods (this was found to be a two-node network from the DLPFC to the VMPFC via the IFG). The authors speculated that the DLPFC is responsible for considerations of long-term goals and is able to modulate the short-term value signal; in a food context it was suggested that this is equivalent to considerations of the healthiness of foods while reducing the value attributed to the tastiness of foods. As the DLPFC has been shown to be involved in both self-regulated food intake (Del Parigi *et al.*, 2007; Hollmann *et al.*, 2012; Weygandt *et al.*, 2013) and response inhibition (Beeli *et al.*, 2008; Boggio *et al.*, 2007; Garavan *et al.*, 2006; Liddle *et al.*, 2001) it seems plausible that this region would be involved in food-related inhibition training.

Furthermore, activation of the DLPFC using brain stimulation techniques has previously been shown to reduce food craving and consumption (Claudino *et al.*, 2011; Fregni *et al.*, 2008; Goldman *et al.*, 2011; Montenegro *et al.*, 2012; Uher *et al.*, 2005; Van den Eynde *et al.*, 2010). Studies using repetitive transcranial magnetic stimulation (rTMS) have shown that while food craving tends to increase with food exposure in a sham group, participants receiving active stimulation to the DLPFC report either no change (Uher *et al.*, 2005) or a reduction in the strength of craving (Van den Eynde *et al.*, 2010). However, it has been argued that these effects may be due to the pain and discomfort experienced during active rTMS rather than the

stimulation per se (Barth *et al.*, 2011). Barth *et al.* (2011) found that when the perceived pain of stimulation was matched in the sham condition using scalp electrodes, the reduction in craving was present for both conditions. An alternative method of stimulation which is weaker than TMS, and is considered to involve a more reliable sham condition, is transcranial direct current stimulation (tDCS; Gandiga *et al.*, 2006; Nitsche *et al.*, 2008).

tDCS involves the delivery of a weak (typically 1-2mA) direct electrical current to the cortex via two scalp electrodes. The effect of tDCS on brain activity is dependent on the stimulation polarity; anodal stimulation is thought to increase cortical excitability by neuronal depolarisation whereas cathodal stimulation is believed to decrease excitability by hyperpolarising neurons (Antal *et al.*, 2007; Liebetanz *et al.*, 2002; Nitsche & Paulus, 2000; Nitsche *et al.*, 2003a; Nitsche *et al.*, 2005; Priori, 2003). In a within-subjects crossover design, Fregni *et al.* (2008) found that while a significant increase in food craving was observed with sham stimulation, bilateral tDCS to the DLPFC resulted in no increase in craving in the anodal left/ cathodal right condition, and a significant decrease in craving for the anodal right/ cathodal left condition. Active stimulation was also associated with a significant reduction in food consumption across both conditions; this reduction was non-significantly greater in the anodal right/ cathodal left condition. This finding was replicated by Goldman *et al.* (2011), who also demonstrated a significantly greater reduction in both food craving and an inability to resist food with active anodal right/ cathodal left stimulation compared to sham stimulation, although they found no difference in food consumption. Using unilateral anodal stimulation of left DLPFC (cathode over the contralateral supraorbital area) Montenegro *et al.* (2012) also found that active stimulation was associated with a reduced desire to eat, especially when combined with physical exercise.

It has also been argued that the effectiveness of tDCS may be improved with the addition of a cognitive target, such as inhibitory control (Alonso-Alonso, 2013; Wiers *et al.*, 2013). Not only is the DLPFC associated with the control of cravings, but it has also been linked to a range of executive functions including successful response inhibition (Beeli *et al.*, 2008; Boggio *et al.*, 2007; Garavan *et al.*, 2006;

Liddle *et al.*, 2001; MacDonald *et al.*, 2000; Wager *et al.*, 2005; Zheng *et al.*, 2008). For example, Beeli *et al.* (2008) demonstrated that cathodal tDCS of the right DLPFC (the anode was placed on the ipsilateral mastoid) increased the number of commission errors on a GNG task; however, they found no improvement in inhibitory control with unilateral anodal stimulation, although it is possible that this was due to a 'floor effect' (the mean number of commission errors was ~0.4). In a sample of depressed patients with a high mean error rate (32%), Boggio *et al.* (2007) found that anodal left DLPFC stimulation improved performance for an affective GNG task by approximately 23%. As tDCS is thought to increase synaptic plasticity and support learning (Stagg & Nitsche, 2011), applying tDCS in conjunction with a task designed to target response inhibition should improve behavioural performance. A recent study which paired stimulation of the inferior frontal gyrus (IFG), another area believed to be involved in response inhibition (Aron *et al.*, 2003, 2004, 2014; Chambers *et al.*, 2006, 2007; Chevrier *et al.*, 2007), with inhibition training using the SST found this combination to more effective at improving performance than just inhibition training alone (Ditye, Jacobson, Walsh & Lavidor, 2012). However, without a stimulation-only group, it is unclear whether this effect was due to the combination of tDCS and inhibition training or just an effect of tDCS.

In the current study food-related inhibition training was combined with tDCS to see whether the two interventions had a cumulative effect on decreasing food consumption compared to inhibition training alone. Participants performed a food-related inhibition task during either active or sham tDCS over two counterbalanced sessions. In accordance with the findings of Fregni *et al.* (2008), bilateral, anodal right/ cathodal left DLPFC stimulation was used. The methods used were as similar as possible to those in Study 3 with two adjustments. Firstly, a measure of state food craving was included before and after the training and tDCS to replicate previous findings demonstrating an effect of tDCS on reduced food craving (Fregni *et al.*, 2008; Goldman *et al.*, 2011; Montenegro *et al.*, 2012); furthermore, it is possible that a reduction in food craving may act as a potential mediator for any effect of tDCS and training on reduced food consumption. Secondly, as a manipulation check for the effect of tDCS on response inhibition, and to see whether any effect on food consumption is mediated by improvements in response inhibition, a second GNG

task at the end of the session was modified to reduce the likelihood of floor effects on this task. This was achieved by increasing the presentation speed and reducing the rate of inhibition to encourage rapid responding and increase the number of commission errors. The primary hypothesis was that participants would consume fewer unhealthy calories when no-go training was paired with active rather than sham tDCS. It was also hypothesised that the active tDCS condition would be associated with a smaller increase (or greater decrease) in food craving and a greater improvement in inhibitory control compared to the sham condition.

A11.2. Method

Figure A11.1 provides a schematic diagram of the procedure for each session (participants were also debriefed at the end of the second session; see section A11.2.3).

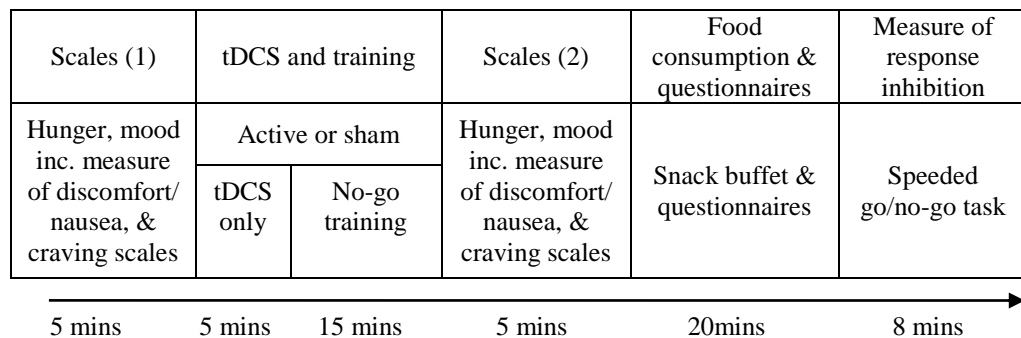


Figure A11.1. Schematic diagram of the study procedure. Participants answered state measures of hunger, mood and general food craving (section 3.2.3). They then received 20 minutes of bilateral tDCS to the DLPFC (right anodal/ left cathodal; see section A11.2.2.1); after five minutes of tDCS had elapsed, participants began the no-go training task (section A11.2.2.3). Following training, participants answered the state measures of hunger, mood and food craving for a second time before being taken to another room where they were presented with a snack buffet (section A11.2.2.4) and several personality questionnaires (section 3.2.2.4). Participants were left alone in this room for 20 minutes, they were then brought back to the original testing room to complete a speeded, food-related GNG task (see section A11.2.2.5).

A11.2.1. Participants

Eight participants (5 females, aged 19-44, $M=23.75$; $SE=2.94$) were recruited from University advertisements and a database of neurologically healthy participants who had already passed safety screening for brain stimulation techniques. To comply with our ethics for brain stimulation techniques, and to maintain a relatively homogenous sample, all participants were aged 18-45, were right-handed and reported no contraindications to tDCS/TMS safety (see Appendix 12 for the TMS/tDCS safety screening form). Participants were screened at least one week in advance for dietary restraint using the Restraint Scale (RS; Herman & Polivy, 1980) and were only considered eligible if they scored 15 or above ($M=17.25$; $SE=0.56$). In addition, participants had to report no history of eating disorders and no current intentions to lose weight through dieting. All participants were reimbursed for their time at a rate of £10 per hour. The study was approved by the School of Psychology Research Ethics Committee, Cardiff University.

A11.2.2. Materials/ Measures

A11.2.2.1. Transcranial Direct Current Stimulation (tDCS)

The tDCS protocol involved a within-subject crossover design, in which participants received either sham or active tDCS across two counterbalanced sessions. Two 7x5cm (35cm²), saline-soaked, sponge electrodes were positioned according to the international 10-20 EEG system, with the anode placed over the right DLPFC (F4) and the cathode over the left DLPFC (F3) to deliver bilateral stimulation (see Figure A11.2.). For the active condition a 2mA current was applied using a battery-driven constant-current stimulator (NeuroConn DC-STIMULATOR PLUS, neuroConn GmbH, Illmenau, Germany) for 20 minutes (with a 10sec ramp up and down). For the sham condition, the electrode montage was identical to the active condition; the stimulator delivered a 2mA current for 30 seconds before being slowly ramped down to 0mA over a 1 minute period. This initial period of stimulation was used to improve the likelihood of participants being blind to the tDCS condition (Gandiga *et al.*, 2006).

Although tDCS is a weaker form of stimulation compared to TMS, there are still a number of commonly reported side-effects including a mild tingling sensation, moderate fatigue and a light itching sensation. Headaches, nausea and insomnia have also been reported but in a minority of cases (11.8%, 2.9% and 0.98%, respectively; Poreisz *et al.*, 2007). In this study the occurrence of side-effects was assessed with post-monitoring forms. In the 24-hour period following tDCS participants were asked to indicate whether they experienced any incidence of seizure, fainting or collapse, dizziness, nausea or vomiting, headache, muscular aches, muscle spasm or twitches, insomnia, sensory problems, difficulties speaking or understanding speech, lack of coordination, slowness or impairment of thought or skin irritation including itching or pain.

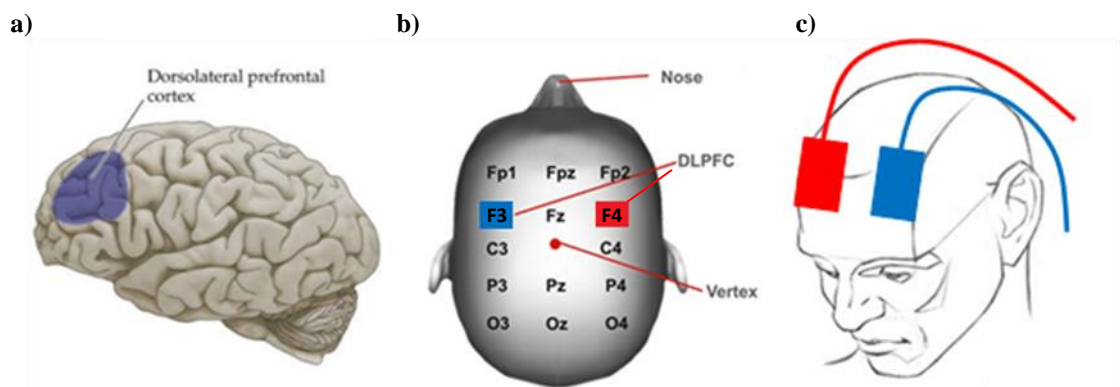


Figure A11.2. tDCS was used to bilaterally stimulate the DLPFC. The anode (red electrode) was placed over the right DLPFC and the cathode (blue electrode) over the left DLPFC using the international 10-20 EEG system (F4 and F3, respectively). Figure shows **a)** the cortical region of the DLPFC, **b)** the international 10-20 system scalp locations and **c)** the electrode montage used.

A11.2.2.2. Stimuli

As this study involved two sessions, the stimuli and snack foods used in Study 3 were divided into two sets. These were split so that there was one unhealthy-sweet, one unhealthy-savoury, one healthy-sweet and one healthy-savoury food in each training task and snack buffet (see Table A11.1; for stimuli presented during the

training task see Appendix 13; for nutritional information and weights provided see Appendix 14). Furthermore, the consumption data for each food from Study 3 was used as a guide so that there was no difference in expected calorie consumption across the two buffets. The food selection for each session was counterbalanced so that half the participants received food selection 1 for session 1 and food selection 2 for session 2, whereas the other half received them in the reverse order. We used two buffets to control for any effects of novelty or familiarity on food consumption.

Table A11.1. The selection of foods presented in the training task and snack buffet for each session. With the exception of the novel foods (which were only present in the snack buffet), all foods were presented as images in the training task as part of a wider category (in parentheses). For example, an image of carrot batons was presented in the training task along with other salad foods such as lettuce and cucumber.

	Food selection 1	Food selection 2
Unhealthy-sweet	Chocolate bites (chocolate)	Mini malted milk biscuits (biscuits)
Unhealthy-savoury	Cheese bites (cheese savouries)	Ready salted crisps (crisps)
Healthy-sweet	Carrot batons (salad vegetables)	Grapes (fruit)
Healthy-savoury	Mini rice cakes (rice cakes/ crackers)	Mini breadsticks (breadsticks)
Unhealthy novel food (buffet only)	Flapjack bites	Victoria sponge cake bites

A11.2.2.3. Go/No-Go Training

All tasks were programmed in Matlab (Mathworks, Natick, MA) using Psychophysics Toolbox (www.psychtoolbox.org) and all stimuli were presented on a 19-inch flat-panel LCD monitor.

The GNG training task consisted of eight blocks of 40 trials (320 trials in total) and lasted approximately 15 minutes with a 15 second break between each block. Each block randomly presented ten images of unhealthy foods (five each of chocolate and cheese savouries in selection 1 and five each of biscuits and crisps in selection 2), ten

images of healthy foods (five each of salad foods and rice cakes in selection 1 and five each of fruit and breadsticks in selection 2) and 20 filler images of clothes (three each of jeans, shirts, jumpers, socks, skirts and ties, plus two blouses that were added from the original task to increase the task length to 15 minutes). One image for each food type corresponded to the food that was presented in the snack buffet. All images were carefully selected so that there were no additional ingredients or packaging, and they were approximately equal in size and visual complexity (for images of the stimuli used see Appendix 13).

With the exception of changes to the stimuli, the task was identical to that used in Study 3 (see Figure 4.2b.ii). Each trial began with the presentation of a central rectangle (fixation; 1250ms). A stimulus was then presented within this rectangle randomly, and with equal probability, to either the left or right hand side. Participants were required to respond to the location of the stimulus as quickly and accurately as possible using their left and right index fingers (using the ‘C’ and ‘M’ keys, respectively). All unhealthy food images (10/10; 100% mapping) and half of the filler images (10/20; 50% mapping) were presented alongside a no-go signal (the fixation rectangle was bold for the duration of the trial), indicating that the participant must withhold their response for that trial. All healthy foods required a location response (0% signal mapping).

A11.2.2.4. Snack Buffet

Following training participants were taken to another testing room and presented with five bowls of food (see Table A11.1) and a jug of water. They were instructed to consume as much food as they liked, as long as they did not feel hungry when the experimenter returned after 20 minutes. They were also asked to fill out a series of questionnaires to keep them occupied during the full 20 minutes (The Big Five Inventory, the Brief Self Control Scale, the Emotion Regulation Questionnaire, the UPPS impulsive behaviour scale, the Attentional Control Questionnaire and the Mood and Anxiety Symptom Questionnaire; see section 3.2.2.4 for full details). The presentation of foods was pseudo-randomised to minimise the effect of proximity on food intake (see Appendix 15). Random sequences in which both the healthy foods and both the unhealthy foods were placed together were eliminated to minimise

participants' awareness of the food categories. Unknown to the participant, all foods were weighed before and after the consumption phase and the difference in weight was multiplied by the food's caloric density to determine food intake.

A11.2.2.5. Speeded Go/No-go Task

In order to assess whether active tDCS had any effect on response inhibition the second GNG task was modified by decreasing the presentation time and rate of inhibition trials. The commission error rate (the percentage of erroneous responses made on no-go trials) on the training task is typically very low (~5%) making it difficult to detect any potential improvements in inhibitory control. In this speeded GNG task, we therefore reduced the presentation time of each stimulus and ITI from 1250ms to 500ms (see Collins & Mullan, 2011) and decreased the percentage of no-go trials from 50% to 33.3%. It was believed that these changes would encourage rapid responding, which, as a result of the speed-accuracy trade-off would also increase the rate of commission errors. A pilot study ($N=13$) with non-food images (e.g. household items including electrical goods and furniture) provided data to support this with a mean commission error rate of 24.44% ($SE=3.53\%$).

The speeded GNG task consisted of eight blocks of 45 trials (a total of 360 trials) and lasted approximately eight minutes with a 15 second break between each block. Each block randomly presented nine unhealthy-old foods (chocolate, crisps and biscuits), nine healthy-old foods (salad vegetables, fruit and rice cakes), nine unhealthy-new foods (pizza, pancakes, deserts etc.) and 18 filler images (clothes). Three images for each food category and six filler images were presented alongside a no-go signal (33.3% mapping).

A11.2.3. Procedure

All participants were informed that they were taking part in a study measuring the effect of blood glucose levels on cognitive performance. They were therefore asked not to eat for three hours prior to the study to ensure that their glucose levels were relatively low. During the study they were informed that they would be given something to eat after the first task so that we could measure performance again when glucose levels were expected to increase. The true purpose for this limit on

prior food intake was to control for levels of hunger (Gibson & Desmond, 1999; Guerrieri *et al.*, 2009; Lawrence *et al.*, under review; Veling *et al.*, 2013a, 2013b). This combined with the cover story was also to encourage participants to eat during the food consumption phase. All testing sessions were therefore in the afternoon/early evening (from 12pm-7.30pm). For tDCS safety reasons participants were also asked to refrain from alcohol and drugs for 12 hours before the study and from caffeine in the preceding two hours (no more than two cups of tea or coffee).

On entering the lab, participants were given a verbal introduction to the methodology and safety aspects of tDCS. Provided they passed safety screening and gave their consent, they were asked to complete scales measuring their hunger (three 100mm VAS scales measuring hunger, fullness and desired to eat) and mood (PANAS; Watson *et al.*, 1988; see section 3.2.3 for full details). Two additional items were added to the PANAS to measure discomfort/pain and nausea. These measures were taken before and after tDCS to rule out any differences in food consumption due to these potential influences of tDCS (see Barth *et al.*, 2011). They also completed a *state* measure of general food craving (G-FCQ-S, Cepeda-Benito *et al.*, 2000; Nijs *et al.*, 2007). The G-FCQ-S is a 15 item questionnaire which measures the extent to which an individual is experiencing momentary food cravings. Participants are asked to rate how strongly they agree with the items “*right now, at this very moment*” on a five point scale from 1 “strongly disagree” to 5 “strongly agree”. There are five subscales: an intense desire to eat (three questions), anticipation of relief from negative states and feelings as a result of eating (three questions), craving as a physiological state (i.e., hunger; three questions), obsessive preoccupation with food or lack of control over eating (three questions) and anticipation of positive reinforcement that may result from eating (three questions).

Participants then received bilateral DLPFC stimulation for five minutes in isolation before starting the training task for a further 15 minutes. Following training participants completed the scales for hunger, mood and food craving once more before being taken to the snack buffet and questionnaires. After 20 minutes they were brought back to the original testing room to complete the speeded GNG task. At the end of each session participants were asked at what time they last ate and

were probed for awareness of the tDCS condition. To reduce the likelihood of answers based on comparisons between sessions, participants were told that the stimulation could be active left, active right, active on both sides or sham tDCS. They were then asked if they thought they received active or sham tDCS and if active whether they believed it was on the right side, left side or both sides. At the end of the second session participants were probed for awareness of the study's aims and their height and weight was recorded to calculate BMI (kg/m^2).

A11.2.4. Statistical Analysis

Initial scores of hunger (VAS measures) and mood (PANAS) as well as hours since food consumption were analysed using paired t-tests to ensure that there were no statistically significant differences between these measures for each tDCS session. Differences in pre- and post- tDCS/training measures of hunger and mood, including measures of nausea and discomfort were also analysed to ensure that these variables did not explain any differences in food consumption or craving between tDCS conditions. In addition, the validity of the sham condition was explored to rule out the possibility of demand characteristics.

Data from the training tasks were also analysed to ensure that participants performed the tasks as expected in both sessions. Exclusions were made if participants' performance on no-signal trials was below 85% accuracy (errors included incorrect locations and missed responses), if their mean reaction time for no-signal trials (GoRT) was $>3\text{SDs}$ from the mean for that tDCS condition or if their commission error rate (failed inhibition trials) on signal trials was $>3\text{SDs}$ from the condition mean. One participant was excluded from the analysis based on the commission error rate in the sham condition; this resulted in a final sample of seven participants (4 female).

A measure of general, momentary food craving was measured using the G-FCQ-S (Cepeda-Benito *et al.*, 2000; Nijs *et al.*, 2007) before and after tDCS and inhibition training. The G-FCQ-S includes five subscales, however, as these scales are highly inter-correlated and the total scale has good internal consistency (Meule *et al.*, 2012b), only the total scale was used. The effect of tDCS and inhibition training was

analysed with a within-subjects 2x2 ANOVA with factors *tDCS condition* (active or sham) and *time* (pre- or post-tDCS/training).

Food consumption data was first checked for statistical outliers. Outliers were examined for each food type and for each tDCS condition separately (e.g. chocolate in the active condition, crisps in the sham condition etc.). Values were considered as outliers if they exceeded 3SDs from the mean and were replaced with the highest non-outlier value for that food and condition +1 (Tabachnick & Fidell, 2007).

Calorie intake was analysed using a within-subjects 2x3 ANOVA with factors *tDCS condition* (active or sham) and *food* (unhealthy-old food, healthy old food and unhealthy-new food).

As a manipulation check for the effect of tDCS on inhibitory control, and to see whether any effects on food consumption were mediated by improvements in inhibitory control, performance was measured on a speeded, food-related GNG task. The rate of commission errors for foods and filler stimuli were analysed using a within-subjects 2x4 ANOVA with factors *tDCS condition* (active or sham) and *stimulus* (unhealthy-old food, healthy old food, unhealthy-new food and filler images).

As the analyses in this pilot study were exploratory, all results are interpreted without corrections for multiple comparisons and all unadjusted significance values are presented. All analyses were carried out using SPSS.

A11.3. Results

A11.3.1. Adverse Effects and Blinding Check

Participants generally responded well to tDCS. Only one minor adverse effect was reported which was a mild twitching of the jaw during stimulation (this was during the active stimulation). Furthermore, no participants reported an increase in pain/discomfort from pre- to post-tDCS for either the sham or active conditions and only one participant reported an increase (+1) in nausea which was reported following sham tDCS. Participants also appeared blind to the tDCS conditions. For

the sham condition only one participant correctly guessed that it was sham tDCS, one participant answered active left, one answered active right and the remaining five participants believed they received stimulation on both sides. The responses for active-both were therefore above chance and the alternative answers, including sham, fell below chance. For the active condition all participants correctly guessed that it was active, two thought it was on the left hand side, five thought it was on the right and one participant was not sure.

A11.3.2. Between-Session Differences

There were no statistically significant differences between the sham and active tDCS conditions for hours since consumption ($t(6)=0.83$, $p=0.44$, $d_z=0.32$) and pre-tDCS/training measures of hunger (all $t_s<1.02$, all $p_s>0.36$, all $d_z<0.42$) or mood (all $t_s<1.48$, all $p_s>0.24$, all $d_z<0.74$; see Table A11.2).

Table A11.2. Between-session differences for hours since food consumption, pre-tDCS/training hunger scores (VAS) and pre-tDCS/training positive and negative affect scores (PANAS), per tDCS condition (SE within parentheses).

	Sham	Active	$t =$	$p =$
Hours since food	4.96 (1.04)	5.64 (1.76)	0.83	0.44
Hunger ¹	5.1 (0.58)	4.32 (1.06)	0.65	0.55
Fullness ¹	0.93 (0.26)	1.92 (0.99)	1.02	0.36
Desire to eat ¹	5.65 (0.38)	4.28 (1.24)	0.94	0.39
Positive affect ³	26.5 (4.92)	30.5 (5.89)	1.26	0.3
Negative affect ³	10.25 (0.25)	12.25 (1.6)	1.48	0.24

Note. Superscript denotes the number of participants missing for that variable.

In addition, there were no statistically significant interactions between tDCS condition and time (pre- and post-tDCS/training) for measures of hunger, fullness or desire to eat (all $F_s<1.48$, all $p_s>0.28$, all $d_z<0.23$; see Figure A11.3 a-c.; one participant was missing from this analysis for not completing the VAS scales prior to tDCS/training). There were, however, significant main effects of hunger ($F(1,6)=54.95$, $p=0.001$, $\eta^2_p=0.92$) and desire to eat ($F(1,63)=76.34$, $p<0.001$,

$\eta^2_p=0.94$) which both increased following tDCS/training. This effect is likely to be due to exposure to images of palatable foods. The main effect for the decrease in fullness was not statistically significant ($F(1,6)=3.32, p=0.13, \eta^2_p=0.4$), although it is possible that this was due to a floor effect after participants were asked not to eat for three hours prior to the study.

There were also no statistically significant interactions between tDCS condition and time (pre- and post-tDCS/training) for measures of either positive ($F(1,3)=0.62, p=0.49, \eta^2_p=0.17$) or negative affect ($F(1,3)=3.00, p=0.18, \eta^2_p=0.5$; see Figure A11.3 d,e; three participants were missing from this analysis for failing to complete the PANAS prior to tDCS/training). There was, however, a significant main effect of time for positive affect ($F(1,3)=18.55, p=0.02, \eta^2_p=0.86$) with participants reporting a decrease in positive affect for both conditions after tDCS/training. The main effect of time for negative affect was not statistically significant ($F(1,3)<0.001, p=1.0, \eta^2_p<0.001$), indicating that tDCS/training reduced positive affect but did not increase negative affect¹⁵.

¹⁵ Correlations between these difference measures for state hunger and mood measures (pre tDCS/training measures were subtracted from post tDCS/training measures) and measures of calorie intake, for unhealthy-old, healthy and unhealthy-new foods, were explored to see whether these differences could explain consumption (see Appendix 16). Generally, these relationships were inconsistent with previous research which has shown positive associations between intake and measures of hunger and negative affect (Nederkoorn *et al.*, 2009a; Tice *et al.*, 2001). With such a small sample it is likely that these relationships are driven by one or two participants and are not reliable – nevertheless, they will be of interest in the final study.

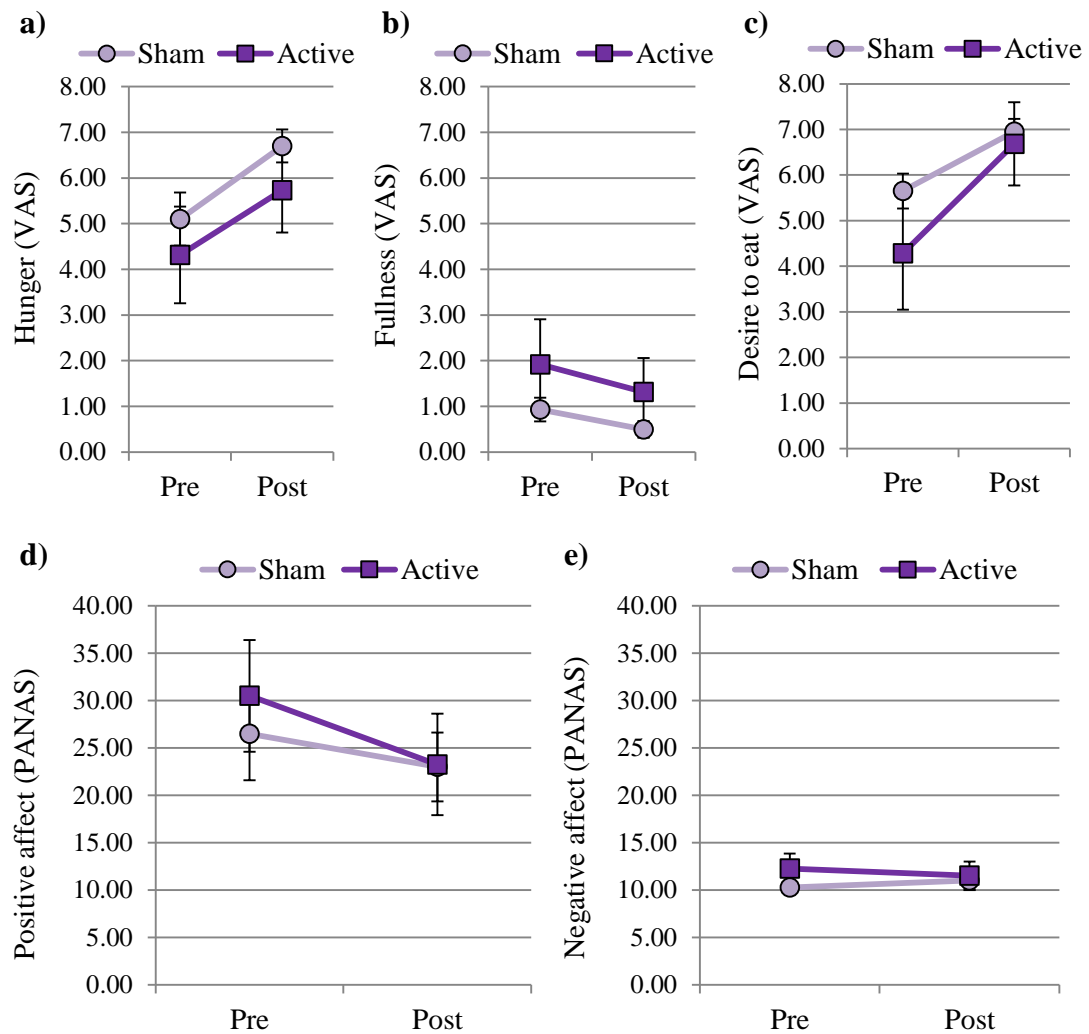


Figure A11.3. Mean pre- and post tDCS/training measures of hunger and mood, using the VAS scales for **a)** hunger, **b)** fullness and **c)** desire to eat, and the PANAS for positive **(d)** and negative **(e)** affect.

A11.3.3. Training Data Analysis

Data for the mean percentage of incorrect responses on signal trials (commission errors), mean reaction time for correct no-signal responses (GoRT) and mean percentage of incorrect no-signal responses are presented in Figure A11.4. Data for incorrect signal and incorrect no-signal responses were analysed using Wilcoxon signed-rank tests as this data was not normally distributed and could not be normalised with a square root or log transformation; the GoRT data was analysed with a paired t-test. The results revealed that participants made fewer commission errors on signal trials in the active condition ($M=3.3$, $SE=0.76$) compared to the

sham condition ($M=6.34$, $SE=1.57$; $Z=2.06$, $p=0.04$, $r=0.78$). However, there was no statistically significant difference in performance between conditions for the no-signal trials; there was no difference in either GoRT ($t(6)=0.96$, $p=0.34$, $d_z=0.36$) or the percentage of incorrect no-signal trials ($Z=0.54$, $p=0.59$, $r=0.22$).

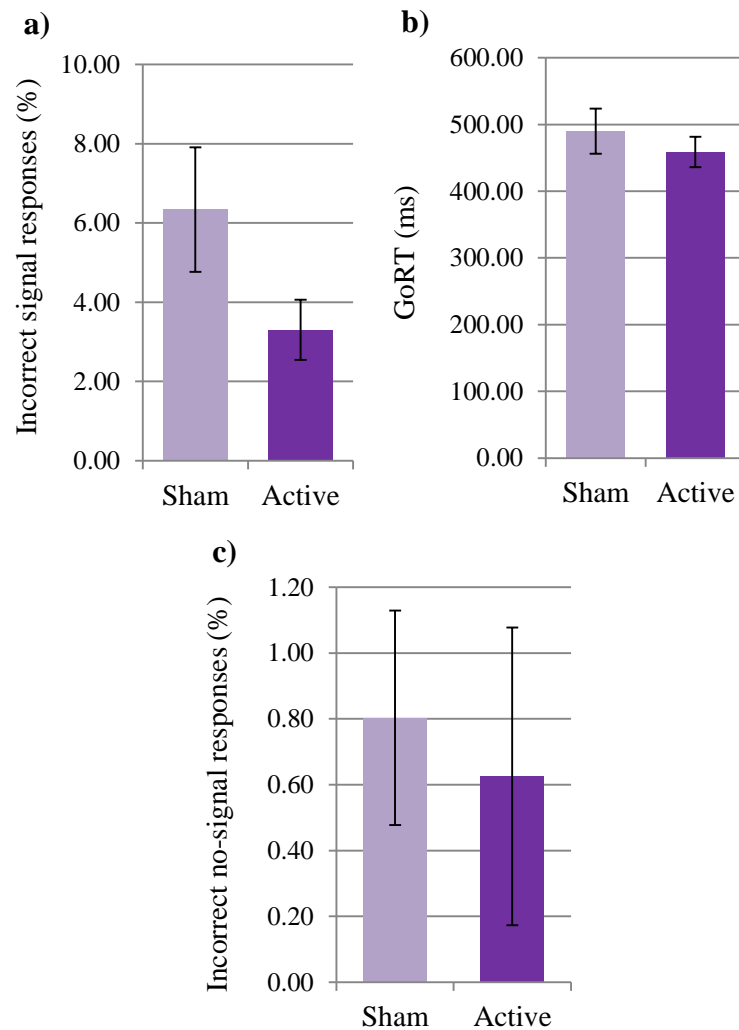


Figure A11.4. Training data for the sham and active conditions showing the mean percentage of commission errors **a)**, the mean GoRT **b)**, and the mean percentage of incorrect no-signal responses **c)**. Error bars show ± 1 SE.

A11.3.4. Consumption Data Analysis

The results for food intake revealed a statistically significant difference for the main effect of food ($F(2,12)=12.43$, $p=0.001$, $\eta^2_p=0.67$) reflecting a greater consumption

of calories for the unhealthy-old foods ($M=391.42$, $SE=81.75$) compared to the healthy foods ($M=70.47$, $SE=9.72$; $p=0.01$) and a greater consumption of the unhealthy-new food ($M=295.15$, $SE=47.61$) compared to the healthy foods ($p=0.01$). There was no statistically significant difference between the consumption of unhealthy-old and unhealthy-new foods ($p=0.74$; see Figure A11.5). There was also no statistically significant main effect of tDCS ($F(1,6)=0.06$, $p=0.82$, $\eta^2_p=0.01$) or interaction between tDCS condition and food type ($F(1.16, 6.96)=0.24$, $p=0.68$, $\eta^2_p=0.04$, with Greenhouse-Geisser correction for non-sphericity (Mauchley's test: $\chi^2(2)=6.44$, $p=0.04$)). A paired t-test also showed that there was no statistically significant difference in the consumption of healthy food intake in grams ($t(6)=0.9$, $p=0.4$, $d_z=0.34$). Although these differences were not statistically significant with this sample, the direction of effect was in the expected direction for total calorie intake; participants consumed fewer calories after receiving active tDCS ($M=733.65$, $SE=466.65$) compared to the when they received sham tDCS ($M=780.43$, $SE=130.95$). However, this effect appears to be primarily due to a reduced consumption of healthy foods in the active condition.

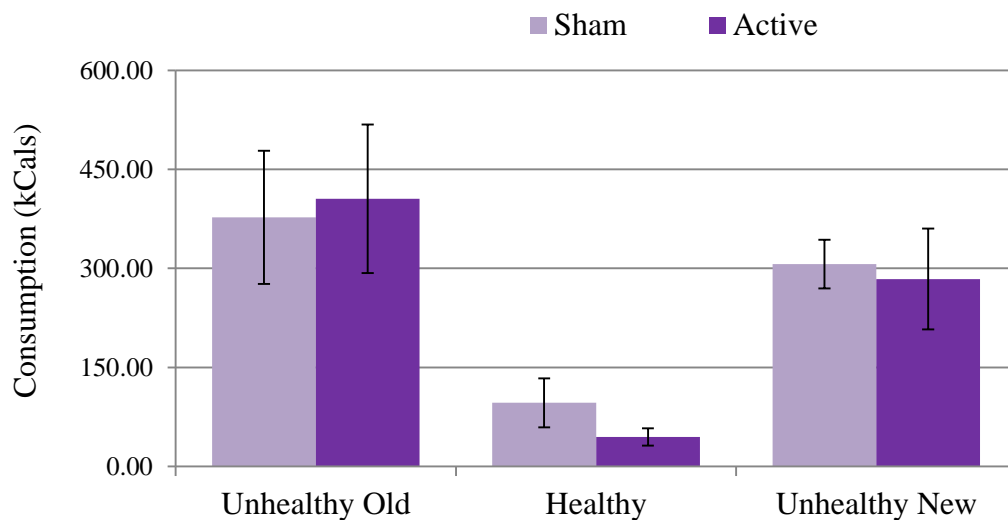


Figure A11.5. Calorie consumption as a function of tDCS condition for the unhealthy-old and healthy foods that were presented in both the training and the snack buffet and the unhealthy-new food. Error bars show ± 1 SE.

A11.3.5. Craving Data Analysis

For state food craving there was a trend towards a main effect of time, with participants reporting a greater level of craving following the tDCS/training ($M=50.67$, $SE=5.02$) than before ($M=44.83$, $SE=5.69$; $F(1,5)=6.16$, $p=0.06$, $\eta^2_p=0.55$; see Figure A11.6). This is consistent with the significant increase in levels of hunger and desire to eat mentioned earlier (see A11.3.2) and is likely to be due to exposure to palatable food images. However, there was no statistically significant main effect of tDCS ($F(1,5)=0.96$, $p=0.37$, $\eta^2_p=0.16$) or interaction between tDCS and time ($F(1,5)=0.41$, $p=0.55$, $\eta^2_p=0.08$). Although the interaction term was not statistically significant, the results were in the expected direction with a greater pre to post increase in craving for the sham condition (from $M=46.17$ ($SE=4.03$) to $M=53.19$ ($SE=3.37$)) compared to the active condition (from $M=43.5$ ($SE=7.69$) to $M=48.17$ ($SE=6.84$)). Moreover, inspection of the pairwise comparisons for the interaction term revealed that this was a significant increase in the sham condition ($p=0.01$) but not the active condition ($p=0.27$).

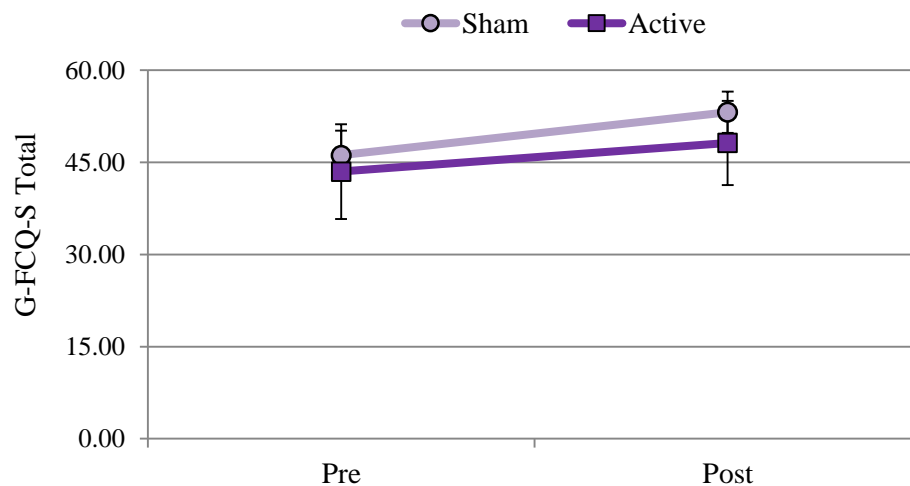


Figure A11.6. Mean state food craving score for the sham and active tDCS conditions, measured before (pre) and after (post) the tDCS and training task. Error bars show ± 1 SE.

Correlations between the difference in food craving (pre tDCS/training G-FCQ-S scores were subtracted from post tDCS/training G-FCQ-S scores) and measures of

calorie intake, for unhealthy-old, healthy and unhealthy-new foods, were explored to see whether these differences could explain consumption (see Appendix 17). Neither the sham (all $r_s < 0.72$, all $p_s > 0.11$) nor the active condition (all $r_s < -0.53$, all $p_s > 0.22$) revealed any statistically significant correlations. However, for the sham condition all correlations were positive between increased craving and intake with a large effect size for the correlation with unhealthy-new food intake ($r=0.72$, $p=0.11$) and a medium effect for healthy food intake ($r=0.37$, $p=0.48$). For the active condition all measures of food intake were negatively correlated with craving with medium-large effect sizes (unhealthy-old: $r=-0.46$, $p=0.29$; unhealthy-new: $r=-0.53$, $p=0.22$; healthy: $r=0.51$, $p=0.24$).

A11.3.6. Speeded GNG Data Analysis

Response inhibition was measured on the speeded GNG task as the percentage of commission errors (the percentage of signal trials in which participants failed to withhold a response). The results from this analysis showed that neither the main effect of tDCS ($F(1,6)=2.85$, $p=0.14$, $\eta^2_p=0.32$) nor the interaction between tDCS and stimulus type ($F(1,6)=1.41$, $p=0.27$, $\eta^2_p=0.19$) were statistically significant. There was, however, a significant main effect of stimulus type ($F(1,6)=5.46$, $p=0.008$, $\eta^2_p=0.48$). Pairwise comparisons showed statistical trends for a lower error rate for unhealthy novel foods ($M=10.42$, $SE=2.27$) compared to both healthy foods ($M=16.07$, $SE=3.0$, $p=0.09$) and filler stimuli ($M=15.33$, $SE=2.03$, $p=0.05$). This unexpected finding, for improved inhibitory control for untrained stimuli may reflect an effect of increased arousal (Pessoa, Padmala, Kenzer & Bauer, 2012). Although the interaction term was not statistically significant, inspection of the pairwise comparisons for this analysis revealed that the differences between stimulus types were only significant in the sham condition and not the active condition (all $p_s > 0.23$). For the sham condition, the error rate for unhealthy-new stimuli was significantly lower than for unhealthy-old stimuli ($p=0.02$), healthy-old stimuli ($p=0.006$) and filler stimuli ($p=0.002$). Visual inspection of the data (see Figure A11.7a) indicates that inhibitory performance was the expected direction; there were fewer commission errors in the active compared to the sham condition for all stimuli that were presented during training but not the novel stimuli. There was no statistically significant difference between tDCS conditions for performance on the

no-signal trials for either the GoRT ($t(6)=0.5$, $p=0.63$, $d_z=0.19$; see Figure A11.7b) or percentage of incorrect responses ($t(6)=0.06$, $p=0.96$, $d_z=0.02$; see Figure A11.7c).

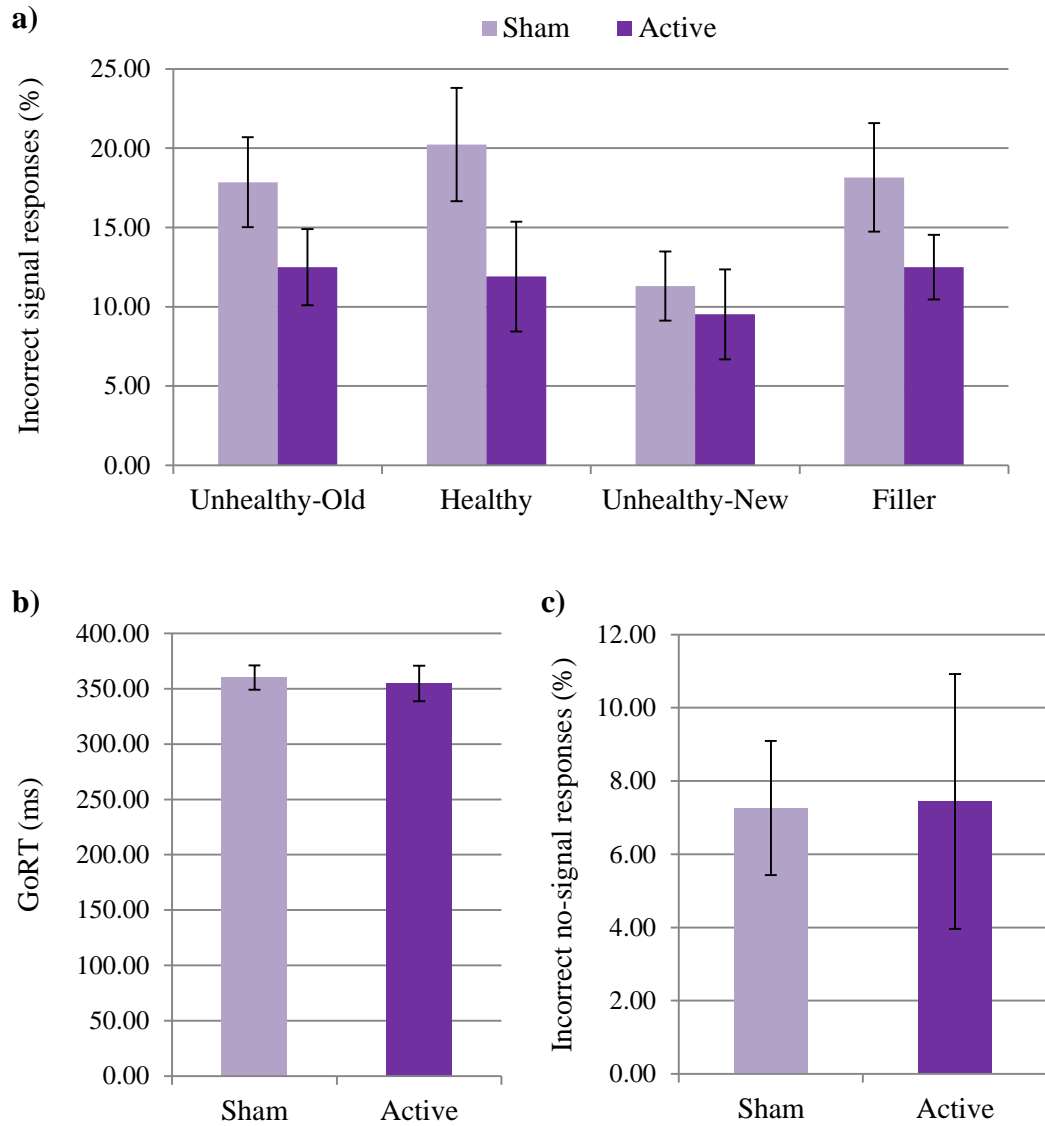


Figure A11.7. Mean performance on the speeded GNG task for **a)** the percentage of incorrect responses on signal trials according to different stimuli types, **b)** the reaction time on no-signal trials (GoRT) and **c)** the percentage of incorrect responses on no-signal trials.

A11.3.7. Consumption Data Analysis According to Session and Buffet Selection

Calorie consumption for the different food types (unhealthy-old, healthy, unhealthy-new) was analysed according to the session (first or second) and buffet selection (see Table A11.1) to explore whether either of these variables were influencing food intake. Firstly, it is important in this within-subjects design to rule out the possibility that returning to the lab for a second time will have the greatest effect on food intake. For example, feeling more comfortable eating in such an environment could result in increased consumption in the second session regardless of the tDCS condition, whereas a greater sense of novelty in the first session could have the opposite effect. A 2x3 within-subjects ANOVA (*session*: first or second; *food*: unhealthy-old, healthy, unhealthy-new) revealed no statistically significant main effect for session ($F(1,6)=2.03, p=0.2, \eta^2_p=0.25$) nor a statistically significant interaction between session and food type ($F(2,12)=0.94, p=0.42, \eta^2_p=0.14$).

Secondly, it was important to explore whether there were any significant differences in intake according to the selection of foods. A 2x3 within-subjects ANOVA (*buffet selection*: one or two; *food*: unhealthy-old, healthy, unhealthy-new) revealed no statistically significant main effect of buffet ($F(1,6)=2.83, p=0.14, \eta^2_p=0.32$); however, there was a statistically significant interaction between buffet and food type ($F(2,12)=9.5, p=0.003, \eta^2_p=0.61$). Pairwise comparisons showed that participants consumed significantly more unhealthy-old calories in buffet selection 1 ($M=514.65, SE=109.02$) compared to buffet selection 2 ($M=268.19, SE=77.37; p=0.04$). There was also a trend towards significance for the healthy foods ($p=0.06$) with participants consuming more healthy calories from buffet selection 2 ($M=108.11, SE=33.87$) compared to buffet selection 1 ($M=32.82, SE=12.09$). The difference in unhealthy-new foods was not statistically significant ($p=0.17$). Exploring the calorie intake for the individual foods showed that these differences appear to reflect a greater preference for the chocolate in buffet 1 ($M=364.57, SE=89.8$) compared to the biscuits in buffet 2 ($M=103.19, SE=35.89$) and a greater preference for the grapes in buffet 2 ($M=49.11, SE=13.14$) compared to the carrots in buffet 1 ($M=10.1, SE=5.12$).

A11.3.8. Speeded GNG Data Analysis According to Session

The influence of session on commission errors in the speeded GNG task was also explored. As more participants received sham stimulation in the first session followed by active stimulation in the second session (5 participants), than vice versa (2 participants following 1 exclusion), it is possible that the effects reported above (section A11.3.6.) are due to practice effects rather than an effect of tDCS. Indeed, a 2x4 within-subjects ANOVA (*session*: first or second; *stimulus*: unhealthy-old, healthy, unhealthy-new, filler) revealed a significant main effect of session ($F(1,6)=6.15, p=0.048, \eta^2_p=0.51$) with a higher commission error rate in the first ($M=17.56, SE=2.97$) compared to the second session ($M=10.94, SE=1.7$). There was no statistically significant interaction between session and stimulus type ($F(3,18)=1.89, p=0.17, \eta^2_p=0.24$).

A11.3.9. Debrief Analysis

During debrief, no participants correctly guessed the aim of the study or mentioned an awareness that food intake was being measured. One participant reported that the task made them hungrier which reflects the results of the pre- and post tDCS/training VAS scores for hunger. Two participants noticed that the unhealthy foods were associated with inhibition signals compared to five participants who were not aware of any associations between the stimuli and inhibition signals.

A11.4. Discussion

The aim of this pilot study was to see whether stimulation of the prefrontal cortex could be used to augment the effect of food-related inhibition training on food consumption. Over two counterbalanced sessions, participants received either sham or active bilateral stimulation of the DLPFC using tDCS (anodal right/ cathodal left), whilst also completing a food-related no-go training task. Measures of state food craving, food consumption and food-related inhibitory control were recorded. The results for food craving were not statistically significant but were in the expected direction with participants reporting a greater increase in food craving following training in the sham compared to the active condition. This finding is consistent with previous research showing both a smaller increase in craving and a greater decrease

in craving following active compared to sham stimulation of the DLPFC (Fregni *et al.*, 2008; Goldman *et al.*, 2011; Montenegro *et al.*, 2012; Van den Eynde *et al.*, 2010; Uher *et al.*, 2005). A similar result was also found for food consumption; although the difference in intake between conditions was not statistically significant, as expected, participants consumed fewer calories in the active compared to the sham condition. Again these results are consistent with previous findings showing an effect of active DLPFC stimulation on reduced food intake compared to sham stimulation (Fregni *et al.*, 2008). However, this difference was due to decreased consumption of the healthy and unhealthy-new foods, and not the unhealthy-old foods that were paired with inhibition during training.

Results for food-related response inhibition on the speeded GNG task also showed some evidence to suggest that participants were better able to inhibit their responses towards foods previously presented during training in the active compared to the sham condition. However, statistical analysis demonstrated that inhibitory control was more efficient for the novel unhealthy foods that were only presented in this speeded GNG task compared to the unhealthy foods that were always presented alongside no-go signals during training. This result is inconsistent with previous studies demonstrating that repeatedly pairing a stimulus with stopping improves the ability to inhibit responses to that stimulus on future trials (Verbruggen & Logan, 2008; see also Chiu *et al.*, 2012; Lenartowicz *et al.*, 2011). It is possible that this result reflects increased arousal for the novel unhealthy stimuli (Possoa *et al.*, 2012); however, there were also novel exemplars that were included in the unhealthy-old and unhealthy-new food categories. Another possible explanation for this finding is the ambiguous nature of some of the foods presented in the unhealthy-new category; for example, three desserts were presented in this category which all included fruit. It is possible, therefore, that participants interpreted these foods as unhealthy, healthy or neutral. This limitation should be addressed in future research with tighter control over the content of these images.

Although these results provide some indication that active stimulation of the DLPFC may be useful for increasing food-related self-control when combined with inhibition training, analyses of between-session and buffet selection effects indicate further

limitations of the within-subjects design in this study. Firstly, further analysis of the results for food consumption indicated that participants consumed more unhealthy-calories from one buffet selection and more healthy calories from the other. Although the buffet selections are counterbalanced across sessions and tDCS conditions, the greater effect size for this comparison compared to that of the tDCS condition indicates that this effect of buffet selection may obscure any effect of tDCS on food intake. For future research this limitation could be rectified by ensuring that the foods in each category and for each buffet are more closely matched for palatability and/ or desirability. Once this limitation is addressed it is also important to ensure that there are no statistically significant effects of session on food intake, which may also obscure any effects of tDCS. Although there was no evidence for an effect of session on intake in this sample, it is possible that any underlying effect may have been overshadowed by the effect of buffet selection.

A significant effect of session was found, however, for the analysis of inhibitory control performance on the speeded GNG task, which showed an improvement in performance on the second compared to the first session. As more participants received active stimulation in the second session than the first, it is possible that this practice effect also explains the earlier mentioned results indicating a reduced commission error rate as a result of active, compared to sham, tDCS. In future, it may be possible to correct for this by either providing participants with an initial training session in which performance can be calibrated, or by including additional practice blocks in this task which can be excluded from the final analysis. However, these solutions do not control for the possibility that response inhibition will benefit from all additional training sessions or that effects of tDCS on performance will be greatest at the beginning of the GNG task. A within-subjects design was employed here to maximise power and was in accordance with previous research (Fregni *et al.*, 2008; Goldman *et al.*, 2011), however, the findings from this pilot study indicate that repeated sessions in this type of study are problematic. To investigate more effectively whether prefrontal stimulation can be used to boost the effect of inhibitory control training on reduced food consumption, the above methodological issues will need to be addressed. Moreover, the sample size will also need to be increased to achieve enough statistical power to detect these effects if they do exist;

as discussed earlier, data collection for the final study will be guided with Bayesian analyses unless a maximum sample of forty four participants is reached (based on 90% power for a medium effect size).

Over the last decade there has been a surge of interest in tDCS as a potential tool for neuro-enhancement (Dubljević, Saigle & Racine, 2014). Compared to TMS, tDCS is safer, cheaper and easier to use – so much so that the device is portable and can be used at home by participants and patients. These advantages are coupled with exciting findings which indicate that tDCS can be used to improve performance across an array of cognitive tasks in both healthy and clinical populations (for a review see Sarkis, Kaur & Camprodon, 2014) – although, there are concerns regarding the limited mechanistic understanding of tDCS and whether these improvements in executive function can be considered genuine (Bestmann, de Berker & Bonaiuto, under review; Sarkis *et al.*, 2014). tDCS has also been endorsed as a potential tool for the treatment of addiction (Feil & Zangen, 2010; Jansen *et al.*, 2013; Nardone *et al.*, 2012). Firstly, it has been shown to improve inhibitory control when stimulating the prefrontal cortex (Boggio *et al.*, 2007; Ditye *et al.*, 2012; Jacobson, Javitt & Lavidor, 2011). As discussed in the literature review, the inability to inhibit basic motor responses has been linked to various impulse-control disorders including addiction and overeating (e.g. Jentsch & Pennington, 2014; Nederkoorn *et al.*, 2006a). Secondly, there is evidence to suggest that stimulation of these prefrontal regions can result in decreased craving for both addictive substances and food (Boggio *et al.*, 2008, 2009, 2010; Fregni *et al.*, 2008; Goldman *et al.*, 2011; Montenegro *et al.*, 2012) and even decreased consumption of palatable foods (Fregni *et al.*, 2008). It seems logical, therefore, to explore whether tDCS can be combined with inhibition training to further reduce food consumption compared to inhibition training alone.

The aim of this pilot study was to explore whether such an experiment would be feasible using a within-subjects design. Although this design affords more power, the present results indicate that it also raises issues concerning repeated sessions. These issues need to be addressed and explored further before moving forward, although, a between-subjects design may be the more plausible solution. With recent studies

showing effects of repeated inhibition training on actual weight loss (Lawrence *et al.*, in preparation; Veling *et al.*, 2014), future research should also consider using more ecologically valid dependent measures to interpret outcomes. These studies also suggest that now may be the time to explore the effects of inhibition training outside of the laboratory over extended periods. These general limitations and possible solutions and future directions are discussed, along with others, in more detail in the general discussion.

Appendix 12: Safety Screening Questionnaire for TMS and tDCS contraindications.

CUBRIC, CARDIFF UNIVERSITY - TMS and TDCS SCREENING FORM

NAME OF PARTICIPANT Sex: M / F

Left or right handed?.....

Date of birth.....

Have you previously had an MRI scan at CUBRIC?.....

If so, are you happy for us to access your existing CUBRIC MRI data in this study?.....

Do you normally wear glasses or contact lenses? (please indicate which).....

Do you have normal colour vision?.....

Transcranial Magnetic Stimulation (TMS) and Transcranial Direct Current Stimulation (TDCS) are methods for safely stimulating the brain using an electric current.

Before receiving TMS or TDCS, please read the following questions carefully and provide answers. For a small number of individuals, these techniques may carry an increased risk of causing a seizure or other symptoms. The purpose of these questions is to make sure that you are not such a person. You have the right to withdraw from the screening and subsequent scanning if you find the questions unacceptably intrusive. The information you provide will be treated as strictly confidential and will be held in secure conditions.

If you are unsure of the answer to any of the questions, please ask the person who gave you this form or the person who will be performing the study. Definitions of some of technical terms are given overleaf.

	<i>Please tick</i>
Have you ever had an adverse reaction to TMS, TDCS, or other form of brain stimulation?	<input type="checkbox"/> Yes <input type="checkbox"/> No
Do you experience claustrophobia?	<input type="checkbox"/> Yes <input type="checkbox"/> No
Have you or has anyone in your family had a seizure?	<input type="checkbox"/> Yes <input type="checkbox"/> No
Have you had a stroke?	<input type="checkbox"/> Yes <input type="checkbox"/> No
Have you had a serious head injury (including neurosurgery) or have you ever been taken to hospital following an injury to the head?	<input type="checkbox"/> Yes <input type="checkbox"/> No
Do you have any metal in your head (outside the mouth) such as shrapnel, surgical clips, or fragments from welding or metalwork?	<input type="checkbox"/> Yes <input type="checkbox"/> No
Do you have any implanted devices such as cardiac pacemakers, aneurysm clips, cochlear implants, medical pumps, deep brain stimulators, or intracardiac lines?	<input type="checkbox"/> Yes <input type="checkbox"/> No
Do you suffer from frequent or severe headaches or have you ever experienced a migraine?	<input type="checkbox"/> Yes <input type="checkbox"/> No
Have you ever had any other brain-related condition?	<input type="checkbox"/> Yes <input type="checkbox"/> No
Have you ever had any illness that caused brain injury?	<input type="checkbox"/> Yes <input type="checkbox"/> No
Are you taking any psychiatric or neuroactive medications (e.g. antidepressants), or do you have a history of drug abuse?	<input type="checkbox"/> Yes <input type="checkbox"/> No
Are you pregnant?	<input type="checkbox"/> Yes <input type="checkbox"/> No
Do you, or does anyone in your family, have epilepsy?	<input type="checkbox"/> Yes <input type="checkbox"/> No
Are you taking any medication, or suffering from any medical condition, that causes dizziness, nausea or balance problems?	<input type="checkbox"/> Yes <input type="checkbox"/> No
Do you suffer from eczema or any other acute skin condition?	<input type="checkbox"/> Yes <input type="checkbox"/> No
Do you hold a heavy goods vehicle driving license, pilot's license, or bus license?	<input type="checkbox"/> Yes <input type="checkbox"/> No

Appendix 13: Food stimuli used in the go/no-go training tasks in Study A1 (Appendix 11).

Note. Foods presented with an asterisk were the foods presented in the snack buffet.

Food Selection 1.

Unhealthy-sweet
Foods
(chocolate)



Unhealthy-savoury
Foods
(cheese savouries)



Healthy-sweet Foods
(salad vegetables)



Healthy-savoury
Foods
(rice cakes/
crackers)



Food Selection 2.

Unhealthy-sweet
Foods
(biscuits)



Unhealthy-savoury
Foods
(crisps)



Healthy-sweet Foods
(fruit)



Healthy-savoury
Foods
(bread sticks)



Appendix 14: Nutritional information and weights for the unhealthy and healthy foods presented per food selection in the snack buffet in Study A1 (Appendix 11).

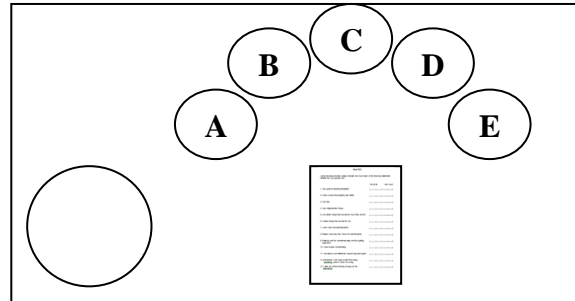
	Food selection 1	Food selection 2
Unhealthy-sweet	<p>Chocolate</p> <p>kCals = 554 (per 100g)</p> <p>fat = 34.2 (per 100g)</p> <p>Weight provided: ~269g</p> <p>- Cadbury 'Bitsa Wispa'</p>	<p>Biscuits (mini)</p> <p>kCals = 484 (per 100g)</p> <p>fat = 21.4 (per 100g)</p> <p>Weight provided: ~158g</p> <p>- Fox's mini malted milk biscuits</p>
Unhealthy-savoury	<p>Cheese bites</p> <p>kCals = 536 (per 100g)</p> <p>fat = 29.2 (per 100g)</p> <p>Weight provided: ~172g</p> <p>- ASDA's cheese bites</p>	<p>Crisps</p> <p>kCals = 550 (per 100g)</p> <p>fat = 36.3 (per 100g)</p> <p>Weight provided: ~76g</p> <p>- Tesco's ready salted crisps</p>
Healthy-sweet	<p>Carrot batons</p> <p>kCals = 42 (per 100g)</p> <p>fat = 0.3 (per 100g)</p> <p>Weight provided: ~279g</p> <p>- pre-cut carrot batons</p>	<p>Grapes</p> <p>kCals = 70 (per 100g)</p> <p>fat = 0.1 (per 100g)</p> <p>Weight provided: ~387g</p> <p>- green grapes</p>
Healthy-savoury	<p>Rice cakes (mini)</p> <p>kCals = 388 (per 100g)</p> <p>fat = 3 (per 100g)</p> <p>Weight provided: ~57g</p> <p>- Boots' organic plain rice cakes</p>	<p>Breadsticks (mini)</p> <p>kCals = 413 (per 100g)</p> <p>fat = 7.4 (per 100g)</p> <p>Weight provided: ~110g</p> <p>- ASDA's mini breadsticks</p>
Novel unhealthy food (buffet only)	<p>Flapjack bites</p> <p>kCals = 462 (per 100g)</p> <p>fat = 21.6 (per 100g)</p> <p>Weight provided: ~300g</p> <p>- Flapjack bites</p>	<p>Cake bites</p> <p>kCals = 443 (per 100g)</p> <p>fat = 15.9 (per 100g)</p> <p>Weight provided: ~155g</p> <p>Mr Kipling Victoria slices (cut into quarters)</p>

Appendix 15: Pseudo-random orders for the presentation of unhealthy and healthy foods in the snack buffet for Study A1 (Appendix 11).

Note. For clarity the orders have been presented here in numerical order – these were randomised for presentation in the study.

Food codes

- 1 Chocolate
- 2 Carrots
- 3 Cheese bites
- 4 Bread sticks (mini)
- 5 Biscuits (mini)
- 6 Grapes
- 7 Crisps
- 8 Rice cakes (mini)
- 9 Flapjack (mini)
- 10 Sponge cake (mini)



Schematic diagram of the buffet layout with 5 food bowls (A-E), questionnaires and a serving plate

Food selection 1

A	B	C	D	E
1	2	3	8	9
1	2	3	9	8
1	2	8	3	9
1	2	8	9	3
1	2	9	3	8
1	2	9	8	3
1	3	2	9	8
1	3	8	9	2
1	8	2	3	9
1	8	2	9	3
1	8	3	2	9
1	8	3	9	2
1	8	9	2	3
1	8	9	3	2
1	9	2	3	8
1	9	2	8	3
1	9	3	2	8
1	9	3	8	2
1	9	8	2	3
1	9	8	3	2
2	1	3	8	9
2	1	3	9	8
2	1	8	3	9
2	1	8	9	3
2	1	9	3	8
2	1	9	8	3
2	3	1	8	9
2	3	1	9	8
2	3	8	1	9
2	3	8	9	1
2	3	9	1	8
2	3	9	8	1

A	B	C	D	E
2	8	1	9	3
2	8	3	9	1
2	9	1	3	8
2	9	1	8	3
2	9	3	1	8
2	9	3	8	1
2	9	8	1	3
2	9	8	3	1
3	1	2	9	8
3	1	8	9	2
3	2	1	8	9
3	2	1	9	8
3	2	8	1	9
3	2	8	9	1
3	2	9	1	8
3	2	9	8	1
3	8	1	2	9
3	8	1	9	2
3	8	2	1	9
3	8	2	9	1
3	8	9	1	2
3	8	9	2	1
3	9	1	2	8
3	9	1	8	2
3	9	2	1	8
3	9	2	8	1
3	9	8	1	2
3	9	8	2	1
8	1	2	3	9
8	1	2	9	3
8	1	3	2	9
8	1	3	9	2

A	B	C	D	E
8	1	9	2	3
8	1	9	3	2
8	2	1	9	3
8	2	3	9	1
8	3	1	2	9
8	3	1	9	2
8	3	2	1	9
8	3	2	9	1
8	3	9	1	2
8	3	9	2	1
8	9	1	2	3
8	9	1	3	2
8	9	2	1	3
8	9	2	3	1
8	9	3	1	2
8	9	3	2	1
9	1	2	3	8
9	1	2	8	3
9	1	8	2	3
9	1	8	3	2
9	2	1	3	8
9	2	1	8	3
9	2	3	1	8
9	2	3	8	1
9	3	2	1	8
9	3	2	8	1
9	3	8	1	2
9	3	8	2	1
9	8	1	2	3
9	8	1	3	2
9	8	3	1	2
9	8	3	2	1

Food selection 2

A	B	C	D	E
4	5	6	7	10
4	5	6	10	7
4	5	7	6	10
4	5	7	10	6
4	5	10	6	7
4	5	10	7	6
4	6	5	10	7
4	6	7	10	5
4	7	5	6	10
4	7	5	10	6
4	7	6	5	10
4	7	6	10	5
4	7	10	5	6
4	7	10	6	5
4	10	5	6	7
4	10	5	7	6
4	10	6	5	7
4	10	6	7	5
4	10	7	5	6
4	10	7	6	5
5	4	6	7	10
5	4	6	10	7
5	4	7	6	10
5	4	7	10	6
5	4	10	6	7
5	4	10	7	6
5	6	4	7	10
5	6	4	10	7
5	6	7	4	10
5	6	7	10	4
5	6	10	4	7
5	6	10	7	4

A	B	C	D	E
5	7	4	10	6
5	7	6	10	4
5	10	4	6	7
5	10	4	7	6
5	10	6	4	7
5	10	6	7	4
5	10	7	4	6
5	10	7	6	4
6	4	5	10	7
6	4	7	10	5
6	5	4	7	10
6	5	4	10	7
6	5	7	4	10
6	5	7	10	4
6	5	10	4	7
6	5	10	7	4
6	7	4	5	10
6	7	4	10	5
6	7	5	4	10
6	7	5	10	4
6	7	10	4	5
6	7	10	5	4
6	10	4	5	7
6	10	4	7	5
6	10	5	4	7
6	10	5	7	4
6	10	7	4	5
6	10	7	5	4
7	4	5	6	10
7	4	5	10	6
7	4	6	5	10
7	4	6	10	5

A	B	C	D	E
7	4	10	5	6
7	4	10	6	5
7	5	4	10	6
7	5	6	10	4
7	6	4	5	10
7	6	4	10	5
7	6	5	4	10
7	6	5	10	4
7	6	10	4	5
7	6	10	5	4
7	10	4	5	6
7	10	4	6	5
7	10	5	4	6
7	10	5	6	4
7	10	6	4	5
7	10	6	5	4
10	4	5	6	7
10	4	5	7	6
10	4	7	5	6
10	4	7	6	5
10	5	4	6	7
10	5	4	7	6
10	5	6	4	7
10	5	6	7	4
10	6	5	4	7
10	6	5	7	4
10	6	7	4	5
10	6	7	5	4
10	7	4	5	6
10	7	4	6	5
10	7	6	4	5
10	7	6	5	4

Appendix 16: Correlation matrices for the difference in pre- and post- tDCS/training measures of state hunger (hunger, fullness and desire to eat; VAS) and mood (positive and negative affect; PANAS) and calorie consumption according to food type, and for total calorie consumption, Study A1 (Appendix 11).

Sham condition

	Unhealthy – old calories	Healthy calories	Unhealthy – new calories	Total calories
Hunger	-0.59	-0.346	0.239	-0.379
Fullness	0.120	0.072	0.041	0.111
Desire to eat	-0.866*	-0.008	-0.048	-0.493
Positive affect	-0.089	0.561	0.250	0.169
Negative affect	-0.454	0.532	-0.652	-0.359

* $p < 0.05$

Active condition

	Unhealthy – old calories	Healthy calories	Unhealthy – new calories	Total calories
Hunger	0.194	-0.047	-0.091	0.081
Fullness	0.573	0.157	0.516	0.601
Desire to eat	-0.457	-0.237	-0.766*	-0.641
Positive affect	-0.222	-0.240	0.495	0.071
Negative affect	-0.549	-0.146	-0.989**	-0.820~

* $p < 0.05$

** $p < 0.01$

Appendix 17: Correlation matrices for the difference in pre- and post- tDCS/training measures of state food craving (G-FCQ-S) and calorie consumption according to food type, and for total calorie consumption, Study A1 (Appendix 11).

Sham condition

	Unhealthy – old calories	Healthy calories	Unhealthy – new calories	Total calories
G-FCQ-S	0.179	0.366	0.716	0.522

Active condition

	Unhealthy – old calories	Healthy calories	Unhealthy – new calories	Total calories
G-FCQ-S	-0.464	-0.514	-0.533	-0.565