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Abbreviations

a.m.	ante meridiem
ANOVA	analysis of variance
CNS	central nervous system
e.g.	exempli gratia
et al.	et alii/et aliae
g	gram(s)
h	hour(s)
HC	high caloric
i.e.	id est
kg	kilogram(s)
LC	low caloric
m	metre(s)
M	mean
ml	millilitre(s)
mm	millimetre(s)
mRNA	messenger RNA
ms	millisecond(s)
n.s.	not significant
OECD	Organisation for Economic Cooperation and Development
p.m.	post meridiem
SD	standard deviation
SEM	standard error of the mean
UZA II	Universitätszentrum Althanstraße II
WHO	World Health Organisation
WLAN	Wireless Local Area Network

1 Introduction and leading question

In Austria, 12 % of adults and even 7 % of schoolchildren are obese with respect to the data collected for the 'Österreichischer Ernährungsbericht 2012' [ELMADFA *et al.*, 2012]. According to the latest OECD health statistics nearly 23 % of total population in OECD nations can be classified as obese with the United States topping this statistics (35 %) [OECD, 2014a]. Detrimentially, the worldwide obesity epidemic continues to grow and none of the OECD nations can reveal reversal trends [OECD, 2014b].

The growing prevalence of obesity paired with a rising awareness of its adverse effects on health and health costs have therefore stimulated research among several disciplines in investigating the major causing determinants for this condition [RICHARD and TIMOFEEVA, 2010].

Most conceptualizations concerning the regulation of food intake propose that two different, but interacting systems influence food intake and therefore both, internal, physiological signals as well as external, environmental signals control food consumption [PETROVICH, 2013].

While the homeostatic system serves to maintain energy balance, the hedonic regulation of food intake is mediated by cortico-limbic brain structures, which are associated to learning, memory, emotions and reward. This system is strongly based on palatability and rewarding capacity of food, leading not only to establishing food preferences and aversions but also influencing motivational concepts to eat [KENNY, 2011; JOHNSON, 2013; PETROVICH, 2013].

The obesogenic environment, which is characterised by a mainly sedentary lifestyle, an overwhelming access to highly palatable, high caloric food and increasing portion sizes, is thought to play a pivotal obesity-inducing role. However, while many individuals are very good at dealing with these conditions, others seem to heavily struggle within their environment in terms of weight control. [DAVIDSON *et al.*, 2005; RICHARD and TIMOFEEVA, 2010; LOXTON *et al.*, 2011].

A major determinant in controlling reward-driven food consumption is an individual's extent of successful self-regulatory processes that aim to override unwanted, predominant impulses such as indulging to tempting foods. Self-regulation as a goal-

directed behaviour is intricately linked to executive functions which are defined as a collection of top-down mental processes [HOFMANN *et al.*, 2012]. They can be seen as gatekeepers for food intake and the selection of food [RIGGS *et al.*, 2010] as they are used when solely relying on instinct would lead to ill-advised or insufficient behaviour. In the complexity of cognitive control, three core executive functions can be assumed: updating, inhibition and shifting [HOFMANN *et al.*, 2012; DIAMOND, 2013].

Updating is linked to the construct of working memory and can therefore be seen as the ability of keeping information in mind and relating them to former and prospective information [HOFMANN *et al.*, 2012; DIAMOND, 2013]. In the context of food intake, working memory allows individuals to hold healthy nutritious plans in the foreground of mind [DAVIDSON *et al.*, 2005]. However, to keep a goal in mind, irrelevant or inappropriate information have to be inhibited. Thus, working memory is linked to inhibition as they co-occur and support each other [HOFMANN *et al.*, 2012; DIAMOND, 2013].

Inhibitory control broadly refers to not acting in accordance with impulses, habits or environmental stimuli but to control one's mind in order to resist temptations [DIAMOND, 2013]. Environmental cues, such as the sight or smell of food and even thinking of palatable food induces appetite and can provoke subsequent eating especially in individuals who are prone to overeating, such as restrained eaters are [JANSEN *et al.*, 2009]. The ability to resist those cues or the urge to eat is mainly based on one's inhibitory control as it plays an important role in cognitive and behavioural control. Inhibition is in turn affected by many aspects of behaviour leading to increased impulsivity [MARTIN and DAVIDSON, 2014].

Set-shifting is the third core of executive functions and is also called cognitive flexibility. It is thought to be the ability for changing perspectives and for flexibly adapting to new rules or demands and is therefore built on working memory and inhibitory control [DIAMOND, 2013].

Neuropsychological research has investigated cognitive flexibility and impulsivity by using a variety of set-shifting paradigms. Within those tasks, participants need to update present rules in certain trials, in order to correctly response to a given stimulus. Task performance is typically poorer during shift blocks, where upcoming stimuli differ from

the previous ones and participants have to both, flexibly reverse response associations as well as maintain behavioural control [BOLTON *et al.*, 2014; MEULE *et al.*, 2014; TESLOVICH *et al.*, 2014].

Furthermore, research on the relation between cognitive priming and subsequent food intake has been conducted. Cognitive priming can be described as a process by which the accessibility of stored concepts or information is increased when participants get recently or repeatedly exposed to similar concepts [ROTENBERG *et al.*, 2005]. It is therefore a commonly used method to enhance the cognitive availability of psychological concepts without participants' conscious awareness [GUERRIERI *et al.*, 2009]. Priming participants with words associated with lack of control resulted in increased subsequent food intake. As priming is an implicit memory process, these findings suggest that activating thoughts of control can causally affect food intake in a non-conscious way [ROTENBERG *et al.*, 2005]. *Guerrieri and colleagues (2008)* used a memory task for manipulating participants' impulsivity and inhibition and found those primed to impulsive items eating more in a subsequent taste test than compared to the others. Those findings indicate that the induction of impulsivity provokes overeating, while inducing inhibition has reverse effects [GUERRIERI *et al.*, 2009].

According to this, the present camouflaged experimental thesis was created in order to investigate the capability of priming effects on an individuals' extent of impulsivity.

Therefore an affective shifting task (go/no-go task), conceptualized in line with the study of *Meule and Kübler (2014)*, was created in order to collect baseline information of participants overall task performance. Within the task, pictures of high caloric and low caloric foods were presented randomly. Participants had to respond to a go stimulus, but withhold their response when a no-go stimulus appeared. The defined target category altered every second block and thus shift blocks (same stimulus-response relation) and non-shift-blocks (reverse stimulus-response relation) were created. Participants' overall reaction times to go-trials, the number of omission errors as well as the number of commission errors were defined as target values.

Referring to *Meule and Kübler*, high number of commission errors, particularly within the more challenging shift-blocks, is associated with low inhibitory control, respectively

with impulsive behaviour. This assumption is supported by the correlation between self-reported trait impulsivity and trait food craving [MEULE and KÜBLER, 2014].

The intention of the current thesis was not only to confirm those findings but to additionally examine whether task performance alters after repeatedly priming participants with high caloric food cues and low caloric food cues as control condition.

Therefore, subsequent to a two weeks priming intervention phase participants had to perform another go/no-go task which provided the main experimental results.

With respect to previous findings [ROTENBERG *et al.*, 2005; GUERRIERI *et al.*, 2009] the author expected that participants which had been previously primed to high caloric food cues committed more errors in response to high caloric food cues during shift-blocks compared to those participants which were assigned to the low caloric control group.

Therefore the following main hypotheses were defined:

- Hypothesis I

Does the priming intervention influence participants' task performance within the second go/no-go task, respectively the number of committed errors within the task as an expression of altered inhibitory control?

H0: There is no difference in the number of commission errors between the first and the second go/no-go task.

H1: There is a difference in the number of commission errors between the first and the second go/no-go task.

- Hypothesis II

Does the priming with high caloric food cues in particular influence the number of commission errors in the second go/no-go task?

H0: There is no difference in the number of commission errors between the HC intervention group and the LC intervention group.

H1: There is a difference in the number of commission errors between the HC intervention group and the LC intervention group.

2 Theoretical background

2.1 Regulation of food intake

Human eating is a complex and broadly varied phenomenon, which is influenced by peripheral metabolic signals, which convey information about the body's energy status to the brain, as well as psychological sensations.

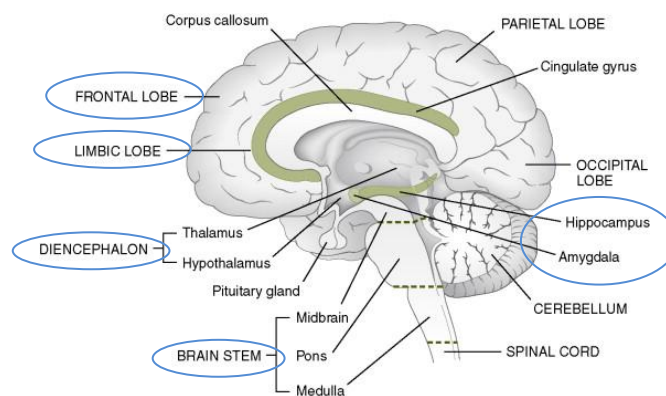
The homeostatic regulation of hunger and satiety aims at maintaining energy balance. It detects the status of energy stores and equalises energy intake with energy expenditure. On the other hand, the hedonic control of appetite is strongly mediated by reward. The power of food-related stimuli in turn is influenced by the body's energy status. Therefore palatable food stimuli are more appetizing in conditions of energy depletion than in conditions of energy surfeit [HARROLD *et al.*, 2012; RICHARD and TIMOFEEVA, 2010].

The following chapters should illustrate the functions of the main brain structures involved in food intake regulation as well as the purpose of the homeostatic and hedonic control of appetite.

2.1.1 Involved brain structures

Several cerebral areas are involved in the complex control of hunger and satiety. The caudal brainstem and the hypothalamus play a key role in the control of food intake due to changes in energy stores, while parts of the cortex and the limbic system are involved in the interaction with the environment [LENARD and BERTHOUD, 2008].

Figure 1: Schematic midsagittal view of the brain [after CECH and MARTIN, 2012]



2.1.1.1 Caudal brainstem

The brainstem is the posterior part of the brain and consists of the hindbrain, which is build up of the pons and the medulla oblongata, and the midbrain. It comprises of three major structures, which are involved in energy homeostasis: the nucleus of the tractus solitarius (NTS), the area postrema (AP) and the motor nucleus of the vagus nerve. Those structures compose the dorsal vagal complex (DVC) [RICHARD and TIMOFEEVA, 2010].

The NTS, located in the medulla oblongata, plays a key role in the integration of vagally transmitted signals conveyed by the intestinal peptide cholecystokinin as well as by the peptides leptin, ghrelin, amylin and the neuropeptide Y (NPY). Also afferent sensory signals from the mouth are conveyed to the NTS via cranial nerves [HARROLD *et al.*, 2012].

The AP is directly connected to the NTS and has been identified as the chemoreceptor zone which is responsible to trigger vomiting reflexes. It is also important in fluid and electrolyte homeostasis and in processing signals from blood and bone substances [YOUNG, 2012].

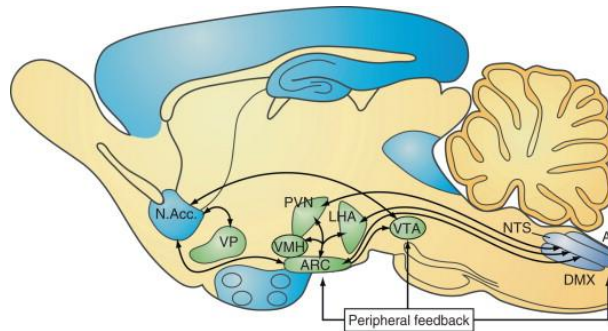
2.1.1.2 Hypothalamus

The hypothalamus is located in the ventral diencephalon directly beneath the thalamus and can be seen as the body's major control centre for nutrient-related signals. They are transmitted from peripheral tissues or mediated through hormones and metabolites or neural pathways mainly from the brainstem. Within the hypothalamus, the received signals are linked with external and internal signals and also compete with other regulatory pathways, such as thermoregulation or fluid homeostasis [LENARD and BERTHOUD, 2008].

The stability of energy stores is ensured by interconnected neurons expressing specific receptors and ion channels as well as producing diverse mediators, such as peptides or neurotransmitters. Within those hypothalamic neurons, glucose, fatty acids, amino acids and other nutrients cause a variety of neurobiological responses. Energy intake and nutrient utilization is regulated due to hypothalamic nutrient sensing neurons, which are mostly found in nuclei. The arcuate nucleus (ARC), the paraventricular nucleus (PVN)

and the lateral nucleus of the hypothalamus (LH) are the most important hypothalamic sites in the regulation of energy balance [BLOUET and SCHWARTZ, 2010;]. The hypothalamic nuclei are interconnected and also receive signals via the NST/AP as shown in *figure 2*.

Figure 2: Interconnected neural regulation of food intake [FRIHAUF *et al.*, 2010]



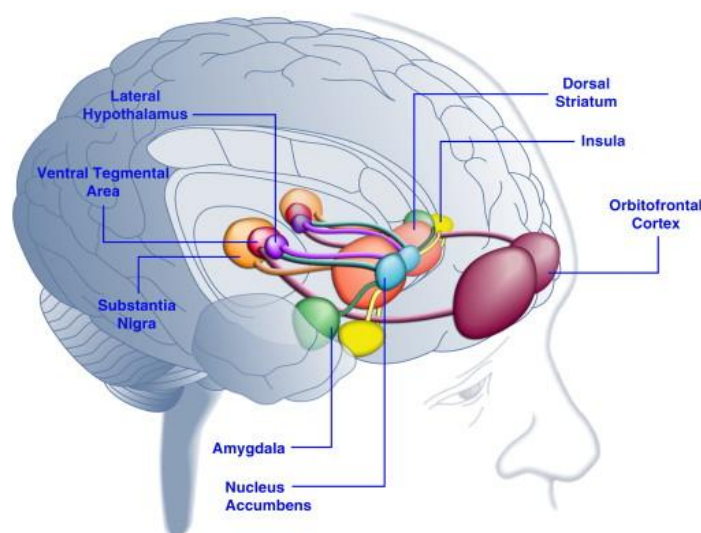
The ARC is considered as the main zone of the hypothalamus for regulating energy intake and energy expenditure. It integrates peripherally transmitted signals and comprises of two populations of neurons of the melanocortin system. The orexigenic neurons synthesize the anabolic peptides NPY and agouti-related peptide (AgRP), whereas the anorexigenic neurons express catabolic peptides, such as proopiomelanocortin (POMC) and cocaine amphetamine-related transcript (CART) [ELIAS *et al.*, 1998; HARROLD *et al.*, 2012]. The PVN integrates signals from the ARC and the LH and communicates with important areas in the brainstem [HARROLD *et al.*, 2012].

The lateral hypothalamic area has a far reaching afferent and efferent connectivity. It receives signals from the ARC and from the caudal brainstem and is also connected to cortico-limbic structures. Therefore it integrates a vast of neural and humoral transmitted information and processes adaptive responses. Similar to the ARC, the LH comprises of neuronal populations expressing either orexigenic neuropeptides, such as the melanin-concentrating hormone (MCH) or anorexigenic neuropeptides, such as neurotensin or CART. Due to its ability to directly adjust cognitive, behavioural, autonomic and endocrine functions, the LH plays a key role in the modulation of energy balance [BERTHOUD and MÜNZBERG, 2011].

2.1.1.3 Limbic structures

As explored, the homeostatic regulation of food intake is mainly formed by the brainstem and the hypothalamus. In contrast, cortico-limbic brain structures act hedonically regulating food intake. Those structures are strongly associated with emotion, memory and sensory sensations [RICHARD and TIMOFEEVA, 2010].

Figure 3: Limbic structures involved in hedonic regulation of food intake [KENNY, 2011]



The nucleus accumbens and the ventral tegmental area

The nucleus accumbens (NAcc), located in the ventral striatum, consists of the anatomically different core and shell components. Within the rostromedial region of the medial shell, “hedonic hotspots” have been identified. The NAcc shell as a whole, but especially the hedonic hotspots play an important role in the nonhomeostatic regulation of food intake by expressing μ -opioid receptors. An activation of those receptors by brain microinjection is associated with enhanced liking reactions towards sensory rewards, such as the sweet taste, and an increased intake of palatable food due to greater motivation to eat [CASTRO and BERRIDGE, 2014; KENNY, 2011].

Besides the opioid system, the mesolimbic dopamine pathway also plays a crucial role in terms of appetite. Dopamine release is associated with the consumption of palatable food and therefore dopamine can be seen as a motivational neurotransmitter, as it

increases the motivation to eat although the stimulation of dopaminergic receptors does not enhance positive reactions to palatable food [FRIHAUF *et al.*, 2010]. The mesolimbic dopamine pathway comprises of a subpopulation of dopamine neurons located in the ventral tegmental area (VTA). The VTA integrates signals from the periphery and the hypothalamus as well as information from parts of the midbrain, hindbrain, the limbic system and the cortex. In turn the VTA projects to the NAcc, ventral pallidum (VP), prefrontal cortex, amygdala and the hippocampus [MEYE and ADAN, 2014]. The NAcc is also interconnected with the LH and thus the motivationally-related limbic regulation is linked to the homeostatic regulation of the hypothalamus and brainstem [FERNANDES *et al.*, 2013].

The Hippocampus, Amygdala and the orbitofrontal cortex

The hippocampus, amygdala and the orbitofrontal cortex are important parts of the brain's reward circuitry, which provides food consumption and the storage of energy despite of homeostatic energy needs [SIEP *et al.*, 2009].

The hippocampus is located in the medial temporal lobe and plays a central role in processes related to emotion and memory [VAGO *et al.*, 2014], but it is also involved in the control of food intake. It integrates external and internal appetite-related signals and creates food-related memories, such as remembering the point in time of last meal consumption and identifying interoceptive states of hunger. Additionally, the hippocampus expresses a variety of peripheral hormones involved in the control of food intake, such as ghrelin and insulin [VOLKOW *et al.*; 2011; FERNANDES *et al.*, 2013].

The amygdala is also located in the medial temporal lobe and it comprises three nuclei - the basolateral amygdala (BLA), the lateral amygdala (LA) and the central amygdala (CeA) [AMUNTS *et al.*, 2005]. It is related to emotional learning, memory formation and to reward processes and therefore plays a central role in integrating sensory-related and physiological stimuli and subsequently engendering the appropriate behavioural responses. The amygdala is reciprocally connected to other limbic structures, such as the hippocampus and the orbitofrontal cortex as well as to hypothalamic areas [FERNANDES *et al.*, 2013; STAMATAKIS *et al.*, 2014].

The orbitofrontal cortex as part of the prefrontal cortex is located in the ventral surface of the frontal lobe of the brain and integrates sensory inputs, modulates autonomic reactions and is fundamentally involved in decision making. It is directly connected to the amygdala and uses projected signals to predict reward outcomes [KRINGELBACH, 2005; SIEP *et al.*, 2009].

2.1.2 Homeostatic regulation of food intake

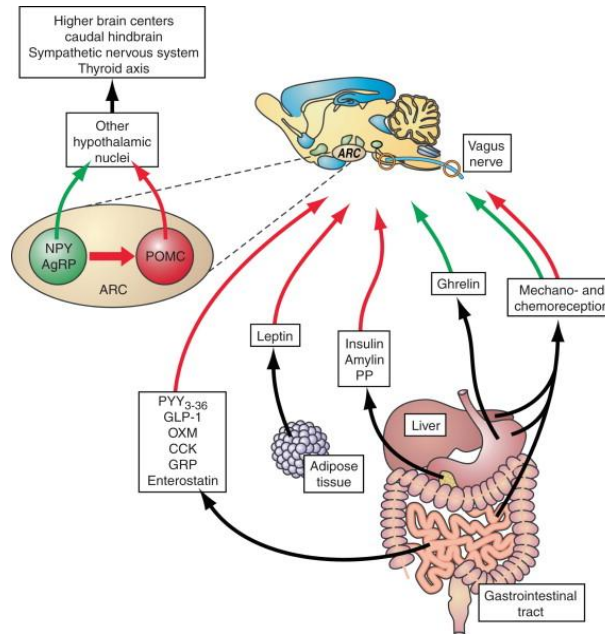
The homeostatic control of energy balance and food intake is regulated by a vast of mechanisms regarding to the gut-brain-axis. Within this regulatory circuitry the hypothalamus and the brainstem are the most important brain structures as they integrate numerous signals in order to determine body's energy resources and initiate the respective behavioural actions [HARROLD, 2004; DEL PRETE *et al.*, 2012]

The occurrence of these signals is caused by sensory sensations of eating as well as by peripheral receptors in the gastrointestinal tract, which are vagally transmitting chemical and mechanical signals to the brain. Mechanical signals from the stomach are released in response to stomach and duodenal distension. Furthermore the nutrient content itself can cause the release of specific peripheral hormones [FRIHAUF *et al.*, 2010].

2.1.2.1 Peripheral Signals

Peripheral hormonal signals play an important role in the homeostatic regulation of food intake, as they mainly emerge in response to food ingestion and activate or inhibit appetite regulating systems in the brainstem and hypothalamus as stated in *figure 4* [FRIHAUF *et al.*, 2010]. According to their duration effect, they can be classified in episodic and tonic signals. Episodic signals arise by recent food consumption and therefore provide short-term inputs, whereas tonic signals provide long-term inputs depending on the body's constant metabolic constitution [HARROLD *et al.*, 2012;].

Figure 4: Peripheral signals and the control of food intake [FRIHAUF *et al.*, 2010]



Episodic signals for satiety

Satiation is defined as the process terminating food consumption and leading to a state of satiety, in which hunger is inhibited [BLUNDELL, 1991]. Satiety can be controlled by several mechanisms including stomach distension, signals arising through nutrient oxidation or by chemical substances occurring during digestion, such as cholecystokinin, glucagon-like peptide-1, peptide YY and amylin [HARROLD *et al.*, 2012].

Cholecystokinin (CKK)

CCK is a peptide released from the I-cells in the proximal intestinal tract post-prandially, at a maximum of 25 minutes after meal initiation. It is primarily secreted in response to fat and protein rich food, leading to termination of hunger and food intake [HARROLD *et al.*, 2012].

CKK can bind to the two G-protein coupled receptors CKK-1 and CKK-2. CKK-1 receptors occur in peripheral organs, such as the pancreas, the gall bladder and the intestines and leads to secretion of digestive enzymes in these organs. Besides this, both receptor types are expressed in the striatum and the NAcc shell region [FRIHAUF *et al.*, 2010; HARROLD *et al.*, 2012; SAM *et al.*, 2012; FERNANDES *et al.*, 2013]. CKK interacts

with the hypothalamus using two different pathways. The main route is determined by signals relayed by the vagus nerve and the brainstem to the hypothalamus [BEGLINGER *et al.*, 2001], whereas the other pathway is modulated by direct communication with the CKK-1 receptors located in the hypothalamus [BLEVINS *et al.*, 2000].

Glucagon-like-peptide-1 (GLP-1)

GLP-1 is an anorexigenic hormone secreted post-prandially by the L-cells in the distal intestinal tract following carbohydrate but also fat ingestion and is co-secreted with peptide YY [LAVIN *et al.*, 1998; FROST *et al.*, 2005].

GLP-1 can bind to GLP-1-receptors (Glp-1R) located both in peripheral tissues as well as in the CNS. In peripheral organs the incretin hormone GLP-1 meets its main function in appetite control by activating the release of insulin and inhibiting the secretion of glucagon [FRIHAUF *et al.*, 2010].

GLP-1R is expressed in different areas of the brain, containing regions of the NST and AP located in the brainstem and the hypothalamic ARC. Throughout projections of GLP-1 neurons in the NST to the NAcc, food intake is inhibited as satiety signals in the hindbrain are linked with food reward related signals in limbic structures [DOSSAT *et al.*, 2011].

Peptide YY (PYY)

PYY is synthesized in the L-cells of the distal intestinal tract and is released in the very end of meal consumption. The endogenously truncated form PYY₃₋₃₆ is the main circulating PYY, which is secreted in response to detection of fatty acids, fibre and bile acid in the intestines leading to decreased food intake by delaying gastric emptying [FRIHAUF *et al.*, 2010].

PYY₃₋₃₆ interacts with Y2 receptors, which are widespread throughout the CNS. Signals from peripheral PYY₃₋₃₆ are mediated via vagal afferents to the NST and therefore performing the effects of PYY₃₋₃₆ in appetite control [RENSHAW and BATTERHAM, 2005]. *Batterham and colleagues* have demonstrated that PYY₃₋₃₆ affects not only the homeostatic regulation of food intake, but also the hedonic reward-related eating behaviour by modulating neural activity in limbic structures [BATTERHAM *et al.*, 2007].

Amylin

Amylin is a peptide co-released with insulin from the pancreatic β -cells dependent on glucose levels in the blood. It can act peripherally as well as centrally via the AP in the brainstem, leading to decreased food intake and delayed gastric emptying and digestive secretion [HARROLD *et al.*, 2012].

Episodic signals for hunger

Hunger can be seen as striving after food consumption and therefore initiating a feeding period. Signals released from sensory sensations, such as sight, smell and mouth feeling of food, are transmitted to the brain via afferent fibres of several cranial nerves and prepare the digestive system for the subsequent food administration. Furthermore, changes in blood glucose levels as well as peripherally acting peptides, such as ghrelin, can provide a signal for meal initiation [HARROLD *et al.*, 2012].

Ghrelin

Ghrelin is a peptide mostly secreted from the fundus cells of the stomach and ϵ -cells of the pancreas, but can also be found in smaller amounts in the brain with maximum circulatory concentrations prior to meal initiation [FRIHAUF *et al.*, 2010].

Ghrelin binds to the G-protein-coupled growth-hormone-secretagogue receptor (GHS-R) and therefore increases sensations of hunger and stimulates food intake. It is also involved in weight status by decreasing fat expenditure and promoting fat storage [HARROLD *et al.*, 2012; WREN *et al.*, 2001].

The orexigenic effects of ghrelin to the homeostatic regulation of eating behaviour are derived from activating neurons, such as NPY expressing neurons, in the hypothalamic ARC and in the NTS of the caudal brainstem. Additionally, ghrelin affects the mesolimbic dopamine pathway and therefore influences the hedonic control of food intake, respectively enhances the motivation to eat [FRIHAUF *et al.*, 2010].

Tonic signals

In contrast to episodic signals, which are released as an acute response to nutrient intake, tonic signals arise as a long-term response to the status of the body's energy stores. Tonic signals are produced from organs involved in energy storage, including the liver, the pancreas and the adipose tissue. The main tonic signal, which provides information about the extent of the body's adipose tissue is leptin [HARROLD *et al.*, 2012].

Leptin

The anorexigenic protein leptin plays a crucial role in the regulation of food intake. It is synthesized from the body's adipocytes. Leptin plasma concentrations are proportional to fat mass. Therefore, an increase in body's fat mass leads to proportionally increased circulatory leptin levels, which subsequently induces a decreased food intake and an increase in energy expenditure. On the other hand food intake to maintain energy stores is stimulated in response to starvation [FRIHAUF *et al.*, 2010].

Leptin binds to three main types of receptors (OB-Ra, OB-Rb, OB-Rc) and provides its anorexigenic effect throughout the interaction with OB-Rb receptors located in the hypothalamus, medulla oblongata and other cerebral areas. Within the hypothalamus, leptin inhibits appetite-stimulating neurons, such as NPY and on the other hand it activates appetite-inhibiting neurons, such as parts of the melanocortin system [HARROLD *et al.*, 2012; FERNANDES *et al.*, 2013].

Furthermore circulatory leptin enhances the anorexigenic effect of CCK in a synergistic way mediated by leptin receptors in the hypothalamic ARC, in the brainstem or by peripheral leptin receptors on vagal afferent fibres. Leptin is also likely to reduce food intake by stimulating the hippocampus and therefore links the homeostatic regulation with the hedonic modelling of food intake [HARROLD *et al.*, 2012].

2.1.2.2 Central signals

The brain structures involved in the homeostatic control of food intake comprise a variety of neurotransmitters and neuropeptides, which affect eating behaviour and weight control [HARROLD *et al.*, 2012].

Central signals for satiety

The melanocortin system

The melanocortin system plays a pivotal role in the regulation of energy homeostasis, as it comprises of receptors, which are expressed in the brain and mediate the anorexigenic effects of the melanocortin peptides. In particular, there are two receptor subtypes (MC3-R, MC4-R) performing these effects, which are located in the hypothalamic nuclei [HARROLD *et al.*, 2012].

The melanocortin peptides are derived from the precursor POMC, which in turn is produced in neurons of the hypothalamic ARC, but also in neurons of the NST in the brainstem. Within the melanocortin peptides, notably the α -melanocyte stimulating hormone (α -MSH) and the β -MSH are potential inhibitors of food intake [ZHENG *et al.*, 2005].

The melanocortin system is regulated by the activity of different peripheral hormones, especially leptin, as the POMC neurons also express a remarkable amount of leptin receptors. Therefore POMC expression increases proportionally to increasing leptin levels as a response to overfeeding [FRIHAUF *et al.*, 2010; HARROLD *et al.*, 2012].

On the other hand POMC neurons receive signals from the naturally occurring antagonist AgRP, which is co-expressed with NPY in the hypothalamic ARC. NPY/AgRP neurons stimulate food intake, as they directly act to orexigenic pathways and additionally override the leptin-induced anorexigenic effect [EBIHARA *et al.*, 1999].

Cocaine amphetamine-related transcript (CART)

The endogenous satiety factor CART is co-expressed in POMC neurons within the ARC and is supposed to be involved in different mechanisms of food intake [HARROLD *et al.*, 2012]. CART modulates the activity of NPY and therefore inhibits NPY-induced

food intake. In turn, CART mRNA expression is stimulated by leptin, enhancing its anorexigenic effects [KRISTENSEN *et al.*, 1998].

Besides this, there are also interactions between CART and endocannabinoids and dopamine, which are both involved in hedonic regulation of food intake [HARROLD *et al.*, 2012].

Serotonin (5-HT)

The serotonin system is closely linked to the activation of peripheral hormones following fat ingestion, such as CKK, and therefore decreases food intake and weight gain as a consequence of these signals [HARROLD *et al.*, 2012]. Additionally, the consumption of carbohydrate rich meals leads to an increased expression of serotonin within the hypothalamic PVN and in turn to decreased levels of NPY within the PVN [LEIBOWITZ *et al.*, 1990].

Serotonin binds to a variety of receptors, whereby the anorexigenic effect of serotonin is mainly mediated by the two receptor subtypes 5-HT_{1B} and 5-HT_{2C} [HALFORD and BLUNDELL, 2000].

Central signals for hunger

Neuropeptide Y (NPY)

The highly potent orexigenic peptide NPY is widely distributed throughout the brain, but is primarily synthesized from the NPY neurons within the hypothalamic ARC, which also project to PVN. NPY binds to specific receptors, but only the Y₁ and Y₅ receptor subtypes mediate the orexigenic effects of the peptide leading to increased food intake, meal duration and enhanced motivation to eat [FRIHAUF *et al.*, 2010; HARROLD *et al.*, 2012].

NPY neurons in the ARC, PVN and hindbrain detect available energy resources by interacting with peripheral and central hormonal signals. The expression is stimulated by the intestinal peptide ghrelin and is in turn inhibited by the anorexigenic protein leptin. In case of energy depletion and subsequently decreased leptin concentrations, the

expression of NPY genes is increased, leading to enhanced food intake, in order to restore body's energy levels [FRIHAUF *et al.*, 2010].

Orexins

Within the orexin system, orexin A and orexin B are mainly involved in the regulation of food intake as they increase striving for food and food consumption, but however less potently than NPY does [FRIHAUF *et al.*, 2010]. The two orexins are synthesized within the LH and bind to specific receptor subtypes, whereby orexin A selectively binds to OX-1 receptors located in the ventromedial hypothalamus (VMH) and ARC, whereas orexin B binds preferably to OX-2 receptors located in the PVN and hindbrain [WANG and LEIBOWITZ, 1997; HARROLD *et al.*, 2012].

Orexin neurons are rapidly stimulated by decreasing blood glucose levels making them sensitive to changes in food intake. Similarly to NPY, the expression of orexins is inhibited by increasing leptin levels. Depending on nutritional sensing, peripheral orexin neurons in the enteric nervous system modulate gastric secretion and motility, in order to prepare the gastrointestinal tract for subsequent food ingestion [FRIHAUF *et al.*, 2010].

The orexin system is furthermore connected to reward-related areas in the brain, as the OX-1 and OX-2 receptors are also expressed within the VTA, which receives projections from orexin neurons [FADEL and DEUTCH; 2002].

Melanin-concentrating hormone (MCH)

The orexigenic peptide MCH is expressed in the hypothalamic LH as well as in the subthalamic zona incerta [NAITO *et al.*, 1988] and binds to the specific MCH-R1 and MCH-R2 receptors. MCH increases food intake in a comparable amount to orexins, as the MCH neurons are stimulated by food restriction and are in turn inhibited by leptin. MCH-R1 receptors are also widely distributed in limbic structures, such as the NAcc and the amygdala, leading to a reward-related connection of food intake behaviour [HARROLD *et al.*, 2012].

2.1.3 Hedonic regulation of food intake

In the regulation of food intake, the hormonal and neural control of feeding, which is mainly regulated throughout hypothalamic and hindbrain systems, constitutes only one side of the medal. On the other side, behavioural, social and environmental factors are connected to food intake and determine ‘what’, ‘when’ and ‘how much’ we eat beyond energy needs [JOHNSON, 2013; FERNANDES *et al.*, 2013].

The neural system for maintaining energy homeostasis is believed to be a potent complex following metabolic needs. When taking the increasing incidence of obesity into account, it is obvious, that other neural processes related to appetitive and rewarding aspects of food might influence eating behaviour in a likewise extent. The reward circuitry is a complex comprising of neuronal systems triggered by palatability and pleasure as they are remarkable motivators for food intake. The most important neuronal systems, involved in the hedonic regulation of food intake are the opioid system and the dopamine system [HARROLD *et al.*, 2012].

Besides the neuronally mediated reward effects, behavioural aspects must be considered too, when talking about the hedonic control of food intake.

2.1.3.1 Emotions and reward

“We recognize pleasure as the first good innate in us, and from pleasure we begin every act of choice and avoidance, and to pleasure we return again, using the feeling as the standard by which we judge every good.” – Epicurus [after FULTON, 2010].

The experience of emotions influences behaviour in general and eating behaviour in particular by affecting choice and avoidance and therefore emotions are capable to powerfully shape future actions [FULTON, 2010].

Emotional responses to food are determined by different factors. Sensory qualities of foods are capable to affect appetite in a way, that taste, smell, appearance and texture of a food elicit different emotions among customers [DESMET and SCHIFFERSTEIN, 2008]. For example, sweet solutions are likely to cause positive feelings, such as happiness, whereas bitter taste is related to feelings of anger and disgust. Emotional responses to food are also affected by the type of food [ROUSMANS *et al.*, 2000] In this context

emotions might range much more as a consequence of personal significance, including feelings of love when remembering a person usually preparing this food or feeling guilty when eating a high caloric food [DESMET and SCHIFFERSTEIN, 2008]. And last but not least, individual's characteristics, such as nutritional state, diet awareness, cultural or educational habits influence emotional responses to food. Within this context, negative emotions as well as pleasurable feelings must be considered as determinants for food intake [JIANG *et al.*, 2014].

Individuals in a negative mood state are prone to excessive food intake in order to attain pleasurable feelings and to escape negative emotions. That is, emotional eaters are less successful in dieting and are more likely to pathologically gain weight than compared to non-emotional eaters, who do not change their food consumption in a negative mood state. Therefore, mechanisms underlying emotional eating are comparable to those seen in addictive behaviour, such as drug or alcohol addiction. Behavioural processes are biased by negative affects during conditions of stress and deprivation and thereby can lead to encouraging addictive behaviour, respectively increasing reward values of food cues and urge to eat [HEPWORTH *et al.*, 2010; BONGERS *et al.*, 2013; WERTHMANN *et al.*, 2014].

Positive feelings do as well affect food intake as they are related to increased eating due to associative learning mechanisms. For example, food is used as a highlight within special events, such as weddings and is consumed with familiar and friendly people. Therefore, socializing and eating become strongly connected, resulting in more enjoyable conditions of eating and consequently in an increase of food consumption. Furthermore, an individual's mental strength can be enhanced by pleasurable emotions, making it much easier to resist rewarding food cues [EVERS *et al.*, 2013; BONGERS *et al.*, 2013].

The term 'pleasure' is related to a variety of positive emotions including happiness, satisfaction, confidence or joy, but is rather difficult to quantify. Thus, the term 'reward' is much more commonly used and refers to objects, which affect behaviour in a way leading to the ongoing of an action. It is determined by a vast of factors including the aspects of hedonic 'liking' and hedonic 'wanting' [KRINGELBACH *et al.*, 2012]. Food-

related rewards are subjectively assessed in terms of sensory qualities as well as in terms of availability and metabolic needs [FULTON, 2010].

Hedonic 'liking' refers to a pleasure-giving value of a food and/or the appreciation of its sensory qualities. Liking for a food is thought to be a long lasting impression and is therefore believed to be substantially involved in modulating the motivational value of a food [FINLAYSON *et al.*, 2008]. Hedonic liking is mediated by the opioid peptide signalling in the hindbrain, midbrain and forebrain regions, such as the NST, the NAcc, the amygdala, the VTA and different hypothalamic nuclei [KANOSKI, 2012].

Otherwise, hedonic 'wanting' is related to the desire or incentive motivation to attain a reward and is triggered by the attraction of a food cue in the environment. Wanting of a food depends on many factors, such as state of hunger or time of the day [FINLAYSON *et al.*, 2008; DALTON and FINLAYSON, 2014]. It is mediated by the neurotransmitter dopamine, respectively by the mesolimbic dopamine system which originates in the brainstem and projects to the NAcc, the prefrontal cortex, the amygdala as well as to the hypothalamus [KANOSKI, 2012].

2.1.3.2 Associative learning processes (Pavlovian conditioning)

Learning cues are primarily common cues, which occur in the environment and are associated with rewarding or punishing sensations and which then gain the ability to control food intake in terms of stimulation or inhibition [PETROVICH, 2011; PETROVICH, 2013].

These findings are typically based on Pavlovian conditioning procedures with rats, which are trained to associate a primarily neutral cue (conditioned stimulus) with a biological important event (unconditioned stimulus). The initially neutral cue becomes a predictive cue and is subsequently used to initiate motivational states, which are capable to override the physiological states. The interaction between the Pavlovian cue and the instrumental behaviour is known as 'Pavlovian-instrumental transfer', by which a feeding environment can stimulate food intake independent from physiological hunger [PETROVICH, 2013; WATSON *et al.*, 2014].

The obesogenic environment in the western world comprises a vast of food-related cues, such as commercials in the television showing palatable, high energy-dense foods. It is

believed to encourage the striving for food and food consumption, as it provides possibilities for associations between foods and cues. Associative learning processes within the obesogenic environment are therefore seen as one of the major causes in the growing number of obesity [JOHNSON, 2013; WATSON *et al.*, 2014].

There are only a few data from human studies to confirm the results of animal studies. However, *Ferriday and Brunstrom (2008)* exposed female participants to smell and sight of pizza for a minute. Participants' prospective and maximum tolerable portion sizes as well as their craving for pizza were interrogated before and after the mentioned cue condition. They were then given a fixed portion of pizza and were instructed afterwards to eat pizza *ad libitum*. Compared to the control group, cueing did increase prospective portion size as well as the subsequent pizza consumption, indicating that cueing is capable to increase the amount of food, above what was actively planned to eat [FERRIDAY and BRUNSTROM, 2008; PETROVICH, 2013].

Furthermore, the setting, in which food is consumed, can lead to specific food-context associations typically known from fast food restaurants, which offer uniform meals across all locations and additionally the restaurants itself are mainly uniform. These circumstances are thought to induce appetite-related associations and might provoke food consumption independent from physiological state of hunger.

Many aspects of eating are driven by learned cues underlying highly sensitive and specific circumstances. Therefore the induced state of hunger cannot be seen as a general state of hunger, but stimulates only the consumption of the signalled food. In turn, the selective consumption of the signalled food contributes to motivational sensations likewise to appetite or food craving [PETROVICH, 2011; PETROVICH, 2013].

2.1.3.3 Memory

As for many learning processes memories are required, they are accepted to be an important influencing factor on food intake and in generating preferences towards foods [HIGGS, 2008].

First striking evidence of the influence of memory on appetitive responses was provided by the results of studies with amnesic patients, such as those of the well studied amnesic patient H.M. in the 1950s. H.M. suffered from memory loss, after major parts

of his medial temporal lobe was removed, including the hippocampus and amygdala, in order to treat his epilepsy. The procedure was successful, but left him in a condition rarely mentioning being hungry. He continued eating another offered meal, even though having eaten recently [HEBBEN *et al.*, 1985; HIGGS and DONOHUE, 2011].

The ability to recall what has been eaten in the past affects food intake in the future in terms of limiting consumption, as the memory of eating a food influences the expectations of satiety and therefore affects the selection of portion size. Situations leading to disturbances in encoding or recalling memories of a meal, such as watching television while eating, might in turn lead to increased food intake along with the next meal [HIGGS, 2008; MARTIN and DAVIDSON, 2014].

Expected satiation belongs to the so called 'semantic memory', dealing with a persons' general knowledge, whilst 'episodic memory' covers autobiographical information. Both types of memory function in an independent way, but nevertheless episodic memory is often biased by semantic memory [HEMMER and STEYVERS, 2009].

Brunstrom and colleagues (2012) investigated the influence of expected satiety to post-meal hunger. Participants were shown either a 300 ml or a 500 ml bowl of soup and then consumed either 300 ml or 500 ml soup. Incongruous eating was achieved by covertly filling or emptying the bowl using a hidden pump, while participants ate. Therefore the amount of actually eaten soup differed from the amount participants perceived to eat in the incongruous eating groups. Appetite was assessed for three hours after the meal consumption and the expected satiation was conducted 24 hours later. Participants who saw 500 ml of soup reported to feel less hungry three hours after the test, than those who saw 300 ml of soup. Similarly, participants who saw 500 ml of soup expected the food to deliver more satiation than those who saw 300 ml of soup. Both results were regardless to the participants' actual soup consumption. Furthermore, those participants who saw 300 ml but actually consumed 500 ml of soup remembered consuming a smaller amount than those participants who saw 500 ml and ate 300 ml of soup. According to these results, memory processes can be defined as an influential factor of satiety and satiation [BRUNSTROM *et al.*, 2012; MARTIN and DAVIDSON, 2014].

Another determinant for controlling subsequent food intake is to suppress unwanted thoughts. Therefore the process of memory inhibition averts the recall of food-related

memories in situations when a person already feels satiated, respectively in situations when food intake is not worthwhile. Individuals who are strictly monitoring their food intake in order not to gain weight are called restrained eaters. Obese restrained eaters are shown to be more likely to experience memories of food and also seem to be more susceptible to environmental food cues. These findings indicate that persons tending to lose control of their food intake also have deficits in memory inhibition. On the other hand, memory inhibition can also lead to a paradoxical increased appearance of intrusive thoughts in obese restrained eaters, who are prone to environmental cues. In other words, when trying to control their memories of food restrained eaters even provoke the upcoming of such thoughts [MARTIN and DAVIDSON, 2014].

2.1.4 Interaction of homeostatic and hedonic regulation of food intake

As mentioned repeatedly on the previous pages, neither the homeostatic nor the hedonic regulation of food intake works exclusively on its own, but rather interact with each other. Metabolic signals are capable to modulate higher brain functions and in turn, reward and cognitive functions can influence the body's metabolic state [Berthoud, 2011].

2.1.4.1 Bottom-up processes

Bottom-up processes refer to conditions when the hedonic reward system is overridden by metabolic signals, leading to weight gain even during reduced caloric intake. Especially food restriction, respectively a negative energy balance increases the occurrence of fasting-induced signals and contemporaneously the rewarding properties of food.

The interaction between homeostatic and hedonic control has been demonstrated in a number of studies using functional neuroimaging techniques, such as functional magnetic resonance imaging (fMRI), in order to investigate changes in the activity of cortico-limbic brain structures. *Goldstone and colleagues (2009)* have demonstrated an increased activation within the ventral striatum, the amygdala, the anterior insula and the medial orbitofrontal cortex when confronting fasting healthy women with visual high-caloric stimuli [GOLDSTONE *et al.*, 2009; BERTHOUD, 2011].

The underlying mechanisms and pathways are widespread and yet not clearly identified. One way of influencing food reward is the modulation of the sensory properties of food, which is mainly derived from leptin, but also from other nutrient agents, such as insulin or GLP-1 [BERTHOUD, 2011]. For example, leptin can suppress sweet taste by influencing leptin responsive lingual taste cells [SHIGEMURA *et al.*, 2004] and can change olfactory qualities by decreasing mucous secretion in olfactory mucosal cells [BADONELL *et al.*, 2009].

Leptin receptors are also capable to affect mesolimbic dopamine neurons, which project to the NAcc and are considerably involved in hedonic ‘wanting’ of food. Conditions of leptin receptor stimulation in the VTA lead to decreased dopamine secretion in the NAcc and inversely leptin receptor silencing increases dopamine release and consequently food intake due to enhanced preference of highly palatable foods [HOMMEL *et al.*, 2006; BERTHOUD, 2011].

Besides this, there is also evidence that lateral hypothalamic orexins and MCH, which act as metabolic signals in energy homeostasis, can interconnect metabolic needs with environmental cues and therefore also control hedonic ‘liking’ [BERTHOUD, 2011]

2.1.4.2 Top-down processes

Top-down processes refer to conditions when peripheral signals and brain systems involved in the homeostatic control of appetite and food intake are overridden by the reward system [BERTHOUD, 2011].

In this context, *la Fleur and colleagues (2010)* attempted an interesting study with overfed rats. Rats which were exposed to a high-fat diet showed expanding plasma levels of the anorexigenic signals leptin and POMC, while plasma concentrations of the orexigenic NPY decreased. Similar plasma concentrations were achieved in rats fed with a high-fat high-sugar diet. An additionally high-sugar diet for one week did not change leptin, POMC or NPY plasma levels. However, although hypothalamic neuropeptide response did indicate an anorexigenic state, rats continued eating and became obese. Thus, these findings underline the previous assumption, that hedonic mechanisms can powerfully override the homeostatic regulation even if only in a rat model [LA FLEUR *et al.*, 2010; BERTHOUD, 2011].

Direct projections from the amygdala, the prefrontal cortex and the NAcc to the lateral hypothalamus are believed as a possible underlying mechanism of top-down processes as the LH receives gustatory and olfactory signals from the mentioned cortico-limbic structures [BERTHOUD, 2011].

Besides the hypothalamic modulation, cortico-limbic structures override homeostatic regulation via subconscious drives including activities within the motor cortex leading to decision-making before the respective signal enters awareness [SOON *et al.*, 2008; BERTHOUD, 2011].

Finally, the connection and crucial interplay between cognitive and emotion related processes, as delineated in *chapter 2.1.3.* have to be taken into account [BERTHOUD, 2011].

2.2 Food intake and behaviour

2.2.1 Executive functions

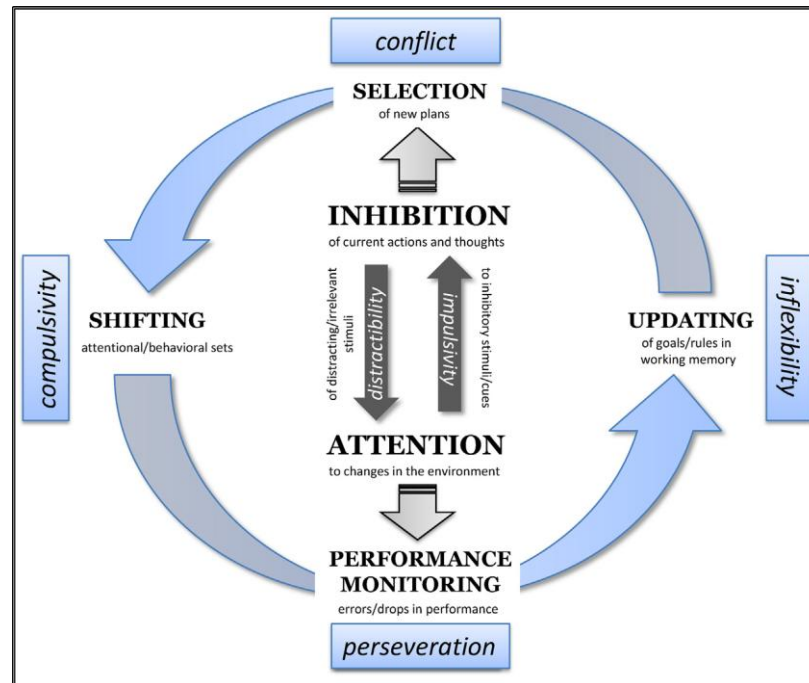
In the industrialized world, highly palatable and high-caloric food is easily obtainable everywhere, which clarifies the pivotal role of efficient self-regulation over food-related temptations in order to prevent weight gain [LOGUE and GOULD, 2013]. There is growing evidence from recent research that obesity is not exclusively due to increased calorie intake and problems in managing weight, but also due to poorer cognitive performance, respectively deficient executive functioning. [CSERJÉSI *et al.*, 2009].

Executive functions are essential for successful self-regulation as they are believed to be a multidimensional complex of higher-order cognitive processes. They enable individuals to adapt to changes in the environment in a sense of regulating reflexive reactions to upcoming stimuli and therefore facilitating goal directed cognitive behaviour. Executive functions include impulse control, response inhibition, attention, working memory, cognitive flexibility, planning, judgement and decision-making. These complex functions are important for normal working in everyday life as they feature future directed, self-controlled and goal directed behaviour [LOGUE and GOULD, 2013; DIAMOND, 2013].

They are mediated by activities of the prefrontal cortex, particularly by the medial prefrontal cortex (mPFC) and the orbital frontal cortex (OFC), and are modulated by neurotransmitter systems such as the dopamine, norepinephrine, serotonin or acetylcholine system. Damages within the frontal lobes result in individuals' poor performance in controlling and regulating complex behaviour. Notably, lesions of the OFC are associated with disrupted response inhibition and reversal learning, whereas lesions of the mPFC affect mainly attentional processes and set-shifting [LOGUE and GOULD, 2013].

For successful functioning of any complex cognitive behaviour process it is furthermore important, that different executive functions are related to each other. As illustrated in *figure 5*, attention and inhibition play a pivotal role in the relationship of executive functions, but only the coordinated action of attention, inhibition and cognitive flexibility maintain an adapted behaviour.

Figure 5: Interaction of executive functions [BARI and ROBBINS, 2013]



Attention is necessary for detecting stimuli respectively signal changes in the internal or external environment and thus requires the inhibition of current actions or thoughts. They are supported by processes, which deliver feedback information about the appropriateness of actions and thoughts and which select and shift to another behavioural set, if necessary. These newly adjusted plans and goals are then again implemented into the interconnected relationship of executive functions and provide new stimuli for attentional processes [BARI and ROBBINS, 2013].

Executive functions can broadly be assumed to three main functions, namely updating, shifting and inhibition. Updating refers to the construct of working memory and enables individuals to withhold information in an active and at all moment retrievable state [HOFMANN *et al.*, 2012; DIAMOND, 2013; ALLOM and MULLAN, 2014]. Working memory is required for reordering items by incorporating previous and actual information into thinking, planning and considering alternatives. It is essential for realising connections between apparently not related information and recombining them in a new way. To maintain focus on goals, goal-related information has to be kept in mind and attention towards tempting stimuli as well as unwanted desires and cravings have to be down-regulated [DIAMOND, 2013; ALLOM and MULLAN, 2014].

Thus, working memory and inhibitory control support each other. To focus on goals, sometimes makes inhibiting internal and external distractions necessary and therefore includes the discipline to complete a task although someone would rather be doing something else [DIAMOND, 2013]. Therefore, there is a strong relation to what is called delaying gratification, which means to voluntarily forego an immediate gratification for the sake of a greater reward later [DIAMOND, 2013; MISCHEL *et al.*, 1989]. In terms of food intake, successful inhibitory control constitutes the ability to resist a highly desirable trigger for food consumption and stop the behavioural response, such as overeating. According to this, weak inhibitory control is associated with more pronounced impulsivity [AMES *et al.*, 2014].

Whilst inhibition is crucial to maintain the focus on goals and plans by inhibiting automatic and prepotent impulses or irrelevant information, shifting is related to the ability to alter goals and perspectives as a consequence of changes in environment [HOFMANN *et al.*, 2012; ALLOM and MULLAN, 2014]. In order to be able to flexibly change perspectives needs to inhibit information or to activate alternative perspectives in mind. Therefore it is obvious, that cognitive flexibility is built on working memory and inhibition and that none of the three core executive functions can work solely on its own [DIAMOND, 2013].

Deficits within any of the executive functions lead to aberrant behavioural conditions. Stimuli-caused strong desires and urges can perform a specific action, which when not inhibited, leads to compulsivity or impulsivity. Compulsivity can be seen as the inability to inhibit inappropriate behaviour and consequently repeating that kind of behaviour over and over again, whilst impulsivity in general means acting without reflection or without considering consequences [BARI and ROBBINS, 2013].

In particular, trait impulsivity comprises of different dimensions including a high sensitivity to reward, difficulties in inhibiting impulses and the contempt of consequences. Notably reward sensitivity and a lack of inhibition are strongly interconnected determinants of food intake and especially of overeating. Therefore, the balance between reward sensitivity, inhibition and the availability of highly palatable foods determine the frequency of eating beyond homeostatic needs as food reward sensitivity is correlated with motivation towards highly palatable foods and accordingly

leads to increased food consumption and obesity. Additionally, enhanced food reward sensitivity reinforces difficulties in inhibiting the strong desire to eat those foods. Inhibition of food-related impulses can occur either automatically, such as during the process of satiation or volitionally, which is associated to knowingly reduce or avoid food intake in order to stop superfluous food consumption. Volitional inhibition is the main mechanism behind restrained eating as increased inhibitory control is associated with decreased hedonically driven food consumption [ELY *et al.*, 2013].

Shifting deficits have been found to be predominant in individuals with *anorexia nervosa* but there is some debate whether this impairment causes *anorexia nervosa* or appears symptomatically. Therefore, Bolton and colleagues have most recently attempted a study to clarify a possible relation between food restriction and the set-shifting ability in healthy women. The newly implemented rule change paradigm within their study allowed comparing participants' set-shifting capacity under conditions of fasting (16 hours prior to the experiment) and satiety. In order to accessorially investigate the potential effect of food itself, they used both food and non-food stimuli. A set of pictures were presented on a screen in connection with a question (Odd?, Even?, High?, Low?) and participants were instructed to press a defined button according to the number of screened pictures. One, two or three pictures were defined as 'low' and 'odd' was the correct answer when one, three or five pictures appeared on the screen. As questions changed periodically the paradigm required mental shifting measured in different reaction times. Shifting deficits were more pronounced under conditions of fasting as well as during shift trials but was not dependent on food stimuli. Furthermore, self-reported weight concern was correlated with decreasing general task performance in the fasted condition. Therefore, the authors concluded that set-shifting deficits might occur in conditions of fasting independently of a given disposition to cognitive inflexibility and that those individuals who are highly concerned with their weight are thought to be more vulnerable towards food deprivation and are more likely to frequently restrict their food intake [BOLTON *et al.*, 2014].

2.2.2 Priming

2.2.2.1 Historical development of priming

The term 'priming' occurred firstly in a 1951 published paper by *Karl Lashley*, who referred the term to a temporary internal activation of response action. He was analysing in which way serial response sequences, such as in speech production, can appear that effortlessly and quickly. He believed that before enunciating a sentence, an aggregate of words is activated. *Lashley* thought priming to be this mediator between the act of intention and the processing of intentional behaviour, which puts the action into the respective serial sequence. He implemented the phenomenon priming into literature as a preparedness of mental representations to serve a response function [LASHLEY, 1951; BARGH and CHARTRAND, 2014].

Lowell Storms unexpectedly discovered the passive influencing capacity of priming in a 1958 conducted study [BARGH and CHARTRAND, 2014]. Participants had to memorize a list of words and then had to freely associate to a list of stimulus words in a subsequent association task. He found that backward associations among previous presented words act as facilitating links and therefore those words became more likely associated. Unfortunately *Storms* could not explain the recency effect [STORMS, 1958].

In 1960 *Segal and Cofer* replicated *Storm's* findings, but did not give explicit recall instructions. Thus, only exposing participants to a list of words without learning them, lead to an increased occurrence of those words within the subsequent conducted association task. Several studies followed in the 1960s and 1970s using priming as an experimental technique for demonstrating the distinction between implicit and explicit forms and uses of memory [BARGH and CHARTRAND, 2014].

In 1977 *Higgins and colleagues* created a masked study which served groundbreaking findings for social psychology as they showed that not only words but also personality trait concepts can be primed. In a first memory task participants were exposed to either positive (e.g. adventurous, self-confident) or negative (e.g. reckless, conceited) terms of personality trait and were then asked to fulfil a reading comprehension task without being aware of the coherence of both test settings. The used paragraph described a person called Donald, who behaved ambiguously to the previous primed personality

trait terms. For instance, he had driven in a demolition derby or did some skydiving and was in general well aware that he could do many things very well. When participants were afterwards asked to describe Donald, those subjects who had been previously primed with positive words also characterised Donald more positively. Inversely, those participants who had been primed to negative terms of personality trait had more negative impressions of the stimulus person. In that way, the study revealed for the first time that a person's previous exposure to prime stimuli can even affect ones perceptual interpretation and subsequent characterisation of another person's behaviour [HIGGINS *et al.*, 1977; BARGH and CHARTRAND, 2014].

More recently studies in cognitive psychology have demonstrated a relation between the perception of a stimulus and the attendant response action, which can be assumed in the term 'priming'. In this manner, a consciously or unconsciously processed stimulus (prime) can alter subsequent behaviour (target) by implicitly activating higher-order concepts [GAILLET *et al.*, 2013].

Priming can affect all stages of stimulus processing, such as attention, comprehension or memory. The processing of a prime stimulus makes the respective content, and furthermore the cognitive concepts used for evaluating and manipulating this content, more accessible. Accessible contents are in turn enabled to influence following judgements, decisions as well as behaviour [JANISZEWSKI and WYER, 2014].

2.2.2.2 Classification of priming

There is a vast of experimental techniques underlying the general term 'priming' and there is no uniform classification of the different priming effects in literature.

A first brief distinction can be done according to whether content is directly or indirectly primed. Direct priming refers to the capacity of the prime stimulus to increase the accessibility of target content as a direct consequence of experiencing the stimulus. Contrary to this, indirect priming occurs when a prime stimulus enhances the accessibility of a content which is associated with the primed content. Therefore, not the primary primed content but the associated content influences subsequent judgements, decisions or behaviour [JANISZEWSKI and WYER, 2014].

Additionally, a commonly used classification of priming refers to the distinction between perceptual and conceptual priming. Perceptual priming is based on the physical features of a prime stimulus and therefore changes in perceptual features of a prime stimulus affect perceptual priming. Furthermore priming effects are greatest when prime stimulus and target are broadly similar. Perceptual prime stimuli include pictures or words as well as visual, auditory or tactile information, but are highly modality specific. A common perceptual priming task is the completion of word fragments after studying a list of words. Previously seen words are more accessible and are therefore more likely to be used for completing the fragments (i.e. Elephant → E_ _ p_ _n_) [FOERDE, 2010].

Conceptual priming in contrast is based on semantic properties of a stimulus and refers to manipulations that activate internal mental representations, which influence a subsequent unrelated content passively and unintended so that the participant does not recognize the relation between activation and subsequent influence. Conceptual priming is not modality specific and increases the deeper the prime stimulus' semantic information is. In other words, previous experiences with the representation-relevant stimuli can facilitate the completion of a task without intentionally using the studied information. For example, answering general knowledge questions can be relieved by the semantic properties of previous studied prime stimuli (i.e. Elephant → What animal did Hannibal use to cross the alps?) [FOERDE, 2010; BARGH and CHARTRAND, 2014].

In general all types of priming paradigms have some common characteristics:

- Priming effects only emerge if both a prime stimulus and a target stimulus exist.
- The prime stimulus, respectively specific characteristics of the prime stimulus, leads to changes in response to the target stimulus.
- The prime stimulus' influence is temporary.
- Priming effects are unintended and arise without participants' awareness [JANISZEWSKI and WYER, 2014].

2.2.2.3 Important types of priming

Semantic priming

Semantic priming is a widely used tool to investigate cognitive processes which are associated with memory and language, perception and attention.

The paradigm is typically used within lexical decision tasks to investigate in which way words are accessed, comprehended and integrated with other words in sentence understanding. A target word, which is preceded by either an unrelated or a semantically related word prime, is presented and participants' response, such as the time needed for a lexical decision, is measured. Responses to target words occur more quickly and accurate after presentation of related than unrelated word primes [LUCAS, 2000; ORTELLS *et al.*, 2006].

The most popular interpretation and explanation of semantic processing is based on the believe that memory comprises of different interconnected nodes. According to the spreading-activation theory of *Collins and Loftus (1975)* the presentation of a prime word firstly activates the corresponding node in memory. Activation then spreads to linked nodes and afterwards to nodes linked to the previous activated nodes. Therefore, activation expands continuously, but with decreasing intensity which is inversely proportional to the accessibility of the links. In other words, the more related the prime stimulus and the target stimulus are, the more closely related are the respective nodes and activation can spread faster [COLLINS and LOFTUS, 1975; LUCAS, 2000; ORTELLS *et al.*, 2006].

Affective priming

Affective priming refers to a facilitated response to a target stimulus when it is preceded by a congruent prime stimulus in terms of affective valence. Therefore, prime stimuli make affective states, such as emotions or mood, more accessible which in turn can influence judgements about unrelated stimuli. The most commonly used affective priming paradigm pertains to categorisation of a word as being either positive or negative [FERRÉ and SÁNCHEZ-CASAS, 2014; JANISZEWSKI *et al.*, 2014].

Ferré and Sánchez-Casas (2014) conducted a lexical decision task in order to investigate whether semantic relatedness contributes to affective priming effects. Affective congruence and semantic relatedness were manipulated between prime and target stimuli. When using concrete words within the task, semantic effects could be observed regardless of affective congruence. When using abstract words participants responded faster when prime and target stimuli were congruent, indicating an affective priming effect [FERRÉ and SÁNCHEZ-CASAS, 2014].

The underlying mechanism of affective priming effects is again related to spreading activation as emotions are represented by a memory node. Thus, the distinction between semantic and affective priming effects is difficult, particularly because affective effects are highly elusive whereas semantic priming is a robust phenomenon [FERRÉ and SÁNCHEZ-CASAS, 2014; JANISZEWSKI *et al.*, 2014].

Goal priming

Goals are represented in memory as knowledge structures and are therefore underlying similar activation pathways than presented in semantic processing. Internal states and external stimuli activate the mental representation of goals and means, such as behaviours that enable someone to pursue his goals. The activation makes goals and means more accessible in memory leading to respectively faster and easier recognition and recall [VAN OSSELAER and JANISZEWSKI, 2012]. The motivational source for goal pursuit is based both on positive affect as well as on deprivation of crucial resources such as fluid or food, and interacts with mental representations [VELTKAMP *et al.*, 2009]. While direct goal priming activates concepts which stimulate respective behaviours in order to attain the goals, indirect goal priming refers to the activation of contents which in turn spread activation to an associated goal [JANISZEWSKI *et al.*, 2014].

Behavioural priming

As previously discussed, behaviour is related to semantic, affective and goal related information and can therefore be indirectly primed. But also direct behavioural priming can occur when observing a specific behaviour makes the mental representation of the behaviour more accessible and therefore more likely to be executed.

A typical form of direct behavioural priming is mimicry, which means that people mirror behaviour of other people, such as verbal behaviours (e.g. accents) or body movements [JANISZEWSKI *et al.*, 2014]. Food consumption and preference can also be influenced by behavioural mimicry as shown by *Tanner and colleagues (2008)*. Participants were told to watch a video in which a confederate described several advertisements and were briefed to memorise the given descriptions for they were subsequently asked about their memory of those advertisements. The confederate as well as the participants had access to a bottle of water and two different types of crackers (animal and goldfish shaped crackers) and were told to freely help themselves to the snacks and water at any time. In a suppositious unrelated task they were then asked to rate their preference of several snacks including the animal and goldfish crackers. During the task the confederate ate only one of two given snacks. Participants mimicked the snacking behaviour of the confederate during watching the videotape and also the participants' preference ratings within the second task were consistent with their snacking behaviour [TANNER *et al.*, 2008].

2.2.2.4 Priming and inhibitory control

Priming effects on subsequent food-related behaviour are mainly investigated based on the specific response of restrained eaters to food cues [GAILLET *et al.*, 2013].

Although restrained eaters are chronically concerned with controlling their weight and with restricting the amount of food they are eating, they are less successful in these attempts. Restrained eaters are reported to have a stronger preference for palatable food than unrestrained eaters and have due to this greater difficulties in resisting tempting foods [PAPIES *et al.*, 2009]. The underlying mechanism is thought to be caused by a higher accessibility of the hedonic aspects of food in dieters' mind guiding their cognitions and behaviour and therefore making the process of consumption more likely. The reward-related activation is more pronounced in restrained eaters than in unrestrained eaters leading to an increased motivation to eat [PAPIES *et al.*, 2007; PAPIES *et al.*, 2009].

Papies and colleagues (2007) have investigated the spontaneous activation of hedonic thoughts in restrained and unrestrained eaters in response to palatable food cues in a lexical decision task. Participants were confronted to experimental sentences both

referring to eating behaviour and palatable food. Furthermore control sentences not referring to those items were created. Each sentence was followed by a hedonic probe word, such as 'delicious', which did not occur in one of the experimental sentences and participants had to indicate as quickly as possible whether the word was part of the previous sentence or not. Restrained eaters reacted significantly slower to hedonic probe words following sentences containing palatable food cues compared to those containing neutral food items, whereas unrestrained eaters did not. Although only confronting restrained eaters to verbal food cues and verbal descriptions of eating behaviour, the findings suggest that perceiving palatable food or reading about it leads to the activation of hedonic thoughts in restrained eaters indicating a rather motivational eating-regulation [PAPIES *et al.*, 2007].

These findings are consistent to the Goal Conflict Model of Eating, which refers to the conflict between two incompatible goals in restrained eating behaviour. On the one hand eating palatable food is highly desirable for restrained eaters whereas on the other hand they chronically want to reduce or control weight. Therefore, in order to succeed in one goal, restrained eaters need to inhibit the other goal. Unfortunately, an increased extent of palatable food stimuli primes the goal of enjoying palatable foods, subsequently resulting in the inhibition of the eating control goal and leading to weight gain [STROEBE *et al.*, 2008].

Gaillet and colleagues (2013) have explored the impact of olfactory food cues on food choice. In two separate experiments melon and pear food aroma was diffused in the laboratory in a low intensity not consciously notable for the participants. After completing a lexical decision task participants were asked to choose a three-course meal from a given list (menu task). Within the lexical decision task, participants who were primed to the melon odour reacted faster to the word 'melon' than to other words and even faster compared to other fruity words. Furthermore they showed a greater tendency for choosing a starter with vegetables than participants of the control group, while there were no differences in the choice of the main course or dessert. When using the pear odour, there were no differences in reaction times within the lexical decision task, but those participants who were primed with the pear odour, chose fruity desserts more often. Although these findings indicate an activation of a specific cognitive concept (fruit and vegetable concept), the priming effects seem to be specific to the food cue.

For instance, pears are more often used for desserts than for other courses and therefore priming with the pear odour led to rather choosing fruity desserts. The stronger the link between the prime and the respective mental representation, the stronger the priming effects are thought to be [GAILLET *et al.*, 2013].

Nevertheless, hyperphagia is considered to be not only determined by a strong reward-related activation of respective brain areas but also by a lack of an individuals' inhibitory control. Therefore, the tendency for overeating might be due to an interrelation of bottom-up processes, such as reward sensitivity and top-down processes such as impulsivity. Individuals who tend to overeat react more impulsive especially when confronted with highly palatable and high caloric food stimuli [MEULE and KÜBLER, 2014; MEULE *et al.*, 2014].

The relationship between trait impulsivity and trait food craving as well as their impact on subsequent food intake has been recently investigated by various authors (e.g. MEULE and KÜBLER, 2014; MEULE *et al.*, 2014; TESLOVICH *et al.*, 2014) using a go/no-go task with pictures of foods. To additionally examine, whether an individuals' extent of impulsivity can be even enhanced by repeated exposure to high caloric food primes, the present thesis was created. The study design for the used go/no-go task was conceptualised analogues to MEULE and KÜBLER (2014) and is outlined in *chapter 3.5.2*.

3 Materials and methods

3.1 Study design

The study was designed as a camouflaged attention test. Therefore the experimenter made the students believe they participate in an attention test among undergraduate students called “attention and food”, while the real interest was whether a two week priming intervention does change the outcome parameters associated with inhibitory control in young females.

3.2 Ethics

The study design was submitted to the Ethics Committee of the University Vienna on the 13th of March 2014 and was approved (reference number: 00064) on the 7th of April 2014. All participants provided informed written consent before participating in the study.

3.3 Participants

The recruitment of the participants was conducted from the 14th of February 2014 until the 26th of March 2014. To attain a preferably homogenous study sample, only female undergraduate students of nutritional sciences, in the age ranging from 18 to 40 years were enlisted.

They were hired via announcements on blackboards at the department of nutritional sciences, postings in the official student forum of nutritional sciences and via the social network *Facebook*.

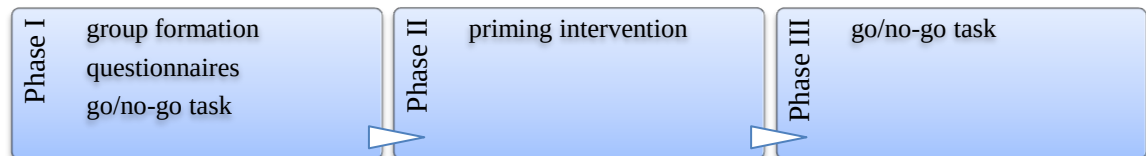
The main inclusion criterion was that participants possess a smartphone supporting the messenger application *WhatsApp*.

Students received a written confirmation for the participation in the study.

3.4 Procedure

The study was conducted from the 17th of March until the 12th of April at the facilities of the Department of Nutritional Sciences (1090 Vienna, Althanstraße 14, UZA II) and comprised three experimental phases as stated in *figure 6*.

Figure 6: Study procedure



3.4.1 Phase I: Go/no-go task

Participants were tested between 9:30 a.m. and 6:30 p.m., whereby testing of each person took at maximum 30 minutes.

Students were divided into two test groups by randomly drawing the number of the group [WEI and LACHIN, 1988]. Group 1 was called the “high caloric group” (HC group) and group 2 was defined as the “low caloric group” (LC group).

They were briefed in the run-up to the task not to consume any food or beverages at least two hours before the experiment and were then firstly invited to drink a 125 ml glass of apple juice (*Höllinger Bio*), in order to ensure comparable blood glucose levels within the study sample [WEISSMANN and BINAH, 2014].

The experimenter gave all necessary information about the following study and participants signed the written informed consent.

The first go/no-go task was created in order to obtain baseline information of the participants overall task performance. Prior to performing the task each participant completed the questionnaires delineated in *chapter 3.5.1*.

3.4.2 Phase II: Priming intervention

The priming intervention phase started immediately on the day after conducting the first go/no-go task. Participants received one modified picture and the appropriate original photo over a time period of 14 days, including weekend, via the messenger *WhatsApp*.

Pictures of high caloric foods or low caloric foods were sent each day between 9.00 a.m. and 10:00 a.m. depending on the group affiliation.

Participants were briefed prior to the first intervention day that there was at least one mistake in each modified picture and they were asked to find as many hidden mistakes in the photos as possible, without knowing the maximum number. The predefined timeframe for completing each task was four hours. Participants returned their results by describing the detected mistakes and by stating the time it took to find all changes.

Spending a maximum of time on analysing the photos, respectively strengthen the priming effect, was of higher importance than detecting all inserted mistakes.

3.4.3 Phase III: Go/no-go task

Within a timeframe of two days after completing the priming intervention phase, participants conducted the second go/no-go task, which was proceeded under the same conditions as described in *chapter 3.4.1*.

3.5 Measurements

3.5.1 Questionnaires

Mood Questionnaire (Aktuelle Stimmungsskala, ASTS)

The ASTS is a self-report questionnaire in German language for assessing the actual subjective mood and can be used for describing mental health. It has been modelled after the well approved “Profile of Mood States” (POMS) and is an abridged version of the existing German short version of POMS.

The ASTS consists of 19 items. Within each item the response scale is divided into seven categories ranging from “very strong” to “not at all”. The items are summed up to the five subscales “depression”, “hopelessness”, “fatigue”, “anger” and “vigour”. The total score, used in the current study, provides the extent of participants’ negative feelings [DALBERT, 2009].

Flexible und rigid control of eating behaviour (FRK)

These scales were developed to assess the extent of restrained eating behaviour, which includes flexible restraint as well as rigid restraint. Flexible dieting can lead to more successful weight control, because foods are limited in quantities when necessary. Contrary to this, rigid restraint can be explained as an all-or-none principle, which means that periods of dieting follow periods where foods are consumed freely. Rigid restraint is associated with less successful long-term weight control [WESTENHÖFER *et al.*, 2013]. In the current study only rigid restraint was assessed by a 16-item scale, consisting of a response scale divided in the categories “applicable” and “not applicable”.

Barratt Impulsiveness Scale – short form (BIS-15)

The BIS-15 is the short version of the 11th version of the Barratt Impulsiveness Scale and consists of 15 items for measuring the construct of impulsivity, which is mainly characterized by rapid und unplanned actions without considering the consequences [MEULE *et al.*, 2011].

Within each item the response scale is divided into four categories ranging from “rarely/never” to “almost always/always”. The items are summed up to the three subscales “attentional impulsivity”, “motor impulsivity” and “non-planning impulsivity”. The total score can be conducted by adding up all subscale scores and was used in the current study.

Perceived Self-Regulatory Success in Dieting Scale (PSRS)

The PSRS questionnaire can be used to distinguish successful dieters from unsuccessful dieters. It consists of three items and a 7-point response scale ranging from “not successful” to “very successful” within each item. Participants have to mark how successful they are in watching their weight, in losing extra weight and how difficult it is to stay in shape [MEULE *et al.*, 2012].

Food Cravings Questionnaire – Trait – reduced version (FCQ-T-r)

This self-report questionnaire to measure the frequency of food craving experiences is the short version of the Food Cravings Questionnaire-Trait. Food craving means a strong desire for food and although it is highly related to hunger, food craving differs from plain hunger in terms of intensity and specificity.

The questionnaire consists of 15 items, which deal with lack of control over eating, thoughts about and intentions to consume food as well as emotions before and during food craving and possible trigger cues for food craving. The 6-point response scale of each item ranges from “never” to “always” [MEULE *et al.*, 2014].

Bratman’s Orthorexia Test (BOT)

The Bratman’s Orthorexia Test is a short screening tool, which allows the diagnosis of the eating disorder *orthorexia nervosa*, which can be considered as a pathologic obsession for naturally pure foods. Affected people pay too much attention to consuming healthy foods and also to quality of foods [BRYTEK-MATERA, 2012]. The questionnaire consists of 10 items with the answer options “yes” or “no”.

Visual Analogue Scale (VAS)

Visual analogue scales are commonly used psychometric tools to measure appetite sensations. They obtain neither objective nor strictly quantitative results but they are able to predict aspects of food intake behaviour [STUBBS *et al.*, 2000].

VAS are most often composed of 100 mm horizontal lines with the extremes of the sensation feeling at each end of the line. Endpoints are expressed by words and/or numbers. Participants quantify their subjective feeling by placing a mark across the line, which can then be quantified by measuring the distance from the left endpoint to the set mark [CHAPUT *et al.*, 2010].

In the current study separate visual analogue scales for hunger (How hungry do you feel?) and fullness (How full do you feel?) with the anchor points “0 = not hungry” and “100 = very hungry” respectively “0 = not full” and “100 = very full” were used at the beginning and in the end of the go/no-go task.

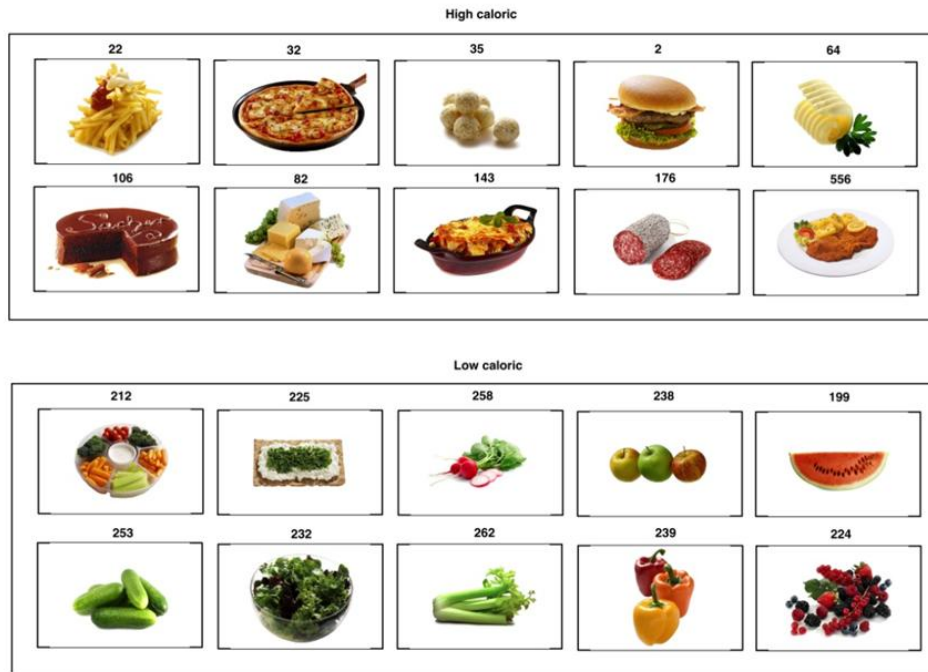
3.5.2 Go/no-go task

The go/no-go task is an often used method to measure the extent of self-control, respectively impulsivity, which means a lower inhibitory control expressed in an inability to suppress inappropriate behaviours for the benefit of appropriate ones. Impulsivity is often accompanied by higher food intake among healthy individuals.

Within the go/no-go task participants have to react as quickly as possible to a shown target but withhold the response to a non-target [TESLOVICH *et al.*, 2014].

In the current study ten pictures of high caloric and ten pictures of low caloric foods were selected from the *food.pics database* (figure 7) and were used for the go/no-go task. The declared numbering of the pictures refers to original numbering within the database [BLECHERT *et al.*, 2014].

Figure 7: Selected high caloric and low caloric pictures for the go/no-go task



The items of each target group differed in calorie content per 100 g as well as calorie content per item (see *table 1*), but did not differ in terms of brightness, contrast, complexity and palatability (all $p > .05$).

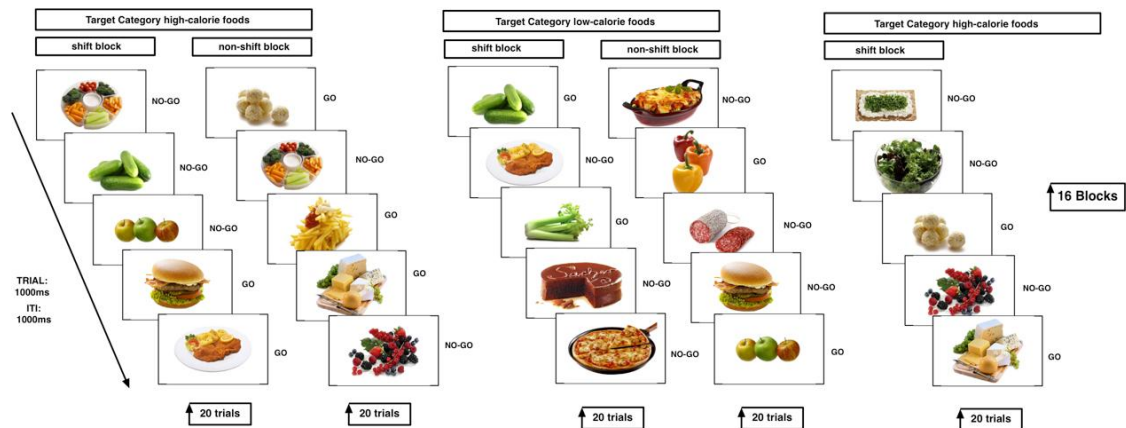
Table 1: Physical features of selected pictures in the go/no-go task

	High caloric food target		Low caloric food target		<i>p</i>
	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	
Calories per 100 g	349.60	196.26	40.40	42.48	.001
Calories per item	1110.90	951.83	106.60	130.53	.009

The go/no-go task was created within the free software program *PsychoPy - Psychology software in Python version 1.79.01* [PEIRCE, 2009] and was displayed on a 23" screen. As stated in *figure 8* the task was composed out of 16 blocks consisting of 20 trials each. The pictures were randomly presented. Each picture appeared once per trial for 1000 ms and a blank screen was shown for 1000 ms during inter-trial interval. Students were asked to press the spacebar as soon as a target item was shown, but to suppress the response when a non-target item, a so called distractor, appeared.

Before each block, the target category was defined and blocks were displayed in two different orders (HC-HC-LC-LC-HC-HC-LC-LC-HC-HC-LC-LC-HC-HC-LC-LC or LC-LC-HC-HC-LC-LC-HC-HC-LC-LC-HC-HC-LC-LC-HC-HC) according to the group belonging of the participants as set out in *chapter 3.4.1*.

Figure 8: Exemplary procedure of the go/no-go task



Those blocks with the same target category than in the block before, and therefore the same stimulus-response relation, were called non-shift blocks, whereas shift blocks were defined as those blocks with reverse stimulus-response relation. Written instructions or the task were presented on the screen. Participants had to do a practice block before the real task started, in order to already arrange the first block as a shift-block. Participants' overall reaction times to go-trials, the number of omission errors (missing responses to target items) as well as the number of commission errors (responses to distractors) were defined as target values. As a high number of commission errors is associated with low inhibitory control, this target value was of particular interest in the current study.

3.5.3 Priming intervention

Pictures used for the priming intervention were selected from *Google search* within the search tool "labelled for reuse with modification". Search criteria were high caloric or low caloric food in general as well as particular dishes. Despite this, photos from *foodspotting.com* were used, after getting the permission for reuse and pictures from the *Foodcast Research Image Database (FRIDa)* [FORONI *et al.*, 2013]. According to the

intervention groups, 14 pictures both of high caloric and low caloric foods were prepared by using the software Adobe® Photoshop® 7.0. Therefore, each photo was modified by inserting five mistakes.

3.6 Data analysis

Data were analysed with *IBM Statistics 21*. Diagrams were partly computed in *Microsoft Excel 2007* and *IBM Statistics 21*.

Sample characteristics (age, BMI and food deprivation), questionnaire measures and priming measures were evaluated using descriptive analysis. Additionally, t-tests for independent samples were conducted to test whether mean values differentiated between both intervention groups.

The extent of hunger and fullness before and after each go/no-go-task was compared using paired-samples t-tests.

In each go/no-go task the dependent variables were defined as reaction times in milliseconds during go-trials, the number of commission errors and the number of omission errors. For each dependent variable a separate 2x2 ANOVA for repeated measures [(target type: HC vs. LC) x (block type: shift vs. non-shift)] was conducted in a first step.

In a second step, a 2x2x2 ANOVA for repeated measures [(target type: HC vs. LC) x (block type: shift vs. non-shift) x (testing phase: phase I vs. phase III)] was used in order to determine, whether the priming intervention did affect the participants' inhibitory control.

Furthermore correlations between body mass index (BMI), food deprivation, questionnaire measures and the number of commission errors were evaluated using Pearson's correlation coefficient.

Linear regression analyses were conducted for evaluating the predicting capacity of measures for the number of commission errors.

3.7 Problems

3.7.1 Go/no-go task

The following conceptual and textual problems occurred during the affective shifting tasks:

First, some of those students, who attended the go/no-go task later in the afternoon, reported difficulties in concentrating on the task. Some of them even gave an advance warning to the experimenter, that there would be many mistakes in their performance due to fatigue.

Second, there were difficulties among some participants in deciding on the pictures' belonging within the affective shifting task to either high caloric or low caloric food. The experimenter was frequently told that it took a few blocks to opt for one target category.

Third, there were a few images within the go/no-go task which were misinterpreted in terms of calorie level and some of the shown foods were difficult to identify:

- *Picture 35 (Raffaello)* was sometimes associated with millet balls or small dumplings. In total 14 % of the participants wrongly assigned this picture to the low calorie category.
- *Picture 64 (butter)* was often assigned to low calorie foods, because only small amounts of butter are eaten during a meal. One participant also argued her choice because of the fatty acid composition of butter. Many participants reported that they were confused by the parsley besides the butter. In total 6 % of the participants wrongly assigned this picture to the low calorie category.
- *Picture 82 (cheese)* was associated with healthy foods and therefore was misinterpreted as low calorie food from 5 % of the participants.
- *Picture 199 (watermelon)* was assigned to high calorie food by one participant due to its high amount on fructose. In total 1 % of the participants wrongly assigned this picture to the high calorie category.

3.7.2 Priming intervention

The following conceptual problems occurred during the priming intervention phase:

First, not all of the students could receive their results within the given time due to participation in other courses. Additionally the task was not completed in time due to lack of WLAN connectivity, when participants did a trip abroad.

Second, participants were briefed prior the intervention, that there was at least one mistake in each picture but they did not know the maximum number of hidden changes. Because some of the modifications were much easier to detect than others, parts of the participants did not spend much time to detect possibly more than the obviously visible mistakes.

3.8 Sub-Hypotheses

In order to carry out the main hypotheses, which have already been stated in *chapter 1*, the following sub-hypotheses were defined:

- Sub-hypothesis I

Does the point in time of last food consumption influence the number of commission errors in the go/no-go task?

H0: Food deprivation does not influence the number of commission errors.

H1: Food deprivation influences the number of commission errors.

- Sub-hypothesis II

Does the state of mood influence the number of commission errors in the go/no-go task?

H0: The state of mood does not influence the number of commission errors.

H1: The state of mood influences the number of commission errors.

- Sub-hypothesis III

Does the self-reported success in dieting influence the number of commission errors in the go/no-go task?

H0: The self-reported success in dieting does not influence the number of commission errors.

H1: The self-reported success in dieting influences the number of commission errors.

- Sub-hypothesis IV

Does the self-reported trait impulsivity influence the number of commission errors in the go/no-go task?

H0: The self-reported trait impulsivity does not influence the number of commission errors.

H1: The self-reported trait impulsivity influences the number of commission errors.

- Sub-hypothesis V

Does the self-reported trait food craving influence the number of commission errors in the go/no-go task?

H0: The self-reported trait food craving does not influence the number of commission errors.

H1: The self-reported trait food craving influences the number of commission errors.

- Sub-hypothesis VI

Does the self-reported restrained eating behaviour affect the number of commission errors in the go/no-go task?

H0: The self-reported restrained eating behaviour does not influence the number of commission errors.

H1: The self-reported restrained eating behaviour influences the number of commission errors.

- Sub-hypothesis VII

Does the self-reported orthorectic eating behaviour influence the number of commission errors in the go/no-go task?

H0: The self-reported orthorectic eating behaviour does not influence the number of commission errors.

H1: The self-reported orthorectic eating behaviour influences the number of commission errors.

4 Results

4.1 Sample size

A total of 86 female undergraduate students of nutritional sciences participated in the current study.

Data from one participant were excluded from further statistics, because there was no stimulus-response registered over a period of two blocks in the first go/no-go task. Therefore the statistical analysis was generated with data from a total of 85 participants ($n = 85$). The HC group comprised 42 students, whereas 43 participants formed the LC group.

Moreover, data from picture 35 and picture 64 shown in the go/no-go task were excluded due to misinterpretation of the target category as described in *chapter 3.7.1*. The cut-off point for exclusion was that less than or as much as 95 % of participants classified the picture as high calorie or low calorie food correctly.

4.2 Sample characteristics

Sample characteristics such as age and BMI in the total sample as well as group-related were analysed.

Age

The mean age of participants was 21.46 years ($SD = 2.85$) ranging from minimum 18 years to maximum 31 years. The median age was 21 years and 50 % of the participants were aged between 19 years and 23 years as illustrated in *figure 9*.

The group-related distribution of participants' age is illustrated in *figure 10*. Therefore the mean age of participants in the HC group was 21.67 years ($SD = 2.79$) ranging from minimum 18 years to maximum 31 years. The median age was 21.50 years and 50 % of the participants were aged between 19.75 years and 23.00 years.

The mean age of participants in the LC group was 21.26 years ($SD = 2.98$) ranging from minimum 18.00 years to maximum 30.00 years. The median age was 21.00 years and 50 % of the participants were aged between 18.73 years and 21.82 years.

Figure 9: Age distribution among participants

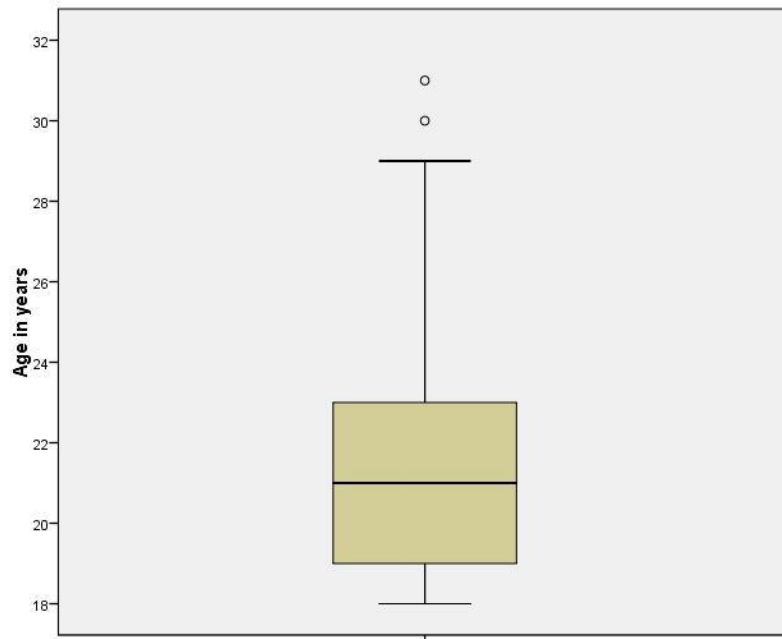
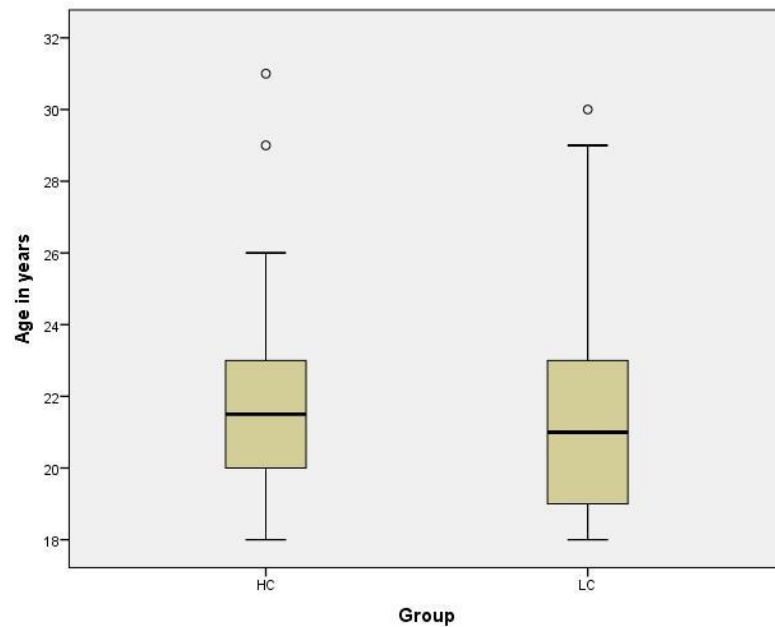


Figure 10: Age distribution among intervention groups



To test whether there were differences in mean age between both intervention groups, a t-test for independent samples was conducted. There were no significant differences between the HC group and the LC group ($t(83) = 0.66, p = .510$).

BMI

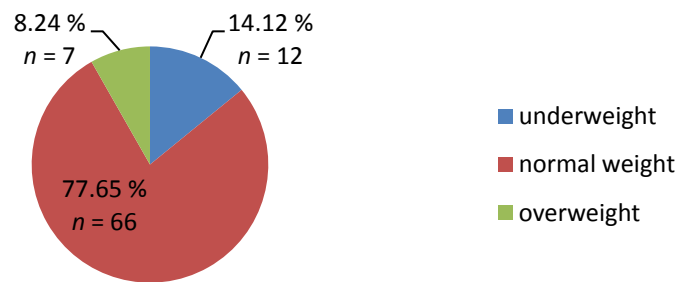
The BMI is a simple tool using weight and height to classify underweight, overweight or obesity in adults. The BMI is independent of age or gender in adults and is calculated using the following formula:

$$\text{BMI [kg/m}^2\text{]} = \text{weight [kg]} / \text{height [m]} * \text{height [m]}$$

The WHO classifies underweight as $\text{BMI} < 18.50$, overweight as $\text{BMI} \geq 25.00$ and obesity as $\text{BMI} \geq 30.00$ [WHO, 2000]. Therefore normal weight ranges between 18.50 and 24.99 kg/m^2 .

Following the WHO classification, 14 % of the participants can be classified as underweight, 78 % as normal weight, 8 % as overweight and none of the participants as obese.

Figure 11: BMI distribution according to WHO classification



The mean BMI of participants in total was 20.97 kg/m^2 ($SD = 2.35$) ranging from minimum 17.01 kg/m^2 to maximum 26.53 kg/m^2 . The median BMI was 20.70 kg/m^2 and 50 % of the participants ranged between 19.00 kg/m^2 and 22.20 kg/m^2 (figure 12).

The mean BMI of participants in the HC group was 20.62 kg/m^2 ($SD = 2.36$) ranging from minimum 17.01 kg/m^2 to maximum 26.53 kg/m^2 . The median BMI was 20.28 kg/m^2 and 50 % of the participants ranged between 18.73 kg/m^2 and 21.82 kg/m^2 as stated in figure 13.

The mean BMI of participants in the LC group was 21.31 kg/m^2 ($SD = 2.35$) ranging from minimum 17.36 kg/m^2 to maximum 26.26 kg/m^2 . The median BMI was 20.76 kg/m^2 and 50 % of the participants ranged between 19.79 kg/m^2 and 22.72 kg/m^2 (figure 13).

Figure 12: BMI distribution among participants in total

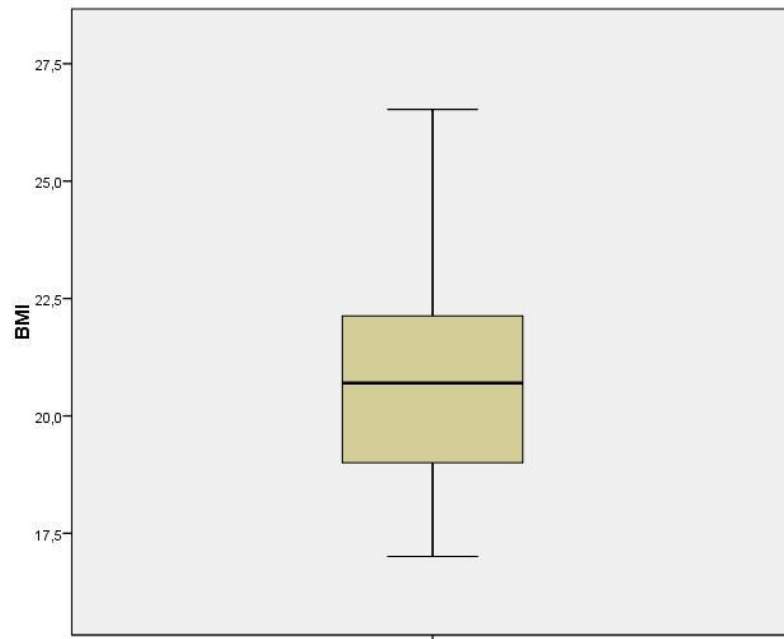
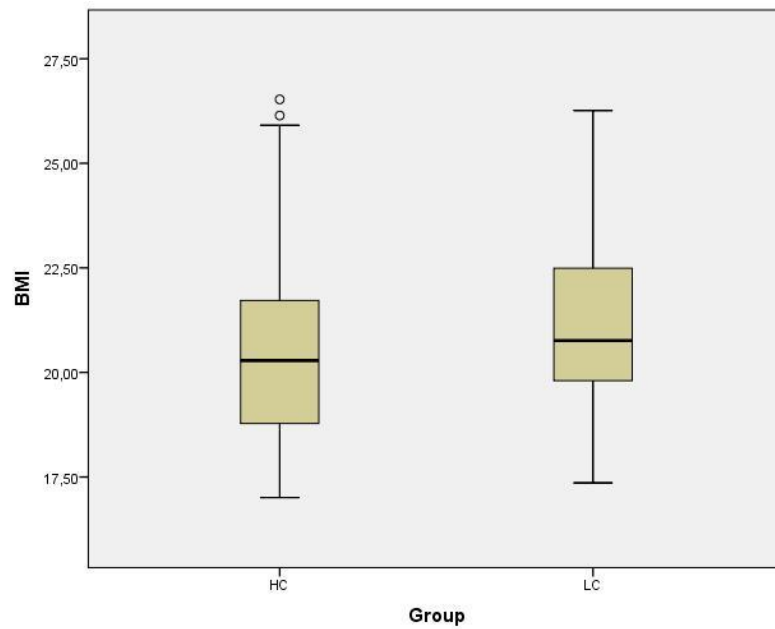


Figure 13: BMI distribution among intervention groups



A t-test for independent samples was conducted to test whether mean BMI differed between the intervention groups. There were no significant differences between the HC group and the LC group ($t(83) = -1.36, p = .179$).

4.3 Questionnaire scores

Prior to the go/no-go task in phase I, participants were asked to complete different questionnaires, in order to assess covariates, which might affect subsequent task performance. Relevant score results were incorporated in further analysis and presented within the following chapters.

Assessment of actual subjective mood

Using the ASTS score participants' actual subjective mood was ascertained and furthermore classified into the subscales "depression", "hopelessness", "fatigue", "anger" and "vigour". Subscales were summed up to total score of negative feelings.

A median split was then conducted to subclassify the frequencies within the subscales and the total score.

As depicted in *table 2*, there were lower scores in the subscales "depression", "hopelessness", "fatigue" and "anger" and higher scores in the subscale "vigour", indicating participants mainly positive mood state, reflected also in total score for negative feelings.

Table 2: Frequencies among the subscales of the ASTS

		Depression		Hopelessness		Fatigue		Anger		Vigour		Total	
		low	high	low	high	low	high	low	high	low	high	low	high
Total	<i>n</i>	46	39	60	25	45	40	72	13	38	47	44	41
	%	54	46	71	29	53	47	85	15	45	55	52	48
HC group (n = 42)	<i>n</i>	23	19	31	11	22	20	36	6	17	25	19	23
	%	55	45	74	26	52	48	86	14	40	60	45	55
LC group (n = 43)	<i>n</i>	23	20	29	14	23	20	36	7	21	22	25	18
	%	54	46	67	33	54	46	85	15	49	51	58	42

Additionally, a t-test for independent samples was conducted to test whether mean total scores differed between the HC group and the LC group. There were no significant differences according to the subjective mood state between both intervention groups ($t(83) = 1.19, p = .239$).

For testing internal consistency and reliability of the score, Cronbach's alpha was used. According to the literature acceptable values range between $\alpha = .70$ and $\alpha = .80$, but

depend on the included number of items and characteristic of the psychometric tool [FIELD, 2005].

Internal consistency of the German version of the scale is $\alpha = .83$ to $\alpha = .94$ [DALBERT, 2009] and was $\alpha = .55$ in the current study using the total score indicating relatively low reliability.

Assessment of rigid restrained eating behaviour

For assessing participants' extent of rigid control of eating, the FRK scale was used. Again a median split was conducted in order to divide the participants into rigid restrained eaters and non-rigid restrained eaters. Therefore, the majority of participants in total as well as group-related could be classified as non-rigid restrained eaters as depicted in *table 3*.

Table 3: Classification of restrained eating behaviour

		Rigid restraint	
		low	high
Total	<i>n</i>	58	27
	%	68	32
HC group (<i>n</i> = 42)	<i>n</i>	27	15
	%	64	36
LC group (<i>n</i> = 43)	<i>n</i>	31	12
	%	72	28

A t-test for independent samples was conducted to test whether mean total scores differed between the HC group and the LC group. There were no significant differences according to the self-reported restrained eating behaviour between both intervention groups ($t(83) = 0.77$, $p = .446$).

Internal consistency of the test in former studies is $\alpha = .81$ [MEULE *et al.*, 2012] and was $\alpha = .60$ in the current study indicating relatively low reliability.

Assessment of trait impulsivity

Using the Barratt Impulsiveness Scale participants' construct of impulsivity was measured. Items were summed up to the three subscales "attentional impulsivity",

“motor impulsivity” and “non-planning impulsivity” and frequencies among the subscales were assessed via a median split. Results for the entirety of participants as well as group-related results are illustrated in *table 4*.

Table 4: Frequencies among the subscales of the BIS-15

		Attentional impulsivity		Motor impulsivity		Non-planning impulsivity		Total impulsivity	
		low	high	low	high	low	high	low	high
Total	<i>n</i>	43	42	49	36	54	31	52	33
	%	51	49	58	42	63	37	61	39
HC group (n = 42)	<i>n</i>	24	18	26	16	30	12	30	12
	%	57	43	62	38	71	29	71	29
LC group (n = 43)	<i>n</i>	19	24	23	20	24	19	22	21
	%	44	56	54	46	56	44	51	49

The extent of attentional impulsivity, indicating an inability to focus concentration, and motor impulsivity, as acting without considering the consequences, was distributed nearly equally among participants. On the contrary there were higher scores of low non-planning impulsivity among students. In total 39 % ($n = 33$) of participants were classified as impulsive.

A t-test for independent samples was conducted to test whether mean total scores of the BIS-15 differed between the HC group and the LC group. There were no significant differences between the intervention groups ($t(83) = -1.94, p = .056$).

Internal consistency of the German version of the test is $\alpha = .81$ [MEULE *et al.*, 2011] and was $\alpha = .80$ in the current study when using the total score, indicating equal reliability.

Assessment of success in dieting

The assessment of participants’ success in dieting was conducted via median split of the total score of the PSRS, indicating a quite equal distribution between successful dieters and unsuccessful dieters as stated in *table 5*.

Table 5: Classification of success in dieting

		Success in dieting	
		low	high
Total	<i>n</i>	43	42
	%	51	49
HC group (n = 42)	<i>n</i>	20	22
	%	48	52
LC group (n = 43)	<i>n</i>	23	20
	%	54	46

Additionally, a t-test for independent samples was conducted to test whether mean total scores differed between the intervention groups. There were no significant differences according to the self-reported success in dieting between the HC group and the LC group ($t(83) = 0.54, p = .594$).

Internal consistency of the German version of the scale is $\alpha > .70$ [MEULE *et al.*, 2012] and was $\alpha = .73$ in the current study, indicating suitable reliability.

Assessment of trait food craving

The extent and frequency of food craving experiences, as an intense desire to eat specific foods and especially highly palatable foods such as chocolate, were measured using the FCQ-T-r scale. Scores were divided via median split, indicating a similar distribution (table 6).

Table 6: Classification of trait food craving

		Trait food craving	
		low	high
Total	<i>n</i>	44	41
	%	52	48
HC group (n = 42)	<i>n</i>	21	21
	%	50	50
LC group (n = 43)	<i>n</i>	23	20
	%	52	48

A t-test for independent samples was conducted to test whether mean total scores in self-reported trait food craving differed between the HC group and the LC group. There

were no significant differences between the formed intervention groups ($t(83) = 0.38, p = .751$).

Internal consistency of the German version of the scale is $\alpha > .94$ [MEULE *et al.*, 2014] and was $\alpha = .89$ in the current study, indicating suitable reliability.

Assessment of orthorexia nervosa

The Bratman's Orthorexia Test was used to assess, whether participants with a pathologic obsession for naturally pure foods participated the study. If participants answered at least four of the ten items with "yes", they were at risk of developing *orthorexia nervosa*, while those who agreed with all items already suffered from the eating disorder [BRYTEK-MATERA, 2012].

Table 7: Classification of *orthorexia nervosa*

		Orthorexia nervosa	
		low	high
Total	<i>n</i>	37	48
	%	43	57
HC group (<i>n</i> = 42)	<i>n</i>	19	23
	%	45	55
LC group (<i>n</i> = 43)	<i>n</i>	18	25
	%	42	58

According to the classification 57 % of participants (*table 7*) were at risk of developing orthorectic eating behaviour, but none of them already suffered from that kind of eating disorder.

Additionally, a t-test for independent samples was conducted to test whether mean total scores differed between the HC group and the LC group. There were no significant differences between both intervention groups ($t(83) = -0.31, p = .757$).

Internal consistency of the scale is $\alpha = .82$ in previous investigations [VARGA *et al.*, 2014] and was $\alpha = .57$ in the current study, indicating relatively low reliability.

4.4 Phase I measurements

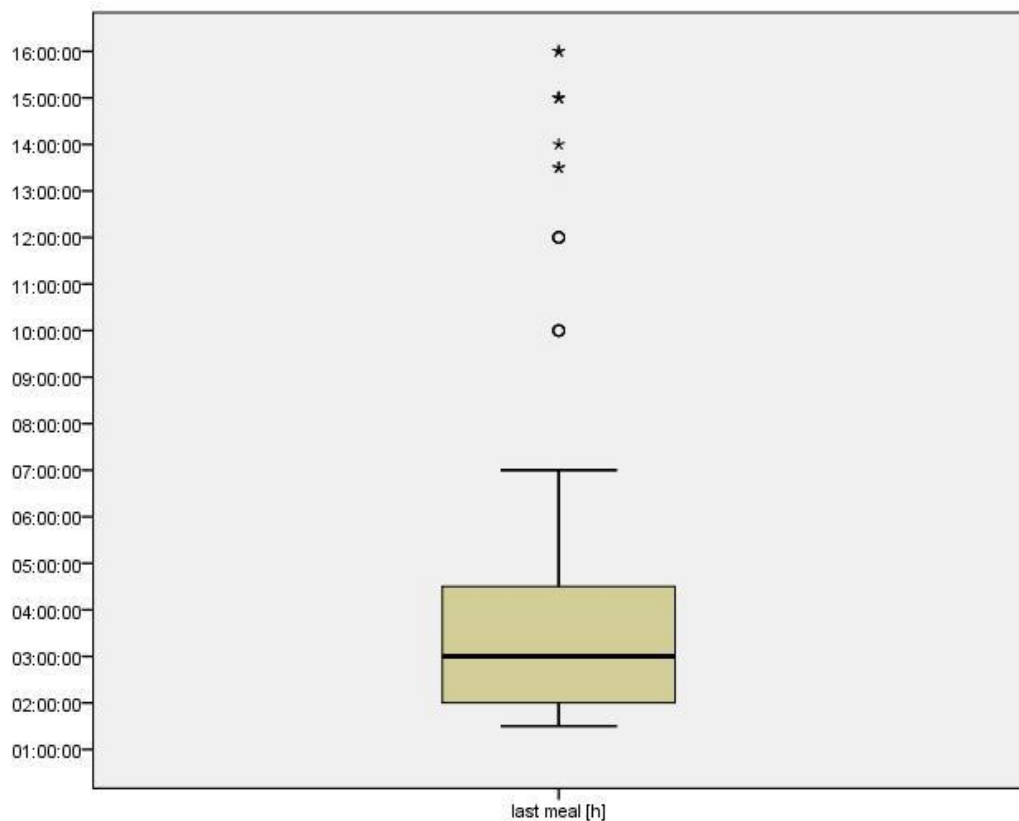
4.4.1 Appetite-related parameters

Food deprivation

Prior to the questionnaires, participants were asked to state the point in time of their last meal. Participants last food consumption was on average 04:38 hours ($SD = 04:01$) before the task, ranging from minimum 01:30 hours to maximum 16:00 hours. The median point in time of last meal was 03:00 hours and 50 % of the participants did not eat between 02:00 hours and 04:30 hours before the go/no-go task.

Furthermore, the mean point in time of last food consumption did not differ between the intervention groups ($t(83) = 0.72, p = .476$) as ascertained using a t-test for independent samples.

Figure 14: Distribution of last food consumption among participants



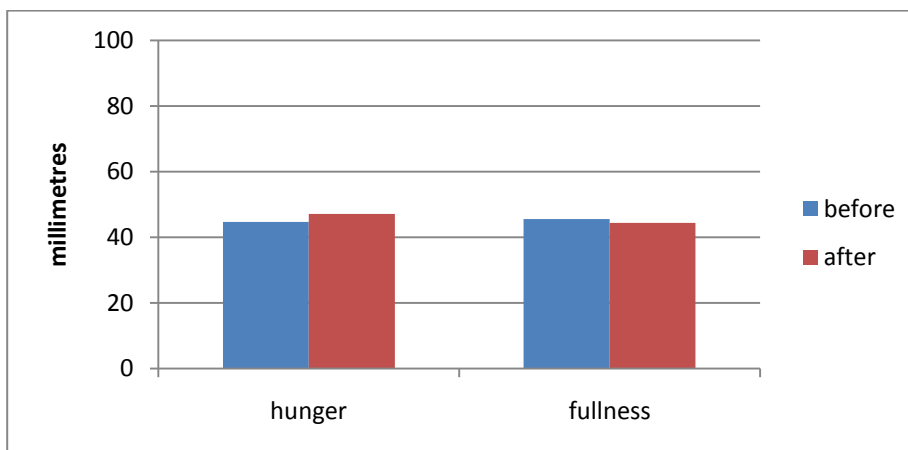
VAS scores for hunger and fullness

Participants mean sensation of hunger at the beginning of the task was 44.65 mm ($SD = 27.99$) and was 47.11 mm ($SD = 30.19$) at the end of the task. Their state of fullness was in average 45.54 mm ($SD = 30.22$) at the beginning and 44.39 mm ($SD = 31.16$) at the end of the go/no-go task.

To test whether mean hunger and fullness sensations changed along the task a paired samples t-test was performed. In addition, a t-test for independent samples was conducted to test whether mean hunger parameters differentiated between the intervention groups.

Neither the mean scores for hunger ($t(84) = -1.06, p = .293$) nor the mean scores for fullness ($t(84) = 0.58, p = .564$) did change significantly during the go/no-go task as illustrated in *figure 15*. Furthermore, there were no significant differences in hunger scores ($t(83) = -0.11, p = .910$) or fullness scores ($t(83) = 0.30, p = .766$) between the HC group and the LC group.

Figure 15: VAS scores at the beginning and at the end of the go/no-go task



Correlation of food deprivation with sensation of hunger and fullness

The correlation of the point in time of the last consumption with the states of hunger and fullness before the go/no-go task were conducted using a correlation analysis (Pearson's correlation coefficient) and are depicted in *figure 16* and *figure 17*.

The mean score of hunger before the go/no-go task was weakly positively correlated with the mean point in time of the last consumed meal ($r(83) = .216, p < .05$). In other words, the longer ago the point in time of the participants last meal was, the higher was the stated sensation of hunger.

The mean score of fullness before the go/no-go task was weakly negatively correlated with the mean point in time of the last consumed meal ($r(83) = -.314, p < .05$). Respectively, the longer ago the participants last meal was, the lower was the stated fullness score.

Figure 16: Correlation of point in time of last meal with hunger scores

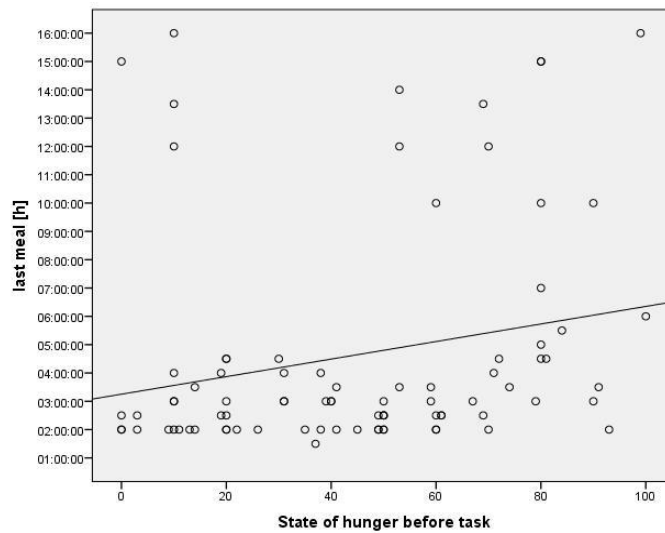
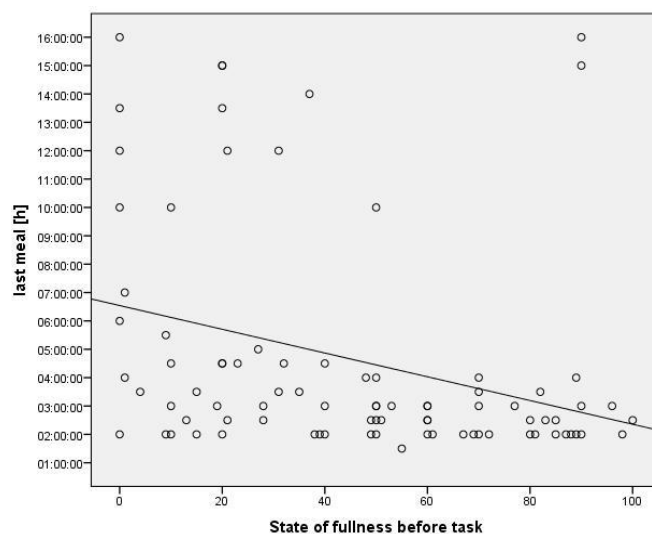


Figure 17: Correlation of point in time of last meal with fullness scores



Correlation of food deprivation with BMI and questionnaire scores

As stated in *table 8*, the point in time of the last meal consumption neither correlated with BMI values, nor with one of the questionnaire scores. Furthermore the actual subjective mood did not correlate with one of the conducted measurements.

Table 8: Correlation between point in time of last meal, BMI and questionnaire measures

<i>n</i> = 85	1.	2.	3.	4.	5.	6.	7.	8.
1. Food deprivation (hours)	-	-.134	.038	-.027	-.018	-.049	.095	-.050
2. BMI (kg/m ²)	-.134	-	-.077	.418**	.197	-.523**	-.024	.259*
3. ASTS	.038	-.077	-	.149	-.070	-.049	-.020	.053
4. FRK	-.027	.418**	.149	-	.035	-.307**	.194	.484**
5. BIS-15	-.018	.197	-.070	.035	-	-.154	.233*	-.029
6. PSRS	-.049	-.523**	-.049	-.307**	-.154	-	-.287**	-.298**
7. FCQ-t	.095	-.024	-.020	.194	.233*	-.287**	-	.321**
8. BOT	-.050	.259*	.053	.484**	-.029	-.298**	.321**	-

**p* < .05

***p* < .01

On the contrary, BMI values were positively correlated with rigid control of eating behaviour ($r(83) = .418, p < .01$). Or to put it differently, the higher the BMI values were, the higher was the extent of rigid restrained eating behaviour in a sense of altered periods of dieting and freely consumption of foods. The scores of the orthorexia test were also positively correlated with BMI ($r(83) = .259, p < .05$), respectively, the pathologic obsession for naturally pure foods increased with higher BMI values. Additionally the BMI was negatively correlated with the scores of the perceived self-regulatory success in dieting ($r(83) = -.523, p < .01$). In other words, the higher the participants BMI values were, the less successful they were in dieting.

The PSRS was additionally negatively correlated with the extent of trait food craving ($r(83) = -.287, p < .01$), which means, the more often food craving experiences occurred, the lower the perceived self-regulatory success in dieting was. Furthermore there was a negative correlation between the perceived self-regulatory success in dieting and the rigid control of eating behaviour scores ($r(83) = -.307, p < .01$) as well as with the orthorexia test ($r(83) = -.298, p < .01$).

Total scores of the BIS-15 test were positively correlated with the extent of trait food craving ($r(83) = .233, p < .05$). In other words, the extent of impulsivity was related to the frequency of food craving experiences.

Furthermore the scores of the orthorexia test were positively correlated with rigid control of eating behaviour ($r(83) = .484, p < .01$) and with the extent of trait food craving ($r(83) = .321, p < .01$).

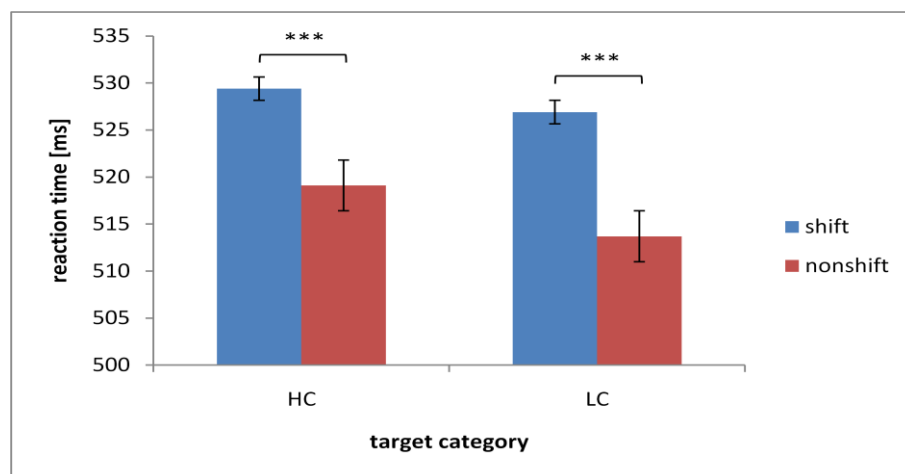
4.4.2 Task-related parameters

Reaction time

As stated in figure 18, there was a significant main effect for block type ($F(1,84) = 21.91, p < .001, np^2 = .207$), which means that participants reacted slower in response to pictures in shift blocks than in non-shift blocks ($M = 528$ ms, $SEM = 4$ vs. $M = 516$ ms, $SEM = 4$).

The main effect for target type ($F(1,84) = 1.96; p = .166; np^2 = .023$) as well as the interaction between target type * block type ($F(1,84) = 0.34; p = .563; np^2 = .004$) was not significant. In other words, reaction times in response to high caloric or low caloric food cues did not differ significantly ($M = 524$ ms, $SEM = 4$ vs. $M = 520$ ms, $SEM = 4$) and reaction times in response to high caloric or low caloric targets did not depend on block type.

Figure 18: Mean reaction times in the go/no-go task



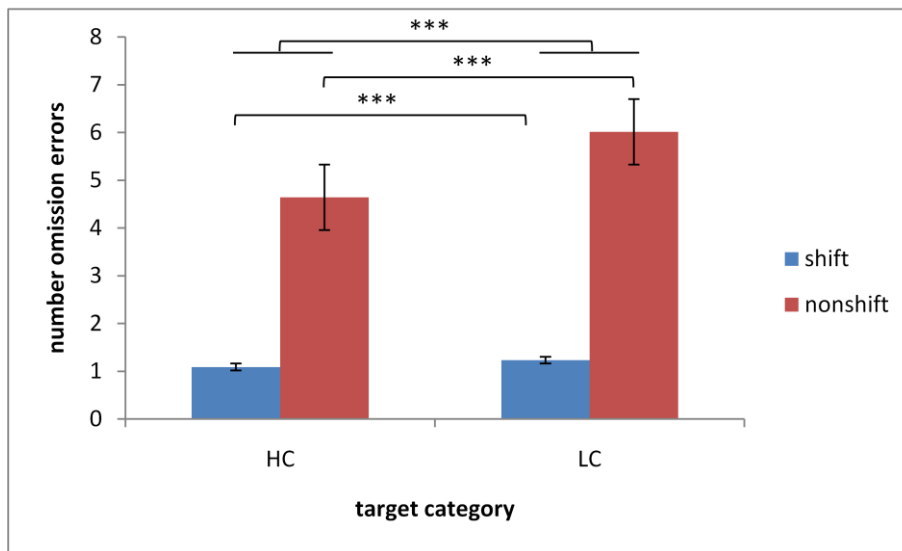
Mean reaction times as a function of target type and block type. Error bars indicating the standard error of the mean. (***) $p < .001$

Omission errors

There were significant main effects for target type ($F(1,84) = 12.41$; $p < .001$; $np^2 = .129$) and block type ($F(1,84) = 129.89$; $p < .001$; $np^2 = .607$) as well as for the interaction of target type * block type ($F(1,84) = 8.24$; $p < .005$; $np^2 = .089$).

As depicted in *figure 19*, in total participants omitted significantly more errors in response to low caloric food targets ($M = 3.62$, $SEM = 0.26$) than to high caloric food targets ($M = 2.87$, $SEM = 0.21$). Furthermore they omitted significantly more errors in non-shift blocks ($M = 5.33$, $SEM = 0.34$) than in shift blocks ($M = 1.16$, $SEM = 0.19$). The significant interactive effect indicates that participants omitted more errors particularly in non-shift blocks in response to low caloric food cues.

Figure 19: Mean number of omission errors in the go/no-go task



Mean number of omission errors as a function of target type and block type. Error bars indicating the standard error of the mean. (***) $p < .001$

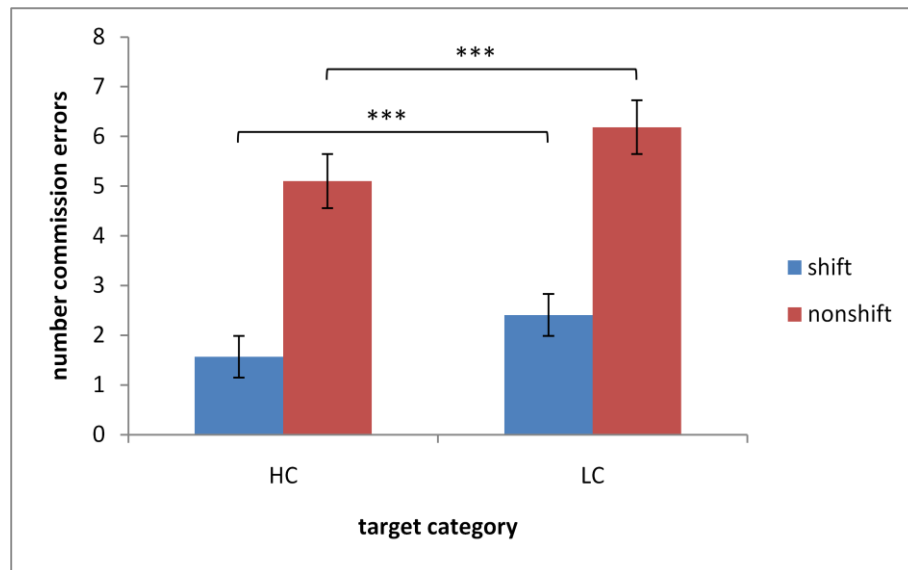
Commission errors

There were significant main effects for target type ($F(1,84) = 23.60$; $p < .001$; $np^2 = .219$) and block type ($F(1,84) = 96.76$; $p < .001$; $np^2 = .535$).

The interaction target type * block type was not significant ($F(1,84) = 0.36$; $p = .553$; $np^2 = .004$).

In other words, participants committed significantly more errors in response to low caloric food targets ($M = 4.30$, $SEM = 0.24$) than to high caloric food targets ($M = 3.33$, $SEM = 0.20$). Furthermore there were significantly more commission errors in non-shift blocks ($M = 5.64$, $SEM = 0.33$) than in shift blocks ($M = 1.99$, $SEM = 0.20$) as stated in figure 20.

Figure 20: Mean number of commission errors in the go/no-go task



Mean number of commission errors as a function of target type and block type. Error bars indicating the standard error of the mean. (***) $p < .001$

Correlation of food deprivation with BMI, questionnaire scores and number of commission errors

As stated in table 9, the point in time of the last meal consumption neither correlated with BMI values or questionnaire scores, nor with the number of committed errors in response to high caloric or low caloric food targets or during shift and non-shift blocks. Furthermore the participants' actual subjective mood, their trait impulsivity (BIS-15) and trait food craving (FCQ-T-r) did not correlate with one of the conducted measurements.

Table 9: Correlation of food deprivation with BMI, questionnaire scores and number of commission errors

	LC food blocks			HC food blocks			Non-Shift	Shift
	Non-Shift	Shift	total	Non-Shift	Shift	total		
Food deprivation (h)	-.029	.189	.090	.064	-.125	-.028	.015	.053
BMI (kg/m ²)	-.044	.097	.024	-.159	.016	-.114	-.101	.079
ASTS	.037	.078	.074	.029	.069	.066	.035	.101
FRK	-.160	.015	-.113	-.209	-.142	-.253*	-.189	-.082
BIS-15	-.090	-.027	-.085	-.058	.050	-.014	-.078	.013
PSRS	.060	-.254*	-.104	.084	.018	.077	.073	-.168
FCQ-t	.019	-.077	-.031	.043	-.114	-.038	.031	-.129
BOT	-.237*	-.114	-.248*	-.247*	-.069	-.237*	-.250*	-.126

* $p < 0.05$

On the contrary, rigid control of eating behaviour (scores of the FRK) was weakly negatively correlated ($r(83) = -.253, p < .05$) with the number of commission errors in response to high caloric food targets in total, indicating that participants committed the more errors in response to high caloric food targets and independent of block type, the less rigidly they controlled their eating.

Participants' success in dieting was negatively correlated with the number of commission errors in response to low caloric food cues during shift blocks ($r(83) = -.254, p < 0.05$). To put it differently, the less successful participants were in dieting, the higher was the number of commission errors in response to low caloric food cues during shift blocks.

Orthorectic eating behaviour was negatively correlated with the number of commission errors in non-shift blocks ($r(83) = -.250, p < .05$), independent of target type. In other words, the higher the scores of the Bratman's orthorexia test were, the more errors were committed in response to low caloric food cues in total ($r(83) = -.248, p < .05$) and especially in non-shift blocks ($r(83) = -.237, p < .05$). Similarly, the higher the scores were, the more errors were committed in response to high caloric food targets in total ($r(83) = -.237, p < .05$) and especially in non-shift blocks ($r(83) = -.247, p < .05$).

Prediction of commission errors

To go further into detail, linear regression analyses were conducted, in order to determine whether appetite-related parameters or questionnaire scores could act as predictors for the number of committed errors in response to low caloric and high caloric food targets as well as during shift blocks and non-shift blocks.

In *table 10* the standardized coefficients (β -values) and significance levels of the conducted coefficients within the linear regression analyses were outlined for giving a better overview of their capacity for predicting commission errors in the go/no-go task, as they are regarded to low inhibitory control.

Table 10: Prediction capacity of measures for the number of commission errors

	LC food blocks				HC food blocks			
	Non-shift		Shift		Non-shift		Shift	
	β	p-value	β	p-value	β	p-value	β	p-value
Food deprivation (h)	-.055	.619	.172	.117	.022	.842	-.116	.312
VAS hunger difference	.159	.150	-.001	.995	.149	.175	.004	.971
BMI (kg/m ²)	.131	.375	-.009	.948	-.012	.937	.053	.726
ASTS	.076	.500	.055	.620	.056	.620	.106	.361
FRK	-.114	.400	.025	.851	-.124	.361	-.185	.191
BIS-15	-.146	.212	-.047	.678	-.086	.463	.070	.559
PSRS	.037	.792	-.337	.015*	.000	1.000	-.021	.885
FCQ-T-r	.166	.199	-.125	.323	.146	.257	-.093	.483
BOT	-.264	.048*	-.180	.168	-.230	.084	.021	.877

	Non-shift total		Shift total		LC total		HC total	
	β	p-value	β	p-value	β	p-value	β	p-value
Food deprivation (h)	-.020	.860	.047	.679	.060	.587	-.056	.618
VAS hunger difference	.160	.147	.002	.985	.120	.268	.120	.279
BMI (kg/m ²)	.066	.653	.028	.851	.094	.518	.024	.869
ASTS	.068	.541	.108	.344	.090	.419	.110	.330
FRK	-.123	.365	-.102	.458	-.072	.590	-.213	.121
BIS-15	-.121	.297	.012	.918	-.139	.229	-.023	.845
PSRS	.020	.885	-.252	.075	-.171	.214	-.013	.925
FCQ-T-r	.162	.209	-.149	.253	.052	.680	.056	.665
BOT	-.256	.054	-.114	.396	-.307	.021*	-.167	.210

*p < .05

Appetite-related parameters, such as food deprivation, BMI or the state of hunger (illustrated as a difference score of state of hunger after the task minus state of hunger before the task) and the actual subjective mood state could not predict the number of committed errors. Additionally, neither the participants' trait impulsivity, respectively the scores of the Barratt Impulsiveness Scale, nor participants' trait food craving could predict the number of commission errors (all $p > .05$).

The extent of orthorectic eating behaviour predicted the number of committed errors in response to low caloric food targets during non-shift blocks ($p = .048$) as well in response to low caloric food cues in total ($p = .021$).

Furthermore, the scores of the PSRS test, indicating participants' success in dieting, could predict the number of commission errors in response to low caloric food targets during shift blocks ($p = .015$). For a better overview, the significant measures of the linear regression analysis are depicted in *table 11*.

Table 11: Significant regression coefficients

	LC Non-Shift	LC Shift	LC Total
(Intercept)	3.370	7.179	10.548
BOT	-.478	n.s	-.730
PSRS	n.s.	-.245	n.s.

Using the linear regression equation $Y = \beta_0 + \beta_1 * x_1 + \varepsilon$ the number of committed errors could be calculated. Therefore negative β -values indicated a decrease in the number of commission errors, if the predictor variables increased by one unit [FIELD, 2005].

According to the prediction capacity of the BOT to the number of commission errors in response to LC food cues during non-shift blocks and in response to LC food cues in total, the following calculation can be done:

- $CE_{(LC/Non-Shift)} = 3.370 - 0.478$
 $CE_{(LC/Non-Shift)} = 2.892$
- $CE_{(LC/Total)} = 10.548 - 0.730$
 $CE_{(LC/Total)} = 9.818$

To put it in words, if the score of the orthorexia test increased by one point, the number of commission errors in response to low caloric food targets during non-shift blocks decreased from 3.37 to 2.89 errors. In response to low caloric food cues in total, the number of commission errors decreased from 10.55 to 9.82 errors.

Similarly, the prediction capacity of the PSRS to the number of commission errors in response to LC food cues during shift blocks can be calculated as follows:

- $CE_{(LC/Shift)} = 7.179 - 0.245$

$$CE_{(LC/Total)} = 6.934$$

Thus, if the scores of the PSRS test increased by one point, the number of commission errors in response to low caloric food targets during shift blocks decreased from 7.18 to 6.93 errors.

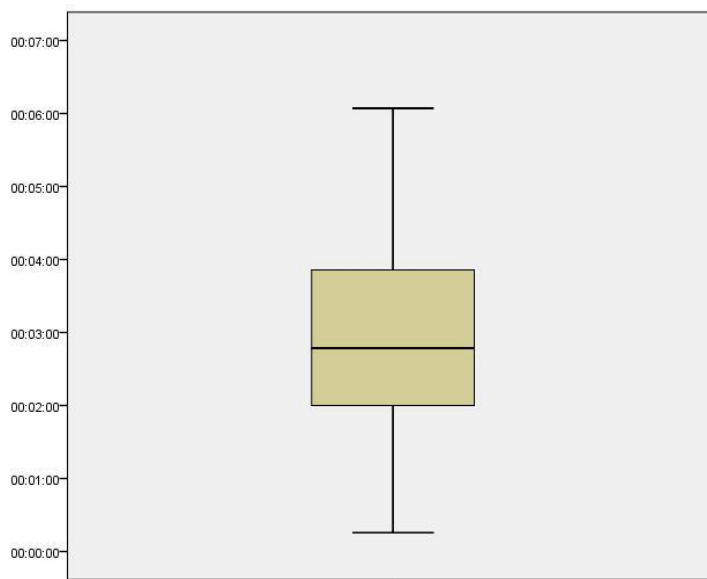
4.5 Phase II measurements

According to group affiliation participants received modified pictures of high caloric or low caloric foods. The number of detected mistakes as well as the time participants needed to find the mistakes were measured.

Reaction time

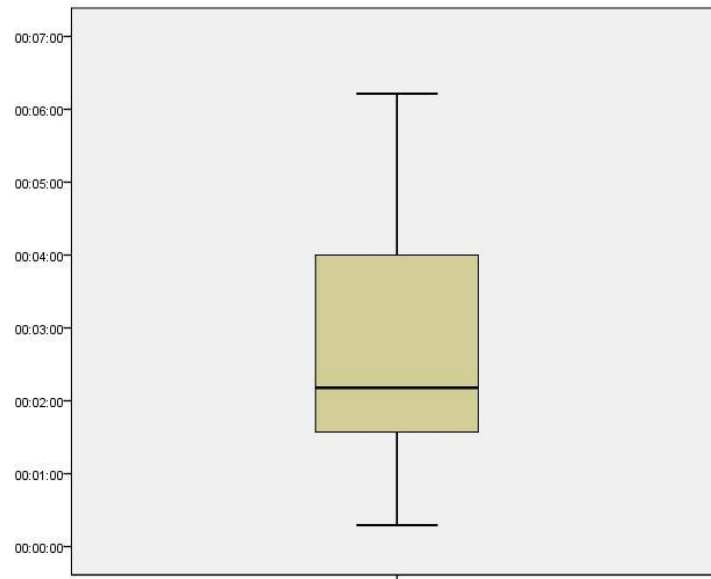
Participants of the HC group spent a mean time of 02:48 minutes ($SD = 01:20$) on inspecting the sent pictures, ranging from a minimum of 00:15 minutes to a maximum timeframe of 06:04 minutes. The median time spent was 02:47 minutes and 50 % of the participants needed between 01:57 minutes and 03:51 minutes to complete the task as stated in *figure 21*.

Figure 21: Time to detect mistakes within the HC group



As depicted in *figure 22*, students belonging to the LC group needed on average 02:41 minutes ($SD = 01:34$) to finish the task with a minimum of 00:17 minutes and a maximum of 06:12 minutes. The median of spent time was 02:10 and 50 % of the participants needed between 00:40 minutes and 04:00 minutes to find the inserted changes.

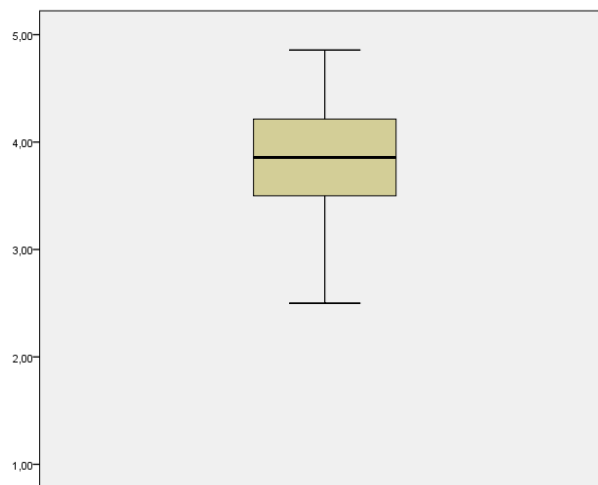
Figure 22: Time to detect mistakes within the LC group



Inserted mistakes

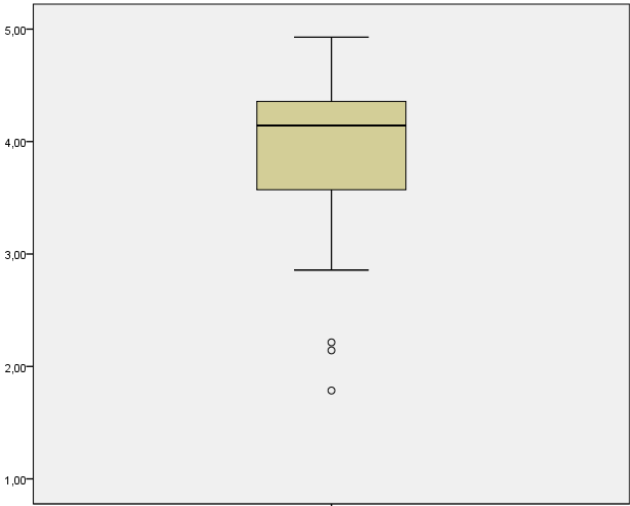
Within the HC group participants detected 3.84 mistakes ($SD = 0.58$) per picture on average, ranging from at least 2.50 changes to a maximum of 4.86 changes. The median of detected mistakes was 3.86. 50 % of the participants belonging to the HC group detected between 3.48 mistakes and 4.21 mistakes from a maximum of five mistakes in each picture (*figure 23*).

Figure 23: Detected mistakes within the HC group



On the contrary, participants of the LC group detected 3.90 inserted mistakes ($SD = 0.71$) on average with a minimum of 1.79 and a maximum of 4.93 changes. The median was 4.14 detected mistakes, whereas 50 % of the participants found between 3.43 changes and 4.46 changes in each sent picture as illustrated in *figure 24*.

Figure 24: Detected mistakes within the LC group



4.6 Phase III measurements

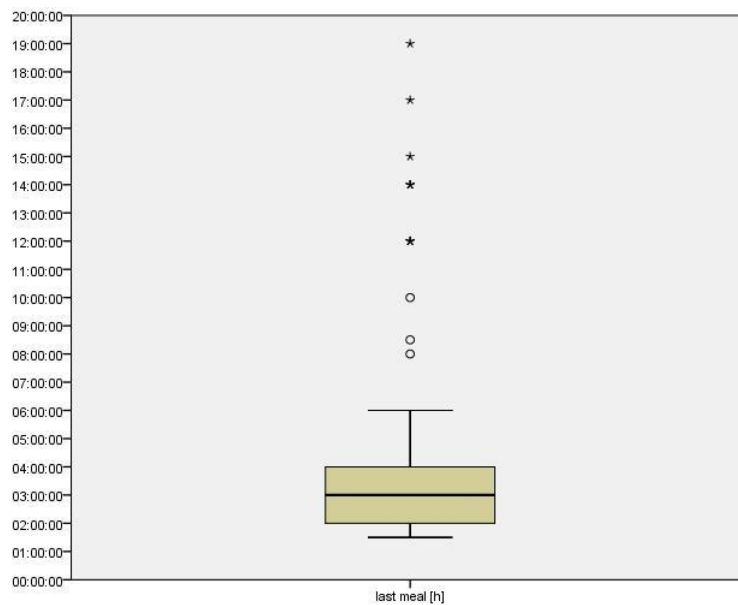
4.6.1 Appetite-related parameters

Food deprivation

Participants were again asked to state the point in time of their last meal consumption before completing the task.

As depicted in *figure 25* participants did not eat in average 04:19 hours ($SD = 03:50$) before the task, ranging from minimum 01:30 hours to maximum 19:00 hours. The median point in time of last meal was 03:00 hours and 50 % of the participants did not eat between 02:00 hours and 04:15 hours before the go/no-go task.

Figure 25: Distribution of last food consumption among participants



VAS scores for hunger and fullness

The participants mean states of hunger and fullness were again conducted using a VAS at the beginning and at the end of the go/no-go task.

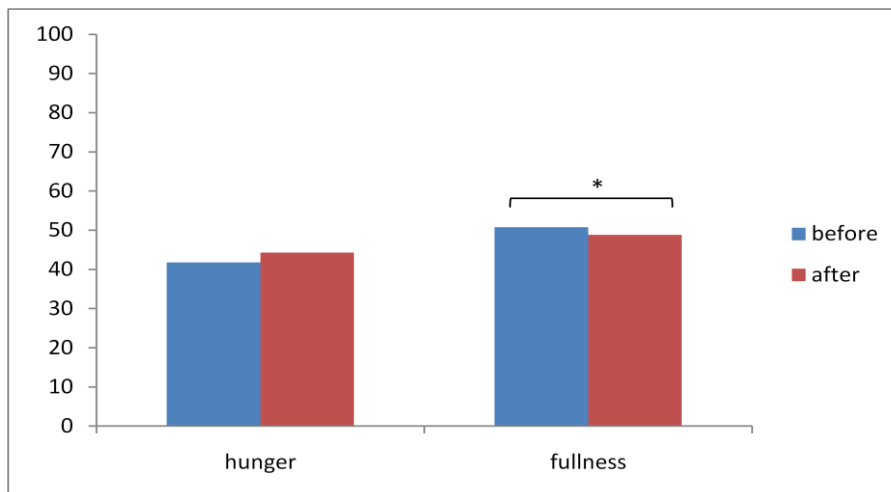
Participants sensation of hunger did not drop significantly from 41.79 mm ($SD = 28.81$) at the beginning of the task to 44.29 mm ($SD = 31.38$; $p = .059$) at the end of the task.

Their mean state of fullness was 50.76 mm ($SD = 30.65$) at the beginning and 48.81 mm ($SD = 31.40$) at the end of the go/no-go task.

Similar to phase I, a paired samples t-test was performed in order to test whether mean hunger and fullness sensations changed along the task. Additionally, a t-test for independent samples was conducted to test whether mean hunger parameters differentiated between the intervention groups.

The mean scores for fullness did change significantly ($t(84) = 2.24, p = .028$) during the task, whereas the mean scores for hunger ($t(84) = -1.91, p = .059$) did not, as illustrated in *figure 26*. Furthermore, there were no significant differences between the HC group and the LC group according to hunger ($t(83) = -0.58, p = .566$) or fullness scores ($t(83) = -0.29, p = .769$).

Figure 26: VAS scores at the beginning and at the end of the go/no-go task



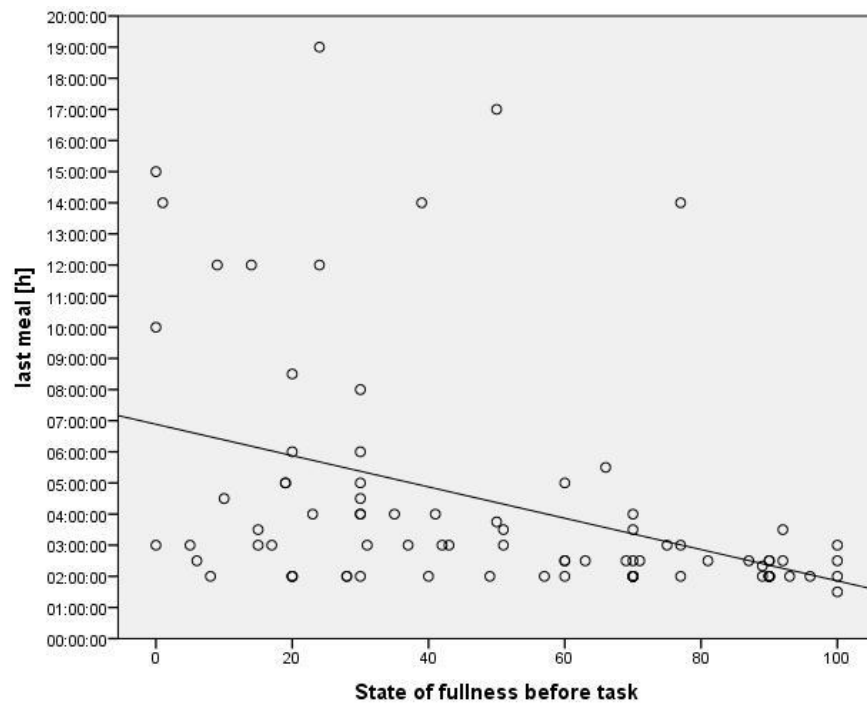
Correlation of food deprivation with sensation of hunger and fullness

As already used for analysis of the correlation of the point in time of last consumption with the states of hunger and fullness before the go/no-go task in phase I, a correlation analysis (Pearson's correlation coefficient) was conducted. Results are depicted in *figure 27*.

The mean score of hunger before the go/no-go task was not significantly correlated with the mean point in time of the last consumed meal ($r(83) = .145, p = .187$).

The mean score of fullness before the go/no-go task was negatively correlated with the mean point in time of the last consumed meal ($r(83) = -.401, p < .001$). Or to put it differently, the longer ago the last meal consumption was, the lower was the stated fullness score.

Figure 27: Correlation of point in time of last meal with fullness scores



Correlation of food deprivation with BMI and questionnaire scores

The correlation of food deprivation with BMI and questionnaire scores was similar to the measurements of phase I.

The point in time of last meal neither correlated with BMI values, nor with the questionnaire scores. Again the actual subjective mood did not correlate with one of the conducted measurements as stated in *table 12*.

Table 12: Correlation between point in time of last meal, BMI and questionnaire measures

<i>n</i> = 85	1.	2.	3.	4.	5.	6.	7.	8.
1. Food deprivation (hours)	-	.174	.117	.167	.185	-.154	-.042	-.050
2. BMI (kg/m²)	.174	-	-.089	.421**	.208	-.512**	-.035	.222*
3. ASTS	.117	-.089	-	.149	-.070	-.049	-.020	.053
4. FRK	.167	.421**	.149	-	.035	-.307**	.194	.484**
5. BIS-15	.185	.208	-.070	.035	-	-.154	.233*	-.029
6. PSRS	-.154	-.512**	-.049	-.307**	-.154	-	-.287**	-.298**
7. FCQ-t	-.042	-.035	-.020	.194	.233*	-.287**	-	.321**
8. BOT	-.050	.222*	.053	.484**	-.029	-.298**	.321**	-

**p* < .05

BMI values were once more positively correlated with rigid control of eating behaviour ($r(83) = .421, p < .01$). In other words, the higher the BMI values were the higher was the extent of rigid restrained eating behaviour. BMI was also positively correlated with the scores of the orthorexia test ($r(83) = .222, p < .05$). Additionally the BMI was negatively correlated with the scores of the PSRS ($r(83) = -.512, p < .01$). Therefore participants were less successful in dieting, the higher their BMI was.

The PSRS was additionally negatively correlated with the extent of trait food craving ($r(83) = -.287, p < .01$), with the rigid control of eating behaviour scores ($r(83) = -.307, p < .01$) as well as with the orthorexia test ($r(83) = -.298, p < .01$).

Total scores of the BIS-15 test were positively correlated with the extent of trait food craving ($r(83) = .233, p < .05$). Additionally the scores of the orthorexia test were positively correlated with rigid control of eating behaviour ($r(83) = .484, p < .01$) and with the extent of trait food craving ($r(83) = .321, p < .01$).

4.6.2 Task-related parameters

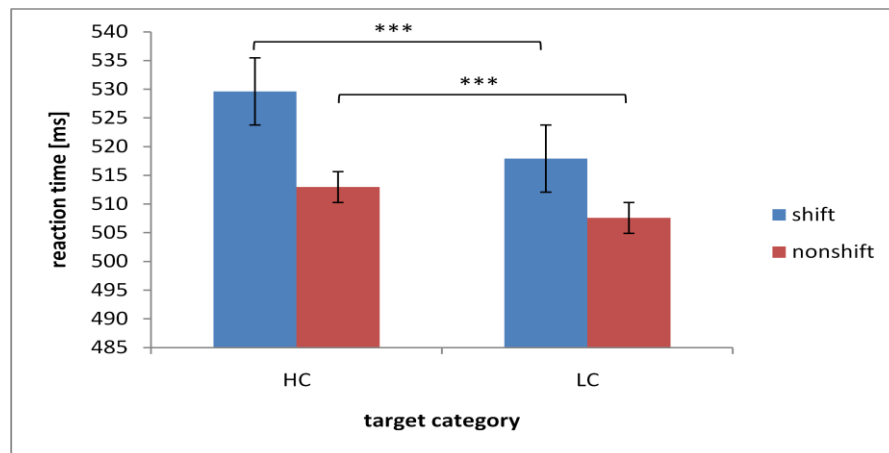
Reaction times

As depicted in figure 28, the main effect for target type was significant ($F(1,84) = 12.06$; $p < .001$; $\eta^2 = .126$), which means that participants reacted faster in response to low caloric targets than to high caloric targets ($M = 513$ ms, $SEM = 5$ vs. $M = 521$ ms, $SEM = 5$).

There was a significant main effect for block type ($F(1,84) = 39.78$; $p < .001$; $\eta^2 = .321$), or to put it differently, participants reacted faster in response to non-shift blocks than to shift blocks ($M = 510$ ms, $SEM = 5$ vs. $M = 524$ ms, $SEM = 5$).

The interaction between target type and block type was not significant ($F(1,84) = 1.94$; $p = .167$; $\eta^2 = .023$). In other words, reaction times in response to high caloric or low caloric targets were not dependent on block type, similar to the first go/no-go task in phase I.

Figure 28: Mean reaction times in the go/no-go task



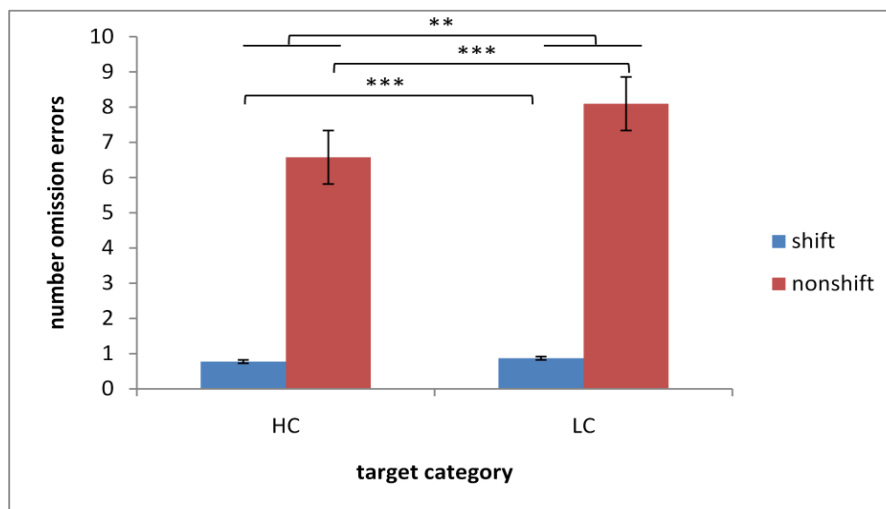
Mean reaction times as a function of target type and block type. Error bars indicating the standard error of the mean. (***) $p < .001$

Omission errors

Similar to the go/no-go task in phase I, there were significant main effects for target type ($F(1,84) = 40.40$; $p < .001$; $\eta^2 = .325$), block type ($F(1,84) = 868.18$; $p < .001$; $\eta^2 = .912$) as well as for the interaction of target type * block type ($F(1,84) = 58.25$; $p < .001$; $\eta^2 = .409$).

As depicted in *figure 29*, in total participants omitted significantly more errors in response to low caloric food targets ($M = 4.48$, $SEM = 0.15$) than to high caloric food targets ($M = 3.67$, $SEM = 0.13$). Furthermore they omitted significantly more errors in non-shift blocks ($M = 7.33$, $SEM = 0.19$) than in shift blocks ($M = 0.82$, $SEM = 0.14$). The significant interaction of target type * block type indicates that participants omitted more errors particularly in non-shift blocks in response to low caloric food cues.

Figure 29: Mean number of omission errors in the go/no-go task



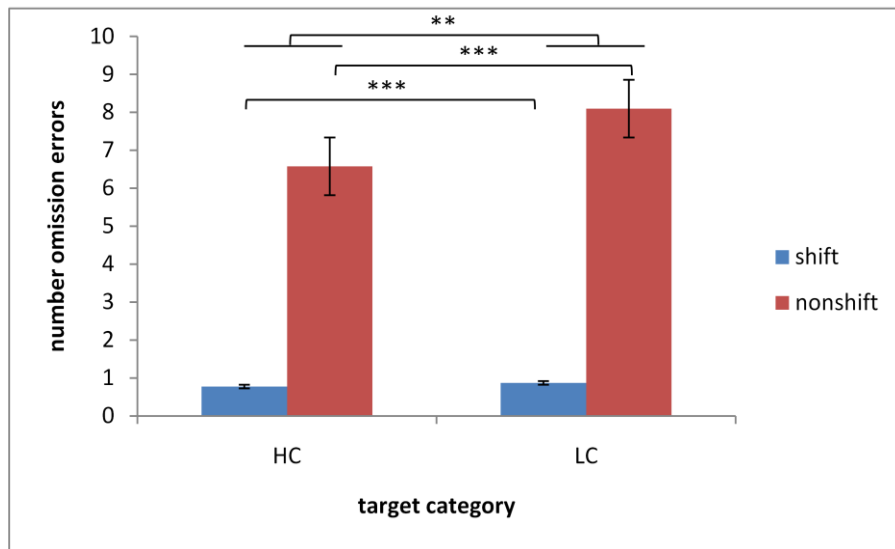
Mean number of omission errors as a function of target type and block type. Error bars indicating the standard error of the mean. (** $p < .01$, *** $p < .001$)

Commission errors

The main effects for target type ($F(1,84) = 56.86$; $p < .001$; $\eta^2 = .404$) and block type ($F(1,84) = 661.88$; $p < .001$; $\eta^2 = .887$) as well as the interaction of target type * block type ($F(1,84) = 21.87$; $p < .001$; $\eta^2 = .207$) were significant.

The results indicate, that participants committed significantly more errors in response to low caloric food targets ($M = 5.17$, $SEM = 0.17$) than to high caloric food targets ($M = 4.08$, $SEM = 0.16$). Similar to the go/no-go task in phase I, they committed significantly more errors in non-shift blocks ($M = 7.71$, $SEM = 0.22$) than in shift blocks ($M = 4.55$, $SEM = 0.16$). Additionally the significant interaction of target type * block type indicates that participants committed more errors in non-shift blocks in response to low caloric food cues as stated in *figure 30*.

Figure 30: Mean number of commission errors in the go/no-go task



Mean number of commission errors as a function of target type and block type. Error bars indicating the standard error of the mean. (** $p < .01$, *** $p < .001$)

Correlation of food deprivation with BMI, questionnaire scores and number of commission errors

As stated in *table 13*, neither the point in time of the last food consumption nor BMI values did correlate with the number of committed errors in response to high caloric or low caloric food targets or during shift and non-shift blocks.

Additionally the participants' actual subjective mood, their trait impulsivity (BIS-15) and the scores of the Bratman's orthorexia test did not correlate with one of the conducted task measurements.

Table 13: Correlation of food deprivation with BMI, questionnaire scores and number of commission errors

	LC food blocks			HC food blocks			Non-Shift	Shift
	Non-Shift	Shift	total	Non-Shift	Shift	total		
Food deprivation (h)	.013	.070	.053	-.002	-.003	-.003	.006	.046
BMI (kg/m²)	-.032	.152	.074	.001	-.001	.000	-.017	.103
ASTS	.005	.062	.043	-.064	-.168	-.137	-.029	-.051
FRK	-.068	.042	-.021	-.149	-.221*	-.224*	-.111	-.094
BIS-15	-.049	-.111	-.104	-.117	-.075	-.121	-.085	-.117
PSRS	-.009	-.237*	-.156	-.050	.063	.001	-.030	-.126
FCQ-t	-.272*	-.182	-.306**	-.172	-.105	-.176	-.234*	-.182
BOT	-.060	.049	-.011	-.020	.066	.023	-.043	.070

* $p < .05$

** $p < .01$

Participants' rigid control of eating behaviour (scores of the FRK) was weakly negatively correlated ($r(83) = -.224, p < .05$) with the number of commission errors in response to high caloric food targets, especially in shift blocks ($r(83) = -.221, p < .05$). In other words, the more errors in response to high caloric food targets participants committed, the less rigid they controlled their eating.

Similar to phase I, participants' success in dieting was negatively correlated with the number of commission errors in response to low caloric food cues during shift blocks ($r(83) = -.237, p < .05$), indicating that participants who were less successful in dieting, committed a higher number of errors in response to low caloric food cues during shift blocks.

While trait food craving was not correlated with the number of commission errors in phase I, it is negatively correlated to committed errors in non-shift blocks

($r(83) = -.234, p < .05$) in phase III and in response to low caloric food targets ($r(83) = -.306, p < .01$). Or to put it differently, the more often and stronger participants' food craving experiences were, the less errors they committed in non-shift blocks and especially in non shift blocks in response to low caloric food targets ($r(83) = -.272, p < .05$).

Prediction of commission errors

For detailed information regarding to the predicting capacity of the measures, similar to phase I, a linear regression analyses was conducted.

The standardized coefficients and their significance levels within the linear regression analyses were outlined in *table 14*.

Once more appetite-related parameters, such as food deprivation, BMI or the state of hunger could not predict the number of committed errors in phase III. Neither did the participants' actual subjective mood state or the extent of their trait impulsivity predict the amount of committed errors (all $p > .05$).

Table 14: Prediction capacity of measures for the number of commission errors

	LC food blocks				HC food blocks			
	Non-shift		Shift		Non-shift		Shift	
	β	<i>p</i> -value	β	<i>p</i> -value	β	<i>p</i> -value	β	<i>p</i> -value
Food deprivation (h)	-.019	.872	.014	.899	.020	.866	.075	.515
VAS hunger difference	.142	.244	.182	.125	.092	.454	.106	.375
BMI (kg/m ²)	-.126	.397	.033	.817	-.005	.975	.118	.416
ASTS	-.001	.991	.061	.586	-.050	.667	-.119	.294
FRK	.020	.890	-.006	.966	-.172	.232	-.331	.019*
BIS-15	.014	.909	-.131	.265	-.108	.376	-.089	.451
PSRS	-.147	.297	-.282	.041*	-.141	.322	.076	.579
FCQ-T-r	-.370	.006**	-.285	.027*	-.204	.127	-.103	.424
BOT	.057	.671	.076	.559	.104	.445	.282	.035*

	Non-shift total		Shift total		LC total		HC total	
	β	<i>p</i> -value	β	<i>p</i> -value	β	<i>p</i> -value	β	<i>p</i> -value
Food deprivation (h)	.000	.997	.051	.655	-.004	.970	.055	.630
VAS hunger difference	.123	.315	.182	.128	.214	.064	.122	.307
BMI (kg/m ²)	-.071	.635	.088	.543	-.067	.632	.063	.665
ASTS	-.026	.826	-.025	.825	.038	.730	-.101	.373
FRK	-.074	.602	-.188	.177	.010	.940	-.302	.032*
BIS-15	-.046	.705	-.138	.243	-.073	.522	-.124	.297
PSRS	-.150	.290	-.148	.282	-.280	.037*	-.054	.696
FCQ-T-r	-.303	.024*	-.250	.053	-.439	.001**	-.196	.128
BOT	.083	.541	.208	.116	.088	.488	.228	.085

* $p < .05$

** $p < .01$

On the contrary, participants' extent of rigid control of eating predicted the number of commission errors in response to high caloric food targets during shift blocks ($p = .035$) as well as in response to high caloric food cues in total ($p = .032$).

The scores of the PSRS test could predict the number of commission errors in response to low caloric food targets during shift blocks ($p = .041$) as well as in response to low caloric food cues in total ($p = .037$).

Furthermore, participants' trait food craving predicted the number of commission errors in response to low caloric food targets during non-shift blocks ($p = .006$) and shift blocks ($p = .027$), as well in response to low caloric food cues in total ($p = .001$) and during non-shift blocks in total ($p = .024$).

Contrary to phase I, the magnitude of orthorectic eating behaviour predicted the number of committed errors in response to high caloric food targets during shift blocks ($p = .035$).

For a better overview, the significant measures of the linear regression analysis are depicted in *table 15*.

Table 15: Significant regression coefficients

	LC Non-shift	LC Shift	LC Total	HC Shift	HC Total	Non-Shift Total
(Intercept)	14.642	5.555	20.196	1.880	13.469	26.230
FRK				-.210	-.343	
PSRS		-.156	-.246			
FCQ-t	-.075	-.052	-.127			-.112
BOT				.246		

The number of committed errors could be calculated by using the linear regression equation $Y = \beta_0 + \beta_1 * x_1 + \varepsilon$. Negative standardized coefficients again indicated a decrease in the number of commission errors, if the predictor variables increased by one unit.

According to the prediction capacity of the FRK to the number of commission errors in response to HC food cues during shift blocks and in response to HC food cues in total, the following calculations can be done:

- $CE_{(HC/ Shift)} = 1.880 - 0.210$
 $CE_{(HC/ Shift)} = 1.670$
- $CE_{(HC/ Total)} = 13.469 - 0.343$
 $CE_{(HC/ Total)} = 13.126$

In other words, if the extent of rigid control of eating increased by one unit, the number of commission errors in response to high caloric food targets during shift blocks decreased from 1.88 to 1.67 errors. In response to high caloric food cues in total, the number of commission errors decreased from 13.47 to 13.13 errors.

The prediction capacity of the PSRS to the number of commission errors in response to LC food cues during shift blocks and in response to LC food cues in total, the following calculations can be done:

- $CE_{(LC/ Shift)} = 5.555 - 0.156$
 $CE_{(LC/ Shift)} = 5.399$
- $CE_{(LC/ Total)} = 20.196 - 0.246$
 $CE_{(LC/ Total)} = 19.950$

Or to put it differently, if the scores of the PSRS test increased by one point, the number of commission errors in response to low caloric food targets during shift blocks decreased from 5.56 to 5.40 errors. In response to low caloric food targets in total the number of commission errors decreased from 20.20 to 19.95 errors.

The FCQ-t questionnaire scores could predict the number of commission errors in response to LC food cues during non-shift blocks as well as during shift-blocks and additionally in response to LC food cues in total and during non-shift blocks in total. Therefore the following calculations can be done:

- $CE_{(LC/ Non-shift)} = 14.642 - 0.075$
 $CE_{(LC/ Non-shift)} = 14.567$
- $CE_{(LC/ Shift)} = 5.555 - 0.052$
 $CE_{(LC/ Shift)} = 5.503$
- $CE_{(LC/ Total)} = 20.196 - 0.127$
 $CE_{(LC/ Total)} = 20.069$

- $CE_{(Non-shift/Total)} = 26.230 - 0.112$
 $CE_{(Non-shift/Total)} = 26.118$

Therefore the number of commission errors in response to low caloric food targets during non-shift blocks decreased from 14.64 to 14.57 errors while it decreased from 5.56 to 5.50 errors during shift blocks regarding to LC food cues. In response to low caloric food cues in total the number of commission errors decreased from 20.20 to 20.07 errors and decreased from 26.23 to 26.12 errors during non-shift blocks in total, if the score of the food craving questionnaire increased by one point.

According to the prediction capacity of the BOT to the number of commission errors in response to HC food cues during shift blocks the following calculation can be done:

- $CE_{(HC/ Shift)} = 1.880 + 0.246$
 $CE_{(HC/ Shift)} = 2.126$

To put it in other words, if the score of the orthorexia test increased by one point, the number of commission errors in response to high caloric food targets during shift blocks also increased from 1.88 to 2.13 errors.

4.7 Impact of the priming intervention

In order to verify the established hypotheses, whether the priming intervention in total and the affiliation to the HC group in particular does affect the number of commission errors, another ANOVA [(target type: HC vs. LC) x (block type: shift vs. non-shift) x (testing phase: phase I vs. phase III)] for repeated measures was conducted.

Furthermore the intervention groups (HC vs. LC) were incorporated as a between-subjects-factor into analysis in a second step to determine, whether the group formation during the priming intervention did affect participants' inhibitory control in a sense of altered measure results.

In order to be able to draw a comparison of the mean values, the respective difference scores of the results of both go/no-go tasks (phase III minus phase I) were calculated in a first step. Therefore positive values indicate slower reaction times or a higher number of omission or commission errors, whereas negative values indicate a faster reaction or a smaller amount of omitted or committed errors during the second task (phase III).

The impact of the intervention was mainly verified by assaying a change in the number of commission errors, which are considered to reflect behavioural disinhibition.

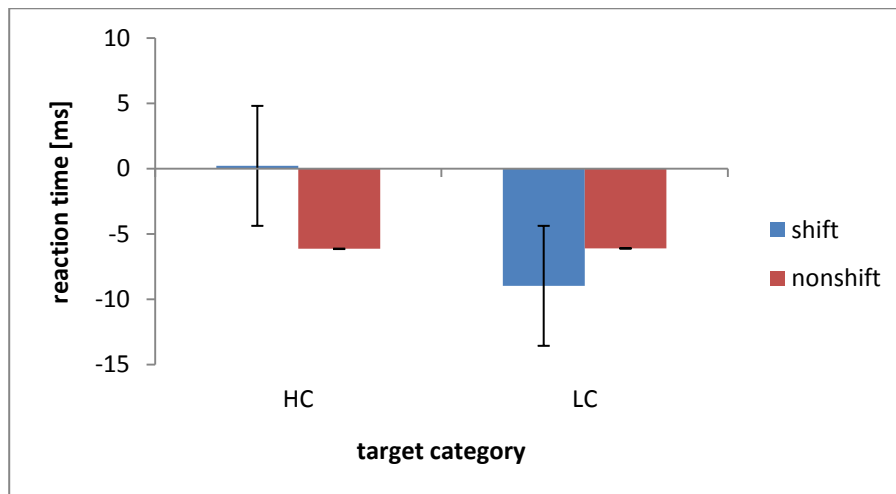
Reaction times

In the comparative measurement, the main effect for testing phase was not significant ($F(1,84) = 2.85$; $p = .095$; $np^2 = .033$). In other words the priming intervention did not affect participants' reaction times in total. Neither was the impact of the formed groups to the task performance, respectively to reaction times, significant ($F(1,84) = 0.81$; $p = .539$; $np^2 = .005$).

The comparative main effects for target type ($F(1,84) = 2.15$; $p = .146$; $np^2 = .025$) and block type ($F(1,84) = 0.28$; $p = .599$; $np^2 = .003$) as well as the interaction effect of target type * block type * testing phase ($F(1,84) = 1.76$; $p = .188$; $np^2 = .021$) were not significant. Taking the different intervention groups into account, neither the main effects for target type ($F(1,84) = 0.41$; $p = .525$; $np^2 = .005$) or block type ($F(1,84) = 3.70$; $p = .058$; $np^2 = .043$), nor the interaction effect for target type * block type * testing phase ($F(1,84) = 0.38$; $p = .541$; $np^2 = .005$) were significant.

Therefore reaction times in response to high caloric ($M = -3$ ms, $SEM = 4$) and low caloric ($M = -8$ ms, $SEM = 3$) food targets did not differ significantly, when comparing both go/no-go tasks. Additionally participants reaction times did not differ significantly in response to shift blocks ($M = -4$ ms, $SEM = 4$) or non-shift blocks ($M = -6$ ms, $SEM = 3$). The interaction effect of target type * block type * testing phase was not significant, indicating that reaction times in response to high caloric or low caloric food targets were not dependent on block type, when comparing both go/no-go tasks. The difference scores of both go/no-go tasks are depicted in *figure 31*.

Figure 31: Difference scores regarding reaction times in both go/no-go tasks

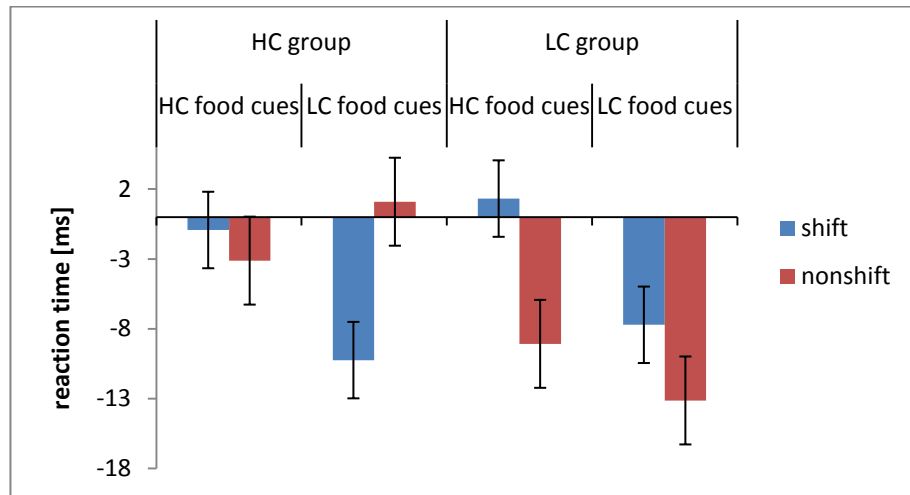


Mean difference scores of reaction times (phase III – phase I) as a function of target type and block type. Error bars indicating the standard error of the mean.

Regarding the intervention groups, neither within the HC group ($M = -2$ ms, $SEM = 6$) nor within the LC group ($M = -4$ ms, $SEM = 6$) were significant differences in reaction times in response to high caloric foods. Similarly to this were the results in response to low caloric foods in the HC group ($M = -5$ ms, $SEM = 4$) and the LC group ($M = -10$ ms, $SEM = 4$) not significant.

Reaction times of the HC group in shift blocks ($M = -6$ ms, $SEM = 5$) and non-shift blocks ($M = -1$ ms, $SEM = 5$) did not differ significantly from those of the LC group in shift blocks ($M = -3$ ms, $SEM = 5$) or non-shift blocks ($M = -11$ ms, $SEM = 5$). Group-related results are depicted in *figure 32*.

Figure 32: Group-related difference scores regarding reaction times



Mean difference scores of reaction times as a function of target type and block type regarding to both intervention groups. Error bars indicating the standard error of the mean.

Omission errors

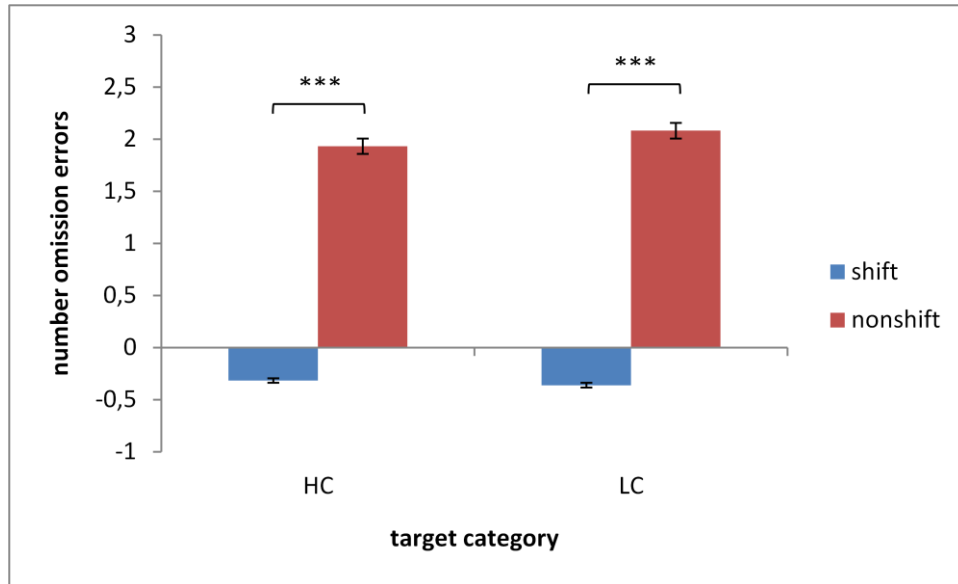
Concerning the number of omission errors in the comparative measurement, the main effect for testing phase was significant ($F(1,84) = 13.20$; $p < .000$; $np^2 = .136$), but not the main effect for group classification ($F(1,84) = 0.04$; $p = .851$; $np^2 = .000$). In other words, the priming intervention did affect participants' performance in the second go/no-go task in terms of missing responses to target issues, but the belonging to either the HC group or the LC group could not be associated with the number of omission errors in particular.

As stated in *figure 33* the main effect for block type ($F(1,84) = 39.46$; $p < .001$; $np^2 = .320$) was significant, indicating that participants omitted significantly more errors in non-shift blocks ($M = 2.01$, $SEM = 0.35$) than in shift blocks ($M = -0.34$, $SEM = 0.22$) when comparing both testing phases.

On the contrary, the main effect for target type ($F(1,84) = 0.05$; $p = .832$; $np^2 = .001$) as well as the interaction effect of target type * block type * testing phase ($F(1,84) = 0.17$; $p = .684$; $np^2 = .002$) were not significant. To put it differently, omitted errors in response to high caloric ($M = 0.81$, $SEM = 0.27$) and low caloric ($M = 0.86$, $SEM = 0.30$) food targets did not differ and the number of omission errors in response to

high caloric or low caloric food targets were not dependent on block type between the tasks.

Figure 33: Difference scores regarding the number of omission errors in both go/no-go tasks



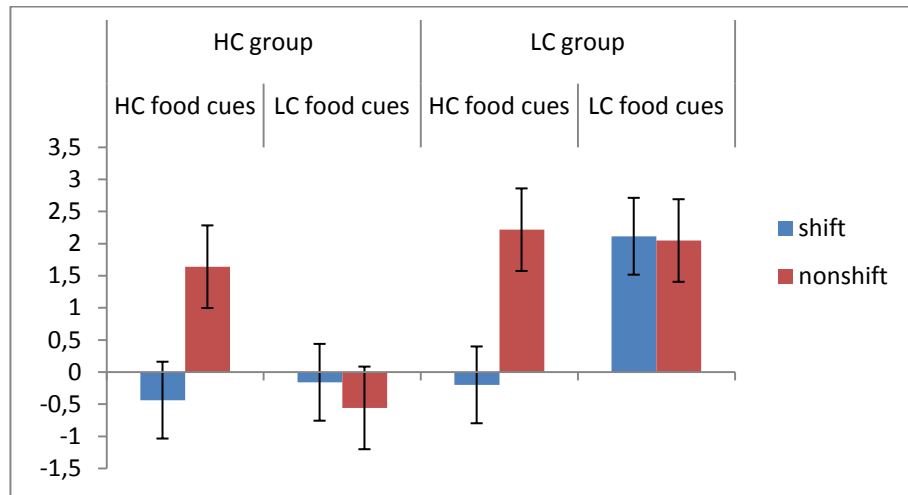
Mean difference scores of omission errors (phase III – phase I) as a function of target type and block type. Error bars indicating the standard error of the mean. (*) $p < .001$**

When taking the intervention groups into account again, the main effects for target type ($F(1,84) = 1.70$; $p = .196$; $np^2 = .20$) and block type ($F(1,84) = 0.20$; $p = .656$; $np^2 = .002$) were not significant and neither was the interaction effect for target type*block type*testing phase ($F(1,84) = 0.00$; $p = .995$; $np^2 = .000$), indicating that the group formation had no particular effect on the number of omitted errors.

As stated in *figure 34*, the difference scores of omitted errors in response to high caloric food targets within the HC group ($M = 0.60$, $SEM = 0.32$) were not significantly different to those within the LC group ($M = 1.01$, $SEM = 0.32$) and as response to low caloric food targets ($M_{HC} = 0.98$, $SEM = 0.42$ vs. $M_{LC} = 0.75$, $SEM = 0.41$).

Participants belonging to the HC group did not omit significantly more errors within shift blocks ($M = -0.30$, $SEM = 0.32$) or non-shift blocks ($M = 1.88$, $SEM = 0.51$) than participants belonging to the LC group ($M_{Shift} = -0.38$, $SEM = 0.32$ vs. $M_{Non-shift} = 2.13$, $SEM = 0.50$).

Figure 34: Group-related difference scores regarding the number of omission errors



Mean difference scores of omission errors as a function of target type and block type regarding to both intervention groups. Error bars indicating the standard error of the mean.

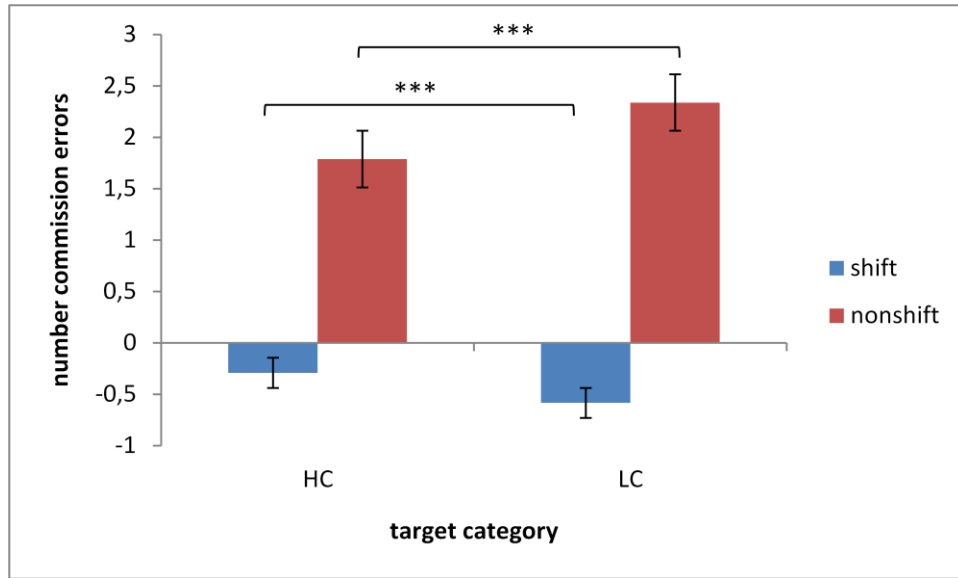
Commission errors

Referring to the number of commission errors, the main effect for testing phase was significant ($F(1,84) = 12.72$; $p < 0.001$; $np^2 = .132$), while the main effect for group classification was not significant ($F(1,84) = 0.05$; $p = .833$; $np^2 = .001$), indicating that the priming intervention between both go/no-go tasks did affect the participants extent of impulsive reactions, but again there was no group-related effect.

Comparing the number of commission errors between both testing phases, the main effect for block type ($F(1,84) = 47.23$; $p < .001$; $np^2 = .360$) was significant again. That implies, that participants committed significantly more errors in non-shift blocks ($M = 2.06$; $SEM = 0.36$) than in shift blocks ($M = -0.44$; $SEM = 0.20$).

The main effect for target type ($F(1,84) = 0.32$; $p = .574$; $np^2 = .004$) as well as the interaction effect of target type * block type * testing phase ($F(1,84) = 3.28$; $p = .074$; $np^2 = .038$) were not significant. Therefore the number of commission errors in response to high caloric ($M = 0.75$; $SEM = 0.25$) and low caloric ($M = 0.88$; $SEM = 0.26$) food targets did not differ, when comparing both testing phases, as stated in *figure 35*. Additionally, committed errors in response to high caloric or low caloric food targets were not dependent on block type between the tasks.

Figure 35: Difference scores regarding the number of commission errors in both go/no-go tasks

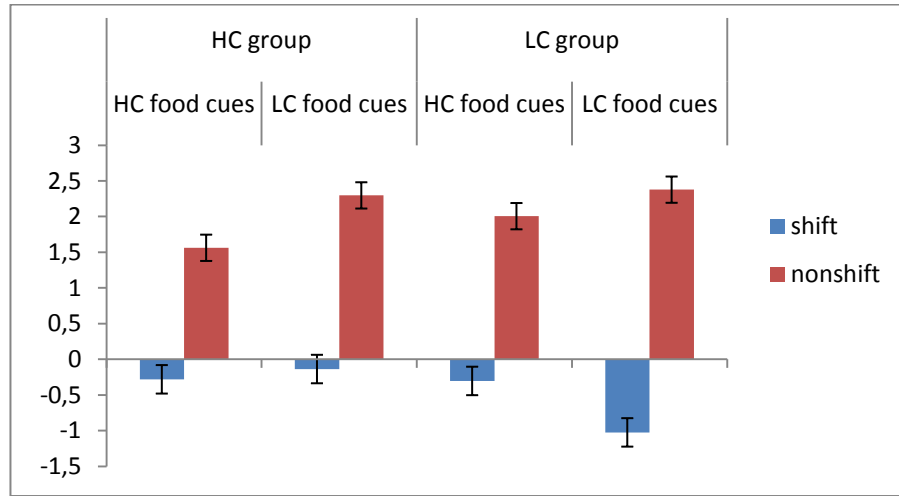


Mean difference scores of commission errors (phase III – phase I) as a function of target type and block type. Error bars indicating the standard error of the mean. (*) $p < .001$**

Regarding the different intervention groups, the main effects for target type ($F(1,84) = 1.84$; $p = .179$; $np^2 = .022$) and block type ($F(1,84) = 0.97$; $p = .328$; $np^2 = .012$) as well as the interaction effect target type * block type * testing phase ($F(1,84) = 0.29$; $p = .593$; $np^2 = .003$) were not significant.

Therefore the number of commission errors in response to high caloric food targets within the HC group ($M = 0.64$; $SEM = 0.36$) was not significantly different to those within the LC group ($M = 0.85$; $SEM = 0.36$). Similar to this, the number of committed errors in response to low caloric food cues did not differ between the formed groups ($M_{HC} = 1.08$; $SEM = 0.37$ vs. $M_{LC} = 0.68$; $SEM = 0.36$). Neither there were significant differences between the results of the HC group within shift blocks ($M = -0.21$; $SEM = 0.29$) and non-shift blocks ($M = 1.93$; $SEM = 0.51$) and the LC group within shift blocks ($M = -0.66$; $SEM = 0.28$) and non-shift blocks ($M = 2.19$; $SEM = 0.51$). Group-related difference scores regarding to the number of omission errors are depicted in *figure 36*.

Figure 36: Group-related difference scores regarding the number of commission errors



Mean difference scores of commission errors as a function of target type and block type regarding to both intervention groups. Error bars indicating the standard error of the mean.

Correlation of the priming measures with questionnaire scores

Only within the LC intervention group the number of detected mistakes correlated with the time spent to find them ($r(83) = .305, p < .05$), while there was no significant correlation between the measures within the HC intervention group.

As illustrated in *table 16*, the scores of the participants' actual subjective mood did not correlate with measurements of the priming intervention, similar to prior conducted correlation analysis between questionnaire scores and task-related measures. Neither did trait impulsivity correlate with reaction times or number of found mistakes within the intervention groups.

Table 16: Correlation of the priming measures with questionnaire scores

	WhatsApp – LC group		WhatsApp – HC group	
	Reaction time	Mistakes	Reaction time	Mistakes
ASTS	.179	-.007	-.059	.234
FRK	-.063	-.415**	-.104	-.011
BIS-15	.077	.047	.250	-.143
PSRS	.032	.260	-.139	.326*
FCQ-T-r	-.197	-.350*	.149	-.094
BOT	-.015	-.351*	.068	-.218

* $p < .05$

** $p < .01$

On the contrary, participants' rigid control of eating behaviour was negatively correlated ($r(83) = -.415, p < .01$) with the number of found mistakes within the LC intervention group. In other words, the higher the extent of restrained eating behaviour among students of the LC group was, the less hidden mistakes they found within the sent pictures.

Furthermore, scores of the trait food craving questionnaire ($r(83) = -.350, p < .05$) and the Bratman's orthorexia test ($r(83) = -.351, p < .05$) were negatively correlated with the number of found mistakes within the LC group. Therefore, the higher the disposition to *orthorexia nervosa* or trait food craving among students of the LC intervention group were, the less hidden changes they detected within the pictures.

The only significant correlation within the HC intervention group was ascertained between the number of found mistakes and the participants' success in dieting ($r(83) = .326, p < .05$). To put it in other words, the more successful students were in dieting, the more hidden mistakes they found.

Correlation of the priming measures with the number of commission errors in the go/no-go task of phase III

When taking the interaction between the priming intervention and the number of commission errors within the subsequent go/no-go task into account, only the number of found mistakes within the HC intervention group was significantly correlated with the total number of committed errors in response to high caloric food targets ($r(83) = -.308, p < .05$). In other words, the more hidden mistakes participants of the high caloric intervention group found, the less often they committed errors within the second go/no-go task in response to high caloric food targets.

As also stated in *table 17*, there was no other significant correlation between task measures of the priming intervention and the number of commission errors in the second affective shifting task.

Table 17: Correlation between the priming intervention and the number of commission errors

	LC food blocks			HC food blocks			Non-Shift	Shift
	Non-Shift	Shift	total	Non-Shift	Shift	total		
LC group - reaction time	.009	-.117	-.057	-.081	-.257	-.213	-.035	-.264
LC group - mistakes	.243	.020	.183	.260	-.122	.100	.262	-.075
HC group - reaction time	-.095	-.019	-.079	-.124	-.044	-.107	-.114	-.031
HC group - mistakes	-.169	-.258	-.299	-.247	-.282	-.308*	-.215	-.293

* $p < .05$

Prediction of commission errors

In addition to the conducted regression analyses in phase I and phase III, the target variables of the priming intervention were undertaken a further linear regression analysis, in order to evaluate the predicting capacity of those measures for the number of commission errors in the second go/no-go task. The standardized coefficients and their significance levels within the linear regression analyses were depicted in *table 18*.

Table 18: Prediction capacity of priming measures for the number of commission errors within the second go/no-go task

	LC food blocks				HC food blocks			
	Non-shift		Shift		Non-shift		Shift	
	β	<i>p</i> -value	β	<i>p</i> -value	β	<i>p</i> -value	β	<i>p</i> -value
LC group – reaction time	.009	.956	-.117	.457	-.081	.607	-.257	.096
LC group – mistakes	.243	.116	.020	.901	.260	.092	-.122	.436
HC group – reaction time	-.095	.548	-.019	.907	-.124	.433	-.044	.783
HC group – mistakes	-.169	.284	-.258	.099	-.247	.115	-.282	.070
	Non-shift total		Shift total		LC total		HC total	
	β	<i>p</i> -value	β	<i>p</i> -value	β	<i>p</i> -value	β	<i>p</i> -value
LC group – reaction time	-.035	.824	-.264	.087	-.057	.717	-.213	.169
LC group – mistakes	.262	.090	-.075	.633	.183	.239	.100	.522
HC group – reaction time	-.114	.473	-.031	.846	-.079	.620	-.107	.499
HC group – mistakes	-.215	.171	-.293	0.60	-.299	.055	-.308	.047*

* $p < .05$

Therefore, the number of found mistakes in the HC intervention group predicted the number of commission errors in response to high caloric food targets in total within the second go/no-go task ($p = .047$). Referring to the linear regression equation $Y = \beta_0 + \beta_1 * x_1 + \varepsilon$, the number of commission errors decreased from 13.71 to 12.18 errors, if the amount of found mistakes within the HC intervention group increased by one unit.

All other priming measures could not predict the number of commission errors (all $p > .05$).

5 Discussion

Lower inhibitory control and higher reward sensitivity, especially in response to highly palatable, high caloric food cues have been shown to be major determinants for overeating. A commonly used tool for measuring the extent of one's impulsivity is an affective shifting task. Within this tool, individuals' motor response inhibition, shifting of attention and the influence of salient cues to executive functions can be examined [MEULE and KÜBLER, 2014; TESLOVICH *et al.*, 2014; NOËL *et al.*, 2007].

Similar to Meule and Kübler (2014) the present affective shifting task has been conceptualised, but in contrast to their work the experimenter gave instructions regarding participants' food intake prior to the affective shifting task as they were briefed not to consume any food or beverages at least two hours prior to testing. To eliminate the influence of food deprivation on task performance and to achieve comparable states of hunger and satiety among the study sample, students received a glass of apple juice before they filled out the questionnaire scores and completed the go/no-go task.

As priming effects are related to a higher accessibility of hedonic aspects of food in restrained eaters [PAPIES *et al.*, 2009] and therefore might additionally influence individuals' inhibitory control, the current study combined a priming paradigm to an affective shifting task (go/no-go task). In both tools high caloric and low caloric food cues were used as pictorial stimuli.

Within preliminary analysis using subset questionnaire scores there were no differences among both intervention groups with regard to self-reported state of mood, rigid control of eating behaviour, trait impulsivity, perceived success in dieting, trait food craving or the disposition to orthorectic eating behaviour. The influence of participants' dieting behaviour on task performance was investigated during the study by incorporating the questionnaire scores into further analyses.

Previous study results suggest that trait impulsivity, trait food craving and the number of commission errors, particularly in high-caloric shift blocks are positively correlated [MEULE and KÜBLER, 2014]. Contrary to those findings, participants' trait impulsivity was not correlated with the number of commission errors in the current study and the

extent of trait food craving was negatively correlated with the number of commission errors in response to low caloric pictures in non-shift blocks.

With respect to the postulated sub-hypotheses, neither the point in time of last food consumption, nor the actual mood state, the trait impulsiveness or the disposition to *orthorexia nervosa* were correlated with the number of committed errors within the second go/no-go task.

As a result, the formulated null hypotheses of the sub-hypotheses I, II, IV & VII were accepted.

On the other hand, the success in dieting, trait food craving as well as participants' rigid control of eating behaviour were significantly correlated to the number of commission errors.

Therefore, the null hypotheses of the sub-hypotheses III, V, VI were rejected in favour of the alternative hypotheses.

Two main hypotheses were formulated, which raised the question whether the priming intervention and particularly the priming with high caloric food cues affects participants' extent of inhibitory control belayed by an increase in committed errors within the second affective shifting task.

The overall task performance within the second affective shifting task provided different results when compared to previous findings of *Meule and Kübler (2014)*. They found participants reacting slower and omitting more errors in response to high caloric food cues and the number of omission and commission errors was higher during shift-blocks [MEULE and KÜBLER, 2014]. In the present study participants reacted slower in response to high caloric food cues as well, but the number of omission and commission errors was higher in response to low caloric food stimuli during non-shift-blocks.

Regarding to the impact of the priming intervention phase, there was no remarkable change in reaction times between both affective shifting tasks, even not when taking the formed intervention groups into account. The number of omitted and committed errors did differ when comparing both go/no-go tasks in terms of a significantly higher

number of errors within the non-shift blocks in the second task. However, both target values were not related to participants' affiliation to the HC group.

Although there was no significant group-related impact to the defined target values of the go/no-go task, an inversely proportioned relationship between the priming target values (i.e. time to detect mistakes and the number of detected mistakes) and the numbers of committed errors during the second affective shifting task was discovered. In particular, the more hidden mistakes were found within the HC intervention group, the less errors participants committed in response to high caloric food targets. Actually, the number of detected mistakes within the HC group predicted the number of commission errors in response to high caloric food stimuli.

With respect to the present results the null hypothesis of the main hypotheses I was rejected. Therefore the priming intervention phase in total did influence impulsivity in participating females, although the detected alterations within the second go/no-go task might be due to a kind of learning effect.

As the priming with pictures of high caloric foods in particular had no effect on the number of committed errors when comparing both affective shifting tasks, the null hypothesis of the main hypotheses II could not be rejected.

The weak priming effects might be due to participants' awareness of the priming event as this condition is related to an adjustment away from the presumed effect. Another explanation might be that the priming manipulations were too exaggerated which increases the possibility to tip-off participants to the nature of the study [BARGH and CHARTRAND, 2014]. Furthermore, recruiting only students of nutritional sciences might have caused the weak effect as they are believed to be more sensitive to food-related items.

5.1 Concluding remark

In the following section the key highlights of the present study will be shortly summarised to give a concluding overview of the significant findings.

When, in a first step, only taking the results of the introductorily affective shifting task into account, participants reacted slower during shift blocks than compared to non-shift blocks whilst the target type of the presented stimuli had no effect on their reaction times. On the contrary, omission errors as well as commission errors were more frequent during non-shift blocks in response to low caloric food cues.

These findings are expected to be modulated by the fact that 57 % of the participating students were classified to be at risk of developing orthorectic eating behaviour, which can be characterised as overwhelming attention to consuming healthy foods and foods of high quality [BRYTEK-MATERA, 2012]. To put it more precisely, orthorectic eating behaviour was proven to predict the number of commission errors in response to low caloric food stimuli across non-shift blocks as well as in total, in a sense of decreasing error frequency when the orthorectic score increased.

Basically similar to this, individuals' perceived success in dieting was capable to predict the number of committed errors during shift blocks in response to low caloric food cues. Thus, the more attention participants paid to healthy foods and the more successful they perceived themselves in dieting the less frequent they committed errors during the first affective shifting task in response to low caloric food stimuli.

Within the second affective shifting task participants' overall task performance was quite similar when compared to the first go/no-go task. Participants reacted slower during shift blocks, particularly in response to high caloric food stimuli and omission errors as well as commission errors were more frequent during non-shift blocks in response to low caloric food cues again.

Analogous to the previous findings of the present study, the number of commission errors in response to low caloric food cues was inversely modulated by participants' perceived success in dieting and trait food craving. Additionally, a higher extent of rigid

control of eating behaviour predicted a decreasing number of commission errors in response to high caloric food stimuli during shift blocks as well as in total.

Interestingly, higher orthorectic scores predicted the frequency of commission errors within the second go/no-go task in an altered way. Therefore, participants committed more errors during the challenging shift blocks in response to high caloric food stimuli if their orthorectic eating behaviour was more distinct. This effect is thought to characterise individuals high in reward sensitivity, respectively individuals with low inhibitory control, as they react more sensitive in response to high caloric food stimuli, particularly during shift blocks when the target category switches [MEULE and KÜBLER, 2014]. Repeatedly priming participants with food stimuli was used to manipulate impulsivity and inhibition and might explain the altered effect among participants with higher risk of developing orthorectic eating behaviour.

When taking the influence of the priming intervention to the overall task performance into account, no group-related findings could be achieved but nevertheless there was a remarkable increase in both, omission and commission errors within the second affective shifting task. But because of the weak priming-related findings this increase might also be an effect of repetition in a sense of reacting more careless within the second go/no-go task.

5.2 Limitations

First, the sample size was rather small to offer statistically representative results.

Second, only healthy, young females attended the study and therefore the interpretation of the present results cannot be generalized for other samples, such as men [HEINRICH *et al.*, 2010]. Nonetheless, the solely recruitment of women avoided a confounding effect of gender and made results comparable to those of *Meule and Kübler (2014)*.

Third, the recruitment of students of nutritional sciences might have confounded the current results as they are possibly more aware of food-related issues than students of other disciplines.

Fourth, the choice of pictures for the high caloric and low caloric category might have influenced results of the go/no-go task as a lack of differentiation regarding that the caloric level was given.

Fifth, within the priming intervention, participants were not briefed to the total number of hidden mistakes. Maybe it would have been better to tell the exact number of changes to extend the timeframe in doing this task, respectively strengthen the priming effects.

5.3 Summary

Background

Human eating is mainly determined by homeostatic and hedonic regulatory pathways which interact with each other. The homeostatic regulation of food intake refers to maintaining energy balance and is controlled by a vast of hormonal and neural signals. Beyond metabolic needs, emotions and reward as well as associative learning and memory processes can powerfully shape one's eating behaviour as they contribute to motivational sensations such as appetite or food craving. The increasing incidence of obesity and the obesogenic environment emphasize the potent role of the hedonic regulation of food intake. Therefore, functioning self-regulation over highly palatable, high caloric food temptations plays a pivotal role in order to prevent weight gain since high reward sensitivity and low inhibitory control are thought to be main determinants for overeating. Furthermore, the perception of a prime stimulus can influence subsequent behaviour as it makes the respective content more accessible in mind and implicitly activates higher-order concepts.

Methods

The current study combined two affective shifting tasks (go/no-go tasks) with an intermediate priming intervention phase, but was designed as a camouflaged attention test. A total of 86 female undergraduate students of nutritional sciences participated in the experiment and were assigned either to the high caloric (HC group) or the low caloric intervention group (LC group) by drawing a number at random. Prior to the first go/no-go task, which provided baseline information, self-reported food deprivation, hunger parameters and questionnaire scores with regard to participants actual mood state, rigid control of eating behaviour, trait impulsiveness, success in dieting, trait food craving and their disposition to *orthorexia nervosa* were assessed. Within the priming intervention phase participants received modified pictures either of high or low caloric meals via the messenger application *WhatsApp* and were instructed to detect as many mistakes as possible in each picture. The pictures were thought to serve as prime stimuli. In order to investigate whether the priming intervention did change outcome parameters associated with inhibitory control in young females a second go/no-go task was conducted. Pictures of high caloric and low caloric foods were used as target and

non-target stimuli. The overall reaction times in go-trials, the number of omission errors and the number of commission errors were defined as target values.

Results

Trait food craving, the success in dieting as well as participants' rigid control of eating behaviour were significantly correlated to the number of commission errors. In a comparative measurement between both affective shifting tasks, participants' overall reaction times did not change significantly ($p = .095$). Subjects omitted and committed (all $p < .001$) more errors during non-shift blocks than during shift-blocks. Regarding to the different intervention groups, no group-related changes in reaction times ($p = .539$), the number of omission errors ($p = .851$) or the number of commission errors ($p = .833$) were detected.

Conclusion

The current findings suggest that although a repeatedly conducted affective shifting task provided altered results in participants' task performance, the intermediate priming intervention in particular was not powerful enough to change impulsivity-related outcome parameters. The weak priming effects might be due to subjects' awareness of the priming event itself or due to conceptual errors within the priming intervention phase.

5.4 Zusammenfassung

Hintergrund

Die menschliche Nahrungsaufnahme wird im Wesentlichen durch homöostatische und hedonische Regelkreise kontrolliert, welche gegenseitigen Wechselwirkungen unterliegen. Das homöostatische Regulationssystem basiert auf der Aufrechterhaltung der Energiereserven sowie des Energieverbrauches und wird durch eine Vielzahl von hormonellen und neuronalen Signalen gesteuert. Auf der anderen Seite üben Emotionen, Gedächtnisleistungen, Belohnungsverhalten sowie affektives Lernen unabhängig vom physiologischen Bedarf einen starken Einfluss auf das individuelle Essverhalten aus, da sich die genannten Funktionen unwillkürlich appetitanregend auswirken können. Die steigende Inzidenz von Übergewicht gepaart mit zunehmend übergewichtsfördernden Umweltfaktoren rückt die Rolle des hedonischen Regulationssystems in den Vordergrund. Dementsprechend wichtig sind gute Selbstregulationsprozesse, um den auftretenden Versuchungen in der Umwelt durch wohlschmeckende hochkalorische Lebensmittel widerstehen zu können. Die Neigung zu Belohnungsverhalten sowie eine geringe Fähigkeit bestehende Verhaltenstendenzen zu unterdrücken, werden als die maßgeblichen Ursachen übermäßigem Essens angesehen. Die Wahrnehmung und kognitive Verarbeitung eines Reizes (*prime Stimulus*) beeinflusst in diesem Zusammenhang zusätzlich nachfolgende Entscheidungen und Verhaltensweisen, indem Reizinhalt präsenter werden.

Methoden

Unter dem Vorwand eines Aufmerksamkeitstests wurde die vorliegende Studie als Kombination zweier *affective shifting tasks (go/no-go tasks)* mit einer dazwischenliegenden Priming Interventionsphase entwickelt. Insgesamt haben 86 weibliche Bachelorstudentinnen des Studiums für Ernährungswissenschaften an der Studie teilgenommen. Diese wurden durch zufälliges Ziehen einer Nummer entweder der hochkalorischen oder der niederkalorischen Interventionsgruppe zugeteilt. Vor Durchführung des ersten go/no-go task, welcher zur Ermittlung von Basisdaten diente, wurde der Zeitpunkt der letzten Nahrungsaufnahme sowie das subjektive Hunger- und Sättigungsgefühl ermittelt. Diverse Fragebögen über die momentane Stimmungslage sowie die subjektive Einschätzung rigider Kontrolle des Essverhaltens, der Impulsivität,

des Erfolges im Diäthalten, des Verlangens nach Essen und der Neigung zur Essstörung *Orthorexia nervosa* wurden ebenfalls ermittelt. Im Zuge der Priming Interventionsphase erhielten die Teilnehmer modifizierte Bilder von hochkalorischen bzw. niederkalorischen Speisen über das Messenger-Programm *WhatsApp*. Die Aufgabenstellung war, möglichst viele versteckte Fehler zu entdecken, während die Bilder tatsächlich als *prime* Stimuli dienten. Ein nachfolgender affective shifting task wurde durchgeführt, um mögliche priming Effekte auf die inhibitorische Kontrolle der Teilnehmerinnen herauszufinden. Hierbei wurden ebenfalls hochkalorische und niederkalorische Nahrungsmittel als Stimuli eingesetzt und die Reaktionszeiten, die Anzahl an Auslassungsfehlern und die Anzahl zusätzlicher Fehler als Zielvariablen definiert.

Ergebnisse

Das subjektive Ausmaß des Verlangens nach Essen, des Erfolges im Diäthalten sowie der rigiden Kontrolle des Essverhaltens korrelierten signifikant mit der Anzahl zusätzlicher Fehler im go/no-go task. Im Vergleich beider affective shifting tasks veränderten sich die Reaktionszeiten der Teilnehmer nicht ($p = .095$), während die Studenten aber mehr Auslassungsfehler und auch mehr zusätzliche Fehler (alle $p < .001$) in non-shift Blöcken machten. In Bezug auf die unterschiedlichen Interventionsgruppen konnten weder Veränderungen in den Reaktionszeiten ($p = .539$), noch in der Anzahl der Auslassungsfehler ($p = .851$) oder zusätzlichen Fehler ($p = .833$) festgestellt werden.

Fazit

Die vorliegenden Studienergebnisse deuten darauf hin, dass sich bei wiederholter Durchführung des go/no-go tasks unterschiedliche Resultate ergeben. Der Einfluss der dazwischenliegenden priming Interventionsphase war jedoch nicht groß genug, um die mit Impulsivität assoziierten Zielparameter zu verändern. Dies könnte einerseits auf möglichem Gewahrsein der Studieninhalte oder auf konzeptuellen Fehlern im Studiendesign der priming Interventionsphase basieren.

6 Appendix

6.1 References

- ALLOM V, MULLAN B.** Individual differences in executive function predict distinct eating. *Appetite*, 2014; 80: 123-130.
- AMES SL, KISBU-SAKARYA Y, REYNOLDS KD, BOYLE S, CAPPELI C, COX MG, DUST M, GRENARD JL, MACKINNON DP, STACY AW.** Inhibitory control effects in adolescent binge eating and consumption of sugar-sweetened beverages and snacks. *Appetite*, 2014; 81: 180-192.
- AMUNTS K, KEDO O, KINDLER M, PIEPERHOFF P, MOHLBERG H, SHAH NJ, HABEL U, SCHNEIDER F, ZILLES K.** Cytoarchitectonic mapping of the human amygdala, hippocampus region and entorhinal cortex: intersubject variability and probability maps. *Anatomy and Embryology*, 2005; 210: 343-352.
- BADONELL K, DURIEUX D, MONNERIE R, GRÉBERT D, SALESSE R, CAILLOL M, BALLY C.** Leptin-sensitive OBP-expressing mucous cells in rat olfactory epithelium: a novel target for olfaction-nutrition crosstalk?
- BARGH JA, CHARTRAND TL.** The Mind in the Middle: A Practical Guide to Priming and Automaticity Research. In: *Handbook of Research Methods in Social and Personality Psychology*. Second Edition (REIS HT, JUDD CM; Eds.), Cambridge University Press, New York, 2014; 311-344.
- BARI A, ROBBINS TW.** Inhibition and impulsivity: Behavioral and neural basis of response control. *Progress in Neurobiology*, 2013; 108: 44-79.
- BATTERHAM RL, FFYTCH DH, ROSENTHAL JM, ZELAYA FO, BARKER GJ, WITHERS DJ, WILLIAMS SC.** PYY modulation of cortical and hypothalamic brain areas predicts feeding behaviour in humans. *Nature*, 2007; 450: 106-109.
- BGLINGER C, DEGEN L, MATZINGER D, D'AMATO M, DREWE J.** Loxiglumide, a CCK-A receptor antagonist, stimulates calorie intake and hunger feelings in humans. *American Journal of Physiology – Regulatory, Integrative and Comparative Physiology*, 2001; 280: R1149-R1154.

- BERTHOUD HR, MÜNZBERG H.** The lateral hypothalamus as integrator of metabolic and environmental needs: From electrical self-stimulation to opto-genetics. *Physiology&Behavior*, 2011; 29-39.
- BERTHOUD HR.** Metabolic and hedonic drives in the neural control of appetite: who is the boss? *Current opinion in Neurobiology*, 2011; 21: 888-896.
- BLECHERT J, MEULE A, BUSCH NA, OHLA K.** Food-pics: an image database for experimental research on eating and appetite. *Frontiers in Psychology*, 2014; 5:617.
- BLEVINS JE, STANLEY BG, REIDELBERGER RD.** Brain regions where cholecystokinin suppresses feeding in rats. *Brain Research*, 2000; 860: 1-10.
- BLOUET C, SCHWARTZ GJ.** Hypothalamic nutrient sensing in the control of energy homeostasis. *Behavioural Brain Research*, 2010; 209: 1-12.
- BLUNDELL J.** Pharmacological approaches to appetite suppression. *Trends in Pharmacological Sciences*, 1991; 12: 147-157.
- BOLTON HM, BURGESS PW, GILBERT SJ, SERPELL L.** Increased Set Shifting Costs in Fasted Healthy Volunteers. *Plos One*, 2014; 9 (7): e101945.
- BONGERS P, JANSEN A, HAVERMANS R, ROEFS A, NEDERKOORN C.** Happy eating. The underestimated role of overeating in a positive mood. *Appetite*, 2013; 67: 74-80.
- BRUNSTROM JM, BURN JF, SELL NR, COLLINGWOOD JM, ROGERS PJ, WILKINSON LL, HINTON EC, MAYNARD OM, FERRIDAY D.** Episodic Memory and Appetite Regulation in Humans. *Plos One*, 2012; 7 (12): e50707.
- BRYTEK-MATERA A.** Orthorexia nervosa – an eating disorder, obsessive-compulsive disorder or disturbed eating habit? *Archives of Psychiatry and Psychotherapy*, 2012; 1: 55-60.
- CASTRO DC, BERRIDGE KC.** Advances in the neurobiological bases for food ‘liking’ versus ‘wanting’. *Physiology & Behavior*, 2014; *In Press*.
- CECH DJ, MARTIN S.** Chapter 9: Nervous System Changes, In: *Functional Movement Development Across the Lifespan* (DUNCAN L, Ed.), Elsevier Saunders, St.Louis, 2012; 174-212.

- CHAPUT JP, GILBERT JA, GREGERSEN NT, PEDERSEN SD, SJÖDIN AM.** Comparison of 150-mm *versus* 100-mm visual analogue scales in free living adult subjects. *Appetite*, 2010; 54: 583-586.
- COLLINS AM, LOFTUS EF.** A Spreading-Activation Theory of Semantic Processing. *Psychological Review*, 1975; 82 (6): 407-428.
- CSERJÉSI R, LUMINET O, PONCELET AS, LÉNÁRD L.** Altered executive function in obesity. Exploration of the role of affective states on cognitive abilities. *Appetite*, 2009; 52: 535-539.
- DALBERT C.** ASTS. Aktuelle Stimmungsskala. Martin-Luther-Universität Halle-Wittenberg, 2009;
[<http://www.erzwiss.uni-halle.de/gliederung/paed/ppsych/instd1.htm> (accessed on 21.05.2014)]
- DALTON M, FINLAYSON G.** Psychological examination of liking and wanting for fat and sweet taste in trait binge eating females. *Physiology&Behavior*, 2014; *In Press*.
- DAVIDSON TL, KANOSKI SE, WALLS EK, JARRARD LE.** Memory inhibition and energy regulation. *Physiology&Behavior*, 2005; 86: 731-746.
- DEL PRETE A, IADEVAIA M, LOGUERCIO C.** The role of gut hormones in controlling the food intake. What is their role in emerging diseases? *Endocrinología y Nutrición*, 2012; 59 (3). 197-206.
- DESMET PMA, SCHIFFERSTEIN HNJ.** Sources of positive and negative emotions in food experience. *Appetite*, 2008; 50: 290-301.
- DOSSAT AM, LILLY N, KAY K, WILLIAMS DL.** Glucagon-Like Peptide 1 Receptors in Nucleus Accumbens Affect Food Intake. *The Journal of Neuroscience*, 2011; 31 (41): 14453-14457.
- EBIHARA K, OGAWA Y, KATSUURA G, NUMATA Y, MASUZAKI H, SATOH N, TAMAKI M, YOSHIOKA T, HAYASE M, MATSUOKA N, AIZAWA-ABE M, YOSHIMASA Y, NAKAO K.** Involvement of agouti-related protein, an endogenous antagonist of hypothalamic melanocortin receptor, in leptin action. *Diabetes*, 1999; 48 (10): 2028-2033.

- ELIAS CF, SAPER CB, MARATOS-FLIER E, TRITOS NA, LEE C, KELLY J, TATRO JB, HOFFMAN GE, OLLMANN MM, BARSH GS, SAKURAI T, YANAGISAWA M, ELMQUIST JK.** Chemically defined projections linking the mediobasal hypothalamus and the lateral hypothalamic area. *The Journal of Comparative Neurology*, 1998; 402 (4): 442-459.
- ELMADFA I, HASENEGGER V, WAGNER K, PUTZ P, WEIDL NM, WOTTAWA D, KUEN T, SEIRINGER G, MEYER AL, STURTZEL B, KIEFER I, ZILBERSZAC A, SGARABOTTOLO V, MEIDLINGER B, RIEDER A.** Österreichischer Ernährungsbericht 2012. Wien, 2012; 2-8.
- ELY AV, WINTER S, LOWE MR.** The generation and inhibition of hedonically-driven food intake: Behavioral and neurophysiological determinants in healthy weight individuals. *Physiology&Behavior*, 2013; 121: 25-34.
- EVERS C, ADRIAANSE M, DE RIDDER DTD, DE WITT HUBERTS JC.** Good mood food. Positive emotion as a neglected trigger for food intake. *Appetite*, 2013; 8: 1-7.
- FADEL J, DEUTCH AY.** Anatomical substrates of orexin-dopamine interactions: lateral hypothalamic projections to the ventral tegmental area. *Neuroscience*, 2002; 111 (2): 379-387.
- FERNANDES MF, SHARMA S, HRYHORCZUK C, AUGUSTE S, FULTON S.** Nutritional Controls of Food Reward. *Canadian Journal of Diabetes*, 2013; 37:260-268.
- FERRÉ P, SÁNCHEZ-CASAS R.** Affective priming in a lexical decision task: Is there an effect of words' concreteness? *Psicológica*, 2014; 35: 117-138.
- FERRIDAY D, BRUNSTROM JM.** How does food-cue exposure lead to larger meal sizes? *British Journal of Nutrition*, 2008; 100: 1325-1332.
- FIELD A.** *Discovering Statistics Using SPSS (and sex, drugs and rock 'n' roll)*. Sage Publications, London, Thousand Oaks, New Delhi, 2005; 668-670 and 143-152.
- FINLAYSON G, KING N, BLUNDELL J.** The role of implicit wanting in relation to explicit liking and wanting for food: Implications for appetite control. *Appetite*, 2008; 50: 120-127.

- FOERDE K.** Implicit Learning and Memory: Psychological and Neural Aspects. In: Encyclopedia of Behavioral Neuroscience (KOOB G, LE MOAL M, THOMPSON R, Eds.), Elsevier Academic Press, London, Burlington, San Diego, 2010; 84-93.
- FORONI F, PERGOLA G, ARGIRIS G, RUMIATI RI.** The Food Cast Research Image Database (FRIDa). *Frontiers in Human Neuroscience*, 2013; 7:51.
[doi: 10.3389/fnhum.2013.00051]
- FRIHAUF JB, ZORILLA EP, FEKETE EM.** Control of Food Intake. *Encyclopedia of Behavioral Neuroscience*, 2010; 335-344.
- FROST GS, BRYNES AE, DHILLO WS, BLOOM SR, MCBURNEY MI.** The effects of fiber enrichment of pasta and fat content on gastric emptying, GLP-1, glucose, and insulin responses to a meal. *European Journal of Clinical Nutrition*, 2003; 57: 293-298.
- FULTON S.** Appetite and reward. *Frontiers in Neuroendocrinology*, 2010; 31: 85-103.
- GAILLET M, SULMONT-ROSSÉ C, ISSANCHOU S, CHABANET C, CHAMBARON S.** Priming effects of an olfactory food cue on subsequent food-related behaviour. *Food Quality and Preference*, 2013; 30: 74-281.
- GOLDSTONE AP, PRECHTL DE HERNANDEZ CG, BEAVER JD, MUHAMMED K, CROESE C, BELL G, DURIGHEL G, HUGHES E, WALDMAN AD, FROST G, BELL JD.** Fasting biases brain reward systems towards high-calorie foods. *European Journal of Neurosciences*, 2009; 30: 1625-1635.
- GUERRIERI R, NEDERKOORN C, SCHROOTEN M, MARTIJN C, JANSEN A.** Inducing impulsivity leads high and low restrained eaters into overeating, whereas current dieters stick to their diet. *Appetite*, 2009; 53: 93-100.
- HALFORD JCG, BLUNDELL JE.** Separate systems for serotonin and leptin in appetite control. *Annals of Medicine*, 2000; 32 (3): 222-232.
- HARROLD JA, Dovey TM, Blundell JE, Halford JCG.** CNS regulation of appetite. *Neuropharmacology*, 2012; 63: 3-17.
- HARROLD JA.** Hypothalamic Control of Energy Balance. *Current Drug Targets*, 2004; 5: 207-219.

- HEBBEN N, CORKIN S, EICHENBAUM H, SHEDLACK K.** Diminishing Ability to Interpret and Report Internal States After Bilateral Median Temporal Resection: Case H.M. *Behavioral Neuroscience*, 1985; 99 (6): 1031-1039.
- HEINRICH J, HEINE SJ, NORENZAYAN A.** The weirdest people in the world? *Behavioral and Brain Sciences*, 2010; 33: 61-135.
- HEMMER P, STEYVERS M.** Integrating episodic memories and prior knowledge at multiple levels of abstraction. *Psychonomic Bulletin&Review*, 2009; 16 (1): 80-87.
- HEPWORTH R, MOGG K, BRIGNELL C, BRADLEY BP.** Negative mood increases selective attention to food cues and subjective appetite. *Appetite*, 2010; 54: 134-142.
- HIGGINS ET, RHOLES WS, JONES CR.** Category Accessibility and Impression Formation. *Journal of Experimental Social Psychology*, 1977 (13): 141-154.
- HIGGS S, DONOHOE JE.** Focusing on food during lunch enhances lunch memory and decreases later snack intake. *Appetite*, 2011; 57: 202-206.
- HIGGS S.** Cognitive influences on food intake: The effects of manipulating memory for recent eating. *Physiology&Behavior*, 2008; 94: 734-739.
- HOFMANN W, SCHMEICHEL BJ, BADDELEY AD.** Executive functions and self-regulation. *Trends in Cognitive Sciences*, 2012; 16 (3): 174-180.
- HOMMEL JD, TRINKO R, SEARS RM, GEORGESCU D, LIU ZW, GAO XB, THURMON JJ, MARINELLI M, DiLEONE RJ.** Leptin Receptor Signaling in Midbrain Dopamine Neurons Regulates Feeding. *Neuron*, 2006; 51: 801-810.
- JANISZEWSKI C, WYER RS.** Content and process priming: A review. *Journal of Consumer Psychology*, 2014; 24 (1): 96-118.
- JIANG Y, KING JM, PRINYAWIWATKUL W.** A review of measurement and relationships between food, eating behavior and emotion. *Trends in Food Science&Technology*, 2014; 36: 15-28.
- JOHNSON AW.** Eating beyond metabolic need: how environmental cues influence feeding behavior. *Trend in Neurosciences*, 2013; 36 (2): 101-109.
- KANOSKI SE.** Cognitive and neuronal systems underlying obesity. *Physiology&Behavior*, 2012; 106: 337-344.

- KENNY PJ.** Reward Mechanisms in Obesity: New Insights and Future Directions. *Neuron*, 2011; 69(4): 664-679.
- KRINGELBACH ML, STEIN A, VAN HARTEVELT TJ.** The functional human neuroanatomy of food pleasure cycles. *Physiology&Behavior*, 2012; 106: 307-316.
- KRISTENSEN P, JUDGE ME, THIM L, RIBEL U, CHRISTJANSEN KN, WULFF BS, CLAUSEN JT, JENSEN PB, MADSEN OD, VRANG N, LARSEN PJ, HASTRUP S.** Hypothalamic CART is a new anorectic peptide regulated by leptin. *Nature*, 1998; 393: 72-76.
- LASHLEY KS.** The Problem of Serial Order in Behaviour. In: *Cerebral mechanisms in behaviour: The Hixon symposium* (JEFFRESS LA; Ed.), Wiley, New York, Chapman&Hall, London, 1951: 112-136.
- LAVIN JH, WITTERT GA, ANDREWS J, YEAP B, WISHART JM, MORRIS HA, MORLEY JE, HOROWITZ M, READ NW.** Interaction of insulin, glucagon-like peptide 1, gastric inhibitory polypeptide, and appetite in response to intraduodenal carbohydrate. *The American Journal of Clinical Nutrition*, 1998; 68: 591-598.
- LEIBOWITZ SF, WEISS GF, SUH JS.** Medial hypothalamic nuclei mediate serotonin's inhibitory effect on feeding behavior. *Pharmacology Biochemistry and Behavior*, 1990; 37 (4): 735-742.
- LENARD N, BERTHOUD HR.** Central and Peripheral Regulation of Food Intake and Physical Activity: Pathways and Genes. *Obesity*, 2008; 16 (3): 11-22.
- LOGUE SF, GOULD TJ.** The neural and genetic basis of executive function: Attention, cognitive flexibility, and response inhibition. *Pharmacology, Biochemistry and Behavior*, 2013; *In Press*.
- LOXTON NJ, DAWE S, CAHILL A.** Does negative mood drive the urge to eat? The contribution of negative mood, exposure to food cues and eating style. *Appetite*, 2011; 56: 368-374.
- LUCAS M.** Semantic priming without association: A meta-analytic review. *Psychonomic Bulletin&Review*, 2000; 7 (4): 618-630.
- MARTIN AA, DAVIDSON TL.** Human cognitive function and the obesogenic environment. *Physiology&Behavior*, 2014; *In Press*.

- MEULE A, HERMANN T, KÜBLER A.** A short version of the Food Cravings Questionnaire-Trait: the FCQ-T-reduced. *Frontiers in Psychology*, 2014; 5 (190): 1-10.
- MEULE A, KÜBLER A.** Double trouble. Trait food craving and impulsivity interactively predict food-cue affected behavioral inhibition. *Appetite*, 2014; 79: 174-182.
- MEULE A, LUTZ A, KRAWIETZ V, STÜTZER J, VÖGELE C, KÜBLER A.** Food-cue affected motor response inhibition and self-reported dieting success: a pictorial affective shifting task. *Frontiers in Psychology*, 2014; 5: 216.
- MEULE A, PAPIES EK, KÜBLER A.** Differentiating between successful and unsuccessful dieters. Validity and reliability of the Perceived Self-Regulatory Success in Dieting Scale. *Appetite*, 2012; 58: 822-826.
- MEULE A, VÖGELE C, KÜBLER A.** Psychometrische Evaluation der deutschen Barratt Impulsiveness Scale – Kurzversion (BIS-15). *Diagnostica*, 2011; 57(3): 126-133.
- MISCHEL W, SHODA Y, RODRIGUEZ ML.** Delay of Gratification in Children. *Science*, 1989; 244 (4907): 933-938.
- MEYE FJ, ADAN R.** Feelings about food: the ventral tegmental area in food reward and emotional eating. *Trends in Pharmacological Science*, 2014; 35(1): 31-40.
- NAITO N, KAWAZOE I, NAKAI Y, KAWAUCHI H.** Melanin-concentrating hormone like immunoreactive material in the rat hypothalamus; characterization and subcellular localization. *Cell and Tissue Research*, 1988; 253: 291-295.
- NOËL X, VAN DER LINDEN M, D'ACREMONT M, BECHARA A, DAN B, HANAK C, VERBANCK P.** Alcohol cues increase cognitive impulsivity in individuals with alcoholism. *Psychopharmacology*, 2007; 192: 291-298.
- OECD.** OECD Health Statistics 2014, 2014a
[Internet: www.oecd.org/health/healthdata, accessed on 22.07.2014)]
- OECD.** OECD Obesity Update 2014, 2014b
[Internet: www.oecd.org/health/obesity-update.htm, accessed on 22.07.2014)]
- ORTELLS JJ, VELLIDO C, DAZA MT, NOUERA C.** Semantic priming effects with and without perceptual awareness. *Psicológica*, 2006; 27: 225-242.

- PAPIES** EK, **STROEBE** W, **AARTS** H. Pleasure in the mind: Restrained eating and spontaneous hedonic thoughts about food. *Journal of Experimental Social Psychology*, 2007; 43: 810-817.
- PAPIES** EK, **STROEBE** W, **AARTS** H. Who likes it more? Restrained eaters' implicit attitudes towards food. *Appetite*, 2009; 53: 279-287.
- PEIRCE** JW. Generating stimuli for neuroscience using PsychoPy. *Frontiers in Neuroinformatics*, 2009; 2:10. doi:10.3389/neuro.11.010.2008
- PETROVICH** GD. Forebrain networks and the control of feeding by environmental learned cues. *Physiology&Behavior*, 2013; 121: 10-18.
- PETROVICH** GD. Learning and the motivation to eat: Forebrain circuitry. *Physiology&Behavior*, 2011; 104: 582-589.
- RENSHAW** D, **BATTERHAM** RL. Peptide YY: A Potential Therapy for Obesity. *Current Drug Targets*, 2005; 6 (2): 171-179.
- RICHARD** D, **TIMOFEEVA** E. Energy Balance Regulation: Complex Interplay between the Autonomic and Cognitive/Limbic Brains to Control Food Intake and Thermogenesis. In: *Obesity Prevention: The Role of Brain and Society on Individual Behavior* (Dube et al., Eds.), Elsevier, Amsterdam, 2010; 299-316.
- RIGGS** NR, **SPRUIJT-METZ** D, **SAKUMA** KL, **CHOU** CP, **PENTZ** MA. Executive Cognitive Function and Food Intake in Children. *Journal of Nutrition Education and Behavior*, 2010; 42 (6): 398-403.
- ROTENBERG** KJ, **LANCASTER** C, **MARSDEN** J, **PRYCE** S, **WILLIAMS** J, **LATTIMORE** P. Effects of priming thoughts about control on anxiety and food intake as moderated by dietary restraint. *Appetite*, 2005; 44: 235-241.
- ROUSMANS** S, **ROBIN** O, **DITTMAR** A, **VERNET-MAURY** E. Autonomic Nervous System Responses Associated with Primary Tastes. *Chemical Senses*, 2000; 25: 709-718.
- SHIGEMURA** N, **OHTA** R, **KUSAKABE** Y, **MIURA** H, **HINO** A, **KOYANO** K, **NAKASHIMA** K, **NINOMIYA** Y. Leptin Modulates Behavioral Responses to Sweet Substances by Influencing Peripheral Taste Structures. *Endocrinology*, 2004; 145 (2): 839-847.

- SIEP N, ROEFS A, ROEBROECKA, HAVERMANS R, BONTE ML, JANSEN A.** Hunger is the best spice: An fMRI study of the effects of attention, hunger and calorie content on food reward processing in the amygdala and orbitofrontal cortex. *Behavioural Brain Research*, 2009; 198: 149-158.
- SOON CS, BRASS M, HEINZE HJ, HAYNES JD.** Unconscious determinants of free decisions in the human brain. *Nature Neuroscience*, 2008; 11 (5): 543-545.
- STAMATAKIS AM, SPARTA DR, JENNINGS JH, MCELLIGOTT ZA, DECOT H, STUBER GD.** Amygdala and bed nucleus of the stria terminalis circuitry: Implications for addiction-related behaviors. *Neuropharmacology*, 2014; 76: 320-328.
- STORMS LH.** Apparent backward association: A situational effect. *Journal of Experimental Psychology*, 1958; 5 (4): 390-395.
- STROEBE W, MENSINK W, AARTS H, SCHUT H, KRUGLANSKI AW.** Why dieters fail: Testing the goal conflict model of eating. *Journal of Experimental Social Psychology*, 2008; 44: 26-36.
- STUBBS RJ, HUGHES DA, JONESTONE AM, ROWLEY E, REID C, ELIA M, STRATTON R, DELARGY H, KING N, BLUNDELL JE.** The use of visual analogue scales to assess motivation to eat in human subjects: a review of their reliability and validity with an evaluation of new hand-held computerized systems for temporal tracking of appetite ratings. *British Journal of Nutrition*, 2000; 84: 405-415.
- TANNER RJ, FERRARO R, CHARTRAND TL, BETTMAN JR, VAN BAAREN R.** Of Chameleons and Consumption: The Impact of Mimicry on Choice and Preferences. *Journal of Consumer Research*, 2008; 34 (6): 754-766.
- TESLOVICH T, FRIEDL EK, KOSTRO K, WEIGEL J, DAVIDOW JY, RIDDLE MC, ROSENBAUM M, WALSH BT, CASEY BJ, MAYERL L.** Probing behavioral responses to food: Development of a food-specific go/no-go task. *Psychiatry research*, 2014; 219 (1): 166-170.
- VAGO DR, WALLENSTEIN GV, MORRIS LS.** Hippocampus. *Encyclopedia of the Neurological Sciences (Second Edition)*, 2014; 566-570.
- VAN OSSELAER SMJ, JANISEWSKI C.** A Goal-Based Model of Product Evaluation and Choice. *Journal of Consumer Research*, 2012; 39 (2): 260-292.

- VARGA M, THEGE BK, DUKAY-SZABÓ S, TÚRY F, VAN FURTH EF.** When eating healthy is not healthy: orthorexia nervosa and its measurement with the ORTO-15 in Hungary. *BMC Psychiatry*, 2014; 14: 59.
- VELTKAMP M, AARTS H, CUSTERS R.** Unravelling the motivational yarn: A framework for understanding the instigation of implicitly motivated behaviour resulting from deprivation and positive affect. *European Review of Social Psychology*, 2009; 20 (1): 345-381.
- VOLKOW ND, WANG GJ, BALER RD.** Reward, dopamine and the control of food intake: implications for obesity. *Trends in Cognitive Sciences*, 2011; 15 (1): 37-46.
- WANG J, LEIBOWITZ KL.** Central insulin inhibits hypothalamic galanin and neuropeptide Y gene expression and peptide release in intact rats. *Brain Research*, 1997; 777: 231-236.
- WATSON P, WIERS RW, HOMMEL B, DE WIT S.** Working for food you don't desire. Cues interfere with goal-directed food-seeking. *Appetite*, 2014; 79: 139-148.
- WEI LJ, LACHIN JM.** Properties of the Urn Randomization in Clinical Trials. *Controlled Clinical Trials*, 1988; 9: 345-364.
- WEISSMANN A, BINAH O.** The fractal nature of blood glucose fluctuations. *Journal of Diabetes and Its Complications*, 2014; In Press.
- WERTHMANN J, RENNER F, ROEFS A, HUIBERS MJH, PLUMANN L, KROTT N, JANSEN A.** Looking at food in sad mood: Do attention biases lead emotional eaters into overeating after a negative mood induction? *Eating Behaviors*, 2014; 15: 230-236.
- WESTENHÖFER J, ENGEL D, HOLST C, LORENZ J, PEACOCK M, STUBBS J, WHYBROW S, RAATS M.** Cognitive and weight-related correlates of flexible and rigid restrained eating behaviour. *Eating Behaviors*, 2013; 14: 69-72.
- WHO.** Obesity: preventing and managing the global epidemic. Report of a WHO Consultation. WHO Technical Report Series 894. Geneva 2000.
[Internet: www.who.int (accessed on 26.05.2014)]

WREN AM, SEAL LJ, COHEN MA, BRYNES AE, FROST GS, MURPHY KG, DHILLO WS, GHATEI MA, BLOOM SR. Ghrelin Enhances Appetite and Increases Food Intake in Humans. The Journal of Clinical Endocrinology&Metabolism, 2001; 86 (12): 5992-5995.

YOUNG AA. Brainstem sensing of meal-related signals in energy homeostasis. Neuropharmacology, 2012; 63:31-45.

ZHENG H, PATTERSON LM, PHIFER CB, BERTHOUD HR. Brain stem melanocortinergeric modulation of meal size and identification of hypothalamic POMC projections. American Journal of Physiology – Regulatory, Integrative and Comparative Physiology, 2005; 289: R247-R258.

<http://www.foodspotting.com/304024-reinhard-reschny> (accessed on 21.05.2014)

„Ich habe mich bemüht, sämtliche Inhaber der Bildrechte ausfindig zu machen und ihre Zustimmung zur Verwendung der Bilder in dieser Arbeit eingeholt. Sollte dennoch eine Urheberrechtsverletzung bekannt werden, ersuche ich um Meldung bei mir.“

6.2 Attachments

6.2.1 Ethical approval

Beschluss der Ethikkommission Decision of the Ethics Committee	 universität wien Ethikkommission
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Antragsteller/Applicant: **Univ.-Prof. Dr. Jürgen König**
Bearbeitungsnummer/Reference Number: **00064**
Projekttitel/Title of Project: **Der Einfluss visueller Primingmethoden auf das Ernährungsverhalten – neue experimentelle Ansätze**

Die Stellungnahme der Ethikkommission erfolgt aufgrund folgender eingereichter Unterlagen/
The decision of the Ethics Committee is based on the following documents:

- Durchführungsplanung_Geisler
- Einwilligungserklärung
- Antragsformular_ek_Geisler
- Projektantrag_Geisler
- Mail_Projektbeginn_korr._Missbach
- Antragsformular_Ek_Geisler_neu
- Aushang
- Einwilligungserklärung_EK_neu

Die Kommission fasst folgenden Beschluss (mit X markiert)/The Ethics Committee has made the following decision (marked with an X):

☒ Zustimmung: Es besteht kein ethischer Einwand gegen die Durchführung der Studien/
Consent: there is no ethical objection to any accomplishments of the project

☐ Negative Beurteilung: Der Antrag wird von der Ethikkommission abgelehnt /negative
evaluation: the proposal is refused by the Ethics Committee

Unterschrift/Signature

Datum/Date

Ilse Kryspin-Exner

07.04.2014

Vorsitzende der Ethikkommission/Chairwoman of the Ethics Committee
emer. o. Univ.-Prof. Dr. Ilse Kryspin-Exner

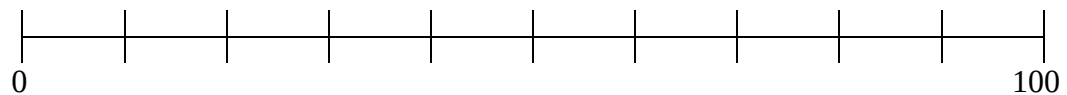
6.2.2 Questionnaires

- Visual Analogue Scale**

Wie hungrig sind Sie?

0 = nicht hungrig

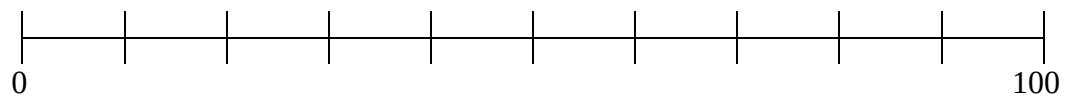
100 = sehr hungrig



Wie satt sind Sie?

0 = nicht satt

100 = sehr satt



- Mood questionnaire**

Bitte bestimme bei den angegebenen Aussagen deine persönliche Stimmungslage durch Markierung an geeigneter Stelle.

zornig	Sehr stark	7	6	5	4	3	2	1	Überhaupt nicht
abgeschlafte	Sehr stark	7	6	5	4	3	2	1	Überhaupt nicht
unglücklich	Sehr stark	7	6	5	4	3	2	1	Überhaupt nicht
traurig	Sehr stark	7	6	5	4	3	2	1	Überhaupt nicht
angenehm	Sehr stark	7	6	5	4	3	2	1	Überhaupt nicht
betrübt	Sehr stark	7	6	5	4	3	2	1	Überhaupt nicht
freudig	Sehr stark	7	6	5	4	3	2	1	Überhaupt nicht
hoffnungslos	Sehr stark	7	6	5	4	3	2	1	Überhaupt nicht

müde	Sehr stark	7	6	5	4	3	2	1	Überhaupt nicht
verärgert	Sehr stark	7	6	5	4	3	2	1	Überhaupt nicht
frohgemut	Sehr stark	7	6	5	4	3	2	1	Überhaupt nicht
entmutigt	Sehr stark	7	6	5	4	3	2	1	Überhaupt nicht
fröhlich	Sehr stark	7	6	5	4	3	2	1	Überhaupt nicht
erschöpft	Sehr stark	7	6	5	4	3	2	1	Überhaupt nicht
heiter	Sehr stark	7	6	5	4	3	2	1	Überhaupt nicht
verzweifelt	Sehr stark	7	6	5	4	3	2	1	Überhaupt nicht
wütend	Sehr stark	7	6	5	4	3	2	1	Überhaupt nicht
entkräftet	Sehr stark	7	6	5	4	3	2	1	Überhaupt nicht
lustig	Sehr stark	7	6	5	4	3	2	1	Überhaupt nicht

- ***Flexible and rigid control of eating behaviour***

Wie oft hast du in deinem Leben bereits Schlankheitsdiäten gemacht?

- ☐ noch nie
- ☐ 1-3 mal
- ☐ 4-8 mal
- ☐ 9-15 mal
- ☐ mehr als 15 mal
- ☐ in regelmäßigen Abständen
- ☐ ich halte so gut wie immer Diät

Im Folgenden siehst du verschiedene Aussagen, bitte gib an wie sehr du den einzelnen Feststellungen zustimmst. Folge dabei bitte deinem ersten Impuls.

1=trifft voll zu

2=trifft überwiegend zu

3=trifft überwiegend nicht zu

4=trifft gar nicht zu

	1	2	3	4
Bei den üblichen Nahrungsmitteln kenne ich ungefähr den Kaloriengehalt.	0	0	0	0
Ich zähle Kalorien, um mein Gewicht unter Kontrolle zu halten.	0	0	0	0
Wenn Sie zu viel gegessen haben, bringen Sie Gewissensbisse dazu, sich eher zurückzuhalten?	0	0	0	0

Achten Sie darauf, dass Sie keinen Vorrat an verlockenden Lebensmitteln haben?	0	0	0	0
Kaufen Sie häufig kalorienarme Lebensmittel?	0	0	0	0
Würden Sie Ihre Lebensweise ändern, wenn Sie eine Gewichtsveränderung von 5 Kilogramm feststellten?	0	0	0	0
Zum Abnehmen wäre mir eine Diät zu langweilig.	0	0	0	0
Ich lasse lieber eine Mahlzeit ausfallen, als nach der Hälfte aufzuhören.	0	0	0	0
Bei mir wechseln sich Phasen, in denen ich streng Diät halte, mit Zeiten ab, bei denen ich esse, was und wie ich will.	0	0	0	0
Manchmal lasse ich Mahlzeiten ausfallen, um nicht zuzunehmen.	0	0	0	0
Es gibt Lebensmittel, die ich grundsätzlich nicht esse, obwohl ich sie mag.	0	0	0	0
Beim Abnehmen versuche ich mich möglichst an einen Plan zu halten.	0	0	0	0
Ich esse Lebensmittel, die schlank machen, auch wenn sie mir nicht besonders schmecken.	0	0	0	0
Ohne Diätplan weiß ich gar nicht, wie ich mein Gewicht in den Griff bekommen kann.	0	0	0	0
Bei einer Diät zählt für mich der schnelle Erfolg.	0	0	0	0

- ***Barratt Impulsiveness Scale – short form***

Bitte folge auch hier deinem ersten Impuls. Als Antwortmöglichkeit steht zur Wahl:

1=selten/nie

2=gelegentlich

3=oft

4=fast immer/immer

	1	2	3	4
Ich plane meine Vorhaben gründlich.	0	0	0	0
Ich mache häufig Dinge ohne vorher darüber nachzudenken.	0	0	0	0
Ich bin unaufmerksam.	0	0	0	0
Ich kann mich gut konzentrieren.	0	0	0	0
Ich sichere mich im Leben in allen Dingen ab.	0	0	0	0
Ich rutsche bei Spielen oder Vorträgen oft hin und her.	0	0	0	0
Ich denke gründlich nach.	0	0	0	0

Ich plane für meine berufliche Sicherheit.	0	0	0	0
Ich sage Dinge ohne darüber nachzudenken.	0	0	0	0
Ich handele spontan.	0	0	0	0
Mir wird beim Lösen von Denkaufgaben schnell langweilig.	0	0	0	0
Ich handele gerne aus dem Moment heraus.	0	0	0	0
Ich kaufe Sachen ganz spontan.	0	0	0	0
Ich werde bei Vorlesungen oder Vorträgen schnell unruhig.	0	0	0	0
Ich plane für die Zukunft.	0	0	0	0

- ***Perceived Self-Regulatory Success in Dieting Scale***

Bitte markiere nun folgende Aussagen an jener Stelle, die für dich am zutreffendsten ist:

Wie gut gelingt es Ihnen auf Ihr Gewicht zu achten?	überhaupt nicht gut	1	2	3	4	5	6	7	sehr gut
Wie gut gelingt es Ihnen abzunehmen?	überhaupt nicht gut	1	2	3	4	5	6	7	sehr gut
Wie schwierig finden Sie es in Form zu bleiben?	überhaupt nicht schwierig	1	2	3	4	5	6	7	sehr schwierig

- ***Food Cravings Questionnaire – Trait – reduced version***

Bei den folgenden Aussagen folge bitte wieder deinem ersten Impuls. Es gilt:

1=nie/nicht anwendbar
2=selten
3=manchmal

4=oft
5=fast immer
6=immer

	1	2	3	4	5	6
Wenn ich ein starkes Verlangen nach etwas verspüre, weiß ich, dass ich nicht mehr aufhören kann zu essen, wenn ich erst mal angefangen habe.	0	0	0	0	0	0
Wenn ich das esse, wonach ich ein starkes Verlangen verspüre, verliere ich oft die Kontrolle und esse zu viel.	0	0	0	0	0	0
Wenn ich ein starkes Verlangen nach bestimmten Nahrungsmitteln verspüre, denke ich ausnahmslos darüber nach, wie ich das bekomme, was ich essen will.	0	0	0	0	0	0

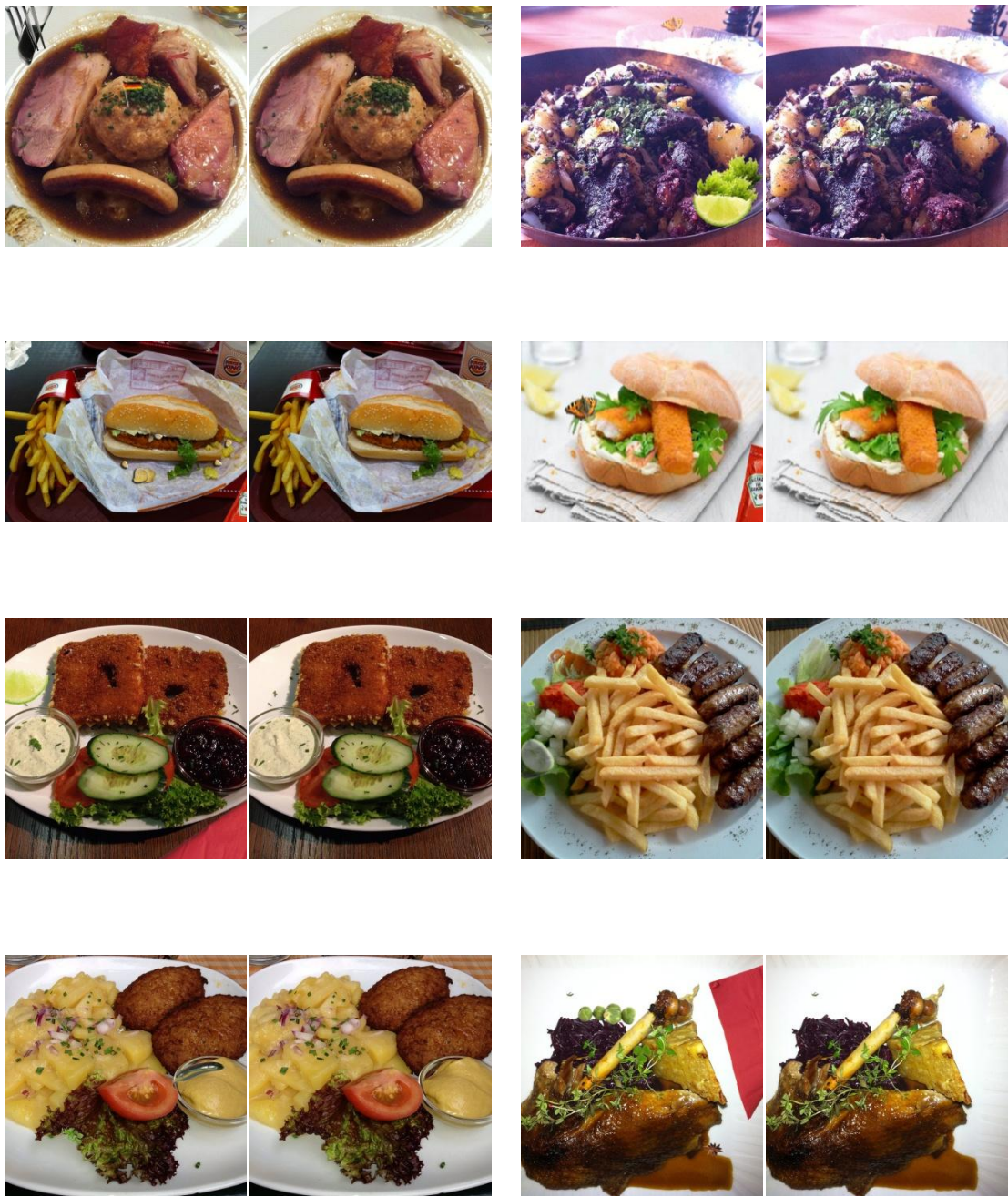
Ich habe das Gefühl, dass ich die ganze Zeit nur Essen im Kopf habe.	0	0	0	0	0	0
Ich ertappe mich dabei, wie ich mich gedanklich ständig mit Essen beschäftige.	0	0	0	0	0	0
Immer wenn ich ein starkes Verlangen nach bestimmten Nahrungsmitteln verspüre, merke ich, dass ich gleich plane etwas zu essen.	0	0	0	0	0	0
Ich verspüre ein starkes Verlangen nach bestimmten Nahrungsmitteln, wenn ich mich gelangweilt, wütend oder traurig fühle.	0	0	0	0	0	0
Ich habe nicht die Willensstärke, um meinen Essensgelüsten widerstehen zu können.	0	0	0	0	0	0
Wenn ich einmal anfangen zu essen, fällt es mir schwer wieder aufzuhören.	0	0	0	0	0	0
Ich kann nicht aufhören übers Essen nachzudenken, wie sehr ich mich auch bemühe.	0	0	0	0	0	0
Wenn ich dem starken Verlangen nach bestimmten Nahrungsmitteln nachgebe, verliere ich jegliche Kontrolle.	0	0	0	0	0	0
Immer wenn ich ein starkes Verlangen nach bestimmten Nahrungsmitteln verspüre, denke ich so lange weiter ans Essen bis ich diese tatsächlich esse.	0	0	0	0	0	0
Wenn ich ein starkes Verlangen nach bestimmten Nahrungsmitteln verspüre, verzehren mich die Gedanken daran diese zu essen geradezu.	0	0	0	0	0	0
Meine Emotionen bringen mich oft dazu etwas essen zu wollen.	0	0	0	0	0	0
Wenn sich appetitliche Nahrungsmittel in meiner Reichweite befinden, fällt es mir schwer der Versuchung zu widerstehen sie zu essen.	0	0	0	0	0	0

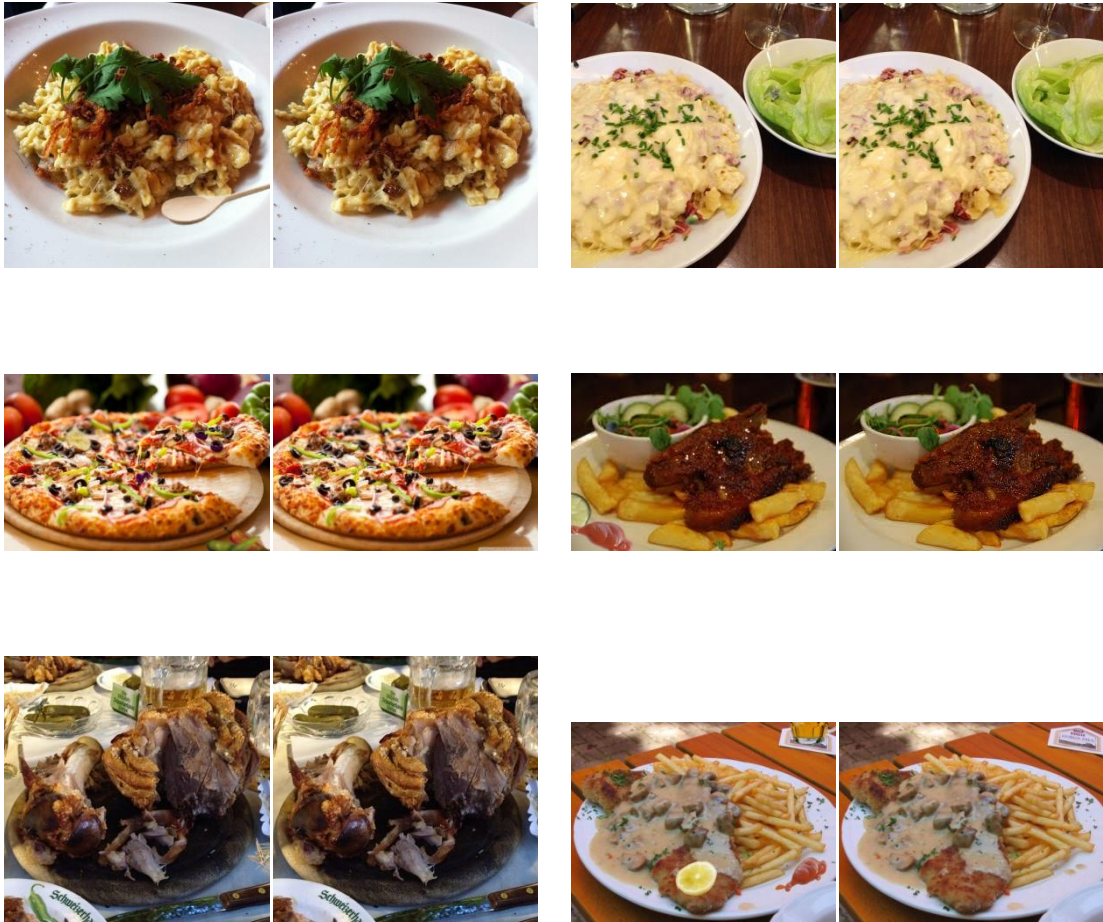
- **Bratman's Orthorexia Test**

	Ja	Nein
Denken Sie mehr als 3 Stunden am Tag über Ihre Ernährung nach?	0	0
Planen Sie Ihre Mahlzeiten mehrere Tage im Voraus?	0	0
Ist Ihnen der ernährungsphysiologische Wert Ihrer Mahlzeit wichtiger als die Freude an deren Verzehr?	0	0
Hat die Steigerung der angenommenen Lebensmittelqualität zu einer Minderung Ihrer Lebensqualität geführt?	0	0
Sind Sie in letzter Zeit mit sich strenger geworden?	0	0
Steigert sich Ihr Selbstwertgefühl durch gesunde Ernährung?	0	0
Verzichten Sie auf Lebensmittel, die Sie früher gerne gegessen haben, um nun „richtige“ Lebensmittel zu essen?	0	0
Haben Sie durch Ihre Essgewohnheiten Probleme auszugehen und distanzieren Sie sich dadurch von Freunden und Familie?	0	0
Fühlen Sie sich schuldig, wenn Sie von Ihrer Diät abweichen?	0	0
Fühlen Sie sich glücklich und unter Kontrolle, wenn Sie sich gesund ernähren?	0	0

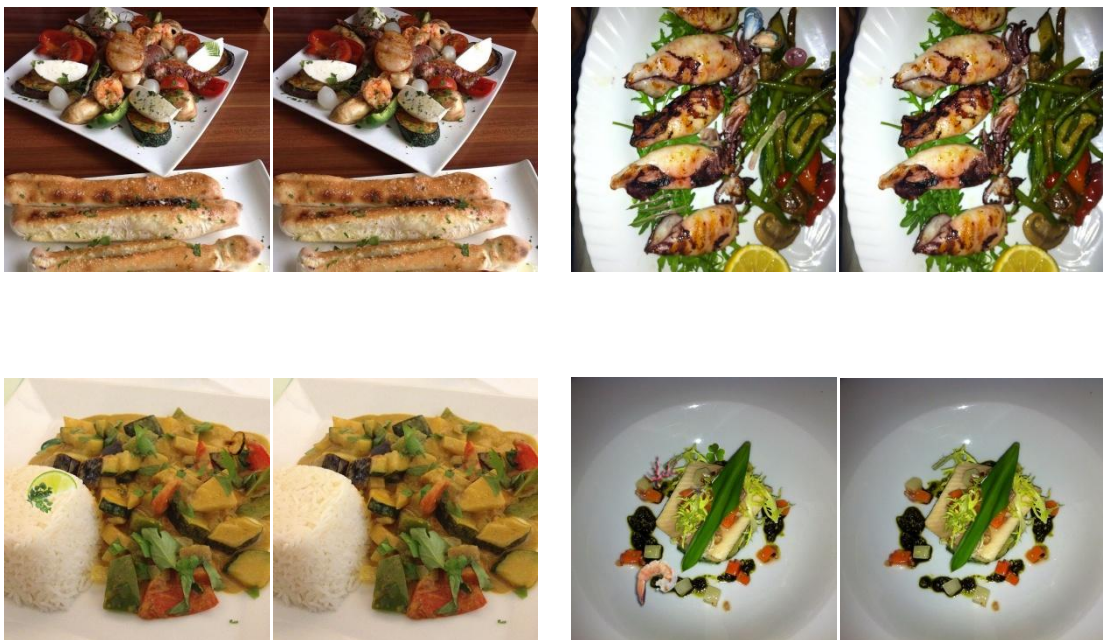
6.2.3 Priming intervention

6.2.3.1 High caloric food pictures





6.2.3.2 Low caloric food pictures





6.2.4 Curriculum Vitae

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Ausbildung

WS 12/13 – SS 14 **Masterstudium Ernährungswissenschaften (Universität Wien)**
Studienzweig Lebensmittelqualität und -sicherheit

WS 07/08– SS 12 **Bakkalaureatsstudium Ernährungswissenschaften (Universität Wien)**
Titel der Bakkalaureatsarbeit: Ist der kritisch Kranke mangelernährt?
Möglichkeiten der Ernährung von Intensivpatienten

10/2006 – 10/2007 **Spezielle Zusatzausbildung in der Intensivpflege**
Wiener Krankenanstaltenverbund, Akademie für Fortbildungen und
Sonderausbildungen – Bereich Pflege, 1090 Wien

09/1999 – 09/2002 **Ausbildung im gehobenen Dienst für Gesundheits- und Krankenpflege**
Schule für allgemeine Gesundheits- und Krankenpflege am
Rudolfinerhaus, 1190 Wien

09/1991 – 06/1999 **Gymnasium und wirtschaftskundliches Realgymnasium**
Schulen der Dominikanerinnen, 1130 Wien
Schulabschluss Matura

Kenntnisse und Qualifikationen

Sprachen Deutsch als Muttersprache
Englisch fließend in Wort und Schrift
Spanisch Grundkenntnisse

Programme gute MS Office und SPSS Kenntnisse
Adobe Photoshop, Adobe Illustrator: Grundkenntnisse