

# MASTERARBEIT

The influence of cognitive processes on eating behaviour -  
illustrated by the example of mental imagery

Verfasser

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## Glossary

ABBREVIATIONS	
A –MSH	$\alpha$ - melanocyte stimulating hormones
ANS	autonomic nervous system
AP	area postrema
AGRP	agouti – related Protein
CART	cocaine – and amphetamine-regulated transcripts
CNS	central nervous system
DV	dependent variable
fMRI	functional magnetic resonance imaging
GIT	gastrointestinal tract
HF	high-fat
HS	high-sugar
IV	independent variable
IQR	interquartile range
LGN	lateral geniculate nucleus
min	minutes
mm	millimeter
NAc	nucleus accumbens
NPY	neuropeptide Y
NTS	nucleus tractus solitarii
OB-R	leptin receptor
PET	positron emission tomography
POMC	pro-opiomelanocortin
sd	standard deviation
VAS	Visual Analogue Scale
DEFINITIONS	
Appetite	the subjective desire to eat food [EDWARD and REXFORD, 2011]
Hedonic control of appetite	mediation of regulatory reward systems which are activated by highly palatable foods[HARROLD et al., 2012]
Homeostatic control of appetite	mediation of biological needs to maintain the body's energy stores[HARROLD et al., 2012]
Hunger	The motivation to seek and consume food. It is often the initiator of a feeding event [HARROLD et al., 2012]
Satiation	the processes that bring episodes of eating behaviour to an end [HARROLD et al., 2012]
Satiety	The process that suppresses the internal drive to eat (appetite) [STAFLEU <i>et al.</i> , 2011]
Sensory-specific satiety	The change in pleasantness to specific foods [ROLLS et al., 1981]

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## 1 Foreword

The brain plays a central role in integrating signals and orchestrating metabolic systems throughout the biological mechanisms of humans. The interest in brain research gained importance in science over the last 20 years, since imaging techniques made it possible to observe the brain's activity while people perform different tasks. This and other new methods opened the gate for different approaches among several disciplines (mostly suffixed by the term “neuro”), and gained popularity for analysing human behaviour patterns.

From a nutritional perspective, the need for more profound explanation models seems to be redundant in the context of rising obesity rates on a national and worldwide scale over the last decades with its burdensome consequences for individuals, health care systems and future generations. No sufficient explanation models for obesity have been provided to this day. Current findings in brain research have already changed the perception of the disease and will probably provide new models.

Coming from a discipline consisting of a vast variety of different fields in itself, nutritional sciences is determined to integrate findings from other disciplines into current models for describing eating behaviour. To tackle such complex topics and provide the best approximations for the problem, “single-discipline approaches” will fall short in comparison to their multi-disciplinary counterparts, and therefore the necessity for merging disciplinary boundaries is indicated. New findings often emerge when disciplines cooperate and focal points are formed.

My driving force to write about *the influence of cognitive top-down processes on eating behaviour - illustrated by the example of mental imagery*, is rooted in the belief that our behaviour is a result of a much more complex interplay between person, food and situation, than what textbooks taught me in my studies. The core questions like what/where/when/with whom and why we eat were asked under the assumption that humans act rationally anywhere, anytime. I'm convinced this is not the case and that our behaviour emerges from much more complex processing without conscious control than we think. The failure of major public health interventions which have been stressing



optimized eating behaviour by means of nutritional guidelines and public health campaigns, may prove my point by now.

Consequently, these questions have to be put into a different context to provide new ways of thinking about food consumption and eating behaviour in a more sophisticated and individual way.

Finally, I want to thank everybody who supported me during my studies - on and off the study desk.

## 2 Introduction and leading question

The construction of the world through our nervous system underlies complex mechanisms with the challenge of processing and integrating external stimuli leading to behavioural output. Therefore, actual perception via our visual system, plus the cognitive task of identifying and distinguishing between information, lays down the basic construction of a highly differentiated system. The factual knowledge being constructed can be of different motor/sensory origin (audible, gustatory, olfactory, tactile or visual), and therefore processed through neural pathways to the brain tissue. Information is projected through hard-wired connections and, as plasticity theory provides, “ever-changing” tissue forms the basis for neural substrates. Wiring mechanism is based on Hebb’s postulation described as “what fires together, wires together”, which is fundamental for contemporary neuroscientific theories [HEBB, 1949].

However, visual processing is in an exceptional position because its intrinsic characteristics in thought processing and symbol manipulation have evolved during human evolution. About half of the cerebral cortex capacity is involved in analysing the visual world [BEAR *et al.*, 2007c].

The process that allows the brain to work as usual in the absence of external stimuli which makes it possible for humans to recreate situations from autobiographical memory; smells, sounds and visualizing the future, is called: *mental imagery* [KOSSLYN *et al.*, 2001].

The evolutionary purpose of scenario visualization is crucial for creative processes and the prediction of future events. These attributes are pivotal in the human problem solving machinery [SCHACHTER and ADDIS, 2007 ; ARP, 2008].

Hence, the brain has the necessary task to distinguish between actual perceptual images (quasi perceptual imagery experiences) and recalled images (imagery representation). Basic research in the last decades about mental imagery in disciplines like cognitive science, psychology and sports sciences, investigated the question to what extent visual processing and mental imagery share the same neural mechanisms and cortical

pathways, how they can be distinguished and how mental imagery can be used as to modulate internal processes [BLOOM *et al.*, 2001 ; PYLYSHYN, 2002].

Over years of research, data accumulated in mental imagery research in diverse disciplines involving philosophical studies (epistemological basis), neurobiological science (biological basis), applied science approaches in sports sciences, and medicine. Those will be described in detail later in the text.

The performance or subjective feeling of mental imagery is also called “imagining”, “visualizing”, or “seeing with the mind’s eye”. The common utilisation of these expressions in our everyday language reflects the importance of mental imagery. People use mental images for different occasions and reasons, with varying frequencies and experience mental images with different degrees of vividness [MARKS, 1973].

Neurological pathologies can lead to impaired mental imagery, as demonstrated by the classical example of patient M.X at the end of the 19<sup>th</sup> century. M.X was a well-educated polyglot using mental images as mnemonics. One day he found he had totally lost his former colourful and vivid images, unable to retrieve them from memory. Neither drawing from memory, nor usage of these images was possible for him anymore, which led the authors to the conclusion that memory is a multimodal faculty consisting of different modality specific components. In the case of M.X, who once could recite the whole first book of *Homer’s Iliad* in ancient Greek, could not even recognize most of the ancient Greek letters anymore [CHARCOT and BERNARD, 1883 ; BARTOLOMEO, 2008].

Mental images can be induced deliberately (e.g. just thinking about a scene and inducing a picture at will), or without cognitive control (e.g. famously illustrated by the example of *Marcel Proust’s “Madeleine passage”* described in the novel “In search of lost time”). The protagonist evoked immediate mental images of his childhood by describing the scenes vividly, clearly and very detailed, after he had tasted Madeleine cookies with a cup of tea, which was the trigger for his mental time-travel experience [PROUST, 1994]).

In the last decades, nutritional sciences acquired new “food for thought” with the help of scientific approaches in sensory, psychology and consumer science describing human

eating behaviour. The question for the main determinants concerning human food intake in a westernized society embedded in an “obesogenic” environment is therefore pivotal [SWINBURN *et al.*, 1999].

How much, how, and when we start and stop eating is still insufficiently explained and research findings presented later in the text provide evidence that models, respectively homeostatic and non-homeostatic eating models, are highly interwoven. Therefore, the assumption that top-down determinants of food intake such as reward, beliefs, attitudes and cognitive mechanisms have enough explanatory power to contribute to the explanation of human eating behaviour.

To investigate one very powerful cognitive tool such as mental imagery and its influence on eating behaviour, an experimentally camouflaged design was created analogously to the preceding publication of *Morewedge et al.* Certain aspects were modified (methods, intervention), hence the presented experimental part of the thesis is more than a replication experiment [MOREWEDGE *et al.*, 2010].

Participants were recruited for a sensory taste-test with gummy bears and divided into four different intervention groups. All groups were given different mental imagery tasks prior to the taste-test, with the difference that two intervention groups imagined eating gummy bears (18 and 36 repetitions), while two control groups imagined putting a 50¢ coin into a laundry machine (18 and 36 repetitions). Additionally, four questionnaires were filled out by the participants, and the amount of gummy bears consumed during the test were weighed by the experimenters (unknownst to the participants) to assess if different interventions influence food consumption during the taste-test.

The rationale reads as follows: performed mental images can lead to physiological responses of a different kind by evoking similar neural substrates as if the event is actually experienced [BARSALOU, 2008].

For example fear responses by people with arachnophobia can be induced when thinking about spiders.

Analogously, thinking about food or food consumption also leads to physiological responses. The question is, what effects does it have on eating behaviour? Thinking

about food or food consumption may intuitively be associated to an increase of appetite. Salivation and stomach rumblings may clearly indicate this answer. However, taking a closer, more differentiated look at the phenomenon, “specificity” may play a crucial role as shown by *Morewedge et al.* Imagining the eating process of any food with subsequent consumption of any other food is termed *unspecific*, while visualizing the eating process of a food with subsequent consumption of the envisioned food is described as *specific* - analogously to sensory-specific-satiety theory provided by *Rolls and colleagues* [ROLLS *et al.*, 1981].

Additional to the formulated main hypotheses, the results postulated by *Morewedge et al.*, 2010 will be augmented by measuring subjective parameters of appetite and individual differences of mental imagery vividness; usage of mental imagery and restrained eating scales will be phrased in sub-hypotheses presented at the end of *chapter 4*.

## **Hypothesis I**

*Does mental imagery influence subsequent gummy bear intake?*

---

H0 = there is no difference in gummy bear consumption between participants who imagined eating gummy bears compared to participants in the control group (motor control task)

H1 = there is a difference in gummy bear consumption between participants who imagined eating gummy bears compared to participants in the control group (motor control task)

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**Hypothesis II**

*Does the implementation of 18 repetitive mental imagery tasks influence subsequent gummy bear intake?*

---

H0 = there is no difference in gummy bear consumption between participants who imagined eating 18 gummy bears compared to participants in the matching control group (motor control task).

H1 = there is a difference in gummy bear consumption between participants who imagined eating 18 gummy bears compared to participants in the matching control group (motor control task).

---

**Hypothesis III**

*Does the implementation of 36 repetitive mental imagery tasks have an influence on subsequent gummy bear intake?*

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H0 = there is no difference in gummy bear consumption between participants who imagined eating 36 gummy bears compared to participants in the matching control group (motor control task).

H1 = there is a difference in gummy bear consumption between participants who imagined eating 36 gummy bears compared to participants in the matching control group (motor control task).

---

### 3 Theoretical background

#### 3.1 Modelling food intake regulation

*“Although humans have the ability to make conscious, voluntary decisions and choices, most of our actions have a subconscious component that escapes voluntary control” [ZHENG and BERTHOUD, 2008].*

The complex mechanism of human eating behaviour depends on a vast variety of different factors which will be interrogated in this chapter. The high degree of complexity demands the combination of animal and human behavioural, neuroimaging and psychological studies to form an integrated model for eating behaviour. Stressing appetite and food intake regulation is important, considering the fact that the obesity epidemic is on the rise worldwide, along with various secondary diseases [KOPELMAN, 2000 ; CUMMINGS and SCHWARTZ, 2003 ; SWINBURN *et al.*, 2011].

The main cause for the obesity epidemic may undoubtful be the imbalance between energy intake and energy expenditure in individuals. Hence, throughout human evolution, only weak physiological mechanisms seem to have evolved to defend humans against excessive energy intake and body weight gain, being a product of the interaction between our ancient eating systems that evolved some thousand years ago, having to deal with an environment that switched from scarce to exuberant. Research that merely investigates the main parameters for homeostatic control of food intake falls short in the context of an “obesogenic” environment because this is where other determinants come to play. In fact, nonhomeostatic and hedonic aspects are of increased importance to determine behavioural traits [SWINBURN *et al.*, 1999 ; BERTHOUD, 2011].

Therefore, an artificial differentiation between the homeostatic and nonhomeostatic control of food intake may be crucial in the context of current research, whereby interaction between the two assumed systems might be more intimately interwoven than

previously thought, with the overarching purpose to maintain an optimal internal milieu in harmony with the external world [SHIN *et al.*, 2009].

Obese patients eat in the absence of metabolic need. Possibly, nonhomeostatic mechanisms may alter food intake patterns and in a long run throw the well-tuned system of body weight off-balance [BERTHOUD, 2011].

To detangle current theories of food intake regulation, this chapter provides an overview of current state of the art research. Therefore, in the first part, important brain structures, which are involved in food intake regulation, will be presented. This is followed by research of the homeostatic and nonhomeostatic regulatory systems, and concludes with crosstalk between these two systems to form an integrated approach.

### **3.1.1 Important brain structures involved in food intake**

#### ***3.1.1.1 Caudal brainstem – the solitary tract and area postrema***

The brain stem consists of the diencephalon (thalamus and hypothalamus), the midbrain (tectum and tegmentum), the pons and the caudal (posterior) part, and the medulla oblongata. The NTS is located along the length of the medulla oblongata. The AP is a circumventricular organ located in the medulla oblongata, deeply connected to the NTS and central for the “vomiting” mechanism. Both structures are involved in mediating autonomic control of behaviour [MILLER and LESLIE, 1994 ; HORN, 2008].

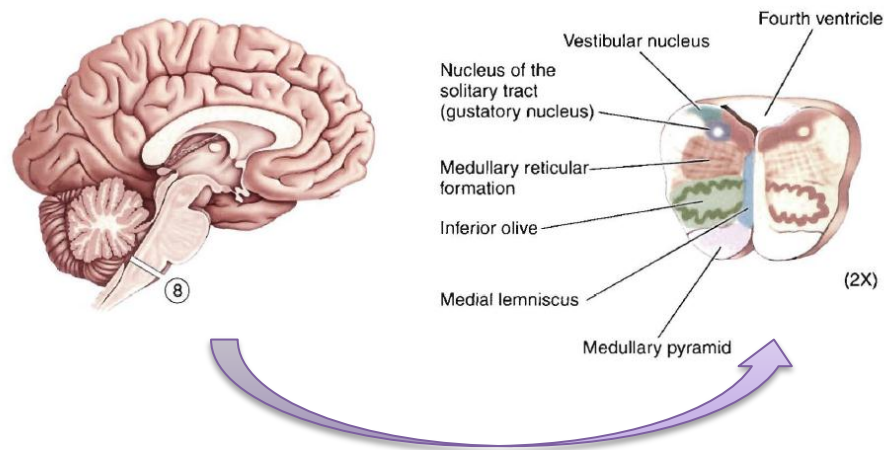
Gut signals (e.g. gastric distension and portal vein glucose levels) are sent via vagal afferents to NTS/AP’s nucleus of the medulla in the brain stem. Additionally, afferent gustatory information from the oral cavity is also projected to the gustatory nucleus, which is part of the NTS. From the NTS/AP, information is projected and received to and from interconnected structures, such as the hypothalamus, amygdala and the cerebral cortex [SCHWARTZ, 2006].

In fact, rodent experiments showed, when disconnecting the brain stem from all other brain structures, basic appetitive (such as ingestion) and aversive reactions (such as facial aversion reactions) can be observed, thus indicating that this region plays a



primordial role for appetitive regulation regulated via the ANS [GRILL and NORGREN, 1978].

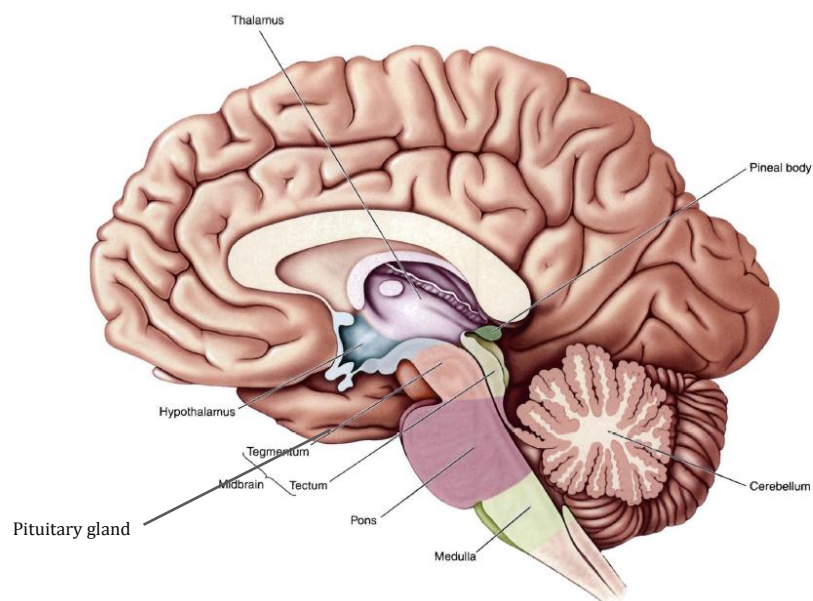
*Figure 1: Cross- sectional view of the mid-medulla [BEAR et al., 2007e]*



### 3.1.1.2 Hypothalamus

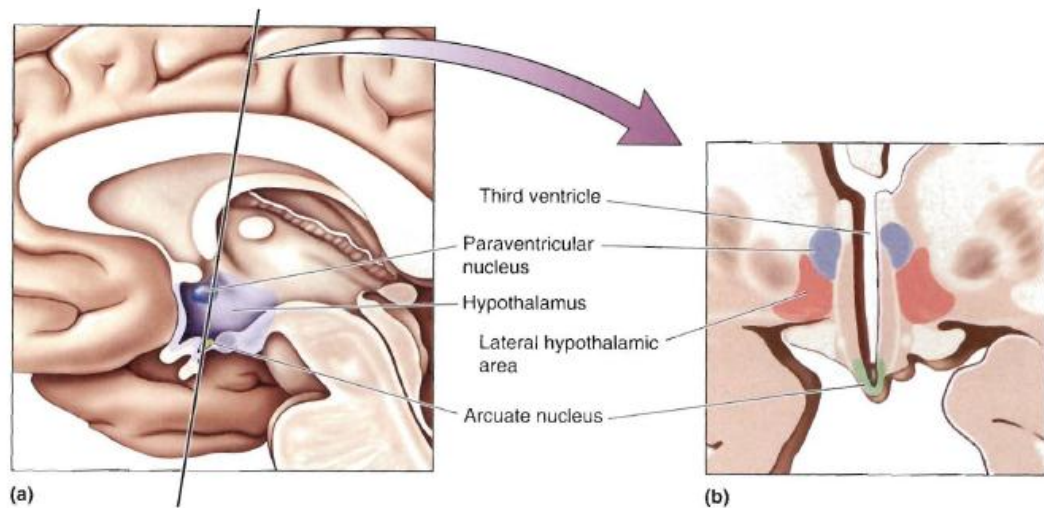
The main regulator which integrates, computes and responds to most of internal and external signals, is the hypothalamus projecting to and receiving information from the NTS/AP in the brain stem and to other sites of the brain. It is part of the limbic system (emotion processing) and localized medial to the thalamus in the brain as seen in *figure 2*.

*Figure 2: Medial surface of the cerebrum (midsagittal cut) [BEAR et al., 2007e]*



The hypothalamus is associated with the function of controlling the ANS (cf. fight-or-flight reaction, heart rate and temperature regulation), and directs the pituitary gland to release hormones directly into the bloodstream. Each side of the hypothalamus has diverse functional zones influencing energy balance, behaviour, autonomic and endocrine outflow and therefore are key downstream structures. The major hub for integrating nutritionally relevant information is the arcuate nucleus (ARC) which is localized in the mediobasal hypothalamus adjacent to the third ventricle [BERTHOUD and MUENZBERG, 2011].

Figure 3: Midsagittal (a) and coronal (b) section of the brain showing three important nuclei in the control of eating behaviour [BEAR et al., 2007e]



The ARC is relevant for integrating peripheral humoral signals and triggering orexigenic pathways such as the NPY/AGRP and anorexigenic pathways like CART/POMC, central in the melanocortin system [CONE, 2005].

The paraventricular nucleus (PVN) and the lateral hypothalamic area (LHA) have extensive connections with the solitary tract of the brain stem involved in the integration of information from areas associated with reward, motivation, learning and memory. These are areas associated with vagal and visceral input, sensory motor coordination and arousal. The LHA receives information from areas associated with reward, motivation, learning and memory such as the orbitofrontal cortex, nucleus accumbens, hippocampus, amygdala and ventral tegmental area; from areas associated with sensory input (insular and olfactory cortex) and visceral sensory input from brainstem areas.

Thus the LHA has the capacity to influence a multitude of neural activity [SHIN *et al.*, 2009 ; BERTHOUD and MUENZBERG, 2011].

The PVN side is localized laterally to the optic tract to regulate the circadian rhythm receiving information from retinal ganglion cells to orchestrate the sympathetic and parasympathetic innervation and to regulate neuroendocrine excretion via the pituitary gland. Hormonal changes being regulated due to anticipated, clock-like rhythms play a crucial role in circadian hormonal secretion of cortisol, leptin and ghrelin [POWER and SCHULKIN, 2011].

In summary, the scope of hypothalamic responses can be characterized as follows:

- i. *Humoral response*: the stimulation and release of pituitary gland hormones.
- ii. *Visceromotor response*: the adjustment to balance parasympathetic and sympathetic output of the ANS (increasing/decreasing sympathetic or parasympathetic activity).
- iii. *Somatic motor response*: generating motor behaviour response which is associated with motivated behaviour (eating, drinking, seeking cold, shivering).

### **3.1.1.3 Forebrain structures**

Forebrain structures are associated with functions like perception, cognition, conscious awareness and voluntary action. Structures involved in reward circuitries, respectively mesolimbic and corticolimbic structures heavily involved in nonhomeostatic food intake, will be presented here.

#### *Ventral tegmental area (VTA) and nucleus accumbens (NAc)*

Both structures are part of the brain's limbic (emotion processing system) associated with reward. The NAc contains dopaminergic and opiodergic pathways which are important in reward circuitries. These structures have been related to food and drug reward systems and therefore share similar pathways [KENNY, 2011].

Extensive connections from the shell of the NAc to the LHA were found which shape the interface-like character between motivation, reward and actual feeding behaviour in rats [STRATFORD *et al.*, 1999 ; SAPER *et al.*, 2002 ; STRATFORD and WIRTSHAFTER, 2012].

The VTA plays a similar role in the reward center of the brain, especially in dopaminergic pathways. Tonic and episodic signalling (such as leptin and ghrelin) can influence firing rates of dopamin neurons in the VTA, leading to adapted feeding behaviour in rats. The administration of leptin decreased and ghrelin increased firing rates of these dopaminergic neurons [NALEID *et al.*, 2005 ; HOMMEL *et al.*, 2006].

Thus, NAc and VTA structures associated with reward circuitry may play an important role in bottom-up cross-talk between homeostatic and nonhomeostatic food intake modelling. Important to notice here is, that other non-dopaminergic pathways could also play an important role in the modulatory cross-talk [CAMERON and DOUCET, 2011].

### *Hippocampus and amygdala*

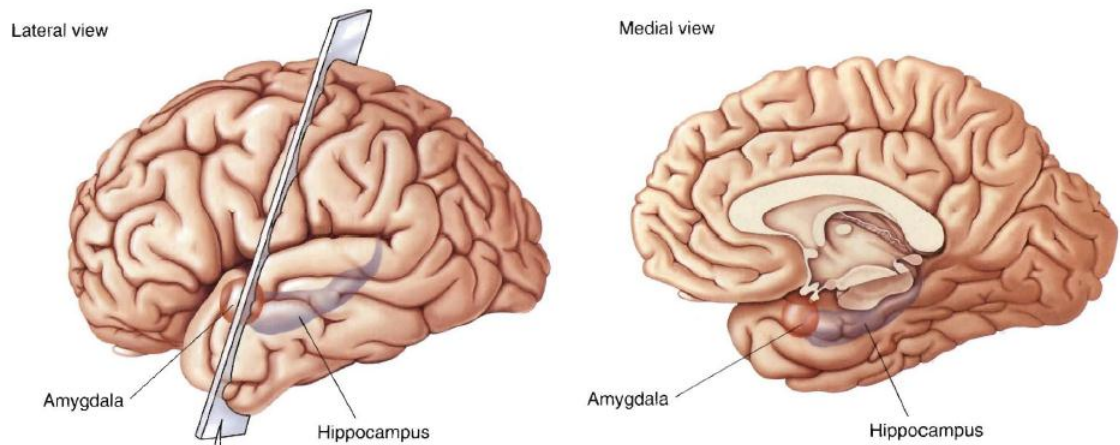
The hippocampus and amygdala are brain structures involved in learning and memory processing. The hippocampus is a cortical structure, located deep in the temporal lobe. Memory is retention of acquired information which is stored in neural substrates. Spatial memory is an essential component of our everyday life, helping us navigate through space [BEAR *et al.*, 2007d].

In hippocampal lesion studies, amnesic patients will consume a second meal immediately after a full meal was finished, indicating that memory for what has recently been eaten is a substantial contributor for eating behaviour [ROZIN *et al.*, 1998].

The amygdala is localized in the pole of the temporal lobe below the cerebral cortex on the medial side. It is divided into three nuclei (basolateral, corticomedial and central), with projections to and from the cerebral cortex and the hippocampus. Two major projections to the hypothalamus can be found: the ventral amygdalofugal pathway and the stria terminalis. The amygdala is central in sensory input integration and associated with learning, memory and emotion processing [BEAR *et al.*, 2007d].

It is involved in basic conditioning mechanisms, where communication between the basolateral hypothalamus and the amygdala is focal. Additionally, reward driven behaviour (opioidergic and dopaminergic) is located in the amygdala as well [PETROVICH *et al.*, 2005 ; BERRIDGE, 2007].

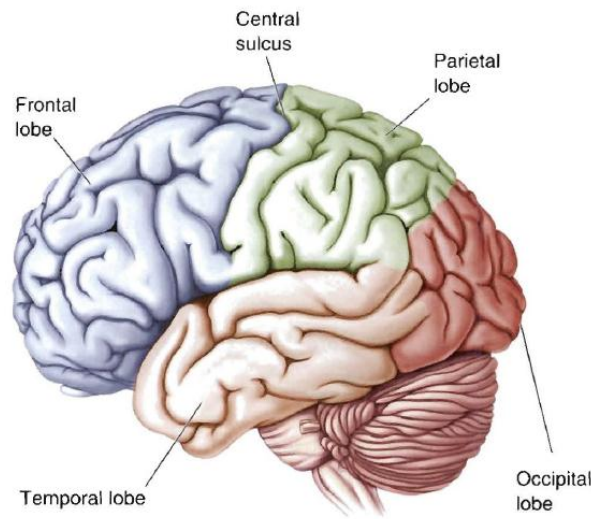
*Figure 4: Lateral and medial view of amygdala/hippocampus [BEAR *et al.*, 2007e]*



### *Cerebral cortex*

The cerebral cortex is the one structure that expanded the most during human evolution and thus plays an important role in the emergence of human behaviour. The cerebral cortex is the sheet of neurons found just below the surface of the cerebrum. The prefrontal cortex (pFC) is the anterior part of the frontal lobe of the brain. It is designated to play a pivotal role in decision making, because of its ideal position in orchestrating sensory information from inside and outside the body. It is connected to the limbic system receiving cognitive and emotional information, as well as connections to cortical areas involved in motor planning and execution [BALLEINE, 2007].

*Figure 5: The cerebral cortex [BEAR et al., 2007e]*



Especially one part of the pFC is important in the linkage to food intake: the orbitofrontal cortex (oFC), which is located in the ventral surface of the frontal lobe. It is associated with emotional processing and hedonic experience, and can therefore be seen as the main nexus in reward and hedonic experience [KRINGELBACH, 2005 ; KRINGELBACH *et al.*, 2012].

Furthermore, the oFC plays a pivotal role in sensory-specific satiety in humans as demonstrated by *Kringelbach and colleagues* [KRINGELBACH *et al.*, 2003].

In conclusion, different brain structures have been identified associated with eating behaviour. This short and inevitably incomplete introduction into the most complex organ of the human body was given to accentuate certain aspects in the context of nonhomeostatic and homeostatic food regulation.

### **3.1.2 Homeostatic modelling**

The term homeostasis is derived from the Greek words “*hómoios*”, meaning *similar* and “*stasis*” meaning standing still – in other words, it describes the process that maintains the body’s internal environment within a narrow physiological range. In this context, the focus is put on energy homeostasis, where especially hypothalamic structures acting






as homeostatic regulator play a crucial role to orchestrate body energy balance [BEAR *et al.*, 2007b].

To assure the balance of energy expenditure and energy intake, every organism has to develop certain strategies to maintain equilibrium and find ways for auto-regulation. Therefore, fail-safe mechanisms have to be developed to meet an organism's need for nutrients.

Human eating behaviour is not random and consequently, people tend to prefer eating meals at certain times of the day. The interval-like structure of eating meals oscillates between times of energy intake and times without energy intake. This rhythm provides the organism with a structure which is reflected by the circadian periodicity and observable hormonal concentration peaks [HARROLD *et al.*, 2012].

In general, meal intake can be divided into three phases, demonstrated on the basis of biochemical determinants:

-  *The pre-cephalic and cephalic phase*, where sensory properties of the food and cognitive stimuli can trigger physiological processes for anticipating the arrival of the food before and while the food is eaten. Consequently parasympathetic and enteric division via vagal circuitry of the ANS are activated leading to excretion of digestive juices and salivation [MATTES, 1997 ; POWLEY, 2000]. Additionally, the mediation of enhanced microcirculation and increasing capillary perfusion in muscular tissue where macronutrients are disposed, is discussed in the literature as yet another anticipatory response [BUSS *et al.*, 2012].
-  *the gastric phase*, where chewing, swallowing and filling of the stomach amplify above mentioned reactions and stimulates the excretion of further digestive enzymes and hormonal responses.
-  *the substrate phase*, when substrate resorption takes place through specialized channel systems in the gut, leading nutrients to be released into the blood stream in the hepatic portal, where the distribution throughout the body begins (cf. Carbohydrates being transported by several different transporter systems of the

GLUT family; Proteins through PEPT1 Transporters) [WOOD and TRAYHURN, 2003 ; DANIEL, 2004].

Once absorbed, macronutrients and their metabolites can be divided into two categories. First, absorbed metabolites can be used for immediate metabolism that can instantly be accessed throughout the body. Secondly, metabolites can serve as energy storage in metabolically active tissue such as the liver or fat tissue, via diverse, well studied biochemical pathways and are re-used when necessary.

### ***3.1.2.1 Nutrient sensing***

Several sensing pathways developed throughout evolution to gain access to and provide current information feedback for the organism. Basically, extero-, proprio- and enteroreceptors represent the three basic receptor groups which form the basis for perception on a conscious and unconscious level.

Most food related information is projected to the major hub of the homeostatic system - the hypothalamus [SHIN *et al.*, 2009].

At first, multimodal sensory food properties are received via olfactory, visual, audible and tactile pathways in the pre-cephalic and cephalic phase. They are integrated via various cranial nerves and computed [VERHAGEN and ENGELN, 2006].

To monitor volumetric and mechanical properties of food, mechanoreceptors are found in the gastrointestinal walls to detect tension and stretch induced by the presence of chyme. This and the information of the enteric division are transported upstream via primary afferents. The principal mediator of the gut-brain axis is the nervous vagus [GREGERSEN and KASSAB, 1996 ; BAIRD *et al.*, 2001].

On the other hand, chemo-sensors are not only found in the oral cavity, but are also present in the gastrointestinal tract (GIT) to provide information about the nutrient composition of the food. These “taste-like” cells are, for example, different types of G-protein-coupled receptors of the T1R and T2R families (which are also found in sweet and bitter receptors), amino acid-sensing, calcium receptors and fatty acid transporters FATP4, CD36 and GPR120 [BERTHOUD, 2008].



Recently, umami taste receptors were found in the GIT, seemingly playing a role in nutrient sensing in the GIT as well [IWATSUKI *et al.*, 2012].

In combination, volumetric and nutrient sensors in the gut are able to quantitatively measure the gut content of each macronutrient available during and after the consumption of food [ZHENG and BERTHOUD, 2008].

Furthermore, findings that glucose sensing occurs in the hepatic vein and other peripheral vascular regions, as well as in the brain stem, indicate that the nutrient sensing system is wide-spread throughout the organism to provide information about available nutrients in the organism [MATVEYENKO and DONOVAN, 2006 ; LEVIN *et al.*, 2011].

### ***3.1.2.2 Peripheral hormonal signals***

Different regions of origin are involved in the regulatory effects which influence eating behaviour, such as circulating signals of nutrients and energy availability, as well as energy expenditure signalling. In this chapter, the main peripheral signals are described without going further into detail about energy expenditure signalling.

The lumen of the GIT is long known to play a crucial role in food availability signalling. Several hormones which are involved in satiety and hunger signalling are produced in this region of the body. These can be classified as either tonic or episodic signals that underpin the express of appetite. All of these hormones are able to regulate numerous functions in the CNS by passing through the blood brain barrier [SMITH and GUMBLETON, 2006].

*Halford and Blundell* introduced the differentiation between tonic/episodic signalling by referring to tonic factors as the constant signalling of the body's' metabolic need over a long-term period (e.g leptin). On the other hand, episodic factors such as cholecystokinin functioning as short-term signals are generated by recent consumption. Moreover, the differentiation between orexigenic (appetite stimulating signalling) and anorexigenic (appetite inhibiting) signalling is used to analyse peripheral physiological

signals related to food intake [HALFORD and BLUNDELL, 2000 ; KOKKINOS *et al.*, 2010].

The hormones insulin and glucagon will be left out of the description.

*Table 1: Overview about major peripheral and central signals for the express of appetite; after [HARROLD et al., 2012]*

	peripheral		central
	<i>Episodic</i>	<i>Tonic</i>	
Orexigenic	Ghrelin	Progesteron	NPY, MCH, Orexins
Anorexigenic	CCK, GLP-1, PYY <sub>3-36</sub>	Leptin	Melanocortin, CART

#### *Episodic satiety signals - CCK, GLP-1, and PYY<sub>3-36</sub>*

Satiety signals (SSs) are signals indicating the termination of a meal and therefore play a pivotal role in food intake regulation.

#### *Cholecystokinin (CCK)*

The anorexigenic hormone CCK is mediating meal termination and possibly early phase satiety - referred to as satiety signal [HARROLD *et al.*, 2012].

CCK is released via the I-cells in the mucosal epithelium of the proximal intestinal tract. The excretion follows the detection of free fatty acids of carbon chain length C12 and protein in the gut. After meal initiation CCK concentrations peak at around 25 minutes post-intake and don't begin to fall until 3h after meal intake [PAIK *et al.*, 2007].

The distributions of CCK receptors are indicating that the hormone uses a dual pathway for communication. This occurs either on the primary route via the vagal nerve and the NTS to the hypothalamus, or by directly binding to CCK-1 receptors of the hypothalamus. Functionally, the effect of CCK can be described with a “gatekeeper

role” by regulating synaptic transmission in different parts of the brain itself [LEE and SOLTESZ, 2011].

Additionally, in afferent neurons of the vagal nerve, CCK can modulate the response capacity to other appetite regulating hormones. Hereby, CCK depletion in the fasting state can lead to an increase of cannabinoid- and melanin concentrating hormone-receptor expression, which are associated with stimulation of food intake [DOCKRAY, 2009].

In other words, if the CCK concentration is low, the behavioural output such as searching for food through increased hunger signalling can be observed.

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

GLP-1 is synthesized in the L-cells of the distal small intestine and in the medulla oblongata in the brain. It is co-excreted with Peptide YY (PYY) and belongs to the incretin family of hormones associated with the ability to stimulate the insulin release. GLP-1 is released into the blood after luminal detection of carbohydrates as well as fat-induced release of GLP-1 has been demonstrated by *Frost and colleagues* in rodent experiments [LAVIN *et al.*, 1998 ; FROST *et al.*, 2003].

The main anorexigenic effect of GLP-1 is to release insulin into the blood stream, to inhibit glucagon production and additionally delaying gastric emptying. The information projection is assumed to be mediated via the vagal nerve, but other more direct pathways to act locally in the brain are discussed as well, and are especially interesting because GLP-1 is also a neuroendocrine peptide produced by neurons in the medulla oblongata with projections to the hypothalamus. Hence it is rather difficult to distinguish between the sources of GLP-1 [ZHENG and BERTHOUD, 2008].

#### *Peptide YY<sub>3-36</sub> (PYY)*

PYY<sub>3-36</sub> is released in the L-cells of the distal intestine and is expressed to appearance of all macronutrients, fibre and bile acid. PYY<sub>3-36</sub> is released towards the end of a meal and its circulation leads to decreased gastric emptying and additionally modulates

appetite circuitry in the hypothalamus, which leads to an inhibitory effect of food intake [BATTERHAM *et al.*, 2002 ; ONAGA *et al.*, 2002].

In obese individuals, PYY<sub>3-36</sub> levels were observed to be lower than in lean individuals, suggesting that PYY<sub>3-36</sub> may be involved in the pathogenesis of obesity [BATTERHAM *et al.*, 2003].

#### *Episodic hunger signal: Ghrelin*

Meal initiation brought up long and controversial discussions about how and when we start to eat. Anyhow, the anticipatory force that leads to diverse hormonal responses during the cephalic and pre-cephalic phases indicate that sensory associations by presenting a gustatory stimulus and cognitive stimuli can jump-start digestive processes and therefore have to be taken into account [BUSS *et al.*, 2012].

However, on a hormonal level, ghrelin is an example for episodic hunger signalling.

#### *Ghrelin*

Ghrelin is an orexigenic hormone functioning as hunger-inducing peptide, mainly synthesized by the fundic cells of the stomach. Ghrelin can be found in the brain and the periphery where its highest concentrations are found in the gut. Human plasma concentrations peak prior to meal intake [CUMMINGS *et al.*, 2001].

*Tschoep and colleagues* demonstrated in rodents that direct injection of ghrelin over time leads to weight gain by reducing fat utilisation and increasing food intake [TSCHOEP *et al.*, 2000].

Hence, the orexigenic effects can be innervated vagally as well as via ghrelin binding receptors (growth hormone secretagogue receptors ; GHSR-1a), which are expressed in high concentrations in the ARC and ventromedial nuclei of the hypothalamus and therefore suggests a dual pathway for ghrelin [CONG *et al.*, 2010].

### *Leptin – the main tonic, anorexigenic hormone*

In contrast to episodic signals, which are short-term signals, tonic signalling reflects the body's energy storage and metabolic processes. The main focus will be held on the primary energy storage of the body: the fat tissue which is represented by plasma leptin concentrations.

Leptin was discovered by *Friedman and colleagues* in 1994 in genetically modified mice, with single-gene mutations inhibiting the expression of leptin and its receptors. Leptin levels in the blood stream are balanced with body fat tissue. This means that greater levels of leptin represent greater size of body fat tissue [ZHANG *et al.*, 1994 ; FRIEDMAN and HALAAS, 1998].

On a cellular level, leptin is mediated by three different leptin receptors (OB-R), which are located in the hypothalamus, the medulla and other sites of the brain. Its anorexigenic effects are dually mediated in the hypothalamus. On one side, leptin interacts with the central neural processes that inhibit appetite-stimulating pathways like NPY/AGRP, and stimulate appetite-inhibiting pathways like CART/POMC [BALTHASAR *et al.*, 2004 ; HARROLD *et al.*, 2012].

On the other hand, interactions between leptin and CCK occur. Leptin may enhance the satiation effect of the episodic hormone CCK, while the underlying mechanisms remain unclear [EMOND *et al.*, 1999].

#### **3.1.2.3 Central hormonal signals**

Endocrine signalling act centrally in the brain by affecting eating behaviour and therefore anorexigenic and orexigenic pathways will be illustrated briefly.

### Anorexigenic signals

#### *CART/POMC pathway*

Originally research on psychoactive drugs, respectively psychomotor stimulants like cocaine and amphetamine, discovered a positive regulatory role of CART in the brain. CART mRNA is centrally distributed in hypothalamic and NAc sites. It is co-localized with POMC, both acting anorexigenic. CART/POMC pathways can be stimulated by the presence of leptin as mentioned above [HARROLD *et al.*, 2012].

#### *Melanocortin system*

The melanocortin system plays a crucial role in regulating energy homeostasis. Therefore, two major receptors (MC3-R ; MC4-R) act synergistically. They are located in the hypothalamus and mediate the melanocortin peptides (all derived from POMC), having an anorexigenic effect in rats [ABBOTT *et al.*, 2000].

Among others, the activity of the system is determined by leptin inhibiting POMC pathways as mentioned above. Additionally, the endogenous agonist AGRP for MC4-R showed orexigenic effect in rats [OLLMANN *et al.*, 1997].

### Orexigenic signals

#### *Neuropeptide Y (NPY)*

In hypothalamic regions associated with food intake regulatory effects, NPY is found in high concentrations, especially in the ARC of the hypothalamus. NPY is associated with promoting meal initiation and delaying the beginning of satiety with the effect that size and duration of meals are increased in rat models and therefore has an orexigenic effect [CLARK *et al.*, 1985].

Additionally, NPY is sensitive to a variety of peripheral signals such as ghrelin and leptin being able to modulate its' orexigenic effect [HARROLD *et al.*, 2012].

### *Melanin-concentrating hormone (MCH)*

MCH is expressed in the hypothalamic regions associated with the regulation of energy homeostasis and co-expressed with CART and the zona incerta. Mouse models showed that high concentrations of MCH injections lead to a dose-dependent increase of food intake, and therefore an orexigenic effect of MCH is suggested [QU *et al.*, 1996].

There are two major receptors identified yet. One of them, MCHR1 is likely to mediate the orexigenic effects of MCH due to the fact that high concentrations of the receptor can be found in the limbic structures associated with energy homeostasis [HARROLD *et al.*, 2012].

### *Orexins*

Orexins and their receptors are found in cerebral structures and the enteric division. The endogenous orexin system consists of two peptides called OX-A and OX-B, along with two G-protein coupled receptors (OX-1 and OX-2). OX-A has a higher affinity to OX-1 receptor than OX-B, while OX-B binds with a higher affinity to receptor OX-2. In mouse models, the stimulation of orexin expression is induced by the falling of plasma glucose levels and fasting, but is inhibited by satiety signals. After intracerebral administration of orexin, a hyperphagic response in rodents was observed. Highest food intakes followed the administration of OX-A [CAI *et al.*, 1999 ; RODGERS *et al.*, 2002].

Peripheral orexins expressed in the enteric division may sense the nutritional status in order to modulate gastrointestinal secretion and motility as demonstrated in rats [BENGTTSSON *et al.*, 2007 ; HARROLD *et al.*, 2012].

Together, both MCH and orexins are associated with the expressions of neurons acting as metabolic sensors in the LHA [ADAMANTIDIS and de LECEA, 2009 ; SILVA *et al.*, 2009].

### 3.1.3 Nonhomeostatic modelling

*“...one antecedent fueling the hyperphagia driving obesity is dietary-induced disruption of the higher-order learned controls of feeding behavior” [KANOSKI, 2012].*

Eating in the absence of metabolic need is called nonhomeostatic eating. Another expression may be “hedonic” eating, implying emotional, rewarding and other cognitive factors being unquestionably involved in food intake related decisions. In this context, the term nonhomeostatic eating will be used [KANOSKI, 2012].

As described in *chapter 3.1.2*, focus on research in metabolic and neural feedback systems representing “homeostatic regulatory mechanism” were conducted extensively in the last decades. In comparison, nonhomeostatic eating pathways located mainly in corticolimbic structures are much less understood. Hence, this chapter presents principle nonhomeostatic mechanisms [SHIN *et al.*, 2009 ; BERTHOUD, 2011 ; HARROLD *et al.*, 2012].

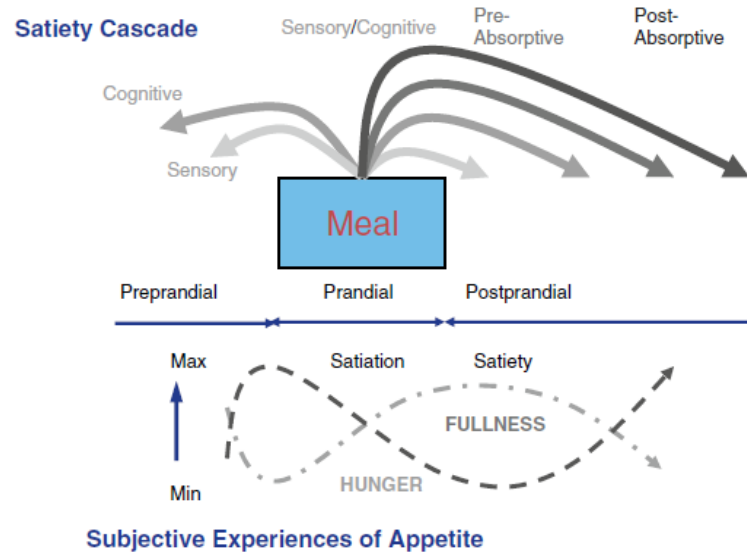
The process of eating can be divided into cephalic, gastric and substrate phase. To extend this model with basic psychological concepts like satiety, satiation, hunger and fullness - *Blundell and colleagues* introduced the satiety cascade adding events that stimulate, happen before, during and follow food intake [BLUNDELL, 1991 ; BLUNDELL and HALFORD, 1994].

In this model, food intake is divided into preprandial, prandial and postprandial sections, where different multimodal sensory properties of food; pre - and postabsorptive, as well as cognitive factors, determine food intake. The cascade analogy illustrates the multicausal character of this process.

The terms “hunger” or “fullness” are used to determine the subjective feeling of appetite, which can be assessed with VAS scale scores. Numerous internal factors influence the expression of the parameters such as prior meal intake, physical activity or temperature [FLINT *et al.*, 2000].



*Figure 7: The satiety cascade after [BLUNDELL, 1991]*



### 3.1.3.1 Main nonhomeostatic mechanisms

#### *Learning*

Conditioning as a basic psychological concept of learning was first formulated by *Pavlov* who showed that condition reflexes involve the formation of learned associations between a conditioned stimuli (CS) and an unconditioned stimuli (US) [PEARSON, 2012].

Learning theory provides that the magnitude of the reinforcer (the unconditioned stimulus) seem to be the most important determinant of the learning process influencing the rate and the extent of learning processes. In other words, the stronger the CS-US association is, the more powerful the memory of the US becomes [KANOSKI, 2012].

Preferences and aversions can be explained through conditioning theory whereby, for example flavour (CS) associations with postingestive malaise or nutrient absorption (US) can be learned, as demonstrated in diverse rat experiments [GARCIA *et al.*, 1955 ; SCLAFANI, 2001].

Further experiments revealed a “cue-potentiated” paradigm, in which rats where trained in conditioning boxes with cues that either signal food access (CS+), or no food access

(CS-). Subsequent presentation of the CS+ in non-deprived rats lead to elevated feeding of rats trained in CS+ states in comparison to CS-. The neural circuitry underlying this pavlovian conditioning is described by direct pathways from basolateral and basomedial nuclei of the amygdala and orbitofrontal cortex to the lateral hypothalamus [PETROVICH *et al.*, 2002 ; PETROVICH *et al.*, 2005 ; PETROVICH *et al.*, 2007].

The role of awareness for pavlovian conditioning is discussed controversially. Findings indicate that associative learning can operate without conscious awareness [PEARSON, 2012 ; RAO *et al.*, 2012].

Additionally, habituation is yet another form of learning. The ability to selectively filter incoming information in higher sensory centers of the brain, evaluating these information and using the stimuli that are needed by decreasing information about unimportant stimuli (see *chapter 3.3.1.1*) [RANKIN *et al.*, 2009].

### *Reward and emotions*

Emotions are said to reinforce advantageous and suppress potentially harmful behaviours. If, for example a food is positively reinforced, it is said to be rewarding. Humans and animals prefer certain foods over others, and there are several factors influencing the palatability of foods. It has been shown that HF/HS - diets are more rewarding than others, while reward in itself is a dynamic and individual-specific state, influenced by numerous individual factors [OLSZEWSKI *et al.*, 2011].

In general, the distinction between opioidergic and dopaminergic pathways underlying reward is made in the literature.

Two hypotheses which are equally supported by data explain the action of midbrain dopamine and excessive food intake. The first is the “gluttony hypothesis”, which suggests that overindulgence or gluttony in pleasurable stimuli relies on a positive correlation between the amounts of dopamine signalling generated and pleasure derived from a sensory experience. The “reward-deficiency hypothesis” on the other hand suggests that overindulgence is an attempt to self-medicate and bring deficient dopamine signalling to pleasurable levels [BERTHOUD, 2011].

Hedonic “liking”, the enhanced appreciation of a reward for certain foods, may be mediated by endogenous opioid peptide signalling. An inhibitory effect of opioid antagonists (Naloxone or Naltrexone), was demonstrated in rodents, followed by the selective reduction of highly palatable food intake such as HF/HS foods. In reverse, the consumption of highly palatable foods stimulate  $\beta$ -endorphin release, leading to the conclusion that the endogenous opioid system may stimulate food intake by modulating the palatability of foods [YEOMANS and GRAY, 2002].

Most results support the hypothesis that the effect of the opioid system on eating is larger in preferred foods than in other, less palatable foods [KANOSKI, 2012].

On the other hand, “wanting” forms another construct of the reward phenomenon. The incentive motivation, where cues associated with rewarding foods, act as motivators to seek for food, is based on a dopaminergic system located in the midbrain and project to the nucleus accumbens, prefrontal cortex and amygdala. Intake of preferred foods increases dopamine levels in various sites regulating natural rewards for environmental cues which are associated with appetitive reinforcement and increasing goal directed behaviour in rats [BERRIDGE, 2007 ; KANOSKI, 2012].

In contrast to the distinction of “liking” and “wanting”, *Havermans* argues that neurophysiological correlates alone do not provide enough evidence that the two mechanisms function independently. Additionally, the validations of the two constructs may still be insufficiently. Hence the differentiation between these two mechanisms may be a form of ad-hoc theorizing. He suggests that both mechanisms are interconnected and act synergistically [HAVERMANS, 2011 ; HAVERMANS, 2012a].

Nevertheless, in summary, basic learning principles can provide explanatory attempts to the question why for example preferred foods maintain greater stimulus control over eating behaviour, compared to less preferred foods. However, the question why certain foods are preferred and over-consumed than others, still has a highly individual and dynamic component to it.

## *Memory*

Memories are essential for the simulation of future events by using autobiographical experience. Why and how experiences are stored in memory is still not fully explained but the construction of memory per se is a highly selective and creative process, utterly susceptible for errors [SCHACHTER and ADDIS, 2007].

As indicated in *chapter 3.1.1.3*, memory plays a key role in eating behaviour. Amnesic patients ate multiple meals in a 10-30 min interval without reporting changes in the subjective feeling of hunger measured with hunger scores [ROZIN *et al.*, 1998].

Furthermore, patient H.M who had parts of his frontal lobe and hippocampus removed to treat his epilepsy resulted in having impaired memory. H.M ate a second dinner when confronted with another one without indicating changes in the subjective feeling of hunger [HEBBEN *et al.*, 1985].

It has been demonstrated that sensory-specific satiety is intact in amnesic patients with impaired memory (medial and frontal lobe lesions). Consequently hyperphagia in these patients may not be due to a decoupling of sensory-specific satiety [HIGGS *et al.*, 2008].

In an evolutionary context, memory is important in the context of food-related behaviour by setting food into a spatial and episodic relation. Information about “place preferences” (also called contextual learning), respectively when and where food was found and can be found again can be processed and stored through memory activation [FERBINTEANU and McDONALD, 2001].

Interestingly, different receptors for episodic and tonic satiety and hunger hormones, such as ghrelin, GLP-1 and leptin can be found in diverse hippocampal sites. This indicates that depending on the hormonal status of these hormones a long or short-term modulation of hippocampal-dependent learning can be modulated through bottom-up signalling [KANOSKI, 2012].

The recall of images, like evoking a mental image is yet another form of memory. Several studies were performed to investigate the relationship of memory about food and its influence on subsequent food intake.

A recall paradigm by *Higgs and colleagues* was developed to investigate effects of memory of recent food intake in a camouflaged taste-test setting on subsequent food intake. Participants were invited to eat pizza for lunch and come back to the laboratory for a taste-test with cookies after 2-5 hours. Prior to the taste-test participants were divided into intervention and control group. Both groups had to perform a recall task (thinking about a certain point in time and writing down their thoughts). While the intervention group had to recall the lunch, participants in the control group could think about everything they wished to think about. After the intervention, participants conducted a taste-test and were told to eat as much as they wanted in a 10 min timespan. The comparison of both groups in terms of the amount of cookies eaten revealed significant differences. Participants recalling the lunch ate significantly less cookies than participants in the control group [HIGGS, 2002].

Additionally, the presence of visual cues influencing food intake has been shown by some elegant experiments made by *Wansink and colleagues* (“chicken wing” experiment ; “bottomless-soup” experiment). Both experiments revealed that the amount of eaten food in the experiments depends on the presence of visual food cues indicating the amount of eaten food. Hereby the memory of what was eaten during the meal seems crucial in determining food intake [WANSINK and CHENEY, 2005 ; WANSINK *et al.*, 2005].

Under a bottom line, memory processing seems to play a pivotal role in short-term food intake indicated by presented studies above.

### ***3.1.3.2 Other cognitive factors***

Early, important studies by *Schachter and colleagues* provided evidence that cognition is essential in the way emotions are processed. In a now famous experiment participants were given epinephrine to evoke physiological arousal. Dependent on their mental state (angry or comic state), participants showed different reactions to the administration of epinephrine - either euphoria or anger. This led the authors conclude that different cognitive contexts can evoke different emotional reactions to the same stimulus [SCHACHTER and SINGER, 1962].

This may also be true in the perception of food or situations involved with food intake where different mental states influence the outcome of associations one makes with the experience with the food leading to an increase or decrease of food intake.

In the context of eating behaviour, *Berthoud and colleagues* created the term “cognitive/emotional brain” referring to an integrator system or interface that reflects several nonhomeostatic regulatory mechanisms. Thus diverse environmental and lifestyle factors additional to individual brain wiring shapes this cognitive/emotional brain. Food related associations are central in describing the connections between cognitions and food. Every autobiographical connection that is made in relation to food evokes mechanisms shaping memory, attitude, awareness, intentions and social habits. Moreover, genetic and epigenetic factors wire the cognitive/emotional brain [BERTHOUD, 2011].

#### *Situation and distraction*

Cognitive control and evaluation processes influencing eating behaviour could be identified by early research of *Schachter and colleagues*. The manipulation of a clock in a laboratory setting led to consummatory adaptations of the participants. In a camouflaged setting, participants were told to eat as many crackers as they wish while participating in a study where different physiological reactions like heart rate and sweat gland activity was measured. The clock on the wall of the room was manipulated letting the participants believe that they snacked either before or past their dinner time (for each participant around 6 o'clock in the evening) – the true timing of the test was identical for every participant. Interestingly obese participants ate more when they thought they were eating past their dinner time compared to obese participants who thought that they were eating before their dinner time. The opposite effect could be observed in normal weight participants who ate less when they thought it was past their dinner time compared to normal weight participants who thought it was before their dinner time. Normal weight participants reported that they didn't want to spoil their dinner and therefore restrained themselves from eating too many of the crackers. Displaying an example for cognitive manipulation [SCHACHTER and GROSS, 1968].

Social settings may also influence food intake such as the presence of other people while eating, and the relationship these people have with each other. When eating in a social setting with a group of people or at least one more person, many different external cues modulate oneself's eating behaviour. These socially derived norms can either have inhibitory or stimulating effects on food intake [HERMAN *et al.*, 2003].

Furthermore the presence of distractors can modulate eating behaviour. For example watching television while eating was shown to increase meal intake. A role for memory was found. Participants who were watching TV during dinner had less vivid memories of the meals compared to control groups indicating the distractional potential of TV-dinners on food intake [HIGGS and WOODWARD, 2009 ; MITTAL *et al.*, 2011].

#### *Food-related associations*

Additionally, cognitions or expectations about specific attributes of foods can alter subsequent food intake. For example the information about the total caloric load of a preload milkshake (a required first course) caused sixteen obese and sixteen normal weight participants to modulate their eating behaviour in a standardized subsequent meal setting. The milkshakes were labeled either as 200 or 600 calories while actually containing the same amount of energy. Participants who believed to have eaten more calories in the preload milkshake subsequently ate less of the standardized meal and reported greater fullness [WOOLEY, 1972].

Furthermore the information about the fat content of a preload yogurt before a meal was associated with a modulation of subsequent meal intake in forty-eight healthy, nondieting women. In this study three different yogurts were presented as preload (low fat with low calories; low-fat and high fat with the same amount of calories). Interestingly, participants who ate yogurts labeled as low-fat (with high amount of calories) subsequently ate more at lunch than those having high-fat yoghurt as preload (with the same amount of calories). The importance of this finding is that the information on the labeled yogurts modulates the amount of subsequent meal consumption [SHIDE and ROLLS, 1995].

In another important study, *Crum and colleagues* could demonstrate that the expectation or mind-set about a consumed food can alter ghrelin response measured in the blood post-intake – providing evidence for a psychological top-down alteration of a physiological parameter. A milkshake with 380kcal was consumed by 48 participants which was either labelled as 620 kcal “indulgent” or 140kcal “sensitive” milkshake. Ghrelin was measured in three time points and a steeper decline of ghrelin levels could be observed in participants who thought they would consume an indulgent milkshake. The finding is consistent with the difference in ghrelin response to beverages with real caloric differences [Le ROUX *et al.*, 2005 ; CRUM *et al.*, 2011]

Modulation of preference could also be shown to be influenced by these top-down processes. Differences in sensory ratings for identical wines with different labels could be shown. Participants in this study rated the wine which was labeled better in quality (higher price, better reputation: “californian wine”) superior compared to an identical wine which was labeled qualitatively lower (lower price, worse reputation: “north dakota wine”). This showed that the expectation of a product influences its sensory property evaluations [WANSINK *et al.*, 2007].

#### **3.1.4 Regulatory cross-talk**

As demonstrated above, the motivation to eat can arise from metabolic needs regulated by hard-wired homeostatic eating regulatory systems or nonhomeostatic motives associated with higher cortico-limbic structures.

An observation in overfed rats may be crucial in this context. Rats were fed to satiety with an increased expression of POMC and decreased expression of NPY in hypothalamic regions reflecting the energy surplus, indicating anorexigenic signalling. Afterwards, rats were presented with a HF/HS – diet and although metabolic signalling indicated a sated metabolic state, the rats were eating and subsequently became obese. This finding indicates the overriding power of nonhomeostatic mechanisms in an animal model and raises the question to what extent this effect may be transferable to humans, as well [La FLEUR *et al.*, 2010].



The importance of the interplay between these two mechanisms has been argued since the early 90's. *Woods* described the paradox nature of food intake producing rewarding and aversive effects. He argues that different physiological and behavioural adaptations aim to regulate food intake and reduce its potentially aversive effects. In this context maintaining metabolic equilibrium and meeting rewarding needs are essential mechanism for regulation [WOODS, 1991].

#### ***3.1.4.1 Bottom-up signalling***

When metabolic signals have a modulatory effect on higher brain functions one refers to bottom-up processes. One well studied phenomenon called incentive salience, meaning that a heightened motivation to eat which can be induced in metabolically hungry individuals, has long been known [BERRIDGE *et al.*, 2009].

One possible explanation could be that orexigenic signalling modulate cortico-limbic structures associated with food reward. Indeed, an observable increase in neural activity in these areas of the brain was detected in humans in a metabolically hungry state when presented with high-caloric pictures of foods compared to low-caloric pictures of foods [GOLDSTONE *et al.*, 2009].

To make foods more compelling for humans one main determinant is its palatability which is mediated through sensory properties. The perceived tastes or odours can be manipulated by peripheral metabolic signalling as well. For example leptin is associated with the modulation of olfaction by changing mucous production in olfactory mucosal cells. Leptin administration was reported to increase mucous production and therefore enhance the detection of odourants by olfactory neurons [BADONNEL *et al.*, 2009].

Other central and peripheral hormones like endocannabinoids, GLP-1 and vasoactive protein (VIP) are also in discussion to modulate sensory properties of foods [BERTHOUD, 2011].

Additional to the change of sensory perception of foods, bottom-up modulation of the dopamine-system can be observed and may be crucial in the connex of homeostatic and nonhomeostatic mechanisms. Basically, metabolic status about the current caloric

supply either up or down regulates for example neurons acting on the midbrain dopamine system. Leptin can act directly on dopamine neurons and indirectly via the LHA in the midbrain modulating rewarding mechanisms [LEINNINGER *et al.*, 2009].

As mentioned in *chapter 3.1.2.2*, MCH and orexin producing neurons in the LHA act as metabolic sensors and therefore are in an ideal position to function as interface between metabolic sensing and behavioural adaptations [BERTHOUD, 2011].

Interestingly, orexins/hypocretins seem to play a role in hippocampal synaptic plasticity and therefore potentially influences memory functioning [SELBACH *et al.*, 2010].

#### ***3.1.4.2 Top-down signalling***

In contrast to bottom-up signalling, top-down processes emerge from neural activity in higher cortical areas inducing modulations in peripheral metabolism and consequently behavioural output.

In *figure 8* possible interactions between neural systems and energy metabolism is depicted. Red arrows facing downwards represent top-down modulation of homeostatic processes. As opposed to the bottom-up signals influencing cognitive, reward and sensory information – top-down signals can modulate metabolic signals through neural activity.

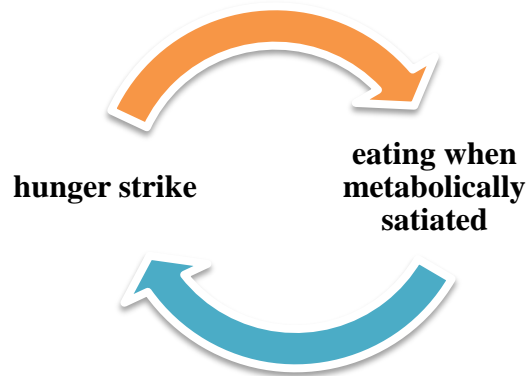
On the one side through modifying the homeostatic regulator systems in hypothalamic and brainstem structures. For example the projections which can be found from sensory input directly to the LHA [NORGREN, 1970].

As seen in *chapter 3.1.3.1* extensive connections between oFC and amygdala to the LHA play a role in conditioning mechanisms described by *Petrovich and colleagues*.

Additionally, hypothalamic structures receive direct input from the NAc shell involved in opioidergic reward-driven food intake as seen in *chapter 3.1.1.3*.



Figure 9: Oscillating cognitive control of food intake



On a subconscious level, top-down processes involve endocrine and ANS regulation mechanisms which are not well understood to this point in time. Knowingly the limbic structures acting as old pathways play a key role in mediating subconscious behavioural control [BERTHOUD, 2011].

In the motor cortex, subconscious processes that escape our awareness could be observed. Even when we think that a free decision is made by ourselves, neural activity in SMA (supplementary motor area; a brain area involved in motor preparation in the cortex) could be observed prior to the actual awareness of the decision itself indicating a following motor-action [SOON *et al.*, 2008].

At last, the interaction between cognitive and emotional processes may be central, especially in the context of the thesis. As demonstrated in *chapter 3.1.3.2* the power of cognitions which have a modulatory effect on food intake behaviour may especially be an interesting research topic in the modern world we live in.

All in all it remains unclear to what extent other nonhomeostatical factors contribute to food intake but research about these factors and additionally the cross-talk between the two systems indicate an enormous potential for explaining major mechanism of regulatory systems.

## 3.2 Mental imagery

Mental imagery certainly is unique without being a culturally dependent phenomenon. Its cross-cultural character can be found from western to non-western societies. Children start to be aware of their own mental images, at around three years of age [ESTES, 1998].

Referring to a quasi-perceptual experience in any sensory mode, the term “imagery” is widely used among cognitive scientists to describe this phenomenon. When humans visualize objects, scenes or processes, a mental mechanism is initiated. This process is called mental imagery.

The problematic nature of the expression, in preliminary and recent research, will be presented in the following chapter.

### 3.2.1 Mental imagery - functions and structures

#### 3.2.1.1 *A brief historic sketch*

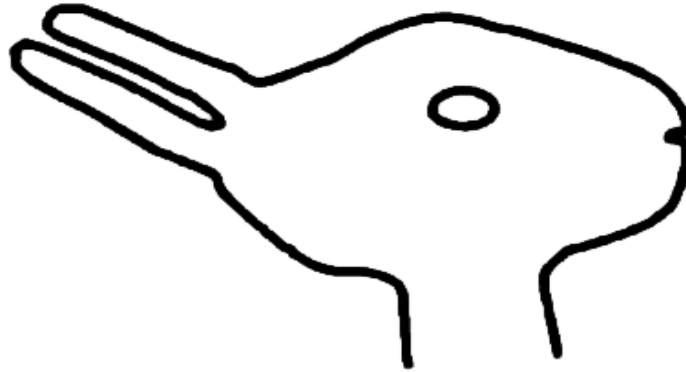
*One could not study the mental image separately. There is not a world of images and not a world of objects. Rather every object, whether it is presented by external perception or it appears to inner sense, is susceptible to functioning as a present reality or as an image, depending on the center of reference that has been chosen [SARTRE, 1948].*

With this quote, Sartre points out the important basic assumption about the construction of reality, which is neither purely represented by inner states, nor by external perception. The question of how humans perceive reality is a rather difficult one to tackle, but the fact that thinking about mental imagery pushes existential epistemological questions to its limits, speaks for the importance of the phenomenon.

The ambiguity of the duck-rabbit (*figure 10*) exemplifies the ambiguity of perception, and points out that our autobiographical knowledge about the world shapes what we

perceive. We perceive these mental images in a highly subjective way, which is shaped even more than actual visual perception on one's autobiographical identity.

Figure 10: The duck-rabbit



The philosophical debate about the nature of the phenomenon of mental imagery played a prominent role in Greek and modern philosophy, because of its incorporated character, which let humans participate vividly in their own cognitive process. The philosophical debate by early Greek philosophers like *Aristotle* to early modern philosophers like *Descartes* and *Hobbes* is interrogated elsewhere :

[<http://plato.stanford.edu/entries/mental-imagery/#PreVieIma>, PreVieIma].

In a yet classical psychological experiment, *C.W. Perky* first demonstrated the overlapping between mental imagery and real perception. He asked participants to fixate on a blank screen and imagine an object (banana or a leaf). Without the participants' knowledge, the experimenter gradually projected an image of that object on the screen. Starting below the threshold of conscious perception and increasing gradually the definiteness of the object on the screen. While all participants continued to believe that they were just imagining the object, many noticed that their imagination changed while another picture was presented, but still thought they saw it with their mind's eye. The so-called "perky-effect" demonstrates the interference between mental imagery and actual perception [BARTOLOMEO, 2002].

The psychological paradigm of behaviourism dominated most of the 20<sup>th</sup> century until cognitivism became a more popular paradigm in the 1960's. Most behaviourists neglected the importance and doubted the existence of mental imagery while assuming

that the human mind is a black box which could not be observed and therefore is not worth investigating. These “iconophobic” tendencies are reflected in the relative lack of studies in this field during that time-span [WATSON, 1913].

From the beginning of cognitivism in the 1960’s, a “dual-code” paradigm was influential by stating that humans either think in pictures or think in words (imaginal and verbal coding) [PAIVIO, 1971].

Findings of the structural and metrical properties of mental images induced by participants, e.g. time spent to mentally rotate an image, is proportional to the angle it has to be rotated, and the time spent to mentally scan an image is proportional to the distance to be scanned, provided additional cornerstones in describing the phenomenon. Consequently a picture-like character of mental imagery was proposed [SHEPARD and METZLER, 1971 ; FINKE and KOSSLYN, 1980].

Mental imagery research findings were integrated into computational and information-processing paradigms. Supported by early fMRI and PET studies identifying picture-like structures in brain regions involved in mental imagery and visual processing, the most developed and popular theory in mental imagery research emerged, referring to a re-perception of a picture with a mind’s eye, the so-called quasi-picture theory [KOSSLYN, 1994 ; GODDALE, 1995].

This view is strongly opposed by the rational of cognitive psychologist *Zenon Pylyshyn*, which is examined in an ongoing “imagery debate” over the last decades. The “grand illusion” of seeing with the mind’s eye and leading to the classical “homunculus fallacy”, is often debated in cognitive sciences and is central in his rationale. The homunculus is often referred to as a “little man” sitting in the back of the head perceiving, experiencing and interpreting mental images. In fact, a theory based on that assumption needs an infinite amount of homunculi interpreting interpretations of the predecessors, and so on. Arguing that the subjective, intuitively most plausible theory about the nature of mental images tells us nothing about the nature it is representing. *Pylyshyn* proposes that mental imagery is a special form of general reasoning with different content or subject matter (cf. optical and geometrical matters), without neglecting the overlapping modalities of visual and mental imagery processing – often

referred to as *propositional description theory* [PYLYSHYN, 1973 ; PYLYSHYN, 2002].

A more general theory has also been proposed, leading to a possible solution of the mental imagery dilemma – the so-called *grounded cognition theory*. In this theory, perceptual-symbol systems, such as higher cognitive processes like language comprehension, memory, and mental imagery, act as simulations of previous perceptual, motor, and introspective states by using the same systems that drive perception and action [BARSALOU, 2008].

### **3.2.1.2 Visual and mental imagery processing**

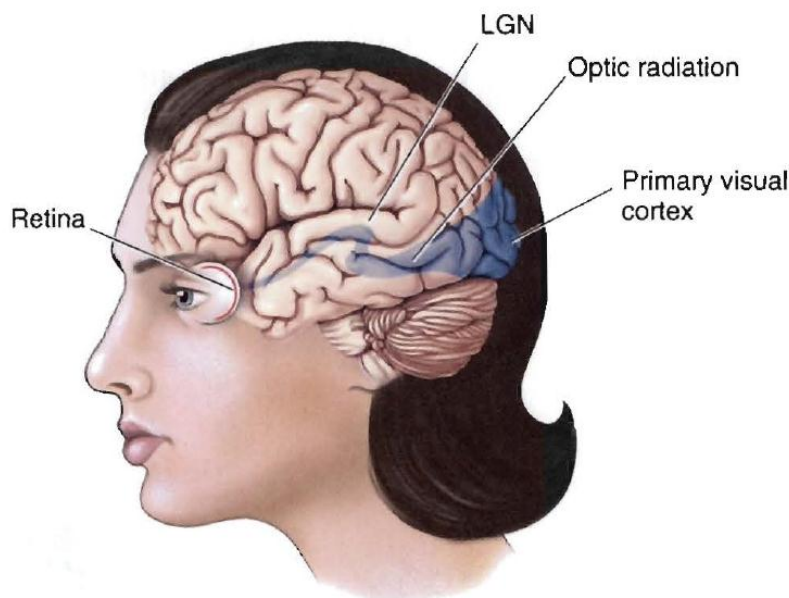
Our everyday visual perception is driven by bottom-up sensory information (external visual information) and top-down signals (internally generated information). As heard before, mental images can be generated without the presence of external visual information. The question, to what extent these two processes share the same neural substrates will be investigated in this chapter.

Visual processing has been extensively studied throughout scientific history. The main assumption is that information is projected from the retina of the eyes to the optic tract innervating the LGN of the thalamus. The LGN is located in the dorsal part of the thalamus, projects information and serves as hub that integrates retinal and extraretinal input. Interestingly, 80% of the LGN input comes from the primary visual cortex (V1) identified as corticofugal feedback pathway [BEAR *et al.*, 2007a].

From V1 two major cortical streams have been identified dissociating action and perception. The *dorsal stream*, which projects towards the parietal lobe (associated with visual motion and visual control of action; action attributes), and the *ventral stream* projecting towards the temporal lobe (associated with object recognition and recognition of visual attributes; perceptual attributes). Both streams are involved in visual processing [MISHKIN and UNGERLEIDER, 1982 ; GOODALE, 2008].



Figure 11. Side view of visual pathway mediating conscious visual perception with retinogeniculocortical pathway [BEAR et al., 2007e]



Another school of thought supersedes the distinction between action and perception and rejects the dissociation of action and perception by referring to an *enactive approach* of perception. Hereby, the brain is not seen as internal representation substrate of the external world passively picking up information. Instead, a more *enactive* approach is suggested stating that what we perceive is a consequence of individuals motor skills letting the environment provoke internal structures [O'REGAN and NOË, 2001 ; NOË, 2004 ; CARDOSO-LEITE and GOREA, 2010].

However, mental imagery processing of any kind is driven by internal stimuli mediated through top-down processes. To disentangle the overlappings and differences between visual and mental imagery processing, imaging techniques like fMRI and PET were offered to observe the brain's activity while performing both tasks. Preliminary studies showed remarkable overlappings in global activation patterns of the brain [GANIS *et al.*, 2004].

On the other hand, differences between mental imagery and visual processing can be exemplified by patients with neurological diseases. In case studies, where patients with damages or lesions in areas of the ventral visual pathway are having problems with the

recognition of objects – so-called object agnosia. The recall of images of objects by drawing from their memory could be observed despite their pathology [BEHRMANN *et al.*, 1992 ; KONEN *et al.*, 2011].

By the same token, patients with intact visual imagery and impaired mental imagery processing have been described. Here the visual acuity and perception was intact, while the retrieval of mental images was impaired [MORO *et al.*, 2008].

Research by *Lee and colleagues* investigated brain activation patterns of five male and six female participants (age  $25 \pm 1$  y) in a event-related design, where 10 common objects were repeatedly imagined or presented as external stimuli (image). The four regions of interests (ROI) associated with the ventral stream of visual processing (object recognition) were identified and activation patterns were compared in both conditions: the lateral occipital (LO), posterior fusiform (pFs), posterior retinotopic cortex (V1) and anterior retinotopic cortex (ES). Major findings of this study were that overlappings of activations in ROI in both conditions could be identified and differences in neural dynamics were observable. The authors concluded that in the absence of external input signalling, alteration of the internal processing in neural dynamics occurs within each region and the contribution from the substrates is different in each condition. In other words, differences in how the tissue is used in both conditions could be observed [LEE *et al.*, 2012].

Interestingly, mental images can induce category specific responses in cortical areas. For example, imagining faces increases neural activity in cortical areas which are associated with external face stimuli [ISHAI *et al.*, 2002]; odour imagery induction which activates the same cortical areas as odour perception [BENSAFI *et al.*, 2007] and gustatory imagery inducing activation in the same brain areas as gustatory perception [KOBAYASHI *et al.*, 2004 ; KOBAYASHI *et al.*, 2011].

In summary, and in the context of the thesis, these theoretical questions regarding the nature of mental images play an important role, because it provides evidence that same neural substrates are evoked during imagining and perception.

### 3.2.2 Research among disciplines

Mental imagery research was conducted in different disciplines over recent years and gained popularity since the post-behaviourist period. Psychological, linguistic, sports and nutritional sciences provided a wide array of studies in which mental imagery is used as an intervention tool to investigate certain phenomena.

This chapter will give a brief overview about mental imagery findings across different disciplines.

#### 3.2.2.1 Sports sciences research

A vast amount of research has been conducted in sports science to demonstrate the enhancement of motor skill performance and improvements in concentration, confidence and decreased anxiety by introducing repetitive imagery task into the practice plans of athletes [WEINBERG, 2008].

One example for increased motor skills performance would be the improvement of free-throw shooting percentage in basketball sports. Hereby, a high-school basketball coach conducted a systematic guided mental imagery exercise with the whole team after half of the games of a regular high-school season. Each imagery practice was conducted once a week and prior to 18 of 36 games of the high school season. Every mental imagery task lasted for 15 minutes and was guided by exact instructions about the conduction of the task. Significant improvement in shooting accuracy could be observed in the second half of the season [POST *et al.*, 2010].

Of course, this study has many limitations (no control group - only reversal study design, second half of the season in general better free-throw percentage than in the first half ?). Nevertheless, it demonstrates an observable effect of imagery on improvement of motor skills which is now fully implemented into best practice guidelines for certain sports [SCHUSTER *et al.*, 2011].

In sports science, two motor-based explanations for mental imagery are provided. First, *psychoneuromuscular theory* explains a basic framework, whereby imagined events of

motor-action movements may produce an exact sequence of muscle activity for the movement. Sports scientists therefore speak of motor imagery that improves motor learning and motor performance. The second approach is the *symbolic learning theory*. The assumption is that movement patterns are symbolically coded in the CNS. Thus mental imagery enables movements to become more familiar and the better the movement is coded, the better the performance will be. A third, more integrated model is the *bioinformational theory*, which states that imagery not only evokes cognitive reactions, but also physiological and emotional responses [MARTIN *et al.*, 1999].

In conclusion, sports science research provides insights about

- i) the importance of mental imagery in the discipline
- ii) the role of repetitive mental imagery tasks in the modulation of motor-skill performance

### **3.2.2.2 Psychological research**

In cognitive psychology research, imagery experiments were conducted in the context of a number of different pathologies associated with emotional, behavioural and cognitive dysfunctions. Cognitive-behavioural therapy (CBT) uses mental imagery techniques as a standard tool.

In general, mental imagery can amplify aversive states such as depression and anxiety disorders. In patients with bipolar disorders, intrusive mental imagery may be an essential feature of the disorder, by using mental imagery more often and more vividly as compared to people without the disease [HOLMES *et al.*, 2011].

On the other hand, mental imagery can function as “cognitive vaccine” when used to boost people’s resistance against depressed moods by evoking positive mental images. Interestingly, mental images have a more powerful effect on the emotional states of patients than verbally guided interventions [HOLMES *et al.*, 2009].

Emotional distress can be induced through so-called memory flashbacks in posttraumatic stress disorder where an image of the traumatic experience is relived through mental imagery [EHLERS and CLARK, 2000].

In the context of nutritional behaviour research, strong implications of mental imagery related to craving periods are indicated. Craving periods are periods of intense desire to eat a specific food. The description through imagery-related prescriptors like “I could picture the pizza in my mind”, or “I could picture eating it” indicate that the nature of craving seems to be primarily of visual nature [TIGGEMANN and KEMPS, 2005].

Additionally, the subjective experience of the craving intensity can be enhanced by thinking about “eating a favourite food” and, according to elaborated intrusion theory, cravings arise from intrusive thoughts about desired substances [HARVEY *et al.*, 2005].

On the other hand, craving periods can be suppressed using imagery techniques to avoiding cravings via associated images and by inducing alternative images. The induction of these images don’t have to be mediated through repetitive mental imagery tasks [KNAUPER *et al.*, 2011].

People who follow a restrictive eating style with the intention to maintain or to achieve a different weight are called *restrained eaters*. This psychological construct was first introduced by *Herman and Mack* and refers to a pattern of dietary intake characterized by cognitively controlling and suppressing physiological hunger signalling through usage of different coping strategies (e.g. mental imagery) with the aim to reduce caloric intake [HERMAN and MACK, 1975].

People who suppress their food-related thoughts may differ in the manifestation of a possible mental imagery effect on subsequent food intake as formulated in sub-hypothesis V at the end of *chapter 4*.

Self-reported inter-individual differences between the vividness of mental imagery have been reported [MARKS, 1973], and differences in brain activation patterns in EEG studies between participants with highly vivid mental imagery in comparison to participants with lower imagery have been reported [HIRSCHFELD *et al.*, 2012].

The inter-individual difference between people with high and low vividness of mental images may influence the mental imagery effect on subsequent food intake as formulated in sub-hypothesis IV at the end of *chapter 4*.

In conclusion, research in this field draws the picture that the induction of mental images can modulate certain behavioural aspects.

### **3.3 Merging mental imagery and food intake behaviour**

Humans make up to 200 food-related decisions on a daily basis. Individuals are mostly unaware of those decisions, as to when we decide to start eating enters our minds. The term “mindless eating”, referring to the nonhomeostatic determinants of food consumption, gained popularity over the last years by describing food intake behaviour through mainly unconscious processes. Therefore, describing alterations of the consumed amount of foods through packaging sizes, meal and plate sizes, lighting and other external sensory cues (colour, visual presentation), or distractional factors (like watching TV during dinner time), are pivotal [WANSINK and CHENEY, 2005 ; WANSINK and KIM, 2005 ; WANSINK, 2010 ; MARCHIORI *et al.*, 2012].

Nonhomeostatic determinants involve cerebral architecture that lead to modification of food consumption. In this context, mental imagery can be seen as an inner representation of the autobiographical experience, and therefore serves as nexus in understanding food intake, because of the intrinsic similarities to visual and memory processing.

Just thinking about various foods activates the salivation processes. Salivation in precephalic and cephalic phases is important for taste transduction and can be induced through environmental cues such as smell or sight of food. Additionally to mental imagery it can be mediated through other top-down stimulation such as labelling [DADDS *et al.*, 1997 ; SPENCE, 2011].

Combining the insights of psychological research on craving periods, which can be influenced by mental imagery in both directions, with insights from sports sciences, where repetitive mental imagery tasks enhance motor skills, becomes the question in the

thesis: How does repetitive mental imagery of food intake influence subsequent food consumption?

### 3.3.1 “Thought for food” Morwedge et al. – findings and implications

*“An intriguing finding suggesting that mental representations of consuming a particular food item may be sufficient to trigger satiety in the absence of actually eating the food item. The paper highlights the importance of higher-order cortical brain sites in regulating the relative incentive value of particular food items” [KENNY, 2011].*

Specifically thinking about eating M&M’s (in a repetitive manner) reduced subsequent M&M intake in participants during a subsequent taste-test compared to participants performing a different mental imagery task (control task) prior to the taste-test. This finding is in line with the findings of *Epstein and colleagues* where actual tasting of the food that would subsequently be consumed led to a decrease in intake [EPSTEIN *et al.*, 2009].

The sensory-specificity for the observed effect could be detected stating that the effect only prevails when the imagined and the consumed food are identical. Decreased motivation (measured through a reinforcement game) was observed while palatability of the food (liking) stayed the same before and after the intervention. This finding let the authors conclude that the decrease of motivation was the result of habituation effects describing the phenomenon. No parameters for satiety and fullness were assessed [MOREWEDGE *et al.*, 2010].

These findings implicate that

- i) mental imagery can reduce short-term subsequent food intake in a laboratory setting
- ii) the proposed mechanism through “habituation” to the food is due to decreased “wanting”, not decreased “liking” of the food

### 3.3.1.1 Habituation

Sensitizing and habituation are two opposed mechanisms describing the response to a stimulus reinforcer, when this reinforcer is presented repeatedly and over a longer period of time. The so-called dual-process theory of response habituation is a form of learning mechanism, also termed the “simplest way of learning” [GROVES and THOMPSON, 1970].

Thus, habituation is defined as a behavioural response decrement that results from repeated stimulation without the involvement of sensory adaptation, sensory fatigue or motor fatigue. Always accompanied by a dishabituation effect describing an immediate recovery of the habituated response to the original response. Habituation allows animals to filter out irrelevant stimuli and focus selectively on more important stimuli. It mainly acts centrally rather than in primary sensory afferents [RANKIN *et al.*, 2009].

In neuroscience, several different characteristics underlying habituation are discussed. Basically the differentiation between long-term and short-term habituation can be made. Long-term habituation refers to the long-term duration of effects of training necessary for changes in protein-synthesis with persisting aspects of habituation over a longer period in time as demonstrated to acoustic signalling for example [MASCHKE *et al.*, 2000].

Short-term habituation on the other hand is a temporary decrement of response due to repeated application of a stimulus.

Additionally, sensory-specific satiety is a form of habituation responding to a specific food without showing dishabituation effects [HAVERMANS, 2012b].

The terms sensory-specific satiety and habituation were used interchangeably in the paper of *Morewedge et al.* describing the effect of mental imagery on food consumption [HAVERMANS, 2011].

Whether sensory-specific satiety as manifestation of habituation or some different process optimally describes the phenomenon seems controversial.



### ***3.3.1.2 Appetite parameters***

The absence of measuring parameters for appetite is noticeable and probably reflects the “non-nutritional” background of the author of the paper. The subjective feeling of hunger and fullness has been described elsewhere in the text. Important to mention here is that the feeling of hunger and fullness is highly subjective, and therefore influenced by a vast variety of parameters [HARROLD *et al.*, 2012].

Arguing that hunger and fullness feelings underlie an overlapping of homeostatic and nonhomeostatic regulatory mechanisms, both feelings may not entirely reflect metabolic need. The question of reliability of appetite parameters should be posed in this context. What does subjective hunger and fullness actually reflect ?

## **4 Materials and methods**

The conducted study design is a product of various methodological considerations, reflecting the fact that only a few studies were conducted stressing the thesis' questioning, the study design is oriented heavily on *Morewedge et al.*, 2010 with two major differences, which will be carved out during the description of the study. Nevertheless, the presented experiment is more than a replicate study [MOREWEDGE *et al.*, 2010].

### **4.1 Participants**

The pre-recruitment phase of the participants was conducted from mid-March 2012 to the beginning of the study on the 19<sup>th</sup> of April. Mainly undergraduate students of nutritional sciences from the University of Vienna participated in the study and were enlisted through announcements in different study courses; notifications on diverse blackboards; advertising in the official student forum of nutritional science and via social networks. No monetary or other incentives were provided for the participants.

Furthermore, on the actual days of the study, additional participants were recruited by the experimenters. Leaflets were handed out throughout the course of the study.

### **4.2 Experimenters**

The author of the thesis conducted the study and was assisted by two undergraduate students of nutritional sciences. Both undergraduate students assisted the author by transmitting accumulated data into the computer during and after the study procedure.

### **4.3 Site of implementation**

The study was implemented at the facilities of sensory research at the University of Vienna, Department of Nutritional Sciences, 1090 Vienna, Althanstraße 14, UZA II, Room 2F549. The sensory laboratory is divided into 10 booths for testing. Participants

were separated from one another, lighting conditions were controlled and standardized throughout the 4 days of the study.

The preparation room next door was used for filling and weighing bowls with gummy bears and to document the participants' data. After the experiment, every participant was debriefed by the author to provide qualitative feedback about the study procedure, problems and thoughts about the research topic.

## **4.4 Study design**

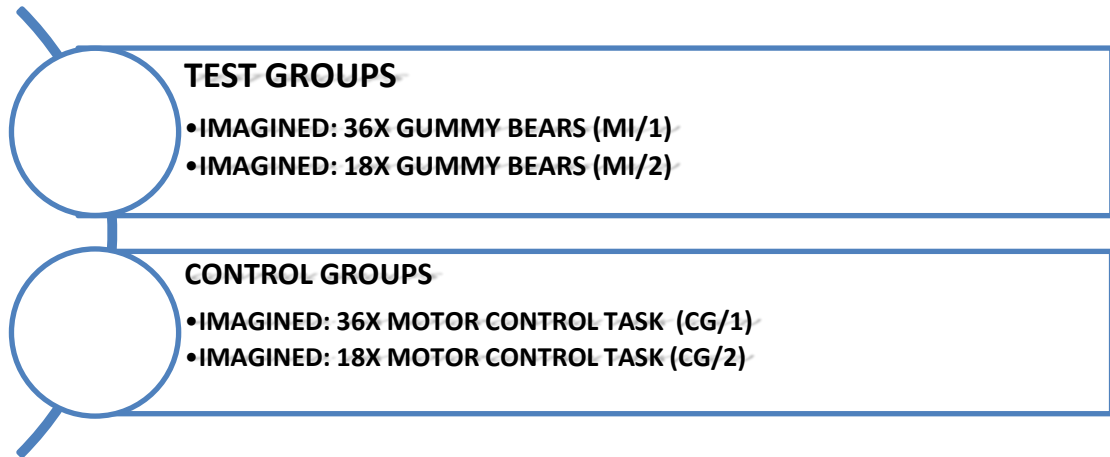
The study is designed as a camouflaged acceptance test, which means that participants thought to participate in a regular acceptance test for gummy bears, while the actual interest of the study was to assess the influence of different interventions on the consumption of gummy bears during the acceptance test. This gives it the experimental character.

In fact, the qualitative outcome of the acceptance test is not of interest for the thesis, however, the actual consumption of each participant while testing the gummy bears in relation to the preceding intervention is important. Additionally, various other parameters were assessed through questionnaires which form the basis for the sub hypotheses stated at the end of *chapter 4*.

### **4.4.1 Procedure**

Participants were divided into four different intervention groups (randomly assigned via lottery). Within each group every participant had to conduct a different mental imagery task as stated in *figure 12*.

*Figure 12: Intervention model*



Prior to the intervention, every participant completed the first questionnaire (Q1).

#### ***4.4.1.1 Imagery task in the test groups***

Participants in MI/1 and MI/2 had to imagine eating gummy bears with a different set of repetitions. The amount of repetitions (36 in MI/1 and 18 in MI/2) was calculated by assessing the amount of gummy bears in one 175 g package of “*Haribo Saftbären*”. One package contains approximately 72 gummy bears. Split in half for MI/1 and divided by four for MI/2 resulted in listed number of repetitions. In other words: participants either imagined eating half or a fourth of a package of gummy bears with the focus to bring the eating process of eating gummy bears vividly in front of the mind’s eye by consciously inducing mental images.

Detailed instructions from the experimenter with the following text was shown prior to the implementation of the task with the purpose to demonstrate the demanded task:

- i. *Position yourself on the chair as comfortable as possible. Keep your head straight.*
- ii. *Mental imagery (one imagery repetition should take at least 15 seconds.*
  - i. *Close your eyes.*
  - ii. *Think of a bowl filled with gummy bears placed in front of you.*
  - iii. *Grab one gummy bear, look at it and smell it.*
  - iv. *Utter and chew the gummy bear – feel the differences in texture and the salivation which increases after chewing for a while.*
  - v. *Swallow the chewed gummy bear.*
  - vi. *Feel the gummy bear slipping down your throat.*
- iii. *Pause for at least 5 seconds.*
- iv. *Replicate mental imagery for given repetition (MI/1 : 36 times ; MI/2: 18 times).*

#### **4.4.1.2 Imagery task in control groups**

Participants belonging to group CG/1 or CG/2 had to imagine putting a 50 ¢ coin into a laundry machine, in each case with the same amount of repetitions. The task to imagine putting a coin into a laundry machine serves as control task while referring to an object that has about the same size as a gummy bear. Furthermore, the involvement of a motor-action (moving your fingers and arms), and the focus on the placement of the object to a specific site (placing the coin into the laundry machine), describes a task with overlapping modalities with similar cerebral activation, and therefore serves as a valid control task [MOREWEDGE *et al.*, 2010].

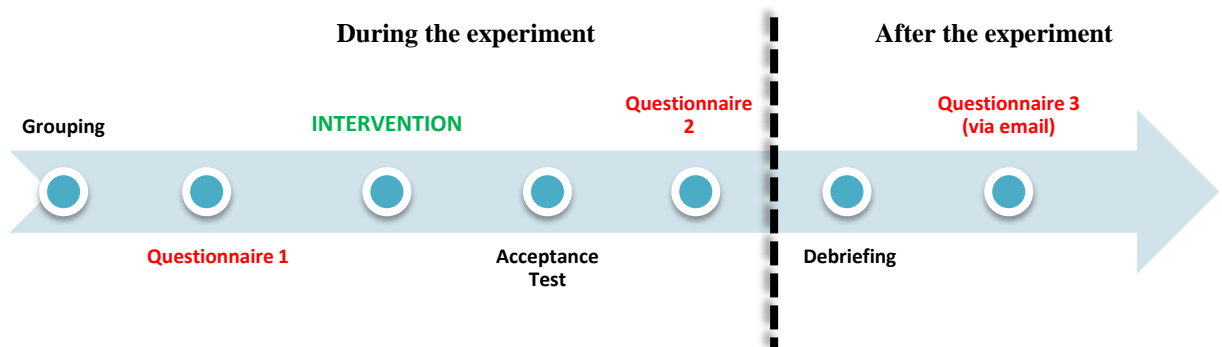
A detailed instruction from the experimenter with following text was shown prior to the implementation of the task:

- i. *Position yourself on the chair as comfortable as possible. Keep your head straight.*
- ii. *Mental imagery (one imagery repetition should take at least 15 seconds).*
  - i. *Close your eyes.*
  - ii. *Think of a bowl filled with 50 ¢ coins placed in front of you.*
  - iii. *Grab one 50 ¢ and take a look at it.*
  - iv. *Put the 50 ¢ coin in a laundry machine in front of you.*
  - v. *Press reset to get back the coin.*
- iii. *Pause for at least 5 seconds.*
- iv. *Replicate mental imagery for given repetition (CG/1 -> 36 times ; CG/2 -> 18 times).*

After the intervention every participant conducted an acceptance test in one isolated taste booth. The provided bowl was filled with 60-80g of gummy bears, measured before and after the sensory test. All participants were told to feel free to eat as many gummy bears as they liked while conducting the test.

At the end of the acceptance test, every participant completed another questionnaire (Q2) and was debriefed in the preparation room. A third questionnaire (Q3) was sent via email, which was filled out and returned on the consecutive days after the experiment.

Figure 13: Experimental procedure



#### 4.4.2 Food Choice

The choice of the right food for the experiment had to involve following criteria:

- *Broad acceptance by participants:* Firstly to bring as many participants into the laboratory, and secondly to minimize acceptance of the product as influencing factor for total gummy bear consumption during the acceptance test.
- *Quasi mono product, no complex meal:* From a theoretical standpoint, this criterion is necessary because a stimulus-specific effect was detected by *Morewedge et al. [MOREWEDGE et al., 2010]*
- *Matching size with the imagined object in the control task:* Sizes of the imagined objects (food and control object) should possess approximately the same size for equal representation.

*Morewedge et al.* chose M&Ms matching the above mentioned criteria .

The author of the thesis chose “gummy bears” matching the above mentioned criteria as well. Additionally, the bear-like shape characteristic serves as object which is easy to imagine, hence the task to imagine gummy bears was rather uncomplicated and easy to invoke by the participants.

From a practical standpoint, the ready purchase (in a regular supermarket), plus the fact that no preparation was needed were additional reasons for choosing this product.

## 4.5 Measurements

### 4.5.1 Questionnaires

#### 4.5.1.1 *Assessment of motivation to eat*

The visual analogue scale (VAS) is used to measure subjective feelings or attitudes by indicating a mark on a 120 millimetre scale with two endpoints representing two contradictory statements. The VAS line was adopted in length (regular VAS scales are 100mm in length) and for practical reasons (number of anchor points). Furthermore, the lines were extended beyond the maximum anchor points. Hunger (four anchor points) and fullness scores (four anchor points) were measured with the VAS before and after the acceptance test in Q1 and Q2 (see appendix).

Depending on the tendency towards one of the two extreme statements, the mark on the line indicates the trend for each statement. Hunger and fullness serve as hypothetical constructs of appetite which are used to conceptualize our perception of sensation or motivations to seek food. In the assessment of pain in pain research, VAS scales serve as golden standard as well as in nutritional sciences to measure appetite parameters [FLINT *et al.*, 2000 ; STUBBS *et al.*, 2000].

#### 4.5.1.2 *Assessment of mental imagery performance*

To assess the vividness of mental imagery, participants were given adapted German versions of the vividness of visual imagery questionnaire (VVIQ) from Marks and colleagues [MARKS, 1973] and the individual difference questionnaire (IDQ) [PAIVIO and HARSHMAN, 1983].

The questionnaires (see appendix) were sent to the participants via email after the study and were filled out and returned within two weeks.

The VVIQ asks participants to imagine and to rate four different scenes by means of the vividness of four different aspects of these scenes on the 5-point Likert scale, ranging from 1 (*No picture at all; you merely know that you are thinking about the object*) to 5



(*Perfectly clear; as vivid as normal vision*). Furthermore, the VVIQ serves as a validated tool to assess individual mental imagery. Correlations between VVIQ scores and brain activity during mental imagery were reported in fMRI studies which reflect the subjective strength of mental imagery, especially in early visual cortex [CUI *et al.*, 2007].

The IDQ consists of 13 statements describing the everyday preference for using visual mental imagery. Hereby, participants rate their agreement to each on the 5-point Likert scale ranging from 1 (*complete agreement*) to 5 (*complete disagreement*). The questionnaire assesses the individual preference for usage of mental images in everyday life.

#### ***4.5.1.3 Assessment of restrained eating***

Participants were given an adapted German restrained eaters scale to stratify participants and test whether or not restrained eating influences possible mental imagery effects. The validity of the German version is discussed elsewhere [DINKEL *et al.*, 2005]

#### ***4.5.1.4 Assessment of consumer acceptance of the product***

The consumer acceptance test is constructed to assess different attributes of the gummy bears on the basis of a 9 – point Likert scale consisting of verbal categories (anchor points). Different sensory characteristics like visual, taste and tactile properties were assessed. Taste (taste of different coloured gummy bears), visual (e.g. size, colour intensity) and haptic characteristics (stickiness, firmness, mouth feeling while chewing) were assessed.

Important to note here is that the data aggregated through the test did not play any further role for the thesis' hypothesis, it only fulfilled the role to camouflage and bring the participants into the laboratory. Additionally, liking of gummy bears (three anchor points) were measured with the VAS before and after the acceptance test in Q1 and Q2 and during the acceptance test (overall acceptance of the product).

Both scales (VAS and overall acceptance of the product) contained the same anchor points, splitting the scales into three equal parts:

- not very much
- either...or
- very much

The term acceptance test and taste-test will be used interchangeably in the text.

#### **4.5.2 Further measurements**

To assess the amount of consumed gummy bears, a standard scale with two decimal places was used to measure the weight of the bowl filled with gummy bears before the acceptance test. When finished, the experimenters consecutively measured the weight of the bowl and calculated the difference of the two values resulting in the consumed amount of gummy bears in grams.

### **4.6 Data**

The aggregated data was evaluated and computed with *IBM SPSS Statistics 20*. Analysis of variances, regression analysis and principal component analysis was used in data exploration. Diagrams and charts were designed with *Microsoft Excel 2010*.

### **4.7 Problems**

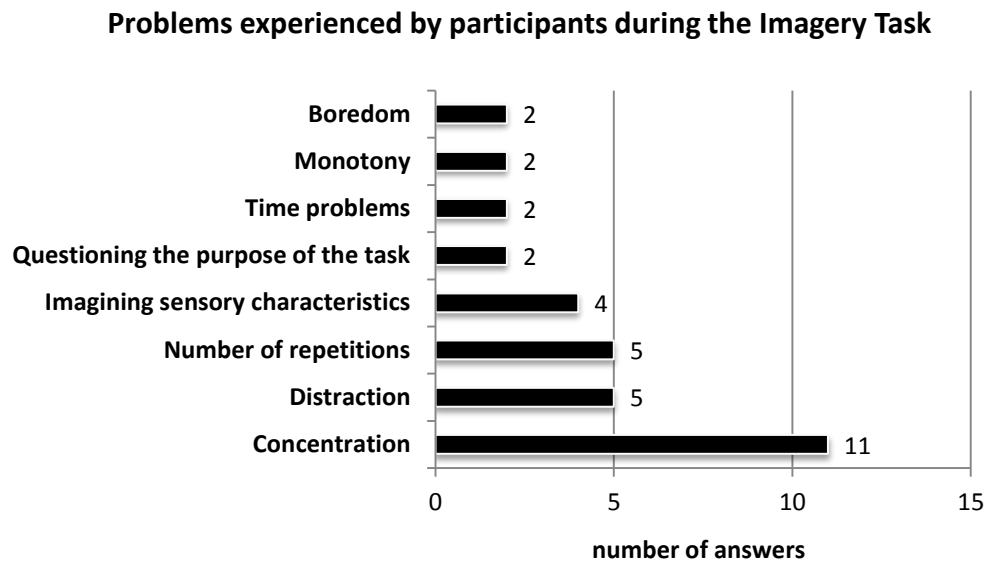
#### **4.7.1 Problems for the participants during the experiment**

Following problems emerged for the participants. They were reported orally to the experimenter after the experiment:

- The ability to concentrate and focus on the task while performing the imagery tasks dropped after several repetitions and made it difficult to perform consistently (especially in the 36 repetition groups).
- Comprehension problems with the instructions of the imagery tasks

Further Problems which emerged for the participants while performing the repetitive tasks were prompted in Q2, clustered in *figure 14*.

*Figure 14: Problems experienced by participants during the imagery Task*



#### **4.7.2 Problems for the experimenters**

Following problems emerged for the experimenters during the implementation of the study:

- When all the taste booths were occupied (max. eight participants at a time), the conduct of a fluent procedure was interrupted from time to time, especially when the time of the debriefing was extended because of enduring discussions about the thesis topic.

- Timing problems occurred because the distance between taste booths and the preparation room which were separated by two doors, and so from time to time the experimenters could not hear the participants when they were ready to proceed with the experiment.
- It was rather difficult to motivate students to come to the laboratory and participate.
- The third questionnaire (Q3) which was sent via email was not sent back by every participant. Five participants failed to send it back, though several reminder emails were sent following the day of participation. The response rate for the third questionnaire was : 95,1%.

#### 4.7.3 Conceptual problems

In the acceptance test five different colours were described, whereas six different colours were present in the sample of gummy bears. Two different nuances of red existed among the gummy bears which was not known to the experimenter at the time when the questionnaires were designed.

### 4.8 Sub-Hypotheses

Following additional sub-hypotheses were formulated prior to the study:

#### **Sub-hypothesis I:**

*Does the status of “hunger” effect the amount of gummy bears consumed?*

---

H0 = hunger scores do not effect gummy bear consumption

H1 = hunger scores do effect gummy bear consumption

---

**Sub-hypothesis II:**

*Does the status of “fullness” effect the amount of gummy bears consumed?*

---

H0 = fullness scores do not effect gummy bear consumption

H1 = fullness scores do effect gummy bear consumption

---

**Sub-hypothesis III:**

*Does the point in time of the last consumed meal have an influence on gummy bear consumption?*

---

H0 = point in time of last consumed meal does not affect gummy bear consumption

H1 = point in time of last consumed meal affects gummy bear consumption

---

**Sub-hypothesis IV:**

*Do VVIQ scores influence subsequent gummy bear consumption in participants performing the mental imagery task?*

---

H0 = VVIQ scores do not affect gummy bear consumption in MI groups

H1 = VVIQ scores does affect gummy bear consumption in MI groups

---

**Sub-hypothesis V:**

*Does restrained eating influence subsequent gummy bear consumption in participants performing the mental imagery task?*

---

H0 = RS scores do not affect gummy bear consumption in MI groups

H1 = RS scores do affect gummy bear consumption in MI groups

---

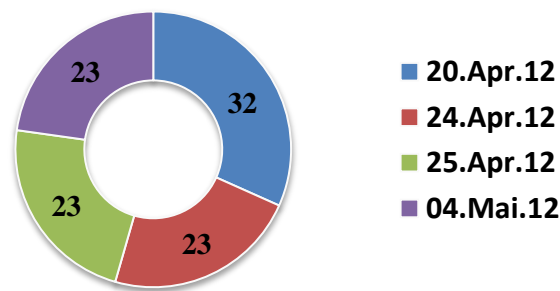
## 5 Results and discussion

### 5.1 Descriptive statistics

#### 5.1.1 Days of conduction, sample size and criteria of exclusion

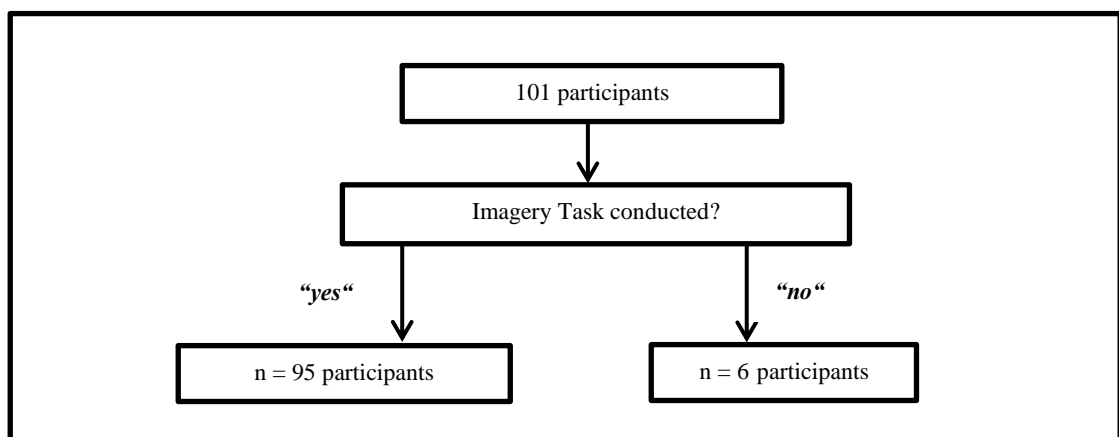
The experiment was conducted on 4 different days at the end of April and beginning of May during the summer term 2012. *Figure 15* represents the number of participants coming into the sensory laboratory each day. Important to note is, that on the first two days of conduction participants mainly attended the experiment who signed in beforehand, while on the last two days the majority of people were recruited directly at the facility buildings of the university.

*Figure 15: Number of participants on days of testing*



In total, 101 participants attended the study. Six participants were excluded from the statistical analysis answering the control question (whether or not they conducted the postulated imagery task ) with "no". This control question was used to assess the reliability of the participants in terms of conducting the claimed intervention.

*Figure 16: Flow chart for exclusion criteria*

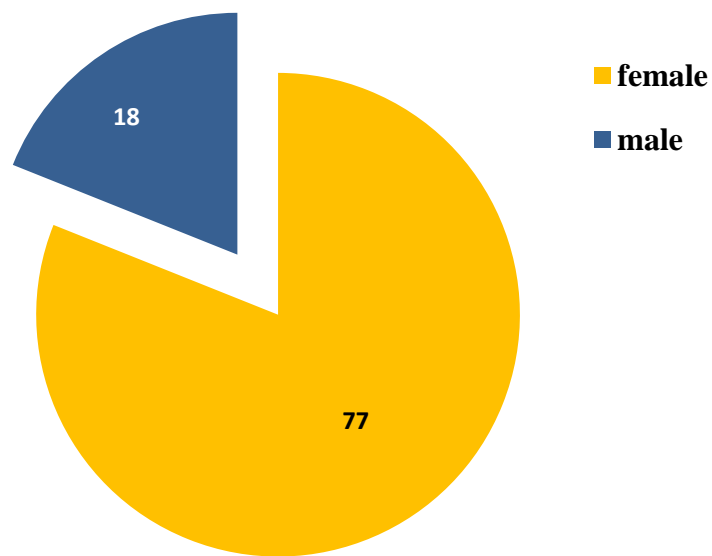


Hence, no further exclusion criteria were applied. The number of participants comprising in the statistical assessment is 95 ( $n = 95$ ).

### 5.1.2 Gender, age and Body Mass Index distribution

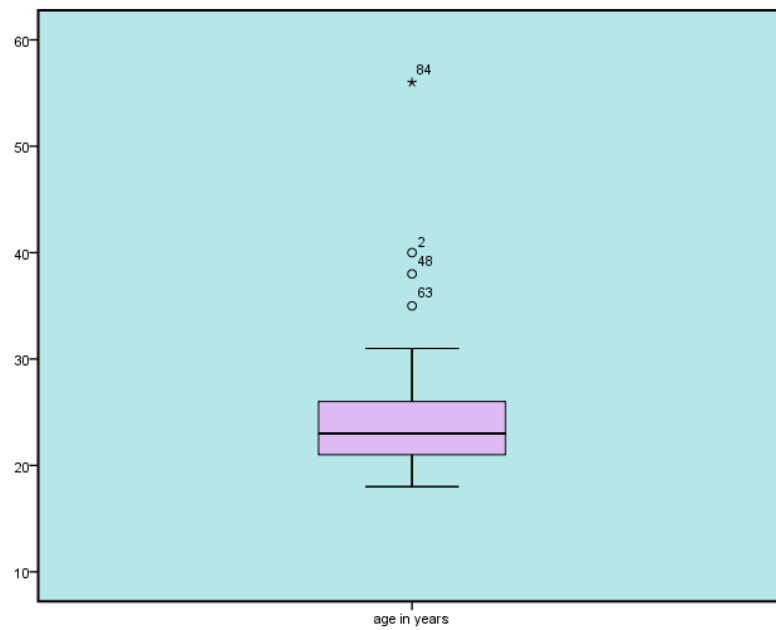
Participants from both sexes participated, whereas 77 female participants compared to 18 male participants conducted the experiment. Due to the fact that there was no actual target group described in the recruitment for the experiment, this distribution of the sexes was expected by the author, because of the high percentage of women studying nutritional sciences at the University of Vienna. Mainly students of this discipline were recruited.

*Figure 17: Gender Distribution among sample*

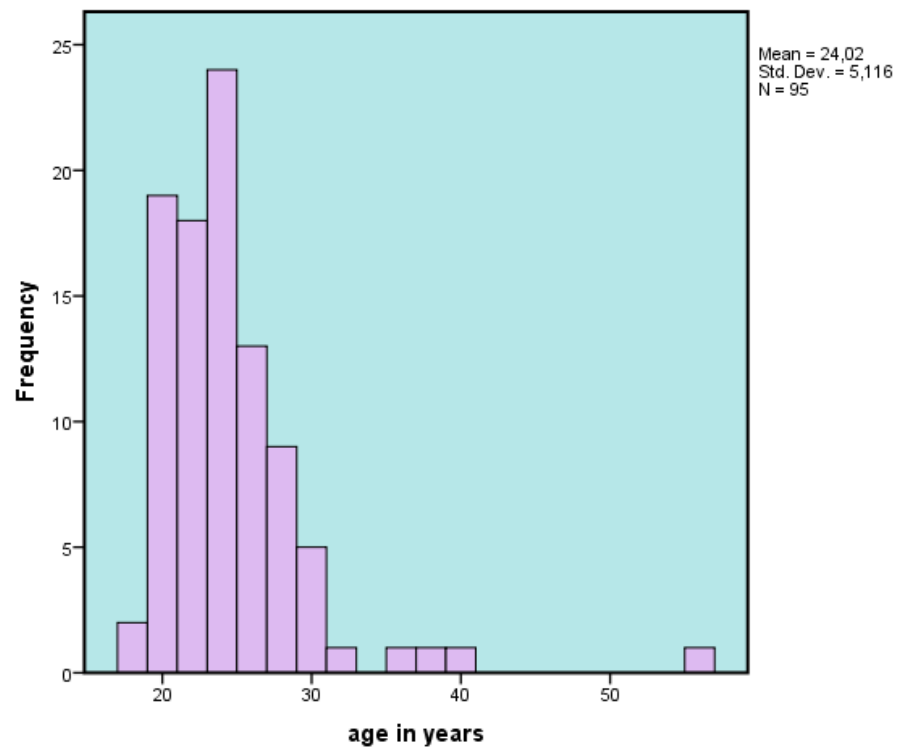


The mean age of the sample was  $24 \pm 5.1y$  (range from 18 to 56 y). 75% of the participants were younger than, or as old as 26 y and 50 % of the participants were between 21 and 26 y old (=IQR). The median age is 23y.

*Figure 18: Age in years - distribution among sample (Boxplot)*



*Figure 19: Age in years – distribution among sample (Histogram)*





Additionally, participants were asked for their height and weight. The Body Mass Index (BMI) was calculated via SPSS using the following formula:

$$\text{Weight (kg)} / (\text{height (m)} * \text{height (m)}) = \text{BMI (in kg/m}^2\text{)}$$

The BMI provides a valid tool for assessing body condition regarding body fat distribution and obesity, although underreporting of self-reported height and weight can be observed among normal weighing adults and especially obese adults [ELGAR *et al.*, 2005].

Keeping this in mind, plus the fact that any other assessment of the BMI concerning body composition (e.g. body fat measurements), would have been out of scope for the thesis. The distribution of the BMI among the participants is described as follows:

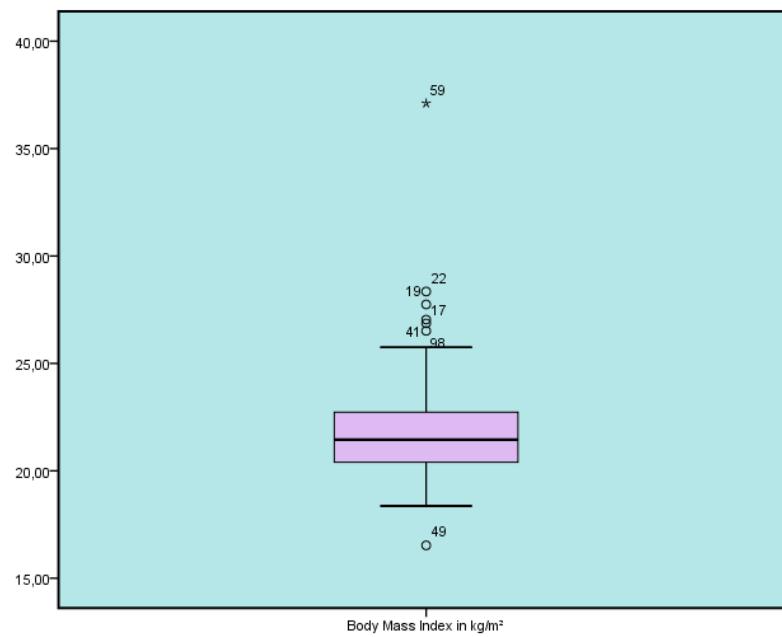
*Table 2: BMI distribution in kg/m<sup>2</sup>*

Mean	22.0
sd	2.7
Median	21.5
Minimum	16.5
Maximum	37.1

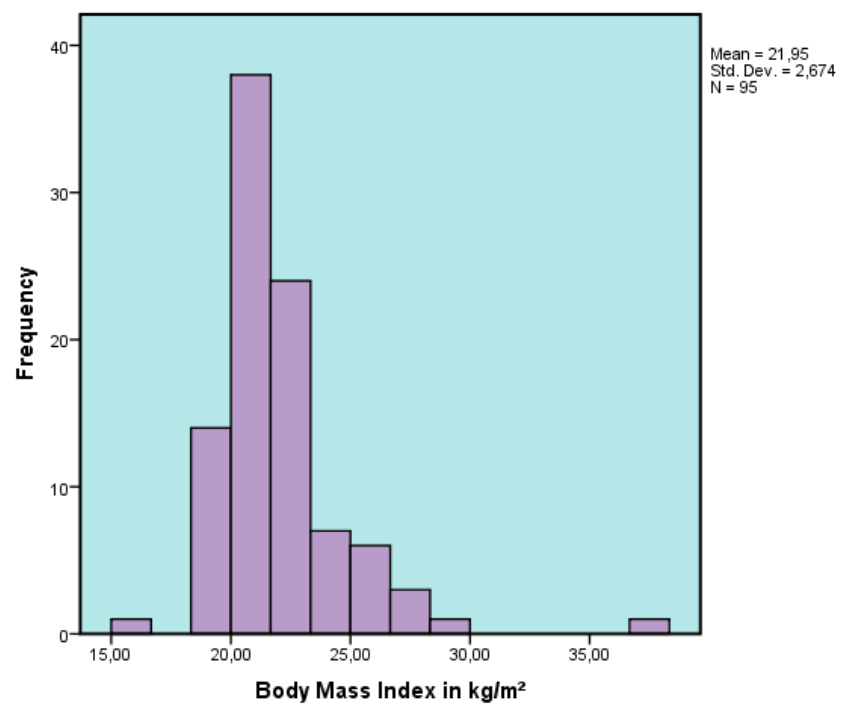
The data are not normally distributed (Kolmogorov-Smirnov ;  $p < 0.05$ ).

The box plot displays the range of values among the participants. 50% of the participants average between 20.4 - 22.9 kg/m<sup>2</sup> (IQR), while 50 % of the participants lie below 21.5 kg/m<sup>2</sup>, with a minimum of 16.5 kg/m<sup>2</sup>.

*Figure 20: BMI distribution among sample (Boxplot)*



*Figure 21: BMI distribution among sample (Histogram)*



According to the WHO BMI classification, the following distribution between male and female participants can be observed in the sample [WHO, 2003]:

*Table 3: BMI distribution according to WHO classification among sample between male and female participants*

	underweight	normal weight	overweight	obese	total
BMI in kg/m <sup>2</sup>	< 18.49	18.5 – 24.99	25 – 29.99	> 30	
female participants	2	69	6	0	77
male participants	0	13	4	1	18
total	2	82	10	1	95

89.6% of the female participants and 72.2% male participants belong to the normal weight group and in total 86.3 % belong to the normal weight group. Two female participants could be identified as underweight, whereas no male participants belong to that group. In total, ten participants were overweight and one male participant was obese.

## 5.2 Exploratory data analysis

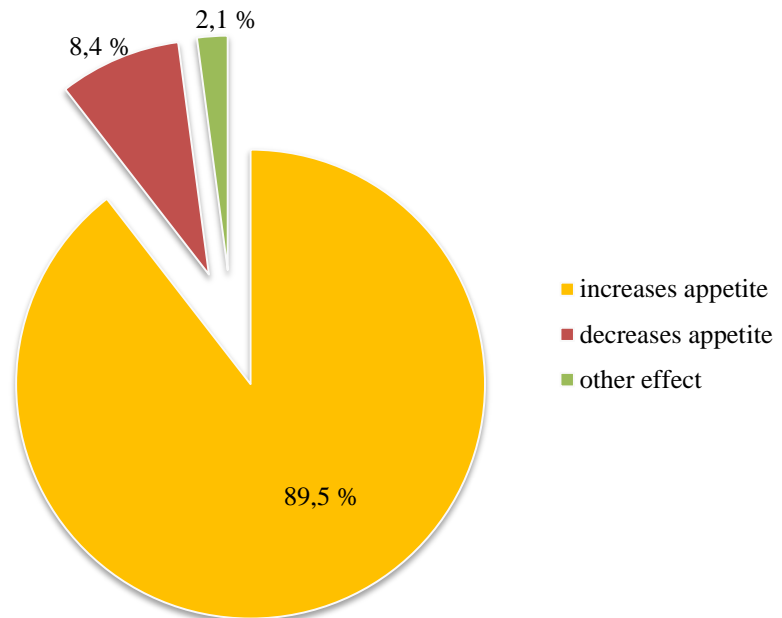
In the course of the text, the consolidation of mental imagery groups (MI-I/MI-II) and control groups (CG-I/CG-II) was made via SPSS. Those new formed groups will be suffixed by the term – *total*.

### 5.2.1 Expectation and intention

In addition to the assessment of gummy bear intake, participants' reasoning about possible effects of mental imagery on food intake was prompted in Q3. They were asked if they think that imagining food intake increases appetite, decreases appetite or has any other effect.

*Figure 22: Expected effect of mental imagery on appetite by participants*

#### What effect does thinking about food intake have on appetite ?



89.5% of the participants assumed that thinking about food intake increases appetite while 8.4 % thought it decreases appetite and 2.1 % of the participants thought it has a different effect. Moreover, participants even described their own theories about the

effect of mental imagery on food consumption for example that it may depend on the situation, the liking of the food or the current satiation.

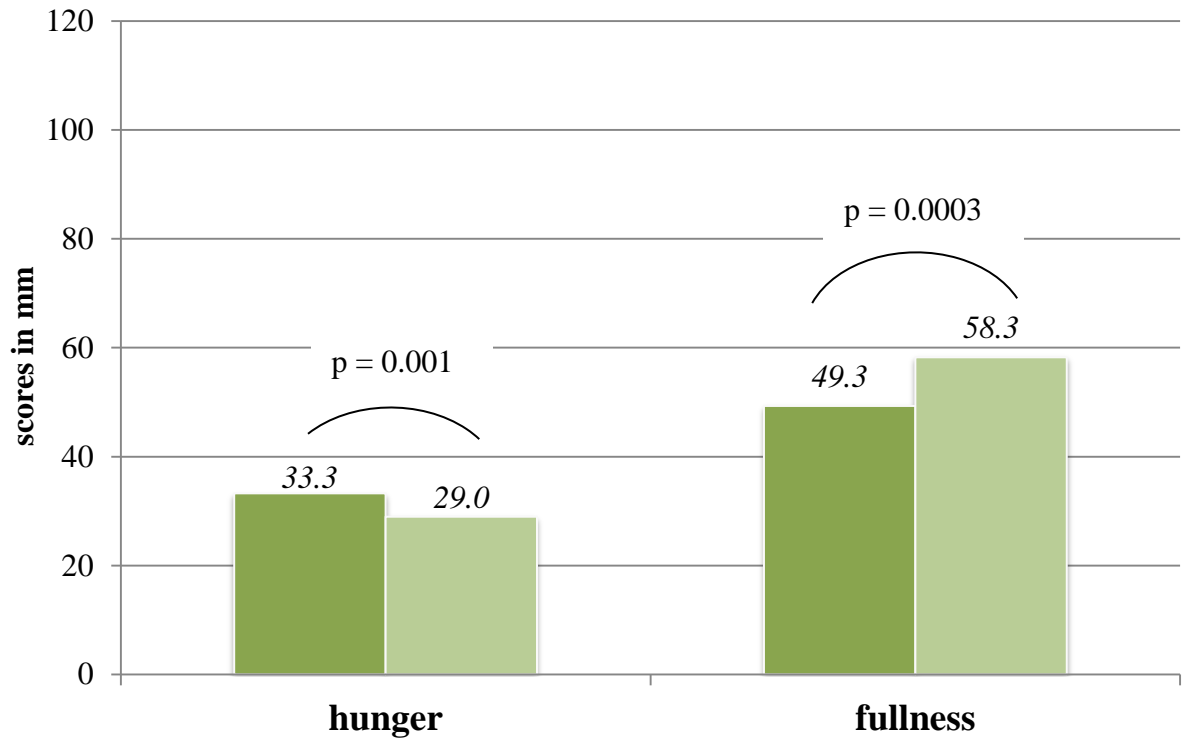
However, most of the participants assumed an appetite inducing effect. It is long known that the phenomenon cognitive bias can influence the outcome of studies. For example the perception of odours can be influenced by positively or negatively biased participants [DALTON *et al.*, 1997]. Therefore, controlled study designs with masked randomisations are developed to reduce these effects.

Cognitive biases in the form of expectations and intentions towards the tested effect may also affect the outcome of the formulated hypothesis I-III and is in itself a form of top-down modulation. Nevertheless it was shown that most participants did assume that thinking about food consumption does increase appetite and therefore expectation or intentional biases about the outcome of the intervention formulated in the hypothesis may be excluded.

### **5.2.2 Measuring subjective appetite parameters**

The assessment of appetite parameters was conducted throughout the experiment. Hunger and fullness states were prompted in Q1 (measurement I) and Q2 (measurement II) - at the beginning and at the end of the experiment. *Figure 23* displays that mean VAS scores for hunger significantly dropped from 33.3 to 29mm ( $p = 0.001$ ) whereas fullness ratings significantly increased from 49.3 to 58.3mm ( $p = 0.0003$ ) after participants conducted the taste-test.

*Figure 23: Pre-and post experimental mean hunger vs mean fullness VAS scores*



The distribution of the data in both scales differed according to relative standard deviation (RSD) which is a standard tool to describe the distribution of the data. In this case, the measurements of hunger VAS scores were less widely distributed than fullness VAS scores as seen in *table 4*.

*Table 4: Relative standard deviation of hunger/fullness VAS scores*

	measurement I	measurement II
RSD hunger	73.4%	81.7%
RSD fullness	53.1%	44.4%

This decline of hunger and the increase of fullness were expected by the author. To test if the decline of hunger and the increase of fullness differentiate among intervention groups, t-test comparisons between mental imagery groups and control groups were conducted and are illustrated in *table 5*.

*Table 5: T-test comparing the decline in hunger and increase of fullness*

	differences hunger scores	p-value	differences fullness scores	p-value
MI - I	8.21	0.781	9.42	0.707
CG - I	6.50		11.33	
MI - II	1.83	0.080	11.56	0.066
CG - II	0,46		3.58	
MI - total	5.08	0.696	10.47	0.367
CG - total	3.48		7.46	

No significant differences between intervention and control groups in the decline of hunger scores respectively increase in fullness scores were detected. This implies that different interventions did not have any influence on the subjective feeling of hunger and fullness felt by the participants and assessed by the VAS measurement.

There is the possibility that the used method may not be sensitive enough to detect these subtle changes in hunger and fullness scores evoked by the consumption of the gummy bears and the intervention. Technically the assessment of subjective hunger and fullness with the VAS serves as ultimate method, respectively being the golden standard for the evaluation of appetite, *among other electronic handheld devices* [GIBBONS *et al.*, 2011]. Assuming that the measurement reflects the actual parameters measured correctly, no differences between intervention and control groups could be detected. In other words, independent from the intervention conducted before the taste-test, participants did not differ in the decline in hunger and increase in fullness scores.

This has major implications for the interpretation of the findings depicted in *chapter 5.2.3.1* and raises the question if a difference in gummy bear consumption between control and mental imagery groups as formulated in hypotheses I – III may be without actual awareness of the participants subjective feeling of fullness and hunger.

### **5.2.3 Parameters influencing gummy bear consumption**

The main purpose of the study was to test the principal hypotheses respectively to analyse whether or not the intervention described in *chapter 4* influences subsequent gummy bear consumption. To unravel this questions, in this chapter a detailed

description of all possible influencing factors which were assessed via several different methods during and after the experiment will be interrogated with the purpose to draw a holistic picture.

### 5.2.3.1 Intervention groups

At first, data will be presented to give an overview about the mean consumption of gummy bears among different intervention groups as shown in *table 6*. The mean consumption among all participants was  $28.83 \pm 14.29\text{g}$ .

One gummy bears' weight is  $2.285 \pm 0.006\text{g}$  (mean weight of 3 repetitive measurements), so the average amount every participant consumed was approximately  $12.5 \pm 6.2$  gummy bears.

*Table 6: Mean grams of gummy bears consumed among intervention groups (table)*

Intervention Group	Mean intake of gummy bears in g	n
MI-I	$24.73 \pm 11.38$	24
MI-II	$24.53 \pm 10.88$	23
CG-I	$35.07 \pm 16.27$	24
CG-II	$30.81 \pm 15.57$	24
MI-total (MI-I + MI-II)	$24.63 \pm 11.02$	47
CG-total (CG-I + CG-II)	$32.94 \pm 15.94$	48

*Due to the study design, every participant must have eaten at least five gummy bears or at least parts of them while conducting the taste-test to evaluate taste differences among different coloured gummy bears. Three repetitive measurements for five gummy bears were conducted.*

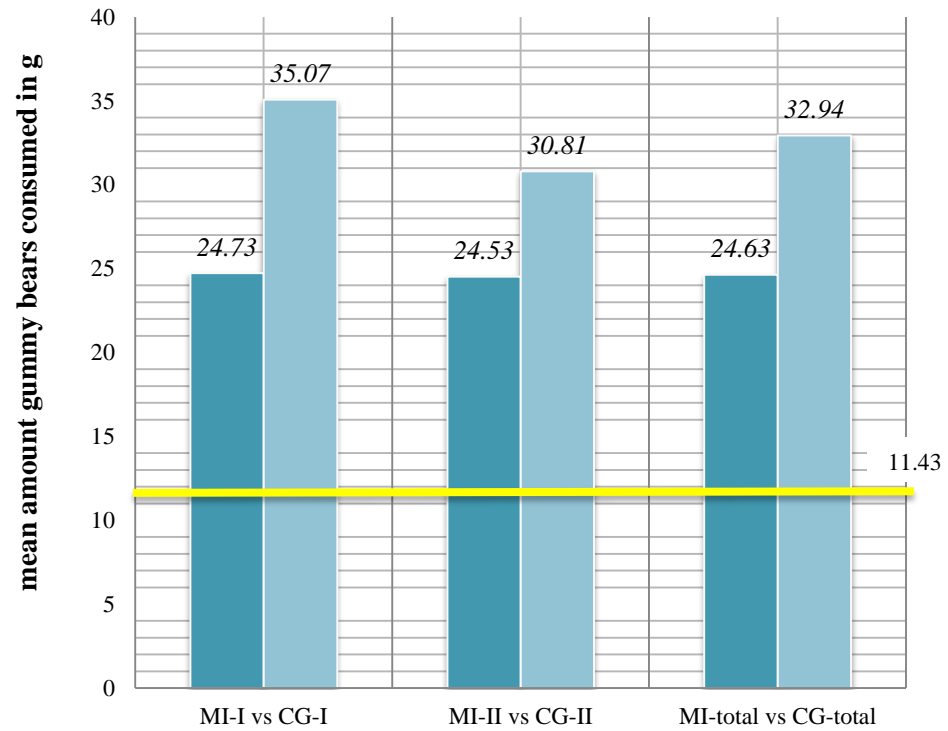
*Weight (5 gummy bears) = 11.43 g*

The yellow line in *figure 24* displays the threshold of 5 gummy bears (11.43 g) . Four participants were below this threshold (4.2% of the participants) belonging to either MI-I or MI-II. The possible explanation for the observed consumption below threshold could be that gummy bears were just partly consumed and put back into the bowl. For



instance, a person eating only half of every gummy bear with a different colour would consume only 5.72 g .

*Figure 24: Mean grams of gummy bears consumed among intervention groups*



To evaluate if observed differences in consumption among the intervention groups is statistically significant, unpaired t-tests were performed.

*Table 7: Comparison of mean values of gummy bear consumption between MI and CG groups*

group comparison	p-value
MI-I vs. CG-I	0.015*
MI-II vs. CG-II	0.117
MI-total vs. CG-total	0.004**
* $\alpha < 0.05$ / ** $\alpha < 0.01$	

Table 7 shows that above observed differences in gummy bear consumption differs significantly between MI-I/CG-I ( $p < 0.05$ ) and MI-total/CG-total ( $p < 0.01$ ) while no significance was found between MI-II/CG-II ( $p > 0.05$ ).

Looking further into detail, regression analysis for the two significant pairs provides useful information about the estimated parameters.

*Table 8: regression analysis for MI-I/CG-I and MI-total/CG-total with amount of gummy bears as dependent variable*

Parameter Estimates						
<i>dependant variable: amount of gummy bears consumed in g</i>						
Parameter	B	Std. Error	t	Sig.	95% Confidence Interval	
					Lower Bound	Upper Bound
Intercept	35.07	2.88	12.19	.000	29.28	40.87
[condition=MI-I]	-10.34	4.07	-2.54	.015	-18.53	-2.15
[condition=CG-I]	0 <sup>a</sup>					
<i>a. This parameter is set to zero because it is redundant.</i>						
Parameter	B	Std. Error	t	Sig.	95% Confidence Interval	
					Lower Bound	Upper Bound
Intercept	32.95	1.98	16.612	.000	29.01	36.88
[condition= MI-total]	-8.31	2.82	-2.95	.004	-13.91	-2.71
[condition= CG-total]	0 <sup>b</sup>					
<i>b. This parameter is set to zero because it is redundant.</i>						

Within a 95% confidence interval participants in the MI-I group consumed 10.34 g [-18.53g ; -2.15g] less gummy bears compared to participants in CG-I.

Regression equation for participants of the MI-I group compared to CG-I

$$\text{Consumption (MI-I)} = 35.073 - 10.338 [-18.53 ; -2.15]$$

In other words, participants thinking about the consumption of 36 gummy bears prior to the experiment ate approximately 4.5 gummy bears less than those performing the control task.

Within a 95% confidence interval participants in the MI-total group consumed 8.31 g [-13.91 ; -2.71] less gummy bears compared to participants in CG-total.

Regression equation for participants of the MI-total group compared to CG-total

$$\text{Consumption (MI-total)} = 32.95 - 8.31 [-13.91 ; -2.71]$$

In other words, participants thinking about the consumption of gummy bears either 18 or 36 times prior to the experiment ate 3.5 gummy bears less than those performing the control task either 18 or 36 times.

Although differences between participants in MI-II and CG-II were not significant, trends for a reduced consumption within participants in the MI-II group could be observed.

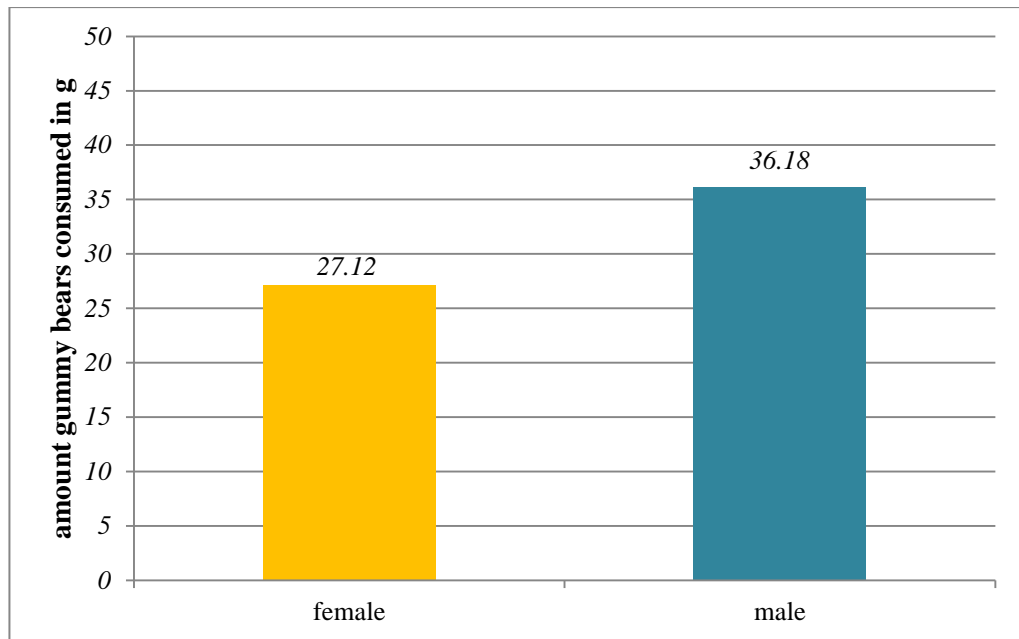
These findings are in line with the findings of *Morewedge and colleagues*, although differences in the number of repetitions of the mental imagery task were made [MOREWEDGE *et al.*, 2010].

#### **5.2.3.2 Gender**

As seen in *chapter 5.1.2*, the distribution between the sexes is in favour of female participants (4.3 female/male participant). Nevertheless, the possible influence of gender on the amount of gummy bears consumed has to be considered.

*Figure 25* shows the mean values of total gummy bears consumed among both groups. Female participants consumed  $27.12 \pm 12.45$  g, male participants consumed  $36.18 \pm 19.10$ g.

*Figure 25: Mean gummy bear consumption among sexes*



To test whether the observed differences in gummy bear consumption between male and female participants is of relevancy, t-test between the two groups was conducted. The test reveals that there is no significant difference between the two groups ( $p = 0.069$  ;  $\alpha = 0.05$ ) although tendencies might be observable.

The percentage of male participants in MI-total was lower than the ratio among CG-total groups (14.95 % vs. 22.95%).

*Table 9: Mean gummy bear consumed among both sexes (Group comparison)*

sex	group	mean	sd	n	% of all participants
Female	MI I	24.17	10.17	21	87.5
	MI II	23.77	11.48	19	82.6
	MI-total	23.97	10.83	40	85.05
	CG I	31.11	10.84	17	70.8
	CG II	30.00	15.60	20	83.3
	CG-total	30.55	13.22	37	77.05
male	MI I	28.68	20.72	3	12.5
	MI II	28.15	7.53	4	17.4
	MI-total	28.42	14.13	7	14.95
	CG I	44.70	23.68	7	29.2
	CG II	34.92	17.04	4	16.7
	CG-total	39.81	20.36	11	22.95

Additionally, one-way ANOVA was conducted to test if differences within intervention groups among male and female consumption are above chance. No significant results were found as seen in *table 10*.

*Table 10: One-way ANOVA, differences in gummy bear consumption among sexes in different intervention groups*

intervention group		Sum of Squares	df	Mean Square	F	Sig.
MI-I	Between Groups	53.472	1	53.472	.402	.533
	Within Groups	2926.505	22	133.023		
	Total	2979.976	23			
MI-II	Between Groups	63.198	1	63.198	.522	.478
	Within Groups	2543.608	21	121.124		
	Total	2606.806	22			
CG-I	Between Groups	915.983	1	915.983	3.842	.063
	Within Groups	5245.622	22	238.437		
	Total	6161.605	23			
CG-II	Between Groups	80.803	1	80.803	.323	.575
	Within Groups	5495.933	22	249.815		
	Total	5576.736	23			
MI-total	Between Groups	115.082	1	115.082	.946	.336
	Within Groups	5472.168	45	121.604		
	Total	5587.249	46			
CG-total	Between Groups	959.263	1	959.263	4.013	.051
	Within Groups	10996.382	46	239.052		
	Total	11955.644	47			

Nevertheless, if male participants may generally consume more during taste-tests is still an open question because tendencies are observable. Possibly, the impact of restrained eating may provide an explanation. Participants who show restrained eating patterns may also restrain themselves from consuming a large amount of gummy bears during the taste-test.

54.5 % of female and 33.3 % of the male participants could be categorised as restrained eaters (assessed through median split of restrained eating scale scores; c.f *chapter 5.2.3.7*). However, the difference between the percentage of male and female restrained eaters is not significant ( $p = 0.107$  ;  $\alpha = 0.05$ ).

In summary, the small number of male participants may be the reason that observed differences in gummy bear consumption may not be significant in this sample, nevertheless the influence of gender differences does not provide enough explanatory power for the amount of gummy bears consumed during testing among all participants or in group comparison.

### 5.2.3.3 Liking gummy bears

To assess if acceptance or liking of the product influences subsequent gummy bear intake among all participants, regression analysis was conducted. Acceptance scores were measured with 9- point Likert scale and liking scores were measured with visual analogue scale (measured twice with Q1 and Q2; average of both scores was used for the analysis).

*Table 11: Regression analysis: gummy bear consumption (DV) with liking (VAS) and acceptance (likert scale) scores [95% confidence interval]*

	Correlation with gummy bear consumption	R -square	model significance	regression equation, gummy bear consumption in g
VAS	0.360	0.130	0.0003	consumption (gummy bears in g) = $10.67g + 0.23 [0.11 ; 0.35] * \text{VAS score}$
Acceptance test	0.305	0.093	0.003	consumption (gummy bears in g) = $13.53g + 2.42 [0.86 ; 3.97] * \text{Acceptance score}$

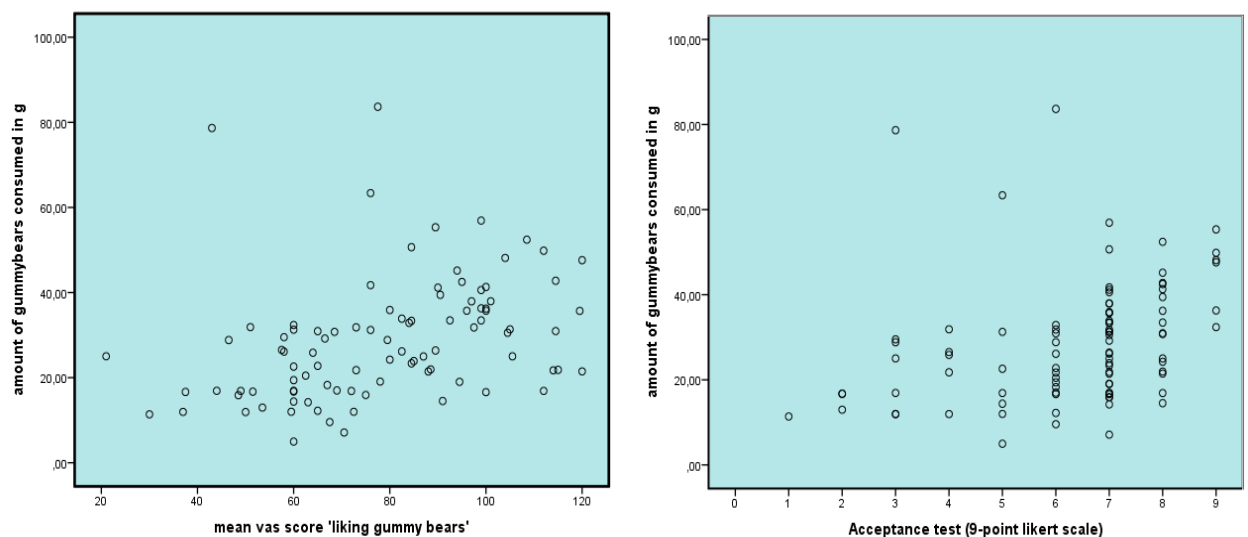
SPSS output for VAS scores in reference to gummy bear consumption shows that VAS scores correlate with  $r = 0.360$  while the model explains 0.130 of the total variance. The model is significant ( $p < 0.05$ ) and regression equation states that additionally to the

constant (y-intercept = 10.67g), every point scored for VAS liking increases the consumption of gummy bears by the factor of 0.23.

SPSS output for acceptance scores in reference to gummy bear consumption shows that scores correlate with  $r = 0.305$  while the model *explains 0.093* of the total variance. The model is significant ( $p < 0.05$ ) and regression equation states that additionally the constant (y-intercept = 13.53g), every point scored on the acceptance test increases the consumption of gummy bears by the factor of 2.42.

The scatterplot for both scales are shown in *figure 26*.

*Figure 26: Scatterplot correlation liking/acceptance scores with consumption*



*In conclusion, both scales detect a relation between liking or the acceptance of the product and the amount of gummy bears consumed during the test. Hence, only small amounts of the variance can be explained by these variables.*

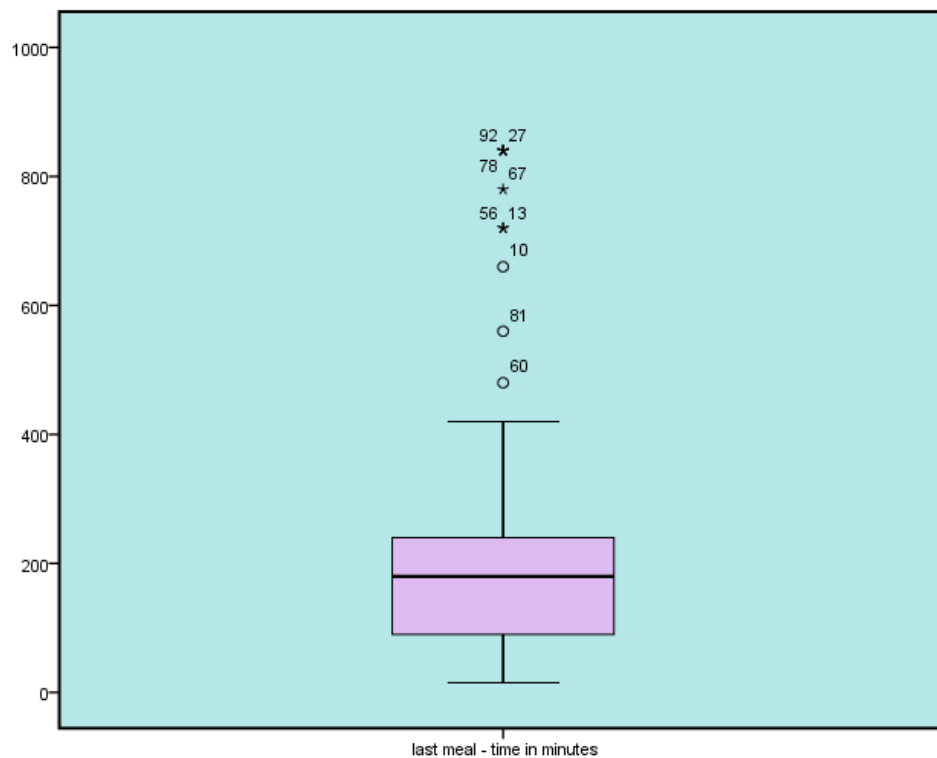
#### **5.2.3.4 Last food intake**

Furthermore participants were asked for the point in time of their last food intake prior to the experiment. In average, participants did not eat or have a meal  $213.5 \pm 200.7$  min

(range from 15 to 815 min): prior to the experiment, equivalent to 3.5 hours. For 50% of the participants the last meal was longer than 180 min (3h) ago while 10 % did not eat 512 min before the experiment (~ 8.5 h).

The point in time when the last meal was consumed correlated significantly with subjective hunger and fullness scores of the VAS, measured prior to the taste-test. Spearman's rho for subjective hunger scores ( $r = 0.396$ ) and fullness ( $r = -0.530$ ) were significant ( $\alpha < 0.05$ ). The correlation reflects the increase/decrease of appetite parameters due to abstinence of meal intake.

*Figure 31: Last meal prior to the experiment - distribution among participants*  
(Boxplot)



After splitting up participants along tertiles ( $Q_1$  vs  $Q_3$  ; lower 33% of last meal intake vs. upper 33% of last meal intake), t-test was conducted ( $\alpha = 0.05$ ) to analyse differences in gummy bear consumption between  $Q_1$  and  $Q_3$ .



*Table 12: Last meal mean values of gummy bears consumed by participants in Q1 and Q3*

	n	mean value gummy bears consumed	sd
Q <sub>1</sub> (≤90min)	19	29.8	15.5
Q <sub>3</sub> (>240min)	25	27.6	15.4

With  $p = 0.647$  no significant difference between participants in Q<sub>1</sub> and Q<sub>3</sub> in terms of gummy bear consumption could be observed.

Excluding participants not having food intake over 400 minutes prior to the experiment (11 participants) and splitting and comparing participants analogously as conducted above (Q<sub>1</sub> vs. Q<sub>3</sub>) likewise did not show any differences in the amount of gummy bears consumed ( $p > 0.05$ ).

In conclusion, the point in time when the last food was eaten does not affect the amount of gummy bears consumed during this experiment. Thus, sub-hypothesis III may therefore be rejected.

#### **5.2.3.5 Appetite parameters**

The VAS measurement of hunger and fullness will be interrogated in relation to gummy bear consumption in this chapter. As seen in *chapter 5.2.3.1*, the type of intervention (mental imagery or control task) does not influence increase or decrease of subjective hunger and increase of fullness rating. The question if participants' hunger/fullness scores prior to the experiment influenced subsequent gummy bear consumption will be reviewed.

Therefore, regression analysis was conducted and most important excerpts will be shown here.

**Fullness scores:**

As shown in *table 13*, fullness scores do not provide any further significant explanation for the amount of gummy bears consumed on a significance level of  $\alpha = 0.05$  among all participants or within intervention groups.

*Table 13: Regression analysis, fullness scores and gummy bear consumption*

ANOVA<sup>a</sup>

Model		Sum of Squares	df	Mean Square	F	p-value
All participants	Regression	51.131	1	51.131	.249	.619 <sup>b</sup>
	Residual	19130.951	93	205.709		
	Total	19182.082	94			
MI - I	Regression	36.428	1	36.428	.272	.607 <sup>b</sup>
	Residual	2943.548	22	133.798		
	Total	2979.976	23			
MI - II	Regression	43.530	1	43.530	.357	.557 <sup>b</sup>
	Residual	2563.276	21	122.061		
	Total	2606.806	22			
CG - I	Regression	34.657	1	34.657	.124	.728 <sup>b</sup>
	Residual	6126.949	22	278.498		
	Total	6161.605	23			
CG - II	Regression	57.254	1	57.254	.228	.638 <sup>b</sup>
	Residual	5519.482	22	250.886		
	Total	5576.736	23			
MI - total	Regression	79.663	1	79.663	.651	.424 <sup>b</sup>
	Residual	5507.587	45	122.391		
	Total	5587.249	46			
CG - total	Regression	119.073	1	119.073	.463	.500 <sup>b</sup>
	Residual	11836.571	46	257.317		

	Total	11955.644	47			
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a. Dependent Variable: amount of gummy bears consumed in g

b. Predictors: (Constant), fullness VAS score before experiment in mm

### Hunger scores:

Table 14 shows that results for hunger scores are analogous to those of fullness scores. In other words, neither among all participants, nor within intervention groups, hunger scores measured prior to the experiment provide any further significant explanation for subsequent gummy bear consumption (significance level ;  $\alpha = 0.05$ ).

*Table 14: Regression analysis, hunger scores and gummy bear consumption*

ANOVA<sup>a</sup>

Model		Sum of Squares	df	Mean Square	F	p-value
All Participants	Regression	251.148	1	251.148	1.234	.270 <sup>b</sup>
	Residual	18930.935	93	203.558		
	Total	19182.082	94			
MI -I	Regression	64.578	1	64.578	.487	.492 <sup>b</sup>
	Residual	2915.399	22	132.518		
	Total	2979.976	23			
MI-II	Regression	284.854	1	284.854	2.576	.123 <sup>b</sup>
	Residual	2321.952	21	110.569		
	Total	2606.806	22			
CG-I	Regression	505.288	1	505.288	1.965	.175 <sup>b</sup>
	Residual	5656.317	22	257.105		
	Total	6161.605	23			
CG-II	Regression	.155	1	.155	.001	.981 <sup>b</sup>
	Residual	5576.581	22	253.481		

	Total	5576.736	23			
MI-total	Regression	20.257	1	20.257	.164	.688 <sup>b</sup>
	Residual	5566.993	45	123.711		
	Total	5587.249	46			
CG-total	Regression	274.591	1	274.591	1.081	.304 <sup>b</sup>
	Residual	11681.054	46	253.936		
	Total	11955.644	47			

a. Dependent Variable: amount of gummy bears consumed in g

b. Predictors: (Constant), hunger VAS score before experiment in mm

All in all, the subjective rating of individual hunger or fullness measured with the VAS prior to the experiment did not influence the amount of gummy bears being consumed during the experiment within all intervention groups. Thus sub-hypotheses I & II may be rejected.

#### 5.2.3.6 BMI

As described in *chapter 5.1.2* according to WHO classification, 86.3 % of the participants belong to the normal weight group.

First, general testing whether BMI scores influence gummy bear consumption among all groups with regression analysis reveal that the BMI is not a significant predictor for subsequent gummy bear intake ( $p = 0.113$ ) within the setting of the present study.

Additionally, possible influence on gummy bear consumption among intervention groups was analysed and is shown in *table 15*:

*Table 15: Regression analysis, BMI scores and gummy bear consumption***ANOVA<sup>a</sup>**

Model		Sum of Squares	df	Mean Square	F	p-value
MI - I	Regression	497.084	1	497.084	4.404	.048 <sup>b</sup>
	Residual	2482.892	22	112.859		
	Total	2979.976	23			
MI - II	Regression	67.711	1	67.711	.560	.463 <sup>b</sup>
	Residual	2539.095	21	120.909		
	Total	2606.806	22			
CG - I	Regression	660.074	1	660.074	2.640	.118 <sup>b</sup>
	Residual	5501.532	22	250.070		
	Total	6161.605	23			
CG - II	Regression	1199.017	1	1199.017	6.026	.022 <sup>b</sup>
	Residual	4377.719	22	198.987		
	Total	5576.736	23			
Mi - total	Regression	453.421	1	453.421	3.974	.052 <sup>b</sup>
	Residual	5133.828	45	114.085		
	Total	5587.249	46			
CG - total	Regression	75.177	1	75.177	.291	.592 <sup>b</sup>
	Residual	11880.468	46	258.271		
	Total	11955.644	47			

a. Dependent Variable: amount of gummy bears consumed in g

b. Predictors: (Constant), Body Mass Index in kg/m<sup>2</sup>

Regression analysis reveals that among participants in MI-I and CG-II, the BMI provides significant explanation to predict the outcome of gummy bear consumption. To look further into detail, coefficients are used to predict the regression equation within those two groups.

*Table 16: Regression analysis, estimated values for BMI scores and gummy bear consumption*

**Coefficients<sup>a</sup>**

intervention group		Unstandardized Coefficients		Standardized Coefficients	t	p-value	95,0% Confidence Interval for B	
		B	Std. Error	Beta			Lower Bound	Upper Bound
MI-I	(Constant)	-18.045	20.499		-0.880	.388	-60.558	24.467
	Body Mass Index in kg/m <sup>2</sup>	1.910	.910	.408	2.099	.048	.023	3.797
CG-II	(Constant)	108.339	31.712		3.416	.002	42.573	174.106
	Body Mass Index in kg/m <sup>2</sup>	-3.629	1.478	-0.464	-2.455	.022	-6.695	-0.563

a. Dependent Variable: amount of gummy bears consumed in g

Table 18 shows that within the MI-I group, the constant term (amount of gummy bears consumed) is not significant within a 95% confidence interval and therefore the effect of the BMI in this group is negligible. On the other hand, for participants in CG-II following regression equation is valid to predict the amount of gummy bears consumed. The lower the BMI, the bigger is the amount of gummy bears consumed.

$$\text{Amount of gummy bears consumed in CG-II} = 108.339 - (3.629 * \text{BMI})$$

Among all other groups, the BMI provides no significant explanatory power for predicting gummy bear intake. It has to be concluded that BMI scores among participants do not have an effect on gummy bear consumption among all participants and additionally among most participants in different intervention groups. For participants within the CG-II group, the BMI serves as a valid predictor of gummy bear consumption which can be described by the above given regression equation.

### 5.2.3.7 Restrained eaters

Results from the restrained eating scale will be presented in this chapter to test sub-hypothesis formulated in *chapter 4*.

Before addressing the question of sub-hypothesis V, whether or not restrained eating influences gummy bear consumption among participants performing mental imagery (resp. MI-I, MI-II and MI-total), basic descriptive data will be presented beforehand.

#### *Internal consistency and reliability of the restrained eating scale*

To test internal consistency of the restrained eating scale, Cronbach's  $\alpha$  testing on reliability over the eight included variables was used.

$\text{Cronbach's } \alpha = 0.67$
------------------------------------

Cronbach's  $\alpha$  values between 0.7 and 0.8 are acceptable in terms of reliability and consistency among psychometric tests but the scores depend strongly on the amount of items in the questionnaire [FIELD, 2005]

Nevertheless, the Cronbachs'  $\alpha$  with 0.67 in presented study is rather low compared to previous studies investigating restrained eating scales (cf. Cronbachs  $\alpha = 0.83$ ) [DINKEL *et al.*, 2005].

#### *Restrained and non-restrained eaters*

In further steps, participants were divided via median split analogous to previous studies (the median serves as a cut-off point along the scores for the restrained eating scale over all variables). Two groups of participants were formed: "restrained eaters" (RE ; n=47) and "non-restrained eaters" (nRE ; n = 48) [DINKEL *et al.*, 2005].

Subsequently, t-tests were conducted to test on differences among groups in terms of gummy bears consumed. Among all participants, no difference in the amount of gummy

bears consumed between restrained and non-restrained eaters could be observed ( $p = 0.235$  ;  $\alpha = 0.05$ ).

*Table 17: Mean amount of gummy bears consumed stratified among intervention group and RE and nRE participants*

intervention group			n	mean	sd	p-value
MI -I	amount of gummy bears consumed in g	nRE	11	26.80	12.28	.424
		RE	13	22.98	10.74	
MI -II	amount of gummy bears consumed in g	nRE	11	25.99	10.67	.552
		RE	12	23.20	11.37	
CG-I	amount of gummy bears consumed in g	nRE	10	30.78	13.55	.287
		RE	14	38.14	17.96	
CG-II	amount of gummy bears consumed in g	nRE	15	36.65	16.61	.014
		RE	9	21.09	6.64	
MI-total	amount of gummy bears consumed in g	nRE	22	26.40	11.23	.310
		RE	25	23.08	10.83	
CG-total	amount of gummy bears consumed in g	nRE	25	34.30	15.44	.544
		RE	23	31.47	16.70	

$\alpha = 0,05$

Table 17 shows the mean values of gummy bear consumption among different intervention groups between participants of the RE and nRE group. It has to be said that in both MI groups no significant differences in consumption between RE and nRE participants could be observed (MI-I,  $p = 0.425$ ; MI-II,  $p = 0.552$ ). Likewise, no differences could be observed in participants in CG-I ( $p = 0.287$ ) and comparing differences between CG-total and MI-total in terms of gummy bear consumption among RE and nRE did not show any significant results, as well. On the other hand in CG-II,



non-restrained eaters ate significantly more gummy bears than restrained eaters ( $p = 0.014$ ).

These findings suggest that restrained eating, which is a form of cognitive control does not affect gummy bear intake neither among all participants, nor in sub-groups. In a laboratory setting, restrained eating may therefore not suppress the amount of consumed food. The formulated sub-hypothesis V may therefore be rejected.

#### 5.2.3.8 VVIQ and IDQ scores

Results from the VVIQ and IDQ will be presented in this chapter testing formulated sub-hypothesis IV from *chapter 4*.

The response rate for the VVIQ and IDQ questionnaire was 95.1% (five participants did not send back the form). At first, the questionnaire's reliabilities were determined with Cronbachs  $\alpha$  testing.

Cronbach's  $\alpha$  (IDQ) = .715

Cronbach's  $\alpha$  (VVIQ) = .934

For the IDQ scale, five items that decreased reliability of the questionnaire were removed due to reliability reasons. No items were excluded in the VVIQ. For remaining items, mean scores were computed and participants were split in three groups along tertiles in both questionnaires illustrated in *table 18*.

*Table 18: Frequency of participants among tertiles of mean scores in VVIQ and IDQ*

	q1	range (q1)	q2	range (q2)	q3	range (q3)
VVIQ	29	[0 ; 3.66]	32	[3.66 ; 4.11]	31	[4.11 ; 4.91]
IDQ	30	[0 ; 3.05]	38	[3.05 ; 3.54]	24	[3.54 ; 4.46]
total	59		70		55	

In further computation, participants of q1 were compared to participants in q3 in terms of mean amount of gummy bears consumed during the experiment regarding differences in the intervention groups while participants of the middle tertile (q2) were left out of the analysis. The downside of this approach is, that 1/3 of the participants get lost for the analysis (q2) but on the other hand groups belonging to the first and third tertile, respectively to both ends of the spectrum can be compared.

Additionally, Spearman's rho correlation between VVIQ and IDQ scores were determined to test whether scores in both questionnaires correlate with each other. Significant, weak correlation between both questionnaire scores were computed:  $r = 0.259$  ;  $p < 0.05$ .

### VVIQ

After grouping participants in high (q3) and low mean scores (q1), differences in the amount of gummy bears eaten were analysed.

*Table 19: One-way ANOVA; amount of gummy bears consumed among participants scoring in the first and third tertile (VVIQ)*

intervention group			n	mean	sd	p-value
MI-total	amount of gummy bears consumed in g	q1	12	28.90	11.85	.077
		q3	18	21.53	10.00	
CG-total	amount of gummy bears consumed in g	q1	17	31.76	16.37	.757
		q3	13	29.97	14.92	
MI-I	amount of gummy bears consumed in g	q1	6	33.13	11.25	.139
		q3	9	23.92	10.98	
MI-II	amount of gummy bears consumed in g	q1	6	24.68	11.81	.319
		q3	9	19.15	8.90	
CG-I	amount of	q1	8	37.59	20.78	.594

	gummy bears consumed in g	q3	7	32.14	17.35	
CG-II	amount of gummy bears consumed in g	q1	9	26.58	9.70	.884
		q3	6	27.44	12.61	

To keep in mind for the interpretation of the results, that due to the stratification into different intervention groups and additionally ignoring one third of the sample (q2), the sample being compared (q1&q3) becomes relatively small. Hence finding significant differences between the groups becomes rather difficult.

Although no significant differences in gummy bear consumption between any group can be found, tendencies can be observed. Interestingly in all MI-groups the amount of gummy bears consumed were higher among participants in q1 than among participants in q3 whereas in CG-II for instance, consumption was higher among participants in q3. Most sound differences in mean intake provides the comparison within MI - total group, between first and third tertile of VVIQ scores with a difference of  $7.37g \pm 10.9g$  ( $p > 0.05$ ) as seen in *table 23* above.

In conclusion, the influence of mental imagery which can be measured with the VVIQ scale could not show any significant differences in terms of gummy bear consumption although tendencies were observable. Therefore, sub-hypothesis IV may be rejected

### *IDQ*

For the sake of completeness, participants' scores for the IDQ scale are presented briefly.

*Table 20: One-way ANOVA; amount of gummy bears consumed among participants scoring in the first and third tertile (IDQ)*

intervention group		n	mean	sd	p-value
MI-total	amount of gummy bears consumed in g	q1	9	25.17	.567
		q3	14	22.82	

CG-total	amount of gummy bears consumed in g	q1	21	32.77	14.78	.198
		q3	10	25.78	11.37	
MI-I	amount of gummy bears consumed in g	q1	6	24.75	8.19	.901
		q3	6	23.98	12.35	
MI-II	amount of gummy bears consumed in g	q1	3	26.00	4.66	.627
		q3	8	21.95	13.27	
CG-I	amount of gummy bears consumed in g	q1	12	37.39	17.08	.117
		q3	4	22.06	10.52	
CG-II	amount of gummy bears consumed in g	q1	9	26.61	8.36	.758
		q3	6	28.27	12.15	

Likewise to VVIQ scores, there is to say that due to the reduction of the sample size, significant results were rather difficult to detect as shown in *table 20*. Hence, no significant differences in gummy bear consumption between q1 and q3 within different intervention groups were detected

#### 5.2.4 Main determinants for gummy bear consumption

As displayed in the statistical analysis, different factors contribute to the explanation of gummy bear consumption during the experiment.

The amount of gummy bears consumed does not depend on:

- State of appetite (hunger or fullness)
- Gender
- Last meal intake
- Restrained or non-restrained eating
- Vividness of mental images scores

Depends on:

- Liking or acceptance of gummy bears
- Intervention

In conclusion, the preference for gummy bears, respectively the liking or acceptance contributes significantly in the explanation of the consumed amount. This finding may be without surprise because who is more fond to gummy bears, may consume more during a taste-test.

More interestingly, different mental imagery interventions prior to the taste-test did affect the amount of consumed gummy bears. Those participants who performed an imagined intake of gummy bears ate significantly less than those performing the control task (in MI-total and MI-I groups).

Other factors like gender, hunger or fullness scores or last meal intake did not have significant effects on the consumed amount.

This finding may be even more intriguing when trying to explain the underlying mechanisms. It seems that the observable decrease in gummy bear consumption may be due to processes on a subconscious level. No differences in decline of hunger or increase of fullness scores could be detected between intervention groups. Additionally, most participants were assuming that the imagining task of eating gummy bears may lead to an increase of appetite. In fact, when talking to participants after the experiment, most of them were surprised to hear what the purpose and the hypotheses of the study were.

## 6 Conclusion and outlook

The famous quote which is ascribed to Apicius (said to be the author of the oldest roman cookbook) saying that: “*the first taste is always with the eyes*” may well be true especially in the context of presented thesis. It raises the question *what is it that exactly tastes for the first time*. A possible role for the mind’s eye may be indicated.

The evocation of mental images is a process of memory retrieval in the absence of external stimuli. It is said to be a part of our problem solving machinery and is involved in creative processes. Memory processing is complex and underlies diverse mechanisms in neural substrates. Among other nonhomeostatic regulation mechanisms, the memory of foods, respectively of what was eaten influences subsequent food intake. On the basis of this rationale, the performance of mental imagery before food is consumed could influence subsequent food intake. Indeed, a stimulus-specific decreasing effect of food intake provoked by prior mental imagery tasks could be observed [MOREWEDGE *et al.*, 2010].

In line with this finding, a study design with three main hypothesis was devised which raised the question if repetitive mental imagery tasks influence subsequent gummy bear intake during a taste test.

The null hypothesis for hypothesis I and III could be rejected. A significant decrease in gummy bear intake for groups imagining eating gummy bears (MI-total and MI-I) compared to matching control groups could be observed. No significant difference in gummy bear intake for MI-II group in comparison to their control group could be detected and therefore the null hypothesis for hypothesis II was accepted.

Moreover, following the rationale of the theoretical part of the thesis, additional sub-hypothesis were formulated and tested.

Neither hunger and fullness scores (sub-hypothesis I & II), nor the point in time when the last meal was eaten significantly influenced the amount of gummy bears consumed during the taste-test (sub-hypothesis III).

Liking of gummy bears on the other hand could predict the amount of consumed gummy bears during the taste-test, but was not formulated in any of the sub-hypothesis.

Furthermore, in mental imagery groups, differences in the vividness of mental images of participants were measured with the VVIQ. The vividness of mental images did not affect the observed decreasing effect of gummy bear intake in participants imagining gummy bear intake prior to the taste-test. Participants in the first tertile of VVIQ scores versus participants in the third tertile were compared (sub-hypothesis IV). Additionally, sub-hypothesis V tested if restrained eating habits influences mental imagery effects on gummy bear consumption. Participants categorised as non-restrained eaters compared to restrained eaters showed no difference in gummy bear intake in mental imagery groups.

The data also provided evidence, that the observed effect may be mediated on a subconscious level. Between mental imagery groups and control groups no significant difference in the decline of hunger scores and increase of fullness scores after the taste-test could be detected. It could be argued, that the differences in observed gummy bear intake should actually be reflected in the change in subjective appetite parameters, if the method is sensitive enough. Participants eating less may have not been aware why they ate less, because no differences in the decline of hunger scores and the increase of fullness scores could be detected. In addition, participants' reasoning about the possible effect of imagining eating gummy bears on subsequent intake is of interest. 89.5 % of the participants assumed that thinking about food or food related processes increases one's appetite. This shows that most of the participants assumed the opposite effect of what was demonstrated through the experiment. Thus, intentional or expectation biases can be ruled out.

The finding that the observed effect may be mediated through subconscious processing fits well into current nonhomeostatic food intake regulation mechanisms as described in *chapter 3.1.3*. The mental imagery tasks conducted by the participants may be *nothing but* some sort of cognitive control - the control and manipulation of thoughts.

Cognitive control in children for goal achieving purposes has been investigated since research findings of *Mischel and colleagues*. Putting children into a room (around the age of four years) with one marshmallow and telling them that they get a second

marshmallow if they can resist eating the one right in front of them for several minutes. Most children could not resist to eat the marshmallow in front of them while others could resist the temptation more easily [MISCHEL *et al.*, 1972].

[<https://www.youtube.com/watch?v=6EjJsPylEOY>]

May gratification also have changed in an obesogenic environment where people are surrounded by ever-stimulating food or other consumption-stimulating cues? Interestingly, by avoiding to look at the marshmallow in front of them, many children were successful in resisting to eat the first marshmallow, indicating a strong role for a visual component in cognitive control and ultimately influencing behavioural traits.



## 7 Summary

### *Background*

The consumption of food is determined by a variety of factors. The current metabolic state is reflected by various peripheral and central hormonal signals, representing a homeostatic regulation system. To know when, with whom, where, what and most importantly how much we eat is of major public health relevance in the face of a growing obesity epidemic on a national and worldwide scale.

A permanent utilisation of nonhomeostatic regulatory mechanisms comes into play by the obesogenic environment humans live in. Nonhomeostatic eating refers to food intake in the absence of metabolic demands, driven by conditioned or rewarding motifs.

Both systems have overriding power over each other (humans can eat without being metabolically hungry; and humans can refuse to eat when metabolically deprived when hunger striking). The so-called regulatory cross-talk can be observed on a micro and macro level, mediated through top-down or bottom-up signalling. It is known that top-down signalling can influence the way we eat, respectively how much we eat. Different cognitive, memory or learning mechanisms can influence human food intake.

Additionally to these factors, a role of mental imagery as a special form of memory processing is discussed in this context. It is known that the simulation of motor-action in sports science through the performance of repetitive imagery tasks can enhance motor skill performance. Other examples from disciplines like behavioural psychology could also detect the influential power of mental images. Translated to the discipline of nutritional sciences, the question if the evocation of mental images can influence eating behaviour is posed in this thesis.

### *Methods*

A camouflaged experimental design was used to make participants think of conducting a conventional taste-test with gummy bears while the actual assessment of the amount of eaten gummy bears was of interest. The weight of consumed gummy bears, self-reported appetite, hunger parameters and questionnaires about restrained eating and the

vividness and usage of mental images was assessed among 95 participants. Four intervention groups were formed. Two groups performed mental imagery about eating gummy bears (18 and 36 repetitions) and two groups performed a control task (imagining putting a 50 ¢ into a laundry machine) with same number of repetitions prior to the taste-test.

### *Results*

Major findings were that all participants in mental imagery groups ate significantly less compared to participants in all control groups ( $p = 0.004$ ). Likewise, participants in the 36 repetition groups ate significantly less gummy bears ( $p = 0.015$ ). Additionally, liking of the product could predict the amount of gummy bears eaten. No differences in the decline in subjective hunger and the increase of fullness scores between participants imaging eating gummy bears and participants in the control groups after the taste-test, could be observed.

### *Conclusion*

The imagination of eating gummy bears influences the amount of subsequent gummy bear consumption in a laboratory setting. The observed decrease in gummy bears eaten may be decoupled of subjective feelings of appetite and may be opposed to participants' reasoning about the possible impact of thinking about eating food and subsequent food intake. All in all, the results of the thesis provide two major implications. It elicits a role for mental imagery in nonhomeostatic food intake regulation and provides evidence that mental imagery and perception may be closer intertwined than assumed.

## 8 Zusammenfassung

### *Hintergrund*

Essen ist ein komplexes Thema, welches von vielen verschiedenen Faktoren abhängig ist bei dem immer die Person selbst, das Lebensmittel und die Situation in der sich ein Mensch befindet, im Mittelpunkt steht. Die zentralen Fragen, warum wir anfangen und aufhören zu essen und warum wir wieviel essen, sind von großem Interesse für die individuelle Gesundheit. Vor allem in Hinblick auf die ansteigende Prävalenz von Übergewichtigen in einem nationalen und internationalen Kontext spielen die Beantwortung dieser Fragen eine wichtige Rolle. Der menschliche Organismus hat zwei Systeme entwickelt, die miteinander interagieren und somit die Nahrungsaufnahme regulieren. Zum einen ein homöostatisches Kontrollsystem, das den metabolischen Zustand über zentral und peripher wirkende Signale steuert und somit einen metabolischen Impuls zur Nahrungsaufnahme geben kann. Zum anderen essen Menschen auch, wenn weder die metabolische Notwendigkeit, noch das subjektive Hungergefühl vorhanden sind. Dabei werden andere Motive bedient, die nicht einer Nährstoffaufnahme dienen, sondern vielmehr Belohnungsmechanismen oder andere Vorgänge in Gang setzen, die oftmals außerhalb unserer Wahrnehmung liegen. Hierbei haben Konditionierungs-, kognitive und Erinnerungsmechanismen einen beachtlichen Einfluss auf individuelles Essverhalten. Das bildliche Vorstellungsvermögen (*mental imagery*) wird als eine Form von Erinnerung diskutiert. Menschen haben die Fähigkeit, bewusst Bilder für unterschiedliche Zwecke zu erzeugen - dieses Phänomen wurde in mehreren wissenschaftlichen Disziplinen untersucht. Vorstellungsübungen werden in den Sportwissenschaften (Verbesserung von motorischen Fähigkeiten), als auch in Therapieformen der Psychologie (Verhaltenstherapie) als Methode angewandt. Die Frage, ob die bildliche Vorstellung von Essen einen Einfluss auf darauffolgendes Essverhalten hat, wurde in dieser Arbeit untersucht.

### *Methoden*

Im durchgeführten Experiment wurden TeilnehmerInnen zu einem sensorischen Test mit Gummibärchen eingeladen, wobei die Menge der während der Überprüfung verzehrten Gummibärchen ermittelt wurde. Des Weiteren wurde das subjektive Hunger-

und Sättigungsgefühl ermittelt und Fragebögen über gezügeltes Essverhalten, Klarheit und Verwendung von bildlichen Vorstellungen der 95 Teilnehmer abgefragt. TeilnehmerInnen wurden per Zufallsprinzip einer der vier Interventionsgruppen zugeordnet, die jeweils unterschiedliche Vorstellungsübungen vor der sensorischen Prüfung durchführen mussten. Dabei stellten sich TeilnehmerInnen in den ersten beiden Gruppen, jeweils mit unterschiedlichen Wiederholungen, vor, Gummibärchen zu verzehren (18 bzw. 36 Wiederholungen). Die beiden anderen Gruppen dienten als Kontrollgruppe, bei der sich die TeilnehmerInnen vorstellten eine 50 ¢-Münze mit der entsprechender Anzahl an Wiederholungen in einen Waschautomaten zu stecken.

### *Ergebnisse*

Alle TeilnehmerInnen, die sich vor der sensorischen Prüfung vorgestellt hatten Gummibärchen zu verzehren, verzehrten während der sensorischen Prüfung im Vergleich zu TeilnehmerInnen in den Kontrollgruppen signifikant weniger Gummibärchen ( $p = 0,004$ ). Ebenfalls signifikant war der Unterschied zwischen den Gruppen mit 36 Wiederholungen ( $p = 0,015$ ). Zusätzlich zur Intervention wurde herausgefunden, dass die Beliebtheit der Gummibärchen die Menge der verzehrten Gummibärchen vorhersagen kann. Der Rückgang des Hungergefühls und der Anstieg des Sättigungsgefühls nach der sensorischen Prüfung hat sich zwischen allen TeilnehmerInnen, die sich vorgestellt hatten Gummibärchen zu verzehren, im Vergleich zu den Kontrollgruppen nicht unterschieden.

### *Fazit*

Es wurde demonstriert, dass in einer Laborumgebung allein die Vorstellung eine gewisse Anzahl an Gummibärchen zu essen, den darauffolgenden Verzehr von Gummibärchen bei einer sensorischen Prüfung verringern kann. Offenbar geschieht diese Verringerung der Verzehrmenge außerhalb unseres Wahrnehmungsbereichs und ist unabhängig von unseren Erwartungen über den Einfluss der Vorstellungsübung. Am Beispiel von *mental imagery* wurde demonstriert, dass die Frage, wieviel wir essen, durchaus von nichthomöostatischen Mechanismen abhängen kann.

## 9 Appendix

### 9.1 Literature

- ABBOTT C. R., ROSSI M., KIM M., ALAHMED S. H., TAYLOR G. M., GHATEI M. A., SMITH D. M. and BLOOM S. R. Investigation of the melanocyte stimulating hormones on food intake. Lack Of evidence to support a role for the melanocortin-3-receptor. *Brain Res* 2000; 869(1-2): 203-210.
- ADAMANTIDIS A. and de LECEA L. The hypocretins as sensors for metabolism and arousal. *J Physiol* 2009; 587(Pt 1): 33-40.
- ARP R. Scenario visualization : an evolutionary account of creative problem solving. MIT Press, Cambridge, Massachussetts. 2008
- BADONNEL K., DURIEUX D., MONNERIE R., GREBERT D., SALESSE R., CAILLOL M. and BALY C. Leptin-sensitive OBP-expressing mucous cells in rat olfactory epithelium: a novel target for olfaction-nutrition crosstalk? *Cell Tissue Res* 2009; 338(1): 53-66.
- BAIRD J. P., TRAVERS J. B. and TRAVERS S. P. Parametric analysis of gastric distension responses in the parabrachial nucleus. *Am J Physiol Regul Integr Comp Physiol* 2001; 281(5): R1568-1580.
- BALLEINE B. W. The neural basis of choice and decision making. *Journal of Neuroscience* 2007; 27(31): 8159-8160.
- BALTHASAR N., COPPARI R., McMINN J., LIU S. M., LEE C. E., TANG V., KENNY C. D., McGOVERN R. A., CHUA S. C., Jr., ELMQUIST J. K. and LOWELL B. B. Leptin receptor signaling in POMC neurons is required for normal body weight homeostasis. *Neuron* 2004; 42(6): 983-991.
- BARSALOU L. W. Grounded cognition. *Annu Rev Psychol* 2008; 59: 617-645.
- BARTOLOMEO P. The relationship between visual perception and visual mental imagery: a reappraisal of the neuropsychological evidence. *Cortex* 2002; 38(3): 357-378.
- BARTOLOMEO P. The neural correlates of visual mental imagery: an ongoing debate. *Cortex* 2008; 44(2): 107-108.
- BATTERHAM R. L., COHEN M. A., ELLIS S. M., Le ROUX C. W., WITHERS D. J., FROST G. S., GHATEI M. A. and BLOOM S. R. Inhibition of food intake in obese subjects by peptide YY3-36. *N Engl J Med* 2003; 349(10): 941-948.
- BATTERHAM R. L., COWLEY M. A., J. S. C., HERZOG H., COHEN M. A., DAKIN C. L., WREN A. M., BRYNES A. E., LOW M. J., GHATEI M. A., CONE R. D. and BLOOM S. R. Gut hormone PYY(3-36) physiologically inhibits food intake. *Nature* 2002; 418(6898): 650-654.
- BEAR M. F., CONNORS B. W. and PARADISO M. A. The central visual system. In: *Neuroscience: exploring the brain*. Lippincott Williams & Wilkins, Philadelphia, 2007a; 10; 310-341.
- BEAR M. F., CONNORS B. W. and PARADISO M. A. Chemical Control of the brain and behavior. In: *Neuroscience: exploring the brain*. Lippincott Williams & Wilkins, Philadelphia, 2007b; 15; 481-509.

- BEAR M. F., CONNORS B. W. and PARADISO M. A. The eye. In: Neuroscience: exploring the brain. Lippincott Williams & Wilkins, Philadelphia, 2007c; 9; 277-309.
- BEAR M. F., CONNORS B. W. and PARADISO M. A. Memory systems. In: Neuroscience: exploring the brain. Lippincott Williams & Wilkins, Philadelphia, 2007d; 24; 725-761.
- BEAR M. F., CONNORS B. W. and PARADISO M. A. Neuroscience: exploring the brain. Lippincott Williams & Wilkins, Philadelphia. 2007e
- BEHRMANN M., WINCOUR G. and MOSCOVITCH M. Dissociation between mental imagery and object recognition in a brain-damaged patient. *Nature* 1992; 359(6396): 636-637.
- BENGTTSSON M. W., MAKELA K., SJOBLUM M., UOTILA S., AKERMAN K. E., HERZIG K. H. and FLEMSTROM G. Food-induced expression of orexin receptors in rat duodenal mucosa regulates the bicarbonate secretory response to orexin-A. *Am J Physiol Gastrointest Liver Physiol* 2007; 293(2): G501-509.
- BENSAFI M., N. S. and KHAN R. M. Hedonic-specific activity in piriform cortex during odor imagery mimics that during odor perception. *Journal of Neurophysiology* 2007; 98(6): 3254-3262.
- BERRIDGE K. C. The debate over dopamine's role in reward: the case for incentive salience. *Psychopharmacology (Berl)* 2007; 191(3): 391-431.
- BERRIDGE K. C., ROBINSON T. E. and ALDRIDGE J. W. Dissecting components of reward: 'liking', 'wanting', and learning. *Curr Opin Pharmacol* 2009; 9(1): 65-73.
- BERTHOUD H. R. Vagal and hormonal gut-brain communication: From satiation to satisfaction. *Neurogastroenterology and Motility* 2008; 20(SUPPL. 1): 64-72.
- BERTHOUD H. R. Metabolic and hedonic drives in the neural control of appetite: who is the boss? *Curr Opin Neurobiol* 2011; 21(6): 888-896.
- BERTHOUD H. R. and MUENZBERG H. The lateral hypothalamus as integrator of metabolic and environmental needs: from electrical self-stimulation to optogenetics. *Physiol Behav* 2011; 104(1): 29-39.
- BLOOM F. E., NELSON C. A., LAZERSON A. and ANNENBERG/CPB Project. Brain, mind, and behavior. Worth Publishers, New York. 2001
- BLUNDELL J. Pharmacological approaches to appetite suppression. *Trends Pharmacol Sci* 1991; 12(4): 147-157.
- BLUNDELL J. E. and HALFORD J. C. Regulation of nutrient supply: the brain and appetite control. *Proc Nutr Soc* 1994; 53(2): 407-418.
- BUSS C., KRAEMER-AGUIAR L. G., MARANHÃO P. A., MARINHO C., de SOUZA M. G., WIERNSPERGER N. and BOUSKELA E. Novel findings in the cephalic phase of digestion: a role for microcirculation? *Physiol Behav* 2012; 105(4): 1082-1087.
- CAI X. J., WIDDOWSON P. S., HARROLD J., WILSON S., BUCKINGHAM R. E., ARCH J. R., TADAYYON M., C. C. J., WILDING J. and WILLIAMS G. Hypothalamic orexin expression: modulation by blood glucose and feeding. *Diabetes* 1999; 48(11): 2132-2137.
- CAMERON J. D. and DOUCET E. Reinforcement and Food Hedonics: A Look at How Energy Deprivation Impacts Food Reward. In: *Handbook of Behavior, Food and Nutrition*. Springer, New York, 2011; 145; 2285 - 2305.

- CARDOSO-LEITE P. and GOREA A. On the perceptual/motor dissociation: a review of concepts, theory, experimental paradigms and data interpretations. *Seeing Perceiving* 2010; 23(2): 89-151.
- CHARCOT J. M. and BERNARD D. Un cas de suppression brusque et isolée de la vision mentale des signes et des objets (formes et couleurs) ; [a case of sudden and isolated suppression of the mental vision of signs and objects (form and colour)]. *Le Progrès Médical* 1883; 11.
- CLARK J. T., KALRA P. S. and KALRA S. P. Neuropeptide Y stimulates feeding but inhibits sexual behavior in rats. *Endocrinology* 1985; 117(6): 2435-2442.
- CONE R. D. Anatomy and regulation of the central melanocortin system. *Nat Neurosci* 2005; 8(5): 571-578.
- CONG W. N., GOLDEN E., PANTALEO N., WHITE C. M., MAUDSLEY S. and MARTIN B. Ghrelin receptor signaling: a promising therapeutic target for metabolic syndrome and cognitive dysfunction. *CNS Neurol Disord Drug Targets* 2010; 9(5): 557-563.
- CRUM A. J., CORBIN W. R., BROWNELL K. D. and SALOVEY P. Mind over milkshakes: mindsets, not just nutrients, determine ghrelin response. *Health Psychol* 2011; 30(4): 424-429; discussion 430-421.
- CUI X., JETER C. B., YANG D. N., MONTAGUE P. R. and EAGLEMAN D. M. Vividness of mental imagery: Individual variability can be measured objectively. *Vision Research* 2007; 47(4): 474-478.
- CUMMINGS D. E., PURNELL J. Q., FRAYO R. S., SCHMIDOVA K., WISSE B. E. and WEIGLE D. S. A preprandial rise in plasma ghrelin levels suggests a role in meal initiation in humans. *Diabetes* 2001; 50(8): 1714-1719.
- CUMMINGS D. E. and SCHWARTZ M. W. Genetics and pathophysiology of human obesity. *Annu Rev Med* 2003; 54: 453-471.
- DADDS M. R., BOVBJERG D. H., REDD W. H. and CUTMORE T. R. Imagery in human classical conditioning. *Psychol Bull* 1997; 122(1): 89-103.
- DALTON P., WYSOCKI C. J., BRODY M. J. and LAWLEY H. J. The influence of cognitive bias on the perceived odor, irritation and health symptoms from chemical exposure. *Int Arch Occup Environ Health* 1997; 69(6): 407-417.
- DANIEL H. Molecular and integrative physiology of intestinal peptide transport. *Annu Rev Physiol* 2004; 66: 361-384.
- DINKEL A., BERTH H., EXNER C., RIEF W. and BALCK F. German version of the Restraint Scale for the assessment of restrained eating. *Diagnostica* 2005; 51(2): 67-74.
- DOCKRAY G. J. Cholecystokinin and gut-brain signalling. *Regul Pept* 2009; 155(1-3): 6-10.
- EDWARD B. L. and REXFORD S. A. Central Regulation of Appetite and Satiety Behavior. In: *Handbook of Behavior, Food and Nutrition*. Springer, New York, 2011; 67; 1023 - 1033.
- EHLERS A. and CLARK D. M. A cognitive model of posttraumatic stress disorder. *Behav Res Ther* 2000; 38(4): 319-345.
- ELGAR F. J., ROBERTS C., TUDOR-SMITH C. and MOORE L. Validity of self-reported height and weight and predictors of bias in adolescents. *J Adolesc Health* 2005; 37(5): 371-375.
- EMOND M., SCHWARTZ G. J., LADENHEIM E. E. and MORAN T. H. Central leptin modulates behavioral and neural responsivity to CCK. *American Journal*

- of Physiology-Regulatory Integrative and Comparative Physiology 1999; 276(5): R1545-R1549.
- EPSTEIN L. H., ROBINSON J. L., TEMPLE J. L., ROEMMICH J. N., MARUSEWSKI A. L. and NADBRZUCH R. L. Variety influences habituation of motivated behavior for food and energy intake in children. *Am J Clin Nutr* 2009; 89(3): 746-754.
- ESTES D. Young children's awareness of their mental activity: the case of mental rotation. *Child Dev* 1998; 69(5): 1345-1360.
- FERBINTEANU J. and McDONALD R. J. Dorsal/ventral hippocampus, fornix, and conditioned place preference. *Hippocampus* 2001; 11(2): 187-200.
- FIELD A. P. Discovering statistics using SPSS : (and sex, drugs and rock 'n' roll). In: *ISM introducing statistical methods*. Sage Publications, London ; Thousand Oaks, Calif., 2005; 666-670.
- FINKE R. A. and KOSSLYN S. M. Mental imagery acuity in the peripheral visual field. *J Exp Psychol Hum Percept Perform* 1980; 6(1): 126-139.
- FLINT A., RABEN A., BLUNDELL J. E. and ASTRUP A. Reproducibility, power and validity of visual analogue scales in assessment of appetite sensations in single test meal studies. *Int J Obes Relat Metab Disord* 2000; 24(1): 38-48.
- FRIEDMAN J. M. and HALAAS J. L. Leptin and the regulation of body weight in mammals. *Nature* 1998; 395(6704): 763-770.
- FROST G. S., BRYNES A. E., DHILLO W. S., BLOOM S. R. and McBURNEY M. I. The effects of fiber enrichment of pasta and fat content on gastric emptying, GLP-1, glucose, and insulin responses to a meal. *Eur J Clin Nutr* 2003; 57(2): 293-298.
- GANIS G., THOMPSON W. L. and KOSSLYN S. M. Brain areas underlying visual mental imagery and visual perception: an fMRI study. *Brain Res Cogn Brain Res* 2004; 20(2): 226-241.
- GARCIA J., KIMELDORF D. J. and KOELLING R. A. Conditioned aversion to saccharin resulting from exposure to gamma radiation. *Science* 1955; 122(3160): 157-158.
- GIBBONS C., CAUDWELL P., FINLAYSON G., KING N. and BLUNDELL J. Validation of a new hand-held electronic data capture method for continuous monitoring of subjective appetite sensations. *Int J Behav Nutr Phys Act* 2011; 8: 57.
- GODDALE M. A. Image and Brain - the Resolution of the Imagery Debate - Kosslyn, Sm. *Journal of Cognitive Neuroscience* 1995; 7(3): 415-420.
- GOLDSTONE A. P., PRECHTL de HERNANDEZ C. G., BEAVER J. D., MUHAMMED K., CROESE C., BELL G., DURIGHEL G., HUGHES E., WALDMAN A. D., FROST G. and BELL J. D. Fasting biases brain reward systems towards high-calorie foods. *Eur J Neurosci* 2009; 30(8): 1625-1635.
- GOODALE M. A. Action without perception in human vision. *Cogn Neuropsychol* 2008; 25(7-8): 891-919.
- GREGERSEN H. and KASSAB G. Biomechanics of the gastrointestinal tract. *Neurogastroenterol Motil* 1996; 8(4): 277-297.
- GRILL H. J. and NORGREN R. The taste reactivity test. II. Mimetic responses to gustatory stimuli in chronic thalamic and chronic decerebrate rats. *Brain Res* 1978; 143(2): 281-297.



- GROVES P. M. and THOMPSON R. F. Habituation: a dual-process theory. *Psychol Rev* 1970; 77(5): 419-450.
- HALFORD J. C. G. and BLUNDELL J. E. Separate systems for serotonin and leptin in appetite control. *Annals of Medicine* 2000; 32(3): 222-232.
- HARROLD J. A., DOVEY T. M., BLUNDELL J. E. and HALFORD J. C. CNS regulation of appetite. *Neuropharmacology* 2012; 63(1): 3-17.
- HARVEY K., KEMPS E. and TIGGEMANN M. The nature of imagery processes underlying food cravings. *Br J Health Psychol* 2005; 10(Pt 1): 49-56.
- HAVERMANS R. C. "You Say it's Liking, I Say it's Wanting ... ". On the difficulty of disentangling food reward in man. *Appetite* 2011; 57(1): 286-294.
- HAVERMANS R. C. How to tell where 'liking' ends and 'wanting' begins. *Appetite* 2012a; 58(1): 252-255.
- HAVERMANS R. C. Stimulus specificity but no dishabituation of sensory-specific satiety. *Appetite* 2012b; 58(3): 852-855.
- HEBB D. O. The organization of behavior; a neuropsychological theory. Wiley, New York, 1949
- HEBBEN N., CORKIN S., EICHENBAUM H. and SHEDLACK K. Diminished ability to interpret and report internal states after bilateral medial temporal resection: case H.M. *Behav Neurosci* 1985; 99(6): 1031-1039.
- HERMAN C. P. and MACK D. Restrained and unrestrained eating. *J Pers* 1975; 43(4): 647-660.
- HERMAN C. P., ROTH D. A. and POLIVY J. Effects of the presence of others on food intake: a normative interpretation. *Psychol Bull* 2003; 129(6): 873-886.
- HIGGS S. Memory for recent eating and its influence on subsequent food intake. *Appetite* 2002; 39(2): 159-166.
- HIGGS S., WILLIAMSON A. C., ROTSCHTEIN P. and HUMPHREYS G. W. Sensory-specific satiety is intact in amnesics who eat multiple meals. *Psychological Science* 2008; 19(7): 623-628.
- HIGGS S. and WOODWARD M. Television watching during lunch increases afternoon snack intake of young women. *Appetite* 2009; 52(1): 39-43.
- HIRSCHFELD G., FELDKER K. and ZWITSERLOOD P. Listening to "flying ducks": individual differences in sentence-picture verification investigated with ERPs. *Psychophysiology* 2012; 49(3): 312-321.
- HOLMES E. A., DEEPROSE C., FAIRBURN C. G., WALLACE-HADRILL S. M., BONSALE M. B., GEDDES J. R. and GODDWIN G. M. Mood stability versus mood instability in bipolar disorder: a possible role for emotional mental imagery. *Behav Res Ther* 2011; 49(10): 707-713.
- HOLMES E. A., LANG T. J. and SHAH D. M. Developing interpretation bias modification as a "cognitive vaccine" for depressed mood: imagining positive events makes you feel better than thinking about them verbally. *J Abnorm Psychol* 2009; 118(1): 76-88.
- HOMMEL J. D., TRINKO R., SEARS R. M., GEORGESCU D., LIU Z. W., GAO X. B., THURMON J. J., MARINELLI M. and DiLEONE R. J. Leptin receptor signaling in midbrain dopamine neurons regulates feeding. *Neuron* 2006; 51(6): 801-810.
- HORN C. C. Why is the neurobiology of nausea and vomiting so important? *Appetite* 2008; 50(2-3): 430-434.

- ISHAI A., HAXBY J. V. and UNGERLEIDER L. G. Visual imagery of famous faces: effects of memory and attention revealed by fMRI. *NeuroImage* 2002; 17(4): 1729-1741.
- IWATSUKI K., ICHIKAWA R., UEMATSU A., KITAMURA A., UNEYAMA H. and TORII K. Detecting sweet and umami tastes in the gastrointestinal tract. *Acta Physiologica* 2012; 204(2): 169-177.
- KANOSKI S. E. Cognitive and neuronal systems underlying obesity. *Physiol Behav* 2012; 106(3): 337-344.
- KENNY P. J. Common cellular and molecular mechanisms in obesity and drug addiction. *Nat Rev Neurosci* 2011; 12(11): 638-651.
- KNAUPER B., PILLAY R., LaCAILLE J., McCOLLAM A. and KELSO E. Replacing craving imagery with alternative pleasant imagery reduces craving intensity. *Appetite* 2011; 57(1): 173-178.
- KOBAYASHI M., SASABE T., SHIGIHARA Y., TANAKA M. and WATANABE Y. Gustatory imagery reveals functional connectivity from the prefrontal to insular cortices traced with magnetoencephalography. *PLoS ONE* 2011; 6(7): e21736.
- KOBAYASHI M., TAKEDA M., HATTORI N., FUKUNAGA M., SASABE T., INOUE N., NAGAI Y., SAWADA T., SADATO N. and WATANABE Y. Functional imaging of gustatory perception and imagery: "top-down" processing of gustatory signals. *NeuroImage* 2004; 23(4): 1271-1282.
- KOKKINOS A., Le ROUX C. W., ALEXIADOU K., TENTOLOURIS N., VINCENT R. P., KYRIAKI D., PERREA D., GHATEI M. A., BLOOM S. R. and KATSILAMBROS N. Eating Slowly Increases the Postprandial Response of the Anorexigenic Gut Hormones, Peptide YY and Glucagon-Like Peptide-1. *Journal of Clinical Endocrinology & Metabolism* 2010; 95(1): 333-337.
- KONEN C. S., BEHRMANN M., NISHIMURA M. and KASTNER S. The functional neuroanatomy of object agnosia: a case study. *Neuron* 2011; 71(1): 49-60.
- KOPELMAN P. G. Obesity as a medical problem. *Nature* 2000; 404(6778): 635-643.
- KOSSLYN S. M. *Image and Brain: The Resolution of the Imagery Debate*. MIT Press, Cambridge. 1994
- KOSSLYN S. M., GANIS G. and THOMPSON W. L. Neural foundations of imagery. *Nat Rev Neurosci* 2001; 2(9): 635-642.
- KRINGELBACH M. L. The human orbitofrontal cortex: linking reward to hedonic experience. *Nat Rev Neurosci* 2005; 6(9): 691-702.
- KRINGELBACH M. L., O'DOHERTY J., ROLLS E. T. and ANDREWS C. Activation of the human orbitofrontal cortex to a liquid food stimulus is correlated with its subjective pleasantness. *Cereb Cortex* 2003; 13(10): 1064-1071.
- KRINGELBACH M. L., STEIN A. and van HARTEVELT T. J. The functional human neuroanatomy of food pleasure cycles. *Physiol Behav* 2012; 106(3): 307-316.
- La FLEUR S. E., van ROZEN A. J., LUIJENDIJK M. C., GROENEWEG F. and ADAN R. A. A free-choice high-fat high-sugar diet induces changes in arcuate neuropeptide expression that support hyperphagia. *Int J Obes (Lond)* 2010; 34(3): 537-546.
- LAVIN J. H., WITTERT G. A., ANDREWS J., YEAP B., WISHART J. M., MORRIS H. A., MORLEY J. E., HOROWITZ M. and READ N. W. Interaction of insulin, glucagon-like peptide 1, gastric inhibitory polypeptide, and appetite in response to intraduodenal carbohydrate. *Am J Clin Nutr* 1998; 68(3): 591-598.

- Le ROUX C. W., PATTERSON M., VINCENT R. P., HUNT C., GHATEI M. A. and BLOOM S. R. Postprandial plasma ghrelin is suppressed proportional to meal calorie content in normal-weight but not obese subjects. *J Clin Endocrinol Metab* 2005; 90(2): 1068-1071.
- LEE S. H., KRAVITZ D. J. and BAKER C. I. Disentangling visual imagery and perception of real-world objects. *NeuroImage* 2012; 59(4): 4064-4073.
- LEE S. Y. and SOLTESZ I. Cholecystokinin: a multi-functional molecular switch of neuronal circuits. *Dev Neurobiol* 2011; 71(1): 83-91.
- LEINNINGER G. M., JO Y. H., LESHAN R. L., LOUIS G. W., YANG H., BARRERA J. G., WILSON H., OPLAND D. M., FAOUZI M. A., GONG Y., JONES J. C., RHODES C. J., CHUA S., Jr., DIANO S., HORVATH T. L., SEELEY R. J., BECKER J. B., MUNZBERG H. and MYERS M. G., Jr. Leptin acts via leptin receptor-expressing lateral hypothalamic neurons to modulate the mesolimbic dopamine system and suppress feeding. *Cell Metab* 2009; 10(2): 89-98.
- LEVIN B. E., MAGNAN C., DUNN-MEYNELL A. and Le FOLL C. Metabolic sensing and the brain: who, what, where, and how? *Endocrinology* 2011; 152(7): 2552-2557.
- MARCHIORI D., CORNEILLE O. and KLEIN O. Container size influences snack food intake independently of portion size. *Appetite* 2012; 58(3): 814-817.
- MARKS D. F. Visual imagery differences in the recall of pictures. *Br J Psychol* 1973; 64(1): 17-24.
- MARTIN K. A., MORITZ S. E. and HALL C. R. Imagery use in sport: A literature review and applied model. *Sport Psychologist* 1999; 13(3): 245-268.
- MASCHKE M., DREPPER J., KINDSVATER K., KOLB F. P., DIENER H. C. and TIMMANN D. Involvement of the human medial cerebellum in long-term habituation of the acoustic startle response. *Exp Brain Res* 2000; 133(3): 359-367.
- MATTES R. D. Physiologic responses to sensory stimulation by food: nutritional implications. *J Am Diet Assoc* 1997; 97(4): 406-413.
- MATVEYENKO A. V. and DONOVAN C. M. Metabolic sensors mediate hypoglycemic detection at the portal vein. *Diabetes* 2006; 55(5): 1276-1282.
- MILLER A. D. and LESLIE R. A. The area postrema and vomiting. *Front Neuroendocrinol* 1994; 15(4): 301-320.
- MISCHEL W., EBBESEN E. B. and ZEISS A. R. Cognitive and attentional mechanisms in delay of gratification. *J Pers Soc Psychol* 1972; 21(2): 204-218.
- MISHKIN M. and UNGERLEIDER L. G. Contribution of striate inputs to the visuospatial functions of parieto-preoccipital cortex in monkeys. *Behav Brain Res* 1982; 6(1): 57-77.
- MITTAL D., STEVENSON R. J., OATEN M. J. and MILLER L. A. Snacking while watching TV impairs food recall and promotes food intake on a later TV free test meal. *Applied Cognitive Psychology* 2011; 25(6): 871-877.
- MONTAGUE P. R. Free will. *Curr Biol* 2008; 18(14): R584-585.
- MOREWEDGE C. K., HUH Y. E. and VOSGERAU J. Thought for food: imagined consumption reduces actual consumption. *Science* 2010; 330(6010): 1530-1533.
- MORO V., BERLUCCHI G., LERCH J., TOMAIUOLO F. and AGLIOTI S. M. Selective deficit of mental visual imagery with intact primary visual cortex and visual perception. *Cortex* 2008; 44(2): 109-118.

- NALEID A. M., GRACE M. K., CUMMINGS D. E. and LEVINE A. S. Ghrelin induces feeding in the mesolimbic reward pathway between the ventral tegmental area and the nucleus accumbens. *Peptides* 2005; 26(11): 2274-2279.
- NOË A. Action in perception. MIT Press, Cambridge, Mass. 2004
- NORGREN R. Gustatory responses in the hypothalamus. *Brain Res* 1970; 21(1): 63-77.
- O'REGAN J. K. and NOE A. A sensorimotor account of vision and visual consciousness. *Behav Brain Sci* 2001; 24(5): 939-973; discussion 973-1031.
- OLLMANN M. M., WILSON B. D., YANG Y. K., KERNS J. A., CHEN Y., GANTZ I. and BARSH G. S. Antagonism of central melanocortin receptors in vitro and in vivo by agouti-related protein. *Science* 1997; 278(5335): 135-138.
- OLSZEWSKI P. K., ALSIO J., SCHIOTH H. B. and LEVINE A. S. Opioids as facilitators of feeding: can any food be rewarding? *Physiol Behav* 2011; 104(1): 105-110.
- ONAGA T., ZABIELSKI R. and KATO S. Multiple regulation of peptide YY secretion in the digestive tract. *Peptides* 2002; 23(2): 279-290.
- PAIK K. H., JIN D. K., LEE K. H., ARMSTRONG L., LEE J. E., OH Y. J., KIM S., KWON E. K. and CHOE Y. H. Peptide YY, cholecystokinin, insulin and ghrelin response to meal did not change, but mean serum levels of insulin is reduced in children with Prader-Willi syndrome. *J Korean Med Sci* 2007; 22(3): 436-441.
- PAIVIO A. Imagery and verbal processes. Holt, New York, 1971
- PAIVIO A. and HARSHMAN R. Factor Analysis of a Questionnaire on Imagery and Verbal Habits and Skills. *Canadian Journal of Psychology* 1983; 37(4): 461-483.
- PEARSON J. Associative Learning: Pavlovian Conditioning without Awareness. *Curr Biol* 2012; 22(12): R495-496.
- PETROVICH G. D., HOLLAND P. C. and GALLAGHER M. Amygdalar and prefrontal pathways to the lateral hypothalamus are activated by a learned cue that stimulates eating. *J Neurosci* 2005; 25(36): 8295-8302.
- PETROVICH G. D., ROSS C. A., GALLAGHER M. and HOLLAND P. C. Learned contextual cue potentiates eating in rats. *Physiol Behav* 2007; 90(2-3): 362-367.
- PETROVICH G. D., SETLOW B., HOLLAND P. C. and GALLAGHER M. Amygdalo-hypothalamic circuit allows learned cues to override satiety and promote eating. *J Neurosci* 2002; 22(19): 8748-8753.
- POST P. G., WRISBERG C. A. and MULLINS S. "A Field Test of the Influence of Pre-Game Imagery on Basketball Free Throw Shooting." 2010, 5(Issue).
- POWER L. M. and SCHULKIN J. Anticipatory Physiological Regulation in Feeding Biology. In: *Handbook of behavior, food and nutrition*. Springer, New York, 2011; 55: 829-844.
- POWLEY T. L. Vagal circuitry mediating cephalic-phase responses to food. *Appetite* 2000; 34(2): 184-188.
- PROUST M. Auf der Suche nach der verlorenen Zeit 1: Unterwegs zu Swann. Suhrkamp Verlag, Frankfurt/Main. 1994; p 66 et seq.
- PYLYSHYN Z. W. What Minds Eye Tells Minds Brain - Critique of Mental Imagery. *Psychological Bulletin* 1973; 80(1): 1-24.
- PYLYSHYN Z. W. Mental imagery: in search of a theory. *Behav Brain Sci* 2002; 25(2): 157-182; discussion 182-237.
- QU D., LUDWIG D. S., GAMMELTOFT S., PIPER M., PELLEYMOUNTER M. A., CULLEN M. J., MATHES W. F., PRZYPEK R., KANAREK R. and

- MARATOS-FLIER E. A role for melanin-concentrating hormone in the central regulation of feeding behaviour. *Nature* 1996; 380(6571): 243-247.
- RAIO C. M., CARMEL D., CARRASCO M. and PHELPS E. A. Nonconscious fear is quickly acquired but swiftly forgotten. *Curr Biol* 2012; 22(12): R477-479.
- RANKIN C. H., ABRAMS T., BARRY R. J., BHATNAGAR S., CLAYTON D. F., COLOMBO J., COPPOLA G., GEYER M. A., GLANZMAN D. L., MARSLAND S., McSWEENEY F. K., WILSON D. A., WU C. F. and THOMPSON R. F. Habituation revisited: an updated and revised description of the behavioral characteristics of habituation. *Neurobiol Learn Mem* 2009; 92(2): 135-138.
- RODGERS R. J., ISHII Y., HALFORD J. C. and BLUNDELL J. E. Orexins and appetite regulation. *Neuropeptides* 2002; 36(5): 303-325.
- ROLLS B. J., ROLLS E. T., ROWE E. A. and SWEENEY K. Sensory specific satiety in man. *Physiology and Behavior* 1981; 27(1): 137-142.
- ROZIN P., DOW S., MOSCOVITCH M. and RAJARAM S. What causes humans to begin and end a meal? A role for memory for what has been eaten, as evidenced by a study of multiple meal eating in amnesic patients. *Psychological Science* 1998; 9(5): 392-396.
- SAPER C. B., CHOU T. C. and ELMQUIST J. K. The need to feed: homeostatic and hedonic control of eating. *Neuron* 2002; 36(2): 199-211.
- SARTRE J. The psychology of imagination. *Philosophical Library*, New York, 1948
- SCHACHTER D. L. and ADDIS D. R. Constructive memory: the ghosts of past and future. *Nature* 2007; 445(7123): 27.
- SCHACHTER S. and GROSS L. P. Manipulated time and eating behavior. *J Pers Soc Psychol* 1968; 10(2): 98-106.
- SCHACHTER S. and SINGER J. E. Cognitive, social, and physiological determinants of emotional state. *Psychol Rev* 1962; 69: 379-399.
- SCHUSTER C., HILFIKER R., AMFT O., SCHEIDHAUER A., ANDREWS B., BUTLER J., KISCHKA U. and ETTLIN T. Best practice for motor imagery: a systematic literature review on motor imagery training elements in five different disciplines. *BMC Med* 2011; 9: 75.
- SCHWARTZ G. J. Integrative capacity of the caudal brainstem in the control of food intake. *Philos Trans R Soc Lond B Biol Sci* 2006; 361(1471): 1275-1280.
- SCLAFANI A. Post-ingestive positive controls of ingestive behavior. *Appetite* 2001; 36(1): 79-83.
- SELBACH O., BOHLA C., BARBARA A., DOREULEE N., ERIKSSON K. S., SERGEEVA O. A. and HAAS H. L. Orexins/hypocretins control bistability of hippocampal long-term synaptic plasticity through co-activation of multiple kinases. *Acta Physiol (Oxf)* 2010; 198(3): 277-285.
- SHEPARD R. N. and METZLER J. Mental rotation of three-dimensional objects. *Science* 1971; 171(3972): 701-703.
- SHIDE D. J. and ROLLS B. J. Information about the fat content of preloads influences energy intake in healthy women. *J Am Diet Assoc* 1995; 95(9): 993-998.
- SHIN A. C., ZHENG H. and BERTHOUD H. R. An expanded view of energy homeostasis: Neural integration of metabolic, cognitive, and emotional drives to eat. *Physiology and Behavior* 2009; 97(5): 572-580.

- SILVA J. P., von MEYENN F., HOWELL J., THORENS B., WOLFRUM C. and STOFFEL M. Regulation of adaptive behaviour during fasting by hypothalamic Foxa2. *Nature* 2009; 462(7273): 646-650.
- SMITH M. W. and GUMBLETON M. Endocytosis at the blood-brain barrier: from basic understanding to drug delivery strategies. *J Drug Target* 2006; 14(4): 191-214.
- SOON C. S., BRASS M., HEINZE H. J. and HAYNES J. D. Unconscious determinants of free decisions in the human brain. *Nat Neurosci* 2008; 11(5): 543-545.
- SPENCE C. Mouth-Watering: The Influence of Environmental and Cognitive Factors on Salivation and Gustatory/Flavor Perception. *Journal of Texture Studies* 2011; 42(2): 157-171.
- STAFLEU A., ZIJLSTRA N., HOGENKAMP P. and MARS M. Texture and Diet Related Behavior: A Focus on Satiation and Satiety. In: *Handbook of behavior, food and nutrition*. Springer, New York, 2011; 10; 133-142.
- STRATFORD T. R., KELLEY A. E. and SIMANSKY K. J. Blockade of GABAA receptors in the medial ventral pallidum elicits feeding in satiated rats. *Brain Res* 1999; 825(1-2): 199-203.
- STRATFORD T. R. and WIRTSHAFTER D. Evidence that the nucleus accumbens shell, ventral pallidum, and lateral hypothalamus are components of a lateralized feeding circuit. *Behav Brain Res* 2012; 226(2): 548-554.
- STUBBS R. J., HUGHES D. A., JOHNSTONE A. M., ROWLEY E., REID C., ELIA M., STRATTON R., DELARGY H., KING N. and BLUNDELL J. E. 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. *Br J Nutr* 2000; 84(4): 405-415.
- SWINBURN B., EGGER G. and RAZA F. Dissecting obesogenic environments: the development and application of a framework for identifying and prioritizing environmental interventions for obesity. *Prev Med* 1999; 29(6 Pt 1): 563-570.
- SWINBURN B. A., SACKS G., HALL K. D., McPHERSON K., FINEGOOD D. T., MOODIE M. L. and GORTMAKER S. L. The global obesity pandemic: shaped by global drivers and local environments. *Lancet* 2011; 378(9793): 804-814.
- TIGGEMANN M. and KEMPS E. The phenomenology of food cravings: the role of mental imagery. *Appetite* 2005; 45(3): 305-313.
- TSCHOEP M., SMILEY D. L. and HEIMAN M. L. Ghrelin induces adiposity in rodents. *Nature* 2000; 407(6806): 908-913.
- VERHAGEN J. V. and ENGELLEN L. The neurocognitive bases of human multimodal food perception: sensory integration. *Neurosci Biobehav Rev* 2006; 30(5): 613-650.
- WANSINK B. From mindless eating to mindlessly eating better. *Physiology and Behavior* 2010; 100(5): 454-463.
- WANSINK B. and CHENEY M. M. Super Bowls: serving bowl size and food consumption. *JAMA* 2005; 293(14): 1727-1728.
- WANSINK B. and KIM J. Bad popcorn in big buckets: portion size can influence intake as much as taste. *J Nutr Educ Behav* 2005; 37(5): 242-245.
- WANSINK B., PAINTER J. E. and NORTH J. Bottomless bowls: why visual cues of portion size may influence intake. *Obes Res* 2005; 13(1): 93-100.

- WANSINK B., PAYNE C. R. and NORTH J. Fine as North Dakota wine: sensory expectations and the intake of companion foods. *Physiol Behav* 2007; 90(5): 712-716.
- WATSON J. B. Psychology as the behaviorist views it. *Psychological Review* 1913; 20(2): 158-177.
- WEINBERG R. "Does Imagery Work? Effects on Performance and Mental Skills." 2008, 3(Issue).
- WHO. Diet, nutrition and the prevention of chronic diseases. *World Health Organ Tech Rep Ser* 2003; 916: 1-149.
- WOOD I. S. and TRAYHURN P. Glucose transporters (GLUT and SGLT): expanded families of sugar transport proteins. *Br J Nutr* 2003; 89(1): 3-9.
- WOODS S. C. The eating paradox: how we tolerate food. *Psychol Rev* 1991; 98(4): 488-505.
- WOOLEY S. C. Physiologic versus cognitive factors in short term food regulation in the obese and nonobese. *Psychosom Med* 1972; 34(1): 62-68.
- YEOMANS M. R. and GRAY R. W. Opioid peptides and the control of human ingestive behaviour. *Neurosci Biobehav Rev* 2002; 26(6): 713-728.
- ZHANG Y., PROENCA R., MAFFEI M., BARONE M., LEOPOLD L. and FRIEDMAN J. M. Positional cloning of the mouse obese gene and its human homologue. *Nature* 1994; 372(6505): 425-432.
- ZHENG H. and BERTHOUD H. R. Neural systems controlling the drive to eat: Mind versus metabolism. *Physiology* 2008; 23(2): 75-83.

Online sources:

<http://plato.stanford.edu/entries/mental-imagery/#PreVieIma> ; accessed on 2012/06/13

<https://www.youtube.com/watch?v=6EjJsPylEOY> ; accessed on 2012/07/01

## 9.2 Attachments

### 9.2.1 Questionnaires

#### Fragebogen Nr.1 (Q1)

Vor wie viel Stunden haben  
Sie das letzte Mal etwas  
gegessen?

Alter: \_\_\_\_\_

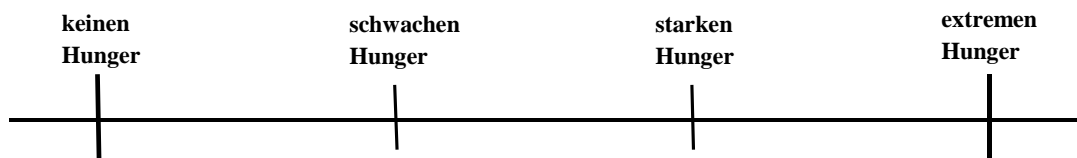
Geschlecht: w ☐ m

☐

Größe: \_\_\_\_\_

Gewicht: \_\_\_\_\_

∴ Wie viel Hunger haben Sie? Beschreiben Sie bitte Ihr Hungergefühl indem Sie auf untenstehender Linie eine Markierung setzen.

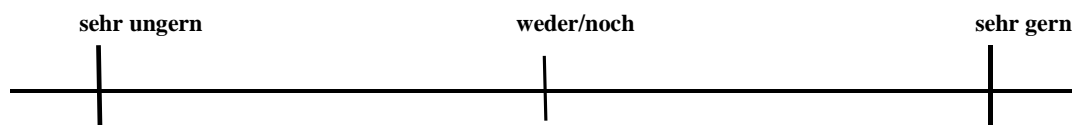


∴ Das Gummibärchen mit Welcher Farbe mögen Sie am liebsten?  
(Mehrfachantworten möglich)

weiß <input type="checkbox"/>	orange <input type="checkbox"/>	grün <input type="checkbox"/>	gelb <input type="checkbox"/>	rot <input type="checkbox"/>
-------------------------------	---------------------------------	-------------------------------	-------------------------------	------------------------------



∴ Wie sehr mögen Sie Gummibärchen?



∴ Wie satt fühlen Sie sich?

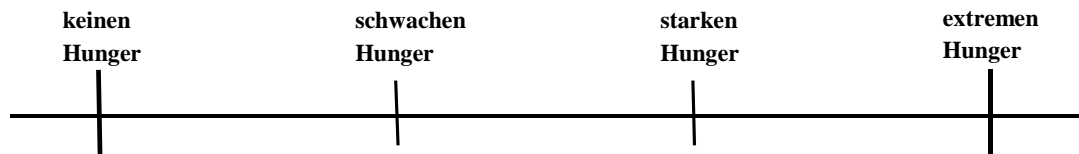


∴ Bitte machen Sie einen Kreis um die zutreffende Antwort:

Wie oft halten sie Diät	Nie	Selten	manchmal	häufig	ständig
Wie viel Gewicht (in kg) haben Sie jemals maximal innerhalb eines Monats verloren?	0,0 -1,9	2,0 -3,9	4,0 -6,4	6,5 -8,9	9,0 +
Wie viel Gewicht (in kg) haben Sie maximal innerhalb einer Woche zugenommen?	0,0 -0,4	0,5-0,9	1,0-1,4	1,5-2,4	2,5+
Wie stark schwankt Ihr Gewicht während einer normalen Woche?	0,0 -0,4	0,5-0,9	1,0-1,4	1,5-2,4	2,5+
Würde eine Gewichtsveränderung von 2kg Ihre Lebensweise beeinflussen?	überhaupt nicht	ein wenig	ziemlich	sehr stark	
Essen Sie kontrolliert, wenn Sie mit anderen zusammen sind und lassen Sie sich dann gehen, wenn Sie alleine sind?	nie	selten	häufig	immer	
Verschenden Sie zu viel Zeit und Gedanken an Essen?	nie	selten	häufig	immer	
Haben Sie Schuldgefühle wenn Sie sich überfressen haben?	nie	selten	häufig	immer	
Wie bewusst achten Sie darauf was Sie essen?	überhaupt nicht	ein wenig	ziemlich	sehr stark	
Wie viel Kilogramm lagen Sie über Ihrem Wunschgewicht, als Sie ihr höchstes Gewicht hatten?	0,0 -0,4	0,5-2,4	2,5-4,9	5,0-9,9	10+

## Fragebogen Nr.2 (Q2)

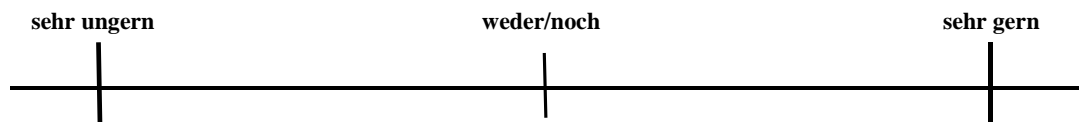
∴ Wie viel Hunger haben Sie? Beschreiben Sie bitte Ihr Hungergefühl indem Sie auf untenstehender Linie eine Markierung setzen.



∴ Das Gummibärchen mit welcher Farbe mögen Sie am liebsten?  
(Mehrfachnennungen möglich)

weiß <input type="checkbox"/>	orange <input type="checkbox"/>	grün <input type="checkbox"/>	gelb <input type="checkbox"/>	rot <input type="checkbox"/>
-------------------------------	---------------------------------	-------------------------------	-------------------------------	------------------------------

∴ Wie sehr mögen Sie Gummibärchen im Allgemeinen?



∴ Wenn Sie an Gummibärchen denken, welche Marke/Sorte würden Sie bevorzugt aus dem Regal nehmen?

\_\_\_\_\_

∴ Haben Sie sich vorhin wirklich das vorgestellt worum wir Sie gebeten haben?

Ja ☐

Nein ☐

∴ Hatten Sie Probleme bei der Durchführung der Vorstellung Probleme? Wenn ja, welche:

Ja : \_\_\_\_\_

Nein ☐

∴ Wie satt fühlen Sie sich?



∴ Welchen Einfluss, denken Sie, haben Gedanken an ein Lebensmittel oder ein Gericht auf den nachfolgenden, tatsächlichen Verzehr der Speise?

einen Appetit anregenden ☐

einen sättigenden ☐

keinen ☐

einen anderen Einfluss:

\_\_\_\_\_

## Akzeptanztest

Liebe TeilnehmerInnen,

Bei diesem Test geht es um eure individuellen Vorlieben für das Lebensmittel. Auf einer Skala von 1-9 wird nach der Akzeptanz von spezifischen Eigenschaften und nach der Gesamtakzeptanz des Gummibärchens gefragt. Bitte lasst euch Zeit mit dem Test.  
**Es dürfen unbegrenzt Gummibärchen verzehrt werden.**

Wie gerne hast du persönlich folgende visuellen Eigenschaften des Produkts?

[illegible]

Wie gerne hast du persönlich folgende texturale Eigenschaften des Produkts?

	missfällt außerordentlich			weder /noch				gefällt außerordentlich	
Festigkeit der Gummibärchen	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Klebrigkeit der Gummibärchen	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Mundgefühl während dem kauen	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>

---

Wie gerne hast du persönlich das Produkt gesamthaft?

	missfällt außerordentlich			weder /noch				gefällt außerordentlich	
Gesamt Akzeptanz	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>

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**Vielen Dank**

**für deine**

**Teilnahme!**

## Fragebogen zur visuellen Vorstellungsfähigkeit (VVIQ)

Visuelle Vorstellungsfähigkeit beschreibt die Fähigkeit sich etwas vorzustellen, also mentale Bilder aufzubauen bzw. mit dem geistigen Auge zu sehen. Es gibt große Unterschiede in der Lebhaftigkeit und der Klarheit dieser Bilder. Diese Unterschiede sind sehr wichtig für psychologische Forschung.

Das Ziel dieses Fragebogens ist es, die Lebhaftigkeit Deiner visuellen Vorstellungsfähigkeit zu messen. Dazu sollst Du im Folgenden einige Selbsteinschätzungen abgeben und danach Deine mentalen Bilder beschreiben.

Die Bearbeitung des Fragebogens dauert ca. 10 min.

**Deine Daten werden anonym ausgewertet.**

**Danke für Deine Mitarbeit!**

## Selbsteinschätzungen

Bitte gebe für jede Aussage an, wie sehr Du ihr zustimmst oder sie ablehnst.

Aussage	Stimme gar nicht zu	Stimme eher nicht zu	Teils teils	Stimme eher zu	Stimme voll zu
Ab und zu wenn ich müde bin, habe ich das Gefühl jemand hätte etwas gesagt, aber wenn ich mich umdrehe ist niemand da.	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Es ist mir schon einmal passiert, dass ich sicher war etwas gesehen zu haben, das dann aber doch nicht da war.	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Manchmal kann ich mir etwas so lebhaft vorstellen, dass ich tatsächlich spüre, wie es passiert.	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Wenn ich Werbung z.B. für Pizza im Fernsehen sehe, kann ich manchmal das Essen schmecken.	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Wenn ich an etwas zu Essen (z. B. eine Zitrone) denke, kann ich diese regelrecht schmecken.	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Wenn ich Werbung z.B. für Pizza im Fernsehen sehe, kann ich manchmal das Essen schmecken.	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Ich stelle mir Dinge oft visuell vor, um sie besser zu behalten.	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Ich denke oft in mentalen Bildern.	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Ich finde es schwierig ein mentales Bild von etwas zu entwickeln.	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>



Wenn ich mich an eine Szene erinnere, verwende ich wörtliche Beschreibung eher als ein mentales Bild.	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Ich benutze fast nie mentale Bilder um Probleme zu lösen.	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Ich benutze mentale Bilder, um mich zu entspannen.	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Mit geschlossenen Augen kann ich mir sehr leicht eine bekannte Szene anschauen.	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Ich glaube, dass die meisten Menschen in mentalen Bildern denken, auch wenn ihnen dies nicht bewusst ist.	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Ich kann mir leicht dynamische Szenen bildhaft vorstellen.	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Ich bilde keine mentalen Bilder, wenn ich etwas lese, oder einer Geschichte zuhöre.	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Wenn mir jemand erzählt, was ihm passiert ist, bemerke ich, wie ich es mir bildhaft vorstelle.	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Ich habe nur von Szenen eine bildhafte Vorstellung, die ich tatsächlich erlebt habe.	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Zuzuhören, wie jemand etwas aus seiner Erfahrung erzählt, erweckt in mir normalerweise keine mentalen Bilder.	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>

## Beschreibung mentaler Bilder

Die Items in diesem Test werden wahrscheinlich mentale Bilder bei Dir erzeugen. Du sollst die Lebhaftigkeit jedes einzelnen auf einer fünf-stufigen Skala beurteilen. Wenn Deine Vorstellung gar vage und unklar ist, gebe eine „1“. Bitte bewerte für jedes Item wie lebhaft es war, einmal mit geschlossenen Augen und einmal mit geöffneten.

Ehe Du beginnst, lese Dir noch aufmerksam die verschiedenen Kategorien durch. Verwende diese Skala für alle Items.

Versuche jedes Item für sich zu beantworten unabhängig davon, wie Du die anderen Items beantwortet hast.

### Skala

Ein mentales Bild kann sein	Rating
Gar kein Bild, Du weißt nur, dass Du an ein Objekt denkst.	1
Vage und unklar	2
Wenig klar und lebhaft	3
Klar und einigermaßen lebhaft	4
Perfekt klar, so lebhaft wie normales sehen	5

Bitte bewerte erst alle Items mit geöffneten Augen, schließe sie dann und fange noch einmal von vorne an. Die zwei Ratings können in den beiden Fällen unterschiedlich sein.



## 9.2.2 CV

**Curriculum Vitae****Benjamin Missbach**

geboren am 01. Januar 1987 in Traunstein,  
Deutschland  
ledig

Hofstattgasse 4/5a  
1180 Wien  
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**Schule**

09/1993 bis 08/1997

Grundschule Unterwössen, Deutschland

09/1997 bis 08/2006

Landschulheim Marquartstein, Deutschland  
Schulabschluss: Abitur

**Studium**

WS 07/08 – SS 10 (Uni Wien)

*Bakkalaureatstudium* Ernährungswissenschaften

WS 10/11 – SS 12 (Uni Wien)

*Masterstudium* Ernährungswissenschaften

**Praktika/Arbeit**

April – Juni 2007

Auslandspraktikum (NGO - LekaGape –  
*Entwicklungshilfe*)

Seit Mai 2010

Freizeitpädagogische Betreuung (*Parkbetreuung  
des Wiener Familienbunds*)

Seit Oktober 2010

Jugendbetreuung (*Jugendzentrum des Wiener  
Familienbunds*)

**Fremdsprachen**

Englisch (sehr gut in Wort und Schrift)  
Spanisch (Grundkenntnisse in Wort und Schrift)  
Französisch (Grundkenntnisse in Wort und Schrift)

**Private Interessen**

Gitarre, Klavier, Basketball