From the Department of Neurobiology, Care science and Society
Section of Applied Neuroendocrinology
Karolinska Institutet, Stockholm, Sweden

EATING, CHEWING AND THE MIND

Ioannis Ioakeimidis

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To my parents, Foteini, Ingrid and Leander

"The consequences of an act affect the probability of its occurring again"
-B.F.Skinner

ABSTRACT

The need for detailed description of eating behavior has become relevant by the limited success of simplified models in genetics and neuroscience to explain and predict eating behavior in humans. Failure of cognitive interventions, combined with the success of treatments normalising eating styles in obesity and eating disorders, demonstrates the central role of eating in dealing with these problems. In continuous recording of eating behavior and satiety over the course of a meal, women have been found to eat either at a decelerated or a constant rate. Linear eaters, unlike decelerated ones, are unable to control their food intake when the rate of eating is experimentally increased or decreased and their rating of satiety become disassociated from the actual food intake. Their responses to these experimental challenges simulate the eating patterns and the satiation ratings of anorexic and binge eating disorder patients. The development of an improved methodology for the analysis of single meals, combining video derived and intake data, allows for the analysis of the distinct behavioral elements of the meal over time. Semi-automation, high validity and reliability make this procedure ideal for comparing eating patterns among different groups of individuals. The chewing frequency, the distribution of chews within the chewing sequences and the pauses between mouthfuls remain stable across the meal both in decelerated and linear eaters. The weight of the mouthfuls decreases and the duration of the chewing sequences increases over time in the decelerated eaters, but not the linear ones, clarifying the nature of deceleration. Additionally, the default chewing frequency, quantified by the use of chewing gum, is lower in linear than in decelerated eaters, indicating that there is a baseline difference in the default chewing frequency between the two groups. It is suggested that linear eating is a behavioral risk factor for the development of disordered eating and it is hypothesized that while repeated disordered eating is the cause of eating disorders, the accompanying chewing characteristics might be the mediator of the emotional profile that characterizes patients with eating disorders.

LIST OF PUBLICATIONS

I. Zandian, M., Ioakimidis, I., Bergh, C., Brodin, U., & Södersten, P. (2009). Decelerated and linear eaters: effect of eating rate on food intake and satiety. *Physiology & Behavior, 96*(2), 270-275.

II. Ioakimidis, I., Zandian, M., Bergh, C., & Södersten, P. (2009). A method for the control of eating rate: a potential intervention in eating disorders. *Behavior Research Methods, 41*(3), 755-760.

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IV. Ioakimidis, I., Zandian, M., Ulbl, F., Åhlund, C., Bergh, C., & Södersten, P. (2011). Food intake and chewing in women. *Journal of Integrated Neuroscience,* (Accepted for publication).

CONTENTS

LIST OF ABBREVIATIONS

AN	Anorexia Nervosa
BED	Binge Eating Disorder
BMI	Body Mass Index
BN	Bulimia Nervosa
CIC	Cumulative Intake Curve
DEB-Q	The Dutch Eating Behaviour Questionnaire
DEC	Decelerated eaters (Figures)
ER+	Increased Eating Rate
ER-	Decreased Eating Rate
LIN	Linear eaters (Figures)
SSRIs	Selective Serotonin Reuptake Inhibitors
STAI-T	State-Trait Anxiety Inventory -Trait
STAI-S	State-Trait Anxiety Inventory -State
TFEQ-R21	21-item Three Factor Eating Questionnaire

INTRODUCTION

The background for research on eating behavior and associated clinical problems will be outlined. Eating behavior is used as an intervention to improve the situation of under- and overweight patients; a translational approach has been found valid in two randomized controlled trials (Bergh, Brodin, Lindberg & Södersten, 2002; Ford et al., 2010). This preliminary success encouraged us to look into eating behavior in further detail.

Thesis framework

The focus on the ever-increasing problem of obesity in our times has led to a corresponding increase of research on every aspect of eating. Great advances have been made in describing the neurobiological, genetic and sociological elements of obesity, each accompanied by various proposed solutions to the problem. Nevertheless, little has been achieved to halt the progress of the so-called "obesity epidemic" (Dietz, 2011). Present demographical data and predictions for the occurrence of obesity in the future are bleak and the sheer prevalence of the problem defies its characterization as a disease or an epidemic (Södersten, Bergh, Zandian & Ioakimidis, 2011). Effective obesity drugs, based upon simplified neurobiological models, inferring direct brain control over eating behavior expressed some time ago (e.g., Bray & Tartaglia, 2000), have proven enormously difficult to develop and even pharmaceutical companies now admit that a “change of fortune may require a change of strategy” (Ledford, 2010). On the other hand, combinations of lifestyle interventions seem to have somewhat positive results, especially in children, even though the reported effects can hardly be generalized due to their dependence on sociological circumstances (Oude Luttikhuis et al., 2009). Finally, surgical interventions, although effective in some cases, are accompanied by various undesirable side effects and the inherent risks of any invasive surgical procedure (Encinosa, Bernard, Du & Steiner, 2009). Hence, they are currently regarded as mere compliments rather than replacements of less invasive interventions (Dixon, Straznicky, Lambert, Schlaich & Lambert, 2011).

The emergence of eating disorders as an important problem afflicting mostly young women and its high profile in western societies (Treasure, Claudino & Zucker, 2010) has not lead to a similar increase of research on the main elements of these disorders, i.e., disordered eating and increased physical activity (Södersten, Bergh & Zandian, 2006). On the contrary, the targeted symptoms for the "mainstream" research concerning Anorexia (AN) and Bulimia Nervosa (BN), i.e., depression, enhanced anxiety and an unspecified, pre-existing mental pathology, might not even be causally related to the disorders (Ioakimidis, Zandian, Ulbl, Bergh, Leon & Södersten, 2011). For this and other reasons (e.g., diagnostic and remission criteria of questionable validity), many interventions have less than satisfying results (Striegel-Moore & Bulik, 2007; Treasure et al., 2010) and high levels of relapse (Berkman, Lohr & Bulik, 2007; Steinhausen, 2002). While the need for the development of a behavioral intervention for eating disorders, based on eating itself, was proposed early on (Bergh, Eklund, Eriksson, Lindberg & Södersten, 1996), its importance is only now starting to be discussed by the rest of the field (Treasure, Cardi & Kan, 2011; Walsh, 2011).

While the connection between obesity and eating disorders have been proposed many times in the past (Kissileff, 1989), the precise relationship of the two conditions remains unclear. Potential underlying elements range from addiction (Wilson, 2010) and reward mechanisms (Berridge, Ho, Richard & DiFeliceantonio, 2010) to various cognitive "progresses" (Riva, 2011). Still, it is noted that the obvious shared characteristic among obese individuals and patients with eating disorders is the development of disordered eating (Day, Ternouth & Collier, 2009). A strong argument for the importance of eating itself in these conditions is the success of interventions based on normalization of the eating pattern through in-meal training (Zandian, Ioakimidis, Bergh & Södersten, 2007). Training patients to eat in a non-pathological way has been proven beneficial both in eating disorders (Bergh et al., 2002) and obesity (Ford, et al., 2010).

The failure of simplified models in genetics and neuroscience to explain and modulate eating regulation and behavior, the obvious relationship between obesity and eating disorders and the subsequent lack of success of cognitive interventions, combined with the success of behaviorally based treatments, suggest that eating behavior itself is of paramount interest. There can be no doubt that excessive over- and under- eating emerge from specific behavioral patterns observed in normal weight individuals (Zandian et al., 2007). Hence, detailed information on eating behavior is required in order to comprehend the eating patterns described in obesity and eating disorders. Better understanding of the specific behavioral elements of human meals might shed light on the underlying relationship between brain and eating and provide the necessary behavioral evidence for the development of a framework bridging the gap between the neurobiological and behavioral substrates of eating disorders (Ioakimidis et al., 2011).

This thesis attempts to thoroughly describe previously reported discrete eating patterns during single meals in normal-weight women and to compare them with eating patterns observed in AN and Binge Eating Disorder (BED) patients. In the meantime, it deals with methodological issues encountered during the collection and the assessment of behavioral data from single meals. Thus, it provides a tool simplifying the collection of information on human eating behavior. Based on the findings of this thesis and supplementary work from our group, the development of a hypothetical model providing insights on the brain-eating relationship is also attempted on a theoretical level.

Microstructural meal analysis

In animals, there is a long history of detailed analysis of eating and drinking, going back at least as far as Skinner (1938). The limitations of simple, cumulative measurements around meals (Davis & Smith, 1992) resulted in the development of more comprehensive methods for the detailed quantification of the microstructural characteristics of the meal.

In 1927, Richter pointed out that behavior is displayed in bouts (Richter, 1927). For example, in rats, drinking is organized in bursts of licking separated by pauses (Hill & Stellar, 1951; Stellar & Hill, 1952) and further organized into clusters when rats are fed with a sugar loaded liquid diet (Davis, 1973; Davis & Smith, 1992). While the duration of bursts and pauses were shown to be fairly constant during similar measuring sessions (Davis, 1973; Stellar & Hill, 1952), they can be modulated by different experimental manipulations (e.g., water deprivation and variation in the concentration of sugar), pointing to the direct control of licking initiation and termination by meal specific characteristics (Berridge & Fentress, 1986; Davis, 1996). Further research has since revealed that licking and mouth movement frequency during the bursts are affected by taste (Berridge, 2000; Smith 2000). Finally, the relationship (Fitzsimons & Le Magnen, 1969; Kissileff,

1969) and the pattern similarities (Hill & Stellar, 1951; Jordan, Wieland, Zebley, Stellar & Stunkard, 1966; Stellar, 1967) between drinking and eating in rats have been described in detail.

In humans, meals have long been recognized as the unit of intake (Woods & Strubbe, 1994) and a lot of research has focused on factors that affect their occurrence, initiation and termination (Strubbe & Woods, 2004; Woods, Schwartz, Baskin & Seeley, 2000). By further dissecting meals into their behavioral components, the study of the progression of the meal (Davis, 1989; Kissileff, 2000) has proven useful in providing information about different aspects of the ingestion of food. Various electromyographic and other oral sensor techniques have offered information about the behavioral details of the meal (Bellisle & Le Magnen, 1980; Stellar & Shrager, 1985) and insights into eating patterns of obese and lean individuals (Bellisle & Le Magnen, 1981; Spiegel, Kaplan, Tomassini & Stellar, 1993), including the effect of food deprivation (Bellisle, Lucas, Amrani & Le Magnen, 1984). However, the invasive nature of the equipment has been a concern (Hennequin et al., 2005) and in many cases, video recording is a better alternative for collecting the data (Bellisle, Guy-Grand & Le Magnen, 2000; Hill, 1974; Llewellyn, Van Jaarsveld, Boniface, Carnell &Wardle, 2008).

Cumulative intake curve

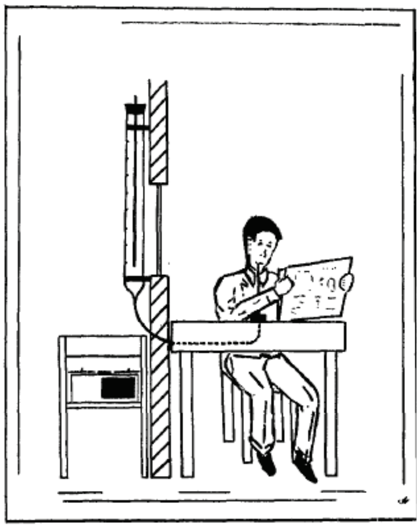
A method for the analysis of single meals in humans was inspired by the research on rats' drinking (Stellar & Hill, 1952). Thus, the "Drinkometer" (Figure 1A) was developed for continuous recording of fluid intake in humans under a variety of experimental conditions (Jordan, et al., 1966; Stellar, 1967). Meyer & Pudel (1972) used a modified version of the same equipment to describe the eating patterns of obese and lean individuals and associated the differences with satiation, being the first to suggest that the cumulative intake curve (CIC) could be modeled by a quadratic equation. Thus the CIC is mathematically expressed as $y=kx^2+lx$, where y is the amount of food ingested, x is the time and the k-coefficient is related to the change of the speed of eating over the course of the meal and the l-coefficient is related to the initial speed of eating.

The procedure and the equipment was refined by Kissileff and his colleagues (Figure 1B), who adapted the machine to solid food diets, renaming it the "Universal Eating Monitor" (Kissileff, Klingsberg &Van Itallie, 1980). They formalized the modeling of intake over time by the use of quadratic equations (Kissileff et al., 1980; Kissileff, Thornton & Becker, 1982), theorizing about the relationship among the equation's coefficients and biological processes. Subsequent variants of the same equipment include the "VIKTOR" (Barkeling, Rössner & Björvell, 1990) and the "Sussex meal pattern monitor" (Yeomans, 1996). The development of the Mandometer® (Figure 2), build on the same principles, added mobility and support for real-time feedback to the user, allowing experimental manipulation of the pattern of the meal (see Materials and Methods).

Hubel and colleagues (Hubel, Laessle, Lehrke & Jass, 2006) have shown that quadratic equations reliably model intake across the meal. Their results, combined with other reports, reveal that curve characteristics are stable across an individual's meals (Barkeling, Rössner & Sjöberg, 1995; Westerterp-Plantenga, 2000). However the rate of deceleration varies among different groups of individuals and across conditions (see below). Thus, the adaptability of the CIC makes it a valuable tool for the analysis of meals (Westerterp-Plantenga, 2000).

In other studies, the l-coefficient has been related to the "eating drive", activated by oral stimulation at the beginning of the meal (Davis & Levine, 1977), or alternatively by the "desire to eat" (Yeomans, 1996; Yeomans, Gray, Mitchell & True, 1997), which depends on palatability (Bobroff & Kissileff, 1986). Also, the eating pattern and, thus, the CIC is affected by the type of the served food, with liquid meals characterized by more linear CICs than solid meals (Guss & Kissileff, 2002). Preparatory and satiety-related components of a meal are affected by smell conditioning (Yeomans, 2006), while meal termination is accelerated by increased retro-nasal aroma release (Ruijschop et al., 2011). Besides, eating rate has been proposed to be a helpful phenotype for the prediction of fat adiposity in children, where higher eating speed was correlated with higher Body Mass Index (BMI; Llewellyn et al., 2008). Furthermore, we have reported sex differences in the CIC characteristics, men being more decelerated than women (Zandian, Ioakimidis, Bergh, Leon & Södersten, 2011; see also Barkeling et al., 1995). Finally, food deprivation prior to the meal affects its CIC characteristics, women eating more linearly and men less so (Zandian et al., 2011).

A



B

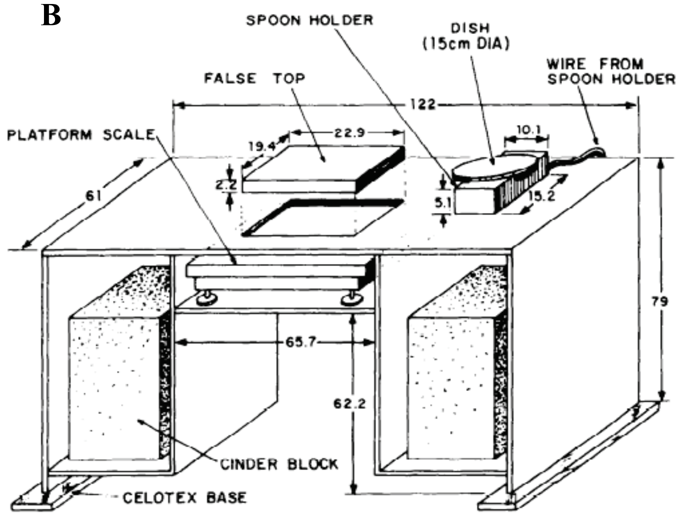


Figure 1. The Drinkometer (A) for analysis of intake of liquid diets and the Universal Eating Monitor (B) for analysis of intake of liquid and solid diets.

Eating styles

The first to describe two different types of CIC were Meyer & Pudel (1972). In normal, underweight and obese individuals the rate of eating remained stable across the meal in one group of subjects, whereas in another there was a reduction in the speed of ingestion towards the end of the meal. Hence, the speed of eating is constant in the first group and decelerated in the second.

The authors linked the decelerated model with the "biological satiation curve", associated linear eating with a disturbed perception of satiation and noted a higher percentage of linearity among the over- and underweight. (We use satiation for the feeling of fullness that develops during the meal. This is in contrast to satiety, which denoted the feeling of fullness between meals.) Although these findings have been replicated (e.g., Adams, Ferguson, Stunkard & Agras, 1978; Barkeling, Ekman & Rössner, 1992; Bellisle & Le Magnen, 1981; Kaplan, 1980; Lindgren et al., 2000), they were not confirmed by a number of other studies (Barkeling et al., 1995; Laessle, Lehrke & Duckers, 2007; Llewellyn et al., 2008; Westerterp, Nicolson, Boots, Mordant &

Westerterp, 1988; Westerterp-Plantenga, Wouters & Ten Hoor, 1990; Zijlstra et al., 2011). Westerterp-Plantenga and colleagues (1988; 1990) suggested that those eating at a decelerated speed should be referred to as Decelerate Eaters and those eating at a constant speed should be referred to as Linear Eaters and reported a correlation between linearity of eating and the cognitive restraint parameter. Dietary restraint is a cognitive process expressed as a drive to control body weight by limiting food intake (Polivy & Herman, 1985). It does not strictly relate to the way a person eats, but refers to the attitude towards food and has even, and surprisingly, been suggested to be outside of physiological control (Bryant, King & Blundell, 2008; see discussion in: Zandian, Ioakimidis, Bergh & Södersten, 2009). Interestingly, cognitive restraint is significantly reduced once Linear Eaters have practiced eating and adopted the decelerated pattern of eating pattern (Zandian et al., 2009), raising questions about the cause-and-effect relationship between eating behavior and cognitive restraint. Although both eating styles appear in normal-weight individuals, it is possible that obese patients, e.g., patients with BED, are Linear Eaters (Westerterp et al., 1988). Reviewing cumulative meal data for BED patients, Walsh & Boudreau (2003) noted that disturbed eating is not limited on binge eating episodes, but spreads across all the patients' meals. However, there is a lack of detailed information on eating styles in this group of patients (Walsh, 2011).

Unlike obesity, there are only a few reports concerning the eating style of eating disorder patients (Walsh, 2011). While there are numerous reports of eating parameters based on external questionnaires (e.g., Wardle, 1987; Wardle et al., 1992), few efforts have been made to describe the eating behavior of these patients. Meyer & Pudel (1972) initially referred to an underweight group of subjects, including mainly anorexic patients, but provided no information about the individuals and did not report details of the pattern of their eating. Hadigan and colleagues (2000) reported that anorexic patients had cumulative intake with fewer calories than controls upon admission to the clinic but no other details. Differences in cumulative intake of AN patients before and after standard treatment have been reported (Mayer, Schebendach, Bodell, Shingleton & Walsh, 2011; Sysko, Walsh, Schebendach & Wilson, 2005). Thus, the total intake increased after the treatment, but it remained lower than intake in the control groups. An attempt to predict the one year outcome in weight-restored AN patients by examining their dietary habits upon weight restoration reported a lower risk of relapse in patients with normalized dietary habits on remission (Schebendach et al., 2008; 2011). By teaching AN patients to eat using Mandometer®, we have normalized their eating behavior, restored their health and prevented relapse over a five year period of follow-up (Zandian et al., 2007).

Chewing, mouthfuls and pauses

While chewing is an important part of the ingestion of food, it has received limited attention in studies of the microstructure of meals, most likely because of technical constraints. Bellisle & Le Magnen (1980;1981) analyzed meals using the "Edograms", i.e., oscillographic recordings of chewing for subjects consuming food items of predefined shape/size and varied palatability (white bread pieces with different tasting pastes) and noted the occurrence of chewing bouts, followed by swallowing and a short pause before the intake of the next food unit (Bellisle et al., 2000). The authors reported an increase of chewing time (but not an increase in "chewing movements") per food unit and an increase of the interval between mouthfuls at the end of the meal, irrespective of palatability. In another two studies based on consumption of standardized bread-based food items, chewing was analyzed using electromyography, but no chewing results across meals were reported (Spiegel, 2000; Spiegel et al., 1993). On average, in meals with smaller food units, the subjects chewed the food longer at an increased frequency, while the size of the food units did not affect the pauses between bites (Spiegel et al., 1993) and the total consumption in the meal. Elsewhere, experimental prolongation of the pauses between bites only marginally affected the size of a meal, but inconsistencies in data reporting and small sample size make these results difficult to evaluate (Kaplan, 1980).

By contrast, Zijlstra and colleagues (Zijlstra, De Wijk, Mars, Stafleu & de Graaf, 2009) reported clear effects of mouthful size and oral processing on the amount of food ingested; lower weight and longer processing time per mouthful result in smaller meals of a semi-liquid, chocolate based drink. Additionally, the size of the mouthfuls decreased over the course of the meal. Westerterp et al. (1980; 1988) reported "chewing time per bite" per individual over the course of the meal using a concealed camera they but did not present data on the progression of this variable across the meals. Mouthful size was stable across meal quarters among linear eaters, decreasing across time among decelerated eaters, while the mouthful frequency remained stable for both groups. In all these studies, the results indicate intra-individual stability of chewing frequency, even across meals with different kinds of food. The size of the mouthful has been examined in people of different body weights with mixed results (Laessle et al., 2007; Llewellyn et al., 2008; Zjilstra et al., 2011). Higher viscosity for liquid food reduces bite size (De Wijk, Zijlstra, Mars, de Graaf & Prinz, 2008) and it has been shown that easily ingested food the total energy intake is higher (Viskaal-van Dongen, Kok &de Graaf, 2011). As whole, methodological differences, diverse groups of participants, a wide variety of test foods and incomplete data reports make conclusions about the relationship among cumulative food intake, mouthful sizes, chewing patterns and pause duration difficult.

Chewing

Mastication is an important issue in dentistry (Soboļeva, Lauriņa & Slaidiņa, 2005A; 2005B). Studies in this field are concerned with chewing movements (rather than meals) and use diverse, usually invasive, techniques with high resolution to describe chewing. These studies use both natural (e.g., Agrawal, Lucas, Bruce & Prinz, 1998) and model foods (e.g., Grigoriadis, Johansson &Trulsson, 2011). The characteristics of chewing sequences have been described thoroughly in animals (e.g., Ootaki et al., 2004; Schwartz, Enomoto, Valiquette & Lund, 1989). Similarly, in humans, chewing can be divided into functional phases based upon characteristic jaw movements, recorded electromyographically (Hiiemae, et al., 1996). These movements adapt depending upon the characteristics of the food (Woda, Mishellany & Peyron, 2006; Wintergerst, Throckmorton & Buschang, 2008) and the subject (Woda, Foster, Mishellany & Peyron, 2006). Thus, the hardness and rheological characteristics of the food and the size of the bolus of the food affect chewing. In the future, development of less invasive equipment with high resolution might allow analysis of chewing in a real-life setting in further detail. For instance, chewing cycles can be recorded based on the sounds of mastication (Amft, Kusserow &Tröster, 2007).

Neurobiology of chewing and mood

It is widely accepted that the rhythmic jaw movements of chewing and licking are controlled by a pattern generator located in the hindbrain (Dellow & Lund, 1971), between the caudal facial nucleus and the trigeminal motor nucleus (Kogo, Funk & Chandler, 1996; Lund & Kotla, 2006; Travers, Herman & Travers, 2010). It is clear that the behavioral expression of the pattern generator is affected by peripheral sensory feedback, probably mediated via adjacent areas such as the serotonin cells in raphe nuclei (Hornung, 2003; Kogo et al., 2006; Stephenson, Hunt, Topple & McGregor, 1999). The activation of chewing according to the sensory characteristics of the food, facilitated by the pre-activation of the masticatory area of the cerebral cortex (Lund & Kotla, 2006; Masuda et al., 1997; Ootaki et al., 2004), suggests the existence of a neural network that is engaged in eating (reviewed in Ioakimidis et al., 2011). For example, during chewing gum use the ventral part of the prefrontal cortex is activated (Kamiya et al., 2010), and experimental activation of that area has been shown to evoke increased activity in the serotonin cell groups in the raphe nuclei (Celada, Puig, Casanovas, Guillazo & Artigas, 2001). Because the raphe nuclei are engaged in a variety of cognitive and emotional processes (Cools, Roberts & Robbins, 2008; Fumoto et al., 2010; Ögren et al., 2008), we have hypothesized that chewing can cause mood changes (Ioakimidis et al., 2011). Supporting evidence is provided by the well known calming effect of breast-feeding in infants (Febo, Numan & Ferris, 2005), the early observation that chewing is relaxing in adults (Hollingworth, 1939) and the more recent report of reduced anxiety after the use of chewing gum to buffer the effect of an acute experimental stress (Scholey et al., 2009). In addition, the rate of chewing affects salivary cortisol levels in a "dose-dependent" manner (Tasaka, Tahara, Sugiyama & Sakurai, 2008). Interestingly, military personnel and professional athletes report high chewing gum use in stressful situations (Wrigley Athlete Study, 2008). The US military forces have been equipped with chewing gum since the first World War (Koehler, 1958) and chewing gum is part of the modern military meal (www.mreinfo.com/us/mre/mres.html). Conversely, the negative effects on mood after disturbances in parts of the chewing network (Stephenson et al., 1999) and the comorbidity of bruxism and mood (Manfredini, Ciapparelli, Dell'Osso & Bosco, 2005) and eating disorders (Vetrugno et al., 2006), support the possible association between chewing and negative mood as well.

Summary

From these introductory considerations it is clear that eating behavior has been studied for a considerable period of time and that humans can be divided into Decelerated and Linear Eaters on the basis of the CIC. Linear Eaters are clinically interesting because they may be at risk of losing control over food intake and, therefore, over body weight. Eating behavior, however, is chewing and swallowing and rather than measuring these, the CIC is a model of cumulative food intake based on information on weight loss from a scale. Previous attempts to measure both chewing and cumulative intake have yielded inconsistent results and in view of the possible role of chewing, not only in food intake but in emotional and cognitive functions, comprehensive study of eating behavior is of interest.

This thesis has studied eating behavior in women because our aim is to understand and treat eating disorders, which mainly affect women. Based on the hypothesis that eating behavior has an important role in the development of disordered eating the thesis has the following aims.

Aims

1. To describe decelerated and linear eating in detail
2. To examine the effect of increasing or decreasing the speed of eating on eating behavior
3. To examine the eating behavior of AN and BED patients
4. To synchronize data on food intake with chewing
5. To analyze chewing in Decelerated and Linear Eaters
6. To formulate a theoretical model for the relationship among eating, chewing and the brain

MATERIALS AND METHODS

Participants

Paper I

Forty-seven normal-weight women (age = 21.2 [19.5-23.1] years; median [range]) with a BMI = 22.2 [20.2-24.3] kg/m2 were recruited by advertisement on a nearby college campus. They completed a health questionnaire to ensure that they were healthy, without a history of eating and anxiety disorders, non-smokers, without food related allergies. Pregnant and lactating women, vegetarians and athletes were excluded. An additional group of thirty women matched for age and body weight was used to test the reliability of the method.

Paper II

Twenty-nine normal-weight women were recruited from a nearby college campus as described above. They were screened according to their rate of deceleration in a control test and sixteen were selected for further testing (age = 19.8 [17.8-24.3] years, BMI = 21.7 [18.5-24.6] kg/m^2).

Sixteen women with AN (age = 14.6 [12.3-26.7] years, BMI = 15.3 [13.4-16.9] kg/m^2, time since diagnosis = 2.6 [0.3-9.5] years, number of previous treatments = 1.5 [0-4]) who were consecutive referrals to our clinic during a period of 6 months were included in the study.

Twelve women with BED (age = 32.2 [17.3-63.3] years; BMI = 39.5 [31.2–62.8] kg/m^2, time since diagnosis = 15.1 [2.2-45.5] years, number of previous treatments = 3 [0-6]) were similarly recruited.

Both patient groups were diagnosed using DSM-IV criteria.

Paper III

Six normal-weight women (age = 24.2 [23.2-24.7] years, BMI = 23.5 [21.2-24.5] kg/m^2) were recruited as described above to participate in the procedure for the validation of the video quantification of the chewing. The participants were visually inspected by a trained dentist for the absence of any outstanding dental conditions.

A similarly selected, matched group of eleven women (age = 22.3 [18.1-24.8] years, BMI = 22.3 [18.6-24.7] kg/m^2) participated in the second experiment. In addition to the previously presented inclusion criteria, all women had a good dental health (self reported absence of dental problems and regular dental controls). They were selected for having k coefficients around -0.25 in the CIC, i.e., between decelerated and linear eaters.

Paper IV

Eighteen normal-weight women were recruited as above (including good dental health). The women were divided into decelerated (n = 9, age = 24.2 [20.7-24.8] years, BMI = 21.5 [20.6-

24.5] kg/m^2, k<-0.3) and linear (n =9, age = 23.9 [20.2-25] years, BMI = 21.1 [19.5-24.2] kg/m^2, k>-0.2) eaters.

A matched sample of 21 normal-weight women was also recruited. They were grouped as decelerated (n = 10, age = 23.6 [19.6-24.6] years, BMI = 20.9 [18.5-24.5] kg/m^2, k<-0.3) and Linear Eaters (n = 11, age = 22.8 [19.5-24.3] years, BMI = 21.4 [19.9-23.7] kg/m^2, k>-0.2) as above.

Food

Food was precooked and reheated before the test meals. Papers I, II and the second group in Paper IV, used a homogenous mix of grilled chicken fillet in cubes (maximum size of the pieces: 2.2 g) with curry-rise and vegetables pieces ("*Nasigoreng*", Findus AB, Bjuv, Sweden: 400 kJ, 4.5 g protein, 15 g carbohydrate and 18 g fat/100 g). Paper III and the first group in the Paper IV used a homogenous mix of grilled chicken fillet and vegetable pieces (maximum weight per piece: 2.6g and 1.2 respectively; "Grönsakspytt med kyckling", Findus AB, Bjuv, Sweden: 426 kJ, 10.7 g protein, 8 g carbohydrate and 2.5 g fat/100 g). The food was selected based on availability, mechanical characteristics (low itemization, small size of components, homogeneity and moderate hardness) and palatability (relatively blunt with acceptable taste).

Mandometer®

Figure 2. Mandometer®, a personal computer connected to a scale recording the weight loss of a plate during the meal. Feedback on how to eat can be provided on the touch screen.

The Mandometer® is a scale connected to a portable computer (Figure 2). It is used for the detailed analysis of single meals, based upon principles first developed in research on rats (Hill & Stellar, 1951) and adapted for research of drinking (Jordan et al., 1966; Stellar & Hill, 1952) and eating in humans (Kissileff et al., 1980; Meyer & Pudel, 1972).

The user places a plate on the scale and puts food on the plate. During the meal, the weight loss on the plate is recorded by the computer every second. A quadratic equation is fitted on the generated weight-loss data series, producing the CIC in real time.

Mandometer® has two modes of operation. During control meals, the user receives no feedback (i.e., empty screen) and the device is used for recording. In test meals, real time feedback, in the form of a weight-loss/time graph is provided to the user. A CIC of predetermined weight/time (i.e., a training curve) is presented on the screen and the user adapts her/his eating behavior to the proposed meal pattern (Figure 3). Deviations >15% from the training curve cause voice and text alerts from the computer, urging the user to eat faster or slower.

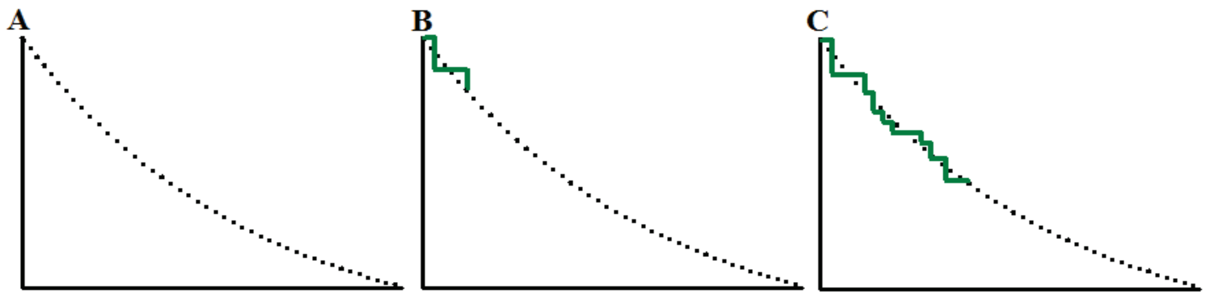


Figure 3. Progression of a meal (green line), following a CIC (dashed curve) on Mandometer®.

Additionally, the Mandometer® features a revised Borg scale (Borg, 1982) and users are asked to rate their satiation on the scale form 0 (nothing at all) to 100 (maximal satiation), which is displayed on the touch screen.

Experimental procedure

Eating

All the meals were served between 11:30 and 13:00 (i.e., typical "lunch" time in Sweden). The women agreed to a certain time of breakfast the morning before the test meals. The ingested amount and the type of food were agreed upon at the beginning of the studies (subject-specific arrangements) and remained stable through consecutive tests. Afterwards, the participants agreed to abstain from eating and drinking, except water. The patients went through a similar procedure.

The recording mode on Mandometer® was used in all Papers to assess the baseline eating pattern and for "Short" and "Interrupt" meals in Paper I. The “Short” meals were reduced by 40%, compared to the control meals. The “Interrupt” meals included one-minute pauses for every 60g of intake. In Papers I and II meals with predefined training curves had individually increased (ER+) or decreased (ER-) rates of eating. The model CICs were preprogrammed with the same duration and 40% more (ER+) or 30% less (ER-) food, compared with the individual’s baseline meals. The experimental conditions are summarized in Table 1.

Table 1. Experimental conditions

	Meal type	Time	Amount of food	Satiety ratings	Food	Special features
Paper I						
Healthy women						
	Control	Unlimited	Unlimited	1 min intervals	Chicken & rice	None
	Short	Limited (Control-40%)	"	"	"	External alarm
	ER+	Limited (same as Control)	Limited (Control+40%)	"	"	Training curve
	ER-	"	Limited (Control-30%)	"	"	"
	Interrupt	Unlimited	Unlimited	"	"	1 min pause / 60g
Paper II						
Healthy women						
	Control	Unlimited	Unlimited	1 min intervals	Chicken & rice	None
	ER+	Limited (same as Control)	Limited (Control+40%)	"	"	Training curve
	ER-	"	Limited (Control-30%)	"	"	"
AN/BED patients						
	Control	Unlimited	Unlimited	Before/after meal	Restaurant food	None
Paper III						
Healthy women						
	Control	Unlimited	Unlimited	Before/after meal	Chicken & vegetables	External cameras
Paper IV						
Healthy women - Group 1						
	Control	Unlimited	Unlimited	Before/after meal	Chicken & vegetables	External cameras
Healthy women - Group 2						
	Control	Unlimited	Unlimited	Before/after meal	Chicken & rice	None