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**GASTRIC MYOELECTRIC ACTIVITY IN OBESE PARTICIPANTS WHILE
VIEWING FOOD IMAGES AND EATING FOOD**

A Thesis in

Psychology

by

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Abstract

Purpose: This study observed the reactions of the stomach to food viewing and food eating in obese people. **Method:** The participants were 20 obese and 20 normal-weighted undergraduate students, who were required to fast for at least 4 hours preceding the experiment. They viewed a video clip of food for 6 minutes; consumed food till full; and viewed the same video clip of food 30 minutes after the meal. Cutaneous electrogastrograms (EGGs) were recorded during the entire procedure. Questionnaires concerning the participants' perception of somatic and affective changes during the tasks were completed. **Results:** The percentages of bradygastric, normal and tachygastric EGG activity were very similar between obese group and normal-weighted group during the baseline period. During the first food image viewing, an increase in normal gastric activity and a decrease in bradygastric activity occurred in both groups, but obese participants showed less of an increase in normal gastric activity than normal-weighted participants and the percentage of bradygastric activity was higher in obese group than in the normal-weighted group. During the 30-minute period following the meal, a higher percentage of bradygastric activity and a lower percentage of normal gastric activity were observed in obese participants than in normal-weighted participants. Furthermore, obese participants showed a different pattern of change in 3 cpm and bradygastric activity than normal-weighted participants during the 30 min. The whole 30-min period was divided into five 6-min periods. Obese participants and normal-weighted participants reacted similarly to the second food image exposure, except that a higher increase in 3 cpm activity was observed in the normal-weighted group in the period after the termination of viewing the food images. **Conclusions:** These data suggest that obese people react to

food images and to food eating differently from normal-weighted people. EGG activity in obese participants needs further investigation.

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INTRODUCTION

1.1. Prevalence and Impact of Obesity

Obesity is an epidemic that is characterized by excess body fat and that contributes to numerous chronic diseases and mortality.

The prevalence of overweight and obesity has escalated in the last 25 years in the United States (Flegal, et al., 2002). According to recent National Health and Nutrition Examination Survey (NHANES 1999-2000), the age-adjusted prevalence of obesity (BMI [Body Mass Index] ≥ 30) was 30.5% in 1999-2000 compared with 22.9% in NHANES III (1988-1994; $P < .001$); the prevalence of overweight (BMI:25.0-29.9) also increased during this period from 55.9% to 64.5% ($P < .001$); and extreme obesity (BMI [Body Mass Index] ≥ 40) increased significantly in the population, from 2.9% to 4.7% (Kuczmarski, et al., 1994). Compared to the data from NHANES (1976-1980), the rate of overweight or obesity increased about 20%.

In addition, obesity is also prevalent in Europe. The most reliable comparative data on the prevalence of overweight and obesity across Europe come from the MONICA (Multinational MONItoring of trends and determinants in CARDiovascular disease) Project (1988). The recent data show that the prevalence of obesity in most European countries has increased by about 10-40% in the past 10 years, ranging from 10-20% in men and 10-25% in women. Furthermore, in Asia, a marked rise in overweight and obesity is being seen in all populations (Popkin, 1994). Therefore, obesity is a world wide problem. A recent estimate is of more than 1 billion overweight adults, and at least 300 million of them obese, based on a global report of the World Health Organization (WHO, 2002).

Overweight and obesity have a very negative impact on health. Individuals with obesity are at a higher risk of developing one or more serious medical conditions, which can cause poor health and premature death (Flegal, et al., 2005). Obesity significantly increases the risk for hypertension, coronary heart disease, lipid disorders, type 2 diabetes mellitus, stroke, gallbladder disease, hypercholesterolemia, osteoarthritis, sleep apnea and respiratory problems, chronic joint pain, back injury, and several cancers (Willett, et al., 1999). The World Health Organization World Health Report estimated that around a third of Coronary Heart Disease (CHD) and ischaemic stroke and almost 60% of hypertensive disease in developed countries were due to overweight and obesity (WHO, 2002). Physical dysfunctions and decreases in quality of life caused by overweight and obesity are also of great importance although they may not be bound to any specific diagnosis. For example, a close association has been observed between overweight and obesity and health-related quality of life (HRQOL) in patients with chronic conditions, after accounting for the effects of depression and medical comorbidities (Fontaine, 2002). Besides, obesity causes a tremendous economic burden to society. The yearly medical cost of obesity is conservatively estimated at \$123 billion in the United States, with consumers spending nearly \$50 billion more on products and services to induce weight loss (Thompson & Wolf, 2002).

1.2. Definition of Obesity

The most commonly used method today for classifying an individual as overweight or obese is based on body mass index (BMI), a value that is determined by dividing body weight (in kilograms) by the square of height (in meters). In adults, overweight is defined

by a BMI of ≥ 25.0 kg/m², and obesity is defined by a BMI of ≥ 30.0 kg/m², regardless of gender (World Health Organization Expert Committee, 1995).

The use of BMI to assess weight-related health risk has gained international acceptance because of the associations between BMI and adiposity. The underlying assumption is that most variation in weight for persons of the same height is due to fat mass. A graded classification of overweight and obesity using BMI values provides valuable information about increasing body fatness.

In addition, BMI is highly correlated with disease risk (Must, et al., 1999) and mortality (Solomon & Manson, 1997). The World Health Organization distinguishes several BMI categories based on increasing health risks. Typically, a BMI of 18.5 to 25 is considered 'healthy', but an individual with a BMI of 25–29 is considered as 'increased risk' of developing associated diseases and one with a BMI of 30 or more is considered as 'moderate to high risk' (see table 1, WHO, 1997).

Weighth Category	BMI (Kg/m²)
Underweight	<18.5
Normal Weight	18.5-24.9
Overweight	25.0-29.9
Obese	≥ 30
Obese Class1	30-34.9
Obese Class2	35.0-39.9
Obese Class3	≥ 40.0

Table 1. World Health Organization's Body Mass Index (BMI) Categories Based on Increasing Health Risks.

Furthermore, the simplicity of determining BMI, which can be calculated from measurements or self-reported values of height and weight, enables comparisons of weight status within and between populations throughout the nation and worldwide. It

also permits identification of priorities for intervention at an individual or community level and for evaluating the effectiveness of such interventions.

The major limitation of BMI is that it does not differentiate between weight that is fat (i.e., fat mass) and weight that is muscle (i.e., fat-free mass), and therefore may lead to misclassification of very muscular individuals as overweight. In addition, older adults may appear to have a healthy BMI despite having excess fat and reduced muscle mass.

1.3. Etiology of Obesity and the Alterations of Gastrointestinal Motility in Obesity

1.3.1. Etiology of Obesity

The underlying etiology for obesity remains obscure except in rare cases of primary endocrine anomalies. People usually consider obesity as a heterogeneous disorder which results from an imbalance between energy intake and energy expenditure (Blundell & Gillett, 2001). However, what causes this imbalance remains relatively poorly understood. The difficulty is due to the fact that obesity results from a combination of genetic, behavioral, environmental, physiological, social, and cultural factors. Body weight seems to be determined by an interaction of these factors acting through the physiological mediators of energy intake and expenditure.

Overconsumption of food relative to calorie requirement is a common feature of most of obese patients. Since all nutrients are absorbed in the gastrointestinal (GI) system, the potential role of the gastrointestinal system in developing and maintaining obesity has caused increasing attention in recent years.

Normal function of the gastrointestinal system depends on the regulation of a complex, interacting network composed of well-coordinated gastrointestinal motility, gut

regulatory peptides and hormones, as well as the autonomic and enteric nervous systems. These regulatory mechanisms not only modulate the rate at which nutrients are processed in the gastrointestinal tract, but it has also been suggested that gastrointestinal motility may participant in the regulation of appetite and satiety (Deutsch, 1985).

Gastrointestinal motility is closely linked to the rate at which nutrients become systemically available. Various segments of the GI tract (esophagus, stomach, and intestines) coordinate in a complex yet precise way to control the process of food consumption, digestion, nutrient transmission and absorption. The current working model suggests that food ingestion stimulates both mechanical (stretch or pressure) and chemical receptors within the stomach that activate vagal fibers (Ritter, 2004). These signals act to decrease appetite and promote satiety. Therefore, well co-coordinated gastrointestinal motility is essential to determine not only how much and how often people eat, but also what kind of food people prefer to eat. Any change in gastric motility may reflect disturbances in the regulatory mechanisms of the gastrointestinal tract, or predispose to overeating and obesity. Altered GI motility has been frequently observed in obese patients (Park & Camilleri, 2004). The significance of these GI motility alterations in obesity is not fully understood, but they have been considered as potential contributing factors in the development and maintenance of obesity and changed eating behavior.

1.3.2. Gastric Emptying of Liquid and Solid Food

The motor functions of the stomach are primarily to (1) mix the ingesta with gastric secretion, (2) agitate the mixture to break down the food into small-sized particles, and (3) empty them into the duodenum at a rate that allows efficient digestion and absorption of food components in the small intestine (Schuster, Crowell & Koch, 2002).

The pattern of gastric emptying and the motor mechanisms differ between solid and liquid food (Kelly, 1980). The gastric motility response to liquids begins with the rapid movement of liquids from the fundus to the antrum, and filling of the gastric antrum. Recurrent gastric peristalsis empties liquids into the duodenum. At the same time, the fundus slowly contracts, which creates a pressure gradient between the stomach and the duodenum. In general, for liquid meals, 50% of gastric emptying can be achieved within 20 to 30 minutes. Liquids of increasing caloric density or viscosity are emptied more slowly than non-nutrient liquids.

Emptying of solid food requires a series of gastric motility responses involving initial grinding through antral contractions, retropulsive and propulsive actions of the distal stomach. Unlike liquid emptying, which mainly relates to the tone of the gastric fundus, emptying of solid also depends on antral contractility. During this process of grinding, solid food is converted to small particles, but very little gastric emptying occurs; this period is called the “lag phase”. The lag phase is followed by a linear phase of emptying, which is similar to that of high calorie or viscosity liquid meals. For convenience, the lag phase is defined as the time for 10% of the gastric solid contents to be emptied (Halkar, et al. 1999), and it is usually around 30 minutes; but the length of a lag phase varies with the meal volume, caloric density, fat content, food particle size, and gastric motility, etc.

1.3.3. Alterations of Gastric Motility in Obesity

1.3.3.1. Gastric Emptying

Many gastric motility studies that are mainly focused on the relationship between gastric capacity, gastric accommodation, gastric emptying and gastric

myoelectrical activity and obesity have been conducted in recent years. However, the results are far from conclusive. For example, gastric emptying in obese subjects has been investigated in several studies, but they did not show consistent results; some studies have suggested more rapid gastric emptying (Johansson & Ekelund, 1976; Wisen & Johansson, 1992), whereas others have showed normal (Huston & Wald, 1993) or even slower gastric emptying (Maddox, et al. 1989) in obese subjects. The few studies that observed gastric myoelectrical activity in obese people have reported inconclusive results (Riezzo, et al., 1998; Jones, et al., 2001). Therefore, how disturbances in gastric motility are involved in the complex pathogenesis of obesity remains unclear.

Subjects who are taller and heavier were suggested to empty liquid more rapidly than lean people. It was found that in normal-weighted subjects, gastric emptying rate has a positive linear relationship to body size (height or weight) (Johansson & Ekelund, 1976). However, it seems that this relationship does not hold for obese subjects because obese subjects empty their meal faster than what would be predicted from their height (Wisen & Johansson, 1992).

Enhanced gastric emptying might predispose to overeating and obesity. In agreement with this theory, the occurrence of obesity in rats with rapid gastric emptying resulting from ventromedial hypothalamic lesions was observed and abnormal gastric emptying was suggested to be the major cause of the obesity (Duggan & Booth, 1986).

Several studies reported rapid gastric emptying in patients with obesity. In one study, 46 obese and 31 nonobese individuals were matched for age-, sex-, and race. Gastric emptying of solids in the obese group was significantly more rapid than that of the nonobese controls, whereas no difference was noted in the gastric emptying of the liquid

test meal between the two groups (Wright, et al., 1983). In another study, a higher percentage of gastric emptying during the initial 30 minutes after eating in the obese participants was observed, although there were no differences found in the overall 3-hour emptying rate (Verdich, et al. (2000)). Several other studies indicated an increased rate of emptying of the first 50% of a solid meal in subjects with morbid obesity compared with lean subjects matched for age and sex (Naslund, et al., 1998).

These and similar findings are, however, challenged by others who found similar or even slower gastric emptying in obese individuals. For example, no significant differences in gastric emptying of both solid and liquid meals between lean and obese subjects were found in Huston and Wald's study (1993). Three of the studies by Horowitz's research group all demonstrated slower emptying of solid food in obese subjects (Horowitz, 1983; Horowitz, 1986; Maddox, 1989). The slowing in total gastric emptying of solid was due to a prolonged lag time, which is contrary to the findings of other researchers (Verdich, 2000; Naslund, 1998; Glasbrenner, 1993). Delayed gastric emptying of the solid and liquid meals was also observed in one study of gastric and oesophageal emptying of obese subjects (Maddox, et al., 1989).

Furthermore, an evaluation of the possible changes in gastric emptying after treatment and weight loss in obese patients provides some evidence about the characteristics of gastric motility in obese patients. Several studies reported delayed gastric emptying in obese patients after a significant weight loss. For example, obese patients showed a slowing of gastric emptying of solids when a significant decrease of body weight has been achieved at the end of the dietetic treatment (Tosetti, et al., 1996; Verdich, et al., 2000). However, in some other studies, this result was not observed. For

instance, gastric emptying rates before and after substantial weight reduction was similar in Huston's study (1993).

These conflicting results from gastric motility studies in obese people may either reflect the heterogeneous mechanisms of obesity, or could be attributed, in part, to the methodology discrepancies, such as the selection of participants, grouping methods, meal composition, meal size, the blood glucose level of subjects at the time of testing, etc. The results from an investigation after an overnight fast may be different from that conducted in the afternoon. The meal might be too small in some studies for the population under investigation. Few studies have collected such information as weight stability, previous diet history, other obese treatment history or life style of the subjects. Therefore, further well-controlled, validated studies are needed to clarify the potential role of gastric motility in obesity.

1.3.3.2. Electrogastrography (EGG)

Gastric myoelectrical activity is composed of spikes and slow waves. The gastric slow waves originate from the proximal stomach and propagating distally. The gastric slow wave determines the frequency and propagation of the gastric contraction.

In contrast to other intrusive methods of measuring gastrointestinal system activity, the cutaneous EGG is a non-invasive method of recording the electrical activity of the stomach, gastric slow waves. The electrodes that are placed on the surface of the skin over the abdomen record signals from the muscular walls of the stomach that reflect gastric myoelectrical activity (Stern, et al., 1985). The frequency of the EGG is identical when it is recorded from the serosal surface of the stomach as when it is recorded cutaneously (Lin, et al., 2000). Recording from the surface of the skin rather than from

the outer surface of the stomach allows for the collection of reliable, accurate, physiological data using a painless, non-invasive procedure. Since the frequency of the EGG signal is identical to the frequency of the contractions of the stomach when they occur (Xu, Wang & Chen, 2002), EGG provides a reliable method to evaluate gastric motility.

Through spectral analysis, the EGG signal is broken down into its component frequencies and power values are assigned to each of the frequency ranges of interest of gastric myoelectrical activity. In healthy humans, the stomach normally contracts three times every minute; the EGG signal that reflects this normal “slow wave” gastric activity is three cycles per minute (3 cpm). Disturbances in gastric slow wave rhythm have been easily identified from the changes in the frequency of the slow waves. For example, during nausea, the frequency of the EGG signal typically increases to four to nine cycles per minute (4-9 cpm), and generally becomes dysrhythmic.

Some studies suggested that abnormal EGG was related to various gastrointestinal symptoms, such as nausea, vomiting, fullness, etc., and was frequently observed in patients with functional disorders of the stomach, such as gastroparesis, functional dyspepsia, anorexia, etc. (Pfaffenbach, et al., 1997) or in motion sickness patients (Stern, et al., 1985). The abnormalities in gastric electrical activity include uncoupling and gastric dysrhythmia, which usually lead to gastric hypomotility. For example, tachygastria (abnormal gastric slow waves with a frequency of higher than 3.75 cycles/min (cpm)) has been consistently shown to cause hypomotility of the stomach in functional dyspepsia patients (Pfaffenbach, Adamek, Bartholomaeus & Wegener, 1997). Furthermore, a reduction or absence of the expected postprandial increase in the EGG

power was reported to be correlated with delayed gastric emptying and antral hypomotility (Dubois, 1989).

However, the extent to which gastric slow waves change in obese patients is still unknown. Presumably an alteration in myoelectrical activity may partly contribute to the accelerated gastric emptying in obesity or predispose one to overeat or obesity.

Very few studies have been conducted to explore the features of gastric myoelectrical activity in obese patients. One study in obese children observed the postprandial pattern in response to a mixed meal and similar responses in EGG were obtained in obese children compared to normal-weighted children (Riezzo, et al., 1998). Another study in adult patients with morbid obesity has revealed an increase in the percentage of gastric bradyarrhythmia in both fasting and fed state. Accelerated gastric emptying of solids in these obese patients was recorded at the same time (Jones, et al., 2001).

Although tachygastria has been suggested to be associated with gastric hypomotility (e.g. You, et al., 1981), the clinical significance of gastric bradyarrhythmia remains unclear. Several studies have associated gastric bradyarrhythmia with strong antral contractions at a reduced frequency (Chen & McCallum, 1993).

Possibly most compelling evidence for gastrointestinal regulation of body weight comes from the studies of the recently developed obesity treatment using an implantable gastric stimulator (IGS), which has achieved body weight reduction in some obese patients. In 1992 Cigaina et al were the first to investigate the potential for gastric electrical stimulation to induce weight loss (Cigaina, et al., 1996). This study showed that gastric electrical stimulation was effective to inhibit weight gain and food intake in

growing swine. The first human study using a gastric stimulator for the treatment of morbid obesity was performed in 1995, and resulted in decreased food intake and a substantial weight loss (Cigaina, 2002). The rationale for this treatment is to reduce appetite and food intake by controlling gastric myoelectrical activity, which hopefully, alters gastric emptying rate and increases satiety (Chen, 2004). With this method, an implantable gastric stimulator (IGS) is placed subcutaneously and it delivers repetitive electrical pulses to the tissue of the stomach via a pair of electrodes implanted on the seromuscular layer of the stomach along the lesser curvature. An IGS may impair or disturb the intrinsic gastric slow waves and synchronize the whole stomach with a frequency of the IGS. The normal postprandial increase in the amplitude of the gastric slow wave after a meal was significantly reduced with gastric electrical stimulation in a canine study (Ouyang, et al., 2003). This result was confirmed in a clinical study which showed a similar reduction in the usual postprandial increase in the amplitude of the gastric slow waves and an increase in the percentage of tachygastria (Lin, et al., 2002). One recent study showed that this treatment reduced the amount of water and food intake and delayed gastric emptying without inducing any unacceptable symptoms (Yao, et al., 2005).

The success of this treatment can not confirm the existence of any type of abnormal gastric myoelectrical activity in obese patients, but, at least, it suggests that gastric myoelectrical activity has a close relationship with the development or maintenance of obesity. Therefore, it is of importance to explore the pattern of the EGG changes in obese patients.

One technical difficulty in EGG measurement in morbidly obese patients is that it

could be hard to record EGG signal from the skin surface of the stomach due to the unusual thickness of the abdomen and the greater distance between the surface-recording electrodes and the stomach. One study in our lab simultaneously recorded EGG from the abdomen and the wrists. The results showed that the amplitude of the EGG from both abdomen and wrists greatly increased after the onset of eating. And individuals with complete gastrectomies failed to show any 3 cpm EGG signals from either abdomen or wrist (Stern & Stacher, 1982). These findings demonstrate that the EGG may be recorded from the surface of the body some distances from the abdomen.

1.4. Cephalic Phase Response in Obesity

Physiological reactions that occur in the gastrointestinal tract just before ingesting food are referred to as “cephalic-phase responses”, which are mediated by cephalic-vagal gastrointestinal reflexes. Cephalic phase responses (CPRs) of the body are elicited by either exposure to the sensory properties of food (e.g., sight, smell and taste) or by simply the thought of eating. Cephalic phase responses prepare the body for a coming meal. For example, exposure to food increases salivation (Nederkoorn, et al., 2000), gastric activity (Stern, et al., 1989), and insulin release in humans (Teff, et al., 1991). Furthermore, a conversion was observed from a fasting to a feeding pattern of gastric motility (Defilippi & Valenzuela, 1981) and gastric myoelectric activity (Stern, Crawford, Stewart, Vasey, & Koch, 1989). Stern et al. observed the effect of modified sham feeding on gastric myoelectric activity. The participants were asked to chew a hot dog several times and then spit it out. The increase in the amplitude of the gastric slow wave was very similar to that after eating, except the increase after sham feeding was

smaller and returned to baseline more rapidly (Stern, et al., 1989). In another study of sham feeding, both the amplitude and the frequency of the gastric slow wave changed, either as an increase or a decrease in amplitude of slow waves (Chen, Pan, & Orr, 1996). Specifically, the participants who showed an increase in the power and the percent of normal 2-4 cpm slow waves after sham feeding were those same participants who showed a similar increase after feeding. This result indicated that the alteration in EGG after food ingestion is significantly correlated with that after sham feeding.

It has been proposed that the cephalic phase response increases the amount of food that the organism is able to eat within one meal. This was demonstrated in a study in rats who consumed smaller meals when CPRs were prevented by making the timing of a meal unpredictable (Woods, 1991). Based on this theory, it might be expected that people with large food intake may have larger cephalic phase response than people who are used to a small amount of food every meal.

Schachter's 'internal-external' theory of obesity suggests that obese individuals, relative to those of normal weight, were over-responsive to external cues (food cues) and under-responsive to internal cues (including feeling of hunger and fullness). It may make obesity-prone individuals more likely to overeat and gain weight (Schachter, 1968; Nisbett, 1968). This theory is consistent with the cue reactivity model that is proposed by investigators studying eating disorders. The cue reactivity model of binge eating predicts that cephalic phase responses to food cues are experienced as craving; thereby food cues increase food intake and induce other abnormal eating behaviors (Wardle, 1990). In other words, binge eating disorder patients respond to food cues with enhanced cephalic phase responses, which thus increase the craving for the food and thereby make it more difficult

for the subject to abstain from eating. The findings demonstrated that when subjects were exposed to food cues, more craving and physiological reactivity was found in the ones with abnormal eating patterns (Karhunen, et al., 1997). In addition, greater salivation response to food exposure and higher sympathetic arousal was observed in patients diagnosed with bulimia nervosa than in non-eating-disordered participants (Legenbauer, et al., 2004). In another study, blood pressure and electrodermal activity significantly increased in binge eating subjects during food exposure (Vogele & Florin, 1997).

Whether the cue reactivity model holds for obese people who overconsume food is far from clear and the questions of whether obese people experience larger cephalic phase responses and what role the cephalic phase responses may play in the development and maintenance of obesity can not be answered at present due to few studies in this area. Further research is required to clarify the relationship between cephalic phase responses and obesity.

1.5. Satiety Phase in Obesity

As stated earlier, the role of potential alterations in gastrointestinal motility in the pathogenesis of obesity is presently not clear. The stomach has been considered to be the most important organ in the gastrointestinal tract in the regulation of satiety. Feelings of hunger and satiety have long been associated with gastric motor and sensory functions. At the turn of the century, Cannon and Washburn described contractions of stomach, which were believed to be the basis for feelings of hunger (Cannon & Washburn, 1912). Boring reported that mechanical distension of the stomach with a balloon could elicit a feeling of fullness (Boring, 1915). As we know now information about the state of

fullness in the stomach is conveyed to the brain by the brain-gut axis; and the gut regulatory peptides, hormones and the autonomic nervous system play a vital role in the cessation of food ingestion and the initiation of the next meal. Furthermore, well-controlled gastric emptying prevents the gut from being overfilled with hyperosmotic solutions and nutrients in excess of the digestive capacity as well as controls the duration of the presence of chyme inside the stomach and maintains the feeling of satiety, which may help reduce meal ingestion frequency.

Rapid gastric emptying reduces the satiety signals from the stomach, which not only lessens the satiation feeling, but also shortens the interval between the consecutive meals (Park & Camilleri, 2004). It is conceivable that altered gastric emptying or abnormal gastric myoelectric activity during ingestion might be associated with reduced postprandial fullness, hence, increase food intake frequency. Furthermore, physiological slowing of gastric emptying appears to be linked with increased feelings of satiety and termination of food intake. Gastrointestinal symptoms of functional dyspepsia (FD) patients with abnormal gastric motility are a good demonstration that gastric motility can influence food intake behavior. FD patients often report upper GI symptoms (early satiety, nausea, and vomiting, etc.) and less food intake. This was suggested to be due to the persistent delayed gastric emptying which prolonged the presence of food within the stomach (Tack, et al., 2001).

Few studies have reported postprandial symptoms and gastric motility in obese subjects, although much attention has been given to the reduced satiation associated with an increased gastric capacity in obesity (Geliebter & Hashim, 2001). One study reported the association of body mass and gastric volumes (fasting and postprandial) with satiation

and postprandial symptoms among 134 participants (81 women and 53 men). In this study, increased body mass was associated with delayed satiation (higher maximum tolerated volume). And this relationship was independent of the influence of increased fasting gastric volume on satiation. In addition, increased body mass index was associated with lower fullness and higher bloating and pain scores 30 minutes after a meal (Delgado-Aros, Cremonini & Castillo, 2004).

Therefore, further studies are needed to explore the regulatory mechanism of satiety by gastric motility.

1.6. Autonomic Nervous System Activity and Eating

The autonomic nervous system (ANS) includes the sympathetic nervous system (SNS) and the parasympathetic nervous system (PNS). They work together with opposing effects on the gastrointestinal system. Keeping balance between these two systems is essential for the normal function of the gastrointestinal system. For example, during a reaction time (RT) task designed to shift autonomic balance toward SNS and away from PNS activity, the gastric myoelectrical activity showed more tachyarrhythmia than during rest as indexed using EGG recording; in contrast, during a cooling of the face (CF) task designed to shift autonomic balance away from SNS and toward PNS activity, less tachyarrhythmia occurred. These complementary findings lend support to the association of tachyarrhythmia in the stomach with an ANS balance shifted toward SNS activity (Muth, Thayer, Stern, Friedman, & Drake, 1998).

Eating alters the activity of SNS and PNS also. In rats, low level, tonic sympathetic nervous system (SNS) activity predicted high food intake, and high level,

tonic SNS activity predicted low food intake (Bray, 1991). Increased SNS activity may affect gastric myoelectrical or contractile function and alter gastric emptying or gastric electrical rhythms. For example, infusions of epinephrine produce bradygastrias (Kim, et al., 1989) and vagotomy results in tachyarrhythmias. The tachyarrhythmias presumably develop because vagal tone is removed and sympathetic nervous system activity is dominant (Stoddard, et al., 1981). The relationship of eating to PNS tone was investigated in one study and the results showed that ingestion of food increased PNS tone (Uijtdehaage, Stern & Koch, 1992).

1.7. Experimental Design and Hypotheses

The purpose of the present study was to explore the possible differences between obese and normal-weighted participants in response to food stimulation measured by gastric myoelectric activity in three phases of ingestion: cephalic phase, postprandial and satiety phase. An independent group, crossover design was employed in which two groups of participants (obese and normal-weighted group) experienced a series of experimental tasks in the same order.

The whole procedure of the experiment was divided into three parts based on the specific experimental objectives.

The first part was designed to explore the characteristic of cephalic phase response of obese participants. The participants were exposed to appetizing food images and were instructed to imagine vividly that they were eating the food.

Electrogastrography, skin conductance level, heart rate variability and subjective reactivity were measured. All the results from obese participants were compared to age

and sex-matched normal weight controls. The group differences were hypothesized to be evidenced by differences in subjective evaluations of the food images, including how strong the desire was to eat the food in the video clip and how much food each subject expected to eat, and by differences in physiological markers for gastric motility, gastric myoelectric activity. The normal stomach activity that frequently accompanies an increase in appetite was hypothesized to be more evident among the obese group participants than the normal-weighted group. Respiratory sinus arrhythmia (RSA), an estimate of parasympathetic nervous system activity was predicted to be higher among participants in the obese group, and skin conductance level (SCL), an index of sympathetic nervous system activity, was predicted to reveal the opposite pattern of results; that is, SCL was expected to be lower among obese group participants.

The objective of the second part of the study was to explore the pattern of gastric motility alteration in the postprandial period. The participants were instructed to eat a large amount of food till they felt very full. Then they sat quietly for 30 minutes and electrogastrogram and subjective reactivity to food were measured. Relationships between the physiological measurements, subjective states, caloric intake and overeating tendency were examined. It was hypothesized that the EGG would reveal different patterns within the 30 minutes after the meal in obese and normal-weighted participants and this difference, to some extent, would reflect the different motility alteration of the stomach in digesting of food between groups.

The third part of the study was designed to test the hypothesis that obese participants had stronger reactions to food stimulation physiologically and (or) psychologically than normal-weighted participants when they were full (in the satiety

period). The participants were instructed to watch the same video clip about appetizing food and follow the same instructions as in the first part of the study.

Electrogastrography, skin conductance level, heart rate variability and subjective reactivity were measured. Physiological estimates of the electrical activity of the stomach, and of the activity of each branch of the autonomic nervous system were expected to differ in ways that would indicate that the obese participants responded intensively to food images in the satiety period, while normal-weighted participants responded rarely to further food stimulation as they were full.

METHODS

2.1. Participants

Participants were 41 undergraduate students at the Pennsylvania State University who were recruited through the Psychology Department subject pool. Twenty of the participants with body mass index (BMI) between 20 and 24.9 were in the normal weight group (male: 11; female: 9) and the other 21 participants with BMI in between 30 to 34.9 were in the obese group (male: 13; female: 8). There were no significant differences in age and gender between the participants in the obese group and the normal-weighted group. The median, minimum and maximum of BMI were 32.1 (30 -34.5) for the obese group and 21.6 (18.5-24.3) for the normal-weighted group respectively (See Table 1). This classification of obesity by BMI is based on a report by the National Institutes of Health (1998).

All participants were at least four hours fasted when they arrived at the laboratory. They were asked to have a light meal such as a small sandwich or two pieces of toast with juice in their last meal before the experiment. None of them had histories of neurological, cardiovascular, or gastrointestinal disorders. The participants had not been in any kind of obesity treatment. They were required not to take any medication, consume alcohol or caffeine, use drugs, or smoke cigarettes within the 24 hours prior to their arrival. No exercise in the three hours of prior to the experiment was allowed. The Institutional Review Board approved the use of human participants for this study. Each participant signed a written consent form before beginning of the experiment and was given a debriefing statement at the end of the study.

2.2. Measurements

2.2.1. Physiological Measurements

2.2.1.1. Electrogastrogram (EGG) and Respiration

EGG signals were collected from three disposable, ConMed Cleartrace electrodes (#1700-001; Haverhill, MA) placed on the surface of the abdomen, over the stomach. Before placement of the silver/silver chloride cutaneous electrodes, the skin was prepared using Omniprep to abrade dead skin cells so that a better signal could be obtained. The electrodes were attached with high conductivity gel in between the electrode and the skin. Two active electrodes were placed on the internal side of each wrist. The reference electrode was placed even with the umbilicus and approximately 10 cm to the right. The electrodes were connected via an Iso-Z isolation head-stage preamplifier to an EGG amplifier (BMA-931 Bioamplifier, 3 CPM). The filter frequency was 0.016Hz-0.25HZ. The signals from the amplifier were sent to one channel of a two-channel recorder (WR 7400, 3 CPM), and recorded on chart paper at a speed of 1 mm/sec.

Respiration was measured via a solid-state pressure transducer placed around participants' midsection. The signal was sent to a transducer amplifier (PA-931, 3 CPM) then to the second channel of the two-channel recorder. The recorder also produced a written record of participants' respiratory responses at a paper speed of 1 mm/s also. The respiration and EGG signals were also sent to a computer with an analog-to-digital board for digitizing the EGG signal and for EGG software analysis programs (EGGSAS-4 program, 3 CPM).

The recording of respiration was not used as a dependent measure; it was monitored so concurrent respiration artifacts in the EGG signal could be identified and

excluded from the analysis.

2.2.1.2. Cardiometer

Electrocardiograms (EKGs) were collected throughout the experiment in order to obtain an index of cardiac vagal activity. Heart rate signals were collected from three silver/silver chloride electrodes (#1700-001; Haverhill, MA). Two active electrodes were placed on the participant's left side at the V5 or V6 level and the sternum. ECG was sampled at 1000 Hz. The reference electrode was placed on the right side of the abdomen. The recording of the ECG signals was accomplished using the EZ-IBI system (UFI, Morro Bay, CA). The EKG signal was sampled at 100 Hz; and Biolog software, which accompanied the UFI ambulatory monitor, calculated a string of interbeat intervals (IBIs) that were ASCII-converted for statistical analysis (Model BCR 100, Morro Bay, CA).

2.2.1.3. Skin Conductance Level (SCL)

Skin conductance responses were recorded from two silver/silver chloride electrodes (BioTac) placed on the volar surface of the medial phalanx of the index and the ring finger of the nondominant hand. The electrodes were connected to a Gould skin conductance coupler (Model: 8188.2202.00). A constant voltage of 0.5 V was applied across the electrodes in conjunction with 0.05 M NaCl electrode. The signal from the skin conductance coupler was then sent to a Gould 3000 recorder which provided a written record at a paper speed of 1mm/s. The SCL estimates were also continuously displayed from a digital readout on the Gould coupler throughout the experiment.

2.2.2. Subjective Measurements

2.2.2.1. Subjective Evaluation Form

The participants' subjective reactions to food images or food were scored on a

100-mm visual analogue scale (VAS) just after food image exposure and food intake (Flint 2000). The following questions were asked in the questionnaire: How hungry do you feel (very weak-very strong)? How strong is your desire to eat? How full do you feel (not at all- very full)? How much food do you think you can eat now (nothing at all- very much). One more question: “How much did you like the food that you have just taken?” was included in the questionnaire that was completed just after the meal.

2.2.2.2. PANAS

The Positive and Negative Affect Schedule (PANAS) questionnaire was utilized to check the change in mood of the participants in response to food image or to food (Watson, Clark, & Tellegen, 1988).

2.3. Procedures

The experiment was composed of seven sections in three parts. Part one contained sections one, two, and three of 6 min each. Part two included section four, which lasted 10-15 minutes, and section five, for 30 minutes. Part three included sections six and seven, with 6 min in each. All the procedures were performed in a quiet room, avoiding all loud noises or distracting voices. During all seven sections EGG and respiration were recorded continually. Except for sections 4 and 5, heart rate variability and skin conductance levels were also recorded (See Figure 1.)

(1) Section one - Baseline

All participants sat in the same comfortable chair. After providing written informed consent and completing questionnaires, they were asked to relax and sit as still as possible for six minutes. They were instructed to keep arms and legs still and to avoid, in

particular, any quick body movement. They were instructed to breathe normally, and keep their mind blank and avoid focusing on any topics. After the first baseline, they were asked to complete the subjective evaluation form and mood questionnaire.

(2) Section two – First food image exposure section

In this section the participants were asked to watch a video clip depicting appetizing food scenes that were edited from food advertisements on TV programs. This was shown to them on a 21-in TV for six minutes. They were instructed to pay attention to the food in the video clip and try to imagine the taste, texture, appearance and smell of the food as vividly as possible while they were imagining that they were eating the food. After this section, they were asked to complete the subjective evaluation form and mood questionnaire.

(3) Section three – First recovery period

During this recovery period, the participants did not need to perform any task. They were asked to sit quietly for six minutes and follow same instructions as in the first section.

(4) Section four - Eating period

The participants received a whole microwave-heated pizza (840g/pizza, one serving (1/6 pizza): calorie 350 cal, fat 14g, protein 16g, carbohydrate 41g) and a bottle of water. They were instructed to eat as much as they could without becoming uncomfortable or sick (they could take a second pizza if they wanted). The eating period was 15 – 20 minutes. The number of pizzas, or the percentage of one pizza, eaten by each participant were recorded. After eating, they completed the subjective evaluation form and mood questionnaire again.

(5) Section five - after meal period

The pizza and water were removed from the room. Thereafter, the subjects sat quietly for another 30 minutes and watched a travel video about Switzerland.

(6) Section six- Second food image exposure section

The same video clip with appetizing food was played again in this section for six minutes. The participants followed the same instructions as in section two, and then evaluate their mood again at the end of this section.

(7) Section seven- Second recovery period

In this section, the participants sat for six minutes remaining quiet following the same instructions as in section one and section three.

Finally, the participants filled in a questionnaire about compliance with the instructions during the whole procedure of the study.

2.4. Data Quantification

2.4.1. Physiological Measurements

2.4.1.1. EGG Data

The EGG data were analyzed by running spectral analysis on the six artifact-free minutes of each period to determine the change in power from each baseline to each task period for normal 2.75-3.75 cpm gastric activity, bradyarrhythmia (i.e., 1-2.75cpm) and tachyarrhythmia (i.e., 3.75-9.00cpm). The first four minutes of each recording period comprised the first epoch to be analyzed. The last 75% (minutes 2, 3, 4) of the first epoch was combined with the first new minute (minute 5) to comprise the second epoch, and so on. Therefore, three epochs were analyzed (minutes 1-4, 2-5, 3-6) and then averaged for

each period. The 75% overlap in each epoch provides a stable spectral estimate of the EGG power. These epochs were analyzed via fast-Fourier transform (FFT) to obtain spectral estimates for each 240 sec epoch. Percent of EGG power was calculated for both the normal EGG power bandwidth, the tachyarrhythmic bandwidth and the bradyarrhythmic bandwidth based on a method described by Uijtdehaage, Stern and Koch (1992). This provided an estimate of the change in normal stomach activity and the change in the abnormal stomach activity that was associated with the food image and food intake.

2.4.1.2. EKG Data

Prior to analysis, all IBIs were edited for artifacts using the artifact detection program of Berntson, Quigley, Jang, and Boysen (1990). A time-series of 500 ms samples was then created from the IBI and RSA was calculated by removing the complex trend in the IBI data with a moving 21-point polynomial filter (Porges & Bohrer, 1990). After filtering, a residual IBI series was created by subtracting the filtered data from the original time series. The natural logarithm of the variance in the residual time series at the respiratory frequency (0.12-0.40 Hz) was taken as the estimate of RSA. This analysis was performed for each minute of each baseline and task period. Group means were then computed by averaging all participants within each experimental group. Essentially, the more one's rhythmic fluctuation of heart rate corresponds to respiratory rate, the more cardiac vagal tone the person showed, and the higher was the estimate of RSA. Decrease in RSA reflects less heart rate variability and a corresponding increased parasympathetic withdrawal (Vybiral, Bryg, Maddens, & Bodden, 1989)

2.4.1.3. Skin Conductance Level (SCL)

The SCL levels were recorded for every minute. The mean value of the each six minutes of recording was used for analysis. Higher values indicate greater skin conductance level and reflect increased sympathetic activation.

2.4.2. Subjective Measurements

2.4.2.1. Subjective Evaluation Form

The participants were instructed to make a vertical mark on each 100 mm VAS line that best matched their subjective reaction to viewing the food images or food at that time. The left end of the line was labeled as ‘not at all’ or ‘very weak’ and the right end of the line was labeled as ‘very strong’ , ‘very much’ or ‘very full’. The VAS score was determined by measuring in millimeters from the left hand end of the line to the point that the participants marks for each question.

2.4.2.2. PANAS

To fill out the PANAS, the participants were instructed to answer "to what extent you feel this way right now, that is, at the present moment" for each adjective on a 100-mm visual analogue scale. “0” means very slightly or not at all, and “100” means extremely.

2.5 Statistical Analysis

The SAS software program version 8.2 was used to perform the data summarization and statistical analyses. Data from the experiment were analyzed using a mixed effects model analysis of variance (ANOVA). The mixed effects model was implemented using SAS Proc Mixed, with REML estimation method, variance-covariance structure of

compound symmetry and Satterth degrees of freedom adjustment. EGG, SCL, RSA and the subjective measurements data were analyzed with group (obese, normal-weighted) and period considered fixed effects and subject within group considered a random effect in the mixed effect model. In the first part of the study, there were three periods: Baseline One, Exposure One, and Recovery One; in the second part of the study, six periods were included in the model: Baseline One, 1st 6-min Post-Meal, 2nd 6-min Post-Meal, 3rd 6-min Post-Meal, 4th 6-min Post-Meal, 5th 6-min Post-Meal; and in the third part of the study, there were three periods: Baseline Two (the fifth 6-minute period after the meal was used as the baseline for the second food images exposure period), Exposure Two, and Recovery Two.

The main and interaction effects of experimental group and period on percent gastric bradyarrhythmia, percent normal gastric activity, percent gastric bradyarrhythmia, RSA, SCL, subjective measurements were detected. Null hypotheses that results in p-values of .05 or less was considered statistically significant, and p-values between .05 and .10 was considered marginally significant. Tukey's post-hoc pairwise comparisons were used to compare differences between the specific periods. Tests of planned contrasts were performed with experimental group as the independent variable in order to explore further the differences between groups during each experimental period. Pearson Product Moment correlations were performed to assess the relationship between physiological measurements (e.g., EGG, SCL, RSA) and subjective responses (e.g., ratings of hunger or desire to eat, how much food each participant expect to eat, and mood change). The amount of food eaten was expressed in terms of the numbers of pizzas eaten by the participants.

For the physiological measurements (e.g. EGG, SCL, RSA) and subjective measurements (e.g. ratings of hunger or desire to eat, how much food each participant expect to eat, and mood change), descriptive statistics (mean and standard error) summarizing data by group were provided in the tables. Graphical presentations of each measurement by period were provided for each group.

RESULTS

3.1. Part One: Effects of First Food Image Exposure in Obesity

Mean physiological and subjective responses to the experimental treatments in both groups are shown in Table 2.1. and 2.2.

3.1.1. Physiological Dependent Measurements

3.1.1.1 Electrogastrography (EGG) Results

3.1.1.1.1. Percent Normal Gastric Activity

As a whole, a significant main effect of experimental period on percent normal gastric activity was observed ($F [1, 36.2] = 7.79, p < 0.001$). Tukey's post-hoc pairwise comparisons revealed that percent normal gastric activity was significantly greater during the food image viewing period than the baseline period ($F [1, 78] = 14.67, p < 0.001$) across the experimental groups. Percent normal gastric activity was not significantly different between the recovery period and the food image exposure period.

There was a marginal significant main effect of group ($F [1, 39] = 3.11, p = 0.09$). The interaction effect of experimental period and experimental group on percent normal gastric activity was significant ($F [2, 78] = 3.54, p = 0.03$), indicating different gastric responses between the two groups.

The tests of planned contrasts examined the differences between the two experimental groups in each experimental period. No significant differences existed during the baseline period. During the food image exposure period, a significant effect of group on percent normal gastric activity was observed ($F [1, 39] = 7.13, p = .01$). Percent normal gastric activity was significantly lower among the participants in the obese group than in the normal-weighted group. The difference in percent normal gastric activity

between the obese and normal-weighted participants during the recovery period was not statistically significant (see Figure 2.1. and 4.6).

3.1.1.1.2. Percent Gastric Bradyarrhythmia

The main effect of experimental period on percent gastric bradyarrhythmia was significant across the experimental groups ($F [2, 78] = 14.74, p < .001$). Tukey's post-hoc pairwise comparisons revealed that percent gastric bradyarrhythmia was significantly lower during the food image exposure period than the baseline period ($F [1, 78] = 18.04, p < .001$) across the experimental groups. During the recovery period, percent gastric bradyarrhythmia did not return to a level similar to that in the baseline period. It continued to decrease after the termination of the food image exposure, although the difference in percent gastric bradyarrhythmia between the recovery period and the food image exposure period was not significant as a whole.

During the baseline period, there was no significant difference in percent gastric bradyarrhythmia between the two groups. However, percent gastric bradyarrhythmia was significantly higher among the participants in the obese group than that in the normal-weighted group in the food images exposure period ($F [1, 39] = 9.24, p = .004$). In other words, percent gastric bradyarrhythmia decreased more in the participants in the normal-weighted group than those in the obese group. In the recovery period, percent gastric bradyarrhythmia increased in both groups and it was similar between the two groups (see Figure 2.2. and 4.7.).

3.1.1.1.3. Percent Gastric Tachyarrhythmia

Percent gastric tachyarrhythmia decreased in the food image exposure period across the experimental groups compared to that in the baseline period; however, the difference

was not significant. The interaction between experimental period and experimental group was not significant (see Figure 2.3. and 4.8). The tests of planned contrasts revealed that no significant difference in percent gastric tachyarrhythmia existed between the two groups in each experimental period.

3.1.1.2. Respiratory Sinus Arrhythmia (RSA) Results

A significant main effect of experimental period on RSA was observed ($F [2, 56.2] = 4.25, p=.02$). Tukey's post-hoc pairwise comparisons revealed that RSA was lower across the experimental groups during the food image exposure period than during both the baseline period and the recovery period ($F [1, 56.3] = 7.93, p=.007, F [1, 56.3] = 4.13, p=.047$). In the recovery period, RAS returned to a level that was similar to that in the baseline period.

RSA of the participants in the obese group was greater than that in the normal-weighted group in each experimental period; however the differences were not significant. The interaction effect of experimental period and experimental group was not significant (see Figure 2.4.).

3.1.1.3. Skin Conductance Level (SCL) Results

A significant main effect of experimental period on SCL was observed ($F [2, 78] = 3.15, p=.05$). Tukey's post-hoc pairwise comparisons revealed that SCL was greater across the experimental groups during the food image exposure period than during the baseline period ($F [1, 78] = 4.92, p=.03$). SCL returned to the baseline level in the recovery period across the experimental groups.

The pattern of changes in SCL during the food image exposure period and the recovery period was quite dissimilar between the two groups. In the participants in the

obese group, food image exposure increased SCL significantly ($F [1, 40] = 6.71, p = .01$), and with the termination of food image exposure SCL decreased. SCL was still higher in the recovery period than in the baseline period and the difference was marginally significant ($F [1, 40] = 2.78, p = .1$). But in the participants in the normal-weighted group, food image exposure did not cause significant change in SCL (see Figure 2.5.).

3.1.2. Subjective Dependent Measurements

3.1.2.1. Food Evaluation Questionnaire Results

No significant difference was observed in the ratings of the feelings of hunger and fullness between the obese group participants and the normal-weighted group participants.

However, while viewing food images, the participants felt much hungrier than in the baseline period ($F [1, 35.6] = 13.86, p < 0.001$). The participants in the obese group and the normal-weighted group reported similar increases in the feelings of hunger during food image exposure period (see Figure 5.2.). Accordingly, the ratings of fullness decreased in the food image exposure period compared to that in the baseline period and the difference between the two groups was not significant (see Figure 5.3.).

The desire to eat and the amount of food that the participants expected to eat increased significantly across the experimental groups in the food image exposure period compared to that in the baseline period ($F [1, 39] = 44.15, p < 0.001$ and $F [1, 39] = 29.01, p < 0.001$, respectively). Differences between groups were observed in the ratings of the desire to eat and the amount of food the participants expected to eat. The participants in obese groups reported a greater increase in the desire to eat ($F [1, 39] = 4.97, p = 0.03$, see Figure 5.4.). They also expected to consume more food ($F [1, 39] = 5.85, p = 0.02$, see

Figure 5.5.) compared to the participants in the normal-weighted group.

The ratings to the question of "How much did you like the food in the video clips" were 73 ± 16.5 as a whole, which indicates that most of the participants liked the food that they were asked to view. No group difference existed in the evaluations of the food images (see Figure 5.1.).

Correlations between hungry level and the amount of food the participants expected to eat and the desire to eat were significant.

3.1.2.2. Effects of First Food Image Exposure on Mood

Analyses of Positive and Negative Affect Schedule (PANAS) questionnaire responses revealed that there were no significant differences in the ratings of feelings of interested, distressed, excited, upset, or nervous, etc. between the experimental groups.

3.1.3. Correlations between Physiological and Subjective Measures

Pearson Product Moment correlations were carried out in order to investigate any associations between the physiological measurements, e.g. EGG, SCL, RSA, and the subjective responses, e.g. ratings of hunger, desire to eat, the amount of food that each participant expected to eat and mood in each experimental period. The results did not show any associations between the physiological measurements and the subjective responses.

3.2. Part Two: Effects of Meal in Obesity

Mean physiological and subjective responses to experimental treatments within the 30 minutes after the meal in both groups are shown in Table 3.1. and 3.2.

3.2.1. Physiological Dependent Measurements--Electrogastrography (EGG) Results

3.2.1.1. Percent Normal Gastric Activity

Percent normal gastric activity of each 6-minute period within the 30 minutes after a meal was compared to that in the baseline period. A significant main effect of experimental period on percent gastric normal activity was observed ($F [5, 187] = 3.11, p = 0.01$) as a whole. Tukey's post-hoc pairwise comparisons revealed that percent normal gastric activity was significantly greater during the 30 minutes after a meal than during the baseline period ($F [1, 187] = 14.71, p < 0.001$) across the experimental groups. However, percent normal gastric activity was not significantly different between each 6-minute period.

There was also a significant main effect of group ($F [1, 38] = 6.51, p = 0.01$) in percent normal gastric activity. The interaction effect of experimental period and experimental group on percent gastric normal activity was not significant. The tests of planned contrasts examined the differences between the experimental groups during each experimental period. Percent normal gastric activity was lower among the obese group participants than among the normal-weighted participants in each 6-minute period within the 30 minutes after a meal. The differences were significant in each 6-minute period after a meal except in the second 6-minute period (see Figure 3.1. and 4.6.).

3.2.1.2. Percent Gastric Bradyarrhythmia

The main effect of experimental period on percent gastric bradyarrhythmia was significant across the experimental groups ($F [5, 187] = 15.79, p < .001$). Tukey's post-hoc pairwise comparisons revealed that percent gastric bradyarrhythmia was significantly lower during the 30 minutes after a meal than during the baseline period ($F [1, 187] = 41.40, p < .001$) across the experimental groups. The interaction effect of experimental

period and experimental group on percent gastric bradyarrhythmia was also significant ($F [1, 187] = 3.18, p = .01$).

Percent gastric bradyarrhythmia was significantly higher among the obese group participants than among the normal-weighted participants in the first four 6-minute periods after a meal. Only in the fifth 6-minute period, did percent gastric bradyarrhythmia in the obese participants decrease significantly ($F [1, 93.6] = 7.64, p = .01$). In other words, in the first 24 minutes after a meal, percent gastric bradyarrhythmia was higher in the participants in the obese group than in the normal-weighted group and only started to decrease in the obese group at around twenty minutes after a meal (see Figure 3.2. and 4.7).

3.2.1.3. Percent Gastric Tachyarrhythmia

Within the 30 minutes after a meal, percent gastric tachyarrhythmia increased continuously and significantly higher in the participants in the obese group than that in the normal-weighted group; however, during this period percent gastric tachyarrhythmia remained the same in the participants in the normal-weighted group ($F [1, 36] = 4.82, p = .03$, see Figure 3.3. and 4.8.).

3.2.2. Subjective Dependent Measurements

3.2.2.1. Food Evaluation Questionnaire Results

The ratings to the question of "How much did you like the food that you have taken?" were not significantly different between the participants in the two groups (69 ± 4.74 and 60 ± 4.74 respectively, see Figure 5.1.).

Compared to the baseline period, the ratings of the feelings of hunger and desire to eat significantly decreased after a meal ($F [1, 149] = 9.17, p = .002$; $F [1, 156] = 17.37$,

$p < .001$, respectively) across the groups. Again, the ratings of hungry level and desire to eat were significantly correlated ($r = 0.88$, $p < .001$).

In addition, there were no differences in the ratings of the feelings of hunger, fullness and desire to eat between the two groups and the amount of food that the participants expected to consume after a meal was similar also (see Figure 5.3.).

3.2.2.2. Effects of Food on Mood

Analyses of the Positive and Negative Affect Schedule (PANAS) questionnaire scores revealed that there were no significant differences between the participants in the obese group and the normal-weighted group in the ratings of feelings of being interested, distressed, excited, upset, or nervous, etc. In other words, food did not cause significant difference in mood between the two groups.

3.2.3. Correlation between Physiological and Subjective Measurements

Again, the associations between electrogastrography parameters and subjective measurements were not significant either across the groups or in each experimental group.

3.3. Part Three: Effects of Second Food Image Exposure in Obesity

Mean physiological and subjective responses to experimental treatments during the first food images viewing period in both groups are shown in Table 4.1 and 4.2.

3.3.1. Physiological Dependent Measurements

3.3.1.1 Electrogastrography (EGG) Results

3.3.1.1.1. Percent Normal Gastric Activity

The analysis of percent normal gastric activity did not show significant changes

while the participants were viewing the same food images for the second time across the groups. The group difference in percent normal gastric activity during the second food image exposure period was not significant. However, with the termination of food images viewing, a significant increase in percent gastric normal activity was shown in the normal-weighted group, but not in the obese group ($F [1, 38] = 5, p = .03$, see Figure 4.1. and 4.6.)

3.3.1.1.2. Percent Gastric Bradyarrhythmia

On the contrary to that in the first food image exposure period, percent gastric bradyarrhythmia increased when the participants were viewing the food images for the second time across the groups and the increase was marginal significant ($F [1, 74.3] = 3.85, p = .054$). No significant difference was observed between the groups in percent gastric bradyarrhythmia during the second food images exposure period (see Figure 4.2. and 4.7.).

3.3.1.1.3. Percent Gastric Tachyarrhythmia

Similar to that in the first food image viewing period, percent gastric tachyarrhythmia decreased in the second food images exposure period across the groups compared to that in the baseline period ($F [1, 73] = 8.19, p = .005$). No significant group differences in percent gastric tachyarrhythmia existed during the food images exposure period, however, the group difference was significant in the recovery period with a higher increase in the obese group ($F [1, 38] = 4.66, p = .04$, see Figure 4.3. and 4.8.).

3.3.1.2. Respiratory Sinus Arrhythmia (RSA) Results

A significant main effect of experimental period on RSA was observed during the second food image exposure period ($F [2, 48.6] = 4.13, p = .02$). Tukey's post-hoc

pairwise comparisons revealed that RSA was lower across the experimental groups during the food image viewing period than during the baseline period ($F [1, 41.8] = 8.26$, $p = .006$). RSA returned to the baseline level in the recovery period across the experimental groups.

There was no significant difference in RSA between the groups; but RSA in the obese group was greater than RSA in the normal-weighted group during the recovery period although the difference was not significant (see Figure 4.4.).

3.3.1.3. Skin Conductance Level (SCL) Results

A significant main effect of experimental period on SCL was observed ($F [2, 78] = 5.16$, $p = .007$). Tukey's post-hoc pairwise comparisons revealed that SCL was greater across the experimental groups during the recovery period than that during the second baseline period ($F [1, 78] = 10.31$, $p = .002$), however, the difference between the food image exposure period and the baseline period was not significant.

No significant difference in SCL was observed during the food image exposure period between the groups. However, with the termination of food image exposure, a significant increase in SCL was observed in the normal-weighted group ($F [1, 38] = 4.26$, $p = .045$), but not in the obese group (see Figure 4.5.).

3.3.2. Subjective Dependent Measures

3.3.2.1. Food Evaluation Questionnaire Results

The participants across the groups rated the extent to which they liked the food in the video clips lower than that during the first food image exposure period (73 ± 16.5 vs 64 ± 3.02) and the difference was significant ($F [1, 77] = 4.25$, $p = .04$, see Figure 5.1.).

Furthermore, the results showed that the ratings of the feelings of hunger and

fullness did not change significantly during the second food image period compared to that in the second baseline period across the experimental groups. Viewing the food images did not make the participants expect to consume more food either. There were no significant differences that existed in the ratings of the feelings of hunger and fullness between the two groups, however, the difference was observed in the ratings of desire to eat with that the participants in the obese group reported marginally stronger desire to eat than the participants in the normal-weighted group while viewing the food images for the second time ($F [1, 39] = 2.95, p = .09$, see Figure 5.3.).

3.3.3. Correlation between Physiological and Subjective Measures

No significant correlations were observed between the physiological measurements and subjective responses during this period either.

DISCUSSION

4.1. Part One: Effects of First Food Image Exposure on Obese People

This part of the study was designed to investigate cephalic phase responses (CPRs) in obese participants, including subjective responses and associated physiological responses, and to test the assumption that obese participants respond with more marked CPRs than normal-weighted participants to the presentation of food images. Two major findings emerged. First, the obese participants were more responsive to the sight of food images with a greater desire to eat and more prospective food consumption compared to that in the normal-weighted participants. Second, the obese participants responded differently to the food images with less normal gastric activity, greater percent gastric bradyarrhythmia, and higher sympathetic nervous system activity.

4.1.1. Interpretation of the Results of the Subjective Measurements

It was hypothesized that the obese participants would experience a stronger desire to eat and expect to consume more food than the normal-weighted participants when fasting. It was also hypothesized that while viewing food images, the obese participants would show a stronger interest in the food in the video clips and that the estimation of prospective consumption at this moment would be more than that of the normal-weighted participants. A food evaluation questionnaire was employed prior to and after exposure to food images in this study in order to evaluate these hypotheses.

The results from the food evaluation questionnaire only partly support our hypotheses. There were no significant differences between the participants in the obese group and in the normal-weighted group in their ratings of the feelings of hunger, desire to eat and prospective consumption during the baseline period, even though the obese

group participants rated higher in their answers to all of these questions than the normal-weighted group participants. These results seem to suggest that the obese participants did not crave food significantly more strongly than the normal-weighted participants when they were feeling a similar extent of hunger as the normal-weighted participants in fast.

However, as predicted, when the participants were exposed to food images, the obese participants reported significantly different subjective responses from the normal-weighted participants. Differences between the participants in the obese group and the normal-weighted group in post-exposure ratings were mainly confined to the desire to eat and the amount of food that they expected to consume when they were exposed to food images, with higher ratings being observed for the obese group. These results support our hypothesis that obese participants respond with more marked cephalic phase responses psychologically than normal-weighted participants in the presence of food images.

As is well known, the cephalic phase is contrasted with the gastric and intestinal phases of digestion; the latter two phases are related to the postingestional consequences of food, whereas the former is associated with the preabsorptive consequences of food. Classically, cephalic phase responses are elicited during exposure to the sensory properties of food (e.g. sight, smell and taste) as well as by simply the thought of eating. In the current study, appetizing food in video clips was presented to the participants and the participants were instructed to imagine eating the food in the video clips while they were viewing the food images. The same video clips have been utilized in one study in our lab previously and the food images in the video clips were proved to be effective food cues to initiate cephalic phase responses in healthy participants (Wang & Stern, 2004). In the current study, analyses of subjective responses revealed increases in the desire to eat

and feelings of hunger in response to the food images in both groups, which confirmed our previous findings on subjective responses to food images.

Cephalic phase responses are believed to prepare the body to optimize digestion, absorption, and the use of ingested nutrients. Whether enhanced cephalic phase responses play a role in the regulation of energy intake and contribute to the development of obesity remains unresolved. It was suggested that for obese individuals, the sight of food or other sensory experiences of food is a more important driving force behind their motivation to eat than hunger ('internal-external' theory of obesity; Schachter, 1968). For example, obese subjects ate more cashew nuts if the light in the room was brightly illuminated compared with when the light in the room was dim (Ross, 1970). In another study, obese subjects ate less food when they were blindfolded without feeling less full (Barkeling, et al., 2003). One explanation for these results is that the removal of sight, therefore, may decline the cephalic phase of ingestion, which, in turn affects eating behavior. The current study showed a stronger desire to eat and more prospective consumption but similar feelings of hunger and fullness in the obese participants compared to the normal-weighted participants. These results suggest that the obese participants, relative to those of normal weight, were over-responsive to external food cues and under-responsive to internal cues (including feelings of hunger and fullness), which was in accordance with the 'internal-external' theory of obesity.

The result of this current study is also consistent with the cue reactivity model that was developed in the eating disorder mechanism exploration (Wardle, 1990). The cue reactivity model of binge eating states that cephalic phase responses are experienced subjectively as cravings food (intense desire to eat a specific food). Therefore, based on

the cue reactivity model, food craving increases food intake and plays a role in abnormal eating behavior. The findings demonstrated that when subjects were exposed to food cues, more craving and physiological reactivity was found in the ones with abnormal eating patterns (Karhunen, et al., 1997). Furthermore, food craving is not restricted to binge eating patients. It is highly prevalent around the world. Close to 100% of young adult females and about 70% of young men reported having experienced one or more food cravings at some time based on surveys in United States, Canada and Great Britain (Pelchat, 1997; Weingarten & Elson, 1991).

It is unclear whether obese people experience food cravings more frequently and more intensely than normal-weighted people. It is also not clear whether the stronger desire to eat that was observed in the current study in the obese participants predicts more craving for food in their everyday life. But it is possible that stronger cephalic phase responses to the food cues which prepare people for food intake make it more difficult for the participants to abstain from eating and thereby add to the continuation of abnormal eating behavior. The role of the predominant subjective experiences to food cues observed in the current study, which distinguished the obese participants from the normal-weighted participants, is still far from clear and needs further exploration.

4.1.2. Interpretation of the Results of the Physiological Measurements

4.1.2.1. Electrogastrography (EGG)

The effects of the food images induced variations in the functioning of the stomach and autonomic nervous system between the obese and normal-weighted participants.

During the baseline period, percent gastric normal activity was not significantly

different between the participants in the obese group and those in the normal-weighted group. Similar percent gastric tachyarrhythmia and percent gastric bradyarrhythmia were also observed between the participants in the obese group and those in the normal-weighted group. During this period, the participants were instructed to avoid focusing on any specific topics while they were sitting quietly. The purpose of this instruction was to reduce the possibility that some of the participants, especially obese participants, might think of food more than other participants, since thinking of food may presumably influence gastric activity in a way similar to that which is caused by food cues (Wang & Stern, 2004). The results of the current study suggest that the stomach functions similarly between the obese participants and normal-weighted participants while fasting.

The findings of the current study were in accordance with previous studies that indicated that obese subjects were not distinguishable from normal-weighted subjects in the frequency of gastric electrical activity while fasting. For example, one previous study showed that the comparison of the mean frequency values of the gastric spectral peak did not reveal statistically significant differences between the non-obese and obese groups (Riezzo, Pezzolla & Giorgio, 1991). In this study, it was also found that gastric spectral power was significantly lower in those who were obese than those who were not. One explanation for the lower gastric spectral power was that EGG spectral power could be affected by the thickness of the tissue situated between the gastric muscular wall and the cutaneous electrode, and hence by the adiposity of the subjects. Therefore, in the current study, EGG signals were recorded through cutaneous electrodes attached to wrists of the participants.

The gastric electrical activity during the period of viewing food images was

compared to that in the baseline period. Across the experimental groups, percent gastric normal activity during the food image exposure period increased significantly compared to that in the baseline period; while the percent of tachygastric activity and bradygastric activity decreased accordingly. These findings indicate that the amount of normal myoelectrical activity in the stomach was greater with the exposure of food images compared to that in a situation without any food-related stimulations across the groups. Accordingly, as a whole, the proportion of gastric activity in the abnormal range was lower during the food image exposure period relative to the baseline period. These results support the findings of Stern et al. (1989) and some other researchers (Chen, Pan & Orr, 1996) about the characteristics of gastric myoelectrical activity identified in cephalic phase responses. Stern et al. have previously investigated the effect of sham feeding on gastric myoelectrical activity and found an increase in 3 cpm EGG power during feeding of appetizing food in healthy human participants. The increase in 3 cpm power during sham feeding was very brief as compared to postprandial power increase. The researchers estimated that the brief increase in 3 cpm was around 1-2 minutes. Chen et al. conducted a similar sham feeding study and concluded that gastric myoelectrical activity can be altered by sham feeding and the response of the EGG to sham feeding was highly correlated with the response to food ingestion (Chen, Pan & Orr, 1996). But this study suggested that cephalic phase responses lasted for 30 minutes after the termination of sham feeding which was different from the previous report on the duration of the effect of sham feeding. In the current study, we only recorded EGG for 6 minutes after the termination of food cues exposure. No significant decrease in percent normal gastric activity was observed with the termination of food image exposure within the 6 minutes

and this result seems to suggest some continuous influence of food cues on EGG in the recovery period.

When EGG activity was compared between obese and normal-weighted groups, differences in the stomach in response to food images were obtained. It was hypothesized that the participants in the obese group would develop more normal gastric activity and less gastric dysarrhythmia than those in the normal-weighted group while viewing food images and imagining eating the food in the video clips. On the contrary, the results show that the participants in the obese group exhibited less increases in normal gastric activity than the participants in the normal-weighted group. A similar pattern of unexpected results was obtained with respect to estimates of percent gastric bradyarrhythmia as well. Obese participants showed more percent gastric bradyarrhythmia than normal-weighted participants while they were watching the food images, although percent gastric bradyarrhythmia decreased in both groups in the food images exposure period compared to that in baseline period. The significantly greater estimates of percent gastric bradyarrhythmia during the food images exposure period among the participants in the obese group than among those in the normal-weighted group suggest the development of abnormal activity in the stomach in obese people when viewing the food images. This is the most intriguing aspect of these results.

It was also hypothesized that normal gastric activity would be maintained within the next 6 minutes following the food images exposure period among the obese group participants; however, the results revealed that the amount of normal gastric activity was maintained only in the participants in the normal-weighted group but not in the participants in the obese group.

Another interesting finding was that obese participants had a significant increase in percent gastric tachyarrhythmia during the recovery period. This was accompanied by a significant decrease in percent gastric bradyarrhythmia in the obese participants, with no significant change in percent normal gastric activity.

Cephalic phase responses are suggested to influence the magnitude of subsequent postingestional gastrointestinal responses. This is supported by reports that the changes of postprandial EGG parameters were significantly correlated with those after sham feeding (Chen, Pan & Orr, 1996) and when CPRs are prevented in rats by making the timing of a meal unpredictable, the rats eat smaller meals (Woods, 1991). Furthermore, based on previous research, the responses triggered in the cephalic phase were anticipatory in the sense that they were in the same direction as those that occur during the gastric and intestinal phases; the only difference might be that they were more rapid in onset, of shorter duration and usually lower in magnitude than physiological reactions stimulated during the gastric and intestinal phases (Stern, et al., 1989).

Since overeating is often reported in obese people and, as stated above, the cephalic phase responses appear to play an important role in the amount of food someone can eat, it has been suggested that obese and non-obese people differ in their preingestive responses to food cues physiologically. Therefore, it was expected that presenting of food cues to the participants would generate a response that was similar to the response after eating, but in the obese participants, the response might be more intense than that in the normal-weighted participants. For example, some research has generally shown that obese persons salivate more to repeated food cues than non-obese persons (Epsten, Paluch & Coleman, 1996). Obese subjects exhibited significantly greater cephalic-phase

insulin release (CPIR) than normal-weighted subjects (Teff, Mattes, Engelman & Mattern, 1993). Furthermore, in response to the sensory experience of food, differences in regional cerebral blood flow (rCBF) were observed in several regions of the brain in obese compared to lean individuals (DelParigi, et al., 2005). These results supported the hypothesis that obese individuals show a stronger physiological response in cephalic phase responses than normal-weighted individuals.

But few previous studies have compared the gastric electrical activity in obese individuals to that in normal-weighted individuals in cephalic phase digestion. It was hypothesized in the current study that the obese participants would respond to the food images with higher percent of 3 cpm activity and less bradygastric and tachygastric activity than normal-weighted participants. However, our results did not confirm this hypothesis. The results of the current study seem to suggest less normal gastric activity in the participants in the obese group than that in the normal-weighted group in cephalic phase digestion. More interesting, a difference between the obese and normal-weighted group in percent bradygastric activity was obtained in this study with higher percent of bradygastric activity in the obese group compared to that in the normal-weighted group.

Explanations for the higher percent bradygastric activity found in the current study in the obese group participants during the food image exposure period are not clear. In addition, the relationship between the observed increase in bradygastic activity in the current study and enhanced salivation, higher insulin release and cerebral blood flow that were reported in other studies during cephalic phase digestion in obesity needs further exploration.

4.1.2.2. Skin Conductance Level (SCL) and Respiratory Sinus Arrhythmia

(RSA)

Skin conductance level is an index of sympathetic nervous system activity. It has been widely used in the evaluation of sympathetic nervous system activities in participants while the participants were experiencing various physiological or psychological stimulations in experimental studies. It is also often utilized in identifying any differences between the experimental groups in regards to responses in the sympathetic nervous system under certain conditions.

In the current study, the skin conductance level was recorded while the participants were viewing food images and the results were compared between the experimental groups in order to assess any sympathetic nervous system activity discrepancy in cephalic phase responses between the obese participants and the normal-weighted participants. The results of SCL in the food images exposure period revealed possible differences in sympathetic nervous system responses between the obese participants and the normal-weighted participants. SCL scores between the participants in the obese group and those in the normal-weighted group during the baseline period were not significantly different. This result seems to suggest that the sympathetic nervous system was equally activated in the obese participants and in the normal-weighted participants when they were not experiencing any food related stimulations.

However, the pattern of changes in the SCL from one experimental period to the next was quite dissimilar between the two groups. Food image exposure increased the skin conductance level significantly in the participants in the obese group; however, during the food image exposure period, the SCL kept in a similar level as to that in the

baseline period in the participants in normal-weighted groups. In addition, in the recovery period, the SCL scores decreased with the termination of food image exposure, but they were still higher than those that were measured in the baseline period for obese participants. These results presumably suggest that the obese participants presented higher sympathetic nervous system activity while viewing food images than the normal-weighted participants. Furthermore, it is hard for the obese participants to return to the same level of sympathetic nervous system activity as that in the baseline period after experiencing food image stimulations.

These results appear to confirm our hypothesis that obese participants responded to food cues with higher sympathetic nervous activity, which can be interpreted as a representation of higher arousal level. In addition, the above results are in accordance with the results from the food evaluation questionnaire showing that obese participants indicated a stronger desire to the food images in the video clips than normal-weighted participants. Based on the belief that vagal withdrawal and/or sympathetic nervous system activation accompany the development of gastric dysarrhythmia (Muth, et al., 1998), the higher activated sympathetic nervous system activity in obese participants may explain, to some extent, the more prominent gastric bradyarrhythmia in the obese participants when they were viewing food images compared to that of the normal-weighted participants.

Respiratory sinus arrhythmia is generally believed to provide an index of vagal activity, and it is found to be influenced by respiratory rate and depth. It is well-established that the initiation and maintenance of the gastric postprandial pattern depend upon an increase of vagal input to increase normal gastric activity (Powley, 2002).

Therefore, the postprandial EGG pattern usually occurs coincidentally with an increase of vagal input to the stomach. The occurrence of gastric dysrhythmias, such as tachyarrhythmia, is thought to be reduced partially by enhanced parasympathetic tone after the meal.

However, in the present study, a mild decrease in parasympathetic nervous system activity was observed when the participants responded to the food images with an increase in normal gastric activity. This result was contrary to previous research. But this result is consistent with the results of one previous study in our lab which observed a similar decrease in RSA when the participants were viewing food images (Wang & Stern, 2004). A possible explanation for this lack of association is that during the baseline condition, the participants sat quietly. In this condition, the parasympathetic nervous system activity is usually higher than that of watching a TV program. The significant increase in the arousal level during the food image viewing task suggested that for the participants viewing food a image was more like a task than being prepared to eat food. Therefore, a decrease instead of an increase in PNS activity was recorded during the food images viewing period. The increase of RSA in the recovery period could easily be attributed to the termination of stimulation provided by the viewing food image task.

Furthermore, this result is explainable given the expectation that the two branches of the autonomic nervous system are thought to function under a reciprocal mode of control. Decreases in RSA that reflect decreased vagal tones were anticipated to correspond with increases in SCL, or increased sympathetic activation. This was observed to be the case in the current study.

This discrepancy may also indicate that the relationship between

electrogastrographic activity and cardiac vagal tone is complex. Gastric motility is influenced by numerous factors other than vagal-cholinergic control, such as sympathetic activity and peptides. The increase in the percent of normal EGG activity in this study may be the combined effects of all the factors. Therefore, the stomach can still function normally with decreased PNS activity.

4.2. Part Two: Effects of a Meal on Obese People

The main purpose of this part of the study was to compare gastric electrical activity between the obese participants and the normal-weighted participants in a postprandial period.

4.2.1. Interpretation of the Results of the Subjective Measurements

As expected, the obese participants consumed more pizza than the normal-weighted participants and the difference was significant. However, although the obese participants consumed more food, they reported similar feelings of fullness, hunger and desire to eat compared to those of the normal-weighted participants measured at the moment after the meal. Both groups rated significantly lower in the feelings of hunger and desire to eat and higher in the feeling of fullness compared to the ratings in pre-meal period.

The current results confirmed previous findings that the obese participants tend to consume more food than the normal-weighted participants in order to reach the same extent of satiety (Delgado-Aros, et al., 2004). It is well known that gastric distension during and after food ingestion contributes to feelings of fullness and satiety (Powley & Phillips, 2004). A stomach with a larger capacity may require a bigger meal to trigger

early postprandial fullness. Some researchers suggest that obese patients tend to have a higher gastric capacity or an enlarged antrum than normal-weighted people. For example, Geliebter et al. measured the stomach capacity in three groups of women - normal, obese, and bulimic - by filling a gastric balloon with water at a rate of 100ml/min until the maximum volume that the subject could tolerate. It was found that the gastric capacity of the obese subjects was higher than in the normal-weighted group, but it is lower than in the bulimic women (Geliebter & Hashim, 2001). Distal gastric volume is also found to be larger in obese individuals in the fasting state from imaging with single photon emission computed tomography, suggesting that the increased gastric volume causes changes in the sensation of satiety with a consequent increase of food intake in these subjects (Kim, 2001).

4.2.2. Interpretation of the Electrogastrography (EGG) Results

We hypothesized that normal gastric activity would be higher in the participants in the obese group than in the normal-weighted group with the ingestion of food. Instead, the results of the current study showed an overall decline in percent normal gastric activity in the obese group as opposed to that in the normal-weighted group within the first 30 minutes after a meal. Furthermore, the obese participants showed higher percent gastric bradyarrhythmia compared to that of the participants in the normal-weighted group after a meal and it started to decline in the last 6-minute period, which was measured between 24 to 30 minutes after a meal. These results presumably suggest that the obese participants tend to have abnormal gastric myoelectrical activity (gastric bradyarrhythmia) than the normal-weighted participants within at least the first 24

minutes after a meal.

The findings of the current study are consistent with one study in adult patients with morbid obesity, which revealed an increase in the percentage of gastric bradyarrhythmia in both the fasting and fed states. Accelerated gastric emptying of solids in these obese patients was recorded at the same time (McCallum, et al., 2001). In this study, 12 healthy patients with morbid obesity ingested a ^{99}Tc -labeled solid meal after an overnight fast. EGG was performed for 30 minutes after the meal and then for 2 hours post-meal with the results compared to normal subjects. Furthermore, 4-hour scintigraphic gastric emptying using percentage intragastric residual with measured counts at 1, 2, 3, and 4 hours post meal was performed. Bradygastria was observed in more than 30% of recording time in 8 of 12 participants. In comparison with normal subjects, the obese participants had a significantly high mean percentage of bradygastria both in the fasting state and after the meal.

However, this result is contrary to the results of another study (Riezzo, et al., 1998), in which similar postprandial patterns and responses in EGG in response to a mixed meal were obtained in obese children compared to normal-weighted children.

The exact origin of gastric bradyarrhythmia is difficult to determine. Although tachygastria has been suggested to be associated with gastric hypomotility (Pfaffenbach, Adamek, Bartholomaeus & Wegener, 1997), the clinical significance of gastric bradyarrhythmia remains unclear. It was suggested that the temperature and volume of ingested liquids slow EGG frequency temporarily after eating. Smout et al. observed that a 'frequency dip' occurred in the first 10 minutes after the 4°C water load, during which the frequency of the EGG decreased transiently into the 2.0- to 2.5-cpm range. However,

when body temperature water (37 °C) was ingested, the ‘frequency dip’ was not induced and the normal 3-cpm EGG activity was initiated immediately after the water load. This study indicated that the temperature of the ingested food has an effect on gastric myoelectrical rhythmicity and the temperature of the food might be a factor that slowed the EGG frequency. However, the participants consumed heated pizza and room-temperature water instead of milliliters of cool water in the current study. The ‘frequency dip’ was not observed and percentage gastric bradyarrhythmia decreased in both groups after the meal. Volume is another factor that induces low-frequency. Lin et al. showed that the normal 4.5-cpm canine pacesetter potential frequency decreased to 2.5 cpm as 1800 ml body temperature water was infused into the stomach over a 5 minutes period (Lin, et al., 1996). This significant decrease in the frequency of the EGG activity after consumption of a high volume of water was not observed either in the current study, although the obese participants showed higher percent gastric bradyarrhythmia compared to that in the normal-weighted participants. However, it is not clear whether the higher percent gastric bradyarrhythmia in the obese participants is related, to some extent, to the higher increase in the volume of the stomach, since the obese participants consumed more food than the normal-weighted participants.

Distension of the antrum also induces bradygastria. In one study, during the basal condition with no balloon distention, 3-cpm EGG signals were recorded. Balloon distension of the antrum in healthy subjects developed bradyarrhythmia in the stomach (Ladabaum, et al., 2001).

Furthermore, several studies have associated gastric bradyarrhythmia with strong antral contractions at a reduced frequency (Chen & McCallum, 1993). These studies

indicate that under certain conditions, gastric bradyarrhythmia waves reflect low-frequency antral contractions because in certain circumstances, the antrum contract at 1.5-1.8 contractions per minute rather than the more recognized 3-per-minute contraction (Sun, et al., 1995). Thus, the low-frequency contractile activity of the antral may be reflected in the low frequency EGG signals in certain situations. One study explored the relationship between bradygastria EGG and low-frequency antral contractions during fasting and after the infusion of erythromycin and found that bradygastria EGG frequently correlated with the low-frequency antral contractions. During fasting, 2-cpm EGG waves occurred and correlated with 2-per-minute antral contractions; after erythromycin infusion, the EGG waves were present at 1.0-1.5 cpm and correlated with stronger antral contractions that occurred at the same frequency (Sun, et al., 1995).

Since antral pressure of the participants was not measured simultaneously with electrogastrogram recording in the current study, there was no way to know if the higher percent gastric bradyarrhythmia that was observed in the obese group in the current study was accompanied by a stronger antral contraction compared to that in the normal-weighted group after the ingestion of food. But it will be very interesting to further study if the bradyarrhythmia and increased antral contraction exist stimulatingly in obesity since antral contraction plays an important role in solid food emptying (Kelly, 1980) and rapid gastric emptying was usually reported in obese participants (e.g. Wright, et al., 1983).

Stomach contractility is regulated by gastric myoelectrical activity, and numerous studies have shown that disturbances in the gastric slow wave are associated with impaired gastric motility. For example, abnormalities in gastric slow waves are

frequently observed in patients with functional disorders of the stomach, such as gastroparesis, functional dyspepsia, anorexia, etc. The gastric abnormalities include uncoupling and gastric dysrhythmia, which usually lead to gastric hypomotility. Tachygastria has been consistently shown to cause hypomotility of the stomach in functional dyspepsia patients (Chen & McCallum, 1994).

However, little is known about the characteristics of gastric electrical activity in hypermotility. Since bradyarrhythmia and rapid gastric emptying were reported to exist in obese participants simultaneously (McCallum, et al., 2001), and, as stated above, bradyarrhythmia may be related to antral contraction, it will also be very interesting to explore the relationship between bradyarrhythmia and rapid gastric emptying in obesity.

Furthermore, the results of the current study support the idea that modifying the gastric electrical rhythm might be considered as a therapy for morbid obesity.

In recent years, a number of studies have been performed to investigate the effects of implantable gastric stimulators on gastric motility and weight loss in obese patients. Cigaina et al were the first to investigate the potential for gastric electrical stimulation to induce weight loss in a porcine model in 1992. In the method proposed by Cigaina, electrical stimulation is performed at the lesser curvature using trains of short pulses with a pulse width in the order of microseconds and pulse frequency of about 40 Hz (2400 cpm). Recently, some researchers have proposed retrograde gastric electrical stimulation (RGES). In this method, electrical stimulation is performed at a tachygastrial frequency using a pair of electrodes placed in the distal antrum, mimicking an ectopic pacemaker generating tachygastria. This artificial ectopic pacemaker may result in electrical waves propagating retrogradely from the antrum to the proximal stomach,

fighting against the normal and physiological electrical waves that propagate from the proximal to the distal stomach. RGEs resulted in decreased gastric accommodation, a 16% reduction in the amount of acute food intake, and delayed gastric emptying in rats (Yin & Chen, 2005).

Basically, gastric electrical stimulation reduced gastric emptying and, therefore, decreased the amount of food obese people usually take in by inducing abnormal tachygastria in the stomach. Preliminary clinical studies by Cigaina et al showed that gastric electrical stimulation induced weight loss in morbidly obese patients (Cigaina, 2002). If there is bradyarrhythmia existing in obese patients, gastric electrical stimulation would result in a correction of gastric bradyarrhythmia and therefore normalize gastric emptying.

The results of the current study suggested more than normal bradygastria activity in obese participants. Presumably, gastric bradyarrhythmia may partly contribute to accelerated gastric emptying in obese patients or predispose one to overeat. More clinical studies need to be conducted to explore the relationship between gastric bradyarrhythmia, hypermotility and obesity. Nevertheless, if such differences in EGG in obesity exist, they may be important in the pathogenesis and/or persistence of obesity. It may also be important in the exploration of the mechanisms of implantable gastric stimulation.

4.3. Part Three: Effects of Second Food Image Exposure on Obese People

The objective of this part of the study was to investigate cephalic phase responses (CPRs) in obesity also. However, the difference between this part and the first part of the study was that in the current part of the study, the cephalic phase responses were

observed under a condition when the participants were full in the stomach while the first cephalic phase responses were observed under a condition when the participants had fasted for at least 4 hours. The purpose of this part of the study was to compare the cephalic phase responses in obese participants under a condition of being full and a condition of fasting. It also aimed to test the assumption that obese participants responded with more marked CPRs than normal-weighted participants to the presentation of food cues when they were full in the stomach.

Subjective responses to food cues and associated physiological responses in obese participants were compared to those in normal-weighted participants. Two major findings were: (1) The obese participants showed a greater desire to eat in response to food cues; (2) The obese participants responded similarly to food cues in gastric electrical activity compared to that in the normal-weighted participants when the participants were full in the stomach.

4.3.1. Interpretation of the Results of the Subjective Measurements

As a whole, the ratings for hunger, fullness and desire to eat were not significantly different from the ratings just after a meal but they were much lower than those during the first baseline period when the participants fasted. The obese participants rated similarly in their feelings of hunger, fullness and desire to eat compared to that in the normal-weighted participants 30 minutes after a meal.

It has been suggested that hypermotility decreased satiety in obesity and rapid gastric emptying lessens the feeling of satiation by reducing the satiety signals from the stomach (Park & Camilleri, 2004). We expected that the obese participants would feel less full in the stomach than the normal-weighted participants 30 minutes after a meal

based on the findings in one study which suggested that increased body mass index was associated with lower fullness scores 30 minutes after a meal (DelgadoAros, et al., 2004). This result was not confirmed by the current study which showed similar fullness ratings in the obese participants and the normal-weighted participants.

Comparing the results in the responses in the first food exposure period, the participants liked the food in the video clips less than when they were viewing the same food images for the first time. Furthermore, as a whole, the second food images exposure did not make the participants feel hungry and expect to consume as much food as the food images did in the first exposure period.

It was predicted that the obese participants would feel a stronger desire to eat and/or expect to eat more food while viewing the food images even though they still felt full in the stomach. This prediction was supported by the findings in the current study. During the second food images exposure period, the obese participants showed a stronger desire to eat than the normal-weighted participants; however, they did not show that they liked the food images more and they did not expect to consume more food than the normal-weighted participants either. Furthermore, the food images did not make them feel hungrier than normal-weighted participants.

This result is consistent with the findings in one study conducted on obese women. This study compared appetite ratings after lunch between obese and normal-weighted women and observed that appetite ratings strongly decreased after lunch in both groups and appetite for a meal or snack after lunch was significantly higher in obese than in normal-weighted subjects (Snoek, et al., 2004).

4.3.2. Interpretation of the Results of the Physiological Measurements

4.3.2.1. Electrogastrography (EGG)

Gastric electrical activity during the second food images viewing period was compared to that in the second baseline period. Across experimental groups, percent gastric normal activity during the food image exposure period did not significantly change compared to that in the second baseline period; while the percent of tachygastric activity decreased significantly and bradygastric activity increased accordingly. The results indicate that the participants responded to the same food cues differently in the stomach while being full compared to that while fasting. More specifically, the participants did not respond to pleasant food with an increase in normal gastric activity in the stomach when they were full.

The lower increase in normal gastric activity can be explained to some extent by the results of subjective measurements. When the participants were exposed to the food images, they still felt relatively full in the stomach; therefore, the food images did not make the participants feel hungry and viewing the food images did not initiate a strong desire to eat in the participants. Under this condition, the food images did not cause strong cephalic phase responses in the stomach. The results of the current study were in accordance with what was found in one study on the stomach's response to unappetizing food in our lab. In this study, cephalic phase responses were measured by changes in gastric myoelectric activity. Percent normal gastric activity decreased when the participants were fed with unappetizing food and increased in the group that was sham fed with appetizing food (Stern, et al., 2001). This study demonstrated 3 cpm EGG did not increase in response to unpleasant food.

Cephalic phase responses are elicited by exposure to food or food cues and cause

adjustments in the body to a coming meal either by direct sensory stimulation or by conditioned processes. The body prepares to facilitate the digestion of the food. However, when the food is not delicious to the subjects or when the subjects were full and did not want to eat more food, food may not cause very strong cephalic phase responses.

Although the obese participants showed a strong desire to eat while viewing the food images for the second time, percent normal gastric activity did not differ between the two groups during the second food images exposure period. However, a significant difference was found in percent normal gastric activity with the termination of food image exposure. More specifically, percent normal gastric activity was much higher in the normal-weighted groups than in the obese group during the second recovery period.

These results seem to be consistent with the ‘internal-external’ theory of obesity (Schachter & Nisbett, 1960). This theory suggests that obese individuals, relative to those of normal weight, were over-responsive to external cues (food cues) and under-responsive to internal cues (including feelings of hunger and satiety signals in body). In response to the food cues, the obese participants showed a stronger desire to eat; however, they did not feel more hungry and did not differ from the normal-weighted participants in the EGG activity in the stomach. EGG records the myoelectrical activity of the stomach, and the gastric myoelectrical activity and gastric contraction are more related to the feeling of hunger (Cannon & Washburn, 1912). Therefore, it is understandable that the participants of the two groups showed similar EGG activity when they felt similar level of hunger or fullness. Furthermore, the obese participants seem to be more reactive to the external cues – the food images and less responsive to the internal cues – feeling of hungry or fullness.

The increase in percent gastric bradyarrhythmia observed across the groups seems hard to explain. When the participants were divided into two groups, the results showed that the significant increase in percent gastric bradyarrhythmia only existed in the obese group but not in the normal-weighted group compared to that in the second baseline period. This result is very interesting since we also observed higher percent bradyarrhythmia in obese participants in the first food images exposure period and postprandial period. In the last 6-minute period after a meal (it is also considered as the second baseline period), percent gastric bradyarrhythmia started to decrease significantly compared to that in the first several 6-minute periods after a meal. However, with the stimulation of food images, percent gastric bradyarrhythmia increased in the obese participants. As stated in the second part of the study, it is not clear whether this increase in percent gastric bradyarrhythmia is related to stronger antral contractions, which further may contribute to hypermotility in the stomach in obese participants.

4.3.2.2. Skin Conductance Level (SCL) and Respiratory Sinus

Arrhythmia (RSA)

Similar to the results in the first food images exposure period, RSA decreased and SCL increased in the second food images exposure period compared to that in the baseline period. However, the difference between the two groups was not statistically significant. The results suggest that the obese participants did not show a stronger increase in sympathetic nervous system activity than the normal-weighted participants while viewing the food images.

4.4. Conclusions

The main purpose of this study was to investigate gastric myoelectrical activity in obesity during different periods of ingestion and to test the assumption that obese participants respond more strongly physiologically and psychologically to the presentation of food images and/or during the ingestion of food. Specifically, we predicted that the obese participants would show greater increases in normal gastric activity in response to food images and/or food compared to the normal-weighted participants.

We examined the cephalic phase responses in the obese participants when fasted. Food exposure caused a strong increase in the feeling of hunger and a decrease in the feeling of fullness in all the participants. The obese participants reported a stronger desire to eat when being exposed to the food images compared to the normal-weighted participants. The stronger desire to eat despite the equal level of hunger in the obese participants compared to that in the normal-weighted group was in line with the obese participants expecting to eat more food. Accordingly, the higher estimation of the amount of food to eat was associated with a higher desire to eat.

As expected, food image exposure also had a pronounced effect on physiology. Normal gastric activity changed appropriately in response to food image exposure in all participants, thus confirming the external validity of the current experimental design in eliciting CPRs. It was expected that different physiological cephalic phase responses would be presented between the obese and normal-weighted participants. The findings of this study support this notion, at least in part. There was a steep increase in percent normal gastric activity levels only in the normal-weighted participants during food

images exposure, and the obese participants showed more gastric bradyarrhythmia during this period.

The obese participants consumed more food than the normal-weighted participants and reached similar levels of fullness and hunger. Contrary to what was hypothesized, normal gastric activity was not higher in the obese participants after eating food; instead, gastric bradyarrhythmia was higher in the obese participants than the normal-weighted participants within the 30 minutes after the meal.

The obese participants showed a stronger desire to eat than the normal-weighted participants when they were viewing the food images while being full in the stomach although the food images did not make the obese participants feel more hungry than the normal-weighted participants. No significant increase in normal gastric activity was observed in response to food images of the obese participants and there was no significant difference in normal gastric activity between the two groups. Although there was no significant difference in percent bradygastria activity between the two groups during the second food image exposure period, an increase in gastric bradyarrhythmia was observed only in the obese group compared to that in the second baseline period.

In conclusion, these data suggest that obese people react to food images and food eating differently from normal-weighted people. We demonstrated that the obese participants showed more marked cephalic phase responses to food images both in the fasting period and satiety period than the normal-weighted participants. Furthermore, the obese participants seem to show more gastric bradyarrhythmia in response to food images and food eating. As of today, there is no definitive consensus on the characteristics of gastric electrical activity in obesity. The current study is only a preliminary exploration of

EKG in obesity. It is not known whether the possible increased gastric bradyarrhythmia in the obese participants observed in the current study is an etiologic factor in the development of obesity or it is simply a result of an adaptive response to overeating. Whether there are alterations in gastric electrical activity in response to a meal in obese patients as a further possible reason for altered food intake remains to be established.

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APPENDIX 1

The Pennsylvania State University
The Department of Psychology
Informed Consent

Title of Investigation: Gastric myoelectrical activity in viewing and eating food in overweight participants

Investigators: Robert M. Stern, Ph.D.

Jiangyue Wang

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Explanation of the Study

Eligibility: Penn State students who do not have a history of brain, heart, stomach, or lung disease are eligible to be in this experiment.

Purpose: **The purpose of this experiment is to investigate the characteristics of gastric activity in obese people. More specifically, we are going to explore the reactions of stomach to food during each phase of digestion in obese people and compare them to that of normal weighted people.**

Description: You will be assigned to one of two groups based on your BMI (Body Mass Index) score. If your BMI score is between 18.5-24.9, you are in the normal weighted group; and if your BMI score is between 30-34.9, you are in the over weighted group. You are required not to eat food in at least 4 hours and not drink alcohol, tea, coffee, cola, or any caffeinated beverage for at least 24 hours preceding the experiment.

If you agree to participate in this study, you will take part in the following procedure: Three sensors will be placed on your skin surface over your stomach in order to monitor your stomach activity. Three more sensors will be placed on your skin to monitor your heart activity. One will be placed over your bottom right rib, one on your sternum, and the third will be placed on your right side of abdomen. Before the sensors are placed on your skin, the area will be rubbed with a rough material to remove any oils so the sensors will stick better. A breathing gauge will also be placed around your mid-section to monitor your breathing. When all the sensors have been placed, you will be seated quietly in a comfortable chair. We will ask you to sit quietly for a 6-minute baseline period, which is followed by watching a video clip about food for 6 minutes. There will then be a 20 minutes recovery period in which you will sit quietly. After this 6-minute period, you will be asked to eat some pizza for 6 minutes, after which you need to sit for 30 minutes. Then you will be asked to watch the video clip again for 6 minutes. You need to sit quietly again in the next 6 minutes,

You will be asked to rate the pleasantness of both the food they were asked to imagine eating, as well as the food they actually ate at the end of watching food section or eating food section.

Discomforts and Risks: There is no risk from the recording of the activity of the stomach. There could be a risk of redness and irritation to the skin from the electrode sensors.

Length of Study: Your participation in the study will take approximately 2 hour.

Compensation: You will receive course credit for participating as specified in the syllabus provided by your instructor. Alternative means for earning this course credit are available as

APPENDIX 2

The Pennsylvania State University
The Department of Psychology
Debriefing Statement

Title of Investigation: Gastric myoelectrical activity in viewing and eating food in obese participants

Investigators: Rober M. Stern, Ph.D.
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According to recent statistical observation, more than 7.5% of the population in the United States suffers from obesity. Obesity has become one of the most common and steadily increasing diseases. Many different studies have been undertaken with a focus on behavioral, genetic, and epidemiological aspects. However, its etiology and characteristics have not yet been fully defined. The study in which you have just participated is an investigation of the characteristics of gastric activity in obese people. More specifically, we are going to explore the reactions of stomach to either food image or food during each phase of digestion in obese people. We also compared the gastric activity in obese participants to that of normal weighted participants.

We hypothesized that obese people react to food in stomach differently from normal weighted people. If any difference exists, we are also interested in that in which phase of digestion that the difference occurs. It is hoped that the results of this study will improve our understanding of the etiology of obesity.

If you would like to talk with someone about this study, please feel free to contact Dr. Robert Stern, 865-1717, rs3@psu.edu, 512 Moore Building or Jiangyue Wang, 865-1580, juw120@psu.edu, 453 Moore Building. Questions about the rights of research participants may be directed to Office for Research Protections (814)-865-1775.

Thank you very much for participating in this study.

APPENDIX 3

Participant Questionnaire

Participant Number: _____ Date: _____

Age: _____ Ethnicity: _____ Time: _____

Sex: _____ **Psychology Course/Section/ Instructor:** _____

You may not have used any alcohol or drugs within the past 24 hours as well as consumed coffee, tea, cola, etc.

If you have done any of these activities, you cannot participate in this research today.

Do you need to reschedule? Y / N

If not, please answer these questions.

When was the last time you ate? _____

What was the last thing you ate? _____

Have you smoked in the past 24 hours? Y / N

Have you exercised in the past 3 hours? Y / N

Do you have any neurological disorders that you are aware of? Y / N

Do you have any cardiovascular disorders that you are aware of? Y / N

Do you have any respiratory disorders that you are aware of? Y / N

Do you have any gastrointestinal disorders that you are aware of? Y / N

APPENDIX 5

PANAS

This scale consists of a number of words that describe different feelings and emotions. Read these words and then put a vertical mark on place that best represents the amount of each emotion experienced during eating the food.

Interested:	
	Not at all	Very much
Distressed:	
	Not at all	Very much
Excited:	
	Not at all	Very much
Upset	
	Not at all	Very much
Strong	
	Not at all	Very much
Guilty	
	Not at all	Very much
Scared	
	Not at all	Very much
Hostile	
	Not at all	Very much
Enthusiastic	
	Not at all	Very much
Proud	
	Not at all	Very much
Irritable	
	Not at all	Very much
Alert	
	Not at all	Very much
Ashamed	
	Not at all	Very much
Inspired	
	Not at all	Very much
Nervous	
	Not at all	Very much
Determined	
	Not at all	Very much
Attentive	
	Not at all	Very much
Jittery	
	Not at all	Very much
Active	
	Not at all	Very much
Afraid	
	Not at all	Very much

Tables

Table1. Characteristics of Obese and Normal-weighted Participants

	Normal-Weighted Group	Obese Group
Body Mass Index* (kg/m²)	21.6 (18.5-24.3)	32.1 (30 -34.5)
Age*	20 (18-23)	19 (18-23)
Gender**		
Male (N)	11	13
Female (N)	9	8

Note: *Values shown in the tables are medians and ranges.

** Values shown in the tables are number of subjects.

Table 2.1. Physiological Dependent Measures from Part I Experiment as a Function of Experimental Period and Experimental Group

Experimental Group	Experimental Period		
	Baseline 1	Food Viewing 1	Recovery 1
	Percent Normal Gastric Activity (2.5-3.75cpm)		
Obese	30.44±2.14	33.46±2.35	33.52±2.87
Normal	29.31±2.49	44.19±3.30	41.51±4.52
Across	29.89±1.62	38.69±2.16	37.42±2.69
	Percent Gastric Tachyarrhythmia (3.75-10cpm)		
Obese	25.83±2.30	24.63±3.24	31.97±1.92
Normal	27.22±1.90	23.27±2.51	26.45±2.63
Across	26.51±1.48	24.99±2.03	29.27±1.65
	Percent Gastric Bradyarrhythmia (1-2.5 cpm)		
Obese	39.08±3.15	33.10±1.89	27.28±2.55
Normal	38.95±3.17	22.66±2.91	24.26±3.64
Across	39.02±2.21	28.01±1.89	25.81±2.19
	Respiratory Sinus Arrhythmia		
Obese	7.48±0.26	7.14±0.25	7.44±0.24
Normal	7.05±0.25	6.82±0.22	6.95±0.23
Across	7.26±0.18	6.98±0.17	7.20±0.17
	Skin Conductance Level		
Obesity	27.98±4.09	37.27±6.28	33.96±4.67
Normal	28.12±5.60	27.93±4.64	27.87±5.27
Across	28.05±3.40	32.71±3.95	30.99±3.50

Note. Values represent lease square means ± standard errors of the means. Respiratory sinus arrhythmia data are presented in ln units. Skin conductance level data are presented in microsiemens (μS).

Table 2.2. Subjective Dependent Measures from Part I Experiment as a Function of Experimental Group

Experimental Group	Baseline 1	Food Viewing 1
	Ratings of Hunger	
Obese	62.00±4.41	74.53±4.07
Normal	60.05±5.12	70.68±4.64
Total	61.05±3.33	72.61±3.06
	Ratings of Fullness	
Obese	18.52±3.36	15.53±4.21
Normal	26.20±4.77	23.42±3.99
Total	22.27±2.92	19.47±2.94
	Ratings of Desire to Eat	
Obese	62.24±5.03	82.33±3.03
Normal	60.25±4.89	70.65±4.32
Total	61.27±3.47	76.63±2.75
	Ratings of The Amount of Food Expected	
Obese	67.52±3.45	77.00±2.51
Normal	61.90±4.15	69.80±3.85
Total	64.78±2.69	75.44±2.41
	Ratings of the Extent of Liking the Clips	
Obese	-	74.26±3.82
Normal	-	71.37±3.83
Total	-	72.82±2.68

Note. Values represent means ± standard errors of the means. Respiratory sinus arrhythmia data are presented in ln units. Skin conductance level data are presented in microsiemens (μS).

Table 3.1. Physiological Dependent Measures from Part II Experiment as a Function of Experimental Period and Experimental Group

	Experimental Period		
Experimental Group	Baseline 1	1 st 6-minute	2 nd 6-minute
	Percent Normal Gastric Activity (2.5-3.75cpm)		
Obese	30.44±2.14	34.57±2.87	34.54±2.89
Normal	29.31±2.49	45.89±4.01	38.44±4.35
Across	29.89±1.62	40.23±2.60	36.49±2.60
	Percent Gastric Tachyarrhythmia (3.75-10cpm)		
Obese	25.83±2.30	24.73±2.30	31.23±2.20
Normal	27.22±1.90	25.27±2.92	31.04±3.94
Across	26.51±1.48	30.60±1.85	31.13±2.23
	Percent Gastric Bradyarrhythmia (1-2.5 cpm)		
Obese	39.08±3.15	30.40±3.48	26.45±2.79
Normal	38.95±3.17	15.83±2.97	19.64±3.01
Across	39.02±2.21	23.11±2.54	23.04±2.10

Note. Values represent means ± standard errors of the means. Respiratory sinus arrhythmia data are presented in ln units. Skin conductance level data are presented in microsiemens (μS).

Table 3.1. Physiological Dependent Measures from Part II Experiment as a Function of Experimental Period and Experimental Group (Cont'd)

	Experimental Period		
Experimental Group	3 rd 6-minute	4 th 6-minute	5 th 6-minute
	Percent Normal Gastric Activity (2.5-3.75cpm)		
Obese	30.26±2.41	28.75±2.68	33.25±2.51
Normal	41.63±4.02	40.50±4.01	39.65±2.80
Across	35.80±2.46	34.78±2.59	36.45±1.93
	Percent Gastric Tachyarrhythmia (3.75-10cpm)		
Obese	36.61±3.05	37.91±2.55	43.37±2.45
Normal	32.86±2.75	32.10±2.52	35.49±2.62
Across	34.78±2.06	34.93±1.83	39.43±1.88
	Percent Gastric Bradyarrhythmia (1-2.5 cpm)		
Obese	27.29±2.66	27.61±2.86	17.11±2.18
Normal	20.42±2.37	18.79±2.83	20.41±1.42
Across	23.94±1.85	23.09±2.11	18.76±1.31

Note. Values represent means ± standard errors of the means. Respiratory sinus arrhythmia data are presented in ln units. Skin conductance level data are presented in microsiemens (μS).

Table 3.2. Subjective Dependent Measures from Part II Experiment as a Function of Experimental Group

Experimental Group	meal
	How hungry
Obese	22.05±4.86
Normal	22.70±3.78
Total	22.38±3.04
	How Full
Obese	76.00±4.79
Normal	73.80±4.96
Total	74.90±3.41
	How strong Desire
Obese	21.24±4.85
Normal	25.20±3.82
Total	23.17±3.08
	How much food expect to eat
Obese	27.90±4.69
Normal	26.55±3.29
Total	27.24±2.86
	How like Pizza
Obese	67.85±4.13
Normal	59.80±5.28
Total	63.83±3.37
	How many pizza (percent)
Obese	85.48±5.66
Normal	67.50±5.80
Total	76.71±2.72

Note. Values represent means ± standard errors of the means. Respiratory sinus arrhythmia data are presented in ln units. Skin conductance level data are presented in microsiemens (μS).

Table 4.1. Physiological Dependent Measures from Part III Experiment as a Function of Experimental Period and Experimental Group

	Experimental Period		
Experimental Group	Baseline 2	Food Viewing 2	Recovery 2
	Percent Normal Gastric Activity (2.5-3.75cpm)		
Obese	33.25±2.51	35.63±3.04	34.74±3.16
Normal	39.65±2.80	40.12±2.98	44.77±3.16
Across	36.45±1.93	37.82±2.13	39.51±2.35
	Percent Gastric Tachyarrhythmia (3.75-10cpm)		
Obese	17.11±2.18	23.70±2.92	20.21±2.98
Normal	20.41±1.42	22.27±2.11	19.69±2.65
Across	18.76±1.31	23.00±1.80	19.96±1.98
	Percent Gastric Bradyarrhythmia (1-2.5 cpm)		
Obese	43.37±2.45	34.19±2.16	38.23±2.50
Normal	35.49±2.62	32.96±1.94	30.58±2.50
Across	39.43±1.88	33.59±1.44	34.60±1.85
	Respiratory Sinus Arrhythmia		
Obese	6.84±0.23	6.52±0.25	6.83±0.19
Normal	6.82±0.20	6.42±0.22	6.47±0.27
Across	6.83±0.15	6.47±0.16	6.68±0.16
	Skin Conductance Level		
Obesity	34.50±6.75	41.20±7.74	40.10±7.24
Normal	32.83±8.94	37.43±6.89	49.24±10.5
Across	33.69±5.50	39.36±5.14	44.56±6.27

Note. Values represent means ± standard errors of the means. Respiratory sinus arrhythmia data are presented in ln units. Skin conductance level data are presented in microsiemens (μS).

Table 4.2. Subjective Dependent Measures from Part III Experiment as a Function of Experimental Group

Experimental Group	Baseline 2	Food Viewing 2
	How hungry	
Obese	20.80±2.77	26.65±5.00
Normal	27.90±5.05	30.20±4.42
Total	24.35±2.90	28.43±3.31
	How Full	
Obese	68.80±4.71	66.65±5.48
Normal	68.60±5.10	72.80±4.05
Total	68.70±3.42	69.73±3.40
	How strong Desire	
Obese	23.86±3.60	38.52±3.74
Normal	26.30±3.97	29.15±3.98
Total	25.05±2.65	33.95±2.79
	How much food expect to eat	
Obese	31.81±4.04	39.55±3.93
Normal	28.10±4.21	32.50±3.67
Total	30.00±2.89	36.03±2.71
	How like the food in the video clips	
Obese	-	67.25±3.70
Normal	-	62.45±4.82
Total	-	64.85±3.02

Note. Values represent means ± standard errors of the means. Respiratory sinus arrhythmia data are presented in ln units. Skin conductance level data are presented in microsiemens (μS).

Figures

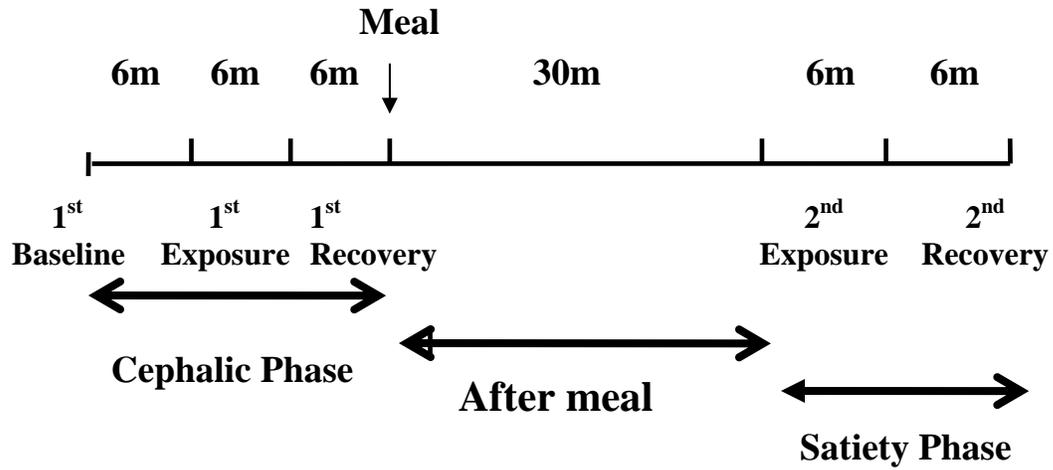


Figure 1. Timeline of the Procedures

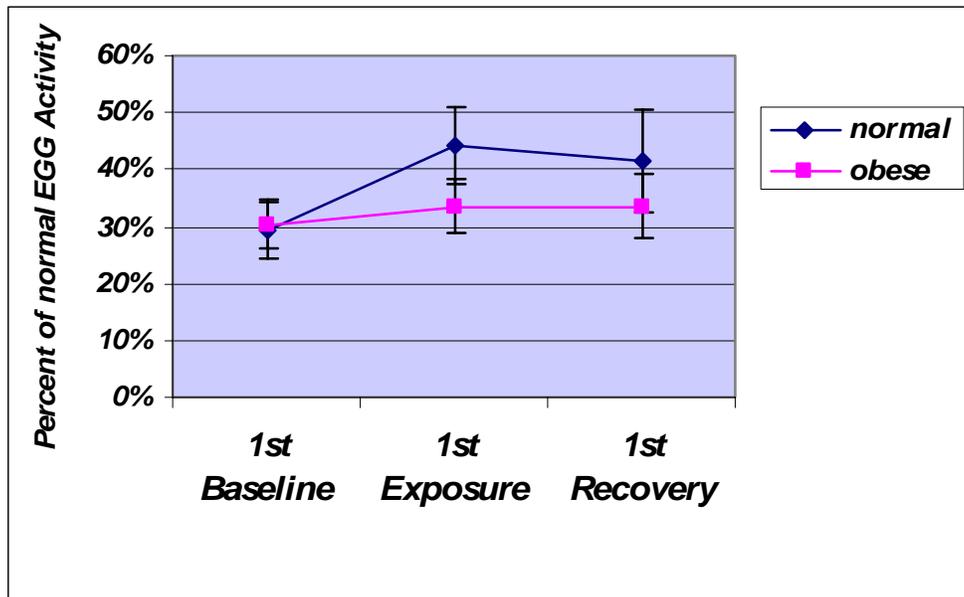


Figure2.1. Percent of Normal Gastric Activity (Part I)

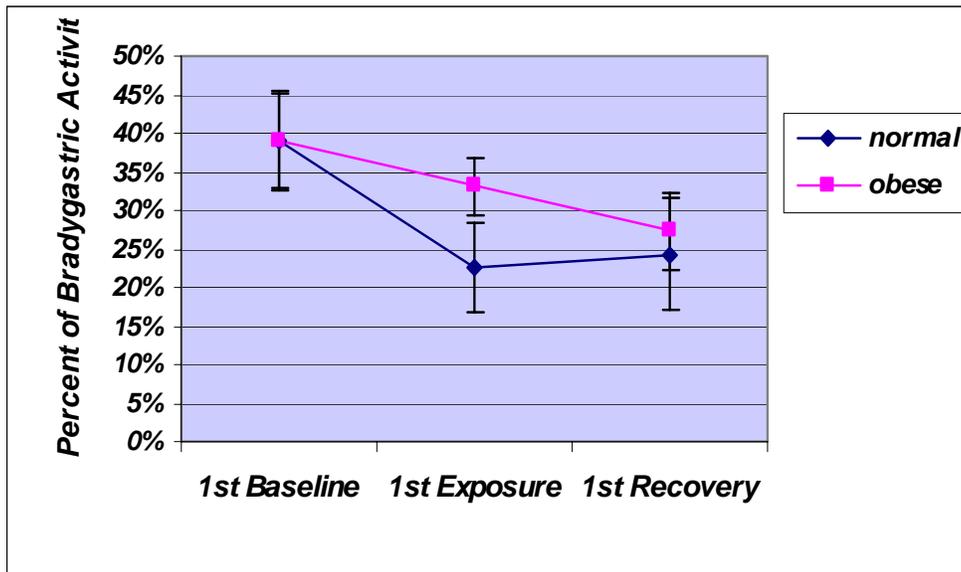


Figure2.2. Percent of Bradygastric Activity (Part I)

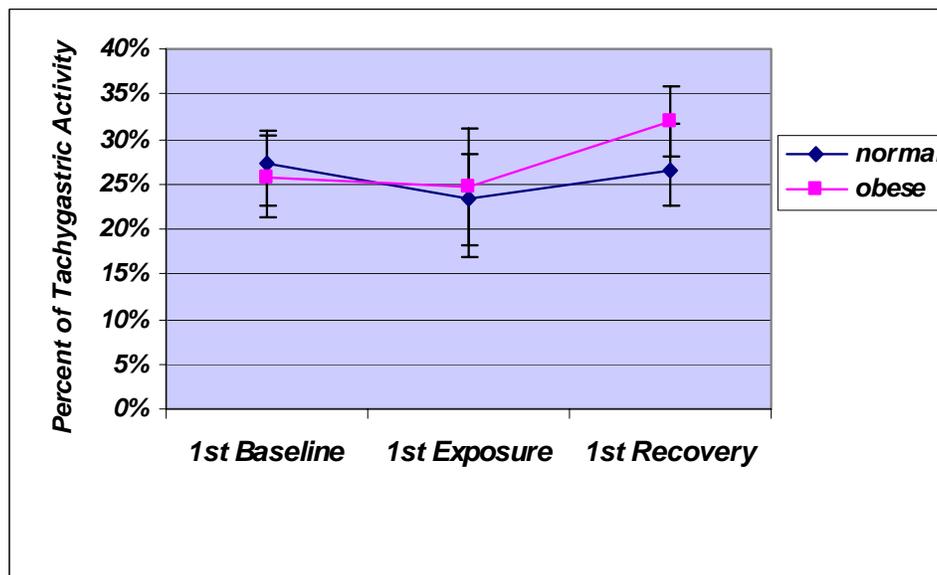


Figure2.3. Percent of Tachygastric Activity (Part I)

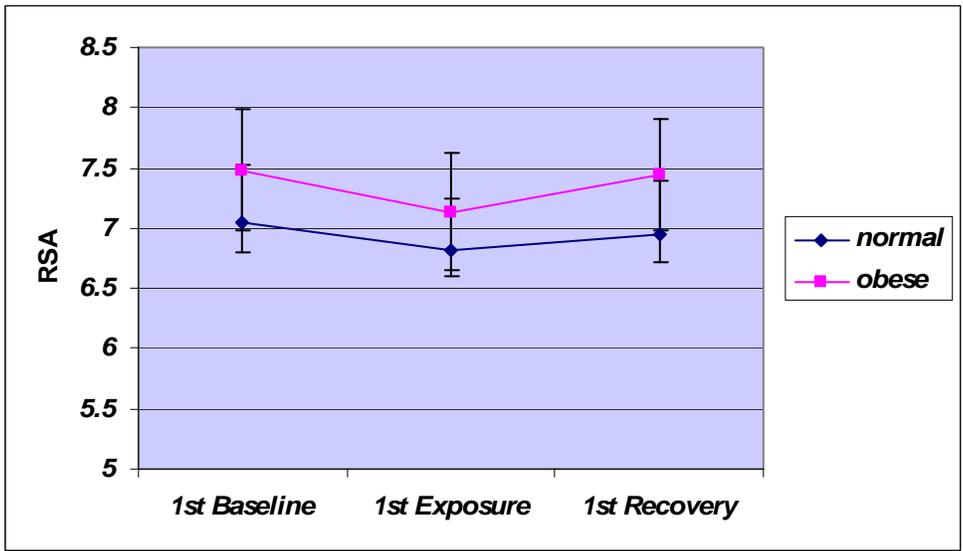


Figure2.4. RSA data (Part I)

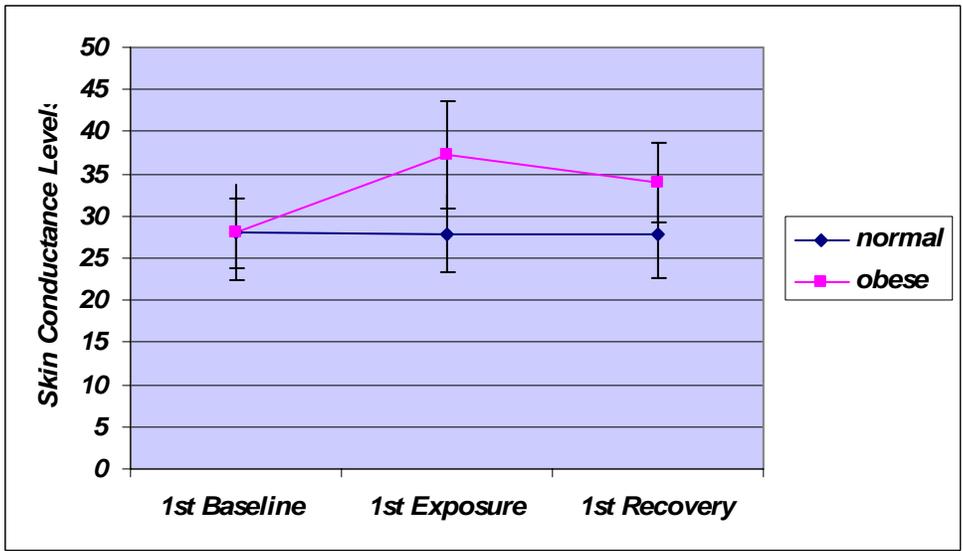


Figure2.5. SCL data (Part I)

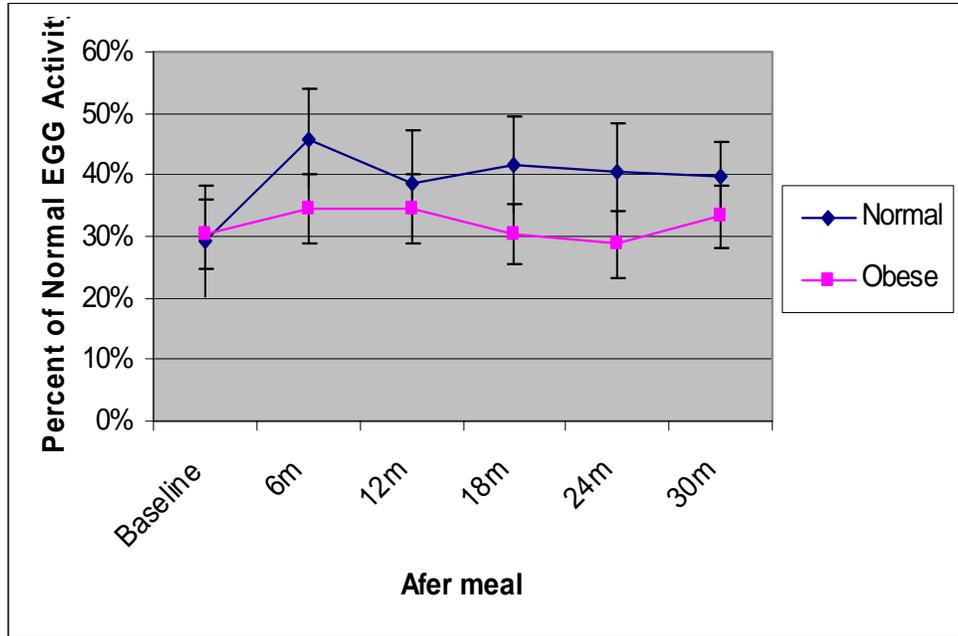


Figure 3.1. Percent of Normal Gastric Activity (Part II)

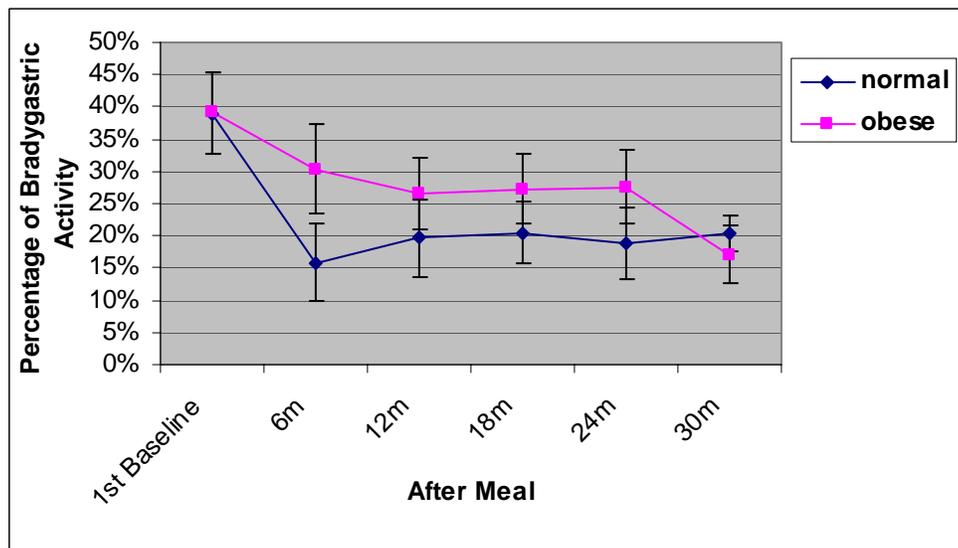


Figure 3.2. Percent of Bradygastric Activity (Part II)

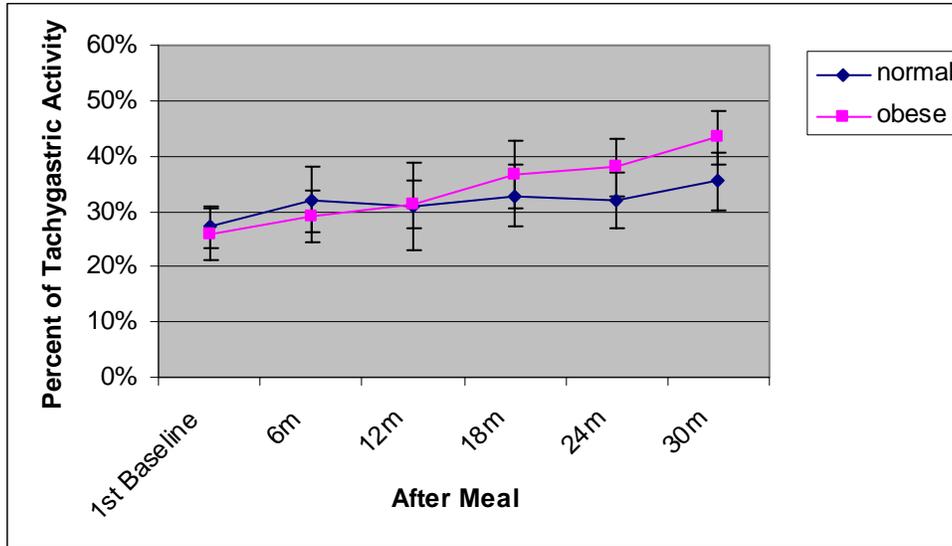


Figure 3.3. Percent of Tachygastric Activity (Part II)

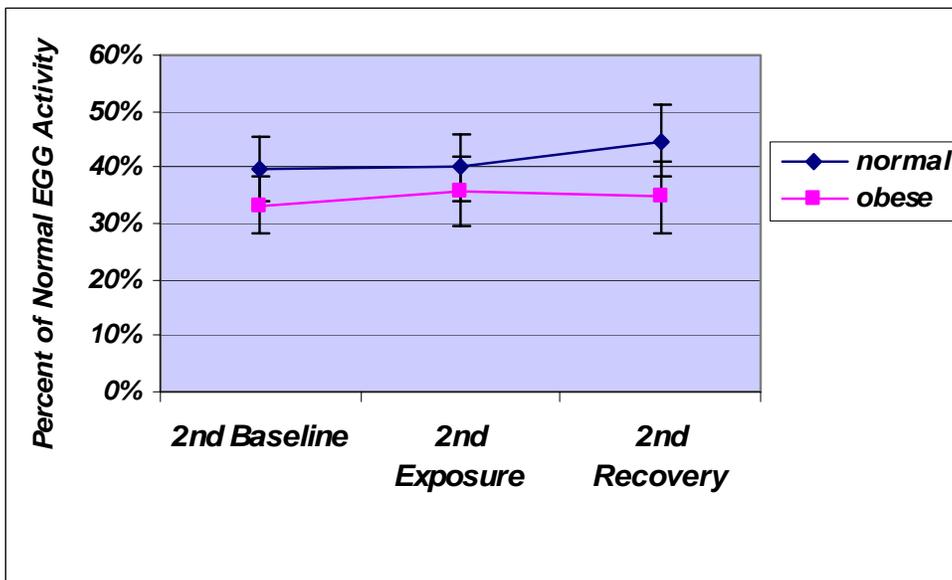


Figure 4.1. Percent of Normal Gastric Activity (Part III)

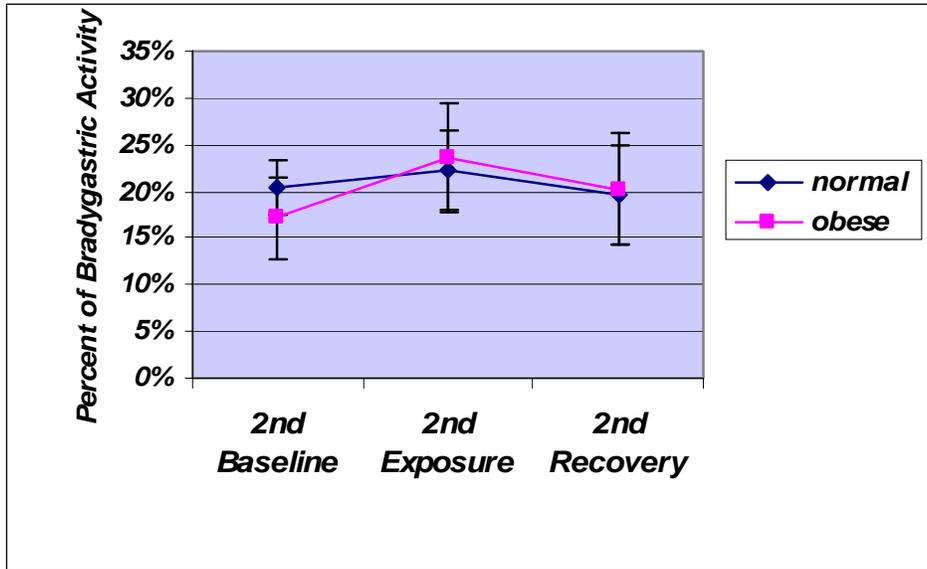


Figure 4.2. Percent of Bradygastric Activity (Part III)

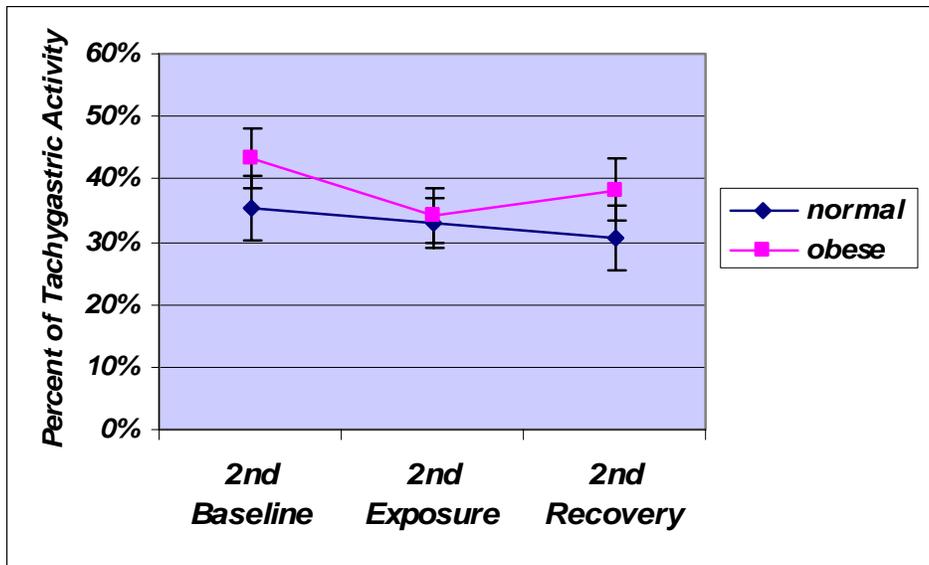


Figure 4.3. Percent of Tachygastric Activity (Part III)

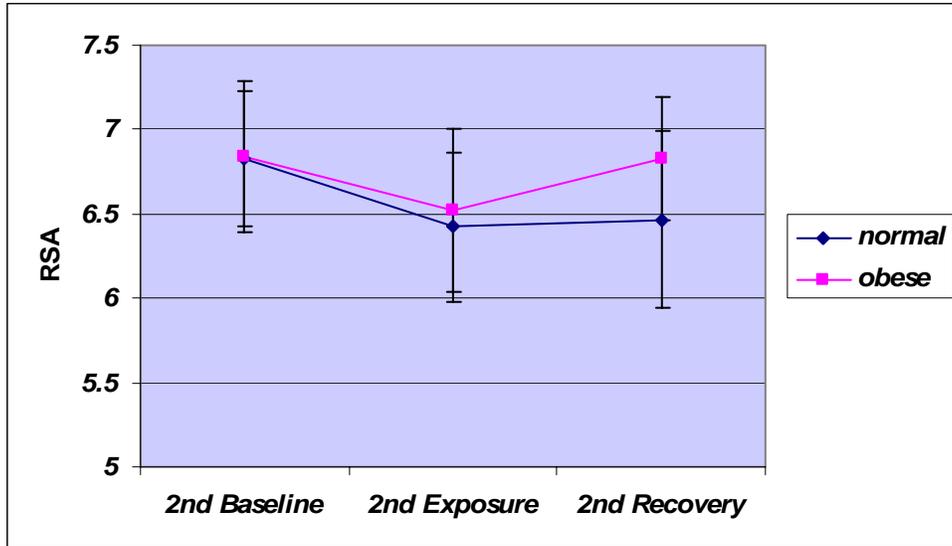


Figure 4.4. RSA data(Part III)

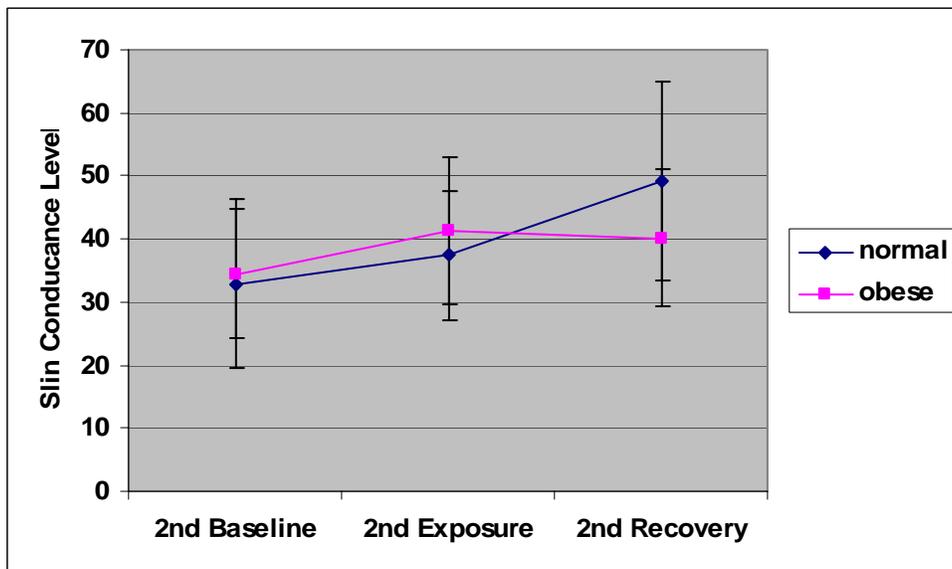


Figure 4.5. SCL data (Part III)

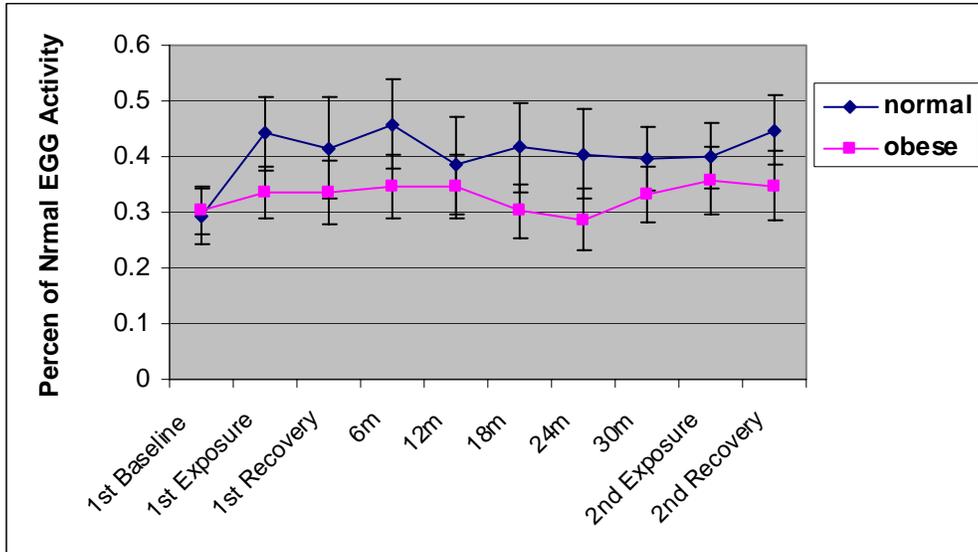


Figure 4.6. Percent of Normal Gastric Activity (Part I, II and III)

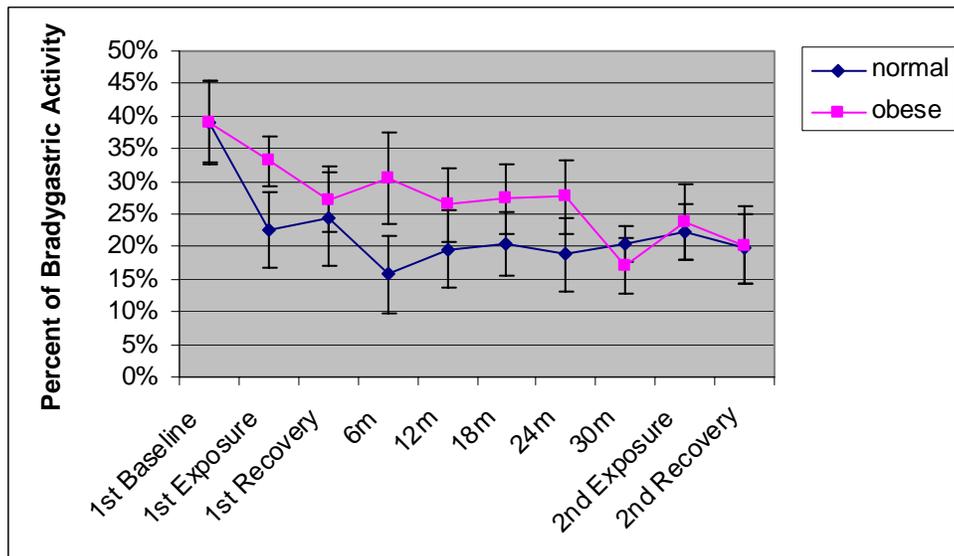


Figure 4.7. Percent of Bradygastric Activity (Part I, II and III)

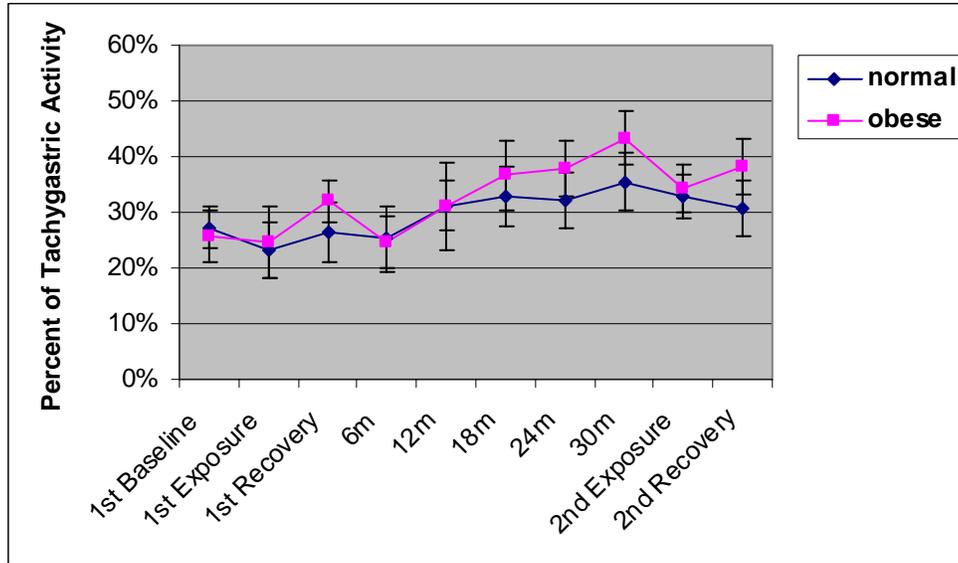


Figure 4.8. Percent of Tachygastric Activity (Part I, II and III)

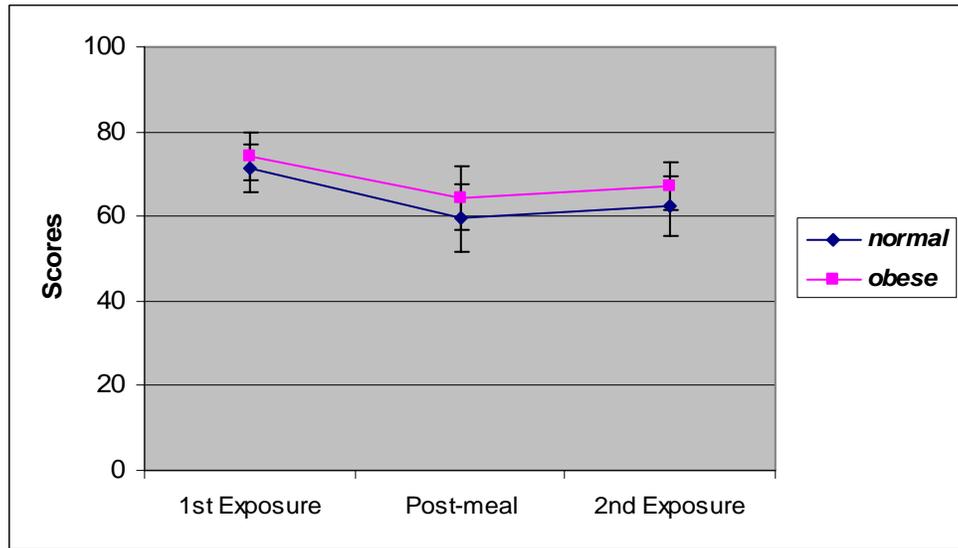


Figure 5. 1. Ratings of the Extent of Likeness of the Food (or Food Images)

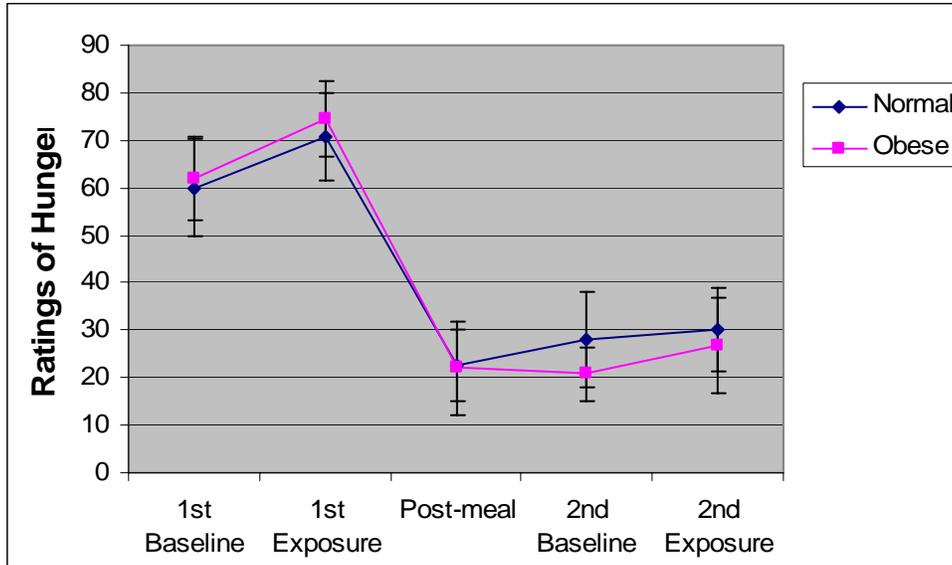


Figure 5.2. Ratings of Hunger

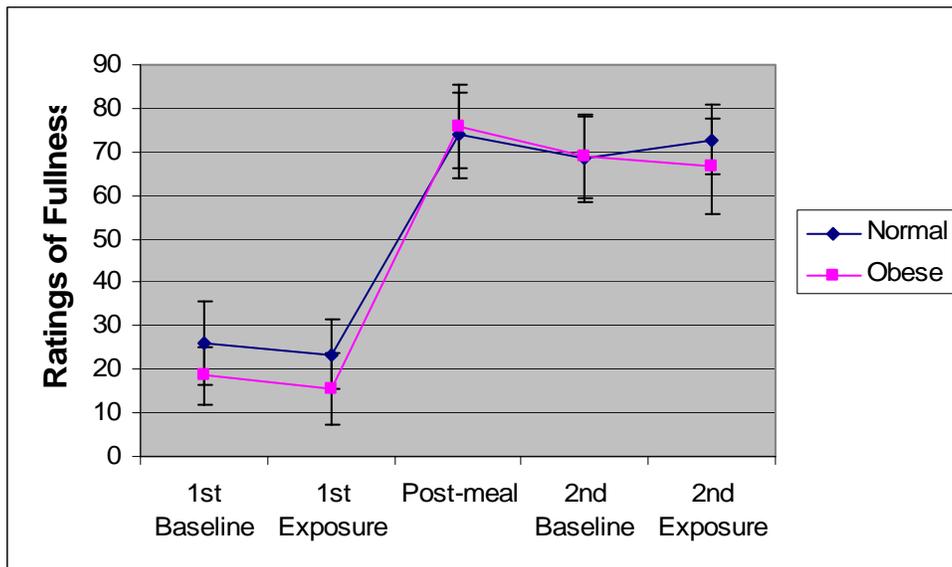


Figure 5. 3. Ratings of Fullness

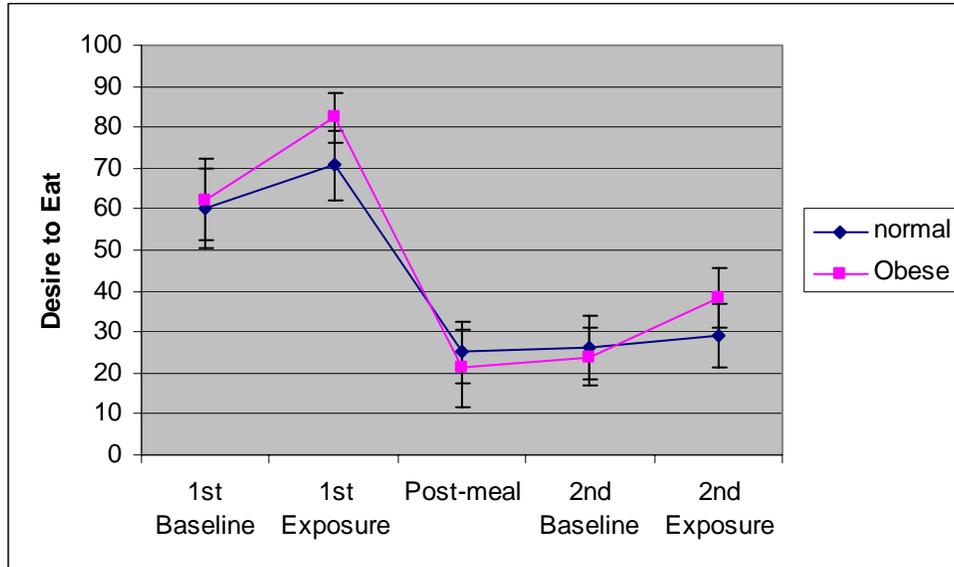


Figure 5. 4. Ratings of Desire to Eat

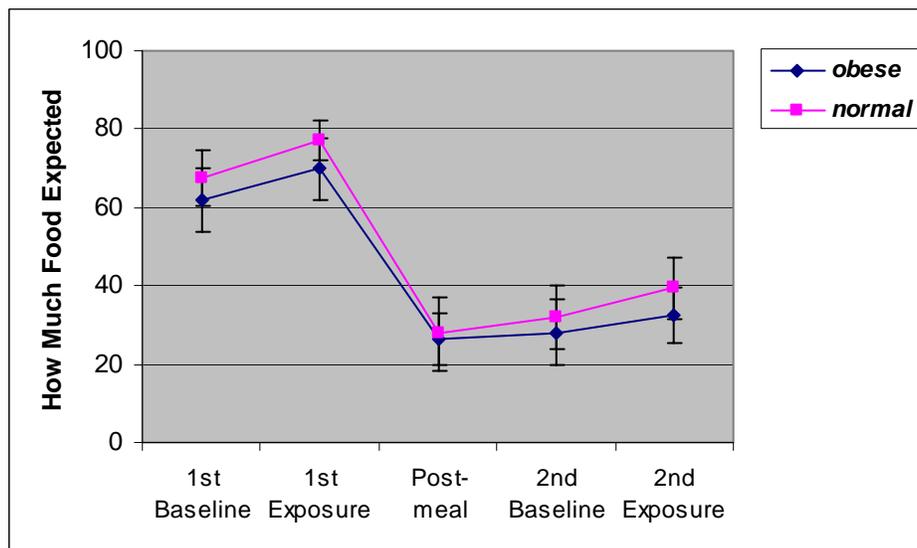


Figure 5. 5. Ratings of the Amount of Food Expected

Vita

Jiangyue Wang received her Medical Doctor degree at the China Medical University, ShengYang, China, in 1999. She worked as a resident physician at the 1st Affiliated Hospital at the Dalian Medical University, DaLian, China from 1999-2001. In August 2001, she was enrolled in the graduate program in Psychology at the Pennsylvania State University and began to pursue her PhD degree. In August 2004, she was enrolled in the master of applied statistics program to pursue her concurrent degree in Statistics. Her research interests include the impact of stress on health, motion sickness, and psychophysiological mechanisms of obesity.