AN EXAMINATION OF DIETARY RESTRAINT, APPETITE AND DISINHIBITION USING ELECTROPHYSIOLOGICAL MEASUREMENT OF PAROTID ACTIVITY

by

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ABSTRACT

The central issue examined by the present research was the degree to which disinhibited eating could be accounted for by cognitive versus physiological factors. The Boundary Model holds that disinhibitory cognitions after the ingestion of a preload lead to excessive ingestion. The present research contends that inconsistent meal sizes, typical of most dieters, leads to a conditioned exaggeration of physiological response to food, making it difficult to stop eating once started. Validation of a measure of physiological appetite (electrophysiological recording of the parotid gland, ESG) is presented: ESG to food reliably distinguished fasted from nonfasted, and restrained from nonrestrained subjects. ESG was then used to test the hypothesis that physiological response to food would be a better predictor of disinhibited eating than either dietary restraint or subjective appetite. Although ESG was the best predictor of amount eaten following a preload, subjective appetite also contributed to the prediction. Dietary restraint did not. It was also found that elevated ESG and subjective appetite were correlated with inconsistent eating patterns on diet records. The results suggest that disinhibited eating is not caused exclusively by disinhibitory cognitions, but that heightened physiological response to food, possibly resulting from the conditioning effects of irregular eating patterns, also contributes.

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GENERAL INTRODUCTION

Over the past two decades there has been an unprecedented expansion of interest in and research on disorders of appetite and body weight regulation. For the most part, the public and popular media have focused their attention on the more severe forms of these disorders, and have typically represented cases of anorexia nervosa and bulimia nervosa in dramatic fashion. This popular interest has also been reflected in the scientific literature in which case studies, clinical reports, and descriptions of treatment approaches have predominated. Many authors introduce their reports with reference to the "epidemic" nature of these disorders, and it is the sense of dealing with a rapidly expanding epidemic which pervades the clinical literature.

Early on, authors such as Bruch (1973), Selvini Palazzoli (1974), and Minuchin (Minuchin, Rosman & Baker, 1978) recognized that there were distinctive difficulties inherent in treating these patients which rendered the usual approaches to treatment, whether psychoanalytic, medical or behavioral, inadequate if not naive. Development of more appropriate treatment strategies was hampered both by a lack of a unified theoretical approach and by a lack of standardized diagnostic categories. Although in the past

few years there has been some success in delineating diagnostic criteria (cf. APA, 1994), advances in the areas of etiology and treatment continue to be hampered by the diversity of theoretical approaches.

Research has tended to remain focused on clinical issues. Among these are behavioral patterns and symptomatology, personality factors and underlying or concomitant psychopathology, intrapsychic structure, defenses and psychodynamics, family functioning and interactions, cognitive styles, and treatment approaches. In contrast to this intense focus on issues directly relevant to clinical management, there has been a relative paucity of research exploring the "eating" aspect of eating disorders. Basic research on body weight regulation, ingestion patterns, appetite, satiety, gastrointestinal activity, and metabolism has lagged behind clinical research.

Although gains have been made in the elaboration and testing of models of ingestive behavior and body weight regulation (cf. Powley, 1977; Cioffi, James, & Van Itallie, 1981; Van Itallie & Kissileff, 1985; Rodin, 1985), few researchers have made more than cursory attempts at bridging the gap between basic research on eating and clinical research on eating disorders. For example, based on the initial assertion by Bruch (1973) that anorexia nervosa patients suffered from deficits in interoceptive awareness

and regulation of internal states, these putative clinical features were assumed to have etiological significance and became the focus of some treatment approaches (Goodsitt, 1983; Davis & Marsh, 1986; Stern, 1986). In fact, little or no direct research which addresses this hypothesis has appeared.

Other researchers have chosen the opposite approach to the problem by developing an etiological model based on analogue research using nonclinical populations of subjects who exhibit milder forms of eating symptoms. The main proponents of this approach, Herman and Polivy (1984), as well as Ruderman (1985a), have developed a model in which attempts to lose weight through reduction of caloric intake, dietary restraint, lead directly to periodic binges, which can become clinically significant in individuals with an unspecified general psychopathology or vulnerability.

Externality Hypothesis

The theory that dietary restraint led to binge-eating, which contradicted the generally held view that dieting was a necessary response to excessive intake, originally developed out of research on obesity which was attempting to show that overweight individuals overate because of a hyper-responsiveness to external cues to eating (Schachter & Rodin, 1974). In support of what later came to be known as the Externality Theory, Schachter and his associates found that overweight individuals were more sensitive to, and

would overeat in response to external cues to eating, such as time of day (Schachter & Gross, 1968), or availability of food (Nisbett, 1968a & b). Later, Schachter proposed a more general form of the theory in which obese individuals were described as being more responsive to a wide variety of external stimuli, not just those relating to food and eating (Schachter, 1971).

Leon and Roth (1977) noted a number of inconsistencies in the findings related to the externality hypothesis. Although a number of studies were able to show reliable response differences between overweight and normal weight subjects (eg. Schachter & Rodin, 1974), many were not (Nisbett & Temoshok, 1976; Wooley, 1972). Ruderman (1986) has elaborated on what appear to be the central problems with this research area in general.

She notes, first of all, a lack of good definitions of external responsiveness in the studies of externality focusing on nonfood issues (i.e. testing the more general form of the externality hypothesis). Second, there is the difficulty of defining internal versus external cues -- in many instances this distinction appears blurred, if not arbitrary. Finally, the intensity of cues has not been objectively established, making it difficult to test the hypothesis that obese individuals were more responsive to intense stimuli. Rodin (1981) provided the additional criticism that much of this research used inconsistent

sampling procedures which allowed for a confound of body weight differences with variability in external responsiveness. Despite inconsistent and contradictory results, the externality hypothesis persisted in the literature, perhaps because, as Wooley and Wooley (1984) suggested, it reflected the popular notions of the time regarding the causes of obesity.

The externality theory was eventually refuted by two lines of research which showed that: 1) the relationship between overeating and overweight was not as straightforward as many assumed, and in fact, many overweight individuals were chronically restricting their intake (Garrow, 1974; Wooley, Wooley & Dyrenforth, 1979); and 2) that the characteristics which Schachter and his colleagues thought were the root of obese overeating, were more likely the result of chronic dietary restraint in overweight individuals (Herman & Mack, 1975; Hibscher & Herman, 1977). Once it became clear that the phenomena of "external" eating -- hyper-responsiveness to stimulus qualities of food, over-dependence on external cues to ingestion, and poor awareness of interoceptive cues to satiety -- were not inherent characteristics of overweight overeaters, researchers began to elaborate on the features of dietary restraint itself.

Restraint Theory

Early in the development of the restraint concept Nisbett (1972) proposed a physiological theory to account

for the finding that some individuals could be chronically restrained in their eating, and as a result appear persistently hungry, yet be above socially-established norms for body size and adiposity. Nisbett introduced the notion of a physiological set point of weight which acted as a biologically determined setting on a homeostatic feedback loop: when body weight was below the set point, even if it was still above the socially-accepted range or normal weight, drive mechanisms were activated which caused hyper-responsiveness to food and hyperphagia, similar to the phenomena found in rats with lesions of the ventromedial hypothalamus (Nisbett & Temoshok, 1976).

Although the initial research on the set point hypothesis seemed promising (Herman & Mack, 1975; Hibscher & Herman, 1977), further investigation revealed no clear evidence of a homeostatic mechanism of body weight regulation in humans (Rodin, Slochower & Fleming, 1977), or a physiological basis for a set point of body weight (Kirtland & Gurr, 1979). The notion of dietary restraint, originally wedded to the idea of a set point of weight, was not abandoned, but reformulated along cognitive lines.

Whereas the original restraint theory proposed that restrained eaters were those who had, through chronic dieting, reduced their body weight below a physiological set point and thus were physiologically deprived, Herman and Polivy (1980) revised this account by proposing that

restrained eaters' inhibitions against eating led to a psychological sense of deprivation, which in turn led to a cognitively-mediated disinhibition effect. Restraint, then, became defined as a cognitive variable in which individuals who worried about their weight, counted calories, and attempted to resist eating fattening foods were high in restraint, and those who essentially ignored their body weight and ate whatever they pleased, were low in restraint. Further research on and theoretical elaboration of this construct led Herman and Polivy to propose the Boundary Model of body weight regulation (Herman and Polivy, 1984).

Boundary Model

According to this model, food intake is regulated by boundaries which determine the upper and lower limits of hunger and satiety. In nonrestrained eaters -- that is, in individuals who do not report concern with body weight or attempts to lose weight through restricting intake -- the decisions to eat, or stop eating, are based on the biological pressures of hunger and satiety, respectively. After a certain period of deprivation, hunger sensations are experienced and these individuals respond by seeking out and ingesting food. The amount of food eaten is determined, at least in part, by the experience of physiological satiety -- feeling "full" -- and so, the individual's intake is maintained between two physiological boundaries, hunger and

satiety. The range between these two boundaries is referred to as the region of "biological indifference."

Restrained eaters, on the other hand, attempt to ignore these biological boundaries by not eating when hungry, and by eating less than an amount necessary to attain satiety. According to this model, these individuals establish an artificial "diet boundary" as the upper limit of intake, which is below that of the biologically determined satiety boundary. The restrained eaters also exhibit a paradoxical tendency to occasionally exceed their own diet boundary by eating excessively, bypassing the satiety boundary and eating to the point of physical capacity. Herman and Polivy refered to this aspect of the boundary model as the "disinhibition hypothesis," (Herman et al, 1987).

According to Herman and Polivy (1984; Polivy & Herman, 1985), under certain conditions, the inhibition which dieters impose on their eating is released and unusually large amounts of food are ingested. A variety of conditions have been shown to act as releasors or disinhibitors of dietary restraint, including moderately large preload meals (Herman & Mack, 1975; Hibscher & Herman, 1977; Herman, Polivy & Silver, 1979), anxiety (Herman, Polivy, Lank & Heatherton, 1987), dysphoric mood (Ruderman, 1985b); and alcohol consumption (Polivy & Herman, 1976). Nondieters regulate their intake by eating less following the ingestion of a preload meal. Hibscher and Herman (1977) initially

described the behavior of dieters under these conditions as "counter-regulatory."

Based on the counter-regulatory conceptualization of ad libitum eating following a preload, dietary restraint was thought to somehow reverse the normal, regulatory effect of a preload. In normally regulated eating, larger preloads lead to smaller amounts eaten afterwards. For counter-regulatory eating, larger preloads should lead to larger amounts eaten. Therefore, if dieters' eating was truly counter-regulatory, the size of the preload should be inversely proportional to the amount eaten afterwards. As Herman, Polivy and Esses (1987) point out, this is not the case. They note that the disinhibition produced by a large preload is not proportional to the size of the preload, but only appears to be at certain, moderate preload levels. At higher levels of preload, dieters show normal regulation of intake, ie. intake inversely proportional to preload size, but to a higher intake boundary.

This research strongly supported the boundary model conceptualization of the disinhibition effect; that dieters had, in effect, two upper boundaries of intake, a lower diet boundary which was cognitively established and mediated, and a higher boundary which was physiologically determined and represented the limits of intake capacity. Polivy and Herman make it clear, however, that the disinhibition effect itself is not the result of physiological processes, but is

based on cognitive factors; the perception of having overeaten is what causes the disinhibited eating in dieters.

Cognitive Factors in Eating Regulation

Support for the view of restraint and disinhibition as cognitive factors is provided by studies which assessed the effects of subjects' beliefs about the caloric content of preloads on further eating, independent of actual variations in preload size. Spencer and Fremouw (1979) for example, presented the same 500 kcal milkshake preload to three groups of restrained and unrestrained subjects: underweight, normal weight and overweight. Half of the subjects were told that the milkshake was very high in calories, and half were told that it was low-cal, made with artificial sweetener. Following the preload, subjects were given free access to three flavors of ice cream in the guise of a taste test. They found no effect of body weight, but a significant interaction effect of instructions regarding caloric content of the preload and restraint. Restrained subjects ate significantly more of ice cream following the preload, but only when they were told it was high in calories. This research was replicated and similar results have been obtained by Woody, Constanzo, Liefer and Conger (1981).

Further support for the cognitive formulation of the disinhibition effect was provided by Ruderman (1985c) who found a significant negative correlation between dietary

restraint and rational thinking. She suggests that the disinhibition effect reflects absolute, rigid thinking in which the dieter perceives dietary restraint in an all-or-nothing fashion. Once the diet boundary has been surpassed, the irrational cognitive style of the dieter leads them to think: "Oh, what the heck, I've already blown my diet, so I might as well go all the way."

Given that cognitions about past consumption, and perceptions of current intake both appeared to affect further intake in restrained subjects, Tomarken and Kirschenbaum (1984) predicted that expectations regarding future consumption might also serve as cognitive disinhibitors. In the first of two studies, restrained and unrestrained subjects were given a standard preload (600 kcal milkshake) and then given an ad libitum taste test of three kinds of nuts. Subjects were assigned to three groups: one of which was led to expect a high calorie meal following the preload and taste test; the second was told they would receive a low calorie meal after the taste test; and a third (control) group was not told to expect a meal at all. As predicted, the subjects in the high calorie meal condition ate significantly more than did subjects in either of the two conditions. Contrary to the experimenters' predictions, however, restrained and unrestrained subjects did not differ in the amount consumed in any of the conditions.

Tomarken and Kirshenbaum felt that their unexpected findings might have reflected the use of a nonsweet taste test food (nuts) or possibly, an insufficiently stringent low-calorie manipulation (it was expected to result in restrained subjects maintaining restraint, and unrestrained subjects eating more in expectation of a small meal, but did not differentiate these groups). Consequently they performed a second experiment in which subjects were given ice cream as the taste test food (more likely to induce disinhibitory eating in restrained subjects) and included an additional very-low calorie dinner group. Despite these manipulations, once again they found little difference in the response patterns of restrained and unrestrained subjects to the expectation manipulation. In fact, the restrained subjects in this experiment did not differ across conditions at all. Only in the control group, where subjects were given the preload and then ad libitum ice cream but were not led to expect a meal afterwards, were the results consistent with previous findings: restrained subjects ate significantly more than unrestrained.

Tomarken and Kirshenbaum attempt to explain this pattern of results by suggesting, first, that there is a "palatability threshold," for restrained eaters, such that the intensity of the disinhibitory stimulus affects whether or not a given subject will exhibit counter-regulatory eating. In other words, the expectation of a low calorie

versus high calorie meal did not significantly influence eating behavior in restrained subjects because the highly palatable taste test food overwhelmed any degree of intact restraint which the low calorie meal condition might have engendered.

Second, they suggest that the dichotomy between restrained and unrestrained may be misleading, and that restraint is more likely a continuum. Notably, subjects included in their unrestrained group (based on a median split of RS scores) had a mean RS score of 10.5 and a high of 14. Clearly, it might be inappropriate to label these subjects "unrestrained," and they might be better labelled, "less restrained." Tomarken and Kirshenbaum suggest that the less restrained subjects responded to the milkshake preload by becoming disinhibited, just as the more restrained subjects did. Unfortunately, the pattern of results was not consistent with this explanation across conditions.

Overall, the results of both studies can be summarized as follows: restrained subjects ate a lot of ice cream after a milkshake preload, regardless of future meal expectations; both restrained and unrestrained subjects ate fewer nuts following a preload, when they were expecting a low calorie meal; unrestrained subjects ate less, regardless of whether it was ice cream or nuts, when they were expecting a smaller meal, while restrained subjects only ate

less in the nut condition. Basically, Tomarken and Kirshenbaum found a three-way interaction of restraint, type of ad lib food, and expectations about meal size. Their explication of this unexpected three-way interaction is, perhaps necessarily, a little convoluted.

Ruderman, Belzer and Halperin (1985) helped clear the muddy waters a bit. They found that the anticipation of a high-calorie meal alone (i.e. without a preload) could induce a disinhibition effect in restrained eaters. They told restrained and unrestrained subjects that they would be eating either a high or low calorie meal, and then allowed them ad libitum access to crackers. Cracker consumption was significantly higher only for the restrained subjects who believed that they were about to violate their restraint by having to eat a high calorie meal.

This finding confirms the hypothesis of Tomarken and Kirshenbaum that expectations regarding future consumption could affect the eating patterns of restrained eaters. It is also consistent with their "palatability threshold" hypothesis in that subjects in the low calorie meal condition were able to maintain restraint when allowed access to a low palatability taste test food (crackers).

Other researchers have also shown that the standard disinhibition effect in dieters can be suppressed by cognitive and social factors. Herman, Polivy, and Silver (1979) exposed subjects in a standard disinhibition study to

a model who maintained her restraint, despite a high-calorie preload. Restrained subjects in this condition did not show the standard disinhibition effect, but appeared to follow the example of the model and maintained their restraint. In a further study, Polivy, Herman, Younger and Erskine (1979) examined the effects of a model who exhibited the disinhibition effect on the eating behavior of restrained and unrestrained subjects. In this case, both groups showed an increase in consumption in the ad libitum condition following a preload. Clearly, the presence of others, and their behavior can influence the disinhibition effect by providing either implied censure or support for unrestrained consumption (Polivy et al, 1979).

Nonsocial influences such as self-monitoring have also been shown to have an impact on restrained eaters' responses to the preload-disinhibition procedure. Kirschenbaum and Tomarken (1982) increased the self-monitoring of restrained subjects by calling their attention to the quantity and caloric content of the ad libitum food. Under these conditions, restrained subjects showed a suppression of the disinhibition effect following a preload.

Taken together with the previous research, this finding suggests that a wide variety of situational factors can influence the disinhibition effect, and that there is likely a large cognitive component to the impact of a preload on dietary restraint. These studies suggest that restrained

eating may be altered by strictly cognitive factors, but that the effects of direct exposure to a palatable food stimulus, *along with ingestion of a preload, are perhaps* more powerful influences. Although the research reported above seems to substantially support Polivy and Herman's assertion that one of the most important factors mediating the relationship between restraint and binge eating is cognitive (Polivy & Herman, 1985), some critics contend that this position may be overstated and that other factors may significantly influence intake patterns in dieters.

Critique of the Boundary Model

Lowe (1986) questioned the validity of the assertion made by Polivy and Herman (1985) that cognitive restraint causes binge eating. His objection is essentially that if Polivy and Herman are correct that higher levels of restraint cause higher levels of binge eating, then there should be virtually no successful dieters. Lowe reported that two groups of researchers have found that individuals who succeeded in losing weight, and maintained that loss over extended periods, showed lower restraint scores and fewer cognitive symptoms of food and weight preoccupation than unsuccessful dieters. The implication is that not all dieters are equal with regard to susceptibility to disinhibitory cognitions.

Lowe also argued that Polivy and Herman may have misinterpreted some of their own findings as being based on

cognitive factors, when in fact, significant physiological influences were at work. In this regard, he noted that elevated cephalic phase reflexes (salivation, and motilin secretion) in restrained subjects responding to food stimuli, found in two studies by the Herman and Polivy group, may in fact underlie the disinhibition effect. In support of this contention, he reported a study by Rodin, Moskowitz and Bray (1976, in Lowe) in which pleasantness of food and amount eaten both increased as a result of weight loss, but only when a small amount of food was consumed prior to presentation (i.e. not large enough to constitute a preload). Lowe argued that the Polivy and Herman research has confounded the effects of cognitive restraint with those of weight loss, pointing out that their measure of restraint, the Restraint Scale (RS), has items reflecting both weight fluctuation and cognitive restraint.

Lowe's criticism of the lack of differentiation of physiological and cognitive factors in research on the boundary model is echoed by Wardle (1986) who questioned the use of the RS as a measure of restraint. Her argument is that the items on the RS, which is used by the Polivy and Herman group in virtually all of their research to identify restrained subjects, have been shown statistically to belong to two different factors: Weight Fluctation (WF) and Concern with Dieting (CD). The use of a single score to represent these factors invariably results in a confound of a tendency

toward fluctuations in weight (possibly a predisposition toward disinhibition) with the desire to lose weight. Consequently, previous research using the RS as a basis for subject categorization would inevitably have been selecting unsuccessful dieters, i.e. those more likely to show a disinhibition effect. It is implied that these two factors may operate differently in determining the effects of a preload, or other disinhibitors, on subsequent consumption.

Lowe and Kleifield (1988) presented the results of research directed at clarifying this particular issue. They measured both weight suppression (taken as: greatest weight ever - current weight/ideal weight x 100) and cognitive restraint (from the Restraint scale of the Three Factor Eating Questionnaire, Stunkard & Messick, 1985), then replicated the standard disinhibition procedure introduced by Herman and Mack (1975). In this study, amount eaten of the ad libitum food was not related to cognitive restraint. Contrary to what Lowe and Kleifield had expected, subjects who showed long term successful suppression of body weight ate less after a preload than did nonsuppressors. They argue that, although initial restriction of food intake and weight loss elevate the risk of disinhibition, over longer periods of time, some dieters are able to maintain a lower body weight through suppression of disinhibition, or maintenance of restraint.

This argument is supported by the finding of LeGoff,

Leichner and Spigelman (1988) that although both anorexia nervosa and bulimia nervosa patients had very high restraint scores, only the bulimic patients, who reported much greater fluctuations in their daily caloric intakes, showed elevated salivary responses to food stimuli. In this instance, the anorexics, who had little variability of their caloric intake, maintained a consistantly low body weight with few fluctuations and rarely experienced binge episodes. As such, they were similar to Lowe and Kleifield's successful dieters. The bulimics were more similar to unsuccessful dieters, such as the restrained subjects in Hibscher and Herman (1977) and in LeGoff and Spigelman (1987), who had fluctuations in both intake level and body weight, and showed elevated salivary responses to food, relative to nonrestrained subjects.

It would appear then that dietary restraint, as a cognitive variable, in and of itself, can not account for the diversity of responses to food stimuli and eating patterns exhibited by individuals who are high in dietary restraint (on either the RS or the Cognitive Restraint Scale of the 3-FEQ). Both in terms of the Restraint Scale itself, and the behaviors with which it is associated, restraint appears to be a multidimensional construct. This does not, strictly speaking, rule out the possibility that restraint is nonetheless primarily, if not wholly, a cognitive construct, as Herman and Polivy have claimed.

Jansen and colleagues have addressed this question directly by attempting to establish whether the cognitive concomitants of disinhibition, which Herman and Polivy (1984) have described as causal, are sufficient and/or necessary for disinhibition to occur. In one study, Jansen et al (1988) examined the cognitive and behavioral style (Irrational Beliefs Test & Rational Behavior Inventory), and reported cognitions (tape recorded self-talk, & Self-Talk Questionnaire) of restrained and unrestrained subjects within a standard disinhibition design (ie. in preload and no preload conditions, with ad lib taste test of ice cream). They found, first, that individuals who were high in restraint and exhibited disinhibited eating were not more likely than unrestrained subjects, or restrained subjects who did not show disinhibition, to report irrational cognitions or behavior. This brings into question the assumption that the underlying cause of disinhibition is a characteristic cognitive style, described by Polivy, Herman and colleagues (Polivy, Herman, Olmsted & Jazwinski, 1990; Polivy & Herman, 1985) as dichotomous thinking, or polarized cognitions.

Second, Jansen et al (1988) found that during episodes of overeating, restrained subjects did not report more thoughts related to disinhibition, such as "I've blown my diet, I might as well continue to eat," than did subjects who did not overeat. Likewise, the restrained subjects who

overate did not recall having had those sorts of thoughts during disinhibited eating more often than other subjects. The authors conclude that there was no evidence to support the widely held view that polarized cognitions were the cause of disinhibited eating in restrained subjects.

In a second study, Jansen and van den Hout (1991) examined whether simply exposing subjects to the smell of palatable food, without the usual preload, or other disinhibitor (eg. alcohol, anxiety, etc.) could induce overeating in restrained subjects. They assigned restrained and unrestrained subjects to two conditions: in one, subjects were asked to smell and rate a series of palatable snack foods, and were later told they could eat as much or as little as they liked; in the control condition, subjects did not smell the foods, but were given the ad libitum snack. They found that restrained subjects in the smell condition ate significantly more than the other groups, thus demonstrating a disinhibition effect without a preload, and, they conclude, without the concomitant cognitions.

These studies appear to contradict the prediction of the boundary model that individuals who attempt to reduce their body weight through restrained eating invariably become susceptible to situational factors which act as releasors in disinhibiting their cognitive restraint and result in disinhibitory cognitions, and consequently, over-eating. These findings suggest the involvement of

factors other than those specified in the boundary model which affect both body weight regulation, and response to external food stimuli.

Jansen and van den Hout hypothesize that disinhibited eating is not primarily determined by cognitions, but by the conditioning of a compensatory decrease in blood sugar levels; a compensatory conditioned response (CCR). They propose that the stimuli repeatedly associated with binge eating (in this case, the smell of food) come to elicit a compensatory drop in blood sugar levels which is experienced cognitively as craving:

> After conditioning, an anticipative decline in blood sugar level (CCR) may be functional: the hypoglycemia is a preparatory response in anticipation of the intake of large amounts of food, thereby compensating for the hyperglycemic effects of subsequent food intake (p. 253).

Although this physiological theory accounts for their data, the Jansen and van den Hout study suffers from design flaws which do not allow for ruling out cognitive interpretations of the same data.

Although they specifically note that subjects in the smell condition were not told that they would be allowed to eat the stimuli, they did not assess the effectiveness of this manipulation. There are sufficient demand characteristics in their procedure, such as presenting

subjects with large bowls of snack foods to smell and asking subjects not to eat for a period of time prior to coming to the lab, to suggest that subjects might have come to the conclusion that they would be permitted to eat the stimuli, regardless of whether they were explicitly told to do so or not. The presentation of actual food to the subjects could well have elicited the expectation of consumption, and thus, represents a replication of previous research which demonstrated that expectations of a high calorie meal could induce disinhibited eating (Ruderman et al, 1985; Tomarken & Kirshenbaum, 1984).

With regard to Jansen et al (1988), their failure to find overt evidence of disinhibited cognitions does not necessarily rule out the possibility that those cognitions occurred and exerted an influence on behavior. As the authors themselves note: "Better instruments may eventually show different data," (p. 398). Given the extensive empirical support for the cognitive restraint theory (Boundary Model), the negative findings of Jansen et al and Lowe do not appear to provide enough counter evidence to warrant discarding the theory. These studies nonetheless suggest the need for further examination of the factors involved in the disinhibition effect, both physiological and cognitive. As Jansen et al imply, this may necessitate the development of better methods and instruments for examining these factors.

An Alternative Model of Restraint and Disinhibition

It seems clear that a more complete account of the relationship between dietary restraint and disinhibition would need to account for the action and interaction of cognitive, physiological and behavioral factors. Both the behavior and the reported experiences of individuals who repeatedly fail in their attempts to reduce their body weights suggest a significant conflict among these factors. Simply (and anthropomorphically) put, it appears as though physiological need rebels against cognitive restraint, usurping control at some point, resulting in an incongruency between cognitive intentions (dietary restraint) and behavioral results (overeating). Ironically, it was Nisbett and his colleagues, early proponents of cognitive restraint theory, who later proposed and substantiated the view that cognitions are as likely to be the result of our behavior as the agents. It may be that disinhibitory cognitions (eg. "I've blown my diet") are the consequence of overeating rather than the cause.

This point was made by LeGoff et al (1988), who reported on the salivary responses to food of anorexic and bulimic patients. The highly variable caloric intake of bulimic patients predicted an exaggerated salivary response to food, and it was suggested that the physiological susceptibility to binge eating led to the subjective awareness of dyscontrol, and the resulting cognitions (eg.

"I can't control my eating") which may in turn exacerbate the loss of control. In anorexic patients, the opposite eating pattern, excessive control, may have enhanced subjective experience of control over eating, and cognitions associated with successful restraint (eg. "I can control my eating").

Of course, it may be argued that the salivary responses of hospitalized eating disorder patients may be affected by a number of potentially confounding factors other than degree of control over eating and the resulting variability of intake. In order to establish the primacy of cognitions or physiology in the disinhibition effect, it was first necessary to establish that cognitive restraint alone could not account for the elevation of appetite in restrained subjects. LeGoff, Cox, Beyerstein and Krane (1989) attempted to establish this by examining the daily intake patterns of normal, noneating-disordered subjects and measuring their salivary responses to food.

Their results indicated that subjects who were restrained in their eating and scored high on the RS, but ate consistently small meals, that is, did not exhibit disinhibition of restraint, salivated significantly less to food than did subjects who also scored high on the RS and restricted their intake, but showed high diet variability. This finding substantially supports the hypothesis of Jansen et al (1988) and LeGoff et al (1988) that the

hyper-responsiveness to food exhibited by restrained eaters does not result from restraint alone, but from a combination of restraint and a history of sporadic eating patterns.

The working hypothesis presented here is that the eating pattern exhibited by individuals who score high on the RS (Polivy and Herman's Restrained subjects) -- i.e. prolonged periods of reduced intake or deprivation followed by brief episodes of rapid ingestion of large amounts of food -- results in a learned over-response to the presence of food and/or the onset of ingestion, such as the CCR proposed by Jansen and van den Hout. This CCR represents a compensatory mechanism, which is adaptive and possibly of evolutionary origin, which ensures the ingestion of adequate nutrition in order to counteract the effects of deprivation. Regardless of the prevailing social pressures and the desires of the individual to be thin, the body's adaptive response to an environment in which food is only sporadically available is to slow down its energy expenditure (Klesges, Isbell & Klesges, 1992; Laessle, Tuschl, Kotthaus & Pirke, 1989), and heighten its physiological response to palatable food, thereby increasing ingestion.

The physiological component of appetite was described by Powley (1977) as a cephalic phase reflex (CPR) -- autonomic and endocrine responses to proximal sensory contact with food stimuli -- which is triggered by the

presence of palatable food, and the anticipation of ingestion, when conditions of nutritional deprivation exist. The disinhibited eating exhibited by restrained individuals may be driven by a CPR which has become exaggerated through the repeated cycle of deprivation followed by binge eating, such as was described by Jansen & van den Hout in their description of the CCR. The cognitions associated with disinhibition, regarding loss of control, relaxation of inhibitions, etc., when present, are not the cause of disinhibition, but the effects of disinhibition. Secondarily, they may become contributory factors, such as in a vicious cycle in which disinhibited eating reinforces disinhibitory cognitions, and via self-reflection and evaluation of self-control, vice versa. In this model, the disinhibitory cognitions are post-hoc reflections upon behavior which may affect self-concept but which are driven by a compensatory physiological mechanism.

The predictions of this model in terms of eating behavior in the lab or real-world settings would not differ significantly from those made by the extant boundary model. The central difference is in the primacy assigned to either a physiological or cognitive basis for the disinhibition effect. The boundary model proposes that it is the cognitions triggered by the disinhibiting stimulus (either preload, mood state, or alcohol) which result in binge-like ingestion. The model proposed here holds that it is the

exaggerated physiological response (CPR) to proximal food stimuli or ingestion of a preload (appetizer effect), which leads to disinhibited consumption. The cognitions of dyscontrol are secondary to this physiologically-driven behavioral response, and likely lead to an exacerbation of dyscontrol through low self-efficacy, predictions of failure, etc.

It should be noted that the strong distinction drawn here between physiological and cognitive factors is overstated. In fact, the distinction, in some instances, becomes quite arbitrary. Nonetheless, with regard to formulating models useful in predicting and controlling eating behavior, most researchers agree to make these distinctions. In the present instance, the distinction between cognitive and physiological might be altneratively construed as a distinction between volitional and involuntary aspects of response to food, or conscious versus nonconscious factors. The terms cognitive and physiological do carry these connotations as well, and for the time-being, there is not the technological methodology available to treat physiological and cognitive variables in the same way. The difference, then arises from the fact that cognitive variables of interest must be both volitional and conscious in order for us to measure them. Physiological variables, conveniently, do not.

In order to assess the validity of this model, and

establish the relative contribution of physiological versus cognitive factors in determining the restraint-disinhibition effect, it is necessary to first establish a methodology which will allow accurate and valid measurement of the relevant variables. The variable of cognitive restraint has already been the focus of considerable attention and seems to be well measured by the Restraint factor items of the 3-FEQ (Stunkard & Messick). The more problematic variable, from both theoretical and psychometric points of view, is the appetite response.

Defining Appetite

Part of the difficulty in establishing a valid measure of appetite is the nature of the construct itself. It has been construed of as a cognitive process (subjective ratings), as a behavior (rate and quantity of ingestion), and as a physiological response (pupil dilation, insulin secretion, salivation). Clearly, a complete description of the construct needs to address all three components. Appetite, for the purposes of the present research, is defined as a physiological state of readiness for ingestion of food, which may give rise to corresponding cognitions and is generally predicted by caloric deprivation and predictive of consumption.

It should be noted that this definition includes each of physiological state, cognitions, and behavior, but that the primary feature of the definition is physiological. In

this way, the definition both accounts for the experimental observations regarding the interrelationship among the components of appetite (see below), and provides the most logical formulation of this interrelation. The ingestion of food is neither a necessary nor sufficient definition of appetite since it is logically possible to eat without an appetite, and to not eat when peckish. Neither is the subjective experience of appetite necessary nor sufficient since people can become aware of an appetite only after they have started eating, or will subjectively feel the need to keep eating despite feeling full. Likewise, appetite is also not adequately accounted for by the variable of caloric deprivation. Although being deprived of calories may lead to an appetite, it does not always or necessarily do so, as is clear in many anorexic conditions including depression and hyperarousal.

Whereas it is neither necessary nor sufficient to account for appetite as ingestion, subjective experience, or deprivation, it is at least necessary for appetite to be a state of the organism. In order for the definition to be complete, the additional components of deprivation and ingestion need to be included, while the subjective component is clearly correlational.

The centrality of the physiological component of appetite is supported by research which shows both the desynchrony of the cognitive, physiological and behavioral

aspects, and the relative strength of the physiological component as predictive of the other aspects. Hodgson and Greene (1980) reported a desynchrony between behavioral, subjective and physiological (salivary) measures of appetite and response to food. More specifically, they noted that measures directed at eating behavior (rate of eating, and rating of pleasantness of food) were correlated with each other, but not with measures of appetite (self ratings of hunger, and saliva flow rate).

These results confirmed a previous finding by Wooley and Wooley (1973) that increases in salivary output were found in response to a palatable stimulus in subjects who had not eaten for a period of time. In contrast to the Wooley and Wooley study, Hodgson and Greene did not find a strong correlation between subjective ratings of hunger and salivation.

A number of studies have since examined the relationship between self-reports of hunger and salivary response to food. Jansen, Boon, Nauta & van den Hout (1992) found that subjective ratings of hunger were not correlated with saliva flow rate to a conditioned stimulus. In this instance, the researchers paired a red light with the presentation of chocolate candies, in order to condition the salivary response to a food stimulus. They did not find that subjective ratings corresponded to the conditioned salivary response. Other findings have been somewhat mixed

(cf. Wooley & Wooley, 1981) although, overall, they seem to indicate that salivation may be a more accurate measure of hunger than self-report since it tends to correlate better with other objective measures such as time since last meal, and rate and amount eaten following deprivation (Hodgson & Greene, 1980). Consequently, although there may be a tenuous relationship between self-reported hunger and salivation (Jansen et al, 1992) this appears to represent more of a problem for self-report measures than for salivation.

Measuring Appetite

Perhaps one of the main impediments to firmly establishing the validity of saliva flow rate to a food stimulus as a measure of appetite has been the nonstandardized and often invasive, or otherwise reactive techniques used to measure it. Wooley and Wooley (1981) reviewed twenty-eight studies which measured saliva flow rate in humans using various techniques and presented a comparison of the reliability of each of these techniques. The predominance of studies used one of two techniques: 1) dental roll absorption: either one, two or three cotton dental rolls placed in the mouth and weighed before and after collection; and 2) Lashley suction cup: a small suction disc fitted with a tube for evacuating saliva placed directly over the parotid duct inside the cheek.

Wooley and Wooley concluded that although either of

these techniques allows for a fairly reliable assessment of flow rate, salivation is sensitive to a wide range of confounding factors including mood, irrelevant cognitions, and nutritional status. The potential utility of this measure is succinctly described by Wooley and Wooley (1981):

> "...saliva flow rate, is a joint function of physiological, metabolic and nutritional status, on the one hand, and environmental, sensory and cognitive (conceptual) conditions on the other. Salivary secretions probably parallel 'more important' secretions, such as insulin and gastric acid, well enough to serve as a rough index of the magnitude of these other metabolic functions ... and could provide one reliable way of studying the disordering effects on appetite of social/familial influences, dieting and food-related beliefs (p. 346).

Despite this overall positive conclusion, a number of studies have reported negative or mixed results. As was pointed out by LeGoff and Spigelman (1987) the inconsistencies in results parallel inconsistencies in methodology.

Some methods involve reactivity, such as whole-mouth suction using a dental ejector likely to elicit anxiety-provoking associations, while others lack sensitivity and dynamic range: single dental roll placed

sublingually; or having subjects expectorate into a receptacle. Given the potential usefulness of salivation as a physiological variable, the lack of standardization and poor reliability of measures for assessing it clearly needs to be addressed.

Although fairly sensitive and reliable results have been obtained in studies in which either the Lashley suction cup or the dental roll technique have been carefully applied (Wooley & Wooley, 1973; Hodgson & Greene, 1979; Klajner et al, 1981; Nirenberg & Miller, 1982; LeGoff & Spigelman, 1987; LeGoff et al, 1988) effect sizes found have been fairly small, and the results have been somewhat inconsistent. Part of this problem likely stems from both the lack of sensitivity of the measures, and the possible reactivity due to invasive procedures.

With both techniques, objects are introduced into the mouth in proximity to if not in direct contact with the salivary ducts. A number of researchers have reported that mechanical stimulation of the ducts (eg. Emmelin & Stromblad, 1954; Young & Van Lennep, 1978), and/or the presence of a stimulating bollus in the mouth (Shannon, 1962) increases salivation rate. In addition, whereas the Lashley technique is invasive and difficult to administer, the more simple cotton swab technique lacks sensitivity and specificity (i.e. it is limited to a single measurement and cannot show variability and dynamic qualities of responses

over time).

For these reasons, an alternative method for assessing salivation rate has been sought. A prestudy to the present investigation determined that the activity of the parotid glands (the largest and most dynamic of the salivary glands) could be measured by placing recording electrodes on the skin surface over the gland. This technique, referred to here as electrosaliography (ESG), proved to be both sensitive to minute changes in parotid activity and noninvasive, as well as capable of recording dynamic changes in activity rate over time. The result of the ESG, the amount of electrophysiological activity of the gland, is referred to here as electrosalivary activity (ESA), and this variable in response to a stimulus is referred to as electrosalivary response, or ESR.

Electrophysiological Measurement of Parotid Activity

This technique involves placing a recording electrode on the skin surface directly over the parotid gland, which is located over the mandible and extends to the upper cheek. This gland introduces saliva into the oral cavity via the parotid duct. Salivary fluid and its associated proteins and acids are produced in the gland by secretory cells which are arranged in cul-de-sacs throughout the gland, and are connected by small ducts. The ducts branch into larger collections of ducts, leading eventually to the central duct (Stenson's Duct) which exits through the oral mucosa into

the oral cavity, opposite the upper molars (Young & Van Lennep, 1978). Fluid is expelled from the gland by the activity of stellate myoepithelial cells which surround the secretory cells and ducts (Garrett & Emmelin, 1979).

These tiny muscle cells contract in synchrony with the secretory cells, having the same parasympathetic ennervation (Emmelin, Garrett, & Ohlin, 1968). The joint action of the secretory and muscle cells in the gland produces a change in electrical potential over the gland, which can be recorded at the skin's surface. Relative activity rates of the gland can be distinguished on the basis of these recordings (Davis, Hing & Co, 1990).

Bauslaugh and Davis (1993a) have demonstrated reliable surface recordings of parotid activity (electrosalivary response, ESR) in humans responding to a lemon juice stimulus. They recorded ESR and saliva flow rate via a modified Lashley cup, and injected either water or lemon juice into subjects' mouths using a vacuum tube. Flow rate and wave-form values for the ESR at both resting and response rates were highly and significantly correlated (mean $r = .80$).

In a further study, Bauslaugh and Davis (1993b) examined the relationship between ESR and saliva flow rate as measured by the Strongin-Hinsie-Peck procedure (Peck, 1958), that is, the use of dental swabs to collect saliva, taking the weights of cotton dental swabs which are placed

in subjects' mouths before and after stimulus presentation. In this study, the authors again found that saliva flow rate was significantly correlated with ESR.

The electrophysiological recording of parotid activity appears to be a significant advancement in the assessment of flow rate in humans, by avoiding the shortcomings of previous techniques: invasiveness and nonresponsiveness. At this point, however, the technique has not been validated as a measure of appetite. That is, it has not been demonstrated that the ESG is sensitive to changes in flow rate which result from presentation of food stimuli to hungry subjects. Similarly, the ESG has not been tested with different subject groups, and so, its ability to distinguish restrained and unrestrained subjects' responses to food is unknown. The initial stages of the present research are a direct examination of these questions.

Goals of Research

The goals of the present research project are threefold: first, the validity of ESG as a measure of physiological appetite will be assessed; second, it will be determined whether the ESG is sensitive to differences in dietary restraint and self-ratings of appetite, which have been found with other measures of salivation; and finally, the research will assess the validity of the alternative model of the restraint-disinhibition effect, which proposes that disinhibited eating is better accounted for by a

physiological overresponsiveness to food stimuli, rather than by cognitive release of restraint.

More specifically, the research was designed to: 1) obtain an accurate measure of the degree of cephalic phase responsivity in human subjects (i.e. appetite), such that it is sensitive to differences in caloric deprivation; 2) assess whether the instrument is sensitive to individual differences in important variables such as cognitive restraint, as well as physiological deprivation; and 3) having established some validity of the methodology, to use it to assess the "appetizer effect" model of disinhibited eating.

The specific hypotheses which will be tested in order to provide evidence to support the proposed model include:

a) that in restrained subjects, ESR to food will show a significant increase as a result of ingesting a high-calorie preload;

b) that ESR to food will predict both the extent of disinhibited eating (i.e. ad lib eating after ingestion of a high calorie preload) as well as normal ingestion (i.e. no preload), better than will either cognitive restraint or subjective appetite ratings;

c) that degree of variability of intake from diet records will predict both elevated ESR to food and disinhibited eating in the laboratory.

EXPERIMENT I

This study was designed to assess the reliability and validity of the ESG as a measure of appetite. Subjects were randomly assigned to one of two groups: in one group, the subjects were asked to refrain from eating for at least four hours, while in the other group, subjects were asked to eat within half an hour of testing. In the lab, they were presented with food and nonfood odours while their parotid gland activity rates were being measured by ESG -- referred to here as electrosaliographic response, or ESR. Afterwards, the response levels to food and nonfood stimuli were compared, as were the differences between these two stimulus sets for the fasted and nonfasted groups. The hypotheses being tested were that the ESR would be higher for food than for nonfood stimuli, and that this difference would be greater in the fasted than in the nonfasted group.

Method

Subjects

Twenty subjects (15 males and 5 females) were recruited from the Simon Fraser University Psychology Department subject pool through a sign-up sheet. They received course credit for their participation. The means for age, body weight and Body Mass Index (BMI = weight in kilograms over height in meters squared) are reported in Table 1. BMI has been shown to be a good measure of degree of adiposity, and

reflects overall body proportion better than weight alone (Straw & Rogers, 1985). Subjects completed a brief screening questionnaire which asked details about weight history, history of eating disorders, and medical problems affecting the salivary glands and gastrointestinal system (see Appendix A). No subjects reported a history of significant eating or physical problems which would have required their exclusion from the study. They were assigned to the fasted or nonfasted group on the basis of a coin toss.

Table 1. Mean age, body weight and Body Mass Index (BMI) for male and female subjects.

	Males (N = 15)		Females (N = 5)	
	Mean	SD	Mean	SD
Age (years)	22.1	1.8	21.5	2.2
Body Weight (Kg)	82.0	5.9	63.8	7.5
BMI (Kg/m^2)	25.3	2.1	21.8	1.6

Instruments and Apparatus

Electrosaliogram (ESG). The electrosaliogram (ESG) was designed to monitor the activity of the parotid salivary gland by recording electrophysiological signals at the skin's surface directly over the parotid gland (i.e. placing an electrode on the cheek, over the parotid). A single silver-silver chloride electrode was placed 1 cm below the ear lobe, and 2 cm forward. A reference electrode was

placed on the ipsilateral mastoid process on the occipital bone of the skull.

The electrical potential difference between these electrodes was recorded by a Zenith personal computer, using HEM Data Corporation's "Snapshot Storage Scope with Snap Calc," software. Data was recorded in millivolts (mV) and was graphically displayed, and analysed using the HEM software. Data analyses using this measure were based on total voltage produced over time. The ESG was recorded for a total of 90 s: 20 s for each of five stimuli, with a lag time of 10 s following the initial presentation. This lag time was determined using pretrial data as the mean length of time required for the parotid gland to become activated following presentation of a food stimulus.

Respiration rate was also recorded during ESG measurement. Respiration was recorded by means of a wishbone strain gauge which was fitted to a stap around the subject's chest. The signal was fed to a strain gauge amplifier, and then to the HEM software. These recordings were made primarily to rule out spurious alterations in the parotid recording due to large potentials produced by breathing. Subjects were requested to refrain from altering their breathing suddenly, such as yawning or coughing, during the ESG recording. Interference from eye movements were eliminated by instructing subjects to close their eyes, and not move them during the stimulus presentation.

Artifactual signals arising from the motor cranial nerves which pass close to the parotid were controlled in part by asking subjects not to move their eyes, jaws or face. More specifically, the nerves most likely to affect the ESG signal are the facial nerve, which controls facial expression, the abducens, which controls eye movements, and the trigeminal nerve which controls jaw movements. Electrophysiological signals which result from the activation of these nerves show up on the ESG recording as large, short-duration spikes which are easily distinguished from the much slower changes produced by the parotid. As a second control against artifactual error, the ESG recordings were visually reviewed and any large, rapid spikes were subtracted from the recording. The mV total for the recording was then extrapolated slightly to compensate for the missing section.

Stimuli. The stimuli used were the same as those used in previous research by the authors. Two sets of stimuli were presented, food and nonfood. The food stimuli consisted of 200 ml containers with 100 ml of particalized food: potato chips, nacho-flavored corn chips, cinnamon bun, donut, and chocolate. The nonfood stimuli were: soap, pine needles, pencil shavings, tobacco and dirt. The food stimuli have been shown to produce a reliable increase in flow rate above the nonfood baseline in previous research (LeGoff & Spigelman, 1987; LeGoff et al, 1988). The order

of presentation of the two sets of stimuli (food first, then nonfood, versus nonfood then food) was alternated across subjects and was counterbalanced between groups (fasted and nonfasted). Each of the stimuli was presented for 20 s, for a total response time of 100 s per stimulus set. During this time, subjects sat with their eyes closed, and were instructed to remain completely still. There was a 5 minute delay between presentation of the first and second odour sets in order to reduce possible carry-over effects.

Design and Data Analysis

The design was a simple between-groups design, with two groups, fasted and nonfasted, and one dependent variable, ESR (electrosaliographic response) to food odours minus ESR to nonfood odours (baseline). More specifically, the area under the curve of recorded parotid activity in mV for the 90 s period of nonfood stimuli presentation was subtracted from the area under the curve for the 90 s food stimuli presentation.

Procedure

For half of the subjects, ESR to the stimuli was measured within half an hour of their having ingested their normal midday meal. For the others, their responses were measured between 4 and 5 hours after their morning meal and before lunch. All subjects were tested at approximately the same time of day (1300 hours).

Subjects were met at the lab's observation room (which

has the appearance of a small sitting room), were weighed and then seated in a large reclining chair. They completed a consent form and the screening form, and were then fitted with electrodes (see above) and asked to sit quietly, keeping extraneous bodily and eye movements to a minimum. At that time, they were instructed as follows: "You are going to smell some different odours. Try not to sniff, or change your breathing, but just pay attention to the odours and try to identify them."

Subjects in the fasted group had been instructed not to ingest anything but water (four cups maximum), between the time of their normal morning meal and the testing time. They were also requested to refrain from physical exercise and to not ingest appetite suppressants, or coffee during this period. At the lab, subjects were asked if they had complied with the fasting and abstention requirements. Nonfasting subjects were asked to come to the lab directly after eating lunch, which was to be what they would normally eat at midday.

Results

The average ESR values for food and nonfood stimuli and differences between food and nonfood, as well as length of time since last meal for all subjects as well as for fasted and nonfasted groups are presented in Table 2. Overall, ESR to the food stimuli was positively and significantly correlated with time since last meal ($r = .660$, $p < .001$).

When ESR to nonfood was used as a baseline and subtracted from food response values, the residual values (response to food above nonfood baseline) were even more strongly correlated with time since last meal ($r = .755$, $p < .0001$).

Table 2. Mean ESR for food and nonfood stimuli, food-nonfood ESR differences, and length of time since last meal for fasted and nonfasted subjects.

	All Subjects (N = 20)	Fasted (N = 10)	Nonfasted (N = 10)
Food Stimuli (mV)	X = .110 SD = .138	X = .193 SD = .141	X = .026 SD = .057
Nonfood Stimuli (mV)	X = .017 SD = .131	X = .025 SD = .151	X = .002 SD = .060
Food-Nonfood (mV)	X = .093 SD = .127	X = .168 SD = .093	X = .024 SD = .031
Hours Since Last Meal	X = 2.37 SD = 1.92	X = 4.16 SD = 0.60	X = 0.52 SD = 0.22

A t-test of ESR to food stimuli and nonfood stimuli indicated that ESR to food was significantly greater than that for nonfood stimuli for all subjects ($t = 4.43$, $p < .0001$). A second t-test revealed that the fasted group had

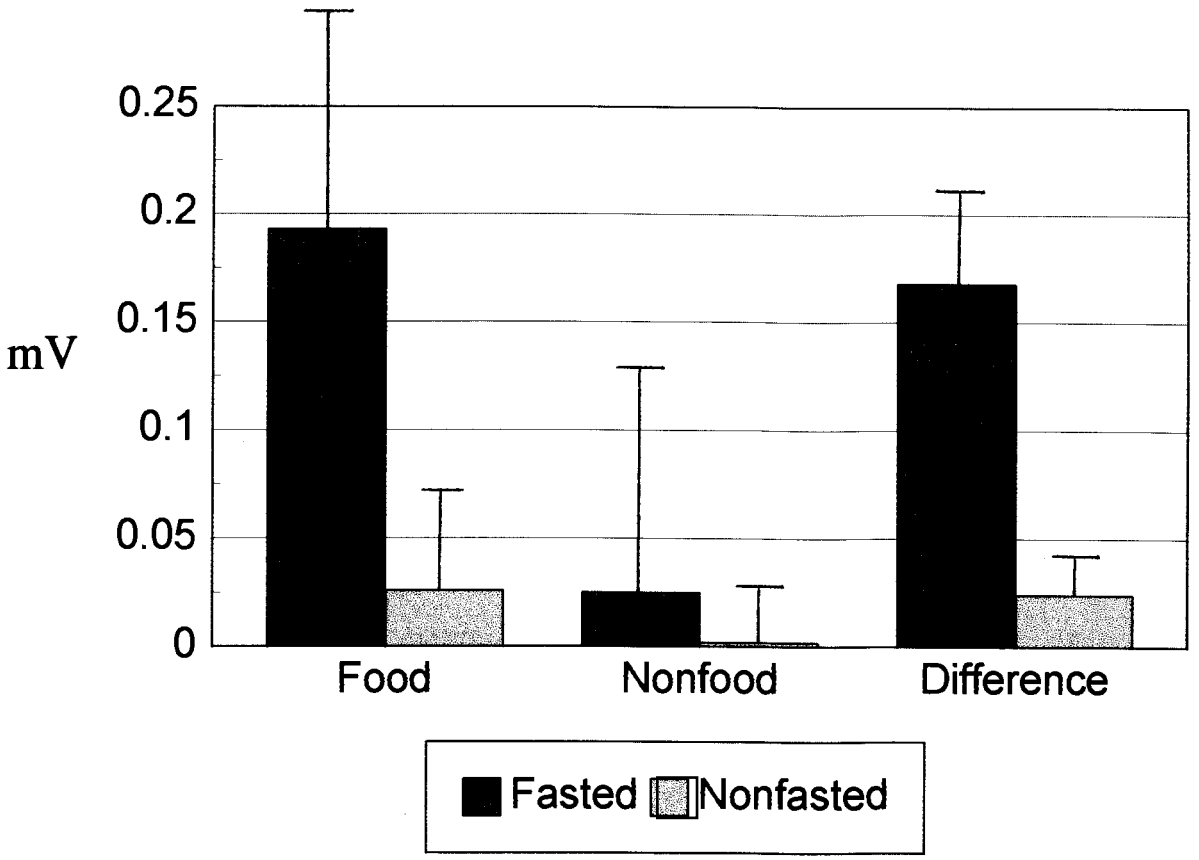
significantly higher ESR to food stimuli, than did the nonfasted group ($t = 2.92$, $p < .01$). The response values for nonfood stimuli were not significantly different across groups ($t = 1.69$, $p > .10$). ESR to food above nonfood baseline (food-nonfood ESR difference) was significantly higher in the fasted group ($t = 3.41$, $p < .005$). The ESR to food, nonfood and differences from baseline for the fasted and nonfasted groups are presented in Figure 1.

Control Measures

Tests of differences between the fasted and nonfasted groups revealed no significant differences on age or body weight ($t < 1$). Although there was a small, nonsignificant correlation ($r = .210$, $p > .10$) between hours of fasting and ESR to nonfood stimuli (baseline), there was no significant difference between the fasted and nonfasted groups on this variable. The correlation between responses to food and nonfood stimuli for all subjects was also nonsignificant ($r = .214$, $p > .10$).

The signal produced by the respiratory transducer was analyzed and a multiple regression analysis was used to assess the relationship between breathing rate and ESR to food and nonfood stimuli in the fasted and nonfasted groups. There were no significant correlations between breathing rate and ESR ($p > .10$, for all correlations). In all cases, respiration accounted for less than 10% of the variance in ESR values.

Figure 1. ESR to food, nonfood, and food - nonfood differences for fasted (n = 10) and nonfasted (n = 10) subjects.



Discussion

As can be seen in Figure 1, the electrophysiological recording of parotid gland activity in fasted and nonfasted subjects in response to food and nonfood stimuli provided two clear findings: 1) ESR to a food stimulus is considerably higher than to a nonfood stimulus, regardless of fasting status; and 2) fasted subjects showed considerably higher ESR to the food stimuli than did nonfasted subjects. This latter effect was not explained by generally higher ESR levels in the fasted subjects, since they did not respond significantly more to nonfood than did the nonfasted subjects. As well, the correlation between fasting time and ESR to nonfood was not significant.

Previous research has not shown such clear effects in demonstrating a relationship between salivary response and either food stimuli or time since last meal. It seems likely that the effects found in the present research reflect changes in methodology, both in terms of the presentation of stimuli, and in the recording of salivary gland activity. Nonetheless, a number of researchers in the past have found similar findings to those presented here using different methods of saliva collection.

Wooley and Wooley (1973) as well as Wooley, Wooley and Dunham (1976) found increases in flow rate using three cotton dental swabs (Strongin-Hinsie-Peck procedure, SHP Test) as a function of duration of food deprivation. Similar results

were reported by Hodgson and Greene (1980). In each of these studies, the food stimuli used were either imaginal (thinking about food) or questionably salient. In the Hodgson and Greene study, subjects looked at and were encouraged to think about eating six chocolate candies.

A negative finding was reported by Brummer and Pudel (1981) in which they found little relationship between saliva flow rate and fasting time. Although these researchers used a highly salient stimulus (pizza), their saliva collection procedures may have been flawed. They used a single sublingual dental roll, which would minimize the amount of saliva collected, especially from the parotid duct as opposed to the sublingual ducts, and thus reduce the chances of finding differences in flow rate. This is particularly significant since the parotid is the largest source of saliva during mastication (Emmelin & Stromblad, 1954).

Klajner, Herman, Polivy and Chhabra (1981) as well as Nirenberg and Miller (1982) reported significant increases in saliva flow rate to palatable food stimuli using whole-mouth suction and the SHP test, respectively. Although there were other factors affecting salivary response to food (dieting history, anxiety, expectations; cf. LeGoff & Spigelman, 1987), their findings generally support those reported here. The larger effect size found here likely reflects the increased sensitivity and reduced error variance of the methodology.

Bauslaugh and Davis (1993a & b) found saliva flow rate

measured directly using a Lashley-cup collector or cotton-swabs correlated with ESR, although in the case of the cotton-swab method, the correlation was only moderate. They found that salivary secretion was increased by the presence of cotton swabs in the mouth, suggesting that the SHP procedure may be a reactive measure which distorts the baseline salivation levels by stimulating flow rate.

The current data, along with those reported by Bauslaugh and Davis, provide evidence that the surface-electrode method provides: 1) a reliable and valid measure of parotid gland activity; 2) a sensitive measure of a physiological response to palatable food stimuli; and 3) a measure sensitive to changes in degree of food deprivation. Having established this preliminary level of validity for the instrument, the next stage of the research was undertaken in which the sensitivity of the ESG to levels of two cognitive variables, self-ratings of appetite and dietary restrained, was examined.

EXPERIMENT II

In this second experiment, the main hypotheses were that ESR to food would be directly related to self-ratings of appetite and to degree of dietary restraint. It was not expected that these would be strong relationships since previous research has not shown high correlations between cognitive variables and other measures of appetite (Jansen et al, 1988). Nonetheless, there has been evidence of a moderate level of relationship between dietary restraint and salivation to food (LeGoff & Spigelman, 1987) as well as subjective hunger ratings and salivary response to food (Wooley et al, 1977). The question addressed here is whether the ESG methodology is sensitive enough to detect differences in cognition which have putative effects on the appetite response to food. This question was addressed by examining the ESR to food and nonfood for a number of subjects, and then correlating that response level with measures of subjective appetite and dietary restraint.

Method

Subjects

The data for this phase of the research also was collected using subjects recruited from the Simon Fraser University Psychology Department subject pool. In total, there were thirty female subjects ranging in age from 19 to 48, who responded to a sign-up sheet asking for female subjects who had concerns with body weight and dieting and

who were currently on a weight-loss diet. The means for age, body weight, and BMI are reported in Table 3.

The physical, questionnaire, and ESG data for this study were collected at one testing time for each subject. The subjects were screened for both current and past eating disorders and obesity by having them complete a brief questionnaire which asked about incidence of eating disorders, weight history, and digestive system illnesses. Subjects were also weighed and their heights were measured at the time of testing and BMI was calculated. No subjects reported a history of or current eating disorder, and no subjects were currently obese.

Table 3. Mean age, body weight and Body Mass Index (BMI) for thirty female subjects

	Mean	SD	Range
Age (years)	24.2	5.9	19 - 48
Body Weight (Kg)	59.8	11.7	45 - 81
BMI (Kg/m^2)	22.01	3.15	18 - 30

Instruments and Apparatus

The equipment (ESG) described for Experiment I as well as the procedure for measuring electrical signals from the parotid gland in response to food and nonfood olfactory stimuli (ESR) were used in this experiment without modification. The olfactory stimuli were also replicated exactly.

The subjects completed a ten-item self-report scale

that was designed to assess degree of concern with dieting and body weight, the Restraint Scale from the Dutch Eating Behavior Questionnaire (DEBQ-R; Van Strien, Frijters, Bergers & Defares, 1986; See Appendix B). The ten items ask questions about eating habits and behaviors and are each scored according to a five-point format: never (1); seldom (2); sometimes (3); often (4) and very often (5). The total for the scale is divided by 10 resulting in an average response value with a range of 1 to 5.

In the standardization sample (N = 1169), the overall mean was 2.21, with a standard deviation of 0.92, a range of 1 to 5, and a standard error of .03. The mean for nonobese females (n = 642) was 2.49, SD = .93, range = 1 - 5, and se = .04. The internal consistency (Cronbach's alpha = .95) and factorial validity of this instrument was established in a large factor-analytic study (N = 978). Two further studies have shown the validity of the DEBQ-R with regard to self-reported caloric intake (Van Strien, Frijters, Staveren, Defares & Deurenberg, 1986; Wardle & Beales, 1987). Laessle, Tuschl, Kotthaus & Pirke (1989) examined the validity of three measures of dietary restraint and concluded that the DEBQ-R was a valid measure of both the intention to diet (cognitive restraint) and successful dieting.

Although the Herman and Mack (1975) Restraint Scale has been used extensively for the purpose of quantifying degree

of concern over dieting and body weight (Heatherton, Herman, Polivy, King & McGree, 1988), the overall scale has been criticized on psychometric grounds for producing a single score which potentially confounds two factors, dietary restraint and weight fluctuation, (Stunkard & Messick, 1985; Wardle, 1986). In the current study, our interest in cognitive restraint as a variable independent of weight fluctuation, warranted the use of the DEBQ-R as opposed to the RS. The fact that our subject selection process was the same as that used in the Polivy and Herman studies (recruiting female university students who report concerns with body weight and are currently attempting to lose weight through restrained eating), suggests that our samples should be comparable on relevant variables.

Subjects were also asked to complete a rating scale which asked: "How hungry are you feeling right now?" on a scale from 1, labelled "not at all," to 10, "very hungry." They were instructed to rate how hungry they felt at the time they were making the rating, which was directly after smelling the food odours. This simple procedure for measuring subjective appetite has been used in a number of recent studies (eg. Mattes, 1990; Jansen et al, 1992).

Design and Data Analysis

This study involved determining whether salivary response to food as measured by the ESG (refered to here as electrosaliographic response, or ESR) was sensitive to

differences in two cognitive variables, concern with dieting and body weight (cognitive dietary restraint) and subjective appetite ratings. In order to account for nonrandom variance in salivation due to age and body weight (the most likely putative covariates), these variables were entered into a regression analysis on ESR and the variance accounted for by these two variables was removed from the ESR and residual values of ESR were generated. The residual ESR values were then used as a criterion variable in each of two regression analyses with the cognitive variables, DEBQ-R and subjective hunger as predictor variables.

Procedure

Thirty female subjects were scheduled to attend individual testing sessions in the lab four hours after the ingestion of their usual morning or midday meal. They were weighed, and then seated in a large reclining chair, in a quiet and dimly lit room. They completed the eating disorder screening questionnaire and the DEBQ-R and were then fitted with recording electrodes, exactly as specified in the ESG recording procedure for Experiment I (refer to Experiment I Procedure section). They were presented with either the food or nonfood stimulus set, and asked to concentrate on identifying the odours. After a five minute break, the second stimulus set was presented. The order of presentation of stimulus sets was counterbalanced across groups. Following the presentation of both sets of stimuli,

subjects were asked to complete the appetite rating scale.

Results

The residualized ESR values for responses to food and nonfood stimuli, as well as the difference scores, the DEBQ-R scores, and the appetite ratings, are presented in Table 4. The results of the regression analyses revealed that DEBQ-R scores reliably predicted ESR values, $r = .583$, $p < .01$, accounting for 34.0% of the variance. Appetite ratings did not reliably predict ESR difference scores, $r = .202$, N.S. The variance in ESR difference scores accounted for by age ($r = .067$) and body weight ($r = .084$) were both nonsignificant and did not make a large difference in the ESR values.

Table 4. Mean ESR values for food, nonfood, and food-nonfood differences, mean DEBQ-R scores & appetite ratings (N = 30)

	Mean	SD	Range
ESR (mV) Nonfood	.022	.163	-.320 - .421
ESR (mV) Food	.276	.302	-.247 - .795
ESR (mV) Diff.	.254	.278	-.138 - .958
DEBQ-R Scores	2.77	.85	1 - 4.2
Appetite Ratings	5.20	1.54	2 - 8

Discussion

The findings reported above are mixed with regard to the sensitivity of ESR differences due to cognitive variables: there is a surprisingly strong relationship

between ESR and DEBQ-R, while there was no reliable relationship between ESR and subjective appetite. The negative finding with regard to the relationship between subjective appetite and ESR may not reflect a deficiency in the sensitivity of the measures, but may be an accurate reflection of the lack of relationship between physiological and subjective components of appetite.

As was noted earlier, self-report measures do not correlate well with other objective measures of appetite such as time since last meal (Wooley & Wooley, 1973) or rate and amount eaten (Hodgson & Greene, 1980). De Castro and Elmore (1988) recorded in vivo subjective hunger ratings and actual ingestion over a seven-day period and found significant, although small, correlations (0.15 to 0.27) depending on how the meals were defined. Mattes (1990) also found no consistent correlation between hunger ratings and actual intake in an in vivo study, suggesting that, for at least the behavioral results of hunger -- ingestion -- subjective experience of hunger is not a causal or even predictive factor. In that study, the average correlation between rating of appetite and ingestion within an hour of rating was 0.16 (N.S.). These findings are reasonably consistent with the result of the current study, although the criterion variables (salivary response versus in vivo ingestion) were different.

The strong relationship between DEBQ-R and ESR to food

confirms and clarifies previous findings (Klajner et al, 1981; LeGoff & Spigelman, 1987) in which elevated salivation to food was found in subjects who scored high on the Restraint Scale. The present finding extends these previous data in that the relationship between the cognitive variable, restraint, and the physiological variable, salivary response to food, is determined to be continuous, and not simply a dichotomous distinction between "dieters," (defined as subjects scoring above the median on the RS) and "nondieters" supporting the finding by Stein (1988) that restraint is a linear, continuous variable.

Further, the finding clarifies that the Restraint Factor of the DEBQ is sufficient to account for elevated salivary responses, independent of the Weight Fluctuation (WF) items on the RS. That is, cognitive restraint alone predicts heightened physiological responsiveness to food, independent of a history of fluctuations in weight. It has been previously argued (eg. Wooley et al, 1981) that the elevated salivary responses of restrained subjects may have reflected high scores on the WF items, and not the CD items, on the RS. In other words, elevated salivary responses to food are associated with the physical variable of weight fluctations, independent of the cognitive variable, concern with dieting. The current finding supports the view of Polivy and Herman that heightened responsivity to food reflects the cognitive set associated dietary restraint.

EXPERIMENT III

This final experiment was designed to extend the previous two sets of findings by applying the ESG methodology to the proposed model of disinhibition in which physiological factors play an important role in the disinhibition effect. The Herman and Polivy Boundary Model proposes that disinbition is the result of specific cognitions ("I've blown my diet I might as well keep eating,") and does not include physiological factors. The model proposed here suggests that an exaggerated CPR to food stimuli results in a stronger drive to ingestion, and therefore disinhibition should be predicted by elevated physiological response to food, in this case, secretion of saliva. This model was assessed by testing a number of hypotheses which are derived from and would be consistent with this model, and not predicted by the Boundary Model. To reiterate these hypotheses, they are as follows:

a) that in self-reported dieters, ESR to food will show a significant increase as a result of ingesting a high-calorie preload;

b) that ESR to food will predict both disinhibited eating (i.e. eating after ingesting a high calorie preload) as well as normal eating (i.e. no preload), better than will cognitive restraint or subjective ratings of appetite;

c) that degree of variability of intake on diet records will predict both elevated ESR to food and disinhibited

eating in the laboratory.

Variability of intake was used in order to assess the degree to which subjects normally ate inconsistent meal sizes. This measure was designed to evaluate the extent of disinhibited-style eating, i.e. periods of time with low calorie intake, followed by periods of time with high calorie intake. This measure reflects relatively unsystematic eating since it is averaged over all four time periods, so that subjects high in variability will tend to eat varying amounts of food throughout the day and across days.

It should be noted, as was pointed out by Polivy and Herman (1987; 1985), that although dietary restraint tends to lead to disinhibited eating, the increased intake exhibited by subjects in their research do not constitute "binges" and therefore extrapolating to clinical manifestations of disinhibition should be made very cautiously. The current research specifically excluded subjects who had a history of eating disorders, and so, the distinction between clinical binge-eating (which occurs within a matrix of other clinical features in bulimia nervosa, cf. Polivy & Herman, 1987) and disinhibited eating is maintained here.

The hypotheses were tested by analysing two data sets: one was comprised of eating diaries which subjects recorded for a week; and the second was laboratory data, including

both ESR and amount of food consumed in a disinhibition protocol. The procedure used in previous research for examining disinhibited ingestion was followed carefully here, including a 500 kcal milkshake preload and ad lib pizza as a test meal. The important change in the methodology in this experiment was the use of the ESG at two points in the protocol in order to assess physiological responsivity to food stimuli, once before and once after the preload. Subjects were also asked to rate their subjective experience of hunger at these times, so that cognitive awareness of hunger, physiological responsivity, and then behavioral response (ingestion) could all be quantified and compared.

Method

Subjects

Twenty female subjects from the undergraduate research pool in the Psychology Department at Simon Fraser University participated in the present study. They were recruited through the use of a sign-up sheet which was posted on the research subject pool bulletin board which provided a brief description of the study and asked for subjects who had "concerns with body weight" and were currently attempting to lose weight through dietary restriction. Subjects were contacted by telephone and individual meetings were scheduled with them in order to give them diet recording sheets and instructions on completing these diet records.

Subjects were then randomly assigned to one of two groups, preload or no preload, according to a coin toss. The subjects were scheduled for testing individually and all subjects received the same information regarding the testing procedures. The mean, standard deviation and range of age, weight, and BMI for the two groups of subjects are presented in Table 5.

Table 5. Mean age, body weight, and Body Mass Index (BMI) for preload (n = 10) and no-preload (n = 10) groups

		Mean	SD	Range
Age (years)	Preload	26.9	8.7	21 - 48
	No preload	22.2	3.9	19 - 36
Weight (Kg)	Preload	61.3	9.0	80.7 - 49.9
	No Preload	55.5	7.7	73.9 - 45.4
BMI (Kg/m^2)	Preload	22.13	3.40	18.9 - 29.5
	No Preload	20.84	2.87	18.4 - 27.8

Instruments and Apparatus

Diet Records. Subjects were asked to record their dietary intake for one week prior to the testing session. Seven recording sheets were provided for this purpose; one record sheet per day (see Appendix C). Subjects were instructed to record the type and quantity of all food and drink, including alcohol, which they consumed during the day. They were told to record their intake at the end of each day, and to record for seven days continuously, including one weekend. Subjects were scheduled to come to

the lab for testing after completion of the eating diary, and the records were collected at that time.

Electrosaliogram. The ESG equipment used in this experiment was identical to that used in both Experiments I and II. No modifications of the recording of parotid gland activity were made, except that respiratory rates were no longer recorded. It was found in the previous experiments that respiration rate did not influence the ESG data and could be ignored. Unusual respirations, body, head or jaw movements (eg. yawning and stretching) did produce a clear change in the recorded signal. These were discouraged at the beginning of each ESR trial, and if they occured, the trial was repeated after a fifteen minute break.

Stimuli. In this study, subjects were presented with the same two sets of olfactory stimuli, food and nonfood, as those used in Experiments I and II. In this experiment, however, subjects' ESR was measured twice: before and after consumption of the preload for subjects in the preload group, and before and after a fifteen minute break for subjects in the no-prelaod group. The timing and order of presentation of stimulus sets was identical to the two previous experiments, except that the recordings were made twice for each subject. Order of presentation of stimulus sets (food-nonfood vs. nonfood-food) was counterbalanced both between and within subjects.

Experiment Food. Subjects assigned to the preload

group were presented with a chocolate milkshake and told to finish it all. The contents of the milkshake were described completely, but the purpose of the milkshake was left vague. Since all subjects had fasted for a few hours prior to coming to the lab, there was no objection to this procedure. Since the goal of this experiment was to examine the effect of a preload on physiological and cognitive aspects of appetite, it was critical to replicate as closely as possible the preload conditions of those studies showing the disinhibition effect (Herman & Mack, 1975; Hibscher & Herman, 1977; Herman, Polivy & Silver, 1979; Herman, Polivy & Esses, 1987; Herman, Polivy, Lank & Heatherton, 1987; Polivy, Heatherton & Herman, 1988; Heatherton, Polivy & Herman, 1989).

Herman & Mack (1975) used a small and a large preload condition, with the large preload effectively producing the disinhibition effect. In this condition, subjects consumed two 7.5 oz milkshakes, one vanilla and one chocolate. Hibscher & Herman (1977), Herman et al (1979), Herman et al (1987), Polivy et al (1988), and Heatherton et al (1989) all used a single 15 oz chocolate milkshake. Although other researchers have used slightly different preloads (eg. Ogden & Wardle, 1990) or preload sizes (eg. Herman et al, 1977; Herman et al, 1987), the standard preload in the bulk of Herman and Polivy's research appears to be one 15 oz chocolate milkshake.

Herman et al (1987) describe the preload as "a 15-oz (600 kcal) chocolate milkshake made by mixing 300 g of chocolate ice cream in a blender with sufficient milk to produce a 15 oz preload," (p. 165). They also comment that the brand of ice cream used in the ad lib condition (which we may assume was also used for the preload) was Baskin-Robbins. These instructions and ingredients were replicated exactly in producing the preload.

Although the most common ad lib food in disinhibition studies has been ice cream, the present study used pizza because of its greater stimulus qualities, olfactory and visual, its variation from the preload stimulus, and because it was consistant with the university lunch-time diet. Pizza has been used in previous research on salivary response to food stimuli in which pre-studies were conducted to determine the most palatable stimulus (Klajner, Herman, Polivy & Chhabra, 1981; Wooley, Wooley & Williams, 1978).

The pizza for the study was acquired from the Simon Fraser University Pub, and each subject was presented with a freshly baked, whole large pizza. Each subject was given the choice of four pizza types at the time of initial contact: ham and pineapple, olive and feta, vegetarian, and pepperoni and mushroom. The contents of each type of pizza were analyzed for caloric content (kcal/g) using Food Processor II software. The weight of pizza consumed by each subject was multiplied by the caloric content to give the

total number of calories ingested. The pizzas were baked fresh at the SFU Pub and were brought directly from the pizza oven to the laboratory. The pizzas were weighed, and then presented to subjects, along with a bottle of mineral water, utensils and napkins. They were told to eat as much of the pizza as they wanted, and were left alone in the lab room to eat. When they were finished eating, the remaining pizza was weighed.

Measures. The subjects in this experiment were asked to complete a body weight and eating disorder screening form (Appendix A), the DEBQ-R (Appendix B), and a standard rating scale for subjective appetite (described in Experiment II). Subjects completed the screening form and DEBQ-R at the time of initial contact. The subjective rating scale was completed twice during the disinhibition procedure, once before the preload or 15 minute wait, and once afterwards, before the ad lib pizza.

Design and Data Analysis

Eating Records Data. The eating records were divided into four time periods, morning, midday, afternoon and evening, in order to cover the time periods in which food is normally eaten -- that is, roughly breakfast, lunch, dinner and evening snack. The number of calories consumed within each of these time periods was then determined using the Food Processor II dietary analysis software package. The calorie totals for each of these time periods over seven

days was entered into a standard deviation equation, giving a variability score for each of the four time periods. These four variability scores were then averaged, giving the average variability of intake for the week.

ESR Data. The responses to the food and nonfood stimuli which were recorded using HEM Snapshot, were analysed here just as they were in Experiments I and II: the values were calculated based on average voltage over time, beginning 10 seconds after the presentation of the first of the five stimuli for a total recording time of 90 seconds. ESR was calculated as the average voltage over time expressed in mV, for 90 seconds, for each of the two stimulus sets.

Ad Lib Ingestion Data. Amount eaten of the ad lib food (pizza) was determined by weighing the pizza before and after the subject had eaten and taking the difference between these two weights (weigh scale accurate to .01 g). Simply using the weights of the pizza consumed was not possible since subjects were offered a choice of four types of pizza (to accomodate individual diets and tastes). The caloric content of the amount eaten was determined by multiplying the weight of pizza eaten by the caloric content of the specific type of pizza: 1) ham and pineapple = 2.51 kcal/g; 2) olive and feta = 2.42 kcal/g; 3) pepparoni and mushroom = 2.60 kcal/g; 4) vegetarian = 2.38 kcal/g.

Manipulation Check and Control Data. To determine

whether the preload manipulation had produced the desired effect on ad lib eating (i.e. increased consumption in the preload group), the amount of pizza eaten in the two conditions were compared using a t-test. In order to assess the putative effects of pre-existing differences in body weight, age, dietary restraint (DEBQ-R), and diet variability between the two groups, these data were also analyzed using t-tests. Further validity checks included t-tests on ESR to food and appetite ratings before the preload, since responsivity to food and appetite were expected to be equivalent between the two groups prior to the preload.

Hypothesis Testing. Hypotheses A, B and C were tested as follows: To determine whether ESR to food showed a significant increase as a result of ingesting a preload, a mixed design was used, in which there was one between subjects variable, preload versus no-preload, and one within subjects variable, first and second presentation of the stimuli. There were two dependent measures in this phase of testing, ESR to food and appetite ratings. These were entered into two separate 2 x 2 ANOVAs.

Hypothesis B was tested by comparing two sets of correlated correlations using the Williams-Hotelling method. First, two sets of correlations were generated for ESR to food, DEBQ-R scores, appetite ratings and ad lib ingestion, for the preload condition alone, and then for the no-preload

group. Then, the correlation between ESR to food and ingestion was compared with that for DEBQ-R and appetite ratings using the Williams-Hotelling statistic for both the preload and no-preload conditions. The Bonferroni correction was used to control for the inflation of the type I error rate due to multiple comparisons.

Finally, hypothesis C was tested using correlational analyses (Pearson's Correlational Coefficients) in which average variability of intake from diet records was correlated with ESR to food and ad lib ingestion after a preload. In hypotheses B and C, comparison of correlations for the purpose of testing whether one correlation was significantly different from another was accomplished using the Williams-Hotelling Test for the difference between correlated correlations.

Procedure

Subjects were contacted and asked to come to an initial individual meeting during which they completed the screening form and DEBQ-R, and were given copies of the eating record sheets and instructed on how to complete them. Laboratory appointment times were scheduled for approximately 7 days later, at either 1145 or 1730. Subjects were requested to eat their normal breakfast or midday meal (depending on the time of testing) exactly 4 hours prior to arriving for the testing session. They were informed that they would be served food during the testing session, and that it was

important not to eat during the four hours prior to testing. Eating records were collected at that time and subjects were weighed and their heights were measured.

Subjects were informed that the research was an investigation of physiological responses to food stimuli before and after eating. After this, the procedure used in Experiments I and II was followed exactly in terms of ESG and stimulus presentation for the first recording: the electrodes were attached, subjects were asked to close their eyes, keep their breathing regular and even, focus on identifying the odours presented, and remain still.

Following the first presentation of food and nonfood odours and ESG recording, subjects in the preload condition were presented with a 15 oz chocolate milkshake, which they were asked to consume within the next 15 minutes. Subjects in the no-preload condition were simply asked to wait for a 15 minute period. The electrodes were left in place during this time.

All subjects were then asked to close their eyes, remain still, breath normally, and were presented with the two stimulus sets a second time, with the order of stimulus sets reversed from the first order. Finally, the electrodes were removed, and subjects were presented with a large pizza of a type previously chosen, as well as a bottle of mineral water, utensils and napkins. The pizza was brought to the lab from the Pub by one of the experimenters so that it was

still hot. Subjects were told that they could eat as much of it as they wished.

The experimenter excused himself at this point, explaining that he had work to do elsewhere, and would return shortly. Subjects were left alone for a period of half an hour. At that time, subjects were allowed to finish eating if they had not already. They were debriefed, and the remaining pizza was weighed to determine amount eaten.

Results

Means for the DEBQ-R, variability of intake from diet records, and amount of ad lib pizza eaten (weight multiplied by caloric content, in kcal), for the preload and no-preload groups are reported in Table 6.

Table 6. Mean DEBQ-R scores, variability of intake, and ad lib ingestion for preload (n = 10) and no-preload (n = 10) groups.

		Mean	SD	Range
DEBQ-R score	Preload	2.81	.76	1.8 - 4.1
	No-preload	2.78	.30	1.9 - 4.3
Variability of Intake	Preload	223.1	129.6	61.2 - 462.8
	No-Preload	209.6	70.2	103.3 - 317.7
Ad lib Pizza (kcal)	Preload	792.0	264.1	459.8 - 1366.1
	No-preload	544.5	135.1	302.5 - 1156.8

A t-test on amount of ad lib pizza consumed by the preload and no-preload groups revealed a significant difference between groups ($t(18) = 2.64$, $p < .02$). The results of t-tests on subject variables performed in order to rule out confounds are reported in Table 7. None of these subject variables were significantly different between groups, although age and body weight were different enough to warrant concern (i.e. probability values less than .25). Effect size analysis (Cohen's *d*), revealed that weight ($d = .69$) and age ($d = .70$) had large effect sizes. The effect sizes of DEBQ-R and diet variability were -.49 and .13 respectively, and were not large enough to warrant concern over potential confound.

Table 7. Results of t-tests of preload and no-preload group differences on body weight, age, DEBQ-R scores, and diet variability.

	t-value	DF	2-tail prob.
Body weight (Kg)	1.54	18	.14
Age (years)	1.57	18	.14
DEBQ-R	-1.09	18	.29
Diet Variability	0.29	18	.78

Consequently, between groups comparisons on dependent measures which could be affected by age and body weight (amount of food eaten, ESR) required that the variance accounted for in the dependent measures by age and body

weight be partialled out and residual values generated for further analyses.

Means for subjective appetite ratings and ESR to food minus nonfood before and after the preload manipulation for preload and no-preload groups are presented in Table 8. The ESR values (food-nonfood) prior to the preload were not significantly different between groups ($t(18) = -.45$, $p > .65$), and neither were appetite ratings ($t(18) = .70$, $p > .50$).

Table 8. Mean appetite ratings and ESR to food minus nonfood before and after preload for preload (n = 10) and no preload (n = 10) groups.

Appetite Ratings		Mean	SD
Preload	Before	5.0	0.8
	After	5.9	1.1
	Difference	+ 0.9	0.3
No-preload	Before	4.8	0.4
	After	4.9	0.7
	Difference	+ 0.1	0.1
ESR to Food (mV/s)		**Mean**	**SD**
Preload	Before	.406	.232
	After	.417	.248
	Difference	+ .011	.060
No-preload	Before	.454	.244
	After	.266	.299
	Difference	- .188	.101

Hypothesis Testing

Hypothesis A. The results of a 2 x 2 ANOVA on ESR to food, with preload versus no-preload, and before versus after preload as independent variables, indicated a significant interaction effect (Table 9). Neither of the main effects, preload versus no preload, and before versus after preload, was significant. An examination of cell means (Table 8) revealed that the interaction effect

Table 9. Analysis of variance on ESR to food

Source	Sum of Squares	DF	F	p
Main Effects	1466.98	2	1.33	.28
Preload vs No-Preload	892.27	1	1.62	.21
Before vs After	574.72	1	1.04	.31
Interaction	2024.64	1	3.70	.05

reflected the fact that subjects who did not receive a preload showed a sharp decrease in ESR to the second presentation of the food stimuli, while the subjects in the preload group showed a small increase in ESR to the second set of stimuli (see Figure 2).

A second 2 x 2 ANOVA with the same independent variables but with subjective appetite ratings as the dependent variable revealed no significant main or interaction effects (Table 10). This result indicates that the appetite ratings were not different between groups and were not affected by the preload or by repetition of the

stimuli and ratings (see figure 3).

Table 10. Analysis of variance on appetite ratings

Source	Sum of Squares	DF	F	p
Main Effects	6.100	2	1.91	.16
Preload vs No-Preload	3.600	1	2.26	.14
Before vs After	2.500	1	1.57	.22
Interaction	1.600	1	1.00	.32

Hypothesis B. In order to test Hypothesis B, two sets of Williams-Hotelling comparisons of correlated correlations were made. The first set involved comparing the predictive abilities of the ESR, DEBQ-R and appetite ratings with respect to amount of food ingested after a preload. In the second, these same correlations were compared for all subjects (i.e. combined across conditions). The correlations are presented in Table 11.

Figure 2. ESR to food - nonfood, first and second presentations of stimuli, for preload (n = 10) and no-preload (n = 10) groups.

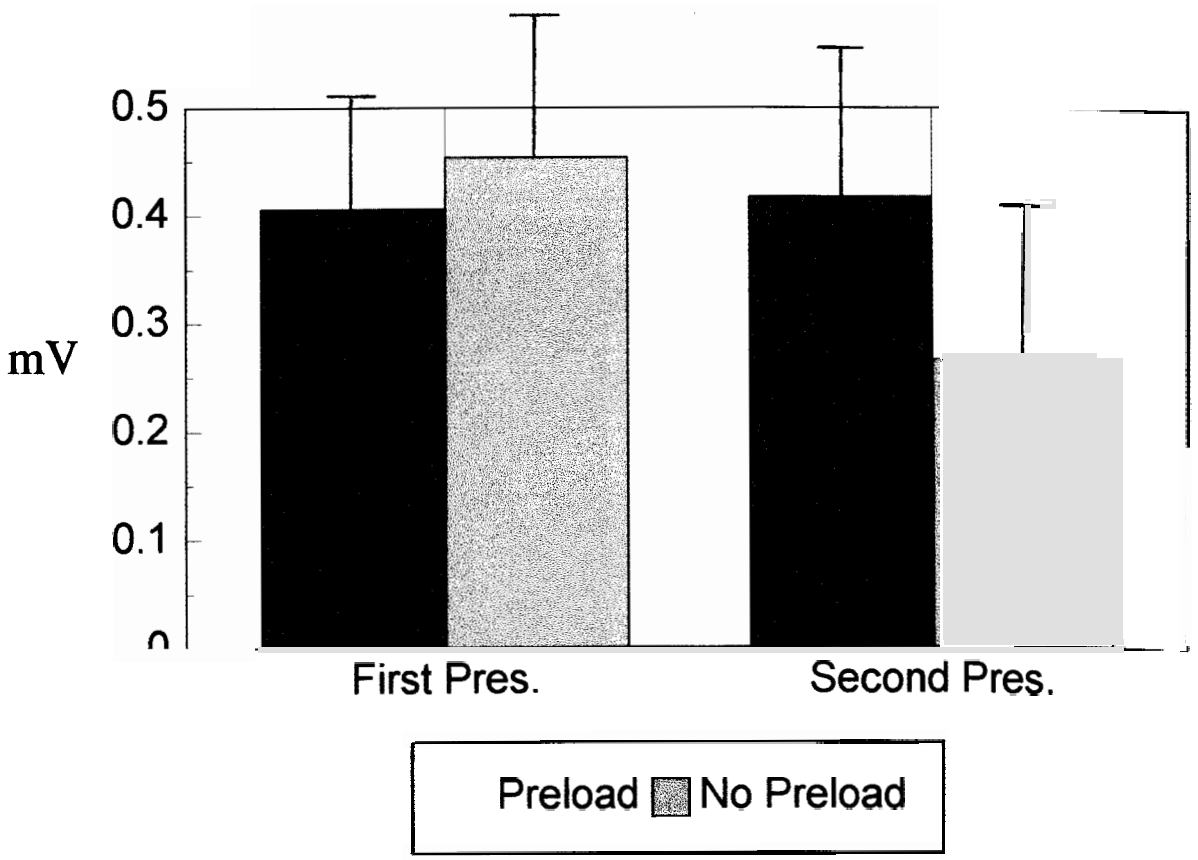


Figure 3. Subjective appetite, first and second ratings, for preload (n = 10) and no-preload (n = 10) groups.

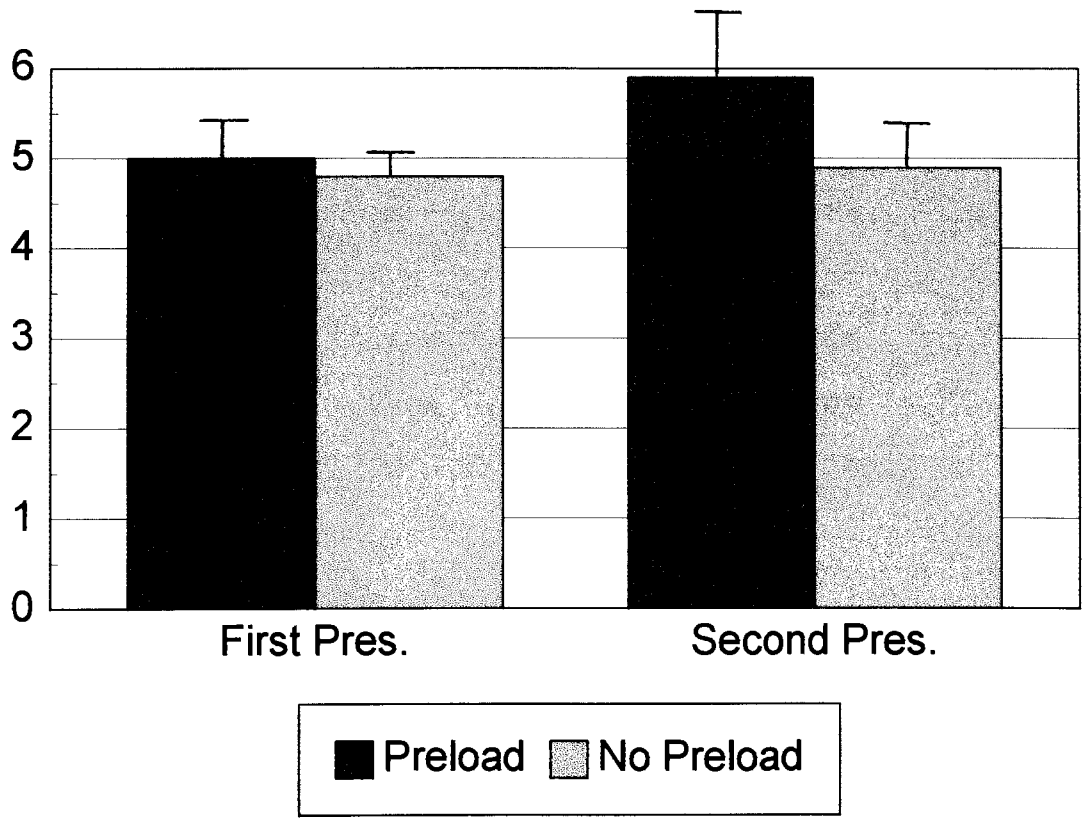


Table 11. Correlation of pizza ingestion with DEBQ-R scores, appetite ratings and ESR to food for preload (n = 10) and no-preload (n = 10) groups.

	Correlation	Variance Accounted For
DEBQ-R scores		
Preload	r = .04	0.2%
No-preload	r = .17	2.9%
Overall	r = -.24	5.8%
Appetite ratings		
Preload	r = .54	29.4%
No-preload	r = .20	4.0%
Overall	r = .51	26.0%
ESR to food		
Preload	r = .38	14.4%
No-preload	r = .27	7.0%
Overall	r = .54*	28.8%

* $p < .005$

A Bonferroni correction for the inflation of type I error rate due to multiple correlations (Bonferroni alpha = alpha divided by the number of comparisons; .05 divided by 9 = .0056) revealed that the only correlation that was statistically significant was that between ESR to food and ingestion, after the preload manipulation, across conditions ($r = .54$, $p < .005$).

For the purposes of testing the first part of the

hypothesis, the relevant correlations between predictor variables and ingestion were ESR to food ($r = .38$), DEBQ-R score ($r = .04$), and appetite ratings ($r = .54$). The relevant cross-correlations were between ESR to food and DEBQ-R score ($r = .34$), and ESR to food and appetite ratings ($r = .04$). The Williams-Hotelling test of differences between correlated correlations found that the difference between the correlations of ESR to food ($r = .38$, N.S.) and DEBQ-R scores ($r = .04$, N.S.) with ingestion was not significant, $t (7) < 1$. Neither was the difference between the correlations of ESR to food, and appetite ratings ($r = .54$), and ingestion, $t (7) < 1$.

The second part of Hypothesis B was also tested using the Williams-Hotelling method. In this case it was used to determine whether ESR to food was a significantly better predictor of ingestion than either DEBQ-R scores or appetite ratings across conditions, that is, for both preload and no-preload groups combined. In this case, the relevant predictive correlations were: ESR to food ($r = .54$), DEBQ-R score ($r = -.24$), and appetite ratings ($r = .51$). The cross-correlations were: ESR to food and DEBQ-R score ($r = -.16$), and ESR to food and appetite ratings ($r = .43$).

Consistent with our hypothesis, ESR to food was a significantly better predictor of ingestion than was DEBQ-R, $t (17) = 2.39$, $p < .025$. The difference between the correlations of ESR to food and appetite ratings with

ingestion did not reach statistical significance, $t(17) = 1.45$, N.S.

Hypothesis C. The average variability of intake from diet records was used as a predictor variable with ESR to food and ingestion of pizza as criterion variables in two separate regression equations. Variability of intake accounted for only 15.3% of the variance in pizza consumption, which was not significant ($r = .39$, N.S., $N = 20$). Diet variability predicted ESR to food somewhat better, accounting for 23.1% of the variance, which was significant ($r = .48$, $p < .05$, $N = 20$).

Williams-Hotelling comparison of these two correlations, taking into account the cross-correlation between ESR to food and ingestion of pizza ($r = .54$), revealed that the correlation of ESR to food with diet variability was not significantly larger than the correlation between ingestion and diet variability ($t < 1$).

An unexpected post-hoc finding was that diet variability and appetite ratings were significantly related, especially after the preload manipulation. Before the preload, the correlation between diet variability and appetite was $r = .43$, N.S., and after the preload, the correlation was $r = .53$, $p < .01$, with a shared variance of 28.1% (Bon Ferroni correction was also applied to these post-hoc correlations).

Discussion

Manipulation Checks. The finding that dieting subjects (selected on the basis of having concerns with body weight and were currently on weight-loss diets) who ingested a high-calorie preload ate more of an ad lib food than did dieters who were not preloaded, is consistent with the findings of Herman and Polivy and others. This finding cannot strictly be considered a replication of the disinhibition effect because: 1) the design did not include a nonrestrained group; 2) the subjects did not have restraint scores comparable to those used by Polivy and Herman, and 3) the ad lib food was different than that used by previous researchers. Nonethless, the subjects in the preload condition ate, on average, 247.5 kcal more pizza than did subjects who did not receive the preload.

Contrary to previous findings in which restrained subjects maintained their restraint in the no-preload condition and ate only small amounts of the ad lib food (Polivy et al, 1988; Herman et al, 1987; Rogers & Hill, 1989), both groups of subjects ate large amounts of the ad lib pizza. A post-hoc examination of the diet records indicated that subjects' average midday meal size was approximately 450 kcal, for both preload and no-preload groups. This suggests that both groups of subjects ate more than an average midday meals' worth of pizza (average consumption of ad lib pizza was 792 kcal for the preload,

and 545 kcal for the no-preload group).

This finding might reflect the fact that the current research used pizza for the ad lib food, as opposed to previous research which had used primarily ice cream, cookies, and in one case, popcorn. These foods clearly suggest that the ad lib ingestion is a "snack," while the large pizza presented here might cue subjects to consider the ad lib food a meal. The pizza might also be presumed to have more potent stimulus properties than would ice cream or cookies.

Control Variables. The DEBQ-R scores for the subjects in the preload and no-preload conditions were very comparable, and both were in the restrained range, according to the norms of the authors (van Strien et al, 1986). The variability of intake values for both groups were also comparable, and similar to the diet variability found in previous research (LeGoff et al, 1989), in terms of both mean and distribution.

Hypothesis Testing

Hypothesis A. The predicted interaction effect in the 2 x 2 ANOVA of preload versus no preload by before and after preload on ESR was found. This result indicated that ESR was sensitive to the differential effects of a preload and the repetition of stimulus presentation. In a similar analysis of appetite ratings, there were no effects, suggesting that ESR to food had greater power of

differentiation of the effects of a preload than did subjective appetite.

However, an examination of the cell means for ESR to food indicated that this interaction effect did not result from an increase in ESR in the preloaded subjects, but by a decrease in ESR in the no-preload group (see Figure 2). The largest change in mean ESR to food was the decrease of .188 mV from first to second presentation in the no-preload group (.454 mV to .266 mV). In this condition, subjects were presented with the food and nonfood stimuli, they waited for 15 minutes, and were presented with a repetition of the same two stimulus sets. This may reflect a decrease in the valence of the stimuli due to the loss of novelty, that is, an habituation effect. In the preload condition, however, subjects responded slightly more (.011 mV) to the second presentation, suggesting that the habituation effect evident in the no-preload condition may have been counteracted by some characteristic of the preload.

Further research could clarify this issue by using two different sets of stimuli in order to avoid habituation effects, and could therefore isolate the effects of the preload, from the effects of repeated exposure to the same stimuli. The fact that subjective appetite did not show the same interaction effect as physiological response, and in fact, showed no significant changes as a result of the preload manipulation, is damaging evidence for the Boundary

Model. The Boundary Model predicts that the preload will be associated with a specifically cognitive mechanism of increased ingestion. In this case, cognitive appraisal (appetite rating) was not significantly affected by the ingestion of a preload, while physiological response (ESR) to food was.

It is concluded that this finding, heightened physiological reactivity to food following ingestion of a preload, is the "appetizer effect" discussed earlier, otherwise described by Jansen and van den Hout as the Compensatory Conditioned Response (CCR). Given that this is the first simultaneous demonstration of exaggerated physiological response and the increased consumption associated with it, replication of these results will likely be necessary.

Hypothesis B. The results of the correlation analyses generally support the physiological model of disinhibition, indicating that ESR to food was a better predictor of disinhibited eating than was DEBQ-R score, and at least as good a predictor as appetite ratings. The strongest support for the hypothesis was the significantly larger correlation between ESR to food and amount of pizza eaten than DEBQ-R, indicating that ingestion was better predicted by elevated physiological response to food than dietary restraint.

In neither the preload nor no-preload conditions did dietary restraint (DEBQ-R score) predict level of ingestion.

The Boundary Model predicts that dietary restraint will be associated with lower consumption in the no-preload condition, and higher consumption in the preload condition. Although this prediction held for group differences, that is, restrained subjects in the preload condition ate more than restrained subjects who did not receive a preload, there was no reliable correlation between restraint score and ingestion.

The findings for appetite ratings and ESR were surprisingly parallel. With both variables, the correlation with ingestion was higher in the preload condition, and was highest for both conditions combined. This finding suggested that there might be a strong relationship between ESR to food and appetite ratings after ingestion of a preload. A post-hoc correlation analysis revealed that ESR to food and appetite ratings, after the preload manipulation, correlated $r = .43$, which was not significant. This does represent a shared variance of 18.5%, however. It was also noted that ESR to food and appetite ratings before the preload were correlated only $r = .23$, N.S. These post-hoc results suggest that the increase in physiological response to food associated with ingesting a high calorie preload is also associated with an increase in subjective appetite, but that this relationship is indirect, and not as strong as the relationship between ingestion and either variable separately.

A post-hoc multiple regression analysis, with ESR to food and appetite ratings used to predict ingestion of pizza after the preload manipulation, revealed that the two variables together accounted for a total of 43.7% of the variance in ingestion of pizza (multiple $r = .66$, $p < .01$). The unique contribution of appetite to ingestion, independent of ESR to food, was 20.3% ($r = .45$). The unique variance of ESR to food to ingestion of pizza, independent of appetite ratings, was 23.3% ($r = .48$).

These findings do not support the hypothesis that subjective appetite is simply a covariate of physiological response to food, and does not contribute unique variance to ingestion. Although ESR to food had a somewhat stronger relationship to ingestion, subjective appetite contributed 20.3% of the total variance, independent of ESR. This implies that both physiological response and subjective experience are important factors in determining ingestion. The two are strongly related to one another, but each contributes unique variance in determining amount of food ingested.

With regard to the models being evaluated, the physiological model and the Boundary Model, both are supported to some degree by these results. The physiological model appears in the best light here, since physiological response to food accounted for more variance in amount ingested than either restraint or subjective

appetite. Nonetheless, the unique contribution of appetite ratings to variance in amount ingested suggests that cognitive factors are important in determining meal size. The Boundary Model does not fair that well with this data, in that it does not account for the physiological component of appetite, independent of cognitive aspects. It also assumes that dietary restraint is a centrally important factor in determining disinhibited eating, and that variable contributed very little to the prediction of eating.

Of course, the predictive ability of this variable may have been reduced by the pre-selection of restrained subjects. It is quite possible that dietary restraint may play a larger role in determining disinhibited eating in the general population, where there are both restrained and nonrestrained subjects. The present research does not allow for any conclusion regarding this possibility. On the basis of the current data, dietary restraint does not appear to play a central role in determining the extent of disinhibition, compared with physiological response and subjective appetite.

Hypothesis C. The strong correlation between diet variability and ESR to food confirmed the first part of this hypothesis, and replicated a previously reported finding (LeGoff et al 1989) that there is an exaggerated salivary response to food in subjects with high diet variability. The moderate, but not statistically significant relationship

between diet variability and disinhibited eating in the laboratory must be considered inconclusive. It seems likely that with a larger sample size, a reliable relationship might be found, however, this would need to be tested.

Factors which could have reduced the likelihood of finding a significant relationship include the following: 1) the small sample size (n = 10); 2) the highly different measures used -- self-reported ingestion and in vivo ingestion; and 3) the use of a relatively new, and unstandardized measure of diet variability. Diet variability has been used previously to predict level of salivary response to food (LeGoff et al, 1989), but this was the first attempted demonstration of a relationship between dietary dyscontrol on eating records, and dysregulation of restraint in the laboratory.

According to the physiological model proposed here, there is a causal relationship between physiological responsivity and amount ingested; the CCR theory. The correlational results, however, do not allow for causal interpretations. Nonetheless, in support of this causal model, it was proposed that there would also be a correlation between diet variability and ad lib ingestion in the laboratory. This relationship, according to the model, is dependent upon the relationship of cephalic phase reflexes to ingestion, so that the relationship between eating patterns and laboratory ingestion would necessarily

be the weaker one. The finding that there was a smaller, nonsignificant correlation between diet variability and ingestion overall, is consistent with the hypothesis that eating patterns and ingestion are putatively mediated by physiological reactivity to food, so that eating patterns and ingestion are one step removed from each other.

In other words, sporadic eating patterns lead directly to an elevated response to food, so diet variability predicts ESR to food well. Physiological response to food is directly associated with ingestion, and so predicts it well, but eating patterns and ingestion are indirectly linked via physiological responsivity, and so their relationship cannot be as strong as either of the direct relationships. This is in fact what was found, providing support for the physiological model of disinhibition.

The Boundary Model, on the other hand, predicts a strong relationship between disinhibitory cognitions and disinhibited eating, and a weaker, more distal relationship between dietary restraint and disinhibition. Unfortunately for the model testing process, these predicted relationships also held true: there was a small, nonsignificant relationship between dietary restraint and amount of ad lib food ingested after a preload ($r = .25$, N.S.), and a significant positive relationship between subjective ratings of appetite and ingestion following a preload ($r = .51$, $p < .05$).

An aspect of the present findings which is clearly inconsistent with the Boundary Model is the fact that restraint overall did not correlate with disinhibited eating, while diet variability did. The Boundary Model specifies that restraint leads to susceptibility to disinhibited eating. In past research, however, the relationship between restraint and disinhibition has been difficult to determine because of the poor construct validity of the Restraint Scale, which combines cognitive restraint items with weight fluctuation items. In the present research, the restraint measure used contained only cognitive restraint items. As a result, it appears that it is not cognitive restraint per se that leads to disinhibition.

The finding of a significant correlation between diet variability and subjective appetite in the preload condition was not predicted. This result might be explained by the correspondence between diet variability and ESR to food, such that subjects who are experiencing a strong hunger drive are more consciously aware of this and are able to be more accurate in their cognitive appraisal of appetite. The Boundary Model does not provide an adequate explanation of how diet variability could lead to more accurate appetite ratings in disinhibited subjects.

Summary. The findings reported above were not entirely consistent with the hypotheses, although they were generally

supportive. The general conclusions that can be made are as follows:

1) ESR to food appears to be increased by the ingestion of a preload, or at least, responsivity is not decreased by repeated presentation of stimuli;

2) level of ESR to food after ingestion of a preload predicts amount eaten of an ad lib food;

3) subjective appetite may also predict amount of consumption after a preload;

4) subjective appetite and ESR to food are related to each other, but both have unique variance associated with ad lib consumption after a preload;

5) dietary restraint did not predict amount of consumption of ad lib food after a preload;

6) dietary restraint did not predict ad lib consumption in general;

7) variability of intake from eating records predicts heightened responsivity to food stimuli after ingestion of a preload;

8) variability of intake from eating records was only weakly associated with amount of food ingested after a preload;

9) variability of intake from eating records predicts heightened subjective appetite after the ingestion of a preload.

GENERAL DISCUSSION

The preceding research has presented an attempt to validate an electrophysiological measure of appetite, and to apply this measure in examining the cognitive, physiological and behavioral components of disinhibited eating. In the first experiment, it was found that the electrophysiological measurement of parotid activity was sensitive to food versus nonfood olfactory stimuli, was correlated with time since last meal, and differentiated fasted from nonfasted subjects, to a significant degree in each case. In the second experiment, cognitive variables were assessed and related to electrosalivary response (ESR) to food. It was found that while dietary restraint was related to differences in ESR, subjective ratings of appetite were not.

These results contain two important findings: 1) that ESR to food is a reliable, sensitive and valid measure of appetite, with appetite operationally defined as time since last meal; and 2) that subjective reports of appetite are not well correlated with this physiological response. The data, however, do not establish whether the lack of correspondence between subjective appetite and physiological response is due to measurement error or an inherent lack of correspondence between cognitive and physiological components of the hunger response.

The fact that dietary restraint (as measured by the DEBQ-R) had a significant correlation with ESR confirmed

previous findings that restrained subjects had heightened salivary responses to food (LeGoff et al, 1989; LeGoff & Spigelman, 1988), without increased subjective awareness of appetite (LeGoff, Cox, Beyerstein and Krane, 1989). These results suggest that trait dimensions of body weight regulation, such as attitudes towards eating and body size, may be important in determining ingestion.

The view that trait factors are important in determining ingestion, in addition to state or situational variables, is supported by the results of the third experiment. In this experiment, the disinhibition protocol was partially replicated; self-reported dieters either were given or were not given a high-calorie preload, and then all subjects were allowed ad libitum access to pizza (non-dieting subjects were not tested). The disinhibition effect for dieters -- greater than usual ingestion in the preload group but not in the no-preload group -- was replicated. The complete disinhibition effect (down-regulated eating in nondieters, and counter-regulatory eating in dieters) was not replicated as there was no non-dieting control group.

The differences between this protocol and previous ones (aside from the exclusion of non-dieters) were: 1) the inclusion of measures of both subjective and physiological appetite both before and after the preload manipulation, and 2) the analysis of eating patterns based on diet records. As was reported above, and has been found in previous

research (LeGoff et al, 1989; LeGoff et al, 1988), subjects whose diet records contain considerable fluctuation in caloric content showed the largest ESR to food in the lab. There was also a tendency for variability of intake on diet records to correlate with increased ingestion of ad lib food. These results suggest that the large amounts of pizza consumed in the lab by some subjects reflects a pattern of hyper-responsivity to food and over-ingestion which occurs in many settings. That is, subjects who ate a lot of pizza in the lab, tend to eat large and small meals in an irregular fashion, presumably because they are less planful, more impulsive, and possibly more physiologically affected by environmental cues to eating than are more regular eaters.

A third, unexpected finding, was the interaction effect on ESR to food, between first and second testing of ESR, and preload versus no-preload conditions. It was found that subjects in the preload condition did not show the expected increase in ESR to food after the preload. The control group, however, who received no preload but had their salivary responses to the stimuli tested twice, showed a significant decrease in ESR to food. A couple of factors may have been affecting the ESR data. First, the repeated presentation of the stimuli may have caused an habituation effect, such that the food odours were less effective in eliciting an appetite response on the second presentation.

Second, congruent with the CCR theory, the preload likely caused a compensatory increase in the cephalic-phase digestive reflexes (i.e. the CPR).

Consequently, the no-preload group showed a reduced ESR, while for the preload group the preload had stimulated a CPR with heightened salivation, so that when ESR was tested a second time, it was still elevated. In the first exposure to the stimuli, ESR was elevated by the food odours, and perhaps by the expectation of consumption. In the second exposure to the odours, the habituation effect reduced the impact on ESR, but the ESR was still affected by both the ingestion of the preload, and the expectation of consumption. This explanation of the results is, of course, post hoc, and will require experimental evaluation in order to verify or disprove it (see below).

Implications for the Cognitive Model of Disinhibition

It is important to note that the amount of the ad lib pizza eaten by subjects was not correlated with dietary restraint, as measured by the DEBQ-R. This measure was designed to assess a cognitively-based attitude towards eating and body weight, and does not include items which reflect fluctuations in body weight or disinhibited eating. As such, it represents a relatively "pure" measure of the subject's characteristic attitude towards food: an intention to restrict their intake in order to control their body weight.

As has been noted by other researchers (eg. Charnock, 1989; Lowe, 1986) it seems that restraint per se is not predictive of disinhibition, although it may be a necessary precondition for the disinhibition effect. That is, more restraint does not lead to greater loss of control over eating after ingestion of a preload, but without any restraint, there is no disinhibition. This point was made by LeGoff et al (1988) with reference to the fact that bulimic and anorexic eating-disordered patients, who had very different patterns of ingestion, and were significantly different in their salivary responses to food, had similar restraint scores. This point was also made by Lowe and Kleifield (1988) and by Charnock (1989), who found that there appeared to be two patterns of dietary restraint: successful and unsuccessful.

This line of argument recalls the early work by Schachter and colleagues on the externality hypothesis in that there appears to be a subgroup of individuals whose eating patterns are unduly influenced by external factors such that they are unable to maintain internal control over their eating. Herman, Polivy and colleagues criticized this perspective, and proposed the concept of dietary restraint in order to account for the disinhibited eating of individuals who were attempting to regulate their body weight through dietary restriction. Although they started with a physiological explanation of disinhibition (Powley's

CPR and Nisbett's set point theory), they eventually abandoned this theory and proposed the Boundary Model, which is a cognitive theory of binge eating.

The current research supports the view proposed by many researchers (Lowe & Kleifield, 1988; Charnock, 1989; Jansen et al, 1992; Weingarten, 1985) that there are both cognitive and physiological factors involved in the disinhibition effect, such that some subjects are better at maintaining restraint than others despite having equivalent levels of restraint (i.e. intention to restrict intake). The most elaborated physiological theory of the disinhibition effect is the CCR theory, proposed by Jansen and Van den Hout (1991) and described earlier. It is not clear in their explanation what role, if any, cognitions play in the disinhibition effect.

The findings reported here, and in previous research, suggests that cognitive factors in the regulation of eating behavior certainly cannot be ruled out altogether. A number of studies have demonstrated that expectations and mental set can increase ingestion and even cause disinhibition (Heatherton et al, 1989; Herman et al, 1987; Herman, Polivy and Esses, 1987). Others have shown the opposite effect -- cognitive factors enhancing the ability to regulate ingestion effectively (Herman et al, 1979; Kirschenbaum & Tomarken, 1982). Rogers and Hill (1989) found that cognitive set can have a mediating effect on both salivary

response to food, and intake. They suggest that self-control strategies may decrease the likelihood of food stimuli eliciting a CPR which in turn makes it more likely that subjects will maintain restraint. This hypothesis is consistent with the finding of LeGoff et al (1988) that anorexic patients salivated very little in response to food, but their responses increased following a period of cognitive-behavioral treatment.

Finally, there is considerable research on the effective use of cognitive treatments of bulimia nervosa, and specifically, cognitive treatment of binge eating (Fairburn, 1981; Garner & Bemis, 1982; Fairburn, 1985; Rossiter & Wilson, 1985; Fairburn et al, 1986; Garner, 1986). In fact, some clinical researchers contend that effective longterm outcome in the treatment of bulimia is more dependent on changes in beliefs and attitudes, than on changes in behavior alone (Agras et al, 1989; Bauer & Anderson, 1989; Craighead & Agras, 1991).

Despite the evidence of the role of cognitions in diet regulation, dietary restraint has not been consistently demonstrated to be a sufficient cause of disinhibited eating in the laboratory (Jansen et al, 1988; Lowe & Kleifield, 1988). This anomaly may be explained by noting that dietary restraint, as it is normally construed, is a trait-type variable and therefore a <u>distal</u> influence on eating behavior. In the research which examined cognitions

directly, they had subjects report their cognitions at the time they were eating, such that they were testing the proximal effects of cognitions on eating.

Although these studies provide evidence which contraindicates a proximal cognitive effect in disinhibited eating, they do not rule out distal cognitive factors. In the results from both the second and third experiments reported here, distal (restraint scores) and not proximal (subjective ratings of hunger) cognitive factors were potentially influential. As such, it seems likely that although cognitions do not necessarily play a proximal role in the disinhibition effect, they appear to have a distal role, in being a necessary though not sufficient precondition for the disinhibition effect to occur.

Two-Factor Cognitive-Physiological Model of Disinhibition

In conclusion, the present state of knowledge about the disinhibition effect, including the current findings, can be summarized by the following two-factor model. The first factor is the cognitive one, dietary restraint, which appears to be a necessary, initial stage in the development of eating disorders, as has been sufficiently demonstrated by Polivy, Herman and colleagues (cf. Polivy & Herman, 1985; 1987). Once having developed the attitudes and beliefs associated with the "dieting mentality," the struggle begins, and individuals have varying degrees of success in maintaining their restraint. Unfortunately, most dieters

are not consistently successful, in fact, the predominance of them vacillate between restriction and over-ingestion (cf. Wooley, 1972; Wooley et al, 1979; Polivy & Herman, 1987), such that almost no diet plans are effective in permanently reducing body weight (Wooley & Wooley, 1984).

This "fence-sitting" (Herman & Polivy, 1980) tends to result in a growing disparity between meal sizes. As has been suggested by Jansen and her colleagues, as well as by LeGoff et al (1989), this pattern of large and small meals results in an exaggerated physiological responsiveness to cues to ingestion, both internal and external. It is hypothesized that this effect is the result of conditioning of the CPR, which needs to increase in order to accommodate larger meal sizes. Consequently, expectations of consumption, and/or disinhibiting stimuli produce an exaggerated CPR which drives the restrained individual to eat more than they would otherwise. The strong physiologically-based appetite response, reflected in elevated salivary activity, as well as other components of the CPR, is in conflict with restrained cognitions and the intention to remain in control of eating is overpowered.

The cognitions which Polivy and Herman suggest are present, and cause the disinhibition, "I've blown my diet, I might as well keep eating," may be secondary to the fact that the individual has lost control over ingestion, and is simply noting the ingestive behavior after the fact. The

sequence of cognitions could therefore be: "Oh no, I've lost control of my restraint. I might as well enjoy this since I can't seem to stop." Consequently, once the compensatory mechanism has been stimulated, and the exaggerated CPR has been released, the individual experiences a loss of cognitive, volitional control over eating. This explanation is consistent with the current data, but is conjecture at this point since there is not sufficient data in the current study to determine the sequence of cognitive versus physiological factors.

The Disinhibition Effect: Compensation or Conditioning

The hypothesis proposed by Jansen et al (1992) that the disinhibition effect is a Conditioned Compensatory Reflex (CCR), is alternatively construed of here as a conditioned "appetizer" effect. According to Jansen et al, disinhibited eating reflects a biologically based mechanism for modulating weight loss due to restrictive dieting. The sustained salivary responses of dieting subjects to food, and the disinhibition effect may reflect this CCR, however, the manner in which this takes place has not been examined.

Rather than reflecting simply the imposition of caloric restraint, the heightened ingestion exhibited by disinhibited subjects may be due to conditioning of the CPR. The CPR needs to release an appropriate amount of digestive enzymes, gastric acid, saliva, insulin, etc., for the size of the meal about to be ingested. Therefore, the CPR needs

a mechanism for predicting the timing and size of meals, such as is provided by the conditioning effect (Powley, 1977).

The fact that the CPR is both based on past meal sizes, and drives current eating through its insulin and enzyme secretion, means that eating patterns tend to become entrenched and difficult to change. Such as is the case with binge-eating and eating at certain times of day. For instance, the past experience of binges in which there may not have been sufficient gastric acid, enzymes or salivation in order to accommodate the intake, results in hypersecretion of saliva, gastric acid, digestive enzymes, insulin, etc. when the next opportunity for a binge arises.

In this case, only those subjects who characteristically eat large meals, and then attempt to control their weight gain by fasting, as opposed to subjects who eat consistently smaller amounts of food and do not binge, will have an elevated CPR, and secondarily, show the "appetizer" effect. The finding of elevated ESR to food in subjects who had highly variable diets, both here and in LeGoff et al (1989) supports this conceptualization. Further research is clearly needed in order to fully assess the relative validity of these hypotheses.

Limitations of the Research

One of the main problems with the present research, as with much of the research in this area, is the insufficient

standardization and validation of measures and procedures. Notably, many researchers have examined the disinhibition effect using a variety of protocols, including different preloads and different ad lib foods. Although there was careful thought given to the selection of the preload and ad lib foods in the present research, the procedure was not based on a replication of a design used by consensus. The protocol here was modelled as closely as possible to the original Herman and Polivy procedures, but other models might have been chosen. One deviation from the Herman and Polivy protocol, which might have implications for the results, was the use of pizza as the ad lib food.

In previous research, the Herman and Polivy group have used a variety of snack foods, including popcorn, cookies and sandwich quarters, as well as pizza. Pizza was chosen for this study in order to allow for maximal ingestion, that is, truly disinhibited eating. It was felt that the snack foods used by Herman and Polivy could cue patients to eat smaller amounts, whereas pizza would be more likely to elicit binge-like disinhibited intake. It might be argued, then, that the lack of correspondence between degree of dietary restraint and amount eaten of the ad lib food might have resulted from an overly effective manipulation, i.e. even non-restrained subjects might overeat under such conditions. The fact that the subjects in the no-preload group ate meal-size amounts of the pizza, despite their

dietary restraint, supports this view.

Nonetheless, pizza was used as the ad lib food in order to increase ecological validity and generalizability. The subjects themselves reported considerable ingestion of pizza on their diet records, and would normally be exposed to situations similar to those created by the experiment.

A second limitation of the methodology was the nonstandardized and crude measure of subjective appetite. Although a number of researchers have used self-ratings of appetite in the past, there is no standardized procedure or validated instrument for recording subjective appetite (Mattes, 1990; Jansen et al, 1994). The lack of correspondence between subjective appetite and ESR in experiment II, and the absence of effect on subjective appetite by the preload manipulation in experiment III might be explained as a result of poor reliability and sensitivity of the measure. Unfortunately, at this point in time, there is no good alternative, and the single self-rating is the "state of the art" measure. This fact may reflect that researchers in this area attribute little importance to subjective appetite as a variable.

A third methodological limitation of the research reflecting the paucity of standardized and validated measures related to the use of diet records, and specifically, the diet variability measure. A thorough review of the literature indicated that there is no extant

standardized procedure or measure for assessing diet patterns, beyond the use of diet records. The technique used here was previously used by LeGoff et al (1989), and the diet variability variable was shown to be related to elevated salivary responses to food. Beyond that, however, the reliability and validity of diet records as a measure of degree of diet impulsivity or impulsive eating in the laboratory has not been established. Perhaps the first step in this validation has been taken here, with the demonstration of at least a small, though nonsignificant, correlation between diet variability on eating records with disinhibited eating in the laboratory.

The restraint model proposed by Polivy and Herman (1984) indicates that it is cognitively-based restraint which leads to disinhibition, and disinhibitory cognitions which trigger the disinhibited eating. A major limitation of the current research was the lack of assessment of these proximal, disinhibitory cognitions. It was assumed that subjective appetite would be the end-result of any and all cognitive reactions to the food presented, such that cognitions such as "I've blown my diet, I might as well keep eating," would be reflected in higher appetite scores. This procedure was used in order to avoid the potential for expectancy and cuing effects, as well as to control for measurement effects on ingestion, i.e. making subjects record their thoughts might inhibit eating. Additional

post-hoc measures might have been used which would have avoided reactance and cuing effects, while allowing for an assessment of such potentially important cognitive variables as perceived control or loss of control, expectancy of ingestion, self-predictions of ingestion, etc. Additional information regarding the interaction of restraint, appetite and ingestion might have been garnered by using a non-restrained control group in experiment III. Although the focus of the research was on the cognitive and physiological reactions of restrained subjects to a disinhibition procedure, the inclusion of non-restrained subjects would have provided a comparison group against which to compare such variables as amount of ad lib food consumed with and without the preload, effects of repeated exposure to food stimuli on ESR, and correspondence between eating record data and ingestion in the laboratory.

With regard to this last variable, there may have been insufficient numbers of subjects in experiment III to provide an adequate test of some of the hypotheses, in particular, the correspondence between diet record and in vivo ingestion data. The relatively low numbers (N = 20) may have reduced the level of power such that these effects were not detectable.

Finally, a general problem with the research is the paucity of operationally defined terms, standardized and valid instruments, and accurately defined models. Much of

the research on the psychological and physiological factors involved in human ingestion and the etiology of eating disorders remains descriptive and limited in terms of generalizability because of this lack of theoretical and empirical rigour. It seems particularly important in an area in which there are so many factors involved -- including social, cultural, learning, environmental, and physiological -- that there be careful operationalization of terms, validation of instruments, and standardization of methodology. Only then will it be possible to undertake the complex model testing necessary to properly understand these difficult problems.

Future Research

The limitations of the present research outlined above suggest some important areas for future research. Primary among these would be the further standardization and validation of the measures used including the ESR, diet records, self-reported appetite, and other relevant cognitive variables using operationalized variables, larger sample sizes, and standardized procedures.

Part of the validation of the ESR should include a comparison of ESR with other measures of the CPR, such as insulin and gastric secretions. As well, further validation of the CPR theory in general might be undertaken, including a test of the theory that heightened CPR is a proximal cause of increased ingestion. This latter area of research will

necessarily require the use of animal subjects, with later correlational designs in order to establish generalizability of results.

A central hypothesis of the two-factor model of disinhibition which needs to be examined is the putative learning effect of large and small meal sizes on CPR. Although it has been twice demonstrated that variability of intake on eating records is related to elevated salivation to food, here and in LeGoff et al (1989), these findings are correlational. Experimental manipulation of eating patterns, likely using animal subjects again, would be necessary to assess the putative learning effect of large and small meal sizes on CPR to food. Although the empirical validation of this model has been initiated in recent research and was continued in the present series of studies, much more is left to be done.

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Appendix A

Eating & Nutritional Disorder Screening Items

1. Do you currently suffer from, or have you previously been diagnosed with any of the following disorders:

   ____ **Anorexia nervosa** (severe restriction of food intake, with significant loss of body-weight; refusal to maintain adequate body-weight; extreme fear of gaining weight).

   ____ **Bulimia nervosa** (alternating periods of severe food restriction or fasting, followed by rapid ingestion of large amounts of food. Feeling out of control of eating. Self-induced vomiting after binges, or laxative abuse, or excessive exercise).

   ____ **Binge-eating disorder** (periods of rapid consumption large amounts of food accompanied by feelings of being out of control; inability to stop eating despite medical or nutritional advice, etc).

2. What is the most you have weighed in the past? _____ lbs

3. How old were you when you weighed this? _____ yrs

4. What is your current height? _____ ft _____ in

5. Have you had any medical problems with your digestive system (i.e. salivary glands, mouth, throat or stomach)?

   _____ yes _____ no

   If yes, please describe:

_____

_____

_____

6. List any current medications:

_____

_____

_____

7. Do you smoke or use other forms of tobacco?

   _____ yes _____ no

Appendix B

The Dutch Eating Behavior Questionnaire - Restrained Eating

1. When you have put on weight, do you eat less than you usually do?

1	2	3	4	5
never	seldom	sometimes	often	very often

2. Do you try to eat less at mealtimes than you would like to eat?

1	2	3	4	5
never	seldom	sometimes	often	very often

3. How often do you refuse food or drink offered because you are concerned about your weight?

1	2	3	4	5
never	seldom	sometimes	often	very often

4. Do you watch exactly what you eat?

1	2	3	4	5
never	seldom	sometimes	often	very often

5. Do you deliberately eat low-cal and diet foods?

1	2	3	4	5
never	seldom	sometimes	often	very often

6. When you have eaten too much, do you eat less than usual the following day?

1	2	3	4	5
never	seldom	sometimes	often	very often

7. Do you deliberately eat less in order not to gain weight?

1	2	3	4	5
never	seldom	sometimes	often	very often

8. How often do you try not to eat between meals because you are watching your weight?

1	2	3	4	5
never	seldom	sometimes	often	very often

9. How often in the evenings do you try not to eat because you are watching your weight?

1	2	3	4	5
never	seldom	sometimes	often	very often

10. Do you take into account your weight when deciding what to eat?

1	2	3	4	5
never	often	sometimes	often	very often

Appendix C

Diet Record Sheet

Date: _____ Name: _____

TIME	FOOD	BEVERAGES

DON'T FORGET:

- Record what you ate and drank at the end of each day.
- Be sure to record the times for each meal.