12-1987

Restrained Eating : The Effects Of Privacy And Perception Of Having Overeaten On Subsequent Food Consumption

Geraldine T. Merola

RESTRAINED EATING: THE EFFECTS OF PRIVACY AND PERCEPTION OF HAVING OVEREATEN ON SUBSEQUENT FOOD CONSUMPTION

by
Geraldine T. Merola

Master of Arts, University of North Dakota, 1984

A Dissertation

Submitted to the Graduate Faculty

of the

University of North Dakota

in partial fulfillment of the requirements

for the degree of

Doctor of Philosophy

Grand Forks, North Dakota

December
1987

This Dissertation submitted by Geraldine T. Merola in partial fulfillment of the requirements for the Degree of Doctor of Philosophy from the University of North Dakota has been read by the Faculty Advisory Committee under whom the work has been done, and is hereby approved.

(Chairperson)

This Dissertation meets the standards for appearance and conforms to the style and format requirements of the Graduate School of the University of North Dakota, and is hereby approved.

9-18-87

Dean of the Graduate School

PERMISSION

Title: Restrained Eating: The Effects of Privacy and Perception of Having Overeaten on Subsequent Food Consumption

Department: Psychology

Degree: Doctor of Philosophy

In presenting this dissertation in partial fulfillment of the requirements for a graduate degree from the University of North Dakota, I agree that the Library of this University shall make it freely available for inspection. I further agree that permission for extensive copying for scholarly purposes may be granted by the professor who supervised my dissertation or, in his absence, by the Chair of the Department or the Dean of the Graduate School. It is understood that any copying or publication or other use of this dissertation or part thereof for financial gain shall not be allowed without my written permission. It is also understood that due recognition shall be given to me and to the University of North Dakota in any scholarly use which may be made of any material in my dissertation.

Signature Geraldine Talerola

Date Sept. 15, 1987

TABLE OF CONTENTS

LIST OF ILLUSTRATIONS

LIST OF TABLES

ACKNOWLEDGMENTS

Thanks to David Stein, Ph.D., my dissertation advisor, for his enthusiastic and capable sharing of experience, expertise, and encouragement. Thanks to William Beckwith, Ph.D., whose immeasurable help with my master's thesis work made it possible for me to pursue my interest in eating disorders, an area that no one in the Department of Psychology was researching at the time. Thanks to Lila Tabor, Ph.D., Douglas Peters, Ph.D., and Donald Lemon, Ed.D., of my dissertation committee, for their valuable suggestions and support. I wish to especially and lovingly thank David Rosenberg for his love, encouragement, assistance, and extraordinary devotion, which helped to make my work delightful as well as rewarding.

ABSTRACT

Herman & Mack (1975) theorized that "restrained eaters," conflicted between social pressure to be thin and biological pressure to be fat, tend to alternately eat very little or a great deal, as they respond to one or the other constraint. According to restraint theory, restrained eaters' chronic dieting induces physiological and psychological states that make them highly susceptible to external disruption of eating controls. Research has shown that when led to believe they have already overeaten (i.e., when they are "preloaded"), restrained eaters will loosen restraints and "counterregulate" (i.e., binge-eat). In contrast, "unrestrained eaters" (i.e., normal eaters) will compensate by subsequently eating less under such conditions. However, while this effect has been shown in normal weight restrained subjects, overweight restrained subjects have not reliably counterregulated. Consequently, questions can be raised as to restraint theory's ability to predict eating behavior of overweight individuals. One study utilizing a private setting found counterregulation in preloaded normal weight and overweight restrained eaters. However, because a no-preload group was not included in this study, it could not be determined whether the preload or the private setting was responsible for the counterregulatory eating.

In the present study, 113 female subjects were told they were participating in a sensory experiment. Normal weight and overweight subjects, who were low restraint or high restraint, either consumed a "high calorie" milkshake as a preload or received no preload. Subjects were subsequently asked to "taste-test" ice cream flavors. Using subtle situational cues, subjects were led to believe the amount of their ice cream consumption would not be easily detected by experimenters. As expected, low restraint-normal weight subjects compensated for a preload by eating less ice cream; and low restraint-overweight subjects ate the same amount regardless of preloading. However, both normal weight- and overweight-high restraint subjects failed to counterregulate after preloading.

The author discusses implications of this and previous findings. It is argued that the restraint dimension may reflect more a cognitive style than a behavioral style. It is suggested that disordered eating may be more productively studied under naturalistic conditions, or longitudinally, than by taking a single measurement in a laboratory situation.

CHAPTER 1

INTRODUCTION AND LITERATURE SURVEY

The question of weight control seems straightforward enough: Those who indulge in too much food, or the wrong kind, become fatter than those who diet. Commonsensical as the idea seems, and notwithstanding the weight-loss industry that has capitalized on it, it is not quite correct.

The vast literature of experimental and clinical reports indicates there is no single etiology of fatness. Weight is determined by a complex of psychological, physiological and genetic variables. Indeed, after reviewing some one hundred studies relying on observational and self-report data, Judith Rodin (1981) was forced to conclude there are few clear (i.e., sufficiently replicated) differences in eating patterns between normal weight and overweight individuals. Thompson, Jarvie, Lahey, & Cureton (1982) also reviewed studies that compared the food consumption of obese and matched normal weight subjects, and reported that while some studies found differences, no eating behaviors reliably differentiated the groups across studies.

It is well to note that most observational studies of eating by the overweight have taken place in public settings, where only a small percentage of total daily food consumption occurs, and where the obese might well be too self-conscious to overeat. Indeed, Thompson et al. caution that such limited observations of meal behavior and self-report data may be insufficient means for quantifying consummatory practices.

Despite the failure to find well replicated differences between normal weight and overweight subjects, obesity and eating disorders continue to be among the most researched areas of the behavioral sciences. To better consider the reasons that experimentally produced differences in normal weight and overweight subjects have been so unreliable, let us survey relevant investigations in the field.

Genetic Contribution to Overweight

It has long been theorized that human obesity can be predisposed by genetic factors influencing total adipose tissue and relative fat distribution, as well as by early feeding habits (Mayer, 1953; Hirsch & Knittle, 1970; Salans, Cushman, & Wiseman, 1973). It seems that the more fat cells an individual develops and the larger the fat cells grow, the heavier that individual is disposed to become.

Chronicity of obesity seems to be heavily influenced by age of onset. Those who became obese in childhood (before age 15) often suffer from a hyperplasic condition, that is, they present with a larger than normal number (two to five times more) of adipocytes (i.e., fat cells). These juvenile-onset obese are also hypertrophic, that is, their adipocytes are significantly larger than normal.

While dieting temporarily reduces the size of fat cells, it cannot decrease the number (Hirsch & Knittle, 1970). During a diet, these nearly depleted adipocytes trigger urgent hunger signals, ultimately causing a return to overeating and weight regain. There is also evidence that following loss of large amounts of weight, the juvenile-onset obese experience greater anxiety, depression, concern over altered body size, preoccupation with food, and decreased energy (Grinker, Hirsch, & Levin, 1973). The fat child is likely to become a "hopelessly" fat adult; whereas the normal weight child can become fat as an adult but still have a good chance of permanently shedding the extra pounds.

The influence of genetic factors on obesity was convincingly demonstrated recently by Albert Stunkard with American and Danish colleagues (Stunkard, Srensen, Hanis, Teasdale, Chakradorty, Schull, & Schulsinger, 1986). These researchers studied 540 Danish adults who had been adopted soon after birth. The researchers found strong correlation between the weight and size of the adoptees and that of

their natural parents. Eighty percent of the offspring of two obese parents became obese. No correlation was found between adoptees' size and weight and that of their adoptive parents.

Stunkard wisely issues the disclaimer that, "Biology is not destiny," stressing that clinicians and researchers must turn their attention to prevention. Although early eating habits may be less responsible for later obesity than had been supposed, good eating habits, learned early, may ameliorate the impact of genetic predisposition.

Griffiths & Payne (1976) studied nonobese children of obese parents, who are (statistically) more likely to become obese than children of normal weight parents. These "preobese" children averaged 770 calories per day lower basal metabolic rate (BMR) than nonobese children of nonobese parents.[1] Low metabolic rate, a genetically transmitted factor in many cases, promotes weight gain and makes weight loss extremely difficult.[2]

One might speculate that this difference in BMR is due to differing levels of exercise. However, in a review of studies that compared exercise levels of preobese and

---

[1] BMR is the amount of energy required by the body at rest to carry out normal physiological processes. BMR uses two-thirds of the body's energy, leaving available the remaining one-third for physical activity.

[2] Low metabolic rate is a gender characteristic of women, partially explainable by their having less lean body mass and a higher proportion of fat than men. Women have a harder time losing weight than do men.

nonobese children, Thompson et al. (1982) unfortunately found inconsistency of methods and results. Thus, it is unclear whether the lower BMR found in preobese children is the result of an innate predisposition for the body to encourage fatness or the result of lower activity level, or both.

The Hypothalamic Endocrine System in Weight Fluctuation

The hypothalamus is the integrating center regulating calorie intake in the long- and short-term (Nisbett, 1972; Bray, 1976). Long-term regulation of hunger, food-seeking behavior, and food consumption may be accomplished by the ventromedial hypothalamus, while short-term caloric balance seems to be a function of the lateral hypothalamus. In choreographing the exquisite balance necessary for optimal nutritional intake, the hypothalamus relies on peripheral signals from the body (i.e., stomach distension and levels of amino acids, glucose, free fatty acids, and glycerol).

Schachter & Rodin (1974) suggested that overweight humans' hyper-responsiveness to environmental food- and nonfood-related stimuli (a more detailed discussion of this phenomenon will follow) is similar to that of rats lesioned in the ventromedial region of the hypothalamus (VMH). To wit, VMH-lesioned rats seem to develop an elevated "set-point" for their adipose tissue stores (the concept of setpoint will be explained in a later context), causing their bodies to reach and maintain a higher weight than the

weight that was maintained before lesioning. VMH-lesioned rats develop fat cells four to five times larger than normal. These rats will grossly overeat and become very fat when offered their preferred foods. However, they are finicky about the taste and texture of their food, and even in a hungry state will eat much less of quinine-adulterated foods than will non-lesioned hungry animals. These rats have a highly efficient metabolism; in effect, because calories are burned slowly, it takes less food to make them fat.

Schachter & Rodin (1974) suggest that these tendencies are analogous to those of obese humans, who tend to have fat cells some two and one-half times larger than normal, and will eat much less of adulterated, unpleasant tasting foods than will normal subjects (Nisbett, 1968b; Decke, 1971; Rodin, 1975). Hashim & Van Itallie (1965) also found that when they offered hospitalized obese subjects a diet that was nutritionally complete but unappetizing in taste, subjects who had previously eaten all they were offered of appetizing food now consumed only 400 to 500 calories daily. In contrast, normal subjects maintained their previous calorie intake. It seemed that obese subjects were not eating to satisfy nutritional requirements of their bodies but instead, like VMH-lesioned rats, were markedly responsive to external stimuli in regulating their food consumption.

In summary, both VMH-lesioned rats and overweight humans tend to grossly overeat preferred foods but avoid unappetizing foods. Also like the VMH-lesioned rats, humans who are obese require fewer calories to gain and maintain weight (Bennett & Gurin, 1982, pp. 64-65).

In contrast to VMH-lesioned rats, rats that were surgically lesioned in the lateral hypothalamus (LH) dramatically decreased food-seeking behaviors. In the absence of endogenous activation (e.g., gastric motility and other internally originated signals to eat), it was necessary to repeatedly supply external food-related stimuli to even minimally reinstate eating (Wolgin, Cytawa, & Teitelbaum, 1976).

The behavior of these LH-lesioned rats is reminiscent of clinically reported behaviors of human patients suffering from anorexia nervosa. However, while anorectics would seem at first glance to abhor food, engaged as they are in self-starvation, they are paradoxically obsessed with food. Merely observing another person eat can make an anorectic feel as though she has consumed a "phantom meal." They are fascinated and preoccupied with all things related to food: recipes, menus, gourmet shops, and the like. Typically, they prepare elaborate, fattening foods for their families, but refuse to share in the meal (Sours, 1980, pp. 234-235). In many cases, the anorectic's state of deprivation makes her periodically susceptibile to the attraction of external food-related cues, which impel eating. At this point, the

anorectic may feel out of control, give up all restraints, and binge-eat. Once she regains control, the anorectic returns to self-deprivation and again, for a time, is able to resist cues to eat.

Thus, like LH-lesioned rats, anorectics can be lured into eating if cues are sufficiently powerful, but the effect is fleetingly salutary. The anorectic's powerful aversion to food ingestion ensures a quick return to self-starvation.

Based on the foregoing, it is tempting to suppose that hypothalamic impairment may underlie some eating disorders. However, as Bray (1976) points out, it is rare to find in humans internally induced, clinically diagnosable hypothalamic dysfunction. There are two crucial clinical markers of patients with anorexia nervosa, which are not present in the hypothalamically lesioned rats whose organic disorders mimic anorexia. These are the anorectic's predisposing rigid, perfectionistic cognitive "set" (which is conducive to dieting as a means to achieve control over self and an exaggerated ideal of thinness), and the family pattern of high expectations and over-closeness with the anorectic patient (Minuchin, Rosman, & Baker, 1978, pp. 51-63; Sours, 1980, pp. 319-330).

Caloric Intake and Expenditure

Common sense rightly tells us that if one consumes 2,000 calories in a day, 2,000 calories must be utilized in

order to maintain weight. It follows that to investigate variables that may influence weight, it is necessary to consider the relationship between calories consumed and calories expended in meeting the requirements of basal metabolism and physical activity. Unfortunately, for logistic reasons, such investigations are extremely difficult to accomplish.

In their review of investigations into obesity and exercise, Thompson et al. (1982) noted that while some researchers found obese children and adults exercising less than nonobese individuals, other researchers did not find a significant difference between groups. Thompson et al. assert that methodological variations--such as diverse measures of activity, general failure to convert measures of activity to caloric expenditure, and failure to adjust for the higher caloric cost of activity for obese subjects relative to normal weight subjects--may help to account for replication difficulties.

Interpretation of the exercise literature is further complicated by the probability that overweight individuals have attempted dieting more frequently, more stringently, and for longer periods than normal weight subjects. This is an important consideration, for each attempt to diet results in a decrease in BMR (Keys, Brozek, Henschel, Mickelson, & Taylor, 1950; Bray, 1970; Garrow, 1978; Wooley, Wooley, & Dyrenforth, 1979). The body interprets a diet as a famine state (Bennett & Gurin, 1982, pp. 84-85)

and reacts defensively, by slowing body metabolism to conserve fat stores and other resources. With each new weight loss attempt, the body becomes more efficient at storing fat, so that it requires fewer and fewer calories to cause a weight gain. Also, obese individuals tend toward hyperinsulemia, a condition that quickens the conversion of blood sugar into fat and leads to more frequent hunger (Rodin, 1981).

Recall the previously mentioned study of Griffiths & Payne (1976), which found that preobese children (i.e., nonobese children of obese parents) averaged 770 calories per day lower BMR than children of nonobese parents. It seems that individuals who are programmed to be fat start out handicapped by an impaired ability to burn calories. Add to this the probability that preobese children will most likely grow up to be dieting adolescents and adults (especially if they are women), and keep in mind that each diet attempt further decelerates metabolic rate. Proportionally more of any regained weight will be fat. With the accumulation of fat goes a tendency toward hyperinsulemia, which will cause more hunger. All in all, the prognosis seems quite discouraging for the overweight.

On a more hopeful note, recent investigations show that moderate exercise tends to decrease appetite and increase metabolic rate for hours afterward (Thompson et al., 1982). Exercise also increases the proportion of muscle to fat, and because muscle burns more calories than

does fat, this buildup increases overall metabolic rate. Thus, exercise offers both short-term and long-term increase of metabolism.

External Responsiveness and Eating

A great deal of obesity research has centered on the idea that the obese are hyperresponsive to external stimuli in feeding regulation. Rotter's (1966) concept of internal/ external locus of control was adapted by researchers investigating obese and normal weight differences in susceptibility to external food- and nonfood-related stimuli. Obese individuals, they hypothesized, were more responsive to environmental cues in initiating eating, whereas the nonobese seemed to attend more to internal cues such as gastric motility. Indeed, food intake by the obese was demonstrated to be influenced by perceived passage of time (Schachter & Gross, 1968), as well as by the sight and taste of food (Decke, 1971; Nisbett, 1968b, Nisbett & Storms, 1975). Nisbett (1968 a & b) found that prominent food stimuli elicited eating by the obese regardless of their hunger state. Obese subjects have also been shown to be more easily distracted from tasks than nonobese subjects (Rodin & Slochower, 1976).

However, other investigations determined that obese individuals did not as a rule change their internal/external responsiveness after substantial weight loss (Rodin, 1975; Rodin, Slochower, & Fleming, 1977).

Consequently, Rodin (1981) concluded that the internal/external split is simplistic, and has been over-applied and mis-applied by researchers of eating behaviors. Rodin concedes that some of the difficulties with replicating the normal weight/obese, internal/external differences discussed above may have been due to dissimilar experimental methods. Nevertheless, she asserts that the locus of eating control model, as it stands, is sorely inadequate in explaining eating behaviors.

The fact that weight loss does not necessarily change an individual's locus of eating control can be explained within a psychodynamic framework. According to Hilde Bruch (1958), a widely read psychodynamic theorist and therapist, external responsiveness can be learned preverbally by infants whose mothers respond indiscriminately to their expressions of need by giving food. These children never learn to differentiate the body's various urges and sensations. Consequently, they eat in response to disparate bodily signals including distress, anxiety and boredom. In the absence of, or in conjunction with, useful body signals, the child uses exoteric signals (e.g., the arrival of dinner time or availability of attractive foods) to determine eating onset and cessation.

Curiously, Nisbett (1972) and Rodin, Herman, & Schachter (1974) found that greatly obese individuals (defined as more than 40% over Metropolitan Life Insurance Company's 1977 weight standards) were no more responsive

than normal weight subjects to food- and nonfood-related stimuli. In fact, more externality is found among moderately overweight persons than among extremely obese and normal weight individuals. Nisbett (1972) suggested that many moderately obese individuals are below their biologically determined setpoint weight. However, he did not account for the fact that external responsiveness is also common in normal weight individuals, as is internal responsiveness in the moderately obese.

Rodin (1981) suggests that internal and external cues may interact in the regulation of eating. For example, external food-related cues may elicit internal, physiological signals that motivate eating. Conversely, externally cued anxiety and arousal may disrupt ongoing behavior, including self-control, thus disinhibiting chronically restrained impulses to eat. In other words, anxiety-based arousal may cause increased responsiveness to salient environmental stimuli.

Indeed, White (1973) found that obese subjects ate significantly more crackers than nonobese subjects after viewing films that produced distress arousal, humorous arousal, and sexual arousal, but not after viewing a "non-arousing" film. Rodin, Elman, & Schachter (1974) had obese and normal weight subjects listen to emotionally disturbing or "undisturbing" tapes, then report their perceived physiological and emotional states. Obese subjects consistently reported being more aroused than nonobese subjects

when listening to emotionally disturbing tapes, and less emotional in response to the "undisturbing" material. Additionally, obese subjects reported being more nervous than normal subjects in response to threats of painful electric shock, and delivery of painful shocks interfered more with their ability to learn a complex task. Herman & Polivy (1975) found that when made anxious, female subjects who were restrained eaters ate more and unrestrained subjects ate less.[3] Ruderman (1985a), also using female subjects, found that restrained eaters ate more when in a dysphoric mood than in a nondysphoric mood, and that unrestrained eaters consumed similar amounts in either mood state. In contrast, Schachter, Goldman, & Gordon (1968) found that while normal weight male subjects ate more when calm than when frightened, and also ate more when food deprived than when sated, obese subjects ate roughly the same amounts despite these experimental manipulations.

As Rodin (1981) asserts, the internal/external responsiveness paradigm is insufficient for explaining the many individual differences within obese and normal weight groups. Rodin argues that it is time to shift orientations toward investigating the etiology of the "several obesities

---

[3] "Restrained" eaters are those who attempt to control their weight by chronically restraining their impulses to eat. When their control is disrupted, restrained eaters tend to "counterregulate," that is, binge-eat. "Unrestrained" eaters are nondieters, who, after eating a large quantity, tend to compensate by simply eating less at a subsequent meal.

that all have fatness as their common observable characteristic" (p. 367). She suggests that restraint theory is one of several promising areas for continued research.

The Body's Resistance to Weight Change

Two remarkable investigations of human physiological and psychological response to weight fluctuations have provided fascinating insights into the effects of dieting and eating disorders.

The first is a study conducted over 35 years ago by Ancel Keys and others at the University of Minnesota (Keys, Brozek, Henschel, Mickelsen, & Taylor, 1950), investigating the effects of starvation. Keys and his colleagues used a subject pool of 36 young, healthy, psychologically normal, male conscientious objectors, who volunteered to participate in the study as an alternative to military service. Subjects were fed normally during the first three months of the study while their behavior, personality, and eating patterns were observed. In the next six months, subjects' calorie intake was reduced to approximately one-half of their former intake. During this period, subjects lost an average of 25% of their original body weight. The men then entered a three-month rehabilitation period, during which they were gradually re-fed. Keys et al. found that starvation affected striking changes in the physical, psychological and social functioning of the men. These effects

varied dramatically among individuals, and generally continued through the rehabilitation phase of the study.

During the starvation period, all subjects became preoccupied with food and food-related matters. For instance, the men primarily conversed, read, and fantasized about food and eating. Some suddenly became interested in collecting coffeepots and kitchen utensils; some began hoarding items, even nonfood-related articles. Subjects began to spend a great deal of their time planning how to eat their daily ration of food. They often felt conflicted between desires to ingest their food ravenously or to savor each bite.

After substantial weight loss, many subjects became unable to control their appetites. These subjects occasionally broke down and compulsively gobbled huge amounts of food, much to their self-disgust. One such individual, who worked in a grocery store, was eventually unable to resist the temptation of available food. After a binge, during which he felt frightened and out of control, he became exceedingly distressed and nauseous, and vomited. When confessing his slip, he was full of self-deprecation. Other subjects experienced similar slips and similar self-disgust.

As we have previously shown, anorectics present with a comparable obsession with food and tendency to transiently lose control and binge-eat. As we shall soon see, other

individuals who diet strenuously experience equivalent reactive states.

At the outset of the Keys et al. study, subjects were assessed as psychologically normal. Therefore, their later psychological states can be attributed to the effects of starvation.

Among many physiological alterations that Keys et al. observed in their subjects during the starvation period was a slowing of body processes, such as temperature, heart rate, respiration, and BMR. Notably, BMR dropped an average of approximately 40%, with the result that available calories were utilized much more slowly and turned to fat more readily than before. While average overall weight dropped 25%, body fat decreased almost 70%, and some 40% of muscle was lost. This loss of muscle was partially responsible for the body's slower burning of calories, because lean body mass burns more calories than does fat. During the rehabilitation phase when weight was regained, an inordinate proportion of the new weight was fat. By the eighth month of rehabilitation, subjects had reached approximately 110% of original body weight and 140% of original body fat, much of it in the abdomen and buttocks.

During the rehabilitation phase, when caloric intake was gradually increased, subjects commonly reported an increase in hunger immediately following a large meal. They found it very difficult to stop eating, resulting in huge daily calorie intakes and weekend binges ranging

between 8,000 and 10,000 calories. Subjects reported an inability to find a point of satiation. In the absence of reliable signals from their bodies to stop eating, they had to cognitively discipline themselves to stay away from food.

Despite the aberrant eating that resulted from their starvation, subjects typically gained back their original weight plus ten percent. By the end of the following year, most had returned to approximately their original weight.

Thus, it seems that the body fights significant deviation below a presumably "set" level, defensively utilizing an arsenal of physiological and psychological imperatives that impel eating and weight regain.

If a setpoint weight truly exists, deviations above a weight setpoint ought to result in corresponding psychological and physiological responses. Indeed, that was the case in a study of induced obesity by Sims, Goldman, Gluck, Horton, Kelleher, & Rowe (1968). Sims et al. used a group of normal weight prison inmates who volunteered to gain 20% to 25% of their original body weight over a six-month period. Subjects consumed 200% of their previous daily caloric intake. Despite their huge consumption, after the initial gains of several pounds, most found it exceedingly difficult to gain weight. Two exceptions were men with family histories of obesity or diabetes, who had a relatively easy time gaining weight.

Analogous to the subjects in Keys et al's. investigation, whose BMRs slowed in response to starvation, Sims et al. found increased BMRs among their overfed subjects. Subjects reported that they felt they were "burning up" and they perspired profusely. Their weight gains only accounted for some 25% of excess calories consumed. It was necessary to consume 50% more calories than their original consumption in order to maintain their higher weights. When allowed to eat as desired, subjects rapidly lost weight and almost to a man, stabilized at their beginning weights. The exceptions were two who had gained weight quickly, and two with family histories of obesity or diabetes.

The Keys et al. and Sims et al. studies, taken together, constitute graphic and substantial evidence that when threatened by deviations below or above a biologically pre-determined level, the body will fight to conserve weight.

Setpoint Theory

Now that we have appraised the effects of prolonged hunger and overfeeding, let us again consider the issue of internality and externality.

Noting the physiological and behavioral response similarities between hungry animals and overweight humans, Nisbett (1972) suggested that the responsiveness of obese humans to external cues might be the result of a chronic

hunger state. Both the food-deprived animal and obese humans are highly taste-responsive; both will consume more good tasting food while rejecting more bad tasting food than the non-hungry animal or the normal weight human; both are more emotional, less active, and less sexually motivated.

Nisbett (1972) concluded that relative responsiveness to internal versus external cues is a function of degree of deprivation. A constant state of deprivation causes the physiological and psychological responses characteristic of hungry organisms. In other words, the obese are chronically hungry.

Nisbett theorized that every organism has a weight setpoint, biologically determined and defended, which is directly related to the number of fat cells in the body. In turn, the number of fat cells is determined by heredity and early feeding patterns.

Nisbett's setpoint concept, while undoubtedly oversimplified, makes sense from an adaptive, evolutionary perspective. Substantial deviation below setpoint evidently triggers signals to conserve body resources, resulting in slowing of the metabolic process. After prolonged food deprivation and significant weight loss, the physiological drive for nutrition begins to be translated into psychological cravings for food. Depleted adipose cells release fatty acids which, when they reach a sensitive ratio with glycerol, seem to alert the hypothalamus that a

state of famine exists. Thus, physiological mechanisms are set in motion to cause the fat restorative behavior required by the adipose tissues. At that point, external food cues become highly salient, virtually irresistible. Eating restraint ultimately breaks down, food seeking becomes the dominant behavior, and one begins to eat a great deal. Ultimately, weight returns to setpoint. Once at setpoint, one is less responsive to external cues for eating initiation and cessation.

Setpoint theory nicely explains striking individual differences in eating styles and weights. An obese individual who is below weight setpoint may be more responsive to external controls and eat much more than an obese individual who is at or above setpoint. Normal weight individuals may likewise eat normally, undereat, or overeat as a function of their deviation from setpoint weight. It makes sense, then, that externality is not a prominent characteristic of the extremely obese (Rodin, 1981). These individuals presumably are at setpoint, and thus not susceptible to physiological and psychological pressures to eat and grow fatter.

It should be borne in mind that setpoint is not a tangible mechanism, but can only be inferred from such indirect evidence as weight history and fatty acid levels (Nisbett, 1972).

When applied to humans, setpoint theory is complicated by an important factor. Current fashion dictates that

individuals, especially females (Orbach, 1979; Allon, 1982), be leaner than nature may have intended. Those whose setpoints are unfashionably high commonly bow to social pressure and attempt to inhibit their eating despite food cravings. In this way, many females maintain weights below their natural body weights, at the cost of enduring a state of chronic hunger.

The Evolution of Fat and Thin

Why is there such a range of body types among humans, from gaunt to corpulent? Why are women proportionately much fatter than men, normally carrying some 20% to 28% body fat as opposed to males, who normally carry 14% to 18% (Bray, 1976; Bennett & Gurin, 1982, pp. 142-143)?

These are some questions raised by Bennett & Gurin (1982), who make a convincing case against dieting. The authors decry dieting to achieve a narrowly defined ideal weight. These attempts are doomed, they say, by the extreme individual variability in natural body weights, or setpoints, which resulted from natural selection within the human species.

Bennett & Gurin (1982, pp. 143-144) argue that fat is not the result of food abundance, as commonly supposed, but rather, evidence of millennia of famines and food scarcity. They cite evidence that the more food is available to a species and the more reliable the food source is, the leaner the species tends to be. For example, animals that

rely on seasonal fruits and vegetables, such as bears and raccoons, tend to get fat. However, animals living in climates that produce a year-round supply of food, such as monkeys and apes, stay lean. Because the food supply of these tree dwellers is fairly consistent, a reserve layer of fat would not be worth the energy required to carry it around.

The ability to fatten was crucial for human survival. Our ancestors survived and passed along their genes because their relatively high setpoints (i.e., fatness) and ability to conserve body resources (by slowing down metabolic rate) enabled them to survive periods of food scarcity.

It was particularly important for females to have high setpoints, in order to ensure adequate nutrition for developing fetuses and to nurse newborns, despite times of food scarcity. The ability to conceive was limited to those females whose fat stores were sufficient to carry a fetus to term and a newborn through the period of suckling. The need for a minimum fat level for childbearing--roughly 20% of body weight, at the least--is illustrated by female anorectics and marathon runners. Both of these groups commonly cease menstruation and consequently cannot conceive when their proportion of fat cells falls below this level.

Fatness is not a constant trait, as are height or color. Fat is stored around the body as an energy reserve available for use in times of food unavailability. An

adaptive organism may become fatter at one time and thinner at another. Thus, it is not the amount of body fat that is subject to natural selection, but rather the particular control system that manages the setpoint.

Bennett & Gurin (pp. 145-146) cite the ruby-throated hummingbird, weighing "less than a nickel," that twice a year journeys 600 miles across the Gulf of Mexico. Before each trip, the bird doubles its weight to supply the 23 calories worth of fat required for the journey. Once at its destination, the extra weight would be a handicap. Therefore, although food is readily available, the hummingbird only regains the fat lost in migration when it is time for the return trip. Thus, the hummingbird is not selected by nature to be either fat or thin, but rather, it has a setpoint mechanism that selects with exquisite precision appropriate weights for its migratory cycle.

According to Bennett & Gurin (p. 146), "A flexible setpoint, which responds to both activity levels and types of food in the diet, has given us [humans] the ability to colonize an endless variety of habitats and to exploit the food resources they have to offer."

There is general agreement among anthropologists that humans began as food-gatherers, consuming a variety of whatever the land and climate yielded--fruits, nuts, vegetation, insects, and possibly small animals. Bennett & Gurin speculate that as large omnivores seeking foods rich in calories and nutrients, our ancestors' setpoints were

highly responsive to the types of food they consumed. A species subjected to periods of food deprivation must be predisposed to eat lots of high-in-calorie, rich-in-nutrient foods when these are accessible. When sweet or fatty foods were available, our ancestors' setpoints would be raised, causing physiological and psychological urgings that would induce increased eating and increased adipose reserves. Thus, our species endured.

It is likely our earliest ancestors were slender--an assumption partially based on their relatively high-activity lifestyle--but physiologically predisposed, for protection against periods of scarcity, to get fatter when they encountered high-yield foods. Of course, the sweets they came upon were generally no more high-density than berries. Food gatherers' diets were usually exceedingly varied and the opportunity for excess was probably rare; therefore, the ability to fatten rarely if ever resulted in obesity.

As Bennett & Gurin lament (p. 152), "Nature had no way of knowing that one day cheesecake would be invented." Obesity became a widespread problem when people moved <u>en masse</u> from rural areas where food in its natural state was plentiful, to densely populated areas reliant on foods subjected to mass production and refining processes. The unfortunate result was unlimited availability of high-density sweets and fats.

A regrettable case in point is the modern-day Sioux tribe, with whom the author had an opportunity to work in 1984-1985, on the Fort Totten Sioux Reservation in North Dakota. The ancient Sioux traditionally lived off the land, hunting and gathering and travelling with the seasons. Tribal members were rarely if ever obese. The Fort Totten Sioux have lived for several generations on the federal reservation. They have radically altered their eating habits and consume a diet of high-density starches, fats and sweets, typical of the diet of many poor Americans. Current-day tribal members are commonly obese and the tribe is plagued with an epidemic level of diabetes.

Restraint Theory

Nisbett (1968b and 1972) observed that many overweight and some normal weight subjects alternately ate very small or very large amounts, as though a switch were turned on and off. Nisbett (1972) hypothesized that these subjects' current weight was below their individual, biologically determined setpoint, and they were consequently under biological pressure to eat food kinds and quantities to increase their weight to its setpoint. Nisbett (1968b) speculated that, "The relative potency of external versus internal controls is a dimension directly related to the degree of overweight."

Herman & Mack (1975) refined Nisbett's theory, predicting that this all-or-none eating style would be most characteristic of those who are conflicted between social pressure to be thin and biological pressure to be fat. These individuals, whom they called restrained eaters, alternately eat very little or a great deal, as they respond to one or the other constraint. In comparison, unrestrained eaters, who are at or near their setpoint weight and are not concerned about dieting, should tend to eat normally.

Further, Herman & Mack (1975) predicted that restrained eaters should eat more in the presence of attractive food cues (i.e., in an externally regulated manner) if their restraint was disinhibited by previous overeating. Unrestrained eaters, on the other hand, should respond to previous overeating by demonstrating internal regulation of subsequent intake.

Herman & Mack (1975) pointed out that corroboration of these predictions would not conclusively prove Nisbett's setpoint theory. They state (p. 649), "It would, however, substantiate the notion (directly derived from Nisbett's theory) that behavioral differences within the population of normal weight individuals ought to be expected; that such differences are related to chronic eating patterns that ought to affect the individual's position vis-a-vis his own set-point [sic]; and that such eating patterns, rather than extent of overweight per se, are perhaps better

predictors of behavior. Furthermore, this study bears specifically on the issue of the nature of the circumstances under which individuals indulge in what at least appears to be 'pathological overeating.'"

According to restraint theory, then, degree of restraint is a better predictor of overeating than is degree of overweight. Normal weight may not reflect a pattern of normal eating, but rather a favorable calorie intake/expenditure ratio over the long term. In other words, a normal weight restrained eater may maintain her weight by overcompensating for periods of overeating with extended periods of starvation. Restrained eating places the self-starving individual in a chronic state of external responsiveness. Once eating is initiated, external food cues become irrestible, restraint is easily broken, and binge-eating may result.

It might seem that an individual would first attempt dieting as a remedy for eating binges. The reverse is true (Polivy, Herman, Olmsted, & Jazwinski, 1984). Females commonly begin to diet in the attempt to bring their weight in line with fashion. Because most females were not meant by nature to be as thin as fashion prescribes, dieting eventually may bring weight below setpoint. The resultant physiological and psychological changes cause food cravings that ultimately result in <u>counterregulation</u>, that is, loosening of restraints and binge-eating. After a binge, the restrained eater attempts to compensate with even more

stringent restriction of intake. Thus, the diet-binge cycle has begun.

To a restrained eater, food is alternately--often simultaneously--highly attractive and extremely dangerous. Food represents nourishment, energy, sedative, social activity, social barrier, reward, punishment, comforter, tormentor, friend, adversary, and more (Orbach, 1979; Laskowitz, 1982). The restrained eater cannot hold a middle ground but is continually being pulled to one extreme or the other.

The restrained eater is terrified of losing control. She feels most in control when rigidly restricting her food intake. When successful, her self-esteem is relatively high. She feels competent, virtuous, and certain that this time her diet will be permanently and <u>perfectly</u> under control. However, food-deprived individuals tend to become aroused by environmental food cues. Inevitably, the perfectionistic restrained eater slips and eats more than the small amount "allowed." She is horrified, and perhaps paradoxically relieved, at having "lost" control and "blown the diet." Her tight restraint broken, she swerves to the opposite behavioral extreme and binge-eats. During the binge, to a degree depending on severity and duration, she may experience exaggerated feelings of loss of control, terror, guilt, humiliation, weight gain, distorted body image, self-disgust, and lowered self-esteem. Her overreaction forms a feedback loop with the overeating. The

worse she feels, the more obsessively she craves food as punishment and consolation. She has been through the cycle before and knows that sooner or later she must return to starvation rations. This knowledge serves to intensify the binge-eating, as she gorges in preparation for the inevitable famine to come. The stage is thus set for the next round of restraint and restraint breaking.

The use of the pronoun "she" in referring to the restrained eater reflects the fact that there are many more female than male restrained eaters, and that female restrained eaters report more distress due to overeating. Hawkins & Clement (1980) found that two-thirds of females and only one-half of males reported occurrences of binge-eating, and that females were much more concerned about their binge-eating. Fewer males reported feeling out of control during a binge or depression after a binge. Although males do counterregulate (Polivy, 1976), females tend to score higher on the Restraint Scale (Herman & Mack, 1975), another indication that males feel less pressure and guilt about their eating. A recent factor analytic study (ref. cited in Nisbett, 1978) suggests there are gender differences within the factor structure of the Restraint Scale. Thus, despite gross similarities in the process of eating disinhibition, subtle gender differences in experimental performance might be expected.

Tests of Restraint Theory

As previously recounted, Herman & Mack (1975) predicted that restrained eaters would eat more in the presence of attractive food cues (i.e., in an externally regulated manner) if their restraint was disinhibited by previous overeating. Unrestrained eaters should respond to previous overeating by demonstrating internal regulation of subsequent intake, that is, by eating somewhat less at a subsequent meal.

To test their predictions, Herman & Mack (1975) simulated a taste perception experiment, using 45 female restrained and unrestrained subjects, most of normal weight (12 subjects were obese). Subjects tasted either zero, one, or two 7.5-ounce milkshakes, as a preload intended to induce the perception of having overeaten. They next were asked to rate flavors of ice cream, tasting ad libitum from three large bowls. Restraint classifications were determined by scoring subjects' answers to ten questions from an eating habits questionnaire (Restraint Scale).

Consistent with Herman & Mack's hypothesis, restrained eaters who did not consume a preload milkshake before tasting ice cream (zero preload condition) ate much less than unrestrained eaters. However, after a preload of one or two milkshakes, restrained eaters counterregulated. Following a preload, unrestrained eaters ate less ice cream proportionate to the amount of milkshake consumed. That is, they regulated their subsequent intake to make up for

excess food consumption. Although relatively few obese subjects were included in this study, their responses were indistinguishable from those of normal weight restrained and unrestrained eaters.

Could the different responses of restrained and unrestrained eaters be attributed to distinctive cognitive responses, or did physiological reactions play a major role? A study by Polivy (1976) of 90 males indicated the counterregulation effect is triggered, initially at least, by cognition. In that study, regardless of actual preload calories consumed, subjects' subsequent eating was in accord with the number of calories they perceived having consumed. Restrained eaters counterregulated only when led to believe they had already overeaten (i.e., they believed the preload was high in calories), regardless of calories actually consumed.

Attempting to replicate Herman & Mack's (1975) findings, Woody, Costanzo, Liefer, & Conger (1981) manipulated taste conditions and calorie information. They tested 100 normal weight (under 15% overweight) women, using Herman & Polivy's (1975) Restraint Scale to identify restrained and unrestrained eaters. Restrained and unrestrained eaters either received no preload or consumed identical preload milkshakes, with one group told it was high in calories and a second group told it was low in calories. The preload and the ice cream subsequently taste-tested by all subjects were either good tasting or bad tasting (adulterated with

quinine sulphate). Both restrained and unrestrained eaters given the bad tasting ice cream ate small amounts. As Herman & Mack (1975) had before them, Woody et al. (1981) found the counterregulatory effect in restrained eaters after a preload, but this time with two qualifiers: counterregulation occurred only when the preload was believed to be high in calories and when the food was good tasting.

Further evidence that counterregulation is cognitively mediated was offered by Kirschenbaum & Tomarken (1982). They had 120 female restrained and unrestrained eaters, most of normal weight, consume a preload milkshake. Subjects had 15 minutes to rate identical amounts of ice cream presented in either small or large bowls, with calorie counts either labeled or unlabeled. The small bowl-labeled condition resulted in regulatory eating in all subjects, presumably because in this condition subjects were more conscious of their food intake. With only one of the two experimental manipulations in effect (i.e., small bowl-unlabeled or large bowl-labeled), restrained eaters used bigger spoonfuls to consume more ice cream than unrestrained eaters, and later underestimated their calorie consumption.

The studies described above included primarily normal weight subjects. Up to this point, no researcher had systematically compared obese and normal weight restrained and unrestrained subjects, even though obese individuals do

self-report as restrained or unrestrained. When obese restrained subjects were included in subject pools, they did not reliably counterregulate. This presented a challenge to restraint theory, which is of course based on the premise that both obese and normal weight restrained eaters will counterregulate when chronic restraints are disinhibited.

Before proceeding, let us note that there is no universally accepted experimental definition of obesity. No criteria are universally accepted, although the ideal weight table in Metropolitan Life Insurance Company's (1959) "Desirable Weights for Men and Women" is used widely as a referent. Many researchers have applied Metropolitan Life's median ideal weights based on height and a medium frame, for defining normal weight subjects. However, cutoff points for weight classifications have varied from researcher to researcher. For example, Herman & Mack (1975) classified as normal weight those subjects less than 10% above ideal weight, and as overweight those subjects more than 15% above ideal weight. Ruderman & Wilson (1979) considered subjects normal weight if less than 10% above the ideal weight, and obese if more than 10% above the ideal weight. Woody et al. (1981) considered as normal weight subjects less than 15% above the ideal weight, and as overweight those more than 15% over the ideal weight (and excluded them from participation in the study). Due

to such wide discrepancies in categorizing subjects according to weight, comparisons among studies can be difficult.

In two experimental attempts to resolve the question of whether the restraint dimension predicts eating behavior equally well for obese and normal weight subjects (Hibscher & Herman, 1977; Ruderman & Wilson, 1979), obese restrained subjects regulated their eating more in response to a preload than did normal weight restrained subjects. Using a within-subjects design, Ruderman & Wilson (1979) found that following a preload, obese and nonobese unrestrained eaters consumed similar amounts, but obese restrained eaters consumed less than normal weight restrained eaters.

To further investigate whether counterregulation is characteristic of the restrained obese, Ruderman & Wilson (1979) performed additional analysis on data from Hibscher & Herman's (1977) and Spencer & Fremouw's (1979) investigations. Their re-analysis of Hibscher & Herman's work showed that contrary to the prediction of restraint theory, after a preload both restrained and unrestrained obese subjects regulated their food consumption. However, unrestrained obese subjects regulated much more effectively than the restrained obese. In the normal weight groups, regulation and counterregulation occurred in the expected directions; that is, restrained eaters counterregulated and unrestrained eaters regulated their ice cream consumption. So, while restraint was a reliable predictor of eating behavior in both obese and nonobese groups, the predicted

directions differed. Only restrained normals showed true counterregulatory eating, while the restrained obese regulated their eating after a preload.

Ruderman & Wilson's re-analysis of Spencer & Fremouw's data also revealed the restraint dimension to be highly predictive of differences among normal but not among obese restrained and unrestrained eaters. As in the Hibscher & Herman study, true counterregulatory behavior was found only among restrained normals. In both studies, the restraint dimension predicted regulation rather than counterregulation in obese subjects.

Accordingly, Ruderman & Wilson concluded that since the obese ate only slightly more than normal weight subjects after a preload, counterregulation is characteristic of restrained normals, but is not a predominant response of the obese. It should be remembered, however, that Herman & Mack's (1975) small number of restrained obese subjects did counterregulate after a preload.

Recently, Ruderman & Christensen (1983) sought to replicate Ruderman & Wilson's (1979) post hoc finding of lack of counterregulation in the overweight, this time using a between-subjects rather than a within-subjects design. Subjects were classified as normal weight and overweight based on a cutoff point of 10% above ideal weight. Subjects were assigned to preload (milkshake) or no preload groups, and subsequently were allowed 10 minutes to rate three flavors of ice cream (vanilla, chocolate, and

peppermint, rather than Herman & Mack's vanilla, chocolate, and strawberry). They found that following a preload, unrestrained eaters demonstrated the predicted regulation. Restrained eaters did not counterregulate, but showed a nonsignificant trend to increase their eating following a preload. No evidence was found that the restraint dimension was associated with different eating patterns among normal weight and overweight subjects. A comparison of normal weight and overweight subjects without considering restraint scores showed that the preload significantly reduced the consumption of the overweight subjects, but did not affect the intake of the normal weight group. Ruderman & Christensen's (1983) post hoc tests of simple effects on the data previously collected by Hibscher & Herman (1977) showed a similar result. In view of their finding that restrained eaters showed a consistent but not significant trend to eat more after a preload, Ruderman & Christensen suggested that restrained eaters' counterregulatory style is a relative rather than an absolute phenomenon.

Self-consciousness As an Inhibitor of Eating

Ruderman & Wilson's (1979) and Ruderman & Christensen's (1983) conclusions that counterregulation is not characteristic of the overweight did not take into account an important factor. Social stigma against the obese is so strong that many overweight individuals feel ashamed and guilty for overeating (Allon, 1982). Over-

weight people may avoid overeating in public, where their consumption would be noticed by others. Even normal weight restrained eaters, concerned with controlling their weight, seeing themselves as fatter than they are, and frequently feeling out of control of their eating, might be highly self-conscious when eating in public.

In the studies re-analyzed by Ruderman & Wilson (1979) and in Ruderman & Christensen (1983), subjects were left alone with three large bowls of ice cream which would remain in plain sight on the table following the taste-test. Although encouraged to eat ad libitum, concerns that their consumption would be noticed might have caused over-weight restrained subjects, and perhaps some normal weight restrained subjects, to keep their eating in check. Unfortunately, there is no way of knowing what and how much subjects consumed after leaving the laboratory setting.

In addition, in an attempt to reinforce subjects' impression that the researchers were studying the influence of prior tastes on subsequent tests, Ruderman & Wilson (1979) required subjects to submit a record of all food eaten on the two experimental days. This manipulation might easily have produced the counterproductive effect of stimulating obese subjects' self-consciousness about their food consumption, thus reinforcing rather than disinhibiting their restraint.

Indeed, attempts to empirically assess eating in public places have repeatedly failed to produce evidence of

overeating in obese adults (Thompson et al., 1982). In a home observation study, Waxman & Stunkard (1980) did find that obese boys consumed more calories than did their nonobese brothers and peers. It is possible that when observed at home, obese boys were not self-conscious and therefore overate as they normally did. Alternatively, it may be that overeating is more easily observable in obese children than adults, simply because children have not yet learned to be ashamed of it.

Herman, Polivy, & Silver (1979) hypothesized that the presence of an observer while the subject was eating would induce self-consciousness and prevent eating disinhibition following a preload. As expected, while unrestrained eaters were unaffected, restrained eaters did not counter-regulate in the observer's presence. Instead, they behaved like the unrestrained eaters, eating more after a small preload and less after a large one. This conformance to social norms lasted only while the experimenter was present. When the observer left the room, restrained eaters, having exceeded their cognitive limit, counter-regulated.

Polivy, Herman, & Hackett (1980) manipulated self-consciousness in an attempt to prevent counterregulation of restrained eaters. All subjects were given a preload and were asked to taste-test candy given to them in wrappers. Control subjects threw their wrappers into a half-filled wastebasket <u>as they ate</u> and "taste-rated" the candies.

Subjects in the self-aware condition threw away their wrappers after they ate, into an already half-filled wastebasket. The assumption to be induced was that their consumption would not be noticed by the researchers. Subjects in the self-conscious and self-aware condition had no place to throw their wrappers and kept them on the table in front of them as they ate. Their presumed assumption was that the experimenter would see the wrappers and know how much had been eaten. The restrained eaters in the control group ate the most, while both experimental groups ate similar smaller amounts. Unrestrained eaters ate small amounts in all three preload conditions, but even they ate less in the self-conscious and self-aware condition.

Corroborating evidence for the inhibiting effects of a public setting come from two investigations by Polivy, Herman, Hackett, & Kuleshnyk (1984). In the first study, a wastebasket was used to induce a sense of privacy. Female restrained eaters ate the most candies after a preload. However, the addition of either self-attention or implied public attention (by supplying no wastebasket for disposing of candy wrappers) significantly inhibited consumption. Unrestrained eaters ate less candy after a preload, and still less under the condition of public attention.

In the second experiment, self-attention and public attention again inhibited cookie consumption of preloaded restrained eaters. However, preloaded unrestrained eaters were not influenced by the attention conditions, eating

less in all conditions. Non-preloaded unrestrained eaters did reduce their consumption in the two attention conditions.

A Test of Self-consciousness As an Inhibitor of Eating by Overweight Subjects

Merola (1984) investigated the effects of a private setting on post-preload eating by normal weight and overweight restrained subjects. She devised a seemingly private laboratory setting, intending to induce overweight restrained subjects to believe that their consumption would not be known to others, and thus to disinhibit post-preload eating.

She assigned 69 female subjects to low or high restraint groups; to normal or overweight groups; and to private or public settings. Subjects' restraint classifications were assessed by administering Herman's (1978) Revised Restraint Scale (RRS). Subjects were classified as normal weight if their weight was 6% or less over, and not more than 15% under, the median ideal weight for their height based on a medium frame, specified by Metropolitan Life Insurance Company's (1959) "Desirable Weights for Men and Women" table; and as overweight if 10% or more over the median ideal weight.

All subjects consumed a preload consisting of a 550 calorie (16 ounce) chocolate milkshake, which they were told contained 800 calories. The deception was intended to maximize subjects' feeling of having overeaten. As in the

prototypal restraint studies, all subjects were instructed to eat as much ice cream as necessary to make their taste-test ratings, and to feel free to help themselves to any remaining ice cream after completing the taste-test.

As was common procedure in most prior restraint studies, subjects in the "public setting" were required to leave the bowls containing any remaining ice cream on the table when they left the laboratory. Merola hypothesized that this manipulation would discourage counterregulation by overweight restrained subjects, by triggering their concern that the researchers would notice their consumption.

Subjects in the "private setting" were instructed to empty any remaining ice cream into a wastebasket after 15 minutes had elapsed, and then to join the experimenter in another room. The wastebasket already contained melted and melting ice cream which, unknown to subjects, was weighed before and after the session. The wastebasket manipulation was intended to promote subjects' belief that their intake would not be noticed, thus inducing the attitude hypothesized necessary for counterregulation by overweight restrained subjects.

Merola found no weight-restraint-setting interaction to support her hypothesis that overweight, restrained subjects will counterregulate in private but not in public, while normal weight restrained subjects will counterregulate in either setting. Accounting for the lack of inter-

action was the finding that the private setting resulted in much more eating by both normal weight and overweight restrained eaters, and even by unrestrained eaters of normal weight (88%, 86%, and 62% more, respectively), than in the public setting. The main effects for restraint and setting, but not for weight, indicate that for both overweight and normal weight subjects, public or private setting, as well as level of restraint, predict post-preload eating behavior. In fact, public or private setting was a stronger predictor of the amount eaten than was restraint classification.

T-tests performed on group means revealed that in the public setting, restrained normal weight subjects consumed more ice cream than unrestrained normal weight subjects, but not significantly more than overweight subjects, whether restrained or unrestrained. As expected, when compared statistically to other groups, restrained overweight subjects did not counterregulate in the public setting.

In the private setting, the restrained overweight group counterregulated compared with their analogue group in the public setting. However, no significant differences were found between consumption of the restrained overweight group in private and any other private setting group. This is partially explained by the increased private setting eating of restrained and unrestrained normal weight groups,

and by high variance within all restrained groups except the restrained-overweight-public setting group.

Merola's (1984) unexpected finding of increased private setting eating of unrestrained normal weight subjects corresponds with the previously discussed findings of Polivy, Herman, & Hackett (1980), and Polivy, Herman, Hackett, & Kuleshnyk (1984), and poses something of a challenge to restraint theory. By definition, unrestrained eaters do not restrain their food intake and should not be susceptible to disruptions of restraint to the point of counterregulation. It seems, though, that all but overweight unrestrained subjects practice some degree of restraint in contrived public eating situations, and that privacy disinhibits this behavior more predictibly than a preload alone. Because the author did not include a no-preload variable, no conclusions can be drawn about the relative predictive power of preload and setting.

It seems that restraint is not a discrete trait, to be diagnosed as "present" or "absent." Rather, dietary restraint is practiced to varying degrees by most people, and especially by women.

Indeed, Wooley & Wooley (1984), in a body image survey done for Glamour magazine, found that 75% of their 33,000 female respondents reported feeling too fat. Only 25% of these women were heavier than they should be, according to the Metropolitan Life Insurance Company (1959) tables of desirable weight. Forty-six percent of respondents re-

ported being self-conscious about their bodies; 88% had used moderate calorie restriction to control weight; and 58% had tried crash diets at some time.

Thus, many women who score below restraint scale cutoff points for classification as restrained eaters nevertheless may practice to some degree the cognitive and eating styles characteristic of the restrained eater.

In the private setting, normal and overweight restrained eaters showed inordinately high, strikingly similar variability (SD = 177.89 and 173.65, respectively). By contrast, variability in public setting eating of normal and overweight groups was dissimilar (SD = 111.53 and 60.34, respectively), with the least variability among overweight subjects, who ate relatively uniform, moderate amounts following a preload. This idiosyncratic pattern of variability may be related to two (or possibly three) factors.

First, individual differences in deviation from set-point weight would have resulted in varying levels of susceptibility and response to a preload. While these differences would be distributed similarly among public and private setting groups, self-consciousness would have prevented public setting subjects from acting on their counterregulatory impulses.

A second factor to consider is individual levels of self-consciousness in the public setting. Predictably, overweight restrained subjects' public setting behavior was

relatively homogeneous. Their restraint was strengthened, rather than disinhibited, by their self-consciousness about eating in a public setting. Thus, in public, self-consiousness would have taken precedence over overweight restrained subjects' urge to counterregulate. Their highly varied individual behavior in the private setting seems to more truly represent their varying susceptibilities (i.e., due to divergent setpoint deviations, lingering self-consciousness etc.) to a preload.

Third, the findings of Herman, Polivy, & Silver (1979); Polivy, Herman, & Hackett (1980); Polivy, Herman, Hackett, & Kuleshnyk (1984); and Merola (1984) provide additional evidence that self-consciousness inhibits food consumption. However, Polivy et al. (1979), Polivy et al. (1980), and Polivy et al. (1984) did not test overweight restrained eaters. Merola (1984) included an overweight restrained condition, but not a no-preload condition which might have indicated whether it was indeed self-consciousness or some unidentified variable that inhibited public setting post-preload counterregulation in overweight restrained eaters. A preload and no-preload comparison might also have shown whether the increased private setting eating of unrestrained normal weight subjects was preload-induced, or merely a matter of reduced self-consciousness.

In a pilot study using 48 preloaded subjects, Stein (1985) had subjects taste-test ice cream, while in a room that either contained an empty wastebasket (public setting)

or a wastebasket partially filled with melted and melting ice cream (private setting). He found that while privacy predicted counterregulation, level of restraint was not predictive of the amount eaten.

Thus, as in much of the research into eating disorders, investigations of restraint theory have been plagued by unexpected findings and unreliable results.

As previously suggested, restraint is not an easy construct to specify. Herman's (1978) Revised Restraint Scale (RRS) has generally been used by researchers of restrained eating, with a median split or a one-third split designating subjects as either restrained or unrestrained. In a personal communication, Polivy (1985) acknowledged the illogic of dichotomizing a trait that essentially exists on a continuum, but defended the practice citing the advantages of using the analysis of variance procedure for statistical analysis.

However, Stein (1987a) repeated the ice cream taste-test experiment, comparing results emerging from using the RRS as an artificial dichotomy (median split) versus as a continuous independent variable in a general linear model. He reported that when restraint was treated as a continuous independent variable, an important joint effect emerged that would have been overlooked in the artificial dichotomy model; and that a greater proportion of the variance could be accounted for. Stein concluded that the dichotomous

approach to restraint classification may be causing researchers to miss important information about relations among variables.

That matter notwithstanding, Ruderman (1983) has questioned the validity of the RRS. Her factor analyses of the RRS tapped a Weight Fluctuation dimension and a Concern with Dieting dimension. The Weight Fluctuation dimension comprises four items assessing weight fluctuations and maximum pounds overweight. These items are scored in pounds rather than percent of body weight, so that the larger the reported weight fluctuation, the greater the number of points scored. Thus, RRS scores for overweight individuals are likely to be inflated. Also, Ruderman (1985a) found that Concern with Dieting scores were more predictive of binge-eating behavior, while Weight Fluctuation was not at all predictive. Therefore, any given restraint score may be associated with less restraint for overweight subjects than for normal weight subjects.

A recently developed scale offers an alternative to the RRS. The Bulimia Test (Bulit), developed by Smith & Thelen (1984), is a 36-item scale that measures five main factors associated with bulimia (see Appendix I). The use of such a scale in restraint research makes intuitive sense, since bulimia represents an ideal <u>behavioral</u> model of extreme dietary restraint, with its problems of counter-regulation. The first factor measured by the Bulit pertains to actual binge behavior, as well as to the fear of

losing control of eating behavior. Factor 2 relates to feelings following eating binges. Factor 3 concerns vomiting behavior. Factor 4 relates to type of food preferred during a binge. Factor 5 concerns weight fluctuations. These five dimensions were shown by Smith and Thelen to reliably and validly predict bulimic versus normal group membership. Stein & Brinza (1987b) showed that this factor structure was reasonably stable across junior high, high school and college student samples.

In using the Bulit for diagnosing bulimia, Smith & Thelen (1984) employ a cutoff score of 102. They use a cutoff of 88 points to identify actual or incipient cases of bulimia before behavior patterns become chronic. In their standardization sample, 13% of subjects met this latter criteria. Not unexpectedly, the authors found that a subject's negative feelings about herself following a binge are as integral a feature of the bulimia syndrome as is binge behavior. In a sample of 635 university women, Stein (1987c) found a .83 correlation between the RRS and the Bulit. Taken together, the available data suggest that the Bulit may represent a viable alternative measure of restraint.

To summarize, Merola (1984) found increased post-preload eating among restrained normal weight, and overweight subjects in a private setting. However, increased private setting eating by unrestrained normal weight subjects lowered statistical power in group comparisons, and

presented a challenge to restraint theory. Statistical power was also lowered by high within-group variance among restrained eaters. The absence of a no-preload condition precludes stating unequivocally that self-consciousness, and not some other variable, inhibited counterregulation of restrained overweight subjects in past research, or that the increased eating of unrestrained normal weight subjects in the private setting was not preload-induced. Finally, it seems that RRS scores may have been inflated for overweight subjects. If so, overweight restrained eaters were actually less restrained than normal weight restrained eaters.

In the present study, an attempt was made to address these considerations.

Research Objective

If restraint theory is correct, normal and overweight restrained eaters should counterregulate when perceiving they have already overeaten, if attractive foods are subsequently available. When normal weight and overweight unrestrained eaters perceive they have overeaten, they should subsequently regulate their intake.

An attempt was made in this study to replicate findings of counterregulation in normal weight restrained groups (Herman & Mack, 1975; Polivy, 1976; Hibscher & Herman, 1977; Ruderman & Wilson, 1979; Spencer & Fremouw, 1979; Woody, Costanzo, & Liefer, 1981; Kirschenbaum &

Tomarken, 1981; Merola, 1984). Further, a private setting, as utilized by Merola (1984), was employed to reduce the self-consciousness that is presumed to inhibit counter-regulation of restrained overweight subjects.

Experimental Hypotheses

1. No-preload condition: All groups ([1] low restraint-normal weight-no preload; [2] low restraint-normal weight-preload; [3] low restraint-overweight-no preload; [4] low restraint-overweight-preload; [5] high restraint-normal weight-no preload; [6] high restraint-normal weight-preload; [7] high restraint-overweight-no preload; [8] high restraint-overweight-preload) should consume similar, relatively small amounts of ice cream.

2. Preload condition: Restrained subjects, both normal weight and overweight, should counterregulate, that is, they should consume more than those consuming no preload. Unrestrained subjects, both normal weight and overweight, should respond to the preload by consuming less than their analogue groups receiving no preload.

CHAPTER 2

METHOD

Subjects

To avoid the possibility of confounded results due to gender differences, only female subjects were included in this study. (As previously discussed, while both females and males have been shown to counterregulate, males score lower on the Revised Restraint Scale, and report experiencing less distress about their eating behaviors.) Subjects were undergraduate students solicited from psychology, sociology and nursing classes at the University of North Dakota. In return for their participation, subjects were given extra credit toward their course grade.

Volunteers were screened for participation by means of a questionnaire battery, which was administered in several group sessions. To ensure subject naivete, along with the Bulit (Smith & Thelen, 1984), which questions food-related attitudes and behaviors, subjects filled in several "decoy", non-food-related questionnaires, and were informed that they were being screened for participation in not one, but several studies.

The typical questionnaire battery was comprised of the Bulit scale and several "decoy" instruments. Restraint classifications were made according to scores on the Bulit.

The mean Bulit score of the initial group screening (N = 256) was 61, with a standard deviation of 18. Initially, subjects scoring at the mean (61) or lower were assigned to the low restraint groups; subjects scoring one standard deviation (79) or more above the mean were assigned to the high restraint groups. Later, after all subjects were run, the mean Bulit score of all respondents (N = 497) was calculated. This new mean was 62, with a standard deviation of 18. In order to maintain the criterion of one standard deviation above the mean for high restraint group assignment, the cutoff point for inclusion in the high restraint group was raised from 79 to 80; and subjects scoring at the new mean (62) or lower were considered low restraint. These new criteria did not necessitate discarding any previously run data. Using the new criteria, 15.8% of those tested qualified as high restraint eaters. The final cutoff point is only slightly lower than Smith & Thelen's (1984) suggested
score of 88 or more for the purpose of screening subclinical or incipient eating disorder cases.

Subjects were considered normal weight if their self-reported weight (which was verified after experimental participation) was 6% or less over, and not more than 15% under, the median ideal weight for their height based on a medium frame, according to Metropolitan Life Insurance Company's (1959 and 1977) "Desirable Weights for Men and

Women".[1] Subjects were classified as overweight if 10% or more over the median ideal weight.

To maximize differences between normal weight and overweight groups, overweight subjects were selected from the upper weight limits of the subject pool. On average, overweight subjects were 20% above their median ideal weight (SD = 9.62, range = +10.2% to +43.8%). Normal weight subjects' mean deviation from their median ideal weight was -2.2% (SD = 5.36, range = -14.8% to +5.6%).

A total of 113 subjects participated in this study. The mean age of subjects was 20.57 (SD = 3.69, range = 18-37).

Five of the eight cells contained 15 subjects. The no-preload-normal weight-restrained group contained 16 subjects. Due to subject pool limitations, only 11 per cell were obtained for the two unrestrained-overweight groups. These unrestrained-overweight subjects' mean age of 23.95 (SD = 5.45) was significantly older ($t[111] = -5.35$, $p < .0001$) than the mean age for all others (excluding unrestrained-overweight subjects) of 19.75 (SD = 2.56).

Merola (1984) encountered a similar difficulty locating unrestrained, overweight subjects. The mean age of her

---

[1] Although Metropolitan Life has published a revised weight chart, most previous studies have used the older versions. The revised version has stirred something of a controversy, with some researchers and clinicians protesting the higher allowable weights. For this reason, and to allow for comparability with previous studies, the 1959 and 1977 version was used in the present study.

eight unrestrained-overweight subjects was 27, higher than the overall mean age of 21.9.

Materials

Furnishings and equipment in the experimental room included the following: table; chair; plastic tablecloth; three .946L capacity stainless steel bowls; stainless steel tablespoon; paper napkin; 250 mL capacity paper cup containing cold water; wastebasket lined with a plastic trash bag; clock; 230 gms each of Bridgeman's brand ice cream in vanilla, chocolate, and strawberry flavors; three Flavor Rating Forms (one for each flavor); and a Flavor Preference Form.

A second room was used for weighing and measuring subjects upon completion of the taste-test. Equipment and materials in this room included a table; two chairs; bathroom-type electronic scale; wall-mounted tape measure; and various blank forms.

A third room was used for ice cream and milkshake preparation. Equipment in this room included a commercial ice cream freezer; two tables; stainless steel bowls; stainless steel tablespoons; blender; ice cream ladles; trays; ice cream; chocolate syrup; whole milk; paper cups; two gram-weight food scales, one small and one larger (the smaller for weighing bowls of ice cream, and the larger for weighing trash bags of ice cream).

Questionnaires used for screening were the following: the Bulit (Smith & Thelen, 1984); "Self-Evaluation Questionnaire (items compiled by the author); "Inventory of Premenstrual Symptoms" (compiled by the author); and "Menstrual Cycle Calendar (see Appendices II, III, and IV).

Design and Procedure

The experimental design was adapted from Herman and Mack's (1975) "ice cream taste-test" paradigm.

A three-way, between-subjects factorial design was used: 2 (low restraint vs. high restraint) X 2 (normal weight vs. overweight) X 2 (no-preload vs. preload). Once criteria for group assignments were met (i.e., restraint and weight), subjects were assigned randomly to no-preload and preload groups.

When contacted by telephone (see Telephone Instructions to Subjects, Appendix V) to schedule experimental participation, potential subjects were told the project was a sensory experiment, examining the effects of previous taste experiences on perception of subsequent taste experiences. Individuals were informed they would be asked to taste and rate flavors of ice cream, and were asked whether they were under any medical or dietary restrictions regarding consumption of sugar. (See transcript of telephone interview, Appendix VI.) Any potential subject answering in the affirmative was eliminated from the subject pool.

Those who agreed to participate were instructed to eat a "normal" meal two to three hours before arrival and to eat or drink nothing except water following that meal. Subjects were run between the hours of 12:00 p.m. and 6:30 p.m.

Subjects were seen by the author and two research assistants, all female. To help control for experimenter effects, insofar as possible only the author gave taste-test instructions; assistants interacted with subjects only after they had completed their experimental task. Experimenter's and assistants' contact with subjects was balanced among all groups. A male assistant telephoned potential subjects to schedule appointments and assisted in the food preparation room, but in order to control for possible inhibiting effects of interacting with a male experimenter, he had no face-to-face contact with subjects.

Experimenters were blind to the restraint classifications of subjects. Insofar as weight classifications were concerned, for obvious reasons the experimenters were not completely "blind." However, since they were blind to subjects' restraint group membership, knowledge or supposition of weight group membership created no likelihood of experimenter effects.

Subjects were run individually. On arrival, a subject was asked to sign a consent form (See Appendix VI). If she was assigned to a preload group, the milkshake was prepared while she read the consent form.

The subject was told that the purpose of the milkshake was to "be sure that prior to the actual taste experiences, all subjects in the experiment will have been exposed to the same taste and volume of intake" (see Preload Instructions to Subjects, Appendix VII). To maximize her feeling of having overeaten, she was informed that the milkshake contained 800 calories, the "approximate equivalent of a filling meal." The 500 mL preload actually contained approximately 550 calories. (The recipe is as follows: one cup whole milk [150 cals.]; one cup vanilla ice cream [260 cals.]; three tablespoons chocolate syrup [140 cals.].) Subjects were led to believe the calorie value was higher than it actually was because, as discussed in Chapter 1, restrained eaters tend to counterregulate more markedly when believing they have eaten a very high-calorie preload (Polivy, 1976; Spencer & Fremouw, 1979; Woody et al., 1981).

After consuming the preload, or after signing the consent form if she was in the no-preload group, the subject was taken to the experimental room. On the table in the room were the following: one bowl (230 gms net weight) each of vanilla, chocolate, and strawberry ice cream; typed Taste-Test Instructions (see Appendix VIII); one cup of cold water; one paper napkin; one stainless steel tablespoon; three Flavor Rating Forms (see Appendix IX), one for each flavor, consisting of a seven-point Likert scale for rating sweetness, sourness, saltiness, bitterness, creami-

ness, and flavor; a Preference Rating Form (see Appendix X); a clock; and a cassette tape recorder.

In addition, the room was equipped with a wastebasket lined with an opaque plastic trash bag containing melted and melting ice cream. Before each subject's arrival, the wastebasket was filled with no less than 300 gms and no more than 1300 gms of melted and melting ice cream, made to look as though it had been emptied from bowls by previous subjects.

Recorded taste-test instructions were played, as the subject read along from the typed sheet. The subject was directed to taste and rate one ice cream flavor at a time, in the specific order presented (vanilla, strawberry, chocolate), and to record all ratings for one flavor before tasting the next. The instructions stressed the importance of making careful, accurate ratings and directed the subject to consume as much of each flavor as necessary to make her ratings. Water could be consumed as desired. Subjects were to stay in the room for all of 15 minutes, "since we must be certain everyone has the same amount of time." After playing the recorded instructions, the experimenter announced the time indicated by the clock on the table, as well as the time it would be in 15 minutes, and instructed the subject to meet her in another room at precisely that time. As she was leaving the room, the experimenter casually informed the subject that once she had made her ratings she should, "Feel free to help yourself to any

leftover ice cream, since we'll only be throwing it away. And before you leave the room, would you please empty any remaining ice cream into the wastebasket."

When the subject reported to the appointed room after the tasting session, rating forms were collected and height and weight measurements were taken. The subject was asked to sign a mailing list to receive a debriefing letter, and was given a credit voucher for participation in the experiment, to be presented to her course instructor.

After the subject departed, the wastebasket was retrieved from the experimental room. The plastic bag was weighed and final weight was subtracted from beginning weight, to calculate grams of ice cream consumed by the subject.

The experimental design is in compliance with the ethical principles of psychologists set by the American Psychological Association (1981).

CHAPTER 3

RESULTS

It was hypothesized as follows: (1) that high restraint subjects of both weight groups would counterregulate following a preload; (2) that low restraint-normal weight subjects would consume less ice cream following a preload than in a non-preloaded state; and (3) that low restraint-overweight subjects would consume the same amount regardless of preloading.

To test the hypotheses, a 2 (restraint) X 2 (weight) X 2 (preload) analysis of variance (ANOVA) was performed, using grams consumed as the dependent variable. A Cochran's test of homogeneity of variance revealed that the groups were homogeneous.

Table 1 presents the results of the ANOVA. Significant main effects were found for restraint ($F[1, 112] = 8.08$, $p = .006$) and for preload ($F[1,112] = 3.66$, $p = .055$). A nonsignificant trend toward a main effect was evident for weight ($F[1,112] = 2.04$, $p = .153$). No interactions were revealed, although a nonsignificant interactional trend was yielded for restraint X weight ($F[1,112] = 2.29$, $p = .129$).

Table 1

Three-Way Analysis of Variance of Mean Grams of Ice Cream Consumed

Source	SSQ	df	MS	F	p
Restraint (A)	79625.6	1	79725.6	8.077	.0056
Weight (B)	20103.46	1	20103.46	2.037	.1528
Preload (C)	36139.94	1	36139.94	3.661	.055
A X B	22621.15	1	22621.15	2.291	.129
A X C	15025.26	1	15025.26	1.522	.218
B X C	3743.25	1	3743.25	.379	.547
A X B X C	14156.32	1	14156.32	1.434	.232
Residual	1036404	105			
Total	1227919	112			

A Newman-Keuls test was performed to compare treatment means. This post hoc test revealed that, as expected, the low restraint-normal weight subjects ate much less when preloaded than when non-preloaded (ps < .05). Counter to expectation, there were no other significant differences between groups.

Table 2 and Figure 1 highlight the results. As expected, low restraint-normal weight subjects ate much less after consuming a preload (M = 100.27 gms, SD = 61.63) than when non-preloaded (M = 221.4 gms, SD = 111.26). Low restraint-overweight subjects also behaved as anticipated, consuming similar, small amounts whether in the no-preload condition (M = 128.63 gms, SD = 55.83) or preload condition (M = 109.91 gms, SD = 73.48). In contrast, high restraint eaters' behavior was antithetic to the expectations of this study, as well as to the predictions of restraint theory. Not only did normal weight-high restraint subjects and overweight-high restraint subjects fail to counterregulate following a preload, but they ate slightly (not significantly) less following a preload than without one. Normal weight-high restraint subjects ate a mean of 206.69 gms (SD = 120.4) when non-preloaded, and 169.27 gms (SD = 113.78) after a preload; overweight-high restraint subjects ate a mean of 181.8 gms in the no-preload condition, and 156.93 gms when preloaded.

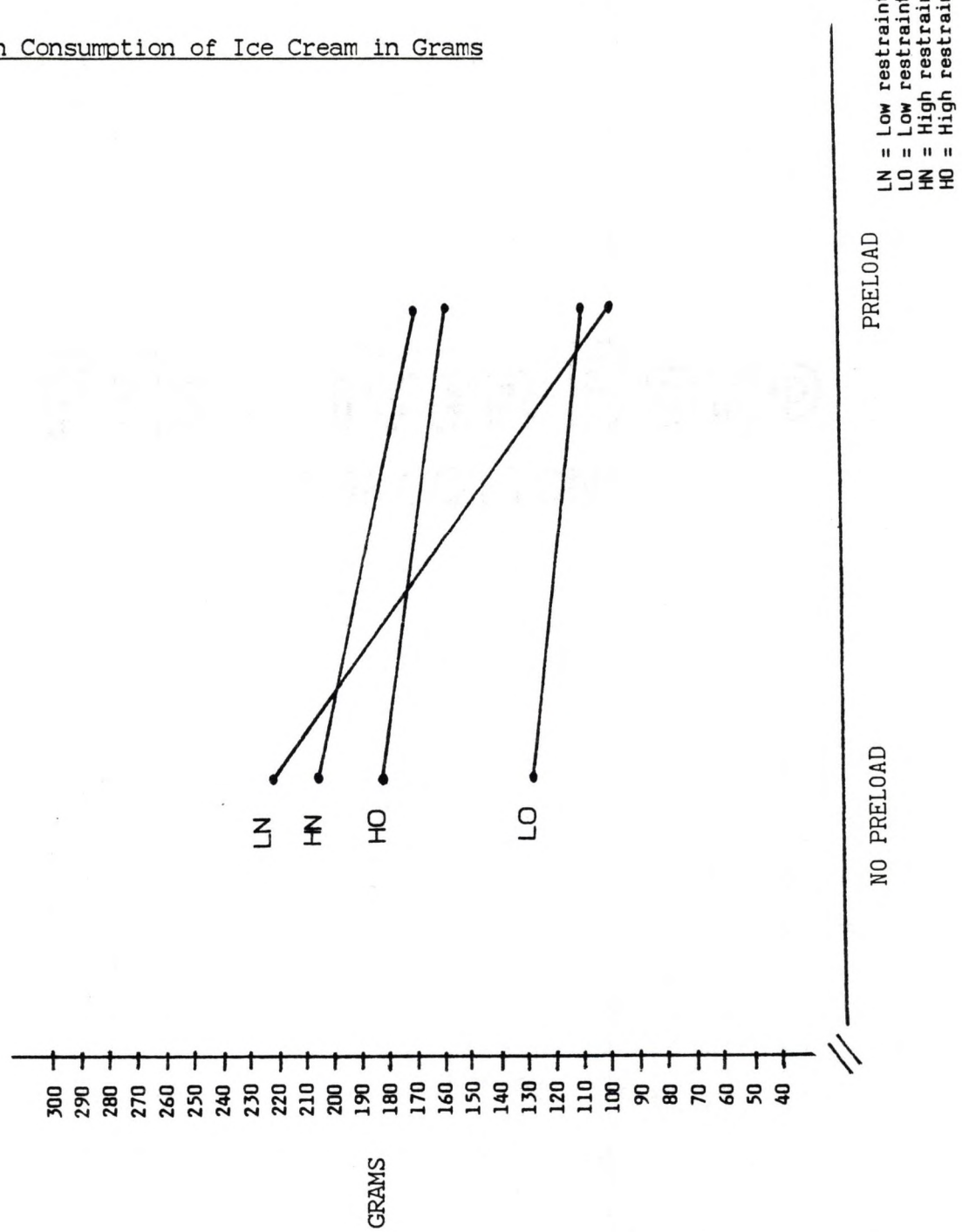
Table 2

Mean Consumption of Ice Cream in Grams

		Normal Weight		Overweight	
		No Preload	Preload	No Preload	Preload
Low Restraint	N=	15	15	11	11
	M=	221.4	100.27	128.63	109.91
	SD=	111.26	61.62	55.83	73.48
	Var=	12378.97	3798.64	3117.25	5398.69
High Restraint	N=	16	15	15	15
	M=	206.69	169.27	181.8	156.93
	SD=	120.4	113.78	112.95	102.63
	Var=	14494.63	12947.78	12756.74	10533.9

Figure 1

Mean Consumption of Ice Cream in Grams



Because several subjects had eaten a good deal more than the others, it was decided to re-analyze the data with these outliers eliminated from the data set. Therefore, subjects scoring more than 2.5 standard deviation points above the mean were eliminated. This resulted in five subjects being dropped, for a new total N of 108.

Table 3 presents the results of the new ANOVA. Although the levels of significance changed slightly, there were no changes in effects. Again, main effects were yielded for preload ($F[1, 107] = 5.6$, $p = .02$) and restraint ($F[1, 107] = 4.2$, $p = .04$), and no interactions were found. A nonsignificant interactional trend was observed in weight by preload ($F[1, 107] = 2.8$, $p = .098$). Bartlett's ($p = .176$) and Cochran's ($p = .159$) tests of homogeneity of variance revealed that the data were homogeneous.

Table 4 highlights the revised descriptive data yielded by discarding the five outliers. In comparing these data with the original data presented in Table 2, it can be seen that all but one of the outliers was in a no-preload group; none was in a low restraint-overweight group. Discarding the outliers' data lowered the mean amount eaten by non-preloaded-low restraint-normal weight subjects from 221.4 to 187.57, and dramatically lowered the standard deviation from 111.26 to 64. The mean for non-preloaded-high restraint- normal weight subjects lowered from 206.69 to 190.64, and the standard deviation fell

Table 3

Three-Way Analysis of Variance of
Mean Grams of Ice Cream Consumed
With Outlying Data (>2.5 S.D. Removed

Source	SSQ	df	MS	F	p
Restraint (A)	28457.67	1	28547.67	4.2	.043
Weight (B)	8662.34	1	8662.34	1.28	.261
Preload (C)	37745.48	1	37745.48	5.57	.02
A X B	1163.47	1	1163.47	.17	.68
A X C	6287.58	1	6287.58	.93	.338
B X C	18905.68	1	18905.68	2.79	.098
A X B X C	1555.57	1	1555.57	.23	.633
Residual	575331.75	100			
Total	678109.54	107			

Table 4

Mean Consumption of Ice Cream in Grams
With Outlying Data (> 2.5 Standard Deviations) Removed

		Normal Weight		Overweight	
		No Preload	Preload	No Preload	Preload
Low Restraint	N=	14	15	11	11
	M=	187.57	100.27	128.63	109.91
	SD=	64.	61.62	55.83	73.48
	Var=	4096.	3798.64	3117.25	5398.69
High Restraint	N=	14	14	14	15
	M=	190.64	149.36	160.21	156.93
	SD=	111.03	86.84	78.81	102.63
	Var=	12327.66	7541.19	6211.02	10533.9

slightly from 120.4 to 111.03. The mean for preloaded-high restraint-normal weight subjects fell from 169.27 to 149.36, with a standard deviation adjustment from 113.78 to 86.84. The mean for non-preloaded-high restraint-over-weight subjects dropped from 181.8 to 160.21, with a standard deviation drop from 112.95 to 78.81.

A multiple regression analysis performed on the new data (see Table 5) also yielded significance for preload and restraint, but not for weight.

Table 5

Multiple Regression Analysis of
Mean Grams of Ice Cream Consumed
With Outlying Data (>2.5 S.D. Removed

Variable	B	SE B	BETA	T	p
Preload (1)	-19.861639	7.947411	-2.33236	-2.499	.014
Restraint (2)	15.679570	7.947411	.183873	1.973	.051
Joint (1 X 2)	8.785284	7.947411	.103184	1.105	.271
(Constant)	148.672647	7.947411		18.707	.000

CHAPTER 4

DISCUSSION

If they had behaved in accord with restraint theory, unrestrained subjects would have regulated their eating subsequent to a preload, and restrained subjects would have counterregulated. That was not the finding of this study. Surprising enough was the failure of high restraint subjects--both normal weight and overweight--to counter-regulate after a preload. Moreover, they changed the directional trend of their consumption, eating less rather than more after a preload, as though making an--albeit weak--effort (after the manner of low restraint-normal weight subjects) to normalize their eating. In short, this study's restrained subjects showed not a tendency to counterregulate, but rather a failure to regulate.

Although main effects were found for restraint and preload, the hypothesized interaction between restraint and preload did not occur. The main effect for restraint is particularly telling. High restraint subjects, who supposedly are dieting much of the time, ate more under all conditions independent of preload or body weight. In other words, the preload was not a necessary factor in precipitating eating; high restraint subjects regularly tended to

eat more than low restraint subjects. This suggests that precipitators such as preload, negative mood states, and alcohol intake are not always prerequisite for the paradoxical eating behavior of high restraint subjects. Indeed, high restraint subjects' self-avowal of weight fluctuation, tendency to overeat, feelings that food is controlling their lives, and other issues relevant to distress over dieting, seem to be behaviorally reflected quite clearly in the laboratory eating paradigm used here.

The control group of low restraint-normal weight subjects did conform to the predictions of restraint theory and the precedent of prior restraint studies. They ate less than half as much after consuming a preload as when non-preloaded. In effect, when offered ice cream on an empty stomach, these nondieting normal weight subjects freely indulged; but after consuming a presumably high calorie milkshake, they sensibly regulated by eating minimal amounts of ice cream.

Low restraint-overweight subjects likewise conformed to precedent, if not to logic: They were curiously unaffected by prior consumption, opting to eat little whether preloaded or not. Over all groups, subjects' weight was unrelated to the amount of food eaten (r = .0101, p = .459).

It is perhaps most remarkable about restraint studies to date that while low restraint subjects have consistently responded as expected, high restraint subjects have not.

Most, but not all, restraint studies described in Chapter 1 did result in counterregulation by preloaded, normal weight, high restraint subjects; and Herman & Mack's (1975) small number of overweight restrained subjects counterregulated following a preload. But Ruderman & Christensen's (1983) normal weight and overweight restrained eaters increased their eating only nonsignificantly after a preload. Spencer & Fremouw's (1979) reanalysis of Hibscher & Herman (1977) showed that only normal weight restrained subjects counterregulated, while overweight subjects (regardless of restraint classification) regulated their eating, albeit low restraint subjects did so much more effectively. Franchina (1987) also reports that restraint status is a poor predictor of amount of food eaten in laboratory eating paradigms.

Why did the present study (among others) fail to find counterregulation among even its normal weight restrained eaters, despite the fact that the phenomenon has been replicated in many studies? This question leads ineluctably to another question: Is the present data anomalous?

We cannot answer by comparing the data with data from most prior studies, because the use of a "private" setting distinguishes this investigation from most prior studies. Fortunately, Merola's 1984 study did utilize a private setting variable. Thus, it is practicable to compare data of equivalent groups from that and the present study. A

finding of similar consumption would demonstrate that the present data is precedented, and therefore not anomalous.

The present study and the 1984 study shared two (out of three) independent variables, restraint and weight. In the present study, all subjects were tested in a private setting and four groups were given a preload. In the earlier study, all groups were preloaded and private setting was a variable common to four groups. Thus, four out of eight groups from each study are essentially identical, enabling comparison of consumption of each of the present study's four preloaded groups with its analogue private setting group from the 1984 study. Each group from the current study was compared with its analogue group from the prior study, as follows: low restraint-normal weight-preload with low restraint-normal weight-private setting; low restraint-overweight-preload with low restraint-overweight-private setting; high restraint-normal weight-preload with high restraint-normal weight-private setting; and high restraint-overweight-preload with high restraint-overweight-private setting. Table 6 presents means and standard deviations of these groups.

T-tests of differences between analogue groups revealed one trend toward significance ($p = .106$), wherein the high restraint-normal weight group in the current study consumed slightly less than their analogue group in the earlier study. There was no difference between high restraint-overweight groups. Thus, the private setting

Table 6

Means and Standard Deviations of Comparable Groups from Merola, 1984 and Merola, 1987

Merola, 1984		compared with	Merola, 1987	
Low restraint- Normal weight- Private group	M = 122.859 SD = 57.819		Low restraint- Normal weight- Preload group	M = 100.27 SD = 61.62
High restraint- Normal weight- Private group	M = 243.385 SD = 177.887		High restraint- Normal weight- Preload group	M = 169.27 SD = 113.78
Low restraint- Overweight Private group	M = 91.366 SD = 84.861		Low restraint- Overweight Preload group	M = 109.91 SD = 73.48
High restraint- Overweight Private group	M = 174.957 SD = 173.652		High restraint Overweight Preload group	M = 156.93 SD = 102.63

data from the two studies are roughly similar, enough so to demonstrate that the present data are not anomalous.

Both this and the prior study (Merola, 1984) utilized as subjects female undergraduates and nursing students at the University of North Dakota. UND students' ethnic backgrounds include a large Norwegian, Swedish, and German representation, along with Polish, Russian, American Indian (especially Sioux), and less than six percent black, Asian, and other minorities. Many of these students were raised in small towns and rural areas and are newly on their own, living in apartments or dormitories in the small city of Grand Forks. This student population seems not unlike that of many universities where restraint research is conducted (e.g., University of Toronto, University of Illinois at Chicago, University of Wisconsin at Madison, University of Cincinnati, Duke University at Durham, North Carolina, and West Virginia University at Morgantown), and there is no apparent reason to question the generalizability of the results based on demographics of the subject pool.

It is possible, of course, that errors in experimental design or implementation are accountable for the results of both investigations. The design of the two studies was adapted from Herman & Mack (1975), the prototype restraint study design which has been utilized with variations by many researchers. This researcher's principal modifications were, in the 1984 study, inclusion of a private setting as an independent variable and, in the present

investigation, use of a private setting for all subjects. Could the private setting have somehow influenced the failure to counterregulate? It will be remembered that in the 1984 study, restrained groups counterregulated after a preload when tested in a private setting (even normal weight nondieters ate slightly more in private) but not in a public setting; and that in the present investigation, high restraint groups ate similar amounts regardless of preloading, while normal weight nondieting subjects ate half as much when preloaded. Since privacy elicited more eating by restrained subjects in the former study, one could not convincingly argue that it somehow inhibited their eating in the later study. Even common sense dictates that privacy should disinhibit more deviant, not more normal, eating behavior. Indeed, Herman, Polivy, & Silver (1979) and Polivy, Herman, Hackett, & Kuleshnyk (1984) also found post-preload disinhibition of eating among normal weight restrained eaters in a private setting, along with reduced consumption under conditions of self-attention or public attention.

Except for independent variable manipulations, specifics of this experiment were similar but not necessarily identical to the prototype design (Herman & Mack, 1975). In comparing features of various restraint studies, no two are identical. Modifications to the prototype study have been freely made, presumably with the intent of improving the design or because of practical impingements. For

example, some have allowed 10 minutes for the taste-test, and others have allowed 15 minutes. Various weight cutoffs have been adopted to determine normal weight and overweight. Either the RRS or the Bulit has been used to assess restraint. Here also, cutoff points have varied; usually researchers have used a median split or an upper and lower one-third split of scores of the particular subject pool. Some researchers have used pudding, crackers, or sandwiches instead of a milkshake and ice cream. When milkshakes and ice cream have been used, amounts presented to subjects have varied within a small range. For the most part, applicable instructions have been similar across studies. These and other variations in restraint study designs, while unavoidable, make comparisons problematic and might be responsible in unknown ways for divergent findings.

As described in Chapter 2, the author personally administered or closely supervised all phases of the study. Assistants were well trained and monitored. To control for experimenter effects, assistant contact with subjects was limited and counterbalanced. Any confounding effects due to the author's contact with subjects were likewise evenly distributed among groups and presumably did not contaminate the high restraint groups' data. Given the overall variation in restraint study designs, nothing in the design and implementation of this study seems remarkably deviant from the prototype restraint study.

Neither can the behavior of this study's restrained subjects be attributed to using the Bulit to assess restraint. As previously shown, Merola (1984) obtained comparable results using the RRS; and Stein found .83 correlation between the RRS and the Bulit. Also, because the 36-item Bulit has a much smaller factor loading attributable to weight fluctuation than has the 10-item RRS, the Bulit should not yield the inflated restraint scores accompanied by less concern for dieting among overweight subjects that Ruderman (1983 and 1985) cited as a problem with studies using the RRS. Indeed, in this study there was no correlation (r = .15, p = .182) between subjects' percent of deviation from ideal weight and degree of restraint. One might also speculate that because the Bulit screens for bulimia, the resultant subject pool of restrained eaters might contain many subjects who anticipated vomiting after eating, and therefore the results might not reflect true consumption. However, there was no correlation between vomiting sub-scores and amount eaten (r = .097, p = .158), or between vomiting sub-scores and Bulit scores (r = .606, p = .146). In support of the Bulit, it can be argued that bulimia represents an excellent model of problematic dieting, restraint and counterregulation.

Is it possible that the recent popular awareness of healthy eating and the dangers of dieting accounts for restrained eaters' failure to counterregulate in this study? To wit, perhaps many former restrained eaters are

now eating with a more normal, less restrained style but are still inordinately fearful of losing control of their eating. While this explanation is not inconceivable, it strains the imagination.

More likely, some seemingly restrained eaters may not eat in a truly "restrained" manner, i.e., with predictable sequences of marked restraint and counterregulation. These quasi-restrained eaters may obtain high restraint scores largely because they manifest the idiosyncratic cognitive style of the restrained eater. As Ruderman (1985b) has shown, the rigid and perfectionistic restrained eater perceives her eating in absolute terms ("I have perfect self-control/I have no self-control."). This cognitive style is reflected in a distorted eating image (comparable to the well-documented distorted body image of individuals with eating disorders). The restrained eater sets unrealistic dieting standards and regards even small digressions as catastrophic failures. These "failures" precipitate counterregulation. Quasi-restrained eaters, on the other hand, may perceive their eating in the all-or-none manner characteristic of "true" restrained eaters; they undoubtedly exhibit some restrained and counterregulatory behaviors, but only moderately, erratically, or in circumscribed situations. Two recent findings support this idea.

First, Crandall (1986) reported fascinating data concerning a recently surfaced phenomenon among college

sorority women. The most popular sorority sisters binge together occasionally as a social activity; they report they feel bad afterward but, unlike restrained eaters, know they can stop if they have to. To the degree that their now-occasional binging bouts are interspersed with distress and compensatory food restriction, these young women may be restrained eaters "in training". Thus, social bingers--who admit to eating binges, guilt, and possibly other cognitive similarities to "true" restrained eaters, but do not exhibit marked restrained eating patterns--might achieve inflated restraint scores. When put to the test, these quasi-restrained subjects would tend to eat normally or in a manner approaching normal, as we may have witnessed in the presumed regulatory attempts of restrained eaters in the present study.

Second, Franchina (1987) found that preloaded restrained subjects salivated more than unrestrained subjects when presented with additional food, yet failed to counter-regulate. Thus, even when restrained eaters are in a state of physiological (and presumably, cognitive) readiness to eat, they often maintain their restraint.

However, the hypothesis is weakened by the fact that the Bulit is considered to yield a low false-positive rate. In addition, in this study, while only mild correlation was found between Bulit score (i.e., restraint score) and consumption (r = .13, p = .091), and there was no correlation between Binge sub-scale scores and consumption (r =

.092, p = .172)., there was a strong correlation between Feelings sub-scale scores and consumption (r = .191, p = .024). Thus, overeating was predicted not by a history of binge behaviors, but by experience of negative feelings concerning perceived overeating. Is it paradoxical, or merely ambiguous, to suggest that feelings (i.e., cognitions) about eating are predictive of counterregulation, and that non-counterregulating subjects' high restraint scores are more attributable to restrained eater-like cognitions than to binge behaviors? The issue warrants further study.

Let us consider the instruments used to measure restraint. Both the Bulit and the RRS are heavily loaded with questions that require subjective answers about subjects' eating, or seek "factual" information without defining terms (e.g., asking about diet frequency without defining a diet). For example, from the Bulit: "I am satisfied with my eating patterns." "I am afraid to eat anything for fear that I won't be able to stop." "I feel sad or blue after eating more than I'd planned to eat." From the RRS: "How often are you dieting?" Would a weight fluctuation of 5 lb. affect the way you live your life?" "Do you give too much time and thought to food?" This is not a criticism of these instruments. Restraint always entails a cognitive stance, which only intermittently results in marked behaviors. It is correct that this

substantial cognitive component be reflected in the assessment instruments.

However, it seems that any given restraint score is best interpreted as diagnostic of a cognitive stance (possibly with physiological correlates or predispositions), but not necessarily as diagnostic or predictive of eating behaviors.

In summary, it seems that we have eliminated as possible explanations of restrained subjects' failure to disinhibit their eating (1) anomalous data; (2) subject pool aberrations; (3) unexpected influences due to the private setting; (4) experimenter influences; (5) effects of variations in experimental design; and (6) artifacts produced by using the Bulit. Possible contributing factors are related to the likelihood that restraint scores are more indicative of a cognitive stance than they are predictive of counterregulatory eating.

Another Look at Restraint Studies

In the many restraint investigations to date, the behavior of nondieting subjects has generally been in accord with the predictions of restraint theory. However, among overweight restrained subjects (and even among normal weight restrained subjects in recent studies), failure to counterregulate has seriously undermined restraint theory. Some contributing variables may still be unaccounted for. Nevertheless, at this point, one must question whether dis-

ordered eating can be validly studied under usual experimental conditions.

Restraint theory focuses in linear fashion on a single aspect of disordered eating, the restraint-counterregulation phenomenon. This linear approach is the basis of the experimental method, often invaluable for identifying one or more factors contributing to an event. However, disordered eating is a complex, context-dependent phenomenon. In forcing a fit within the constraints of the experimental method, are we destroying the essential reality and consequently missing more than we find?

On examining the several restraint studies in which dieters demonstrated preload responses other than counterregulation, one may wonder whether restraint theory neglects a repertoire of eating behaviors utilized by the restrained eater. Indeed, the restrained eater is in a sense misnamed, for her attempts to cope with her eating impulses embody more than pathological, failed attempts to self-starve. As we may have witnessed in this study, many restrained eaters also employ relatively healthy behaviors, such as attempts at true regulation.[1]

It might be a useful undertaking to longitudinally study and compare subjects' behavioral repertoires. Every

---

[1] The striving to model regulatory eating can be translated into clinical technique. For example, a therapist might work to increase and strengthen this and other adaptive behaviors that exist, however weakly, in a patient's behavioral repertoire.

person is characterized by an idiosyncratic network of factors, all interacting and transfiguring in an ongoing, interrelational process. How one responds to stimuli at any given time is not necessarily how one will respond next time. Eating--like most behaviors--is a continuing process, not a definitive response that can be accurately measured once and for all. One's eating today is additively and interactively affected by what one ate yesterday and what one anticipates eating later; by one's learning history, psychodynamic history, and social history; by long- and short-term physiological processes; by the eating of companions; by the public or private nature of the setting; by self-awareness; by attempts at self-control; by food-related cues; by current dieting status; and more.

A subject's perceptions, fantasies and emotions concerning her eating may be as important as what she eats in the laboratory. For example, some restrained subjects might consider they had binged just by having the milkshake. Some may not presently be dieting, while others may be in different phases or intensities of a diet, and consequently more or less likely to eat right then. Subjects who have not recently eaten sugary foods may experience the sugar load of the milkshake or ice cream as distasteful and eat little. A preload might not immediately disrupt some dieters' restraint, but might result in a binge after leaving the laboratory. Some may experience the preload as a disinhibitor but wait until leaving

the laboratory to indulge, perhaps planning to binge on a preferred food. Others may eat a great deal in the laboratory, intending to eat little for the rest of the day. Some may be late afternoon or night bingers and less susceptible to restraint-breaking manipulations at other times. Others' binge-eating may be stylized, occurring only in specific places, under specific conditions, involving specific foods. Even the phase of the menstrual cycle may have a bearing on consumption, for evidence indicates that women's carbohydrate consumption is greater following ovulation (i.e., premenstrually) than before ovulation (Dalvit-McPhillips, 1983).

It bears reiterating that, in merely measuring eating on a one-shot basis in a laboratory, researchers may miss more than they find. In an ideal world, a restraint researcher would take a complete diet and weight history, assess recent and current diet status, recent food intake (e.g., is eating on the increase or decrease?), perceptions regarding the experiment and laboratory eating, and plans and thoughts about later eating. Our ideal-world researcher would somehow follow each subject (literally or figuratively) for several days or more after she leaves the laboratory, monitoring her cognitions and consumption to more truly assess the effects of an experimental manipulation.

The Puzzle of the Low Restraint-Overweight Subject

The low restraint-overweight subjects are an enigmatic lot, for several reasons. Very little data exist regarding this group. Being older than the typical university population (their mean age is 23.96, compared with all other subjects' mean age of 19.75), they are difficult to recruit as subjects. Their eating behavior seems curiously independent of the factors that influence other groups. Regardless of public or private setting or previous consumption, when offered their fill of free, readily available ice cream that "will only be thrown away" if not eaten, they have remained impervious and eaten only small amounts. Most puzzling of all is that these overweight women, in a culture that essentially treats fat women as pariahs, do not report distress or concern with restricting food intake in the service of dieting.

These observations prompt several questions: Why are low restraint-overweight subjects older than other subjects? Why are they uniquely unaffected by previous consumption and external food-related stimuli? To what, if not to overeating, is their overweight attributable? These questions can at best be answered speculatively here. Nevertheless, they are important to raise, if only because the answers may cast light on other groups' eating styles.

Let us begin with the question of these low restraint-overweight subjects being older than the typical college student. The obsession with thinness has predominantly

characterized urban upper and middle classes. Only in recent years has weight obsession seeped to remaining facets of our society (Benson, 1984). It may be that non-dieting overweight subjects were simply born before fads about weight and dieting began in their environs. One might argue that there are few nondieting overweight subjects of college age because at that age, most females have not yet "given up" the attempt at slimness and accepted their bodies as is. This reasoning is admittedly specious. It is not credible that 24-year-old women (roughly the mean age of low restraint-overweight subjects in this investigation), who were only a few years ago concerned about their weight and dieting, now are as unconcerned and consistent in their low consumption as nondieting overweight subjects seem to be.

We will again raise the question of age as it relates to the next question. Let us now consider these overweight nondieters' seeming indifference to their previous consumption and to food-related stimuli. Early socialization is a likely contributor here. It is conceivable that many low restraint-overweight individuals were raised in rural or ethnic subcultures where fatness is more prevalent and acceptable. They would hence have accepted their heaviness, never learning the ethos conducive to attempting the thin middle- and upper-class standard.

It is likewise conceivable that their early environments were unusually supportive and secure, enabling them

to withstand the larger society's prejudice against fatness and remain unconcerned with restricting their eating.

Another possible answer is suggested by a finding of O'Neil, Paine, Riddle, Currey, Malcolm, & Sexauer (1981). These authors observed that juvenile-onset obese subjects scored higher on the RRS than adult-onset obese individuals. They also found a correlation between duration of obesity and restraint; present age, percent overweight, and absolute body weight were not related to restraint. In light of overweight-unrestrained subjects' being older, it is interesting to note that the subjects found by O'Neil et al. to be adult-onset were significantly older than the juvenile-onset obese subjects. It may be that many low restraint-overweight subjects became obese as adults. Adult-onset obese individuals would be less accustomed to considering themselves overweight and being considered overweight by others. Consequently, they may not have internalized a fat self-image, and they would probably be less accustomed to focusing on and restricting food intake. This explanation accounts for the relative lack of concern with dieting, and seeming indifference to previous consumption and external food-related stimuli. The foregoing speculations could be straightforwardly tested by surveying subjects' backgrounds and early relationships.

Alternatively, it is conceivable that these subjects are at their setpoint weight and have never dieted below that point; consequently, they would not be under physio-

logical pressure to eat. Unfortunately, despite convincing arguments for the existence of a weight setpoint, the construct is difficult to concretize, and can only be inferred from indicators such as weight history, fatty acid levels, and metabolic changes accompanying weight fluctuation. Recall, however, Rodin's (1981) report that externality is rarely found among the extremely obese. If low restraint-overweight subjects presented with more extreme obesity than high restraint-overweight subjects, it would make sense to infer that the nondieters are indeed at their setpoint weight, while the dieters are below theirs, thus neatly explaining the difference in susceptibility to external cues. This is not the case, however. A *t*-test between unrestrained and restrained overweight groups in this study showed no difference in deviation from ideal weight. Thus, no conclusions can be drawn about low restraint-overweight subjects' relationship to setpoint weight.

The Future of Restraint Theory

In brief, restraint theory and restraint research seem to need rethinking. Questions remain to be asked and answered. Are current measures of the restraint construct valid? How does restrained eating relate to bulimia and anorexia nervosa? How do cognitive, behavioral, psychodynamic, social, physiological, and genetic variables interact in normal and disordered eating? To better test

theoretical assertions, experimenters need to build into their designs some approximation of the complex reality of normal and disordered eating. In fact, researchers may need to move out of the laboratory and "into the closets." If it is not yet time, it soon will be, to subsume restraint theory under a more encompassing theoretical explanation of disordered eating.

APPENDIX I

THE BULIT[1]

Answer each question on the following pages by filling in the appropriate circles on the computer answer sheet. Please respond to each item as honestly as possible; remember, all of the information you provide will be kept strictly confidential.

1. Do you ever eat uncontrollably to the point of stuffing yourself (i.e., going on eating binges)?
   (a) Once a month or less (or never)
   (b) 2-3 times a month
   (c) Once or twice a week
   (d) 3-6 times a week
   +(e) Once a day or more

2. I am satisfied with my eating patterns.
   (a) Agree
   (b) Neutral
   (c) Disagree a little
   (d) Disagree
   +(e) Disagree strongly

3. Have you ever kept eating until you thought you'd explode?
   +(a) Practically every time I eat
   (b) Very frequently
   (c) Often
   (d) Sometimes
   (e) Seldom or never

4. Would you presently call yourself a "binge eater"?
   +(a) Yes, absolutely
   (b) Yes
   (c) Yes, probably
   (d) Yes, possibly
   (e) No, probably not

5. I prefer to eat:
   +(a) At home alone
   (b) At home with others

---

[1] Reprinted by permission of the authors. First published as Smith & Thelen, 1984.

(c) In a public restaurant
(d) At a friend's house
(e) Doesn't matter

6. Do you feel you have control over the amount of food you consume?
(a) Most or all of the time
(b) A lot of the time
(c) Occasionally
(d) Rarely
+(e) Never

7. I use laxatives or suppositories to help control my weight.
+(a) Once a day or more
(b) 3-6 times a week
(c) Once or twice a week
(d) 2-3 times a month
(e) Once a month or less (or never)

8. I eat until I feel too tired to continue.
+(a) At least once a day
(b) 3-6 times a week
(c) Once or twice a week
(d) 2-3 times a month
(e) Once a month or less (or never)

9. How often do you prefer eating ice cream, milkshakes, or puddings during a binge?
+(a) Always
(b) Frequently
(c) Sometimes
(d) Seldom or never
(e) I don't binge

10. How much are you concerned about your eating binges?
(a) I don't binge
(b) Bothers me a little
(c) Moderate concern
(d) Major concern
+(e) Probably the biggest concern in my life

11. Most people I know would be amazed if they knew how much food I can consume at one sitting.
+(a) Without a doubt
(b) Very probably
(c) Probably
(d) Possibly
(e) No

12. Do you ever eat to the point of feeling sick?
+(a) Very frequently
(b) Frequently
(c) Fairly often
(d) Occasionally
(e) Rarely or never

13. I am afraid to eat anything for fear that I won't be able to stop.
+(a) Always
(b) Almost always
(c) Frequently
(d) Sometimes
(e) Seldom or never

14. I don't like myself after I eat too much.
+(a) Always
(b) Frequently
(c) Sometimes
(d) Seldom or never
(e) I don't eat too much

15. How often do you intentionally vomit after eating?
+(a) 2 or more times a week
(b) Once a week
(c) 2-3 times a month
(d) Once a month
(e) Less than once a month (or never)

16. Which of the following describes your feelings after binge eating?
(a) I don't binge eat
(b) I feel O.K.
(c) I feel mildly upset with myself
(d) I feel quite upset with myself
+(e) I hate myself

17. I eat a lot of food when I'm not even hungry.
+(a) Very frequently
(b) Frequently
(c) Occasionally
(d) Sometimes
(e) Seldom or never

18. My eating patterns are different from eating patterns of most people.
+(a) Always
(b) Almost always
(c) Frequently
(d) Sometimes
(e) Seldom or never

19. I have tried to lose weight by fasting or going on "crash" diets.
(a) Not in the past year
(b) Once in the past year
(c) 2-3 times in the past year
(d) 4-5 times in the past year
+(e) More than 5 times in the past year

20. I feel sad or blue after eating more than I'd planned to eat.
+(a) Always
(b) Almost always
(c) Frequently
(d) Sometimes
(e) Seldom, never, or not applicable

21. When engaged in an eating binge, I tend to eat foods that are high in carbohydrates (sweets and starches).
+(a) Always
(b) Almost always
(c) Frequently
(d) Sometimes
(e) Seldom, or I don't binge

22. Compared to most people, my ability to control my eating behavior seems to be:
(a) Greater than others' ability
(b) About the same
(c) Less
(d) Much less
+(e) I have absolutely no control

23. One of your best friends suddenly suggests that you both eat at a new restaurant buffet that night. Although you'd planned on eating something light at home, you go ahead and eat out, eating quite a lot and feeling uncomfortably full. How would you feel about yourself on the ride home?
(a) Fine, glad I'd tried that new restaurant
(b) A little regretful that I'd eaten so much
(c) Somewhat disappointed in myself
(d) Upset with myself
+(e) Totally disgusted with myself

24. I would presently label myself a "compulsive eater" (one who engages in episodes of uncontrolled eating).
+(a) Absolutely
(b) Yes
(c) Yes, probably
(d) Yes, possibly
(e) No, probably not

25. What is the most weight you've ever lost in 1 month?
+(a) Over 20 pounds
(b) 12-20 pounds
(c) 8-11 pounds
(d) 4-7 pounds
(e) Less than 4 pounds

26. If I eat too much at night I feel depressed the next morning.
+(a) Always
(b) Frequently
(c) Sometimes
(d) Seldom or never
(e) I don't eat too much at night

27. Do you believe that it is easier for you to vomit than it is for most people?
+(a) Yes, it's no problem at all for me
(b) Yes, it's easier
(c) Yes, it's a little easier
(d) About the same
(e) No, it's less easy

28. I feel that food controls my life.
+(a) Always
(b) Almost always
(c) Frequently
(d) Sometimes
(e) Seldom or never

29. I feel depressed immediately after I eat too much.
+(a) Always
(b) Frequently
(c) Sometimes
(d) Seldom or never
(e) I don't eat too much

30. How often do you vomit after eating in order to lose weight?
(a) Less than once a month (or never)
(b) Once a month
(c) 2-3 times a month
(d) Once a week
+(e) 2 or more times a week

31. When consuming a large quantity of food, at what rate of speed do you usually eat?
+(a) More rapidly than most people have ever eaten in their lives
(b) A lot more rapidly than most people
(c) A little more rapidly than most people
(d) About the same rate as most people
(e) More slowly than most people (or not applicable)

32. What is the most weight you've ever gained in 1 month?
+(a) Over 20 pounds
(b) 12-20 pounds
(c) 8-11 pounds
(d) 4-7 pounds
(e) Less than 4 pounds

33. (Females only.) My last menstrual period was
(a) Within the past month
(b) Within the past 2 months
(c) Within the past 4 months
(d) Within the past 6 months
(e) Not within the past 6 months

34. I use diuretics (water pills) to help control my weight.
+(a) Once a day or more
(b) 3-6 times a week
(c) Once or twice a week
(d) 2-3 times a month
(e) Once a month or less (or never)

35. How do you think your appetite compares with that of most people you know?
+(a) Many times larger than most
(b) Much larger
(c) A little larger
(d) About the same
(e) Smaller than most

36. (Females only.) My menstrual cycles occur once a month:
(a) Always
(b) Usually
(c) Sometimes
(d) Seldom
+(e) Never

APPENDIX II

SELF-EVALUATION QUESTIONNAIRE

DIRECTIONS: Read each statement and then blacken in the appropriate circle on your computer form, to indicate how you generally feel. There are no right or wrong answers. Do not spend too much time on any one statement but give the answer which seems to describe how you generally feel.

	Never	Sometimes	Often	Always
1. I feel pleasant.	A	B	C	D
2. I tire quickly.	A	B	C	D
3. I feel like crying.	A	B	C	D
4. I wish I could be as happy as others seem to be.	A	B	C	D
5. I feel rested.	A	B	C	D
6. I am "calm, cool and collected."	A	B	C	D
7. I worry too much about things that don't matter.	A	B	C	D
8. I am happy.	A	B	C	D
9. I am inclined to take things hard.	A	B	C	D
10. I feel secure.	A	B	C	D
11. I try to avoid facing a crisis or difficulty.	A	B	C	D
12. I feel blue.	A	B	C	D
13. I take disappointments so keenly that I can't put them out of my mind.	A	B	C	D

14. I become tense and upset when I think about my present concerns.	A	B	C	D
15. Some unimportant thought runs through my mind and bothers me.	A	B	C	D

APPENDIX III

INVENTORY OF PREMENSTRUAL SYMPTOMS

The following are symptoms some women experience from one day to two weeks before their periods. Please indicate how affected you are by each symptom, by blackening on your answer sheet the appropriate letter following each statement.

<u>One day to two weeks before my menstrual period:</u>

	Never	Some-times	Often	Always	Don't Know
1. I seem to get moodier.	A	B	C	D	E
2. Things bother me that usually do not.	A	B	C	D	E
3. I feel like crying more often than usually.	A	B	C	D	E
4. I lose my temper more easily than usually.	A	B	C	D	E
5. I say or do things I later feel sorry about.	A	B	C	D	E
6. Sweet foods (e.g., cake, candy, ice cream) seem more appealing.	A	B	C	D	E
7. Carbohydrates (e.g., bread, potatoes) seem more appealing.	A	B	C	D	E
8. I seem to crave more salt on food.	A	B	C	D	E

APPENDIX IV

MENSTRUAL CYCLE CALENDAR

ID #_____(Experimenter use)

Name _____

Phone_____

In females, variations in hormone levels occur throughout the menstrual cycle. These hormone level variations may affect moods and perceptions, and we may need to control for them.

Please indicate on the calendar below, the day or days you expect your menstrual period to begin. Since this is sometimes difficult to estimate, you may wish to indicate a range of 3-5 days. All we need is an estimate.

FEBRUARY/MARCH (circle one)

1	2	3	4	5	6	7	8
9	10	11	12	13	14	15	16
17	18	19	20	21	22	23	24
25	26	27	28	29	30	31	

What is your height?_____

What is your weight?_____

What is your age? _____

APPENDIX V

TELEPHONE INSTRUCTIONS TO SUBJECTS

This is _____(name)_____ from the Psychology Department at the University of North Dakota. You filled in some questionnaires for participation in research. I am calling you to schedule your participation in a research project that would take less than one hour. In return for your participation, _____(specify)_____. Do you have any time available on (day and date)?

You will be participating in a perception experiment. Specifically, we are interested in the effects of previous taste experiences on perception of subsequent taste experiences. You will need to eat a normal meal two to three hours before your appointment time and eat or drink nothing, except water, following that meal. When you arrive, you will be asked to sample flavors of ice cream and rate them as to flavor, texture, preference, and so on. We are interested in how taste perception is affected by the order in which flavors are sampled.

Before we set an appointment time, are you under any medical or other restrictions regarding sugar intake that would prevent you from participating? (If yes, discon-

tinue.) Do you have a pencil? (Set time.) Come to the Psychology Building, Corwin-Larimore Hall, Room 417, fourth floor.

Please remember to eat a normal meal two to three hours before your scheduled time. After that, eat or drink nothing except water. Also, it is very important that you remember your appointment, and that you arrive on time. If you have any problems, please notify us 24 hours in advance. Here are some numbers to call if you have any problems. (Give phone numbers.) Alright, then, we will see you at (time) on (day and date). Thanks.

APPENDIX VI

CONSENT FORM FOR PARTICIPATION IN EXPERIMENT ENTITLED "INFLUENCE OF PREVIOUS SENSORY EXPERIENCE ON SUBSEQUENT PERCEPTION IN SAME SENSORY MODALITY"

1. RESEARCH PROCEDURE: This experiment conforms to the ethical guidelines of the American Psychological Association. You will be asked to taste and rate three flavors of ice cream, with or without having first tasted and consumed a specially prepared milkshake. Upon completion of the experiment, you will be mailed a detailed description of the research and group results. At that time, you will have an opportunity to learn your individual results.

2. DISCOMFORTS AND RISKS: There are no known physical risks associated with participation in this experiment. However, if you are under medical restrictions regarding sugar intake (e.g., diabetics), you must make this known to the experimenter, and you <u>should not</u> participate in this study. While no substantial discomforts are anticipated, you are encouraged to discuss with the experimenter any problems or discomforts encountered as a result of participation (call Geraldine Merola, at 777-3451).

3. FREEDOM TO WITHDRAW: You may elect to terminate your participation at any time, without penalty.

4. CONFIDENTIALITY: All information and results are strictly confidential. To preserve your anonymity, your name will not be associated with experimental results, unless you later request to be told your individual result.

5. COMPENSATION FOR PARTICIPATION: Your participation in this research project should normally take less than one hour. You will receive a credit slip for participating, worth one credit toward your course grade.

6. QUESTIONS: If you have any questions concerning this project, please call Geraldine Merola, at 777-3451.

7. Your signature below indicates that you have read the information above and agree to participate in this research project. You will receive a copy of this consent form. Thanks.

_____	_____
Name (print)	Witness
_____	_____
Signature	Date

APPENDIX VII

PRELOAD INSTRUCTIONS TO SUBJECTS

As you know, we are interested in the way characteristics of one flavor affect the perception of subsequent flavors. To be sure our findings are valid, we must control for previous flavors which participants have been exposed to. This is a specially prepared milkshake containing 800 calories, the approximate equivalent of a filling meal. Please consume the entire shake. That way, we can be sure that prior to the actual taste experiences, all subjects in the experiment will have been exposed to the same taste and volume of intake. Take your time. I will be back soon.

APPENDIX VIII

TASTE TEST INSTRUCTIONS

We are interested in the effects of previous taste experiences on perception of subsequent taste experiences.

1. Please taste and rate the flavors one at a time, in the order presented: #1 Vanilla; #2 Strawberry; #3 Chocolate. You may drink as much or as little of the water as you wish while you make your ratings.

2. Record your ratings on the flavor rating forms provided.

3. Complete your rating for one flavor before moving on to the next.

4. Once you have rated a flavor and begun sampling the next flavor, do not return to previous flavors.

5. Do not change previously recorded ratings once you have moved on to the next flavor.

6. It is very important that your ratings accurately reflect your perceptions. Sample as much or as little of each flavor as necessary to make careful, accurate ratings.

7. Drink as much water as you wish while making your ratings.

8. You have 15 minutes to accomplish your ratings. When the 15 minutes have elapsed--but not before then, please, since we must be certain everyone has the same amount of time--take all of your rating sheets to the experimenter in Room ___.

Thank you.

APPENDIX IX

FLAVOR RATING FORM
VANILLA/CHOCOLATE/STRAWBERRY (circle one)

Circle the appropriate choice for each:

1. SWEETNESS

1	2	3	4	5	6	7
Not at all SWEET						Extremely SWEET

2. SOURNESS

1	2	3	4	5	6	7
Not at all SOUR						Extremely SOUR

3. SALTINESS

1	2	3	4	5	6	7
Not at all SALTY						Extremely SALTY

4. BITTERNESS

1	2	3	4	5	6	7
Not at all BITTER						Extremely BITTER

5. CREAMINESS

1	2	3	4	5	6	7
Not at all CREAMY						Extremely CREAMY

6. GOODNESS

1	2	3	4	5	6	7
Not at all GOOD						Extremely GOOD

APPENDIX X

PREFERENCE RATING FORM

Rate each flavor in order of preference. #1 = most preferred;
#3 = least preferred.

Example:

SWEETNESS LEVEL PREFERENCE

Vanilla	3
Chocolate	1
Strawberry	2

1. SWEETNESS LEVEL PREFERENCE

Vanilla	_____
Chocolate	_____
Strawberry	_____

2. CREAMINESS PREFERENCE

Vanilla	_____
Chocolate	_____
Strawberry	_____

3. FLAVOR PREFERENCE

Vanilla	_____
Chocolate	_____
Strawberry	_____

4. OVERALL PREFERENCE

Vanilla	_____
Chocolate	_____
Strawberry	_____

REFERENCES

Allon, N. (1982). The stigma of overweight in everyday life. In B. B. Wolman (Ed.), Psychological aspects of obesity (pp. 130-174). New York: Van Nostrand Reinhold.

American Psychological Association. (1981). Ethical principles of psychologists. American Psychologist, 36, 633-638.

Benson, A. (1984, January 19). "Anorexia and Bulemia," a Colloquium presented by the Center for Study of Anorexia and Bulemia, for Bellevue Hospital, New York, New York.

Bennett, W., & Gurin, J. (1982). The dieter's dilemma: Eating less and weighing more. New York: Basic Books, Inc.

Bjorntorp, P. (1976). Effects of physical conditioning in obesity. In G. A. Bray (Ed.), Obesity in perspective (Fogarty International Center Series of Preventative Medicine, Vol. II, Part II, pp. 838-849). Washington, D.C.: U.S. Government Printing Office.

Bray, G. A. (1976). The overweight patient. In G. H. Stollerman (Ed.), Advances in internal medicine (Vol. 21, pp. 267-307). Chicago: Year Book Medical Publishers.

Bray, G. A. (1970). The myth of diet in the management of obesity. American Journal of Clinical Nutrition, 23, 1141-1148.

Bruch, H. (1958). "Developmental Obesity and Schizophrenia." Psychiatry, 21, 65-70.

Crandall, C. (1986, September). Do binge eaters have more friends? American Health, p. 20.

Dalvit-McPhillips, S. P. (1983). The effect of the human menstrual cycle on nutrient intake. Physiology & Behavior, 31, 209-212.

Decke, E. (1971). Effects of taste on the eating behavior of obese and normal persons. In S. Schachter (Ed.), Emotion, Obesity and Crime (pp. 94-111). New York: Academic Press.

Franchina, J. J. (1987, May). Personal communication to David Stein. Department of Psychology, Virginia Polytechnic Institute and State University.

Garrow, J. S. (1978). Energy balance and obesity in man (2nd ed.). Amsterdam: Elsevier.

Griffiths, M., & Payne, P. R. (1976). Energy expenditure in small children of obese and nonobese parents. Nature, 260, 698-700.

Grinker, J., Hirsch, J., & Levin, B. (1973). The affective responsesof obese patients to weight reduction: A differentiation based on age at onset of obesity. Psychosomatic Medicine, 35, 57-63.

Hanna, C. F., Loro, A. D., Jr., & Power, D. D. (1981). Differences in the degree of overweight: a note on its importance. Addictive Behaviors, 6, 61-62.

Hashim, S. A., & Van Itallie, T. B. (1965). Studies in normal and obese subjects with a monitored food dispensary device. Annals of the New York Academy of Science, 131, 654-661.

Hawkins, R. C., & Clement, P. F. (1980). Development and construct validation of a self-report measure of binge eating tendencies. Addictive Behaviors, 5, 219-226.

Herman, C. P. (1978, December). Restrained eating. Symposium on obesity: Basic mechanisms and treatment. Psychiatric Clinics of North America, 1, 3.

Herman, C. P., & Mack, D. (1975). Restrained and unrestrained eating. Journal of Personality, 43, 647-660.

Herman, C. P., & Polivy, J. (1975). Anxiety, restraint and eating behavior. Journal of Abnormal Psychology, 84, 666-672.

Herman, C. P., Polivy, J., & Silver, R. (1979). Effects of an observer on eating behavior: The induction of sensible eating. Journal of Personality, 47, 85-99.

Hibscher, J. A., & Herman, C. P. (1977). Obesity, dieting, and the expression of "obese" characteristics. Journal of Comparative Physiological Psychology, 91, 374-380.

Hirsch, J., & Knittle, J. L. (1970). Cellularity of obese and nonobese human adipose tissue. Federal Proceedings, 29, 1516-1521.

Keys, A., Brozek, J., Henschel, A., Mickelsen, O., & Taylor, H. L. (1950). The biology of human starvation. Minneapolis: University of Minnesota Press.

Kirschenbaum, D. S., & Tomarken, A. J. (1982). Some antecedents of regulatory eating by restrained and unrestrained eaters. Journal of Abnormal Psychology, 91 (5), 326-336.

Laskowitz, D. (1982). Obesity as a barricade against social stress: An Adlerian view. In B. B. Wolman (Ed.), Psychological aspects of obesity (pp. 118-129). New York: Van Nostrand Reinhold.

Mayer, J. (1953). Genetic, traumatic and environmental factors in the etiology of obesity. Physiological Review, 33, 472-508.

Merola, G. T. (1984). Restrained eating: Public private behavior in normal weight and overweight individuals. Unpublished masters thesis, Department of Psychology, University of North Dakota, Grand Forks.

Metropolitan Life Insurance Company. (1959). New weight standards for men and women. Statistical Bulletin, 43, 3.

Minuchin, S., Rosman, B. L., & Baker, L. (1978). Psychosomatic families: Anorexia nervosa in context (pp. 51-73). Cambridge, MA: Harvard University Press.

Nisbett, R. E. (1968a). Determinants of food intake in human obesity. Science, 159, 1254-1255.

Nisbett, R. E. (1968b). Taste, deprivation and weight determinants of eating behavior. Journal of Personality and Social Psychology, 12, pp. 289-294.

Nisbett, R. E. (1972). Hunger, obesity, and the ventromedial hypothalamus. Psychological Review, 79, 443-453.

Nisbett, R. E. (1978, December). Symposium on obesity: basic mechanisms and treatment. Psychiatric Clinics of North America, 1, 3.

Nisbett, R. E., & Storms, M. D. (1975). Cognitive, social, and psychological determinants of food intake. In H. London & R. E. Nisbett (Eds.), Cognitive modification of emotional behavior (pp. 226-241). Chicago: Aldine.

Nisbett, R. E., & Temoshok, L. (1976). Is there an external style? Journal of Personality and Social Psychology, 33, 36-47.

O'Neil, P. M., Paine, P. M., Riddle, F. E., Currey, H. S., Malcolm, R., & Sexauer, J. D. (1981). Restraint and age at onset of obesity. Addictive Behaviors, 6, 135-138.

Orbach, S. (1979). Fat is a feminist issue: The anti-diet guide to permanent weight loss. New York: Paddington Press.

Polivy, J. (1976). Perception of calories and regulation of intake in restrained and unrestrained subjects. Addictive Behaviors, 1, 237-243.

Polivy, J. (1985, February 27). Personal communication to Geraldine Merola. University of Toronto.

Polivy, J., Herman, C. P., & Hackett, R. (1980). Self-awareness, self-consciousness, and the inhibition of eating. Unpublished manuscript, University of Toronto.

Polivy, J., Herman, C. P., Hackett, R., & Kuleshnyk, I. (1984). The effects of self-attention and public attention on eating in restrained and unrestrained subjects. Unpublished manuscript, University of Toronto.

Polivy, J., Herman, C. P., Olmsted, M. P., & Jazwinski, C. (1984). Restraint and binge eating. In R. C. Hawkins, W. J. Fremouw, & P. F. Clement (Eds.), The binge-purge syndrome: Diagnosis, treatment, and research, Springer Series on Behavior Therapy & Behavioral Medicine (pp. 104-122). New York: Springer Publishing Company.

Polivy, J., Herman, C. P., & Younger, J. C. (1978). Effects of a model on eating behavior: The induction of a restrained eating style. Journal of Personality, 47, 742-751.

Rodin, J. (1975). The effects of obesity and set point on taste responsiveness and intake in humans. Journal of Comparative and Physiological Psychology, 89, 1003-1009.

Rodin, J. (1981, April). Current status of the internal-external hypothalamus for obesity: What went wrong? American Psychologist, pp. 361-372.

Rodin, J. (1982). Obesity: Why the losing battle? In B. B. Wolman (Ed.), Psychological Aspects of Obesity (pp. 30-87). New York: Van Nostrand Reinhold.

Rodin, J., Elman, D., & Schachter, S. (1974). Emotionality and obesity. In S. Schachter & J. Rodin (Eds.), Obese humans and rats (pp. 177-184). Washington, D. C.: Erlbaum/Halsted

Rodin, J., Herman, C. P., & Schachter, S. (1974). Obesity and various tests of external sensitivity. In S. Schachter & J. Rodin (Eds.), Obese humans and rats (pp. 293-309). Washington, D.C.: Erlbaum/Halsted.

Rodin, J., & Slochower, J. (1976). Externality in the nonobese: The effects of environmental responsiveness on weight. Journal of Personality and Social Psychology, 29, 557-565.

Rodin, J., Slochower, J., & Fleming, B. (1977). The effects of degree of obesity, age of onset and energy deficit on external responsiveness. Journal of Comparative and Physiological Psychology, 91 (3), 586-597.

Rotter, J. B. (1966). Generalized expectancies for internal versus external control of reinforcement. Psychological Monographs: General and Applied, 80 (1), p. 609.

Ruderman, A. J. (1983). The Restraint Scale: A psychometric investigation. Behavior Research and Therapy, 21, 258-283.

Ruderman, A. J., (1985a). Dysphoric mood and binge eating: A test of restraint theory's disinhibition hypothesis. Journal of Abnormal Psychology, 94, 339-345.

Ruderman, A. J. (1985b). Restraint and irrational cognitions. Behavior Research and Therapy, 23 (5), 557-561.

Ruderman, A. J., & Christensen, H. (1983). Restraint theory and its applicability to overweight individuals. Journal of Abnormal Psychology, 92 (2), 210-215.

Ruderman, A. J., & Wilson, G. T. (1979). Weight, restraint, cognitions and counterregulation. Behavior Research and Therapy, 17, 581-590.

Salans, L. B., Cushman, S. W., & Wiseman, R. E. (1973). Studies of human adipose cell size and number in nonobese and obese patients. Journal of Clinical Investigation, 52, 929.

Schachter, S., Goldman, R., & Gordon, A. (1968). Effects of fear, food deprivation, and obesity on eating. Journal of Personality and Social Psychology, 10, 91-97.

Schachter, S., & Gross, L. (1968). Manipulated time and eating behavior. Journal of Personality and Social Psychology, 10, 98-106.

Schachter, S., & Rodin, J. (1974). Obese humans and rats. Washington, D.C.: Erlbaum/Halsted.

Sims, E. A. H., Goldman, R., Gluck, C., Horton, E. S., Kelleher, P., & Rowe, D. (1968). Experimental obesity in man. Transcript of the Association of American Physicians, 81, 153.

Smith, M. C., & Thelen, M. H. (1984). Development and validation of a test for bulimia. Journal of Consulting and Clinical Psychology. 52, (5), 863-872.

Sours, J. A. (1980). Starving to death in a sea of objects: The anorexia nervosa syndrome. New York: Jason Aronson.

Spencer, J., & Fremouw, W. (1979, June). Binge eating as a function of restraint and weight classification. Journal of Abnormal Psychology, 88, (3), 262-267.

Stein, D. M. (1985). Restraint and privacy in an eating paradigm. Unpublished manuscript, University of North Dakota, Grand Forks.

Stein, D. M. (1987a). A scaling issue in the assessment of dietary restraint. In press, International Journal of Eating Disorders.

Stein, D. M. (1987c). Preliminary concurrent validity of the Health & Development Inventory (ABI). Unpublished manuscript, University of North Dakota, Grand Forks.

Stein, D. M., & Brinza, C. (1987b). Factor structure of the Bulimia Test given to junior high school, high school, and college women. Manuscript under review, Psychological Medicine.

Stunkard, A., Srensen, T. I., Hanis, C., Teasdale, T. W., Chakradorty, R., Schull, W. J., Schulsinger, F. (1986). An adoption study of human obesity. New England Journal of Medicine, 314, (4), 193-198.

Thompson, J. K., Jarvie, G. J., Lahey, B. B., & Cureton, K. J. (1982). Exercise and obesity: etiology, physiology and intervention. Psychological Bulletin, 91, (1), 55-79.

Waxman, M., & Stunkard, A. J. (1980). Calorie intake and expenditure of obese boys. The Journal of Pediatrics, 96, (2), 187-193.

White, C. (1973). The effects of viewing films of different arousal content on the eating behavior of obese andnormal weight subjects. Dissertation Abstracts International, 34, 5B, p. 2324.

Wolgin, D. L., Cytawa, J., & Teitelbaum, P. (1976). The role of activation in the regulation of food intake. In D. Novin, W. Wyrwicka, & G. A. Bray (Eds.), Hunger: Basic mechanisms and clinical implications (pp. 67-81). New York: Raven Press.

Woody, E. Z., Costanzo, P. R., Liefer, H., & Conger, J. (1981). The effects of taste and calorie perceptions on the eating behavior of restrained and unrestrained subjects. Cognitive Therapy and Research, 5 (4), 381-390.

Wooley, S. C., Wooley, O. W., & Dyrenforth, S. R. (1979). Theoretical, practical, and social issues in behavioral treatments of obesity. Journal of Applied Behavior Analysis, 12, 3-25.

Wooley, S. C., & Wooley, O. W. (1984, February). Feeling fat in a thin society. Glamour, 61-66.