Binge eating : notes and data

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binge eating

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notes and data

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how to read this book

For forming an idea of the subject of the present study chapters 1 and 2 are recommended. Besides the clinical notes (sect. 1) and the laboratory model (sect. 2) the problem under study is formulated in chapter 2.

The author considers chapter 5 to be the most important part of the dissertation. In chapter 5, the present author's empirical contributions to the question why dieters counterregulate / binge eat are collected. Looking back, the author feels that sections 5.8 and 5.3 may be the most important ones. In 5.8 an *experimentum crucis* is presented. The findings contradict generally accepted ideas and put the etiology of counterregulation in a new light. In 5.3 a theory that achieved great popularity, is subjected to a straightforward test. Chapter 5 is, logically, preceded by chapter 4, in which an overview of the empirical studies is presented.

The interested reader should also read chapter 3. In sections 3.2, 3.3 & 3.4 popular theories for explaining counterregulation/binge eating are closely examined. In 3.2 the blind acceptance of the set point theory is criticized; in 3.3 the biopsychiatric 5-HT explanation of binge eating is critically examined; in 3.4 the cognitive view is presented and criticized for its lack of empirical background. In 3.5 an alternative model to explain overeating after exposure to various stimuli is presented to the reader. The model states that overeating results from a learned response.

In chapter 6 the main empirical findings are discussed. It is concluded that future research should concentrate on the model presented in 3.5.

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1 the clinical picture

1.1 a historical note

Binge eating, i.e. the rapid consumption of large amounts of food in a discrete period of time (APA, 1987), is widely believed to be of rather recent origin. A popular idea, especially noticeable in the lay press, is that binge eating primarily emerged during the last twenty years, and that the roots of binge eating are, in fact, socio-cultural (Lowenkopf, 1982; Orbach, 1978; 1986). However, although the prevalence may have increased recently (cf. 1.3), the very occurrence of episodic binge eating is certainly nothing new. The well-known Asterix and Obelix comic strips illustrate the old Roman custom to indulge in excessive eating and to vomit afterwards, the latter being a commonplace method of controlling weight. But better documented historical antecedents of the present concept of binge eating can also be found in writings from the last three centuries. In the early part of the 18th century, several physical and medical dictionaries paid attention to the voracious appetite. In several reports, binge eating appeared on the scene under the headings of '*fames canina*' (hunger of a dog), '*boulimus*' (hunger of an ox) and '*caninus appetitus*', the last designation referring to 'boulimus' followed by vomiting (for overviews: Stein & Laakso, 1988; Pope & Hudson, 1984). Craving for food and pathological overeating were discussed extensively by Robert Whytt in 1764 (Silverman, 1987), and in 1797 the term '*bulimy*', i.e. "*a disease in which the patient is affected with an insatiable and perpetual desire of eating*" was included in the Encyclopaedia Britannica. In the 19th century, Lasègue (1873) and Gull (1873, 1888) observed that binge eating could be part of the anorexia nervosa syndrome. In France '*boulimie*' and in Germany '*hyperorexia*' were discussed (see Stein & Laakso, 1988). Clearly, binge eating was already present centuries ago; from the beginning of the 18th century up to and including the 19th century, several medical textbooks more or less briefly referred to what we now call '*binge eating*'.

In the early part of the present century, binge eating was described more extensively in several case histories. The most familiar ones are "Nadia" described by Janet in 1903, "Ellen West", recorded by Binswanger in 1944, and "Laura" noted down by Lindner in 1954 (Casper, 1983; Pope & Hudson, 1984; Pope et al., 1985; Stein & Laakso, 1988). In the descriptions of these three individual cases, it is impressive how they are characterized by a typical and similar symptom pattern, which also strongly resembles case descriptions of binge eating individuals today.

Nadia, Ellen, and Laura were preoccupied with food and were often plagued by an irresistible craving for food which usually ended up as a binge. They alternated periods of merely dieting and losing weight with binge eating episodes. The hate of their bodies was prominent. Apparently, binge eating was not an isolated phenomenon but was accompanied by dieting practices and abhorrence of body shape.

1.2 diagnostic remarks

Interest in the clinical picture, characterized by a typical pattern of binge eating alternating with attempts at controlling body weight, experienced an upsurge during the last twenty years. The syndrome was described under a variety of diagnostic labels, such as '*dysorexia*' (Guiora, 1967), '*thin fat people*' (Bruch, 1973), '*compulsive eating*' (Rau & Green, 1975), '*bulimarexia*' (Boskind-Lodahl, 1976), '*dietary chaos syndrome*' (Palmer, 1979), '*bulimia*' (APA, 1980), '*binge-eating*' (Wardle & Beinart, 1981) and '*bulimia nervosa*' (Russell, 1979; APA, 1987). The clinical descriptions to which these labels refer hardly differ and three core features were always emphasized in them: first, the occurrence of episodic binge eating, associated with a subjective loss of control over food intake; second, the presence of behavior designed to control body weight; and third, extreme concerns about body shape and weight (Fairburn & Garner, 1986; Russell, 1985).

In the Diagnostic and Statistical Manual of Mental Disorders (DSM) the clinical picture appeared for the first time in the third edition. The term '*bulimia*' was used (APA, 1980). The DSM III criteria for bulimia were particularly criticized for the reason that here '*bulimia*' was, in fact, merely a description of binge eating, thereby referring to one of the symptoms instead of the full syndrome in which three core features (binge eating, behavior designed to control body weight, and extreme concern about shape and weight) are present (Fairburn & Garner, 1986; Russell, 1985). In the revised edition of the DSM III (APA, 1987), the term '*bulimia nervosa*' (BN) took the place of '*bulimia*'. At the moment, this label is generally accepted and widely used. According to the DSM III-R, BN is characterized by recurrent episodes of uncontrollable binge eating in which large amounts of food are rapidly consumed in a discrete period of time. The bouts of overeating are followed by strategies to prevent weight gain, such as self-induced vomiting, intake of laxatives and/or diuretics, vigorous exercise, and strict dieting (APA, 1987). Recently, chewing and spitting out food without swallowing it has also been identified as a common behavior to prevent weight gain (Mitchell et al., 1988). BN is independent of actual weight; individuals within the normal weight range, as well as under- and overweight subjects, can meet the criteria of BN (APA, 1987; Wardle & Beinart, 1981). Unlike DSM III, the revised edition

does include the criteria of behavior designed to control body weight and extreme concern about body shape and weight.

Although the DSM III-R apparently represents a step forward in the description and classification of BN, at least three issues call for elucidation through research.

1 In the diagnostic manual, binges are defined as "*rapid consumption of a large amount of food in a discrete period of time*" (APA, 1987; p. 67). One of the main and intriguing points in studies on binge eating is the question whether a particular eating bout should be interpreted as a binge or not. What exactly determines whether an eating episode is a binge? It is clear that especially the adjectives "rapid" and "large" are subject to highly personal interpretation.

2 "*A depressive mood that may be part of a Depressive Disorder is commonly observed*" (APA, 1987; p. 67). Earlier, in the 18th century, the specification of an "*extra-ordinary appetite, usually accompanied by a defection of the spirits*" (Stein & Laakso, 1988, p.207), indicated that depressive symptoms were already found to be associated with binge eating centuries ago. And throughout the historical descriptions, links between depression and bulimia were a recurrent theme; most descriptions of bulimia also refer to symptoms that could be indicative of depression. Recent empirical data confirmed the high prevalence of depression among binge eating individuals (Swift et al., 1985; Lee et al., 1985; Cooper & Fairburn, 1986). In the past decade, the role of depression in the etiology of bulimia nervosa has been discussed extensively, and special attention was paid to the question whether depression is a cause, an epiphenomenon, or a consequence of binge eating. Some authors even wonder whether bulimia nervosa actually *is* a mood disorder. Although an extensive review of the relationship between binge eating and depression is beyond the scope of this dissertation, the importance of this relationship is acknowledged and it is mentioned obliquely in connection with the biological and cognitive views on the etiology of BN.

3 "*Some people with this disorder are subject to Psychoactive Substance Abuse or Dependence, most frequently involving sedatives, amphetamines, cocaine, or alcohol*" (APA, 1987; p.67). The co-occurrence of addictive behaviors and overeating and similarities between them have recently attracted attention (Brisman & Siegel, 1984; Wardle, 1987c; Lacey & Mourel, 1986; Lacey & Evans, 1986; Filstead, Parela & Ebbitt, 1988). However, so far the alleged analogy between binge eating and addictive behaviors has not been an object of direct tests. The present dissertation mentions several efforts to distinguish possible common pathogenic processes in addictions and BN experimentally. Though refining DSM III-R was not the first aim of the studies included in this book, the issues mentioned above and raised by DSM III-R were touched upon in some of the present empirical investigations (for (1) see 5.1, for (2) see 3.4.3 & 5.4, for (3) see 3.5, 5.7, 5.8 & 5.9).

1.3 prevalence

As the early clinical descriptions showed, binge eating already occurred before the modern cultural stress on thinness of Western society came up. However, today binge eating is claimed to be far more prevalent than several centuries or even years ago (Casper, 1983). Whether this alleged increase in binge eating reflects an actual increase in prevalence or whether it is merely a matter of improved observation is unknown. Indirect observations, such as the number of clinical registrations and the growing attention for binge eating in popular magazines, suggest an increase in the prevalence of binge eating, but empirical findings on the prevalence rates of bingeing and binge-related behaviors vary extremely: percentages between 1% and 79% were reported (Halmi et al., 1981; Hawkins & Clement, 1980; Pope, 1984; Pope & Hudson, 1984; Healy et al., 1985; Pyle et al., 1981, 1983; Cooper & Fairburn, 1983; Fairburn & Cooper, 1984; Cooper et al., 1987; Thelen et al., 1987; Rosenzweig & Spruill, 1987). The divergence may be partly due to sample procedures (e.g. using student samples vs representative samples). However, the confusion about a rather fundamental issue may be at least as important: what exact combination of features constitutes a 'case'? The diagnostic criteria used in various studies differed in strictness as to the inclusion, definition, and frequency of symptoms. Concerning the *inclusion*, some studies merely inquired into 'binge eating' and the presence of strategies to control weight gain (e.g. Thelen et al., 1987; Rosenzweig & Spruill, 1987; Cooper, Charnock & Taylor, 1987), while others also investigated concern about shape and weight (e.g. Cooper & Fairburn, 1983; Fairburn & Cooper, 1983; Fairburn & Cooper, 1984; Healy et al., 1985). Furthermore, it may be clear that especially the *definition* of 'binge eating' is subject to highly personal interpretation. And as to *frequency*, some authors used strict criteria of at least one binge eating episode a week (e.g. Pyle et al., 1983; Healy et al., 1985), while others also included individuals who 'sometimes' binged (e.g. Fairburn & Cooper, 1983). As could be expected, the use of more stringent criteria concerning the inclusion, definition, and frequency of symptoms, made the number of cases decline significantly. It is therefore unclear to what extent 'cases' found in various prevalence studies are comparable with clinical cases; the community rate of clinically relevant binge eating remains unknown (Cooper et al., 1987; Healy et al., 1985). Wardle (1980) pointed out that food craving, difficulty in stopping to eat once started and bingeing were present in normal young people: *"dietary restriction, craving for food, and eating binges are not of themselves abnormal, although their frequency may be higher in the clinical population. In addition, the content of the binges may differentiate normal and clinical groups, although no data exist on this issue."* (Wardle & Beinart, 1981, p.102). Obtaining such data was the aim of one of the empirical studies included in this dissertation (see 5.1).

In sum, binge eating was already noticed long ago but its prevalence rate is supposed to have increased lately. Reliable data are lacking, however. When binge eating is not an isolated phenomenon but is accompanied by extreme concern about body weight/shape and behavior designed to control body weight, it is called bulimia nervosa. In the present dissertation, attention is focussed on bulimia nervosa and, more specifically, on the isolated act of binge eating.

2 a laboratory model

2.1 introduction

First of all attention will be restricted to the isolated act of bingeing and, secondly, the focus will almost entirely be on well-controlled lab studies. Two decades ago, Abramson & Seligman (1977) argued that psychopathological phenomena might be understood better through laboratory research. And experiments on psychopathological phenomena, such as anxiety, depression and obsessive-compulsive disorders really led to new and interesting insights into the etiology and maintenance of these disorders (see Abramson & Seligman, 1977). Binge eating also can be mimicked in the laboratory. The miniature binge that can be provoked is supposed to be analogous to the naturally occurring binge, while the conditions of its occurrence can be controlled and studied optimally.

Historically, the binge model has its roots in Schachter's (1968, 1971) work on obesity. Schachter studied the eating behavior of obese and normal weight people after various experimental manipulations, such as the induction of anxiety or the consumption of a preload. The 2 (*weight status*) x 2 (*experimental manipulation*) factorial design, with the amounts eaten by the subjects as the dependent variable, demonstrated remarkable differences in eating behavior between the experimental (*obese*) and control (*normal weight*) groups. In the next section of the present chapter, Schachter's main experimental findings will be presented briefly and his *externality hypothesis* will be discussed. It should be noted that, although his original hypothesis could not stand critical tests and replication studies, Schachter was the pioneer of a fruitful series of experimental studies on eating behavior. His classic experimental paradigm is very elegant. Although by and by it became clear that it was not obesity per se, but the restraint in food intake that determined the amount eaten after various experimental manipulations, Schachter's experimental paradigm remained very well applicable and is still widely used. In the third section, restraint and the *disinhibition hypothesis* are discussed. The large variety of experimental manipulations which elicit an overeating response in restrained subjects is reviewed in the fourth section. After reading the fifth section, the reader will unfortunately still be in the dark as to *why* restrained subjects overeat after various experimental manipulations.

2.2 obesity and the externality hypothesis

In the late sixties, Schachter and his co-workers carried out a series of lab studies in which they studied the eating response of obese and normal weight subjects after food loading, fear induction, or adrenalin injections (Schachter et al., 1968; Schachter 1971; Nisbett, 1968a, 1968b). Food loading, fear induction, and adrenalin injection all increase sympathetic activation. In this way, gastric motility is inhibited and blood sugar will increase, that is, a physiological state of low hunger emerges (see Cannon, 1920). Schachter (1971) found that low hunger manipulations, remarkably, did not have any effect on the amount eaten by obese subjects but it certainly did decrease the amount eaten by normal weight individuals. Two studies likewise showed that after experimental manipulation of anxiety, normal subjects ate less while obese subjects ate the same amount (McKenna, 1972; Abramson & Wunderlich, 1972).

It appeared that the eating response of the obese was not related to any internal sensation. Moreover, manipulations of presumed 'dinner time', palatability, smell, sight, and visual prominence of food cues demonstrated that the eating behavior of obese people was primarily externally controlled or stimulus bound (Wooley & Wooley, 1975; Rodin, 1980). The 'externality hypothesis' was born: *"eating by the obese seems unrelated to any internal, visceral state, but is determined by external food-relevant cues such as the sight, smell, and taste of food"* (Schachter, 1971, p.130). The eating behavior of normal weight people, on the other hand, appeared to be mainly determined by internal stimuli.

Schachter's externality hypothesis has been criticized for two reasons. First, support for Schachter's hypothesis was equivocal. Many replication studies and also new tests of the hypothesis failed to demonstrate a clear relationship between overweight and external responsiveness on the one hand, and normal weight and internal responsiveness on the other (Pudel, 1975; Price et al, 1975; see also the overviews by Wooley & Wooley, 1975; Rodin, 1981; Bennet & Gurin, 1982; Ruderman, 1986b). Instead of the overweight-external vs. normal weight-internal dichotomy, externally as well as internally responsive subjects were found in each weight category (Herman & Mack, 1975; Hibscher & Herman, 1977; for overviews: Rodin, 1981; van Strien, 1986; Ruderman, 1986b).

Second, it is not correct to draw a strict like between external and internal stimuli. With regard to food intake, external and internal cues are closely related. In the Schachter studies, sight, smell, and taste of food were claimed to be external stimuli. However, these 'external' cues can easily trigger internal physiological responses. It has been demonstrated that 'external' factors such as the sight of food or thoughts about food, stimulate physiological hunger responses, such as an insulin response, which whets the appetite (Powley, 1977; Rodin, 1985). The eating response following the insulin release can reasonably be regarded as internally determined. Therefore the argument in Polivy et al. (1978) that

the elevated external responsiveness serves to compensate for the deficit in internal responsiveness is not necessarily true: being hyperresponsive to 'external' cues does not automatically mean an unresponsiveness to internal cues.

In sum, from a series of lab studies Schachter hypothesized that obese people are externally oriented in their eating behavior. Normal weight individuals, on the other hand, were considered to be internally oriented. The merit of the externality hypothesis is that it stimulated a massive amount of research. However, empirical data showed that the internal vs. external dichotomy was far too simple a hypothesis to account for the processes underlying differences in 'obese' and 'normal weight' eating behavior (Rodin, 1981; Ruderman, 1986b).

2.3 restraint and the disinhibition hypothesis

Random deprivation of food, i.e. scarce and unpredictable food availability, heightens the external responsiveness of rats to food, even under conditions of abundance (see Rodin, 1980). Rodin speculates *"that an externally imposed schedule combining both scarcity and unpredictability might be expected to encourage dependence on external cues for the regulation of eating. Given the uncertainty of such a schedule, it would be best to eat whenever possible, regardless of internal needs."* (Rodin, 1980, p.235). In the early seventies, the idea emerged that external responsiveness was not related to overweight per se, but that it was a consequence of food deprivation (Nisbett, 1972). The attention of researchers gradually shifted from eating behavior as a function of body weight to the role of deprivation. More specifically, deprivation appeared to be a key factor in the determination of eating responses. In the mid-seventies, Herman & Mack (1975) developed the construct of restraint. Restraint literally means self-control and, with respect to eating behavior, restraint refers to dietary self-control (Ruderman, 1983). People vary in the extent to which they are restrained eaters. Highly restrained eaters (dieters), on the one end of the continuum, constantly try to restrict their food intake, and always worry about what they eat. At the other end of the continuum, low restrained eaters (non-dieters) eat whatever they like and do not bother about it (Herman & Mack, 1975; Herman & Polivy, 1980).

Relative deprivation as a consequence of the deliberate restriction of food intake (i.e. dieting) is independent of weight: both overweight and normal weight subjects may be dieters. It became clear that the inconsistency of the above-mentioned findings on the externality of the obese could be due to differences in level of restraint rather than to differences in weight. Externality was claimed to be a correlate of restraint rather than a correlate of obesity (Ruderman, 1986b).

With the emergence of the concept of restraint, a new hypothesis concerning the relationship between weight, externality, and eating patterns was developed. It was argued that, independent of actual weight, individuals who rated themselves as dieters showed the 'external' behaviors previously sometimes found as characteristic of the obese. Moreover, it was noted that restrained eaters were not merely characterized by dieting, but by an eating pattern alternating between dieting episodes and periodic overindulgence (Herman & Polivy, 1980; Ruderman, 1986b). Herman & Polivy (1980) formulated the *disinhibition hypothesis*, which states that breaking the self-control of restrained eaters by so-called disinhibitors will result in overeating.

2.4 many stimuli are disinhibiting

Restrained eating refers to attempts to suppress eating impulses. However, attempts to suppress food intake are not always successful: restraint can be broken. Breaking restraint, i.e. the disinhibition of inhibited (restrained) eating, has been the focus of much laboratory research in the last fifteen years. The most common experimental paradigm is in accordance with Schachter's design: a 2 (*restraint: yes/no*) x 2 (*experimental manipulation: yes/no*) factorial design, in which the consumption by subjects is measured after the experimental or the control manipulation. The disinhibition hypothesis boils down to the prediction that restraint and manipulation interact: restrained subjects will increase consumption after the manipulation, while unrestrained subjects will not. Subjects are usually classified as restrained or unrestrained on the basis of the median split of their restraint scores and the study is usually presented as a taste test. A variety of experimental manipulations has been studied, such as food and alcohol preloads, beliefs and emotional states. A paradigmatic experiment was reported by Herman & Mack (1975). Fifty-seven normal weight subjects scored the Restraint Scale (see Herman & Polivy, 1980) and were divided into low and high restraint subjects on the basis of median split. One-third of each group were given a preload of two milk shakes, one-third consumed a preload of one milk shake, and one-third were given no preload at all. After the preload (or without a preload) subjects had to 'taste' three types of ice cream. The amount eaten by each subject was registered. Results are given in figure 1.

After unrestrained eaters had eaten a large amount of preload, they ingested little ice cream, after they had eaten a little or no preload, they ingested much more ice cream. For restrained eaters the pattern was precisely the opposite: the more preload they had ingested, the more ice cream they consumed. And, *vice versa*, the less preload they had had, the less ice cream they took. Note that it is the interaction disinhibited (yes/no) X restraint (yes/no) which is important. In the present section, an

Fig. 1: ice cream consumption of restrained and unrestrained eaters after two, one or no preload. (Reprinted from Herman & Polivy, 1980, without permission).

overview of the experimental manipulations leading to the (dis)inhibition of restraint is presented.

preloads

It is a robust, well-known finding that dieters do not regulate their food intake as non-dieters do in the so-called 'taste test' (Herman & Mack, 1975; Hibscher & Herman, 1977; Herman & Polivy, 1980; Ruderman & Christensen, 1983). After non-dieters have eaten a preload of e.g. milk shakes, they will eat less ice cream than when they are not preloaded. In other words, non-dieters regulate their food intake. Interestingly, dieters show the opposite pattern in the studies by Herman and co-workers: they ate significantly more ice cream after a high-caloric preload than after no preload (Herman & Mack, 1975; Hibscher & Herman, 1977). This counter-intuitive and apparently illogical behavior is called counterregulation. Others found that dieters non-regulated instead of counterregulating, that is, dieters ate the same amount with or without a preload (Ruderman & Christensen, 1983). In both counterregulation and non-regulation, however, the interesting thing is the interaction between preload (yes/no) and restraint (yes/no).

beliefs

Dieters also counterregulate when they merely *perceive* the preload as high-caloric, regardless of the actual caloric content of the preload (Spencer & Fremouw, 1979; Woody, Constanzo, Liefer & Conger, 1981). Though Polivy (1976) originally hypothesized an effect of instructions, in

which the subjects were told that the preload was high- or low-caloric, she failed to find a significant effect. Therefore, Polivy (1976) reanalyzed her data *post hoc*. After reclassification of subjects on the basis of how calorie-rich they perceived the preload to be, i.e. regardless of the instructions, Polivy (1976) argued that dieters counterregulated when they merely thought they had consumed a high-caloric preload, regardless of the actual caloric content of the preload. But Polivy's conclusions (1976) were, at least, premature. First, the *post hoc* analysis merely showed a marginally significant interaction between restraint and perceived amount of calories ($p < 0.07$) and, second, no further statistical analysis of the simple effects underlying this interaction was presented. Third, it should be noted that *post hoc* analysis should only be considered suggestive. Meanwhile, two replication studies showed that a break of restraint did occur when the preload was *believed* to be high-caloric and did not when the preload was believed to be low-caloric (Spencer & Fremouw, 1979; Woody et al., 1981). Ruderman & Wilson (1979), on the other hand, were unable to find any significant effects due to the, otherwise successful, manipulation of beliefs about the caloric content of the preload. The conflicting results may be due to Ruderman & Wilson's (1979) experimental design: whereas Spencer & Fremouw (1979) and Woody et al. (1981) used a *between*-subject design, Ruderman & Wilson (1979) used a *within*-subject design. The anticipation of another 'taste test' may have been such a horrible prospect for highly restrained subjects that it elicited disinhibition during the very first challenge (cf. 3: anticipated consumption).

Recently, Heatherton, Polivy & Herman (1989) have instructed restrained and unrestrained subjects to feel either hungry or satisfied (experimental condition) or the subjects did not receive any instruction anything (control condition) after the ingestion of a vitamin capsule. When told to feel hungry, restrained subjects ate significantly more ice cream than when told to feel satisfied or when not told anything at all. The other way around, and surprisingly, unrestrained eaters ate significantly less when they were told to feel hungry than when they were told to feel satisfied or when not told anything at all.

anticipated consumption

To test whether anticipated dietary violation will disinhibit dieters, Ruderman, Belzer and Halperin (1985) studied cracker consumption by subjects expecting to consume a milk shake (diet-breaking), a salad (diet-compatible), or nothing at all after 'tasting' the crackers. Dieters actually increased their consumption in anticipation of a milk shake, whereas non-dieters decreased their food intake when expecting a milk shake (Ruderman, Belzer and Halperin, 1985). Although Ruderman and co-workers' data (1985) suggest that dieters respond to anticipated dietary violations in the same way as they do to actual and alleged dietary violation, data from Tomarken & Kirschenbaum (1984) point in a different

table 1: laboratory research on the regulation of food intake in dieters and non-dieters after stimulus A or B.

stimulus A	stimulus B	dieters food intake	non-dieters food intake	hypothetical explanation	authors
preload	no preload	A > B	A < B	being below set point	Herman & Mack 1975
		A > B	A < B	being below set point	Hibschler & Herman 1977
		A = B	A < B	—	Ruderman & Christensen 1983
belief in high-calorie preload	belief in low-calorie preload	A > B	A < B	disinhibitive thoughts	Polivy 1976
		A > B	A = B	disinhibitive thoughts	Spencer & Fremouw 1979
		A > B	A = B	disinhibitive thoughts	Woody, Constanzo Liefer & Conger 1981
		A = B	A = B	—	Ruderman & Wilson 1979
placebo hunger	placebo fullness	A > B	A < B	unresponsive to internal cues overresponsive to cognitive cues	Heatherton, Polivy & Herman 1989
anticipating high calorie consumption	anticipating low calorie consumption	A > B	A > B	disinhibitive thoughts	Tomarken & Kirschen- baum 1984 exp.1
		A = B	A > B	disinhibitive thoughts	Tomarken & Kirschen- baum 1984 exp.2
		A > B	A < B	disinhibitive thoughts	Ruderman, Belzer & Halperin 1985
alcohol	placebo	A < B	A > B	absence of cognitive label	Polivy & Herman 1976a

Continuation table 1

stimulus A	stimulus B	dieters food intake	non-dieters food intake	hypothetical explanation	authors
aware of drinking alcohol	non-aware of drinking alcohol	A > B	A < B	interaction between physiological change and cognitive label	Polivy & Herman 1976b
depressed	non-depressed	A > B	A < B	reinforcers lose their effectiveness	Baucom & Aiken 1981
		A > B	A = B	loss of self-control	Frost et al. 1982
		A > B	A = B	loss of self-control	Ruderman 1985a
anxious	non-anxious	A = B	A < B	loss of self-control	Herman & Polivy 1975
		A > B	A < B	boundary model	Herman et al 1987
monitoring food intake	non moni- toring of intake	A < B	A < B	altered self- monitoring of food intake during counterregulation	Kirschenbaum & Tomarken 1982
explicit attention	no attention	A < B	A = B	monitoring reintroduces diet consciousness	Polivy, Herman Hackett & Kuleshnyk 1986
overeating model	non- overeating model	A > B	A > B	restraint is a state which may be changed by social influences	Polivy, Herman, Younger & Erskine, 1979

direction. In one experiment, both dieters and non-dieters ate significantly more when anticipating a high-calorie meal than when expecting to eat a low-calorie meal or no meal at all. In the next experiment, restrained eaters ate the same amount when anticipating a high-calorie as when anticipating a low-calorie meal, whereas unrestrained subjects again ate more only when anticipating a high calorie meal (Tomarken & Kirschenbaum, 1984). From the inconsistency between the findings in the Ruderman et al. (1985) and the Tomarken & Kirschenbaum (1984) studies it

can be concluded that anticipated dietary violations disinhibit restrained eaters merely under some circumstances (Ruderman, 1986b). It still has to be determined what are the crucial conditions for overeating to occur after anticipation of dietary violation.

alcohol

The knowledge that alcohol is a classical inhibitor led Polivy & Herman (1976a,b) to hypothesize that the consumption of alcohol should disinhibit restrained eaters and make them overeat. But Polivy & Herman's hypothesis could not be corroborated (Polivy & Herman, 1976a). In contrast with what had been predicted, restrained eaters ate marginally less after the consumption of alcohol than after a non-alcoholic drink, whereas unrestrained eaters ate more after the alcoholic drink than after the non-alcoholic drink. According to Polivy & Herman (1976a), it was a post hoc seemingly important point that subjects were not aware that they were drinking alcohol. In another study, Polivy & Herman (1976b) manipulated subjects' beliefs about the alcoholic content of the drink. They hypothesized that it was the *perception* of drinking alcohol instead of the actual alcoholic content which determined the subsequent eating response. It was found that when restrained eaters drank alcohol and the drink was presented as alcohol, they subsequently ate more than when they thought that they had consumed vitamin C. Unrestrained eaters, on the other hand, ate less when they thought they had ingested alcohol than when they thought they had ingested vitamin C (Polivy & Herman, 1976b).

emotional states

It has repeatedly been demonstrated that, apart from eating or anticipating an (alleged) high-caloric food or alcohol preload, the induction of certain emotional states also elicits an overeating response in dieters (Herman & Polivy, 1975; Baucom & Aiken, 1981; Frost et al., 1982; Ruderman, 1985a; Herman et al., 1987). Herman & Polivy (1975) found that anxious unrestrained eaters (expecting painful shocks) ate less than 'non-anxious' unrestrained eaters (expecting mild shocks), whereas anxious restrained eaters ate the same amount as 'non-anxious' restrained eaters. More recently, Herman et al. (1987) have refined their anxiety hypothesis. They found that anxious non-dieters only ate less when they were initially hungry and not when satiated. And anxiety increased eating in dieters when the dieters were initially hungry, but not when satiated. Other researchers manipulated mood, e.g. by manipulating success or failure in a cognitive problem solving task (Baucom & Aiken, 1981; Ruderman, 1985a) or by having the subjects read self-referring statements, i.e. the Velten mood induction procedure (Frost et al., 1982). The general finding is that restrained eaters eat more when depressed, whereas mood hardly influences the amount eaten by unre-

strained eaters. In conclusion, negative affective states generally increase the consumption of restrained eaters (Ruderman, 1986b).

monitoring

Research did not only focus on the stimuli which disinhibit dieters; attention was also paid to experimental manipulations which prevent dieters from the typical non- or counterregulatory response. More specifically, the manipulation of monitoring practices (either self-monitoring or monitoring by others) was found to influence the dieters' food consumption. Kirschenbaum & Tomarken (1982) showed that the introduction of self-monitoring cues could prevent dieters from overeating. Preloaded dieters who were subsequently given ice cream in a small bowl with a label giving accurate information about calorie content were able to regulate their food intake, whereas preloaded dieters who subsequently ate from a large bowl without a calorie label overate. It was a rather unexpected finding that without any self-monitoring cues (large bowls and no calorie label) restrained as well as unrestrained subjects ate much. When one cue was present, restrained eaters ate significantly more than unrestrained ones.

Polivy et al. (1986) found that heightened self-attention (the subject exactly registered how much she ate) as well as the awareness of public attention (the experimenter could see exactly how much was eaten) inhibited the consumption by dieters after a preload. Non-dieters' food consumption, on the other hand, was not influenced.

Earlier, Polivy and co-workers (1979) showed that the food intake of dieters and non-dieters after a preload is influenced by the presence of a model. In the presence of a stooge who overate, dieters, as well as non-dieters ate, significantly more than in the presence of a stooge who did not overeat (Polivy et al., 1979).

Self-observation may serve as an effective inhibitor of overeating in dieters, because the dieter's attention is refocussed on dietary concerns.

In sum, laboratory research shows that restrained eaters can be disinhibited by a motley collection of stimuli. Overeating, i.e. non-regulation of food intake or even counterregulation, is easily induced in dieters; stimuli which may act as disinhibitors are (1) actually eating a high-calorie preload, (2) the perception (wrong or not) of having eaten a high-calorie preload, (3) the notion that a high-calorie preload will be eaten, (4) the idea of hunger (5) the notion that alcohol has been drunk, (6) being anxious and (7) being depressed. Furthermore, it has been shown that self-monitoring practices prevent the dieter from overeating.

Although the data presented concern miniature binges in the laboratory, they are valuable. The model appears to be a good model. The antecedent stimuli for clinical binges seem remarkably similar to the kinds of stimuli investigated in the laboratory (Polivy & Herman, 1985). But why use a model? A model is relatively easy to understand. It is a good means

of learning more about reality on condition that the mechanism c.q. the causal processes are understood. The next question, then, is: how can the disinhibition of dieters be explained? In the next section current explanations of the experimental data on the disinhibition of restrained eaters are reviewed briefly (cf. table 1).

2.5 how does the 'disinhibitive' mechanism work?

From table 1, it becomes clear in how many different ways the identical phenomenon of dieters' overeating after a variety of stimuli is explained. When details are ignored, the different explanations can roughly be divided into four main views: (1) a physiological view, (2) a cognitive view, (3) a view that is no view (pseudo-explanations), and (4) a blind spot (forgotten explanation).

Ad 1. The physiological explanation states that dieters are chronically hungry. Their actual weight is supposed to be below their biologically determined set point level for body weight, which should trigger an urge to binge under appropriate circumstances, e.g. after various stimuli (Herman & Mack, 1975; Hibscher & Herman, 1977). This explanation will be discussed in 3.2.

Ad 2. The cognitive explanation states that the perception of having overeaten disinhibits the dieter. That is, dieters reportedly perceive the (alleged) high-calorie preload as a violation of their diet and are said to respond to that with thoughts such as *"I've blown my diet, I might as well continue to eat"*. Such thoughts are supposed to disinhibit the dieter temporarily (Polivy, 1976; Spencer & Fremouw, 1979; Woody et al., 1981). Anticipated dietary violations reportedly have the same cognitive effect as real or perceived dietary violations (Tomarken & Kirschenbaum, 1984; Ruderman et al., 1985). This cognitive explanation will be discussed in 3.4.

Ad 3. The third series of explanations is called pseudo because they are, in fact, descriptive and do not explain anything. Dieters' non-regulation or counterregulation after the induction of an anxious or depressed mood is usually 'explained' as a loss of self-control (Herman & Polivy, 1975; Frost et al., 1982; Ruderman, 1985). Strong emotions allegedly disrupt the cognitively mediated self-control processes, and consequently restraint, resulting in overeating. Clearly, this tautological description lacks the slightest explanation of counterregulation.

Herman & Polivy (1984) have recently integrated the different explanations in a biopsychological boundary model. The model states that consumption is regulated by biological needs as well as by psychological influences. When hunger or satiety passes a certain boundary, it is aversive and people start or stop eating. Between the boundaries of hunger and satiety there is an area of biological indifference. Normally an organism strives to be in the zone of biological indifference: the aversive-

ness of hunger keeps consumption above a minimum level while the aversiveness of satiety keeps consumption under a maximum level. In the zone of biological indifference, psychological (e.g. cognitive and social) factors dominate the determination of human eating behavior. It is crucial that, according to the boundary model, dieters and non-dieters differ in the place of their boundaries. In dieters, the area of biological indifference is wider than in non-dieters: dieters have lower hunger boundaries and higher satiety boundaries, i.e. they have to be more deprived to report hunger and they can eat more before reporting satiety. Furthermore, dieters have a 'diet' boundary. Passing this diet boundary, i.e. breaking the diet, means excessive eating. The diet boundary is a cognitive one: the boundary determines the maximum amount that dieters wish to eat. But, when this diet boundary is passed, dieters eat until they reach their (high!) satiety boundary. According to Herman & Polivy (1984), the proposal of a cognitive diet boundary may account for counterregulation: normally (i.e. when not dieting), a person eats until the satiety boundary is reached. That is, non-dieters will eat less after a preload than without one. However, dieters usually eat until they reach their diet boundary. That is, without a preload they eat little. But after a preload, the diet boundary is passed and the dieter will eat to the (high) satiety boundary is reached (Herman & Polivy, 1984).

Post hoc, the boundary model appears to cover the findings on counterregulation after an (alleged) high-calorie preload reasonably well. The perception of having passed the diet boundary may lead the dieters to eat until their high satiety levels have been reached. However, the boundary model can be criticized for several reasons. First, when it comes to covering the non-regulation of anxious or depressed dieters, the model only holds by virtue of an auxiliary hypothesis. According to the makers of this model, anxious or depressed dieters counterregulate because emotions are more urgent than dietary concerns; the dieter has to decide how to cope with the stressor, thereby forgetting to diet (Herman & Polivy, 1984).

Second, the model can be criticized for its lack of explanatory power. The boundary model is of a descriptive nature and does not explain *why* dieters regularly pass the diet boundary.

The model predicts that dieters will only overeat when they pass their diet boundary. In other words: the model will be falsified when dieters counterregulate without passing their diet boundary (cf. 5.8).

Ad 4. The blind spot of the forgotten explanation. Curiously, the series of explanations given for laboratory data on counterregulation does not exactly parallel the different explanatory models for clinical binge eating. The physiological set point theory and the cognitive explanation appear both in the literature on lab counterregulation and in that on clinical binge eating. As was mentioned before, these explanations will be discussed more extensively in the next chapter (cf. 3.2 & 3.4). However, attention will also be paid to a biopsychiatric explanation that is hardly ever mentioned

in the literature on experimental research, but is a popular theory when it comes to explaining clinical binge eating. This biopsychiatric theory claims that a lowered serotonin level is responsible for binge eating and the accompanying depression (cf.3.3).

In sum, dieters counterregulate after the introduction of certain stimuli. The question is why. Many explanations are offered. The explanations of laboratory data can roughly be divided into physiological, cognitive, pseudo, and forgotten explanations. The pseudo-explanations were especially criticized for their merely descriptive nature and their lack of explanatory power. Physiological and cognitive theories, as well as the forgotten explanation, will be discussed in the next chapter.

2.6 summary

With the introduction of a laboratory model, Schachter gave the first impulse to decades of fruitful experimental research on eating behavior. But Schachter's original hypothesis, that obese people eat in response to external stimuli and not as a consequence of internal cues, could not stand critical (replication) tests. The amount of self-reported restraint in eating behavior appeared to be a more critical determinant of eating responses. The disinhibition hypothesis was put forward: restrained eaters usually diet but they may temporarily overeat after the introduction of a so-called disinhibitor. A variety of disinhibitors (e.g. preloads, cognitions, and emotions) have been found to trigger the overeating response in dieters but not in non-dieters. Why? Several explanations have been presented. Except for the boundary model, which is claimed to be descriptive and does not explain anything, the different theories will be examined critically in the next chapter.

3 on the disinhibitive mechanism

3.1 introduction

In the past two decades, several hypotheses which should account for counterregulation or binge eating were formulated. In the present chapter, three important explanations that have been proposed to account for counterregulation, or episodes of binge eating, are reviewed. According to the *set point hypothesis* (cf.3.2) dieters are below their biologically determined set point for body weight, which is supposed to trigger the urge to overeat. The *5-HT hypothesis* (cf.3.3) claims that binge eating is the consequence of a diet-induced hyposerotonergic brain state. The *cognitive explanation* (cf.3.4) states that dysfunctional thoughts concerning food intake are of primary importance in triggering an eating binge or counterregulation. The different hypotheses will be examined critically with respect to their explanatory power and the predictions that follow from them.

In section 3.5 an alternative explanation of counterregulation is presented. The alternative model states that counterregulation or binge eating is cue-controlled. Five postulates of the model are discussed.

3.2 being below set point

3.2.1 *the set point theory in the eighties*

"...in the population of normal weight individuals there exists a subpopulation who is actively restraining their eating to maintain a socially sanctioned weight that is low relative to their biologically set or physiologically optimal level." (Woody et al., 1981, p. 382).

"Persons who score high on measures of Dietary Restraint are thought to be those whose weights are below their 'biological setpoints' (Nisbett 1972) because they vigilantly monitor and restrict their food intake." (Blanchard & Frost, 1983, p.259).

"An alternative model presented by Nisbett (1972) proposed that everyone has a biologically determined ideal weight or set point." (Ruderman & Christensen, 1983, p 210).

"Based largely on Nisbett's (1972) set-point hypothesis, restrained eaters are considered to be in a state of chronic hunger as a result of continual

attempts to suppress their weight below a physiologically determined set point." (Tomarken & Kirschenbaum, 1984, p.459).

"....following Nisbett (1972)" it is "suggested that restrained eaters (dieters) strive to maintain body weights below set point, whereas unrestrained eaters (nondieters) do not." (Esses & Herman, 1984, p.712).

"Weight appears to be physiologically regulated around a 'set point', or a weight that one's body tries to 'defend'" (Garner et al., 1985, p.515).

"The logic of Nisbett's (1972) argument that the majority of obese resemble starved organisms because of their attempts to suppress their weight below natural levels may be extended to all chronic dieters, regardless of their weight" (Garner et al., 1985, p.539).

"Successful dieting produces weight loss, which in turn might create a state of chronic hunger, especially if such weight loss leaves the dieter at a weight below the set-point weight that is defended physiologically." (Polivy & Herman, 1985, p.196).

"Despite these negative findings (to support Nisbett's predictions, A.J.), Nisbett's basic concept of a homeostatically defended body weight remains viable" (Ruderman, 1986b, p.248).

It is clear: Nisbett scores high on the citation index for the literature on counterregulation, c.q. binge eating. The above citations illustrate that, even in the eighties, Nisbett's set point theory is still a popular theory when it comes to the explaining of counterregulation in the lab and clinical binge eating. Researchers on counterregulation and binge eating repeatedly refer to Nisbett's set point theory to explain dieters' overeating. The set point theory, as it is cited above and described below, is a rather old-fashioned conceptualization, but it is still a leading theory in counterregulation literature. In the present section it will be argued that it is wrong to take the set point theory uncritically as being true, as too many psychologists do.

3.2.2 *the set point hypothesis*

The set point hypothesis for body weight or its correlate body fat stems from the idea that many physiological processes are regulated by homeostatic mechanisms. In the early seventies the idea took shape that an organism's amount of body fat is regulated by a homeostatic mechanism, just as there is a thermoregulatory set point. The theory states that every organism is prepared to possess a characteristic amount of fat. The idea is that the amount of body fat is registered constantly and that, via feedback processes, a homeostatic control mechanism is informed about the amounts of body fat actually present in the organism. If deviations

from the set point are registered, i.e. if more or less fat is present than the set point level requires, processes will be started up to restore the amount of fat to the set point level.

Pioneers of the notion that there is a set point for body fat argued that the eating pattern of overweight people (cf. 2.2 & 2.3) is, in fact, an epiphenomenon of dieting (Cabanac 1971; Cabanac & Duclaux, 1970; Cabanac, Duclaux & Spector, 1971; Nisbett 1972, 1974). Nisbett pointed out that there are remarkable similarities between obese and hungry individuals: both were found to be hyperemotional, hypoactive and showed decreased sexual interest. His assumption, which seems paradoxical at first sight, was that many fat people were actually hungry and 'underweight'. According to Nisbett's theory, these people should have a high set point for body weight, i.e. a high biologically preprogrammed body weight. This set point weight apparently does not fit in with the socio-cultural ideal of slenderness. With the intention of reaching the ideal weight, people go on a diet and bring down their actual weight below set point level.

Four lines of evidence that back up set point theory can be distinguished:

Cabanac's alliesthesia method

Cabanac (Cabanac 1971; Cabanac & Duclaux, 1970; Cabanac, Duclaux & Spector, 1971) stressed the importance of sensory feedback processes in the regulation of food intake. In several experiments it was found that the pleasantness ratings of the taste of sucrose normally decreased significantly after subjects had eaten a glucose preload. Fasting subjects and a subsample of obese subjects, however, still rated the sucrose stimulus as very pleasant after a preload of glucose (Cabanac & Duclaux, 1970; Cabanac, Duclaux & Spector, 1971; Guy-Grand & Sitt, 1975). The normal decrease in perceived pleasantness of sweet stimuli after a glucose preload, is called *negative alliesthesia* by Cabanac (1971) and is supposed to be functional in determining the moment people stop eating because of satiety. Cabanac (1971, p.1103) argued that "*a stimulus can feel pleasant or unpleasant depending upon its usefulness as determined by internal signals*". He supposed that negative alliesthesia is dependent on the (dis)agreement between the actual body weight and its set point value. When actual weight corresponds to set point weight, sweet stimuli will be evaluated as unpleasant after a glucose preload, because further food intake would lead to a body weight higher than preset. With the reduction of body weight to below set point level negative alliesthesia (the taste of sugar becoming less pleasant after a glucose preload) disappears. For, when people are below their set point for body weight, further food intake is necessary for reaching the preset level of body weight again. Cabanac claimed that the set point of obese people is, naturally, set at a higher body weight. "*If the obese person, for health or social reasons, combats his obesity, he will lower his body weight below the set*

'obese' level, negative alliesthesia will disappear and satiety will be impaired." (Cabanac, 1971, p.1107).

stability of body weight under natural conditions

The idea of body weight being regulated at a specific level or 'set point' has strong face value: the commonsensical intuitive appeal that there has to be 'something like a set point for body weight' is mainly based on the notion that body weight remains relatively constant over long periods and under various circumstances. It is astonishing that, through the years and despite enormous inter- and intra-individual differences in food intake and energy expenditure, body weight is quite stable (Keesey, 1980, 1986). The regulation of body weight is based on the balance between energy intake and energy expenditure; a stable body weight means that energy intake and energy expenditure are in balance. Despite divergent living conditions, body weight of people living in technologically advanced societies (with much 'junk' food and sedentary lives) is usually little different from the body weight of, say, Eskimo (who do not eat any junk food and expend much energy on activity). And even intra-individual variations in food intake do not lead to weight changes expected because of caloric excess/debt (Keesey, 1980, Sims, 1974).

resistance to displacement from body weight

Normally, people strongly resist deliberate attempts at weight loss or weight gain. Dieters can lose weight temporarily but will soon regain it, whereas overeaters can gain weight temporarily but will soon lose it again. When dieters at reduced body weight restore their calorie intake to normal, they gain weight many times more rapidly than non-dieting normal weight subjects eating the same amount, even without overeating (Keesey, 1980). And, the other way around, normal weight students who wanted to gain weight had difficulty in adding even 10% to their normal weight (Sims, 1974). Gaining weight was difficult (for gaining weight double calorie intake was required) but it was even more difficult to maintain the increased weight: continued overeating and a larger and larger intake of food was required to maintain the overweight. After force-feeding was stopped, body weight rapidly returned to normal level, even without dieting. Energy was used less efficiently by the increase of the rate of metabolism, the so-called *luxury-consumption*. It is clearly difficult to maintain the displacement of weight from the normally maintained level (cf. the alleged set point). Organisms resist being displaced from their body weight set points by adjusting their rates of energy expenditure: energy is used more or less efficiently, by respectively reducing or increasing the speed of metabolism.

stability and defense of a new, experimentally induced, body weight

After experimentally induced lowering or elevation of the alleged set point level, this new level is defended as effectively as the original set point was (Keesey, 1980, 1986). After a lesion in the lateral hypothalamus (LH), rats lose weight rapidly. The weight loss of LH-lesioned rats is due to higher rates of energy expenditure immediately after the lesioning (Keesey & Corbett, 1984) and decreased food intake. The hypermetabolic state persists until body weight is reduced to the level which will eventually be maintained (Keesey & Corbett, 1984). Thus, weight stabilizes at a lower level and the energy balance is normal again after a while. It should be noted that non-lesioned animals will be hypometabolic when their weight is lowered to the level of LH-lesioned animals. But LH-lesioned rats show normal rates of heat production and metabolic defense at these reduced levels. And, moreover, the LH-lesioned rat does not only regulate its 'new' weight precisely at the lower level, but it also defends the lowered body weight effectively when challenged, e.g. after force-feeding or dietary restriction. When an LH-lesioned animal's weight drops below its new set point, hypometabolic adjustments occur and the animal displays the same hypermetabolic response to diet-induced weight gain as a non-lesioned animal does (Keesey 1980).

The genetically determined obese Zucker rat (with an abnormally high number of large-sized fat cells), on the other hand, maintains and defends a high body weight. At this high body weight, food intake is normal and energy requirements for maintenance are normal. When challenged, i.e. in case of dietary restriction or force-feeding, the obese Zucker rat effectively defends its high weight by metabolic adjustments. These observations are interpreted as consistent with the idea that body weight is regulated around an elevated set point in the Zucker rat (Keesey, 1980).

In sum, set point theory postulates the existence of a mechanism that determines how fat a body will be. The mechanism aims at a balance in energy input and energy output. A change in set point in fact means a change in the regulation of the input-output balance at a higher or lower body weight level. Arguments for the existence of a such a mechanism are: (1) dieters do not show the normal decrease in sweet preference after ingestion of glucose (2) the stability of body weight over long periods, (3) organisms have great difficulty in keeping their reduced or heightened body weight for a long time, and (4) after the 'set point' is experimentally lowered or elevated, this new level is defended as effectively as the original 'set point' was.

However, several critical remarks can be made about the set point idea. First, the concept of a set point will be critically examined. Second, it is questionable whether the set point theory is appropriate for explaining counterregulatory or binge eating behavior: being below set point re-

quires successful dieting. But is successful dieting really as important as set point theory implies?

3.2.3 *four critical comments on the set point concept*

the significance of alliesthesia

Cabanac's concept of alliesthesia can be criticized for at least two reasons. First, it should be noted that the experimental findings on alliesthesia are unequivocal. Esses & Herman (1984) found that both dieters and non-dieters showed negative alliesthesia. After a glucose preload, dieters even valued the sucrose solutions as less pleasant than non-dieters. Second, the basic idea is that negative alliesthesia has biological significance. It is assumed to be functional in the regulation of body weight. Wooley, Wooley & Dunham (1972b), however, challenged this idea: they found a similar decrease in perceived pleasantness after intake of a non-caloric but equally sweet solution as the glucose preload. The authors are right in arguing that *"it is highly unlikely that ingestion of 200 ml of 25% glucose (approximately 200 calories) would eliminate all need for food (...) regardless of physiological need, such subjects may still find certain nonsweet tastes (i.e. bacon and eggs) pleasant even after ingestion of a considerable amount of glucose solution.."* (Wooley, Wooley & Dunham, 1972b, p.768).

post hoc selection of fitting and unfitting findings

As was mentioned before, after a lesion in the lateral hypothalamus, rats regulate and defend their new weight precisely. According to set point theorists, this finding is a shining example of a new set point. After a lesion in its ventromedial hypothalamus (VMH), however, a rat is hyperphagic until it has reached a higher body weight, but stable weight is maintained only when the quality and the nature of the diet (i.e. the flavor, texture, sweetness, and smell) remain the same. In the case of dietary manipulations, VMH-lesioned rats do not show metabolic adjustments to defend the induced obesity. That is, when the diet is less palatable, the body weight is soon restored to normal levels, without metabolic defense of the former weight (Keesey & Corbett, 1984). And, *vice versa*, when the diet is very palatable, VMH-lesioned rats become very obese (Keesey, 1980). Although Bennet & Gurin (1982, p.96) claim that, in response to the palatability of the diet, VMH-lesioned rats *"reset their set point and then eat as much as they need to keep the fat store constant at the new level"* (sic!), Keesey (1980, p.153) argued that *"it seems inappropriate to view the effects of such lesions in terms of an altered set point"*. According to this prominent set point theorist, *"the point is that VMH-lesioned rats do display certain regulatory deficits, particularly in the defense of their obesity. For this reason they are not well suited for studying the issue now under consideration, namely, how body weight is regulated around a high*

set point" (Keesey, 1980, p.154). This poses serious problems for the theory. Why are LH-lesions (data fitting with the theory) in advance far more convenient than VMH-lesions (non-fitting data)?

circularity

With respect to individual diagnoses (how should someone's set point for body fat be determined?) and with respect to the exact mechanism of action people do not shrink from putting forward circular arguments. Concerning the first issue, set point theory stemmed from the observations that body weight is normally very stable and difficult to change over longer periods. Because of this it is circular to reason that the set point of someone's body weight is the body weight to which he spontaneously returns (cf. Bennet & Gurin, 1982) or to claim that the actual weight of an organism is below its set point *"when they become more efficient at utilizing a given amount of food"* (Mrosovsky, 1983, p. 201-202). Mrosovsky (1983) even seriously proposed to determine whether someone is at or below his set point by using Cabanac's alliesthesia method. Both criteria (a more efficient utilization of energy, and Cabanac's alliesthesia method) were the very observations that led to the notion of an alleged set point. Note that here 'set point' is a descriptive label that is merely a redescription of other phenomena and does not offer any new information.

A more formal option is offered by Kleiber (see Keesey, 1980; Keesey & Corbett, 1984). Energy needs are expressed as a power function of body weight: Kleiber's rule. This rule states that energy needs are proportional to the $3/4$ power of body weight, which only holds when an organism is at its set point level for body weight. Again, but now in formal arithmetical terms, it is described that with weight loss or weight gain, adjustments in energy output take place. The observation of energy adaptation, an observation that once led to the introduction of the concept of set point, is now presented as evidence of the existence of a set point level. This is like saying that someone acts crazy because he is schizophrenic and that we know that he is schizophrenic because he acts crazy. So far, independent determination of the alleged set point for body weight has not been possible. And the model lacks any unique and testable predictions when it comes to this.

The second series of circular reasoning concerns the exact mechanism of action. Set point theory claims that *"..the body does 'care'. Built into every person is a control system dictating how much fat he or she could carry. We'll speak of this control system as determining a setpoint for body fat.."* (Bennet & Gurin, 1982, p.6). However, the exact mechanism of action is still unknown. The anthropomorphic notion that there is a system in the body which attempts to maintain a 'preferred' fat content gets bogged down in homuncular reasoning. The functional details of the system are not yet understood. *What 'mechanism' is informed in what way about what state of the organism?*

the precision of the model

The term set point presumes a fairly precise, accurate and hardly changeable mechanism. However, this does not fit in with all the facts.

Empirical data show a remarkable facility changing the set point for body weight by a change in the diet. Set point can be changed by a more or less varied and palatable diet (Sclafani, 1980; Bennet & Gurin, 1982) and it can be heightened after long-term fat feeding, that is after months of high-fat diet (Van Itallie, 1984). Moreover, exercise has been found to lower 'set point' level (Bennet & Gurin, 1982). It is not appropriate to talk about a fairly precise energy regulatory mechanism when this mechanism can relatively easily be changed by variations in energy input or output. It is questionable whether observations that could falsify the theory are possible. This is slightly reminiscent of psychoanalysis: everything is possible.

In sum, the set point concept lacks preciseness and falls a prey to post hoc and circular reasoning. Mrosovsky & Powley (1977) hit the nail on the head when they, tentatively, stated that using of the term set point is just *"a redescription of the data that adds nothing but the illusion of understanding"*. The concept of 'set point' is merely a non-explanatory classification of observations. As the physiological basis of the concept set point is not yet understood, it is of great importance that the concept be used appropriately. As a descriptive term, 'set point' could be convenient. In fact, set point refers to an adaptation in energy metabolism after weight has been challenged. But when it is claimed to be explanatory, it should lead to unique predictions, i.e. predictions other than adaptation in energy metabolism when weight has been challenged. If the physiological mechanisms by which weight or fat are regulated exactly are discovered, the idea of a set point may even prove unnecessary.

Meanwhile, the set point theory has generated and stimulated much sophisticated research on energy metabolism (see e.g. Scheurink, 1989). In this research, short-term regulation is differentiated from long-term regulation and, in the latter, insulin appears to play an important part. Researchers of counterregulation/binge eating should first update their knowledge before concluding uncritically that dieters counterregulate and bulimics binge eat because *"they are below their biologically determined set point for body weight"*.

3.2.4 *set point theory and counterregulation in the lab*

Set point theory implies that a person's body weight will be lowered to a sub set point level after a period of successful dieting and, that at that very moment, the body tries to restore its weight to set point level by means of binge eating. Dieting and bingeing do usually co-occur (Polivy & Herman, 1985). Repeated dieting is claimed to be a risk factor for bulimia nervosa and about 80% of patients with bulimia nervosa tried to lose weight by strict dieting prior to the onset of binge eating (Pyle et al., 1981; Fairburn

& Cooper, 1982; Striegel-Moore, Silberstein & Rodin, 1986; Rossiter, Wilson & Goldstein, 1989; Mizes, 1985; Fairburn, Cooper & Cooper, 1986). Furthermore, about 50% of the anorexia nervosa patients develop binge eating episodes about 9 to 18 months after the onset of the strict dieting practices (Wardle & Beinart, 1981; Polivy & Herman, 1985; Laessle et al., 1989), and non-clinical dieters report significantly more binge eating than non-dieters (Wardle, 1980). The above-mentioned data on the co-occurrence of dieting and bingeing have been derived retrospectively from clinical reports. They therefore have to be handled with caution when it comes to the chronology and temporal relationship between dieting and bingeing. Also, the laboratory studies on counterregulation presented earlier (cf. chapter 2), which show that especially dieters are prone to disinhibition and overeating, are in fact of a correlational nature. It should therefore not be ruled out that bingeing causes dieting or that there is a third underlying mechanism which explains the co-occurrence of dieting and bingeing. There are few direct data supporting the causality between dieting and binge eating (Fairburn, Cooper & Cooper, 1986); only two empirical studies were carried out. In the early fifties, Keys and co-workers (1950) studied 36 soldiers on a semi-starvation diet for 24 weeks. After several months the soldiers had lost 26% of their initial weight. When food was available again, after the experiment, the soldiers indulged in severe binge eating episodes. It was not until the amount of body fat was normal again that the excessive food intake stopped. And lately, Wardle & Beales (1988) have studied the effects of dieting on laboratory food intake regulation. Overweight subjects took part in a weight loss treatment program, in which some of them dieted and some did not. After three weeks, only the 'dieters', who had lost more weight than the non-dieters, showed the typical laboratory phenomenon of counterregulation.

These data support the idea that dieting is a sufficient condition for binge eating. However, there is one anomaly which the set point theory can not account for. If a set point for body weight really exists, anorexia nervosa patients are certainly below their set point. However, 50% of the anorexia nervosa patients never binge eat, despite the fact that they are starving themselves. Clearly, being below set point is not a sufficient factor for binge eating.

In sum, retrospective data suggest that dieting increases the chance of binge eating. However, empirical data are scarce and a serious anomaly (50% non-bingeing anorexia nervosa patients) cannot be covered by the set point theory.

When the set point theory is put forward to explain the lab data on counterregulation, the implicit assumption is made that counterregulating, highly restrained subjects are, in fact, underweight or at least significantly food-deprived. In this case the classification of subjects into dieters and non-dieters is crucial. The experimental data on counterregulation are

mainly based on the classification of subjects into dieters and non-dieters by means of the empirically derived Restraint Scale (RS; Polivy, Herman & Warsh, 1978). Lately, however, the RS has been subject to psychometric as well conceptual criticism (Blanchard & Frost, 1983; Johnson, Lake & Mahan, 1983; Ruderman, 1983, 1985b; Stunkard & Messick, 1985; van Strien et al., 1986a, 1986b; Wardle, 1986, 1987a; Ganley, 1988; Heatherton et al., 1988). *Psychometrically*, the RS has been criticized for its factor structure and, *conceptually*, for its seemingly paradoxical entanglement of dietary restriction and overeating. For the sake of relevance, attention will only be paid to the last point of criticism.

Set point theory presumes that counterregulating subjects are below their set point, whereas regulating subjects are not. In laboratory experiments high scorers on the RS, i.e. dieters, were found to counterregulate, in contrast to low scorers, i.e. non-dieters. This presupposes that high scorers on the RS are below their set point weight, whereas low scorers are not. However, the RS has been criticized because the scale may measure both the tendency to overeat and the intention to restrict food intake. Then, there may be a tautological element in the finding of counterregulation in high scorers: they were beforehand selected on disinhibitory tendencies. Heatherton et al. (1988) only found the typical counterregulation pattern when subjects were classified as restrained or unrestrained by the RS, which measures both intention to restrict and overeating (see above). The pattern disappeared when subjects were re-classified on the basis of pure measures of restraint (i.e. the TFEQ: *Three Factor Eating Questionnaire*). Likewise, Wardle & Beales (1987) did not find counterregulation at all in high scorers on another pure measure of restraint (i.e. the restraint subscale of the DEBQ: *Dutch Eating Behavior Questionnaire*). From these lab studies on counterregulation it appears that dieting (and consequently being below the hypothetical set point) is insufficient for predicting counterregulation: what counts in these studies is a combination of intention to diet and an overeating tendency. This issue will be further dealt with in 5.2.

3.3 a lowered 5-HT level¹

3.3.1 5-HT and binge eating

In the previous section it was noted that the restriction of calorie intake and binge eating is certainly not a one-to-one relationship. In this section the possible role of a selective restriction of carbohydrate intake in binge eating is reviewed in detail.

¹ This section is a marginally changed version of the submitted article "Food, Mood and 5-HT" written by A. Jansen, M.A. van den Hout, A. Tuiten & F. ten Hoor.

In the past decade an intriguing hypothesis about serotonin (= 5-hydroxy-tryptamine = 5-HT), depression and carbohydrate craving has gained in popularity. Fernstrom & Wurtman (1972) found a relationship between the composition of meals, i.e. consumption of proteins and carbohydrates, and the synthesis of 5-HT in the rat brain. Eating a meal which is rich in carbohydrates and poor in protein is conducive to the synthesis of central 5-HT, while a meal which is rich in protein and poor in carbohydrates decreases the synthesis of serotonin. In its turn, a low level of 5-HT in the brain is found to stimulate carbohydrate intake, while a high level of this neurotransmitter stimulates the intake of protein.

According to Wurtman and co-workers (1981; 1982; 1984b; 1985; 1986), carbohydrate craving may be caused by a disturbance in this serotonergic regulation. More specifically, they suggest that, since there is an overlap between appetitive and affective disorders, carbohydrate craving *"reflects a misuse of the foods as though they were drugs, i.e., to produce by increasing brain serotonin synthesis, mood changes associated with serotonergic anti-depressants"* (Wurtman & Wurtman, 1986; p.99). Indirect support for this view comes from the often found association between depression and a lowered 5-HT activity (van Praag et al., 1987a, 1987b) on the one hand, and, on the other hand the finding that approximately 70% of the obese carbohydrate cravers satisfy the DSM criteria for major depression (Leckie & Withers, 1967). Also, Stunkard reported about the *'dieting depression'* in the obese as early as 1957. Moreover, Rosenthal & Heffernan (1986) reported that at least a subgroup of depressives periodically overeat themselves, in particular on carbohydrates. Thus, there are some clinical data to support the hypothesized food, mood, and 5-HT connection.

Recently, some authors (Kaye et al., 1984; Kaye & Gwirtsman, 1985; Rosenthal & Heffernan, 1986; Rock & Yager, 1987; Goldbloom, 1987) extrapolated the hypothesis about carbohydrate craving and a lowered serotonergic functioning to bulimia nervosa (BN). Clinical reports, psychometric data and family studies show that BN goes together with depression more often than could be suspected on the basis of chance (Hudson et al., 1982; Leckie & Withers, 1967; Swift et al., 1985; Cooper & Fairburn 1986; Lee et al., 1985). Following Wurtman and co-workers, Rosenthal & Heffernan (1986), Kaye et al. (1984), Kaye & Gwirtsman (1985), Rock & Yager (1987) and Goldbloom (1987) hypothesized that BN and depression may share a decreased activity of the serotonergic system. This hypothesis of hyposerotonergic activity in BN fits in with the observations that dieting is an important factor that predisposes people to the development of binge-eating (Wardle, 1980; Polivy & Herman, 1985; Ruderman, 1986), while the composition of the diet of periodically bingeing people is supposed to be characterized especially by the avoidance of carbohydrates (Beumont et al., 1981; Pope & Hudson, 1984; Johnson et al., 1982; Goldbloom, 1987). Consumption of some carbohydrates by these alleged carbohydrate-avoiding dieters is supposed to lead, almost

unavoidably, to an excessive binge on carbohydrates. It was speculated that the avoidance of carbohydrates by dieters will lower the serotonergic activity in the brain and will end up in a carbohydrate-rich binge in order to heighten the 5-HT level again (Wurtman & Wurtman, 1984a; Kaye et al., 1984; Kaye & Gwirtsman, 1985; Goldbloom, 1987; Goldbloom et al., 1988; Brewerton et al., 1988; Hansen et al., 1988). This hypothesis has been tested by the present author (cf. 5.3).

Furthermore, concerning the 5-HT mood association, carbohydrates appear to have an effect on the improvement of mood (Spring et al., 1987). Schweiger and co-workers (1986b) experimentally confirmed that changes in mood during dieting are dependent on macronutrient intake: mood elevated with increasing carbohydrate proportions of nutritional intake. Thus, though it remains to be established directly if serotonergic pathways are involved, there is at least one experimentally demonstrated association between mood and carbohydrate consumption.

In sum, from a variety of sources there is converging evidence that carbohydrate bingeing and depression are empirically related. It is hypothesized that the linking biological marker for these disorders is a lowered 5-HT transmission. The carbohydrate-binge is thought to be a form of "self-medication" (Kaye et al., 1984; Kaye & Gwirtsman, 1985; Rosenthal & Heffernan, 1986; Wurtman & Wurtman, 1986; Rock & Yager, 1987; Goldbloom, 1987). While some people with 5-HT dysfunctioning will mainly complain of depression and consider their carbohydrate bingeing and weight gain as secondary, others primarily complain of obesity or BN and consider their dysphoria as secondary. In both forms the bingeing of carbohydrates raises the central synthesis of 5-HT and will thus diminish the dysphoria (Wurtman & Wurtman, 1986; Rosenthal & Heffernan, 1986).

State generally, it is hypothesized that a particular disregulated brain state **A** [5-HT deficiency] produces a particular mood state **B**¹ [depressed mood] as well as a particular behavior **B**² [bingeing]. The function of **B**² is considered to restore **A** and thus to improve **B**¹.

figure 2: 5HT model

Here we will concentrate on the assumption that lowered 5-HT induces carbohydrate bingeing, which in its turn raise central 5-HT function. First, this line of reasoning will be critically examined. Secondly, it is discussed how the 5-HT model deals with counterregulation in the lab.

3.3.2 *five critical notes*

increased functionally active 5-HT through bingeing is unproven

In the seventies, it was documented that increasing the ratio of the large neutral amino acid tryptophan (TRP) to the other large neutral amino acids (tyrosin, phenylalanin, leucin, isoleucin, valine, and methionin) raises the TRP, and thus 5-HT, level in the brains of rats (Fernstrom & Wurtman, 1972). The TRP ratio can be heightened either by the consumption of a carbohydrate-rich/protein-poor meal, or by direct administration of tryptophan.

A remarkable and unexpected finding is, then, that oral administration of TRP in obese carbohydrate cravers (Wurtman & Wurtman, 1981; Wurtman et al., 1981b; Heraief et al., 1985) and bulimics (Krahn & Mitchell, 1985) did not have a diminishing effect on their craving for carbohydrates, resp. bingeing behavior. Following the 5-HT hypothesis about carbohydrate bingeing, one would suspect that increasing the plasma TRP ratio, either by consumption of a carbohydrate-rich/protein-poor meal or by administration of TRP, to result in an increased 5-HT turnover and thus in a lowered preference for/intake of carbohydrates. According to Heraief et al. (1985) "The mechanisms that (...) caused the drug (i.e. fenfluramin) to be more effective than the nutrient amino acid, have not been clarified" (p.289). This unexpected finding, that an increased TRP ratio does not have a diminishing effect on carbohydrate bingeing behavior, raises a crucial question: *does eating carbohydrates or directly having TRP injected, really lead to a sufficient heightening of the 5-HT activity in the brain for behavioral effects to occur?* There are several reasons why this can be doubted. Four crucial steps will be discussed: (1) does eating carbohydrates heighten the plasma TRP ratio?, (2) does an increased plasma TRP ratio lead to an increased TRP level in the brain?, (3) does an increased level of TRP in the brain raise the level of central 5-HT?, and (4) is a heightened 5-HT level in the brain really functionally active?

About the first step; it was shown in several studies (Fernstrom et al., 1979; Heraief et al., 1983; Moller, 1985; Lieberman et al., 1986; Schweiger et al., 1986a, 1986b) that changing the composition of a meal indeed alters the plasma TRP ratio in humans. A meal which is rich in carbohydrates and poor in protein will lead to an increased TRP ratio, while a meal which is poor in carbohydrates and rich in proteins will decrease the TRP ratio. Differences in plasma TRP ratios after differently composed meals merely vary between 0.065 and 0.160 (Fernstrom et al., 1979).

The second step deals with what happens with TRP after the plasma ratio has been enhanced, e.g. by direct administration of TRP or by the consumption of a carbohydrate-rich/protein-poor meal. It should be noted that TRP is the precursor of both serotonin and nicotinic acid ribonucleotide. BN is often accompanied by depression, and depression is associated with an enhanced cortisol concentration. Cortisol activates the enzyme TRP pyrrolase (Hoes et al., 1981). Induction of TRP pyrrolase means that more TRP is used for the biosynthesis of nicotinic acid ribonucleotide instead of for the biosynthesis of 5-HT (Hoes et al., 1981). Therefore it is reasonable to argue that in bulimia and depression the intake of carbohydrates or simply loading of TRP does not result in enhanced 5-HT synthesis: TRP pyrrolase activity will be induced and the TRP will especially be used for synthesis of nicotinic acid ribonucleotide instead of for that of 5-HT.

Moreover, administration of TRP may even endanger the synthesis of 5-HT: in the synthesis of TRP to 5-HT, but also in the synthesis of TRP to nicotinic acid ribonucleotide, pyridoxalphosphate is an important co-factor. It is the co-factor of 5-HTP to 5-HT and it is the co-factor of many transformations on the nicotinic acid pathway. When pyridoxalphosphate is only marginally present, which is often the case when people are dieting, and the nicotinic acid pathway is activated, too, e.g. through administration of TRP, especially the TRP 5-HT pathway will be at risk. There will be a decreased transformation of 5-HTP to 5-HT because especially the enzyme 5-HTP decarboxylase is sensitive to depletion of the co-factor pyridoxalphosphate (Scriver & Whelan, 1969).

All in all, it may thus be questioned whether the elevated TRP ratio heightens the synthesis of 5-HT in depressed or dieting subjects.

The third step deals with the question whether an increased level of TRP in the brain raises the level of central 5-HT. Crucial is the supposed involvement of feedback processes in the control of 5-HT release. A change in brain TRP concentrations leads to an (in)activation of the enzyme tryptophan hydroxylation, by which brain 5-HT levels are maintained at normal concentrations. Green (1978) reported that central synthesis of 5-HT is controlled by the amount of released 5-HT: increased 5-HT concentrations in the brain are followed by a decrease in 5-HT synthesis. According to Sanders-Bush (1982), increasing TRP concentration enhances the rate of 5-HT synthesis but does not necessarily enhance 5-HT release. However, Fernstrom (1985) argues that the end-product-inhibition by which the formation of 5-HT is decreased or increased as a consequence of suppression or stimulation of the enzyme TRP hydroxylation is a relatively impotent mechanism of control and that recent data do not support the idea of rate of TRP hydroxylation control by end-product-inhibition. Thus, data concerning the involvement of feedback processes are equivocal.

With respect to the fourth step, whether a heightened 5-HT level in the brain is indeed functionally active, Green (1978) argued that turnover

rates for 5-HT calculated of 5-HT synthesis do not reflect functional activity, since the amine normally is synthesized in excess of functional needs. The amount of 5-HT released is only a proportion of that which is synthesized. There are also indications for relatively stable intraneuronal serotonin pools, which are unavailable for release (Green, 1978; Sanders-Bush, 1982). And Aghajanian (1972) observed that administration of large doses of TRP result in a marked decrease in firing frequency of the raphe neurons. In sum, it might be a rewarding enterprise to unravel the complicated issue of feedback mechanisms, end-product-inhibition, functional activity, and so on.

Finally, concerning the fourth step of a really functionally active 5-HT level in the brain, the observations of Trulson (1985) should be mentioned. By studying the effects of dietary experiments on the functional activity of 5-HT neurons in cats, Trulson (1985) did not find any change in activity of 5-HT containing neurons despite significant changes in brain 5-HT and 5-HIAA content. The increased 5-HT concentration following a TRP diet without competitive neutral amino acids was not functionally active and seemed to be catabolized intraneuronally (Trulson, 1985). Neither did the decreased 5-HT level after a TRP-free diet affect the functionally active 5-HT level. *"The present data suggest that manipulations of dietary TRP does not alter the functional activity of brain serotonergic neurons in contrast to previous hypothesis."* (Trulson, 1985; p. 1071).

To sum up, it has been shown that eating a carbohydrate-rich/protein-poor meal elevates the plasma TRP ratio in people. But there is a plausible alternative pathway for TRP to go; the extra TRP may be used for the synthesis of nicotinic acid ribonucleotide instead of for that of 5-HT. Furthermore, when more 5-HT is synthesized it is important to know if more 5-HT is released, too. There may be feedback mechanisms at work. In addition, it may be doubted whether extra synthesis of 5-HT will be actually functional. There are indications (Trulson, 1985) that heightening the TRP ratio in the periphery does not influence the central 5-HT activity. It is, therefore, doubtful whether eating carbohydrates results in functionally active 5-HT levels in the brain.

It is unclear if carbohydrate craving results from lowered 5-HT, heightened NA, or both

There are indications that noradrenaline (NA) is involved in carbohydrate bingeing. A robust phenomenon was demonstrated by Leibowitz and co-workers (1985a, 1985b). They found that stimulation of the noradrenergic activity in the PVN of the medial hypothalamus resulted in a selective preference for carbohydrates, and an actual 59% increase in carbohydrate intake, whereas the intake of protein showed a decrease of 26%. Leibowitz (1983, 1984) claims that episodes of overeating are accompanied by a general increase in peripheral sympathetic activation, mediated by enhanced NA-ergic functioning.

In addition, it should be noted that administration of TRP in high doses interferes with the entrance of tyrosine into the brain (Growdon, 1979; van Praag & Lemus, 1986). Tyrosine is the precursor of catecholamines (CA = dopamine, noradrenaline and adrenaline). Because tyrosine is a large neutral amino acid, just like TRP, it competes with the other LNAAs, and thus with TRP, for a place on the transport mechanism for uptake in the brain. The ratio of tyrosine to the other LNAAs determines the amount of tyrosine in the brain available for the synthesis of the catecholamines. Oral administration of TRP should lead to enhanced TRP and thus 5-HT levels in the brain but may also decrease the uptake of tyrosine, because of their shared carrier. The decreased tyrosine uptake will result in diminished CA synthesis (van Praag & Lemus, 1986); an oral dose of 2 to 10 g TRP is expected to slow catecholamine synthesis down (Growdon, 1979). Moreover, eating foods which are rich in carbohydrates, increases the synthesis of serotonin (Wurtman & Wurtman, 1984a), but, according to Leibowitz & Shor-Posner (1986a, 1986b) also inhibits the release of NA in the PVN of the hypothalamus.

Thus, eating carbohydrates or loading TRP result in both an increased 5-HT synthesis and a decreased NA functioning in the PVN. Which of them is responsible for a diminished carbohydrate intake remains a crucial question. It may be speculated that the interaction between the NA-ergic and 5-HT systems is of crucial importance; during a binge, heightened NA-ergic activation may be interrelated with diminished serotonergic functioning.

In sum, an increase of the noradrenergic activity in the paraventricular nucleus of the medial hypothalamus may also be related to an increased carbohydrate intake and a lowered intake of protein.

A lowered 5-HT functioning is not required for bingeing

Pharmacological findings suggest that lowering the 5-HT level in the brain is not always needed to induce carbohydrate craving. In tables 2 & 3 the effects of pharmacological interventions are summarized.

It is striking that tricyclic antidepressives as well as MAOIs, which both enhance central 5-HT activity either by inhibiting reuptake or breakdown of the neurotransmitter, have a reverse effect in different populations: in obese carbohydrate cravers and bulimics this enhanced serotonergic functioning leads to a decreased craving for carbohydrates, and even to a significant reduction in frequency of binge-eating episodes (see table 1). This fits in very well with the hypothesis that a decreased 5-HT activity will lead to carbohydrate craving. However, in normals, depressives, as well as in anorexia nervosa patients the enhanced 5-HT activity results in weight gain and even in carbohydrate craving. Clearly, carbohydrate bingeing can also happen when the 5-HT functioning is not reduced. Even when 5-HT levels are pharmacologically enhanced, carbohydrate bingeing may take place. Again, the interaction between the NA-ergic and 5-HT system may be of importance; the reported effects of tricyclics and MAOIs

table 2: interventions which enhance central 5-HT activity in obese carbohydrate cravers and bulimics.

source	subjects	drug	dose/day	outcome
Wurtman & Wurtman 1981*	N=11 normal weight carbohydrate cravers	-TRP -dl-FF -placebo	2 g 20 mg	no change on placebo and on TRP, significant less carbohydrate craving on FF
Wurtman et al. 1981	N=23 obese carbohydrate cravers	-l-TRP -dl-FF -placebo	2400 mg 45-60 mg	no change on placebo and on TRP, significant less carbohydrate craving on FF
Heraief et al. 1985*	N=62 obese	-TRP -placebo	1500 mg	less weight loss on placebo
Abell et al. 1986*	N=40 obese	-fluvoxamine -placebo	≤200 mg	weight loss on drug and on placebo
Mitchell & Groat 1984*	N=32 bulimia	-amitriptyline -placebo	150 mg	binge-eating and vomiting: 52% ↓ on placebo 72% ↓ on drug, difference is not significant
Wermuth et al. 1977*	N=19 bulimia	-phenytoin -placebo	300 mg	number of binges binges decreased during drug use, no relapse on placebo
Sabine et al. 1983*	N=50 bulimia	-mianserin -placebo	60 mg	no difference in binge eating between drug and placebo
Agras et al. 1987*	N=20 bulimia	-imipramine -placebo	≤300 mg	binge-eating: 72% ↓ on drug 43% ↓ on placebo p≤0.05
Hughes et al. 1986*	N=22 bulimia	-desipramine -placebo	200 mg	binge-eating 91% ↓ on drug 19% ↑ on placebo significant
Kaplan et al. 1983*	N=6 bulimia	-carbamazepine -placebo		no effect

continuation table 2

source	subjects	drug	dose/day	outcome
Pope et al. 1983*	N=19 bulimia	-imipramine -placebo	200 mg	binge-eating 70%on drug 0% on placebo significant
Pope et al 1986.	N=10 bulimia	-nomifensine	200-400 mg	binge-eating on drug 50%
Pope & Hudson 1982	N=8 bulimia	-imipramine	300 mg	binge-eating 50%
Nassr 1986	N=5 bulimia	-nomifensine	300 mg	4 patients completely free of bulimia
Moore 1977	N=1 bulimia	-diphenyl- hydantoin -imipramine -amitriptyline	100 mg 150 mg 150 mg	recovery of bulimia only on amitriptyline
Walsh et al. 1985*	N=30 bulimia	-phenelzine -placebo	40-90 mg	significant reduction in binge eating while on drug, no reduction on placebo

* Studies marked with an asterisk were double blind, placebo controlled.

table 3: interventions which lower central 5-HT activity in anorexia nervosa patients

source	subjects	drug	dose/day	outcome
Halmi et al. 1986	N=72 anorexia nervosa: 33 bulimics, 39 restricters.	amitriptyline cyproheptadine placebo	160 mg 32 mg	restricters: weight gain on cyproheptadine, bulimics no weight gain
Vigersky & Loriaux 1977	N=24 anorexia nervosa	cyproheptadine placebo	12 mg	no difference in weight gain

are presumably due to their combined effects on 5-HT and NA metabolism. All tricyclic antidepressives and MAOIs influence both the 5-HT and the NA-system in the brain, the latter usually by means of their metabolites (van Praag & Lemus, 1986).

In sum, pharmacological interventions show that an increase of the serotonergic activity in the brain does indeed sometimes result in a lowering of carbohydrate craving. However, in other studies an opposite effect has been documented: the heightened serotonergic activity even caused a craving for carbohydrates. Clearly, a lowered 5-HT activity is not always necessary to induce a binge on carbohydrates.

It is unclear whether hyposerotonergic activity is sufficient for bingeing

The effect of the administration of cyproheptadine, a 5-HT-antagonist, has been studied twice in patients with anorexia nervosa (see table 4). In the first study, Vigersky & Loriaux (1977) treated 24 anorexia nervosa patients with either cyproheptadine or a placebo, in a doubleblind experimental design. There was no significant difference in weight gain between both groups, so the authors concluded that cyproheptadine is not more effective in promoting weight gain than a placebo is. However, the authors did not match the experimental and placebo group very well. The group receiving the drug was significantly older, had been ill for a significantly longer time and used more methods for loss of weight, like drugs, diuretics and laxatives. In the placebo group, exercise and abstinence predominated. These are precisely those differences which are indicative of the bulimic-restrictor distinction! And treating bulimics with a 5-HT antagonist is not very effective in light of the hypothesis and could have distorted the results. The more so as Halmi et al. (1986) found an interesting differential effect of cyproheptadine for these subgroups (see table 4). Cyproheptadine significantly increased treatment efficiency (=weight gain) for the non-bulimic patients and significantly impaired treatment efficiency for the bulimic patients as compared with groups treated with amitriptyline and placebo. This finding seems to fit in with the hypothesis: in anorexia nervosa patients with bulimia, lowering of the 5-HT activity is not useful for weight gain, presumably because it is already lowered. In restricting patients it can be of significance to lower central serotonergic functioning and heighten carbohydrate intake. Unfortunately, the authors only studied weight changes and did not study changes in frequency of carbohydrate bingeing. Though these results are suggestive as to the issue of sufficiency, a crucial empirical question remains yet unanswered. If the hypothesized mechanism is activated experimentally, would normal healthy subjects then turn into responders, i.e. is it possible to induce carbohydrate craving in normals by lowering their central serotonergic activity?

table 4: interventions which enhance central 5-HT activity in normals, depressive and anorexia nervosa patients.

source	subjects	drug	dose/day	outcome
Nakra et al. 1977	N=6 normals	amitriptyline	100 mg	no change in weight, in 2 subjects craving for sweets and appetite
Paykel et al. 1973*	N=51 depressives	amitriptyline	100-150 mg	weight gain and carbo- hydrate craving
Kendler 1978	N=1 anorexia nervosa	amitriptyline	100 mg	weight gain resulting in obesity
Needleman & Waber 1977	N=6 anorexia nervosa	amitriptyline	75-150 mg	weight gain
Berken et al. 1984	N=40 depression	amitriptyline nortriptyline imipramine	56 mg 37 mg 41 mg	on all drugs preference for sweets, hyperphagia and weight gain
Nakra & Gross- berg 1986	N=2 depression	maprotiline	150 mg	carbohydrate craving and weight gain
Katz & Walsh 1978	N=1 depressive anorexia nervosa	chlorimipramine & TRP		resulted in hypomanic obese

* studies marked with an asterisk were double blind, placebo controlled.

hyposerotonergic functioning is not exclusive for bingeing

A decreased 5-HT turnover in the brain does not merely result in carbohydrate bingeing and nothing else. Major depressive disorders, anxiety disorders, obsessive-compulsive disorders, chronic pain, suicide, aggression, and schizophrenia have all been associated with a lowered 5-HT turnover (van Praag et al. 1987a, 1987b; Herbert, 1987). Clearly, a lowered 5-HT activity in the brain does not correspond to a specific nosological syndrome. Van Praag et al. (1986, 1987) speculate that a disturbed aggression or impulse regulation is the broader behavioral correlate of a low 5-HIAA concentration in the brain.

- 3.3.3 *how does the 5-HT model deal with counterregulation in the lab?*
The 5-HT model predicts that dieters will especially counterregulate after relatively carbohydrate-rich food items to compensate for their lowered

5-HT level. But laboratory research on counterregulation has hardly concentrated on the 5-HT hypothesis. One attempt was made by Huon & Wootton (1987), but their data are uninterpretable; no statistics whatsoever are given and their paper does not warrant further attention.

Some speculations can be made. In typical laboratory studies, the experimental manipulation is usually followed by food especially rich in carbohydrates (e.g. ice cream or sweets). (By the way, it could be argued that, apart from being rich in carbohydrates, this food is also very palatable. But in the few studies available in which the taste test consisted of less palatable carbohydrate-rich food (e.g. crackers: Ruderman, 1985; Baucum & Aiken, 1981; or sandwiches: Polivy, 1976) disinhibition of restraint was also found). It is of interest, and in line with the 5-HT hypothesis, that ice cream had a far more pronounced effect on counterregulation than the consumption of palatable low carbohydrate/high protein food (i.e. nuts: Tomarken & Kirschenbaum, 1984).

In a well-controlled study, Spitzer and Rodin (1987) found that subjects who ate a glucose preload ('fast' carbohydrates, producing a steep increment in blood insulin level followed by a steep decline) subsequently ingested significantly more calories than subjects who were given a fructose preload ('slow' carbohydrates, leading to a gradual increase in blood insulin level without subsequent counterdecline). Subjects who had ingested fructose regulated their subsequent food intake very well, whereas subjects preloaded with glucose 'counterregulated'. The proportion between calories ingested as carbohydrates, fats, or proteins was not influenced by the type of preload. These data suggest that the type of nutrient ingested may determine subsequent food intake because of other metabolic consequences (effects on insulin and blood sugar levels; see also section 3.5) than that following from the proportional division of macronutrients. More data on the issue of macronutrient intake after participation in a laboratory experiment are presented in 5.6.

In sum, the hypothesis stating that a lowered 5-HT level induces a carbohydrate binge and that the binge on carbohydrates is in its turn effective in raising the central synthesis of 5-HT, has limited explanatory power. The first and most important question is whether binges of BN patients are indeed carbohydrate-rich and protein-poor. This question is answered in section 5.3. If so, the second question is whether the effect of a carbohydrate-rich binge is a really enhanced functioning of central serotonergic neurons. The appears to be doubtful. The third point of interest is that carbohydrate intake may also be promoted by NA-ergic stimulation of the PVN in the medial hypothalamus. Fourth, an increase of the serotonergic activity in the brain may also result in a craving for carbohydrates. And fifth, a lowered serotonergic activity in the brain is apparently associated with many kinds of disturbed behavior, and does not exclusively predict a craving for carbohydrates.

Counterregulation in the laboratory has hardly come up in the 5-HT discussion. Minimal and merely speculative data suggest, however, that macronutrient intake, and 5-HT levels, are of no significance when it comes to explaining lab counterregulation. As to the aspect of a lowered 5-HT level being sufficient for bingeing and counterregulation to occur, experimental tests would be welcomed. It will be of interest to know whether a pharmacologically induced lowering of the 5-HT system in normals will result in a "binge" on carbohydrates.

3.4 dysfunctional thoughts

3.4.1 *introduction*

An alternative view on the etiology of uncontrollable binge eating and lab counterregulation is a cognitive one. The primary importance of cognitive factors in the etiology and continuance of eating disorders has been stressed by several authors (Bruch, 1973; Polivy 1976; Garfinkel & Garner, 1982; Fairburn, 1984; Rossiter & Wilson, 1985; Garner, 1986; Ruderman, 1985c, 1986a; Wilson, 1986; Johnson & Pure, 1986; Fairburn, Cooper & Cooper, 1986; Strauss & Ryan, 1988). The cognitive factors may include dysfunctional thoughts (e.g., Fairburn, Cooper & Cooper, 1986), perceptual distortions (e.g., Bruch, 1973; Garfinkel & Garner, 1982), as well as poor interpersonal problem solving (e.g., Johnson & Pure, 1986). However, in the present section, attention will only be focussed on the dysfunctional thoughts because of their alleged direct relevance to binge eating episodes.

Dysfunctional thoughts can roughly be divided into *general* and *specific* dysfunctional thoughts. General dysfunctional thoughts concern matters not directly related to food intake, such as the idea that one has to be perfect in all aspects of behavior. The specific thoughts, on the other hand, *directly* concern food intake behavior. Note that the two may be related; e.g. eating one cookie may be perceived as a total lack of control (*specific idea*: food rich in calories is 'forbidden' food, *general idea*: one must have perfect self-control).

3.4.2 *general dysfunctional thoughts*

The cognitive approach states that the *eating disorder* follows from generally distorted patterns of thought based on erroneous underlying assumptions. Clinicians have repeatedly pointed out the unrealistic thoughts, attitudes, and beliefs concerning body weight, body shape, and eating patterns (e.g. Chiodo, 1987; Fairburn, Cooper & Cooper, 1986). Over-valued ideals of slenderness and rigid food rules are prominent. Self-esteem is evaluated in terms of shape and weight, and success and failure are believed to be largely determined by appearance. Being fat is associated with being a failure, unattractive, and unhappy, while being thin is connected with being successful, attractive, and happy. Such ideas

are, of course, directly related to eating and eating problems but they may be specific instances of the more general assumption that one has to be perfect all-round and have total self-control.

Clinical notes indicate that the bulimic's faulty patterns of thought mainly concern logical errors, such as dichotomous thinking, overgeneralization and errors of attribution (Garner & Bemis, 1982; Garner, 1986; Fairburn, Cooper & Cooper, 1986) (see table 5).

table 5: dysfunctional thoughts relevant to eating disorders

logical error	example	
dichotomous thinking (=all-or-none reasoning)	thinking in extreme and absolute terms	"If I'm not in complete control I lose all control"
overgeneralization	extracting a rule on the basis of one event and applying it to other dissimilar situations	"I used to be of normal weight, and I wasn't happy. So I know gaining weight isn't going to make me feel better"
selective abstraction	basing a conclusion on isolated details while ignoring contradictory and more salient evidence	"I am special if I am thin"
magnification	overestimation of the significance of undesirable consequent events	"I've gained two pounds, so I can't wear shorts anymore".
personalization	egocentric interpretations of impersonal events or overinterpretation of events relating to the self	"I am embarrassed when other people see me eat"
superstitious thinking	believing in the cause-effect relationship of noncontingent events	"If I eat a sweet, it will be converted instantly into stomach fat"

After Garner & Bemis, 1982, without permission.

However, the clinical impressions received little empirical attention. Ruderman (1985c, 1986a) showed that scores on a questionnaire measuring restrained eating patterns (*the Restraint Scale; RS*) and scores on a questionnaire measuring bulimic symptoms (*the Bulimia Test: BULIT*) were significant and negatively related to scores on a questionnaire measuring rationality (*the Rational Beliefs Inventory; RBI*). Thus, the more distorted the eating patterns that were reported, the more rigid, perfectionist and irrational the beliefs that were subscribed to. According to Ruderman (1986b), it is "*consistent with the observation that bulimics tend to react to minor dietary violations or other frustrating situations by*

binge eating" (p. 196) that people scoring high on the RS and BULIT also scored high on the RBI-factor 'reactions to frustrating situations'. However, this *ad hoc* interpretation may be somewhat premature. Strauss and Ryan (1988), for instance, did not find any empirical support for the notion that normal weight bulimics and people with subclinical eating disorders are characterized by a greater number of logical errors (overgeneralization, selective abstraction, catastrophizing, personalization) than normal controls. Please note that a non-eating disordered psychiatric control group was used in none of the studies. Irrationality may be a more general characteristic of (sub)clinical psychopathology and should, therefore, not be considered as etiologically specific straight off.

3.4.3 *general dysfunctional thoughts lead to binge eating via depression*

At first sight, the logical errors that binge eaters often make closely resemble the dysfunctional thoughts found to be associated with depression (Fairburn, Cooper and Cooper, 1986). The co-occurrence of bulimia nervosa and depression (cf. 1.2) has particularly stimulated research on prevalence and neuro-endocrinological aspects (Halmi, 1985; Altshuler & Weiner, 1985; Cooper & Fairburn, 1986; Hinz & Williamson, 1987; Levy et al., 1989) but research has hardly concentrated on the similarity of dysfunctional thoughts. In two studies, however, the hypothesis of shared cognitive dysfunction in bulimia and depression was studied but could not be supported. Schlesier-Carter et al. (1989) controlled statistically for the level of depression and found that the maladaptive cognitions and behaviors in bulimia are not simply expressions of an affective disorder but reflect a distinct disorder (i.e. bulimia). Earlier, Ruderman (1986a) had pointed out that although binge eating subjects often showed a depressed mood, they did not show the typical depressive distortions in thought which characterize people with a primary depressive disorder. Thus, the irrational cognitions found in bulimia nervosa are not identical to the depressive thoughts that characterize primary depressive disorders. Although, no evidence for the similarity between cognitive distortion in bulimia nervosa and in depression could be demonstrated, Laessle et al. (1988) argued that bulimics' depression may follow from the irrational attitudes and beliefs characteristic of bulimia. In particular the pursuit of perfectionism and body dissatisfaction are supposed to lead to depression (Laessle et al., 1988). These cognitive characteristics of patients with eating disorders were really significantly related to depression scores (Laessle et al., 1988; Strauss & Ryan, 1988). Because Greenberg (1986) has shown that it is in particular the co-occurrence of depression and dietary restraint which predicts binge eating behavior, it might be of interest to further investigate the role of depression in binge eating/counterregulation of dieters. Exploration of this issue will be presented in 5.4.

3.4.4 *specific dysfunctional thoughts and binge eating/counter-regulation*

According to cognitive theory, dieters possess a cognitive 'diet boundary' (Herman & Polivy, 1984; cf. 2.5). The start of a binge is held to be related to this boundary: dieters overeat because they think they have passed their diet boundary. The idea of passing the boundary leads to a complete loss of control: *"The slightest transgression from rigidly prescribed dieting leads the patient to conclude that she might as well give in to the urge to eat since perfect self-control has been 'blown'."* (Garner, 1986, p.319). Even minor dietary indiscretions are considered to be indicative of a complete absence of self-control: *"I try not to eat. But if I had pizza, it's over for the day", and "If I ate that much, I might as well go ahead and eat more. Usually it ends up in a binge"* (Chiodo, 1987, p.42). Analogous to the notion that disinhibitive thoughts such as *"I've blown my diet, I might as well continue to eat"* are held to be the antecedent of clinical binge eating behavior, it is also assumed that such disinhibitive thoughts trigger the counterregulatory response in the laboratory (cf. 2.5).

3.4.5 *critical comment*

The notion that disinhibitive thoughts trigger a clinical binge or lab counterregulation has never been tested directly. *"Despite the paucity of supporting evidence, the cognitive explanation for the putative association between dieting and overeating has a remarkable degree of acceptance. This is probably because it is credible to clinicians. Patients with bulimia nervosa describe abandoning their controls after breaking their rules."* (Fairburn, Cooper & Cooper, 1986, p.401).

One of the aims of the present author was to subject the cognitive explanation of counterregulation to direct experimental tests. From the cognitive theory several predictions can be deduced. First, the most straightforward assumption is that dieters showing disinhibitive behavior will be characterized by disinhibitive thoughts such as *"I've blown my diet, I might as well continue to eat"*. By measuring thoughts during behavioral tests, the exact relationship between such 'disinhibiting' thoughts and 'disinhibited' behavior is tested in 5.5.

Second, cognitive theory states that dieters only overeat when they perceive their diet as broken. A logical implication of this assumption is that dieters who do not perceive their diet as broken will not overeat. Data are presented in 5.8.

Third, the cognitive theory has another experimental implication. If dieters have eaten 'too much' in the lab, this lab experience counts as diet breaking and will stimulate high intake immediately afterwards. Note that subsequent counterregulation is a unique prediction which follows from cognitive theory and not from the set point theory (cf. 3.2) and/or the 5-HT theory (cf. 3.3). A test of the prediction that disinhibited dieters will counterregulate outside the laboratory will be described in 5.6.

In sum, cognitive theory postulates a link between dieting and bingeing. Intense body image concern leads patients to try and keep to extreme dietary rules. Dieters' negative self-concept predisposes them to depression, whereas dieting and depression together predict binge eating. Minor deviations from the dietary rules (passing the 'diet boundary') are interpreted as catastrophic and result in abandonment of all self-control. However, there is only meager empirical evidence for the presence of overall distorted cognitions and its relationship to counterregulation. The notion that disinhibitive thoughts are responsible for clinical binge eating or lab counterregulation has hitherto not even been subjected to direct experimental tests.

3.5 an alternative account of counterregulation: classically conditioned compensatory responses

3.5.1 *Introduction*

The set point theory (cf. 3.2), 5-HT theory (cf. 3.3), as well as the idea of cognitive dysfunctions (cf. 3.4) do not explain counterregulation c.q. binge eating satisfactorily (cf. Table 6).

If set point, 5-HT, and cognitive theory are not workable, how can counterregulation be explained?

Some characteristics of counterregulating or binge eating subjects have hardly been discussed, but they may put the phenomenon of counterregulation in a different light. Recently, some authors have pointed out that addicts and dieters share several characteristics: both are characterized by a preoccupation with the substance, a craving for it, a loss of control over intake and relapse (Bemis, 1985; Norré & Vandereycken, 1986; Wardle, 1987c). Moreover, epidemiological data show that bulimia and alcohol addiction often co-occur and are easily interchanged (Brisman & Siegel, 1984; Beary, Lacey & Merry, 1986; Lacey & Mourel, 1986; Jonas & Gold, 1988). Experimental support for the addiction analogy is presented in 5.7.

3.5.2 *conditioned compensatory responses (CCRs)*

The addiction analogy may be useful for explaining counterregulation of dieters. Recent developments in the experimental field of addictions support a classical conditioning model of craving and tolerance. In short, the theory states that, after repeated association of cues specifically related to the substance taken (UCS), these cues (CS) will elicit a physiological response which is opposite to the unconditioned response (UCR) of the substance² (Siegel, 1983, 1984; Siegel et al., 1981, 1982; MacCrea

2 Note that, in 1927, Pavlov referred to this phenomenon in terms of 'paradoxical conditioning'.

table 6: theories: explanations & predictions

theory	explanation of binge eating / counterregulation	comments	specific predictions dealt with in this dissertation
set point	being below set point	why merely counterregulating/ binge eating after exposure to a stimulus? descriptive instead of explanatory	especially unsuccessful dieters will counterregulate
5-HT	having a lowered 5-HT level	why merely counterregulating/ binge eating after exposure to a stimulus? no empirical (lab) data	binges will be relatively rich in carbohydrates
cognitive	perceived break of diet	why counterregulating/ binge eating after mood induction? no empirical data	no binge/counterregulation after smelling a 'preload' counterregulating dieters are characterized by disinhibitive thoughts counterregulation will continue outside the lab

et al., 1987). For example, experiments show that dogs repeatedly injected with epinephrine (UCS) show the usual tachycardia response (UCR) to the epinephrine administrations. The injection ritual is a cue which is specifically associated with the UCS and after a series of injection rituals (CS) and drug (UCS) pairings, the CS has become a reliable predictor of the UCS. At that very moment, dogs display a *compensatory* response in anticipation of the epinephrine: they show a bradycardia in advance (CCR) (see Siegel, 1983). This counterdirectional response is an *anticipative conditioned compensatory response* (CCR): it prepares the organism for the intake of a substance. Compensatory CRs thus contribute much to tolerance: they increasingly cancel the effects of the substance taken, so that the response to a drug decreases in the course of successive administrations (Siegel, 1983). The CCR is supposed to be subjectively experienced as *craving*.

The notion of preparatory responses associated with the intake of food is not new. Years ago the idea already took shape that exaggerated

cephalic³ phase responses (CPRs) to food stimuli stimulated overeating. As will soon be clear, the CPR-model does not explain counterregulation/binge eating adequately. However, CPRs may be related directly to the CCRs. Therefore, attention will first be focussed on the preparatory CPRs, before the compensatory model is outlined.

3.5.3 *cephalic phase responses (CPRs)*

CPRs are the earliest physiological reactions of an organism which is exposed to food (Powley, 1977; Powley & Berthoud, 1985). More specifically, CPRs are predigestive hormonal reflexes which occur in response to food cues such as smell, taste, and appearance. They are fast responses which start in the first minute after stimulus presentation, peak within five minutes and return to baseline within ten minutes (Powley, 1977; Powley & Berthoud, 1985). The responses are of parasympathetic neural origin, are organized in the lateral hypothalamus (LH) and can be inhibited by the activity in nuclei of the ventromedial hypothalamus (VMH) (Powley, 1977). Lesions in the VMH of animals elicit exaggerated CPRs (note that VMH lesions may be followed by hyperphagia and obesity, cf. 3.2).

CPRs play an important metabolic anticipatory role. Functionally, they prepare the organism for the consumption of food by priming the mechanisms that will have to transport, break down, and store incoming nutrients (Berthoud et al., 1981; Tordoff & Friedman, 1989). CPRs include secretion of saliva, insulin, gastric juices, Free Fatty Acids (FFA) mobilization and greater gastric motility (Powley & Berthoud, 1985; Rodin, 1985). Relevant human research has mainly concentrated on the cephalic salivation response.

anticipated salivation responses

Fifteen years ago, Wooley & Wooley (1973, 1975) indicated salivation responses as an allegedly objective and useful new measure of hunger. A general finding is that, in response to palatable food stimuli, subjects show an increase in salivation as compared to baseline salivation levels (Guy-grand & Goga, 1981; Nirenberg & Miller, 1982; Christensen & Navazesh, 1984). However, despite the initial optimism, research data on salivation as an objective measure of hunger are confusing and, at least, equivocal. In various studies, significant correlations between subjective hunger ratings and salivation measures were found (Wooley & Wooley, 1973; Durrant and Royston, 1979; Hodgson & Greene, 1980; Booth & Fuller in Sahakian, 1981) whereas no significant correlations were reported in other studies (Guy-grand & Goga, 1981; Blundell & Freeman in

3 The word 'cephalic' means 'pertaining to the head' and here it refers to the activities of the afferent limb originating in the olfactory, visual and gustatory receptors in the head (Berthoud et al., 1981).

Sahakian, 1981; Nirenberg & Miller, 1982; Christensen & Navazesh, 1984). And, as might be clear from table 7, the salivation response has been found to be sensitive to many factors.

The duration of food deprivation, manner of stimulus presentation, experimental instructions, palatability of the food items presented, caloric content of a preload and the use of appetite suppressant drugs may play a role (Wooley & Wooley, 1973, 1975; Wooley, Wooley & Woods 1975; Wooley, Wooley & Williams, 1976; Sahakian, 1981; Wooley & Wooley, 1981; Herman et al., 1981; Klajner et al., 1981; Legoff et al., 1987, 1988).

dieting, heightened salivation, and counterregulation

CPR theory states that dieting or chronic hunger leads to an overresponsiveness of CPRs which, in fact, reflect an irresistible biological imperative to eat (Johnson & Wildman, 1983; Klajner et al., 1981; Herman et al., 1981). According to the theory, the exaggerated CPRs function as a corrective physiological defense against the energy shortage resulting from food deprivation: oversecretion (exaggerated CPRs) leads the organism to ingest enough calories to restore the metabolic balance.

Thus, CPR theory predicts that (1) dieters will show stronger salivation responses than non-dieters and (2) these exaggerated salivation responses lead to an actually higher intake of food. Let us review the evidence.

table 7: the influence of various variables on salivation response

variable	test situation A	test situation B	salivation	subjects	study
food deprivation	more deprived	less deprived	A = B		Wooley, Wooley & Williams, 1976
	more deprived	less deprived	A = B		Hodgson & Greene, 1980
	more deprived	less deprived	A = B	n=20 -/-	Christensen & Navazesh, 1984
	more deprived	less deprived	A = B	n=19 -/-	Brummer & Pudol, 1981
	more deprived	less deprived	A > B	n=19 -/-	Franchina & Slank, 1988
	more deprived	less deprived	A > B		Wooley, Wooley & Williams, 1976

continuation table 7

variable	test situation A	test situation B	salivation	subjects	study
stimulus presentation	no food stimuli	mere presentation of food cues	A < B		Wooley, Wooley & Williams, 1976
	no food stimuli	mere presentation of food cues	A < B	n=10 -/? n=14 +/?	Guy-Grand & Goga, 1981
	no food stimuli	mere presentation of food cues	A < B	n=25 +/?	Nirenberg & Miller, 1982
	no food stimuli	mere presentation of food cues	A < B	n=20 -/-	Christensen & Navazesh, 1984
	no food stimuli	mere presentation of food cues	A = B		Hodgson & Greene, 1980
	no food stimuli	mere presentation of food cues	A = B		Pangborn, Whiterly & Jones, 1979
	no food stimuli	looking at, sniffing and touching	A = B	n=10 -/-	Brummer & Pudol, 1981
	no food stimuli	olfactory and tactile stimulation	A < B		Pangborn, Whiterly & Jones, 1979
	no food stimuli	tasting the food	A < B	(hungry)	Hodgson & Greene, 1980
	no food stimuli	imagination food	A = B	-/+	Wooley et al., 1978
	no food stimuli	thinking of food	A < B	n=4 2 +/? 2 -/?	Wooley & Wooley, 1973
	no food stimuli	hypnosis	A < B	n=7 -/-	Goldfine et al., 1970
palatability	unpalatable	palatable	A = B	n= ?/+	Klajner et al., 1981
	unpalatable	palatable	A < B	n=20 -/-	Christensen & Navazesh, 1984

continuation table 7

variable	test situation A	test situation B	salivation	subjects	study
preload	high calorie preload	low calorie preload	A < B	n=10 +/-	Wooley, Wooley & Williams, 1976
	high calorie preload	low calorie preload	A = B	n=10 +/-	Wooley, Wooley & Williams, 1976
	high calorie preload	low calorie preload	A = B	n=17?/+ n=8?/-	Wooley, Wooley & Williams, 1978
	high calorie preload	low calorie preload	A > B	n= +/-	Herman et al., 1981
	high calorie preload	low calorie preload	A < B	n=18 +/-	Durrant & Royston, 1979
appetite-suppressant drugs	without drugs	with drugs	A > B		Wooley, Wooley & Williams, 1976
	without drugs	with drugs	A < B	n=14 +/- (hungry)	Sahakian, 1981
	without drugs	with drugs	A > B		Wooley & Wooley, 1981
instructions	expectation to eat	expectation not to eat	A > B	n=2 +/- n=2 -/-	Wooley & Wooley, 1973
	expectation to eat	expectation not to eat	A > B		Wooley, Wooley & Williams, 1976
	expectation to eat	expectation not to eat	A = B	n=20 +/-	Christensen & Navazesh, 1984
	expectation to eat	expectation not to eat	A = B	n=15 +/- n= 9 +/-	Powers et al., 1981
	being forced to eat	non-forced to eat	A < B		Wooley et al., 1981
	being forced to eat	non-forced to eat	A > B		Herman et al., 1981

obesity/restraintness: -/- = non-obese/non-restraint; +/- = obese/non-restraint; -/+ = non-obese/restraint; +/+ = obese/restraint; ? = unknown

dieters showing stronger salivation responses than non-dieters

This prediction is not always supported. In several studies it was found that dieters did salivate more than non-dieters in response to palatable food items (Klajner et al., 1981; Herman et al., 1981; Legoff & Spigelman 1987). In other studies, however, subjects on low-calorie diets showed lower salivation responses than control subjects (Rosen, 1981 en Durrant, 1981 in Herman et al., 1981; Wooley & Wooley, 1981). The inconsistent findings are probably due to the types of dieters participating in the different studies. In the '*low-salivation studies*', dieters were institutionalized and put on a strict diet, whereas dieters were selected for the '*high-salivation studies*' on the basis of their self-report scores on the Restraint Scale (RS). Dieters who are on a strict diet are really food deprived whereas dieters identified by the RS usually alternate periods of deprivation with spells of overeating. In line with this, LeGoff, Leichner, and Spigelman (1988) found that dieters who are on a highly unvaried diet (i.e. anorexia nervosa restricters) salivated significantly less to food cues than dieters on a highly varied diet (bulimic patients). Note that this finding is in flat contradiction with CPR theory, which states that dieting per se results in exaggerated CPRs. Data indicate that successful dieting produces low CPRs (salivation responses) but that unsuccessful dieting, i.e. alternation of dieting and overeating, produces exaggerated CPRs (salivation responses).

the exaggerated salivation response leads to an actually higher intake of food

A crucial prediction of CPR theory is that exaggerated CPRs will be followed by overeating. Animal studies show that the higher the cephalic response of rats before they are placed on a 'snack bar' diet (chocolate chip cookies, salami, cheese), the greater the increase in body weight (Berthoud et al., 1981). The authors assume a causal relationship between the amplitude of CPRs, appetite and likelihood that obesity will develop in the rat, and they stress the similarity to human obesity. However, empirical data on CPRs and human eating behavior is scarce. In contrast to the relationship between subjective hunger and salivation levels, on which the bulk of research has concentrated (see earlier), the correlation between salivation and the amount of food ingested has received surprisingly little empirical attention. So far, two studies were carried out. Klajner et al. (1981) found that heightened salivation in dieters after exposure to palatable food was *not* followed by overeating. Wardle (1987b) found a correlation between subjective hunger ratings and food intake ($r=0.56$) but, like Klajner et al., she failed to find a significant correlation between salivation and food intake. These findings contradict the CPR theory, which predicts that food cues affect physiological reflexes (CPRs) and subsequent behavior.

In sum, empirical studies on the relationship between exaggerated salivation responses and actual food intake do not support the CPR assumption that exaggerated salivation has to be eaten away. Moreover, the findings of low salivation levels in 'real' dieters and high salivation flows in dieters who regularly overeat do not support the assumption of CPR theory that it is deprivation which triggers exaggerated anticipatory responses. They rather suggest that it is the behavior with occasional overeating which results in exaggerated CPRs. What is the cause of this? Acknowledgement of the learned nature of CPRs may be fruitful.

3.5.4 *CPRs are learned responses*

The intake of food (*Unconditioned Stimulus = UCS*) triggers metabolic responses (*Unconditioned Responses = UCRs*). Sight, smell, taste, chewing, or swallowing of food naturally have a temporal contingency with feeding. These stimuli may therefore start to act as *Conditioned Stimuli (CSs)* which can trigger a *Conditioned Response (= CR)*, i.e. a CPR. Obviously (*remember Pavlov⁴*), CPRs are conditioned responses: they can be under control of any arbitrary external stimulus that previously reliably preceded the actual ingestion of food (Wooley & Wooley 1973, 1981; Powley, 1977; Booth, 1981; Berthoud et al., 1981; Rodin, 1985). The mere anticipation of food or sham feeding (i.e. the sight, smell, taste, chewing, or swallowing of food without it entering the gastrointestinal tract) and cognitive processes (e.g. the thought of food or even hypnotic suggestion of food) have been found to elicit responses (CPRs) in order to prepare the organism for the digestion of food (Goldfine et al., 1970; Simon et al., 1986; Powley, 1977; Sahakian et al., 1981; Johnson & Wildman, 1983; Rodin, 1985).

When the learned nature of CPRs is taken into account, i.e. CPRs being classically conditioned responses to cues that have come to predict food intake, it becomes understandable why anorexia nervosa restricters and systematically deprived subjects show decreased CPRs while unsuccessful dieters and bulimics show elevated CPRs (see page). In the case of 'total' deprivation, i.e. a low-calorie and unvaried diet, food cues (CS) are less predictive of extreme food intake (UCS) than in the case of a diet-splurge eating pattern, which is usually found in high scorers on the RS. Clearly, it is not dietary restraint in itself, but it may be the nature of the CS (food cues) UCS (extreme food intake) probability relationship which determines the salivation flow. Note that when someone starts to restrict food intake successfully (e.g. anorectics), food cues (CSs), like smell and sight, become less predictive of food intake (UCS). Classical conditioning theory then predicts an extinction of the salivation CR. In the cases of bulimics or unsuccessful dieters, CSs are regularly followed by

4 In fact, this is the way in which Pavlov and co-workers discovered the principle of classical conditioning (Windholz, 1989).

particularly strong UCSs (large/extreme food intake). UCS strength is a powerful determinant of the strength of the CR. Classical conditioning theory therefore correctly predicts relatively high salivation CRs in bulimics and unsuccessful dieters. Intermittent dieting (diet-splurge) will never fulfill the preconditions necessary for inhibitory conditioning or extinction, whereas real dieting does.

In sum, bingers and addicts share some characteristics. This observation drew attention to a possible common mechanism underlying craving, tolerance, and excessive substance intake. In the case of addictions, as well as in eating behavior, classically learned preparatory responses are relevant. These responses are functional in preparing the organism for the intake of a substance. The idea of learned preparatory responses explains the differences in salivation responses between restricters and bulimics quite well. But can the model also explain counterregulation satisfactorily?

3.5.5 *a new model for binge eating & counterregulation: CCR (hypoglycemia) following from CPR (insulin release)*

One of the anticipatory responses (CPRs) is the release of insulin. Insulin, a small protein produced in the β cells of the pancreas, facilitates the entry of glucose in cells and, in that way, lowers the amount of glucose in the blood. Anticipatory insulin release can be elicited by any stimulus which has temporarily been associated with feeding (Deutsch, 1974). This is convenient: food intake heightens blood sugar level and anticipative insulin release allows a better tolerance of the glucose. Especially the taste of sweetness stimulates insulin release, which subsequently lowers blood sugar level and stimulates appetite (Deutsch, 1974; Berthoud et al., 1981; Geiselman & Novin, 1982). Thus, CSs such as tasting or smelling food trigger a CPR (anticipative insulin release) which, in its turn, elicits a decline in blood sugar level. Normally, food intake heightens blood sugar level: the hypoglycemia in anticipation of food intake is therefore, in fact, *a conditioned compensatory response* (CCR). The hypoglycemia (CCR) may be subjectively experienced as hunger/craving and, in that way, stimulate food intake in order to heighten blood sugar level⁵.

5 Note that the intake of food (UCS) leads to the release of insulin (UCR1) as well as to an increase in blood sugar (UCR2). Apart from insulin being a conditioned reaction (CR) to food stimuli such as the smell of food (CS), the release of insulin may also be considered an unconditioned stimulus (UCS2), leading to a decline in blood sugar level (UCR3). In the strict sense, then, insulin release is a CR *and* a UCS. Although use of the term 'compensatory response' may, therefore, be debatable (Eikelboom & Stewart, 1982) in terms of physiological processes, from a functional perspective the use of the term 'compensatory response' is useful. When the temporal dynamics of the conditioning process are considered the ultimate response is always a decline in blood sugar level.

Empirical data support the notion that it is possible to condition blood sugar level. After repeated intravenous injections of insulin (which are usually followed by a decline in blood sugar level) rats learn to show a hyperglycemic response to a placebo (Siegel, 1972). Rats thus learn to anticipate the insulin administration (and subsequent hypoglycemia) with a compensatory, hyperglycemic, response. And, the other way around, after repeated intravenous injections with glucose, rats learn to show a hypoglycemic response to a placebo (Deutsch, 1974). Rats thus learn to anticipate the glucose administration with a compensatory, hypoglycemic, response. Deutsch (1974) also showed that, in rats, an initially neutral taste cue can elicit a glycometabolic effect if it has been paired with glucose administration in a classical conditioning paradigm. Human obesity has repeatedly been attributed to hyperinsulinemia and the hypoglycemia resulting from it (Geiselman & Novin, 1982; Tordoff & Friedman, 1989). In the literature on bulimics' binge eating, however, the notion of a learned hypoglycemia in anticipation of a binge has hardly been pointed out. Some authors touched upon the idea of hypoglycemia (e.g. Wooley & Wooley, 1985) but were unable to specify the exact, i.e. learned, nature of the alleged hypoglycemia.

Others studied bulimics' glucose and insulin levels after a meal or a glucose injection. The data are equivocal. Normal blood glucose and insulin levels in bulimics were reported by Hohlstein et al. (1986) and Weingarten et al. (1988), whereas Schweiger et al. (1987) reported abnormally high insulin and glucose levels in bulimics who had eaten a meal. It should be noted, however, that the present author's CCR model for binge eating/counterregulation specifies *anticipative* responses and not responses following a meal. Moreover, the studies mentioned were one-off, and did not use explicit CSs, which means that the *learned* nature of hypoglycemic responses was not taken into account. As far as the present author knows, there is only one study on anticipative glucose and insulin levels in bulimic subjects: in line with the hypothesis stated earlier, Thompson et al. (1988) found an elevated insulin/glucose ratio in counterregulating restrained subjects after they had eaten a preload.

The model of conditioned compensatory hypoglycemia may explain counterregulation in the lab and it may have clinical implications.

3.5.6 *CCRs and counterregulation*

The CCR-model is unique in the sense that it has, hitherto, been the only model which offers one unifying and satisfactory explanation for the motley collection of disinhibitors that have been identified. With respect to the explanation of counterregulation after the different disinhibitors, it was noted before (see section 2.3) that counterregulation occurs on several occasions. One single behavior, counterregulation, is repeatedly explained in different ways, depending on which disinhibitor attention is focussed. However, for sake of parsimony one single mechanism that

underlies all the counterregulatory challenges should be looked for. It is hypothesized by the present author that the different disinhibitors all act as conditioned stimuli with an identical consequence:

CSs ('disinhibitors') → CPR (insulin oversecretion) → CCR (hypoglycemia) → counterregulation.

Eating a calorie-rich preload, the perceived breach of a diet, being in a low mood or being anxious are all stimuli which could be conditioned to the excessive intake of food (UCS) in dieters. These CSs may therefore elicit a hyperinsulinemia response (CPR) which, in its turn, will be followed by a hypoglycemia (CCR). The hypoglycemia is subjectively experienced as craving or hunger and, therefore, leads to counterregulation.

As well as covering existing findings quite well *post hoc*, the model also provides straightforward and unique predictions. One implication is that if certain cues (olfactory, taste, visual CSs) are sometimes followed by particularly strong UCSs (high-calorie intake) it is to be expected that unsuccessful dieters will show strong conditioned responses (CCRs) to any cue that typically predicts food intake. It follows that counterregulation in dieters should occur not only if an actual high calorie preload is consumed or anticipated (as e.g. cognitive theory would predict). It is predicted that, in contradiction with any current cognitive theory, counterregulation will also occur when dieters are merely smelling a preload. Data of this *experimentum crucis* are given in 5.8.

3.5.7 CCRs and clinical implications

The clinical implications follow from the assumption that binges result from exaggerated CCRs that are due to probabilistic CS (cue) UCS (extreme food intake) contingencies. It would be expected that the urge to binge is cue-dependent, i.e. binges will occur in stereotyped stimulus configurations. There are clinical impressions that back up this inference and there is a therapeutic implication. If bingeing results from prior CS-UCS contingencies it follows that when the probabilistic connection between CS (cue) and UCS (extreme food intake) is broken, the exaggerated CCRs will be extinguished and so will actual bingeing behavior. Controlled testing of this therapeutic implication is beyond the scope of this dissertation. Meanwhile in section 5.9 a case study is presented of a bulimic patient who was successfully treated along the lines traced out above. Though this *n=1* study is, of course, far from decisive, it at least suggests that the classical conditioning model of CCRs is therapeutically useful and warrants clinical trials.

In sum, the classically conditioned compensatory model appears to provide a promising explanation of binge eating. The neo-Pavlovian response model of binge eating and counterregulation postulates that:

CPRs are conditioned responses to food cues

[hyperinsulinemia is a CPR]

CPRs mediate compensatory responses (CCRs)

[hyperinsulinemia elicits hypoglycemia]

CPRs/CCRs are particularly strong if strong UCSs are presented

[hypoglycemia is particularly strong in the case of intermittent dieting/overeating]

CPRs/CCRs are experienced as hunger or craving

[hyperinsulinemia/hypoglycemia is experienced as hunger or craving]

CPRs/CCRs promotes overeating

[hyperinsulinemia/hypoglycemia promotes bingeing/counterregulation]

3.6 summary

Dieting and bingeing usually co-occur. Even in the eighties, an outdated variant of the set point hypothesis is repeatedly put forward to explain this co-occurrence. The set point theory states that every individual has his/her own, biologically predetermined, 'set point' for body weight. When someone's body weight drops below its alleged 'set point', processes are started up to return to the 'set point'. One of these processes is overeating. Four lines that back up the set point theory were outlined: (1) some people do not show the usual decline in sweet preference after eating a glucose preload, (2) body weight remains relatively constant over long periods and under various circumstances, (3) organisms show a strong resistance to weight loss or weight gain, (4) experimentally induced new body weights are also stable and are defended effectively. However, researchers' application of the set point theory to counterregulation can be criticized for several reasons: (1) the decrease of perceived pleasantness after intake of a sweet solution is not always supported in dieters and the relevance to the alleged set point weight is doubtful, (2) the literature promotes the idea that data have been selected *post hoc*, (3) there is some circular/homuncular reasoning when it comes to determining someone's set point for body weight, (4) the model lacks preciseness: a set point can relatively easily be changed by diet composition and/or exercise level. Especially when it comes to explaining the lab data on counterregulation, three problems of using the theory emerge: (1) it is untestable whether counterregulating dieters, who are usually *unsuccessful* dieters, are below their set point for body weight, and (2) some dieters counterregulate and other dieters do not, which depends on the presence of a 'disinhibitor', (3) there are also *successful* dieters. Because of this, auxiliary hypotheses will always be needed. It is con-

cluded that the 'set point' notion is not a satisfactory explanation of counterregulation.

Because of the fact that not all dieters overeat, attention is turned to the selective restriction of carbohydrate intake, instead of to the restriction of total calorie intake. The restriction of the relative carbohydrate intake lowers central 5-HT level. This diet-induced hyposerotonergic functioning is assumed to lower mood and to elicit a carbohydrate-rich binge: by eating a relatively large amount of carbohydrates during a binge, 5-HT level should be raised. In this way, mood will also be improved. However, several critical comments can be given on the 5-HT idea: (1) the assumption that bingeing is functional in raising the 5-HT level is doubtful, (2) it is unclear whether carbohydrate craving results from a lowered 5-HT level, a heightened NA level, or the combination of the two, (3) a lowered 5-HT level is not needed to induce bingeing, (4) it is unclear whether a lowered 5-HT level is sufficient for bingeing and (5) hyposerotonergic functioning is not sufficient for bingeing; other neurotic disorders (e.g. the obsessive compulsive disorders) have also been associated with a lowered 5-HT turnover. The 5-HT hypothesis originally focussed on bulimics' binge eating; there is no laboratory research on counterregulation and the 5-HT model.

Just like the set point hypothesis, the 5-HT model does not explain the observation that dieters do not always overeat, but merely do so after the introduction of a so-called 'disinhibitor'. Cognitive theory, on the other hand, postulates an explanation for the 'disinhibition' of merely challenged dieters. According to the cognitive idea, dieters perceive their diet as broken after 'disinhibition' whereas non-disinhibited dieters, of course, do not. The perception of having broken one's diet is supposed to be a stimulus to overeating more. However, the notion that disinhibitive thoughts such as *"I've blown my diet, I might as well continue to eat"* trigger an overeating response has never been tested. Furthermore, cognitive theory fails to explain dieters' overeating after mood induction satisfactorily.

An alternative model was presented. The underlying idea is that counter-regulation/binge eating responses are cue-controlled. After organisms have been confronted with stimuli that are probabilistically related to subsequent food intake, they show anticipative physiological responses (e.g. salivation) which prepare the organism for the intake of food. These physiological anticipatory responses are classically conditioned responses to cues. The learned anticipatory responses will be stronger in proportion to the extent to which the cues are predictive of large food intake. One of the anticipative responses is the release of insulin. Insulin, released in anticipation of the food cues, leads to a decline in blood sugar level. This decrease is viewed as a compensatory response: after eating, the blood sugar level will increase. It is therefore convenient to lower blood sugar level in advance, especially when cues predict large intake. The classically conditioned compensatory response (hypoglycemia) will

subjectively be experienced as craving or hunger and will stimulate overeating in order to restore blood sugar level. The model explains why dieters overeat after various disinhibitors: all these challengers act as cues which elicit the preparatory (insulin release) and compensatory responses (decline in blood sugar level).

4 overview of the empirical studies

Dieting and bingeing often co-occur. In the laboratory, dieters counter-regulate after a challenge whereas non-dieters do not. A current view is that dieters overeat because they restrict their food intake and are chronically hungry because of that (cf. 3.2). Section 5.1 (clinical and non-clinical binges) describes an investigation of the question whether hunger really is an important antecedent of a binge. Furthermore, when subjects are classified as dieters or non-dieters, the pitfall of tautology should be avoided: classification is usually done by means of the Restraint Scale (RS), which has been found to entangle the intention to diet and the tendency to overeat. Therefore, the question is whether it is the intention to diet or the tendency to overeat which is important in the occurrence of counterregulation. This question is answered in 5.2 (non-regulation of food intake in restrained, emotional, and external eaters). The studies showed that hunger is not a determinant of bingeing (cf. 5.1) and that successful dieting is not a prerequisite for counterregulation to occur (cf. 5.2).

Clearly, actual restriction of calorie intake is not a crucial factor in the occurrence of binge eating & counterregulation. This observation drew attention to the selective restriction of carbohydrate intake (cf. 3.3). The underlying idea was that dieters usually deprive themselves of the intake of carbohydrates (accompanied by a relatively high protein intake) and are in this way characterized by a lowered central 5-HT level. During a binge, dieters allegedly ingest a large amount of carbohydrates (and little protein) in order to restore 5-HT level. In section 5.3 (does bingeing restore bulimics' alleged 5HT-deficiency?) this was investigated. No support was found for the notion that dieters usually ingest relatively little carbohydrates and especially binge on carbohydrates.

Thus, the notions of hunger, actual restriction of calorie intake, and a selective carbohydrate restriction could not be supported in the studies described in chapter 5. Attention was focussed on the part that mood plays. Section 5.4 (Is counterregulation of dieters after forced consumption of a preload dependent on mood?) describes an investigation of the question whether counterregulation of dieters after forced consumption of a preload depends on mood. It did not.

It is widely held that dieters overeat after a challenge because they perceive their diet as broken (cf. 3.4). In section 5.5 (cognitions and self-talk during food intake of restrained and unrestrained eaters) it was tested whether counterregulating dieters think in a more disinhibitive way than non-dieters and non-disinhibited dieters do. They did not. Neither

was any evidence found for the cognitive notion that, after breaking their diet in the lab, subjects will overeat for the rest of the day (cf. 5.6: no evidence that dieters counterregulate outside the laboratory).

Dieters were not characterized by so-called disinhibitive cognitions but they did report a loss of control during the confrontation with food. This loss of control was reminiscent of addictivelike behavior (cf. 3.5). Some authors touched upon the idea, but no experimental tests on the addiction analogy have been carried out so far. In section 5.7 (restrained eaters are rapidly habituating sensation seekers), experimental support was found for the notion that overeating can be viewed as addictionlike behavior. Restrained eaters habituated more rapidly to a series of neutral stimuli than unrestrained subjects did. Moreover, restrained eaters showed higher sensation seeking tendencies than unrestrained eaters. These two tendencies have been found to characterize addicts.

I have been guided by these findings. Recent experimental research on the etiology and maintenance of addictions underline the learned nature of craving and tolerance. An analogous model of classically conditioned compensatory responses was outlined for counterregulation & binge eating (cf. 3.5). Among other things, this model predicts that cues (CSs) reliably and specifically associated with the intake of large amounts of ('forbidden') food (UCS) will elicit craving and an overeating response in dieters. In section 5.8 (on being led into temptation: 'counterregulation' of dieters after smelling a 'preload') the hypothesis was supported that dieters also counterregulate after a CS which reliably predicts food intake (the smell of food), but that has nothing to do with breaking the diet (which was most important according to cognitive notions). In 5.9 (a case of bulimia successfully treated with cue-exposure) it is demonstrated that breaking the CS-UCS connection may be an effective treatment of bulimia.

5 empirical data

11/11/2019

5.1 clinical and non-clinical binges

Anita Jansen¹, Marcel van den Hout¹ & Eric Griez²

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abstract

In the present study, it was investigated if and in what respects clinical binges (reported by bulimic subjects) are different from non-clinical binges (reported by a non-clinical population). Furthermore, the study prospectively tested whether the DSM-III-R criteria and other variables that are retrospectively claimed to be defining characteristics indeed differentiate binges from non-binge eating episodes. Clinical binges appeared to differ from non-clinical binges as to amount of calories ingested and duration. With respect to subjective experience, clinical and non-clinical binges did not differ. The DSM-III-R criteria for binge eating discriminate clinical and non-clinical binges quite well from non-binges. With the addition of other seemingly important variables, binges are not discriminated better from non-binges than with the DSM-III-R variables alone. It is argued that the difference between pathological binge eating and non-clinical binge eating is mainly a matter of severity.

introduction

An essential feature of the eating disorder Bulimia Nervosa (BN) is the recurrence of binge eating episodes. Binge eating refers to the rapid consumption of a large amount of food in a discrete period of time, during which a lack of control over eating behavior is experienced (APA, 1987). Apart from these DSM-III-R criteria for binge eating, other characteristics of a binge, as well as its antecedents and its consequences, are held to be important defining characteristics. Before a binge, 40% to 59% of the patients should feel bored and depressed (Johnson et al., 1982; Abraham & Beumont, 1982), 78% of the patients should crave for food and 44% should feel hungry (Abraham & Beumont, 1982). During a binge, many

1 Dept. of Mental Health Sciences / Experimental Psychopathology, Limburg University, P.O. Box 616, 6200 MD Maastricht, the Netherlands

2 Dept. of Clinical Psychiatry / Experimental Psychopathology, Limburg University, the Netherlands.

patients should stuff the food into their mouth and eat too quickly to taste anything (Abraham & Beumont, 1982; APA, 1987). And, following a binge, a depressed mood is often prominent (Cooper & Bowskill, 1986; APA, 1987).

It is obvious that especially the DSM-III-R criteria for binge eating (rapid consumption, large amount of food, and the lack of control over eating behavior) are subject to a very personal interpretation. Eating much, eating rapidly, and finding it difficult to stop eating once started is not at all abnormal; non-psychiatric subjects also experience eating binges (Wardle & Beinart, 1981; Rand & Kulda, 1986). *Are there any differences between pathological binge eating and binge eating that is of no clinical significance?* So far there are no data on this issue. Therefore, the first aim of the present study was to investigate whether 'non-clinical' binges (reported by non-bulimic subjects) are different from 'clinical' binges (reported by bulimic subjects) with respect to amount of calories ingested, duration, and subjective experience.

Furthermore, the DSM-III-R criteria and the characteristics of a binge that were listed above were obtained retrospectively from clinical reports but have never been tested prospectively. The second aim of the present study was, therefore, to determine prospectively whether the DSM-III-R criteria and/or the other characteristics listed above can indeed differentiate bulimic, as well as normal binges from non-binge eating episodes.

method

subjects

Data on *clinical* binges were obtained from 9 female subjects with BN who were awaiting cognitive-behavioral therapy at the local psychiatric hospital. They were diagnosed as suffering from BN by the hospital staff and the first author according to DSM-III-R criteria. Data on *non-clinical* binges were obtained from 19 female subjects and 1 male subject who reacted to a call for binge eaters in the university newspaper¹. The subjects were interviewed by the first author to make sure that they did experience (self-defined) binge eating episodes but did *not* satisfy the DSM-criteria for any of the eating disorders: it was made sure that the normal subjects did not exhibit such a persistent over-concern with body shape and weight as bulimic patients do. Furthermore, they only sporadically engaged in self-induced vomiting and never used laxatives and/or diuretics. They gave the impression that they did not suffer from their binges as much as patients did.

1 The text of the advertisement word to word run as follows: "Do you have eating binges regularly ? And are you willing to take part in a questionnaire survey for a consideration? Everything is confidential and anonymity will be guaranteed."

table 1: subject characteristics

	patients n=9	normals n=20	t	p
BMI ¹	21.6 (6.9)	21.7 (2.4)	-0.1	NS
age	27 (4.5)	22.2 (3.5)	3.1	<0.01
time since onset of binges (years)	7.7 (4.0)	4.2 (3.0)	2.6	<0.05
total restraint score	24.3 (5.4)	16.6 (5.6)	3.5	<0.01
concern for dieting	14.3 (4.5)	10.1 (3.9)	2.6	<0.05
weight fluctuations	10 (4.9)	6.5 (2.9)	2.5	<0.05
depression score (BDI)	27.3 (6.9)	6.1 (4.9)	9.5	<0.001

¹ BMI = Body Mass Index (= weight/height²)

Subject characteristics are presented in table 1.

assessment

The following instruments were used:

Restraint Scale (RS; Polivy et al., 1978); measuring (a) Concern for Dieting (CD), shown in e.g. attitude towards eating and frequency of dieting and (b) Weight Fluctuations (WF).

Beck Depression Inventory (BDI) measuring level of depressed mood.

Eating Diary in which subjects wrote down everything they ate and drank during 7 days. The Eating Diary asked for the time and duration of eating; the sort and amount of food ingested; whether it was a binge, a normal meal, or a snack; and whether consumption was followed by vomiting or the intake of laxatives.

Pre-eating State Form. An eating episode was defined as a period of eating marked off in time. Before each eating episode, subjects indicated whether they were bored, depressed, hungry, and craved for food on a 0 ('not at all') to 100 mm ('extremely') Visual Analogue Scales (VAsS).

Post-eating State Form. After each eating episode subjects wrote down what and how much they had eaten and whether it was a binge or not. On 0 ('not at all') to 100 mm ('extremely') VASs they noted how depressed they were, whether they had eaten much, whether they had eaten more than planned, whether they had eaten rapidly, whether they had stuffed themselves with the food, whether they had tasted the food, and whether they had lost control over their eating behavior.

The variables on the Pre and Post-eating State Forms correspond with the DSM-III-R criteria and the characteristics/antecedents/consequences held to be important by several authors (see introduction).

procedure

All subjects were interviewed by the first author. At the start of the interview subjects filled in the RS and the BDI. During the interview, information concerning eating pattern, eating problems, time of onset, vomiting, use of laxatives, height, weight, concern with weight and appearance was gathered. At the end of the interview it was explained how they should fill in the eating diary and the pre- and post-eating forms. Subjects kept the eating diary for 7 days. From the morning of the 5th day on, they also filled in the Pre- and Post-eating State Forms before and after the first 10 eating episodes. After the registration period the subjects returned the eating diary and the forms. Unclear issues were discussed.

analysis

1. The eating diaries provided 86 binges of patients and 106 binges of normal controls. To investigate whether 'non-clinical' binges are different from 'clinical' binges in caloric content and duration (question 1), mean calorie intake during a binge, mean duration of a binge, and mean amount of calories ingested per minute during a binge were calculated for every subject. With t-Tests it was tested whether groups differed as to these variables.

2. 32 binges of 9 patients, 43 non-binges of 8 patients, 33 binges of 17 controls and 137 non-binges of 20 controls were gathered by the forms. On the remaining forms (15 of the patients, i.e. 16%, and 30 of the normals, i.e. 15%) subjects were unable to answer the question whether the eating episode was a binge or not: they stated that these were 'semibinges'. The semibinges were not included in the data analysis (it would be interesting to further investigate the phenomenon '*semibinges*'). To investigate whether 'non-clinical' binges are different from 'clinical' binges with respect to subjective experience (question 1) the mean score for the 11 variables (boredom, depression before eating, hunger, craving, depression after eating, eating much, more than planned, rapidly, stuffing the food into the mouth, tasting the food, and the loss of control over eating behaviour) were calculated for binge eating and non-binge eating episodes for each subject. On the mean scores Analyses of Variance

(ANOVAs) were carried out. Main Factors were Group (Patient vs. Controls) and Eating Episode (Binge vs. Non-binge). Because the forms did not obtain non-binge eating episodes from all patients and binge-eating episodes from all normals, the ANOVAs were carried out on the means of 8 patients and 17 control subjects.

3. To determine prospectively whether the DSM-III-R criteria and the other characteristics listed above are reliable predictors of clinical and non-clinical binges (question 2), a factor analysis was carried out on the 11-item form scores of the 75 patients' binges and non-binges. The factor analysis provided only one factor (Eigenvalue 4.3). Only the variable 'hunger' did not contribute to the factor. Therefore, sum scores over all variables except for 'hunger' were used in a subsequent Discriminant Analysis. By means of the patient's Discriminant Analyses (over all factors as well as only over the DSM factors) it was investigated whether normal, i.e. non-clinical, binges could be discriminated from normal, i.e. non-clinical, non-binges.

results

are non-clinical binges different from clinical binges (question 1) with respect to calorie intake and duration?

In table 2 mean calorie intake during a binge, mean duration of a binge, and mean calorie intake per minute during a binge are given for patients (clinical binges) and normals (non-clinical binges) respectively. Clinical binges were characterized by a significantly larger calorie intake and they lasted longer than non-clinical binges. Mean amount of calories ingested per minute during a binge was not different in the two groups.

table 2: quantity of clinical and non-clinical binges.

	clinical binges n=9	non-clinical binges n=20	t	p
calories eaten during a binge	2631.7 (1347.1)	985.7 (512.6)	4.8	<0.001
duration of a binge (minutes)	55.7 (32.4)	33.5 (18.9)	2.1	<0.05
calories eaten per minute during a binge	60.6 (28.8)	44.9 (17.6)	1.7	NS

are non-clinical binges different from clinical binges (question 1) with respect to subjective experience?

Data are represented graphically in Fig. 1.

There were obviously clear differences between binges and non-binges. As to the difference between patients and controls: a similar pattern emerged. Subjectively controls experienced the same as patients, but on a lower level. All parameters were tested with a two-way ANOVA. Results are given in table 3.

table 3: F-values of the ANOVAs carried out on the 11 variables.

boredom	depression before eating	craving	hunger	eating much	eating more than planned	taste	rapidity of eating	stuffing the food	depression after eating	loss of control
main effect for group										
8.6**	16.2***	5.3*	NS	NS	NS	6.2*	9.3**	18.0***	37.7***	12.6***
main effect for eating episode										
10.8**	5.1*	30.7***	NS	123.3***	44.1***	16.6***	25.1***	37.9***	12.0***	80.0***
interaction effect group X eating episode										
NS	NS	NS	NS	NS	NS	NS	NS	6.3*	NS	7.9**

* = $p < 0.05$; ** = $p < 0.01$; *** = $p < 0.001$

As can be seen, main effects for Group emerged on boredom, craving, depression before eating, depression after eating, taste, eating rapidity, stuffing the food into the mouth, and loss of control. On these variables, patients scored higher than the controls, irrespective of the binge/non-binge dichotomy.

Main effects for Eating Episode emerged on all variables, except for hunger, indicating overall differences between binges and non-binges. Interactions would be of particular interest, as they would reflect different *patterns* in eating episodes for patients and controls. However, merely two interaction effects emerged (on 'stuffing the food' and 'controllability') and both were due to the difference between binges and non-binges being greater for patients than for normals.

do the DSM-III-R criteria and/or the other characteristics listed above predict clinical and non-clinical binges? (question 2)

With the aid of the Discriminant Analysis over the 10 variables, 90.7% of the patients' eating episodes could be classified correctly as a binge or a non-binge eating episode; 28 out of the 32 binges (87.5%) and 40 out of the 43 non-binges (93%) were classified correctly. By means of only the

figure 1: the subjective experiences before, during and after a binge or non-binge for 8 patients and 17 controls.

3 DSM-III-R criteria (eating much, eating rapidly, and losing control), 89.3% could be classified correctly (28 binges (87.5%) and 39 non-binges (90.7%)).

The patients' Discriminant Analysis over all variables predicted merely 10 out of the 33 non-clinical binges (30.3%) correctly, whereas all non-binges were predicted correctly. By application of the patients' DSM-III-R Discriminant Analysis to the eating episodes of the normals, 19 out of the 33 binges (57.6%) were classified correctly, whereas 5 out of the 137 non-binges (3.6%) were wrongly classified as binges.

discussion

The present study led to four main findings. First, clinical binges (reported by bulimic subjects) differ from non-clinical binges (reported by 'normal' binge eaters) as to amount of calories ingested and duration. During clinical binges more calories are ingested and the binges take more time than non-clinical binges.

Second, clinical binges are not convincingly different from non-clinical binges as to quality. The same pattern of subjective experience emerged. Both clinical binges and non-clinical binges are preceded by more boredom, depression and craving than non-binge eating episodes. During clinical, as well as during non-clinical binges, less is tasted, food is eaten more rapidly, subjects stuffed themselves more with the food and more loss of control is experienced than during non-binges. After a binge, clinical as well as normal subjects are more depressed and they both report that they ate more, and more than planned, than when they were not having a binge. Except with respect to the last two variables (eating more and eating more than planned) patients merely differed from normals in the greater intensity of these feelings. Patients experienced more boredom, depression and craving before eating. They tasted the food better, ate more rapidly, and stuffed themselves more with the food than normals did. They also lost more control during eating and, afterwards, they felt more depressed.

Third, the DSM-III-R criteria for binge eating (rapid consumption of a large amount of food during which a lack of control over eating behavior is experienced) discriminate clinical binges quite well from non-binges. The addition of other variables recognized as important by clinicians (boredom, depression, craving, tasting, eating more than planned, and stuffing the food into the mouth) did not provide a better prediction than the DSM-III-R variables alone.

Fourth, with the aid of the patients' Discriminant Analysis (including all variables) not even one-third of the non-clinical binges could be predicted correctly. When only the DSM-III-R variables were used in the Discriminant Analysis, considerably more non-clinical binges (58%) could be classified correctly. The latter supports the relevance of the use of the

DSM variables, and *not* the other ones, for identification of a binge. In addition to the data of the ANOVAs and t-Tests, the Discriminant Analysis (with DSM variables) implies that the difference between pathological binge eating and binge eating that is of no clinical significance is mainly a matter of severity, that is, clinical binges are more extensive and are preceded/ accompanied/ followed by more negative feelings.

Assuming that severity is the main discriminant of pathological binge eating and binge eating that is of no clinical significance, the question may be asked what makes a binger into a bulimic. Patients did not only differ from normal subjects in having an eating disorder, but also in level of restraint, period after onset of eating binges, age, and depression. Although normal subjects were highly restrained, patients were significantly more restrained. Could high restraint in patients explain the patient/control differences observed? It has been argued that extreme dieters overeat because they are chronically hungry (Polivy & Herman, 1985) and that more extreme bingeing follows from more extreme hunger as a consequence of more extreme dieting. However, in the present study, hunger did not contribute to the 'binge' factor identified by the factor analysis. Further analysis of 'hunger' showed that no more hunger was experienced before a binge than before a normal eating episode, neither by patients nor by normals. Clearly, bingeing has nothing to do with being hungry.

Patients were older and had suffered from binge eating for a longer period than normals. The transition from non-clinical into clinical problems might progress along a continuum. This would imply that some of the as yet non-clinical subjects will be clinical subjects in a few years. It would be interesting to identify exactly which factors contribute to a development from non-clinical into clinical. The fact that patients were much more depressed than normal subjects does not mean that depression is an important factor. The present study had a correlational nature and, therefore, comes quite close to the chicken-and-egg problem. The understanding of the two issues, the development of eating disorders from non-clinical into clinical and the role depression plays in this, would benefit from more empirical attention.

In sum, clinical binges differ from non-clinical binges in duration and the amount of calories ingested, but they are not convincingly different with respect to subjective experience. The DSM-III-R criteria for binge eating discriminate both clinical and non-clinical binges quite well from non-binges. The addition of other seemingly important variables does not discriminate binges better from non-binges than the DSM-III-R variables alone do. The difference between pathological binge eating and binge eating that is of no clinical significance appears to be mainly a matter of quantity and intensity along a continuum.

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Nonregulation of Food Intake in Restrained, Emotional, and External Eaters

Anita Jansen,¹ Jaap Oosterlaan,¹
Harald Merckelbach,¹ and Marcel van den Hout¹

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In the present study, regulation of ice cream intake after a preload was examined in 20 high and 20 low scorers (median split) on subscales of the Restraint Scale (RS) (Herman, Polivy, Pliner, Threshold, & Muncie, 1978) and the Dutch Eating Behavior Questionnaire (DEBQ) (van Strien, 1986). For each subscale it was hypothesized that low scorers would regulate their ice cream intake. High scorers on "disinhibitive" scales (i.e., the RS and the DEBQ-emotional and -external subscales) were expected to counterregulate after a preload. In contrast, high scorers on the "inhibitive" DEBQ-restraint scale were expected to regulate their ice cream intake. A robust effect was found: high scorers never regulated ice cream intake after a preload on either the disinhibitive or the inhibitive scales, while low scorers always did. For the RS a significant interaction effect was found [$F(1,39) = 4.97, p = .03$]. None of the other subscales showed interaction effects. The t tests showed that low scorers on all subscales ate significantly less after a preload than without one, while high scorers on all subscales did not differ in intake after a preload or without one. This nonregulation appears to be characteristic of people preoccupied with caloric intake, regardless of the content of the preoccupation.

KEY WORDS: nonregulation of food intake; restrained eating; external eating; emotional eating.

¹Department of Mental Health Sciences/Experimental Psychology, Limburg University, P.O. Box 616, 6200 MD Maastricht, the Netherlands.

INTRODUCTION

A series of laboratory studies revealed that high scorers on the Restraint Scale (RS) (Herman, Polivy, Pliner, Threshold, & Muncie, 1978) eat more ice cream after a preload of milkshakes than without a preload, while low scorers eat less after a preload than without one (Herman & Mack, 1975; Hibscher & Herman, 1977). Restraint theorists claim that this so-called "counterregulation" is due to disinhibition of cognitive dietary self-control or "restraint" (Ruderman, 1986). As a consequence of their restrained eating style, restrained eaters are supposed to have a body weight below their biological body weight "set-point." By disrupting their restraint by taking a preload of, for example, milkshakes, they tend to indulge in a biological urge to overeat. However, to consider the experimental results on counterregulation as solid proof of restraint theory is, for several reasons, premature. First, several factor-analysis studies have identified two subscales of the RS: "concern for dieting" (CD) and "weight fluctuations" (WF) (Blanchard & Frost, 1983; Ruderman, 1985; Wardle, 1986). Therefore, the RS cannot be seen as a pure measure of restraint. It remains to be determined whether counterregulation in samples scoring high on the RS is due to concern for dieting, weight fluctuations, or a combination of the two. Second, Wardle (1987a) and van Strien (1986) argue that inspection of the RS items reveals that it is not restraint, as such, that may be measured but rather unsuccessful dieting or disinhibitive restraint. The observation that high scorers on items such as "Do you eat sensibly in front of others and splurge alone?" and "Do you have feelings of guilt after overeating?" are in fact counterregulating in laboratory experiments underlines the predictive value of the RS. The Wardle (1987a) and van Strien (1986) reinterpretation of the RS raises an interesting issue about the association between high RS scores and counterregulation. Is it high restraint per se that predisposes to counterregulation, as restraint theorists claim, or does only a subgroup of high-restrained eaters counterregulate, as the Wardle and van Strien view suggests? More specifically, following Wardle and van Strien, one wonders if unsuccessful dieters, as identified by the RS, may counterregulate in laboratory experiments, while successful dieters may, in laboratory situations, remain able to resist the temptation to overeat. Indirect support for this interpretation of the RS/counterregulation data comes from recent experiments by Shrager *et al.* (1983; cited by van Strien, Frijters, van Staveren, Defares, & Deurenberg, 1986) and Wardle and Beales (1987). By using the Three Factor Eating Questionnaire (TFEQ) of Stunkard and Messick (1985), Shrager and co-workers demonstrated that it was not the "cognitive restraint of food intake" (factor I) but the "disinhibition of cognitive restraint" (factor II) that was associated with overeating (van Strien *et al.*, 1986). Wardle and Beales used the restrained subscale of the Dutch Eating Behavior Questionnaire (DEBQ) as a purer measure of restraint; high

scorers on the DEBQ-restraint subscale may be considered successful dieters (van Strien, 1986). And in spite of the high correlation between the DEBQ-restraint subscale and the RS (.72; see Wardle, 1986) and its validity in a British population (Wardle, 1987a), Wardle and Beales (1987) could not replicate the earlier laboratory findings of counterregulation by using the DEBQ-restraint subscale. Wardle and Beales (1987) found no difference in regulation between high and low DEBQ-restraint scorers. However, reanalyzing their data showed that their high-restrained subjects did not regulate after the preload [$t(23) = .71$, NS], while their low scorers counterregulated even after a preload [$t(27) = 1.96$, $p < .05$]. Counterregulation in low scorers is in sharp contrast with what was found in comparable studies. This curious behavior of low scorers, plus the fact that the ice cream intake in this study was extremely low in all groups, suggests that the Wardle and Beales findings were derived from an atypical subject sample. This study needs replication before firm conclusions can be drawn.

The DEBQ has proven to give valid indications of restrained, as well as emotional and external eating (van Strien, 1986; Wardle, 1987a). While the DEBQ-restraint subscale measures successful inhibitive eating, the emotional and external eating subscales measure disinhibitive eating patterns. Therefore, one would expect "disinhibitive eaters" (i.e., emotional and external eaters) to show the disinhibitive eating pattern of counterregulation. On the basis of the above-mentioned considerations, it was hypothesized that (1) high scorers on scales measuring disinhibitive eating patterns (i.e., the RS, DEBQ-emotional, and DEBQ-external scales) would counterregulate, while low scorers on these scales would regulate after a preload, and (2) both high and low scorers on the scale measuring inhibitive eating patterns (i.e., the DEBQ-restraint scale) would regulate after the preload. In the present study, attention was also directed at determining whether counterregulation of high scorers on the RS is due to concern for dieting, weight fluctuations, or a combination of the two.

METHOD

Assessment

Subjects were classified as high or low scorers on the basis of the median split score on the following subscales.

Restraint Scale (RS) (Herman et al., 1978)

- (a) Total score on the RS: 10 items measuring attitudes toward eating, frequency of dieting, and weight fluctuations.

- (b) RS subscale Concern for Dieting (CD) (see Wardle, 1986): six items of the RS measuring attitudes toward eating and frequency of dieting (e.g., "How often are you dieting?").
- (c) RS subscale Weight Fluctuations (WF) (see Wardle, 1986): four items of the RS concerning weight fluctuations (e.g., "In a typical week, how much does your weight fluctuate?").

Dutch Eating Behavior Questionnaire (DEBQ) (van Strien, 1986)

- (a) DEBQ-restraint subscale: reflecting the degree to which one eats less than he or she actually would like to eat (e.g., "Do you try to eat less at mealtimes than you would like to eat?").
- (b) DEBQ-emotional subscale: measuring overeating due to "confusion between internal arousal states accompanying emotional states and physiological states of hunger and satiety" (van Strien, 1986, p. 137) (e.g., "Do you have a desire to eat when you are feeling lonely?"). A subdivision was made between eating in response to diffuse emotional states and eating in response to clearly labeled emotions.
- (c) DEBQ-external subscale: reflecting overeating due to a "hyperresponsiveness to food-related cues in the environment together with unresponsiveness to internal cues of hunger or satiety" (van Strien, 1986, p. 137) (e.g., "If you see or smell something delicious, do you have a desire to eat it?").

The dependent variable was the number of grams of ice cream consumed.

Subjects

Forty female students and employees of Limburg University volunteered to participate in what they believed to be a taste test. Their mean age was 25.5 years ($SD = 8.1$ years) and their mean body mass index ($BMI = \text{weight/height}^2$) was 22.8 ($SD = 2.7$). High and low scorers (median split) on all subscales did not differ in age or height, but on the RS, RS/WF, and DEBQ-external scales, the BMI of high scorers was significantly higher than the BMI of low scorers.

Procedure

Subjects were asked to have breakfast, lunch, or dinner 1 to 3 hr before participating in the study. Ice cream consumption thus served as a ca-

loric overload and was not influenced by deprivation. After entering the laboratory, subjects were asked to complete a questionnaire. In this questionnaire, items of the RS were hidden between questions about demographic variables and items concerning general aspects of life (e.g., frequency of sporting, smoking and drinking habits, date of last menstruation, taking medicine), in order to keep the subjects blind to the purpose of the study. Subjects 1 to 20 were randomly assigned to the preload or no-preload condition; subjects 21 to 40 were assigned to one of these conditions on the basis of their RS score in relation to the median split score of the subsample of subjects already tested. Thus, being preloaded or not was determined by the median split score on the RS. Each of the four cells (RS high scorers/RS low scorers $\times$ with/without preload) included 10 subjects. Subjects in the preload condition were asked to drink two milkshakes—one vanilla and one chocolate—each containing 150 cal. Ten minutes later, they were presented with three dishes, each containing 600 g of ice cream (chocolate, vanilla, and strawberry). Subjects in the no-preload condition were offered only the ice cream. They were instructed to taste and rate the different flavors. Subjects were encouraged to eat as much ice cream as they wanted and needed to make good taste ratings. Each subjects was left in isolation for 10 min. After that, the ice cream that was left over was reweighed and the subject completed the DEBQ. Then, the subject's weight and height were measured. Finally, before debriefing and payment for participation, we asked subjects to state their ideas on the purpose of the study and what they had eaten before participating in the study. All subjects had eaten their usual meal; there was no reason to believe that the groups differed in level of deprivation. Also, all subjects believed that it was a taste test. Nobody guessed that we measured the amount of ice cream eaten.

RESULTS

The median split scores for the different (sub)scales were as follows: RS/total, 12; RS/CD, 7; RS/WF, 4.5; DEBQ-restraint, 2.9; DEBQ-emotional total, 2.7; DEBQ-diffuse emotions, 3.0; DEBQ-clearly labeled emotions, 2.6; and DEBQ-external, 3.1. The results are depicted in Fig 1. For every subscale, a 2×2 ANOVA was carried out on the food intake data.² Main factors were Group (Low vs. High Scorers) and Condition (Preload vs. No-Preload). Because low and high scorers on the RS, RS/WF, and DEBQ-external scales differed significantly in BMI, an analysis of covariance was carried out with BMI as the covariate. On neither of the questionnaire sub-

²Further details on this study will be published elsewhere.

scales were main effects found. For the RS, the ANCOVA showed a significant interaction effect [$F(1,39) = 4.97, p = .03$]. On none of the other subscales were interaction effects found. However, t tests showed that low scorers on all RS and DEBQ measures regulated for the preload. Regardless of the subscale used, they ate significantly less after a preload than without one. In contrast, high scorers never regulated or counterregulated after the preload, for t tests showed no difference in intake after a preload or without one.³

DISCUSSION

Low scorers on all RS and DEBQ subscales ate significantly less ice cream in a preload condition than in a no-preload condition. Yet high RS and DEBQ scorers in a preload condition did not differ in ice cream intake from high scorers in the no-preload condition. High scorers, in other words, did not regulate their food intake. On the RS, and only on the RS, an analysis of covariance showed a significant Group $\times$ Condition interaction. However, here, too a t test analysis showed that, in high scorers, nonregulation rather than counterregulation took place. The finding of nonregulation instead of counterregulation is in line with the observation reported by Ruderman and Christensen (1983). However, the present findings are different from those of Wardle and Beales (1987), who found no difference in regulation between high and low DEBQ-restraint scorers. The finding of regulation of low-restraint scorers supports our suggestion (see Introduction) that the Wardle and Beales findings were derived from an atypical subject sample.

The fact that, globally, identical patterns were found on all analyses (cf. Fig. 1) could have been due to common variance of the different subscales. However, intercorrelations between the scales were surprisingly low (cf. Fig. 1). Contrary to our hypothesis, differences in regulation were not confined to high vs. low scorers on the disinhibitive scales, the RS, DEBQ-emotional subscale, and DEBQ-external subscale. Identical differences were observed on the inhibitive DEBQ-restraint subscale. It seems, therefore, that the occurrence of nonregulation is a feature of people preoccupied with caloric intake, for whatever reason. In passing, it may be noted that the similarity of the patterns shown in Fig. 1 indicates that the DEBQ subclassification lacks predictive validity when it comes to behavior in test situations such as the present one.

The observation that both successful and unsuccessful restrained eaters displayed the pattern of nonregulation seems a *prima facie* evidence for the disinhibition hypothesis of restraint theorists. However, we found *non*-regulation instead of *counter*regulation. Therefore, the crucial point of

³Numerical details may be obtained from the first author.

RESTRAINED SCALE

RS / CONCERN FOR DIETING

RS / WEIGHT FLUCTUATIONS

DEBQ-RESTRAINT SCALE

DEBQ-EMOTIONAL SCALE

DEBQ - EXTERNAL SCALE

DEBQ-DIFFUSE EMOTIONS

DEBQ-CLEARLY LABELED EMOTIONS

PEARSON P-M CORRELATION COEFFICIENTS				
	RS	DEBQ-R	DEBQ-EM	DEBQ-EX
RS	-	.53*	.47*	.40*
DEBQ-R		-	.34*	.19
DEBQ-EM			-	.44*
DEBQ-EX				-

* $p < 0.05$, one-tailed for $N = 38$

Fig. 1. Grams of ice cream consumed by high scorers (HS) and low scorers (LS). Numbers of subjects in each condition of the subscales were as follows (HS preloaded, HS without preload, LS preloaded, LS without preload): RS—10, 10, 10, 10; RS/CD subscale—10, 10, 10, 10; RS/WF subscale—9, 11, 11, 9; DEBQ-restraint subscale—10, 9, 9, 10; DEBQ-diffuse emotions subscale—12, 7, 7, 12; DEBQ-clearly labeled emotions subscale—11, 8, 8, 11; DEBQ-emotions subscale—13, 6, 6, 13; and DEBQ-external subscale—9, 10, 10, 9. DEBQ data are missing for one low and one high scorer on the RS.

the disinhibition hypothesis is not unequivocally supported: disrupting the restraint or self-control of restrained eaters by a preload did not "disinhibit" them. The finding of nonregulation instead of counterregulation may be due to our low median split scores on the different scales. Median split scores in the studies by Herman and Mack (1975) and Spencer and Fremouw (1979) were, respectively, 17 and 16. In these studies counterregulation was found. Although Polivy (1976) and Hibscher and Herman (1977) found counterregulation in their restrained samples with median split scores of, respectively, 11.5 and 14, nonregulation was found in the study by Ruderman and Christensen (1983) and in our own study. Median split scores were, respectively, 13 and 12. In their study, Ruderman, Belzer, and Halperin (1985) found that for subjects with restraint scores below 17.3, consumption did not differ in the milkshake and no-milkshake conditions, whereas for those with scores above 17.3 it did. All in all, there is a trend for counterregulation to occur in studies with higher medians, and nonregulation in studies with a lower median.

How should nonregulation in people scoring high on the RS or DEBQ subscales be explained? A possible explanation for the observed nonregulation is the cognitive set of restrained subjects. It has been suggested (Polivy, 1976; Polivy & Herman, 1985; Ruderman, 1985, 1986; Ruderman & Wilson, 1979; Ruderman *et al.*, 1985) that after disrupting dietary restraint by a preload, specific cognitive ideation occurs, a prototypical self-statement being, "I've blown it—I might as well continue to eat." However, contrary to this suggestion, it was observed that thinking aloud after a preload was thematically identical from restrained to unrestrained eaters. Retrospectively, too, preloaded restrained eaters did not recall more disinhibiting thoughts compared to nonpreloaded restrained eaters and preloaded as well as nonpreloaded unrestrained eaters (Jansen, Merckelbach, Oosterlaan, Tuiten, & van den Hout, 1988). Nevertheless, analysis of reported thoughts of restrained vs. unrestrained eaters during ice cream intake revealed a difference in perceived controllability. Restrained eaters in both conditions, independent from a disinhibiting preload, reported significantly more often that they could not control themselves during ice cream tasting (Jansen *et al.*, 1988).

This perceived lack of control during eating may link the literature on nonregulation/counterregulation to addiction model of restrained eating. Wardle (1987b) pointed out that loss of control over food intake, together with food craving and preoccupation with eating, parallels addictions. Support for the fruitfulness of the addiction analogy is given by data showing that restrained eaters, just like addicts, are likely to score high on sensation-seeking measures and to habituate fast to neutral stimuli (Jansen, Klaver, Merckelbach, & van den Hout, 1989).

From the addiction analogy, it seems that, just like small doses of drugs increase craving in the addict, eating small amounts produces increased craving in restrained eaters. Craving for food may be experienced as lack of control over food intake and may result in nonregulation. Studies should be welcomed that evaluate the explanatory power of the addiction model when it comes to restrained eating in general and experimentally induced nonregulation in particular.

DOES BINGEING RESTORE BULIMICS' ALLEGED 5-HT-DEFICIENCY?

A. JANSEN^{*1}, M. A. VAN DEN HOUT¹ and E. GRIEZ²

¹Department of Mental Health Sciences/Experimental Psychopathology, Limburg University, P.O. Box 616, MD 6200 Maastricht and ²Department of Clinical Psychiatry/Experimental Psychopathology, Limburg University, The Netherlands

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Summary—The co-occurrence of bingeing and depressed mood in bulimics may be explained by one pathogenic process: hyposerotonergic functioning. A relatively high carbohydrate intake improves mood and raises 5-HT level. Therefore, several authors suggested that the carbohydrate intake during binges may be a form of self-medication to compensate for bulimics' alleged hyposerotonergic functioning, thereby improving mood. This view, in which bingeing compensates for central 5-HT deficiencies, assumes that bulimics' binges contain relatively more carbohydrates than bulimics' non-binge-eating episodes. In the present study, bulimics' binge-eating and non-binge-eating episodes were compared as to macronutrient and calorie intake. However, no support was found for the hypothesis that binge-eating enhances brain serotonin.

INTRODUCTION

Bulimia nervosa is characterized by recurrent episodes of binge-eating and is often accompanied by a depressed mood (APA, 1987). The co-occurrence of bingeing and depressed mood in bulimics may be explained by *one* pathogenic process to which both the depressed mood and bingeing are functionally related. Carbohydrate intake and 5-HT (dys)functioning may be relevant here.

Schweiger, Laessle, Kittl, Dickhaut, Schweiger and Pirke (1986) showed experimentally that mood improves with increasing proportions of carbohydrate intake. The authors hypothesized that the mood changes induced by the intake of carbohydrates were mediated by central serotonergic functioning: eating episodes which are rich in carbohydrates and poor in protein will increase central 5-HT level, whereas eating episodes which are poor in carbohydrates and rich in protein will decrease the brain's serotonergic activity. Considering the relationship between depression and hyposerotonergic functioning (Rosenthal and Heffernan, 1986; van Praag, Lemus and Kahn, 1987a, b), Schweiger *et al.* (1986) argued that the improved mood after having eaten relatively more carbohydrates, is mediated by a heightened central 5-HT level, while a worsened mood follows from eating episodes which are relatively poor in carbohydrates and thus a lowered central 5-HT level.

Considering the depressive mood and binges of bulimics on the one hand and the relationship between carbohydrate intake, 5-HT level and mood on the other hand, several authors (Kaye, Ebert, Swirtsman and Weiss, 1984, 1985; Rosenthal and Heffernan, 1986; Goldbloom, 1987; Rock and Yager, 1987) suggested that the bingeing behavior and depressed mood of bulimics follow from hyposerotonergic functioning in the brain. "It is possible that binge-eating of carbohydrates is an attempt to enhance brain serotonin and, in this manner, serves as a form of self-medication" (Kaye *et al.*, 1984, p. 1601). Thus, binges are supposed to be relatively rich in carbohydrates to compensate for hyposerotonergic functioning, thereby improving mood. Moreover, Rock and Yager (1987) claim that "The carbohydrate-low diet of the bulimic, except for spurts during binges, may impede normal brain serotonin metabolism. Reduced serotonin synthesis in the brain may result from the low calorie, low carbohydrate intake commonly found as the usual nonpurged diet of bulimic patients, and this pattern of modified neurotransmitter synthesis has been proposed and is being explored as a physiologic trigger of binge eating" (p. 269).

^{*}To whom all correspondence should be addressed.

When it is claimed that carbohydrate intake during binges is an effective means to heighten the central 5-HT level, which is otherwise low, because bulimics' non-binge-eating is carbohydrate-poor/protein-rich, the straightforward assumption is made that, comparatively, bulimics' binges contain significantly more carbohydrates than bulimics' non-binge-eating episodes. Without this assumption, the theory that regards binge-eating as a mechanism activated to compensate central 5-HT deficiencies loses its power of explaining the (co-)occurrence of bingeing and depressed mood in bulimics. Is it true that binges contain comparatively more carbohydrates than bulimics' non-binge-eating episodes? Indeed, it has been claimed by patients and therapists that bulimics predominantly binge on carbohydrate-rich foods (Beumont, Chambers, Rouse and Abraham, 1981; Pope and Hudson, 1984; Johnson, Stuckey, Lewis and Schwartz, 1982; Goldbloom, 1987). However, these data are merely based on *impressions* of bulimics. Apart from the impression that binges are especially rich in carbohydrates, several studies also found relatively high fat and protein intake during binges (Mitchell, Pyle and Eckert, 1981; Abraham and Beumont, 1982; Kaye and Gwirtsman, 1985). Unfortunately, only the macronutrient composition of binges was analyzed in these studies, and the macronutrient intake during non-binge-eating episodes was not. Because of this, it was neither proved nor negated that the carbohydrate intake during binges is relatively higher than that between binges, and consequently the effective enhancement of 5-HT by means of a binge, was neither established nor ruled out. The primary aim of the present study was to compare binge-eating with non-binge-eating episodes of bulimic patients as to macronutrient intake. On the basis of the arguments of Kaye *et al.* (1984) and Rock and Yager (1987) it was hypothesized that (1) patients' binges contain relatively more carbohydrates than their non-binge-eating episodes, and (2) non-binge-eating episodes of bulimic patients contain relatively less carbohydrates than the diet of unrestrained eating control *Ss*. Furthermore, (3) the authors wanted to test whether mood is positively correlated with the proportion of carbohydrates in a diet.

METHOD

Subjects

Eight female patients with bulimia nervosa, according to the criteria of DSM III-R (APA, 1987), participated in the study. For one patient the diagnosis anorexia nervosa was also made. At the time of the study, the patients were not receiving any treatment but were awaiting cognitive-behavior therapy at the Academic Anxiety Center of Maastricht.

The control group consisted of eight healthy, unrestrained eating female students and employees of Limburg University. They reported that they did not have any eating problems, which was confirmed by their low scores* on the Dutch Eating Behaviour Questionnaire (van Strien, Frijters, Bergers and Defares, 1986). *S* characteristics are shown in Table 1.

Procedure and analysis

Seven patients kept a food diary and a mood diary during 7 days and one patient kept her diary for 10 days. During these days, patients exactly monitored their eating and drinking behavior, time of intake, whether it was a binge or not, and whether they vomited or used laxatives afterwards. Before going to sleep, *Ss* rated their global mood during the past day on a 0–100 ('extremely bad' to 'extremely good') Visual Analogue Scale (VAS). Control *Ss* kept identical registrations during 4 days, while at least one of these days was a 'free' day, on which they were not working or studying.

*Detailed data can be obtained from the first author.

Table 1. *S* characteristics

	Patients (<i>n</i> = 8)		Control <i>Ss</i> (<i>n</i> = 8)		<i>t</i>
	M	(SD)	M	(SD)	
Age	26.1	(5.0)	22.5	(2.9)	NS
BMI*	20.3	(3.1)	19.3	(0.97)	NS

*BMI = Body Mass Index (= weight/height²).

Binge-eating episodes were defined by the patients themselves, non-binge eating was defined by the authors as the total intake over one day, exclusive of binges. During the registration period 63 binges were recorded over all Ss ($M = 7.9$ per patient, with a range from 3 to 15). For each S, the mean macronutrient intake during binges, and the mean macronutrient intake during non-bingeing episodes were calculated, with the use of the computerized Becel Food Program (version NL 026) of the Unilever Research Laboratory. In addition to the proportional intake of carbohydrates, this program also yielded the amount of calories ingested and the proportional intake of protein, fat and alcohol. *T*-tests were carried out for the food intake data. Because of the large number of *t*-tests the authors used a 1% rejection level.

In order to test whether the proportional intake of carbohydrates and mood ratings were related to each other, Pearson product-moment correlations were calculated. The daily carbohydrate intake of patients *exclusive of vomiting periods*, and the daily carbohydrate intake of control Ss were calculated. The daily percentage of carbohydrate intake was correlated with mood over this day for patients and control Ss separately.

RESULTS

The results for macronutrient intake are shown in Fig. 1.

Carbohydrates

Binges were expected to contain relatively more carbohydrates than non-binge-eating episodes. However, we did not find the slightest support for this hypothesis: the patients' carbohydrate intake during bingeing episodes was as high as their carbohydrate intake during non-binge-eating episodes [$t(14) = 0.06$, NS]. Furthermore, no differences were found between relative carbohydrate intake of patients during non-binge-eating episodes and carbohydrate intake of control Ss [$t(14) = 1.22$, NS]. Neither did the proportional intake of carbohydrates during binges differ from the control Ss' carbohydrate intake [$t(14) = 1.55$, NS].

Fat

No differences in fat intake were found between the groups. Binge-eating and non-binge-eating episodes of bulimics contained the same amount of fat [$t(14) = 0.97$, NS]. The patients' intake of fat during non-binge-eating episodes did not differ from the fat intake of the control Ss [$t(14) = 1.43$, $P = NS$]. Neither was any difference found between fat intake during binges and fat intake of the control Ss [$t(14) = 0.74$, NS].

Fig. 1. Relative macronutrient intake during bulimics' binge-eating and non-binge-eating episodes, and during eating episodes of control Ss.

Protein

Patients ate the same amount of protein during binges as during non-binge-eating episodes [$t(14) = 1.93$, NS]. Neither was the protein intake during non-binge-eating episodes different from the protein intake of the control *SS* [$t(14) = 0.63$, NS]. Furthermore, no difference emerged between normals' protein intake and patients' protein intake during binges [$t(14) = 2.38$, NS].

Alcohol

Proportionally, there was no difference between the patients' alcohol intake during binge-eating episodes and non-binge-eating episodes [$t(14) = 0.35$, NS]. Neither did patients, during binge-eating and non-binge-eating episodes differ from control *SS* as to the intake of alcohol [$t(14) = 0.56$, NS] and [$t(14) = 0.77$, NS] respectively.

Calories

Data on calorie intake are depicted in Fig. 2. No difference was found in patients' calorie intake during binge-eating and non-binge-eating episodes [$t(14) = 1.62$, NS]. The calorie intake during patients' binges was as high as that of control *SS* over one day [$t(14) = 0.56$, NS]. During non-bingeing episodes, patients ate less than control *SS*. This difference approached significance [$t(14) = 2.73$, $P = 0.02$].

Carbohydrate intake and mood

A significant correlation was found between carbohydrate intake and mood of control *SS* [$r = 0.44$, $P = 0.006$]. In contrast to this, no correlation was found between patients carbohydrate intake during non-vomiting episodes and mood ($r = 0.06$, NS).

DISCUSSION

The first hypothesis, stating that the food that is consumed during binge-eating episodes of bulimic patients contains a larger proportion of carbohydrates than that consumed during non-binge-eating episodes, was not supported. No differences were found in the carbohydrate intake between binge-eating and non-binge-eating episodes. Neither was the second hypothesis confirmed, that the carbohydrate intake in the usual diet of bulimic patients during non-binge-eating episodes is low. The bulimics' usual diet between binges contained the same proportion of

Fig. 2. Calorie intake during bulimics' binge-eating and non-binge-eating episodes, and during eating episodes of control *SS*.

carbohydrates as the food intake of unrestrained eating control Ss. Finally, the third hypothesis, stating that carbohydrate intake and mood are positively correlated, was confirmed for control Ss only. For patients, no correlation between carbohydrate intake and mood was found.

From the absence of a difference in carbohydrate intake during binge-eating and non-binge-eating-episodes, it may be inferred that binge-eating does not enhance brain serotonin and serve as a form of self-medication. Moreover, as the data on carbohydrate intake of control Ss and on that of patients between binges show, Rock and Yagers' claim of a "carbohydrate-low diet of the bulimic, except for spurts during binges" was not supported: during non-binge-eating episodes, patients ate the same amount of carbohydrates as control Ss did.

The issue of the validity of the Ss' diaries deserves some attention. Although "prospective self-monitoring provides accurate information on the frequency and extent of substance use" (Wilson, 1987, p. 341), self-monitoring is likely to be reactive; it may affect the rate of binge-eating episodes (Wilson, 1987). However, although self-monitoring may influence the *rate*, and perhaps the *extent* of binge-eating, it seems highly unlikely that self-monitoring will change the *macro-nutrient composition* of binges.

On none of the macronutrients did the patients' intake during non-binge-eating episodes differ from the control Ss' intake. If it is not the macronutrient composition of the diet of bulimics which triggers a binge, how then, should we explain the occurrence of binges? Finding a marginally lower calorie intake of patients during non-binge-eating episodes as compared with eating episodes of control Ss, supports the common notion that binges are related to dieting (Polivy and Herman, 1985). However, that dieting and bingeing are somehow related, still does not offer an explanation. A popular explanation of the urge to binge is given by the set point theory, which states that dieters are below their set point for body weight, and that this induces a biologically determined urge in them to binge, in order to restore their body weight to the set point level. However, the set point theory is rather difficult to test, and although there certainly is a correlation between dieting and bingeing (most bulimics diet, and many dieters binge-eat sometimes), the relationship between reduced calorie intake and binge-eating is certainly not a one-to-one relationship. Not all people who diet (e.g. anorexia nervosa restricters and successful restrained eaters) are plagued by binge-eating episodes. The precise mechanism which links dieting to binge-eating is still to be discovered.

Though it remains to be demonstrated directly that serotonergic pathways are involved, mood and carbohydrate intake are related to each other. For our control Ss, the correlation between mood and carbohydrate intake was highly significant. Of special interest is, therefore, the lack of a positive correlation between carbohydrate intake (exclusive of vomiting episodes) and mood in bulimic patients. A commonsensical psychological explanation is that the patients' generally more depressed mood is primarily determined by their negative evaluation of binge-eating episodes; they are doing things they actually do not want to do and thus end up with a bad mood. The influence of carbohydrate intake on patients' mood may be subordinate.

All in all, these findings on macronutrient intake do not provide any support for the hypothesized function of a binge for heightening people's serotonergic level. Neither does this study support the notion that bulimics' non-binge-eating episodes decrease the central level of 5-HT, because they are relatively poor in carbohydrates. However, the authors are well aware that these data do not exclude the possibility of hyposerotonergic functioning in case of bulimia. Still, the fact that many psychopathologies seem to be accompanied by serotonergic dysregulation poses fundamental questions as to the explanatory power of this alleged 5-HT dysregulation in bulimia. Major depressive disorders, anxiety disorders, obsessive-compulsive disorders, chronic pain, suicide, aggression, and schizophrenia; they have all been associated with a lowered 5-HT turnover (van Praag 1987a, b). And these disorders are not only characterized by 5-HT dysregulation; like bulimia nervosa, they all often go together with depression. Why then, do patients with these other psychiatric complaints, supposed hyposerotonergic brain functions and depression, not binge on carbohydrates in order to induce a better mood?

Finally, during the analysis of the food diaries, the authors were puzzled by an intriguing issue: what determines whether an eating episode is a binge or not? The patients themselves defined which eating episode was a binge and which one was not, but the authors were not able to detect any objective discriminating parameters between binge-eating and non-binge-eating 'meals'.

Repeatedly, patients noted that an eating episode was a 'bad' eating episode, but maintained it was not a binge. When patients were asked retrospectively why a certain eating episode was not a binge, whereas another episode was, they were often doubtful and unable to reply. This may suggest that bingeing is not an 'all-or-nothing' phenomenon, as it is usually claimed to be. In contrast to the dichotomous view that eating episodes either are or are not binges, eating episodes may be more or less binges. It might be a rewarding enterprise to explore this hypothesized continuity of bulimics' eating episodes as more or less binges: what is a binge?

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5.4 is counterregulation of dieters after forced consumption of a preload dependent on mood?

Anita Jansen¹, Ilse Gubbens¹, Sandra Gransier¹, Peter de Jong¹,
Jaap Oosterlaan¹ & Arnoud Arntz¹.

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abstract

In the present study, the question of whether the counterregulation of dieters after forced consumption of a preload is dependent on mood was examined in a 2 (dieter vs non-dieter) x 2 (happy vs depressed) x 2 (preload vs no-preload) factorial design. Subjects were 83 Dutch housewives (mean age 42 years) who were classified as dieter or non-dieter by the median split score on the Restraint Scale of the Dutch Eating Behaviour Questionnaire (DEBQ). A happy or a depressed mood was induced with the aid of music. Half of the subjects were forced to eat a preload. It was hypothesized that forced consumption of a preload will disinhibit the dieter when mood is depressed, but will elicit a normal regulatory response when the dieter is happy. It was found that happy dieters did indeed regulate their food consumption normally after a preload. However, depressed dieters also ate marginally less after a preload. Furthermore, the finding that non-dieters never regulated their food intake, neither when happy, nor when depressed casts some doubts on the validity of the present findings. The curious results may be due to the relatively high age of the subject sample used. A replication study using a younger subject sample is desirable before firm conclusions can be drawn as to the mood-dependence of counterregulation after a preload.

introduction

Dieters are chronically concerned with their weight and they constantly try to control, i.e. restrict, their calorie intake. In laboratory studies, it has repeatedly been shown that the eating behavior of dieters can be disin-

1 Dept. of Mental Health Sciences / Experimental Psychopathology, Limburg University, P.O. Box 616, 6200 MD Maastricht, the Netherlands.

hibited. The most typical finding is that after diet violation, e.g., forced consumption of a preload, dieters eat more than without one, whereas the food consumption of non-dieters is smaller after a preload than without one (Herman & Mack, 1975; Hibscher & Herman, 1977).

A rather intriguing question is *why* the dieter eats more after the diet has been broken by forced consumption of a preload. Eating a preload rich in calories would be a good motive for restricting further food intake. Eating even more after a forced preload is counterintuitive. Interestingly, experimentally induced emotional upheaval is also found to be a potent disinhibitor in dieters. Anxious non-dieters are found to eat less than non-anxious non-dieters whereas anxious dieters eat as much as non-anxious dieters (Herman & Polivy, 1975). Dysphoric dieters eat significantly more than non-dysphoric dieters, and the intake of dysphoric non-dieters was marginally less than the intake of non-dysphoric non-dieters (Baucôm & Aiken, 1981; Frost, Goolkasian, Ely & Blanchard, 1982; Ruderman, 1985). Although the exact process by which a negative affect disinhibits eating in dieters is still unknown (Ruderman, 1986), the experimental findings on counterregulation of dieters during emotional upheaval may help to elucidate counterregulation in dieters after forced consumption of a preload. After having been forced to eat a calorie-rich preload, the notion of having overeaten may elicit a dysphoric mood in dieters. It might be this dysphoric mood rather than being preloaded per se that exerts the disinhibiting effect. A straightforward implication of this assumption is that preloaded but non-dysphoric dieters will not counterregulate. Therefore, it is hypothesized that (1) forced consumption of a preload disinhibits eating in the dieter when he or she is in a depressed mood, while (2) forced consumption of a preload will elicit a normal regulatory response when the dieter is happy. Furthermore, it is hypothesized that (3) non-dieters will regulate their food intake after a preload independently of mood.

method

subjects

Subjects were 83 Dutch females, most of them housewives with a low level of education. Ss were recruited by means of a city newspaper advertisement, asking for females who were willing to participate in "a study on taste experiences". Mean age was 42.4 years ($SD=10.0$, range 20 to 61). Ss were classified as dieter or non-dieter by the median split score (3.1) of the present sample on the Restraint Scale of the DEBQ (van Strien, 1986).

Subject characteristics are given in Table 1. Dieters were significantly younger and their BMI was lower than corresponding values in the non-dieting group.

table 1: subject characteristics.

	non-dieters n=40	dieters n=43	t
Age	38.8 (10.0)	45.9 (8.9)	3.43***
BMI*	23.1 (4.1)	25.0 (3.1)	2.45**

*BMI= Body Mass Index (= weight/height²); ** p<0.05; *** p=0.000

assessment and materials

The following instruments and materials were used:

A questionnaire on demographic variables and way of life was composed by the authors. In this questionnaire the "Concern for Dieting" (CD) items of the Restraint Scale (RS; Polivy, Herman & Warsh, 1978) were interspersed among fake items that asked about the way of life with regard to e.g. practice of sport, smoking and drinking habits and taste preferences. The CD items measure attitudes towards eating and frequency of dieting.

The Dutch Eating Behaviour Questionnaire (DEBQ; van Strien, 1986). The Restraint subscale reflects the degree to which a person eats less than he or she would actually like to eat. The Emotional and External subscales reflect the amount of overeating as a reaction to emotional arousal respectively external stimuli (Van Strien, 1986).

A tape with "happy" music was compiled by the authors. Among other things, the tape included fragments by Charlie Chaplin and Mozart (detailed information can be obtained from the first author). In a pilot study, the happy music proved to be effective in that it induced a happier mood.

A tape with "depressing" music. The music was 'Russia under the Moncholian yoke' by Prokofiev, played at half speed, as recommended by Clark (1988). In a pilot study, the depressing music proved to be effective in that it induced a more depressed mood.

A preload of 100 grams of cookies, i.e. 520 kcal.

The **dependent variable** was the number of grams of cookies consumed during the "taste test".

procedure

Subjects were sent a questionnaire with items asking about demographic variables and way of life, in which the "Concern for Dieting" items from the Restraint Scale (Polivy et al., 1978) were hidden. Thus, the RS/CD items would not appear salient and the subjects were kept in the dark about the purpose of the study. On the basis of their scores on the CD items of the RS, Ss were provisionally classified as dieter or non-dieter, in order to

enhance the probability of ending up with enough Ss in each condition. During the experiment subjects were randomly assigned to subgroups. Subjects were asked to have breakfast, lunch, or dinner 1 to 3 hours before participating in the study in order to prevent experimental food intake from being influenced by pre-experimental deprivation. After entering the laboratory, subjects in the preload condition were given the preload and were instructed to eat it all. After tasting and rating the cookies, the subjects rated their mood on a 0 ("extremely sad") to 100 ("extremely happy") Visual Analogue Scale (VAS). Then they were left in isolation for 10 minutes listening to the tape with either the "happy" or "depressing" music. Subjects were encouraged to give in to the mood evoked by the music. In order to minimize demand characteristics, the hypothesized mood change was not explicitly mentioned, i.e. the subjects were not told whether they should become depressed or elated. Subjects in the no-preload condition started listening to the music directly after they had rated their mood. After 10 minutes, the experimenter instructed the subject through the intercom to rate her mood again. Then the experimenter returned and the subject was offered three dishes, each containing 100 grams of cookies of different tastes. The cookies had been broken into small pieces to prevent the subject and the experimenter from seeing clearly how much had been eaten, later in the experiment. The subject had to taste each of the three sorts of cookies and was encouraged to eat as many cookies as she wanted and needed for making good taste ratings. She was left in isolation for ten minutes. During taste ratings, music was still played. After ten minutes, the cookies which were left were taken away and weighed again. Finally, the subject completed the DEBQ, the subject's height and weight were measured, and the subject was paid for participation. Subjects were asked if, and when, they had eaten before participation, and all subjects reported that they had complied with the instruction to eat between 1 and 3 hours before participation. To prevent subjects from talking about the experiment with other participants, debriefing took place after all subjects had participated in the experiment.

results

manipulation check

Mood induction was found to be successful. Before mood induction, subjects did not differ in mood level. After the mood induction, subjects in the depressive condition showed significantly lower mood scores than subjects in the happy condition [$t(79)=4.38$, $p<0.000$]. This effect was mainly due to a significant lowering of mood in the depressive condition [$t(40)=4.9$, $p<0.000$]. The mood scores in the happy condition did not change significantly [$t(39)=0.76$, NS].

figure 1: food intake of dieters and non-dieters after a preload or no preload and in a depressed or happy mood.

table 2: mean number of grams (sd) eaten in the different groups.

	depressed preload	no-preload	happy preload	no-preload
dieters	31.5 (19.6) n=10	39.9 (16.2) n=9	21.0 (9.9) n=11	29.7 (14.1) n=13
non-dieters	27.0 (18.5) n=12	30.3 (20.7) n=11	25.5 (10.0) n=8	24.9 (11.7) n=9

food intake

Results are given in Figure 1 and Table 2.

The hypotheses were tested in two ways. First, t-tests were carried out. As can be seen from Fig. 1, the first hypothesis, that dieters counterregulate when they are in a depressed mood, was not confirmed. Depressed dieters ate marginally less after a preload than without one [$t(17)=1.0$, NS]. Evidence was found in support of the second hypothesis, stating that forced consumption of a preload will lead to normal regulation when the dieter is happy. Dieters in the happy mood condition ate significantly less after a preload than without one [$t(22)=1.72$, $p=0.05$]. Hypothesis 3 states that non-dieters will regulate their food intake after a preload, independently of mood. It was found, however, that non-dieters did not regulate their food intake after a preload, neither when depressed [$t(21)=0.41$, NS], nor when happy [$t(15)=0.1$, NS].

Second, an analysis of covariance (ANCOVA) was carried out in order to make a closer examination of the interaction between dietary status,

preload, and mood. Main factors were Group (Dieters vs Non-dieters), Condition 1 (Preload vs No-preload), and Condition 2 (Depressed vs Happy Mood), with BMI and Age as covariates. A marginally significant main effect was found for mood [$F(1,73)=3.6$, $p=0.06$], which indicated that individuals in a depressed mood ate more than happy subjects. No further main effects were found, and neither the 2-way, nor the 3-way interactions were significant.

discussion

The present study investigated whether disinhibition of eating in dieters after a preload is mediated by a lowered mood. The hypothesis that the dieters' food intake after a preload is dependent on mood was supported: t-tests showed that dieters failed to regulate their food intake after a preload when their mood was depressed, but they did regulate their intake normally when they were happy. However, the absence of normal regulatory behavior in our non-dieting subjects, and the absence of a significant interaction effect between preload, mood, and dietary status seriously calls the validity of this finding into question. At least, the hypothesis that the non-regulation of dieters is dependent on mood was not unequivocally supported.

In addition, and most importantly, the present study does not provide any support for some typical and well-documented findings. First, by the marginally significant interaction effect between mood and dietary status we could not convincingly confirm the finding that dieters eat more when depressed and non-dieters eat the same or less (Baucom & Aiken, 1981; Frost et al., 1982; Ruderman, 1985). In the present study, dieters *as well as* non-dieters ate slightly more when depressed. Second, the absence of a significant interaction effect between preload and dietary status is in direct conflict with the typical finding that dieters eat more after a preload and non-dieters eat less (Herman & Mack, 1975; Hibscher & Herman, 1977). In the present study, both dieters and non-dieters ate the same amount with or without a preload. Although non-regulation in dieters has been documented earlier (Ruderman & Christensen, 1983; Jansen, Oosterlaan, Merckelbach & van den Hout, 1988), the finding of non-regulation in non-dieters is a curious one. Non-regulation in non-dieters has only been documented once (Wardle & Beales, 1987). How can this curious behavior of non-dieters be explained? Some possibilities are discussed below.

First, the present study shows some weaknesses. Ruderman & Wilson (1979) showed that obese and non-obese subjects react different to the consumption of a preload. Although the subjects in the present study were not extremely obese, dieters had a significantly higher BMI than non-dieters. The authors tried to minimize the effect of this difference by using BMI as covariate in the ANOVA. However, with that, the influence

of weight cannot totally be ruled out. In the same way, age differences may have influenced the data.

Furthermore, the authors did not gather more detailed information on the actual deprivation time of subjects. Subjects all agreed with the instruction to eat between 1 and 3 hours before participating in the experiment. It should be noted, however, that the inter-individual variation between 1 and 3 hours may be large.

Second, Wardle and Beales (1987) argued that the non-regulation of non-dieters in their study could be due to a "floor effect", i.e. all subjects ate the minimum necessary. Indeed, the amounts eaten in their study were fairly small, and so are the amounts eaten in the present study. However, the cookies were rated as palatable and the mean intake of 29 grams shows that the non-dieters were not eating the absolute minimum, i.e. about 10 grams. They clearly ate more cookies than necessary for completing the taste ratings.

Third, in the present study, dietary status was assessed with the DEBQ (van Strien, 1986). In earlier studies on counterregulation, dietary status was assessed by means of the Restraint Scale (RS; Polivy et al., 1978). Remarkably, Wardle and Beales (1987) also classified their subjects as dieter or non-dieter on the basis of the median split on the DEBQ-restraint scale and they also found non-regulation of food intake in non-dieters. Earlier, Wardle (1987) reported that the restraint scale of the DEBQ measures successful dieting irrespective of tendencies towards disinhibition, whereas the RS in fact measures unsuccessful dieting, or proneness to disinhibited eating. It sounds plausible that the overeating of high scorers on the RS is due to their proneness to disinhibition and that the subjects scoring low regulate their food intake because they do not show these disinhibited tendencies.

Classifying subjects according to dietary status by means of the DEBQ may be independent of a tendency to disinhibition. Non-dieters may then, despite their low dietary status, still be prone to 'overeating' in the absence of dieting. To test this, we *post hoc* re-classified our subjects as highly or little emotional and external eaters by means of their scores on the disinhibited "emotional" and "external" scales of the DEBQ. High scores on these scales reflect proneness to disinhibited overeating as a reaction to emotional or external stimuli. However, the *post hoc* classifications did not reveal any differences in outcome. Low scorers on "disinhibited" scales did not regulate their food intake in the present study.

Fourth, a median restraint score of 3.1 on the DEBQ is above average according to the norm scores (see van Strien, Frijters, van Staveren, defares & Deurenberg, 1986). It may therefore be questioned whether our non-dieters were *really* unrestrained. Therefore, we compared the food intake of the lowest scorers on the DEBQ-restraint scale with the food intake of the highest scorers. Again no differences emerged.

Fifth, the calorie content of the preload was high, i.e. 500 kcal. Milk shake preloads usually vary from 250 kcal to 440 kcal (Wardle and Beales,

1987). The higher the calorie content of the preload, the larger the differences are expected between the preloaded and nonpreloaded non-dieters. Then, of course, preloaded non-dieters would be expected to eat significantly less than non-preloaded ones, which was not the case.

Finally, the subject sample that participated in the present study is quite different from subject samples usually participating in laboratory experiments on food intake. Usually, students 20 to 30 years old and with a high level of education are the subjects of the examination. In contrast to this, the participants in the present study were significantly older and most of them had a low level of education. A commonsensical explanation for the present findings may be that older women often report an *intention* to lose some weight, but they are *actually* only making a marginal effort to reach it. For younger people, weight and appearance may in fact be more important, which will result in more extreme and rigid dieting actually put into practice.

All in all, the present study did not provide convincing support for the notion that the disinhibiting effect of the forced consumption of a preload is dependent on mood. With respect to the influence of age, it should be noted that eating disorders such as anorexia nervosa and bulimia nervosa are more prevalent in puberty and young adulthood than later in life (APA, 1987). Therefore, the present data should be interpreted cautiously. A replication study using a younger subject sample is desirable before firm conclusions can be drawn as to the mood-dependence of counterregulation after a preload.

COGNITIONS AND SELF-TALK DURING FOOD INTAKE OF RESTRAINED AND UNRESTRAINED EATERS

A. JANSEN¹*, H. MERCKELBACH¹, J. OOSTERLAAN¹, A. TUITEN² and M. VAN DEN HOUT¹

¹Department of Medical Psychology/Experimental Psychopathology, Limburg University, PO Box 616, 6200 MD Maastricht and ²Department of Theoretical Psychology and Psychonomics, Utrecht University, The Netherlands

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Summary—In the present study, restrained subjects failed to regulate their ice-cream intake after a preload of milkshakes, while unrestrained subjects succeeded. Restraint was not related to irrational thinking patterns, as reflected by scores on the Rational Behavior Inventory (RBI) and the Irrational Belief Test (IBT). During the disinhibitive challenge, the self-talk of the subjects was recorded on tape. Restrained subjects in a disinhibition challenge, i.e. preload condition, were expected to report significantly more disinhibitive thoughts than restrained subjects in a no-preload condition. Unrestrained subjects were expected to report no disinhibitive self-talk at all. The authors failed to find experimental confirmation for the widely held view that dichotomous thoughts such as "I've blown my diet, I might as well continue to eat" inhibit restrained eaters.

INTRODUCTION

It has repeatedly been reported that milkshake preloads inhibit the dietary constraint of restrained eaters. High scorers on the Restraint Scale [RS] (Herman *et al.*, 1978) eat more ice-cream after a preload of milkshakes than they do without one (Herman and Mack, 1975; Hibscher and Herman, 1977; Herman and Polivy, 1980). By manipulating subjects' beliefs concerning the caloric content of the preload, Polivy (1976) and Spencer and Fremouw (1979) demonstrated that this so-called 'counterregulation' of restrained eaters also occurs when they only *think* they have overeaten. Therefore, these authors conclude that counterregulation of restrained eaters is cognitive in nature. The maladaptive eating behavior is supposed to follow from an irrational thinking style and dichotomous, all-or-nothing reasoning. Dietary disinhibitors, such as milkshake preloads, should induce such thoughts as "The day is wasted", "My diet is ruined for the next two days. I may as well give up now," and "I've blown my diet; I might as well continue to eat", thereby leading restrained eaters to overeat (Polivy, 1976; Polivy and Herman, 1985; Ruderman 1985, 1986; Ruderman and Wilson, 1979; Ruderman, Belzer and Halperin, 1985).

However, self-talk during overeating has never been investigated experimentally (Wardle, 1987). Although Ruderman (1985) demonstrated that irrational beliefs, as measured by the Rational Behavior Inventory [RBI] (Shorkey and Whiteman, 1977), are more prevalent among restrained than unrestrained eaters, Sanderman *et al.* (1987) reasoned that self-statements in certain situations may not be reflected in RBI-scores.

Considering both the absence of direct evidence showing that disinhibitive self-talk is responsible for counterregulation and the imperfect association between irrational thinking and types of self-talk, the aim of the present study was 2-fold. First, the authors wanted to replicate Ruderman's findings that there is a close relationship between restraint and irrational thinking as measured by the RBI. Second, we wanted to examine self-talk of subjects during a disinhibitive challenge. It was hypothesized that (1) restrained eaters score significantly higher on measurements of irrationality than unrestrained eaters and that (2) restrained eaters in a disinhibition challenge, i.e. preload condition, report significantly more disinhibitive thoughts than restrained subjects in a no-preload condition. Unrestrained subjects were expected to report no disinhibitive thoughts at all.

*Requests for reprints.

Table 1. Subject characteristics

	Unrestrained (<i>n</i> = 20)		Restrained (<i>n</i> = 20)		<i>t</i>
	M	(SD)	M	(SD)	
Age	24.6	(6.9)	26.3	(9.2)	NS
BMI†	21.5	(1.9)	24.2	(2.6)	3.77*

**P* ≤ 0.001.†BMI = Body Mass Index (= weight/height²).

METHOD

Subjects

Forty female students and employees of Limburg University volunteered to participate in what they believed to be a study on 'the influence of thoughts on taste-testing'. Their mean age was 25.5 yr (SD = 8.1) and their mean body mass index (BMI = weight/height²) was 22.8 (SD = 2.7). Participants were classified as restrained or unrestrained on the basis of the median split score on the RS. Subject characteristics are shown in Table 1.

Assessment

The following instruments were used:

1. The *Restraint Scale (RS)* (Herman *et al.*, 1978), measuring attitudes toward eating, frequency of dieting, and weight fluctuations.
2. The *Rational Behavior Inventory (RBI)* (Shorkey and Whiteman, 1977), measuring irrational thinking patterns. The total score can be broken down into 11 factors, each of which measures a different dimension of rationality: (1) catastrophizing, (2) guilt, (3) perfectionism, (4) need for approval, (5) caring and helping, (6) blame and punishment, (7) inertia and avoidance, (8) independence, (9) self-degradation, (10) projected misfortune and (11) control of emotions.
3. The *Irrational Belief Test (IBT)* (Jones, 1968), also measuring irrational thinking patterns. The total score can be broken down into 10 factors, each of which, again, measures a different dimension of rationality: (1) demand for approval, (2) high self-expectations, (3) blame, (4) frustration, (5) emotional irresponsibility, (6) anxious overconcern, (7) problem avoidance, (8) dependency, (9) helplessness with regard to change and (10) perfectionism.
4. The *Think Aloud on Tape (TAP)*. Subjects were asked to provide continuous verbal expression of their thoughts during the 10-min taste-testing period. The reported thoughts were registered on tape.
5. The *Self-Talk Questionnaire (STQ)*, a list of disinhibitive self-statements composed by the authors (see Table 2). 4 of the 25 statements were taken from the literature; 21 were based on an *n* = 1 pilot study and intuitively chosen. For each self-statement, the frequency of having had this thought during ice-cream tasting was rated between 0–100 ("never" to "very, very often") on a visual analogue scale (VAS).
6. The *amount of ice-cream consumed* was registered.

Procedure

Subjects were asked to have breakfast, lunch, or dinner 1–3 h before participating in the study. Taste-testing thus served as a caloric overload and was not influenced by deprivation. After entering the laboratory, subjects were asked to complete a questionnaire in which items of the RS were hidden. Subjects 1–20 were randomly assigned to the preload or no-preload condition; subjects 21–40 were assigned to one of these conditions on the basis of their RS scores in relation to the median split score of the subsample of subjects already tested. Each of the four cells (restrained/unrestrained × with/without preload) included 10 subjects.

All subjects completed the RBI. Then, subjects in the preload condition were asked to drink two milkshakes—one vanilla and one chocolate—each containing 150 calories. During the 10-min interval between drinking the milkshakes and tasting the ice-cream, subjects completed the IBT. Participants in the no-preload condition completed the IBT directly after the RBI. All subjects were then presented with three dishes of ice-cream—one chocolate, one vanilla, and one strawberry—

Table 2. Self-talk questionnaire

1.	This is a wasted day
2.	I don't have myself under control
3.	I've already eaten too much, let me have another bite
4.	I'm really not supposed to have ice-cream because ice-cream is bad for me
5.	It's going all wrong
6.	I'm giving in completely
7.	I'll sneak another bite
8.	I cannot control myself
9.	It's hard not to eat it all
10.	Ice-cream is really 'off-limits'
11.	My diet is ruined for the next two days. I may as well give up now
12.	Everything has got to be eaten
13.	The experimenter had better come back before I finish it all off!
14.	I'll start my dieting again tomorrow
15.	I must not eat too much
16.	My day is already shot, I may as well keep eating
17.	I don't have a hold of myself
18.	This has been a bad day. I've eaten too much
19.	I haven't kept to my good dieting resolutions
20.	I'm afraid I'm going to eat it all up
21.	I'm really full, but I'll have another bite
22.	I can't help it that I have to eat ice-cream. Let me take advantage of this opportunity
23.	I've already gone over my limit, let me just keep on going
24.	I'm afraid I can't stop eating any more
25.	I've failed

weighing 600 g. The subjects were instructed to taste and rate the different flavors. At the same time, they were asked to express all of their thoughts aloud into the microphone of the audiotape. Subjects were encouraged to eat as much ice-cream as they wanted and needed in order to make good taste testings.

After starting the audiotape, the experimenter left the laboratory for 10 min. When the experimenter came back, the ice-cream that remained was reweighed and the subjects completed the STQ. Then, each subject's weight and height were measured. Finally, the subjects were debriefed and paid for their participation.

RESULTS

1. RS

The median split score on the RS was 12. Subjects scoring 12 or more were classified as restrained eaters. Scorers below 12 were classified as unrestrained eaters.

2. Ice-cream intake

Because restrained and unrestrained subjects differed significantly in BMI (see Table 1), a 2×2 analysis of covariance was carried out on ice-cream data. Main factors were Group (restrained vs unrestrained) and Condition (preload vs no-preload), with BMI as covariate. The ANCOVA showed no main effects. The interaction Group $\times$ Condition was significant [$F(1,39) = 4.97$, $P = 0.03$] (see Fig. 1). *t*-Tests showed that unrestrained subjects regulated for a preload. In contrast, restrained subjects did not eat significantly more or less after a preload than after no preload (see Table 3).

3. IBT and RBI

No significant differences in irrationality were found between restrained and unrestrained subjects (see Table 4). Pearson P-M correlations between the RS, IBT, and RBI (total and factor scores) were calculated. Neither the RS-IBT and RS-RBI total scores (see Table 5) nor the RS-IBT and RS-RBI factor scores revealed significant correlations.

4. STQ

On each of the 25 STQ self-statements, a 2×2 ANCOVA (Group $\times$ Condition with BMI as covariate) was carried out. A main effect for Group was found for the statement "I cannot control myself" [$F(1,39) = 4.97$, $P \leq 0.05$]. Thus, restrained subjects in both conditions reported significantly more frequently than unrestrained subjects that they could not control themselves

Fig. 1. Mean ice-cream consumption of high (HS) and low (LS) scorers on the Restraint Scale.

Table 3. *t*-Tests on ice-cream intake

		Preload		<i>t</i>
		-	+	
Unrestrained	M	130.1	50.2	3.26*
	SD	50.5	58.9	
	N	10	10	
Restrained	M	83.3	93.5	NS
	SD	52.9	82.9	
	N	10	10	

* $P \leq 0.001$.

during ice-cream testing. Main effects for Condition were found for the following statements: "I'll start my dieting again tomorrow" (!) [$F(1,39) = 4.94$, $P \leq 0.05$]; "I must not eat too much" [$F(1,39) = 5.11$, $P \leq 0.05$]; "This has been a bad day. I've eaten too much" [$F(1,39) = 3.94$, $P = 0.055$]; "I am really full, but I'll have another bite" [$F(1,39) = 5.69$, $P \leq 0.05$]. These thoughts were significantly more often reported in the preload condition, independent from the restraint of subjects. No significant Group $\times$ Condition interaction effects were found.

5. TAP

The total number of reported thoughts was divided into 5 exhaustive and mutually exclusive categories:

- Non-food statements (e.g. "I really need to trim my finger nails"),
- Statements literally read from the taste-testing list (e.g. "Flavor one is really quite sweet"),
- Task-relevant statements concerning taste (e.g. "delicious") or test behavior (e.g. "I'll have another bite"),
- Statements concerning associative past or future food situations (e.g. "Ice-cream makes me think of going on vacation, of sitting at a café in the sun"), and
- Statements concerning food or eating behavior which were not relevant to the experimental task of tasting (e.g. "I have to force myself not to finish the strawberry ice-cream").

Statements from categories a, b, c, and d were either a result of the experimental task or concerned past or future events. They were considered not to contain disinhibitive thoughts. We did, however, expect to find disinhibitive thoughts in category 'e' statements. Concerning the proportion of food items that were classified as 'e' statements, no difference emerged between restrained and unrestrained subjects; in the restrained group, 9.2% of the 1379 statements was

Table 4. *t*-Tests on measurements of irrationality

		Unrestrained	Restrained	<i>t</i>
RBI	M	28.2	28.5	NS
	SD	4.9	5.4	
	N	20	20	
IBT	M	275.6	271.1	NS
	SD	26.7	30.7	
	N	20	20	

Table 5. Pearson P-M correlations of the RQ with IBT and RBI ($N = 40$)

	RBI Total						Factor				
		I	II	III	IV	V	VI	VII	VIII	IX	X
RQ	-0.07	0.01	-0.04	-0.08	-0.11	-0.11	-0.09	-0.11	0.14	-0.21	-0.08
	IBT Total						Factor				
		I	II	III	IV	V	VI	VII	VIII	IX	X
RQ	0.06	0.003	0.1	0.24	0.004	0.06	0.21	-0.07	-0.15	0.15	-0.15

classified as 'e'; in the unrestrained group the figures were 8.9% from 1339 statements. The binomial distribution indicates that this difference is not significant. Though the idea that category 'e' statements should contain all disinhibitive thoughts seems plausible, the absence of a difference between restrained and unrestrained eaters led to an examination of the *content* of the category 'e' statements in both groups. As a first step in this content analysis two psychologists were asked to rate the statements on dichotomy. Interrater reliability between the two psychologists was low (Cohen's Kappa = 0.41). Then, 5 psychologists with experience in cognitive therapy were asked, after a short training, to rate the statements independently from one another. In the meantime, in order to detect whether or not the dichotomous thoughts were actually disinhibitive, and could be expected to lead to counterregulation, two bulimia nervosa patients were asked if they had ever had comparable thoughts during a binge. If they had, this was considered an indication of disinhibiteness. However, interrater reliability among the 5 cognitive psychologists was low (Cohen's Kappa = 0.31), as was the interrater reliability between the bulimia nervosa patients (Cohen's Kappa = 0.42). To examine whether it was at all possible to distinguish restrained from unrestrained self-talk, a behavior therapist who works with anorexia and bulimia nervosa patients and a psychology student who was well acquainted with the concept of restraint, were asked to identify the thoughts as coming from a restrained or from an unrestrained eater. Again, interrater reliability was very low (Cohen's Kappa = 0.19).

DISCUSSION

As expected, unrestrained subjects regulated for a preload; they ate significantly more ice-cream in a no-preload condition than in a preload condition. However, ice-cream intake in restrained subjects without a preload did not differ from intake of those with a preload. Therefore, it was concluded that in restrained eaters, non-regulation rather than counterregulation took place. This finding is a fairly exact replication of the data obtained by Ruderman and Christensen (1983).

On the other hand, contrary to Ruderman's findings (1985), we did not find any relationship between restraint and irrationality. Restrained subjects scored just as high as unrestrained subjects. Moreover, we found no significant correlations between restraint and factor scores on the RBI and IBT. Therefore, we conclude that irrational thinking, as measured by the RBI and IBT, is not a necessary prerequisite for the occurrence of non-regulation.

Restrained eaters are supposed to think in a dichotomous way, and dichotomous thinking is believed to promote disinhibitive eating behavior. That is, overeating is thought to occur because restrained eaters think that they have already broken their diets.

The authors first examined whether the supposedly disinhibitive thoughts were more prevalent in restrained preloaded subjects than in subjects in the other conditions. We did, indeed, find differences in the frequency of disinhibitive thoughts between experimental groups, but independently from the restraint of subjects. With one exception, ("I cannot control myself"), the occurrence of disinhibitive thoughts was reported as often by preloaded, unrestrained subjects as by preloaded, restrained ones. Although all preloaded subjects reported having had these thoughts more often than subjects without a preload, unrestrained subjects regulated for their preload while restrained eaters did not. Clearly, 'disinhibitive' thoughts, as reflected in self-talk are not responsible for the occurrence of non-regulation.

In the present study, it was by no means possible to differentiate restrained from unrestrained self-talk. On dimensions of dichotomy, self-talk of restrained subjects could not reliably be differentiated from that of unrestrained subjects, not even by trained psychologists with experience in cognitive therapy. Nor did bulimic patients agree about having comparable thoughts during binges.

Before jumping to conclusions, we must say that we are aware of the methodological problems concerning the measurement of cognitions. It is questionable whether people actually do verbalize all of the things they are thinking to themselves (e.g. Nisbett and Wilson, 1977); even when asked to do so. However if they do not report these thoughts during eating, how can we be sure that these are, in fact, the thoughts that inhibit restrained eaters? It is interesting that, even retrospectively, restrained subjects did not report having had these thoughts.

Retrospectively, restrained subjects reported more often than unrestrained subjects that they could not control themselves during ice-cream tasting. This perceived uncontrollability was not dependent upon being preloaded or not. Clearly, this perceived uncontrollability is a better predictor for the occurrence of non-regulation in experimental situations like these than the supposedly 'disinhibitive' thoughts. This is also clearly in line with the finding of Ruderman *et al.* (1985), which suggested that "disinhibition is not an all-or-non phenomenon and that some degree of loss of control typifies restrained eaters in many situations involving food" (p. 555).

All in all, the present data provide robust confirmation of non-regulation in restrained eaters vis-à-vis unrestrained eaters. However, no differences were observed on questionnaire scores believed to reflect 'irrational' beliefs or thinking. The widely held and popular view that dichotomous thinking is responsible for non-regulation could not be corroborated. It is acknowledged that the interrater agreement on classifying self-statements as disinhibitive was fairly low. Still, the fact that hypothetical, disinhibitive cognitions are apparently not freely accessible to subjects poses fundamental methodological problems for a cognitive explanation of experimental disinhibitive eating. Of course, better instruments may eventually show different data. The fact that restrained eaters reported significantly more often not being able to control themselves was not dependent upon having had a 'disinhibitor', but was a more general characteristic of our restrained eaters. Exploration of this perceived uncontrollability in non-regulation might prove most rewarding.

5.6 no evidence that dieters counterregulate outside the laboratory

Anita Jansen¹, Henriëtte Swijman² & Marcel van den Hout¹.

abstract

In the present study, it was investigated whether dieters who counterregulate in the laboratory also overeat outside the lab. Pre- and post-experimental food intake of 27 dieters and 22 non-dieters who participated in a typical laboratory experiment on counterregulation were examined. It became clear that experimentally disinhibited dieters did not counterregulate outside the lab; neither in the short term (i.e. within 4 hours after the experiment), nor in the long term (i.e. within 24 hours after the experiment) and nor during the rest of the day. Neither did any shifts in macronutrient intake take place after the experiment.

introduction

In the laboratory, dieters do not regulate their food intake as non-dieters do. After a preload of e.g. milk shakes, non-dieters typically eat less than without such a preload: non-dieters regulate their food intake. Dieters, however, eat the same amount of food or even more after the consumption of a preload than without one: dieters do not regulate or they even counterregulate (Herman & Mack, 1975; Hibscher & Herman, 1977; Ruderman & Christensen, 1983; Jansen et al., 1988). Dieters' laboratory behavior has generally been considered as an appropriate model of bulimics' bingeing behavior (Polivy & Herman, 1985).

Several theories have been proposed to account for this counterintuitive and apparently illogical behavior in bingers/dieters. A rather popular theory states that, after dieters have started to eat, they are unable to stop eating because of a dominating biological urge. According to this theory,

1 Dept. of Mental Health Sciences / Experimental Psychopathology, Limburg University, P.O. Box 616, 6200 MD Maastricht, the Netherlands

2 Institute for Drugs, Safety and Behavior, Limburg University, the Netherlands

extreme dieters are characterized by a lowered serotonin (5-HT) level which can temporarily be restored by a binge. The ingestion of a relatively large amount of carbohydrates and little protein during a binge should heighten dieters' lowered serotonin level (Rosenthal & Heffernan, 1986; Kaye et al., 1984; Goldbloom, 1987).

Others, however, state that overeating after a preload can best be explained in cognitive terms (Tomarken & Kirschenbaum, 1984; Polivy & Herman, 1985; Spencer & Fremouw, 1979; Woody et al., 1981; Ruderman, Belzer & Halperin, 1985). In the laboratory it was found that the actual consumption of a high-calorie preload is not the only food stimulus which disinhibits dieters: the mere belief that a high-calorie preload has been eaten (Spencer & Fremouw, 1979; Woody et al., 1981), or even the anticipation of eating a high-calorie preload (Ruderman, Belzer & Halperin, 1985) also makes dieters overeat whereas this does not make non-dieters overeat. These findings led some authors to conclude that it is just the idea of a broken diet that led to non- c.q. counterregulation in non-dieters. After dieters consider their diet broken, they supposedly think *"I've ruined my diet for today, I might as well continue to eat"* (Tomarken & Kirschenbaum, 1984). Thus the idea of a lost day is supposed to lead dieters to overeat for the rest of the day.

If this is true, it follows that, after breaking their diet in a typical preload laboratory experiment, dieters will continue to overeat outside the laboratory as well. Two studies reported on this issue. Wardle & Beales (1987) studied whether experimentally preloaded dieters counterregulated outside the laboratory. They compared the pre-experimental calorie intake of dieters and non-dieters with the calorie intake on the day of the experiment. Outside the laboratory, both dieters and non-dieters neither counterregulated nor compensated for their food intake during the experiment. This finding seems to contradict the notion that disinhibited dieters overeat in the long term. It should, however, be noted that Wardle & Beales' subjects did not even counterregulate in the lab, so that overeating outside the building would have been miraculous in this sample. Recently, Weber, Klesges & Klesges (1988) also studied whether dieters who overate in the lab continued to overeat outside the lab. The authors found that experimentally disinhibited dieters overate outside the lab in the short term, that is, within 4 hours after participation in a laboratory experiment. They failed to find any effect in the long term: within 8 hours after the experiment all differences had disappeared.

From these studies it therefore appears that disinhibited dieters continue to overeat outside the lab, but only in the short term and not during the rest of the day. The absence of counterregulation during the rest of the day draws attention to possible biological mechanisms. 5-HT theory predicts that dieters will overeat in the short term, that is, until their 5-HT level is heightened again. 5-HT theory also predicts a shift in macronutrient intake: during counterregulation relatively large amounts of carbohydrates and little protein will be ingested. It is possible that dieters'

short-term overeating had the function of increasing their 5-HT level by the ingestion of relatively excessive amounts of carbohydrates. However, Weber et al. (1988) did not provide any data on macronutrient intake, and therefore we cannot be sure.

In the present study it was examined whether dieters whose diet is broken in the laboratory will overeat later on. It was hypothesized, according to cognitive notions, that experimentally disinhibited dieters will overeat in the long term (for the rest of the day or even longer). Dieters who do not break their diet and non-dieters are not expected to overeat outside the lab. In order to demonstrate a possible biological function of counterregulation, macronutrient intake in the short and in the long term was analyzed.

method

subjects

Forty-nine female students of health sciences at Limburg University participated in the present study. Mean age was 22 years ($SD=4.6$), mean BMI (Body Mass Index = $\text{weight}/\text{height}^2$) was 22.5 ($SD=3.3$). Subjects were classified as dieter or non-dieter on the basis of the median split score on the Restraint Scale (Polivy et al., 1978). Dieters ($n=27$) scored 17.4 points on the RS, whereas non-dieters' ($n=22$) mean score was 8. According to the hypothesis, not all dieters counterregulate outside the laboratory; only those who overeat in the lab. Therefore, subjects were classified as either disinhibited or non-disinhibited according to the median split score of the experimental calorie intake. Mean food intake of disinhibited subjects ($n=24$) in the lab was 367.4 calories, whereas non-disinhibited subjects ($n=25$) ingested 56.6 calories.

procedure

Subjects participated voluntarily in a study on 'food preferences': they intensively smelled a food 'preload' and their food intake was measured afterwards (data will be published elsewhere). Subjects were seen individually between 9.00 a.m. and 5.00 p.m. and the number of subjects in each condition was balanced at various times of the day. After the subject had participated in the laboratory experiment, she was subjected to an inquiry into her food intake by a trained interviewer. During the dietary interview, detailed information was obtained about the kind and the quantity of the subject's food intake from the day before up to the start of the laboratory experiment. After finishing the dietary recall, the subject was asked to monitor her food intake for the rest of that day and the next day. The eating diary was given a number and the subject was assured that the eating diary would remain anonymous. Moreover, she was given a reply-paid envelope for returning the diary by post, in the hope that these actions would heighten the reliability of the diaries.

analysis

Originally, 51 subjects participated in the laboratory experiment. All subjects except one returned the eating diary. One subject's diary could not be used because she had fallen ill. Thus, 49 subjects remained for the present data analysis. The food diaries were analyzed for calorie content and macronutrient composition with the use of the computerized Becel Food Program (NL 026) from the Dutch Unilever Research Laboratory. To this end, computer codes were assigned to the circa 3000 food items by two independent raters. The raters were blind to both the subject's dieting status and her food intake during the experiment. The interrater reliability was high (*Cohen's Kappa* = 0.85).

Macronutrient intake is presented in energy percentages while calorie intake is given in absolute amounts. Long-term food intake was defined as all intake of food and drink within 24 hours before and after the experiment. Food intake during the rest of the day was defined as all intake of food and drink after the experiment until the subject went to sleep. Short-term food intake was defined as all intake of food and drink within four hours after the end of the experiment. Difference scores between calorie/macronutrient intake on the (post-)experimental day vs calorie/macronutrient intake on the pre-experimental day over the same period were calculated. On the difference scores in calorie, carbohydrate, protein, and fat intake ANOVAs were carried out. Main factors were Group (Dieters vs Non-dieters) and Condition (Disinhibited vs Non-disinhibited). For analyzing simple effects, calorie and macronutrient intake on the (post-)experimental day were compared with calorie and macronutrient intake on the pre-experimental day over the same period by t-Tests for paired observations. Because of the large number of tests, a 1% rejection level was used.

results

long-term calorie intake

Calorie intake in the 24 hours before participation in the study was compared with calorie intake in the 24 hours after participation. The ANOVA revealed neither significant Main effects (Group: [$F(1,48)=0.27$, NS], Condition: [$F(1,48)=0.2$, NS]) nor a significant Interaction effect [$F(1,48)=0.76$, NS]. t-Tests did not show any effects either: in all conditions calorie intake in the 24 hours before participation in the study was equal to calorie intake in the 24 hours after participation.

calorie intake during the rest of the day

Calorie intake in the hours after participation in the study until the subject went to bed was compared with calorie intake in comparable hours on the previous day. Again the ANOVA did not reveal any significant Main effects (Group: [$F(1,48)=0.66$, NS], Condition: [$F(1,48)=0.39$, NS]) and no

**z
change
from
baseline**

figure 1: dieters' and non-dieters' post-experimental food intake in the long term, expressed in percentage change from their pre-experimental intake.

significant Interaction effect [$F(1,48)=0.18$, NS] emerged. Neither did t-Tests show any effects: in all conditions calorie intake during the rest of the day after participation in the study was equal to calorie intake on comparable times the day before.

short-term calorie intake

Calorie intake in the 4 hours after participation in the study was compared with calorie intake in comparable hours on the previous day. Again no significant Group [$F(1,48)=1.0$, NS] and Condition [$F(1,48)=0.32$, NS] effects emerged. The Interaction effect was not significant either [$F(1,48)=0.8$, NS]. t-Tests showed a trend for disinhibited dieters to eat less calories in the four hours after participation in the study than in comparable times on the previous day [$t(9)=2.35$, $p<0.05$]. No further effects were found: in the remaining three conditions, calorie intake in the 4 hours after participation in the study was equal to calorie intake on comparable times the day before.

is there a shift in macronutrient intake?

ANOVAs as well as t-Tests did not reveal any shifts in macronutrient intake after participation in the experiment; neither during the rest of the day, nor in the short and in the long term.

z
change
from
baseline

figure 2: dieters' and non-dieters' post-experimental food intake during the rest of the day, expressed in percentage change from their pre-experimental intake.

z
change
from
baseline

figure 3: dieters' and non-dieters' post-experimental food intake in the short term, expressed in percentage change from their pre-experimental intake.

discussion

In the present study, dieters who overate in the laboratory did not counterregulate outside the laboratory. They did not do so in the short term, i.e. within 4 hours after participation in the experiment, during the rest of the day and not in the long term either, i.e. within 24 hours after the experiment. The data do not support the notion that dieters overeat during the rest of the day once they consider their diet broken. 5-HT theory could not be confirmed either: no shift in macronutrient intake took place after the experiment.

Why do dieters not counterregulate outside the laboratory? It might be speculated that dieters stopped overeating after they left the laboratory because their hunger has been satisfied in the laboratory. If dieters' experimental overeating is considered as a consequence of a biological need (i.e. hunger), laboratory overeating should be sufficient to satisfy the biological need. But the idea of a biological need which has to be satisfied raises the question why non-disinhibited dieters do not overeat inside the laboratory. Non-disinhibited dieters were also confronted with food and were given the opportunity to eat but they could resist the alleged urge to eat.

Attention should be paid to the method of data gathering. Subjects were asked to *recall* their pre-experimental food intake, whereas they *monitored* their post-experimental intake. The post-experimental monitoring practices might have prevented dieters from overeating. The authors tried to restrict the monitoring-effect by assuring the subjects of optimal anonymity, but we cannot be sure that there was no intake reducing effect.

Nevertheless, the present study does not provide support for either the idea of (sustained) cognitive disinhibition or for the notion of a (transient) biological hunger which has to be checked. It might be hypothesized that dieters' overeating is stimulus specific. The presence of certain stimuli, such as a preload of ice cream or, as was the case in the present study, the intense smelling of food, might elicit craving in the dieter. As a result, the dieter will only overeat in the presence of certain stimuli, some of which were saliently present in the lab. After the dieter has left the lab, the stimulus is also left behind and craving will diminish. Because of this, overeating will also fail to occur. Testing this alternative and *post-hoc* explanation of stimulus-specific counterregulation might be fruitful.

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RESTRAINED EATERS ARE RAPIDLY HABITUATING SENSATION SEEKERS

ANITA JANSEN*, JACQUELINE KLAVER, HARALD MERCKELBACH and
MARCEL VAN DEN HOUT

Department of Mental Health Sciences, Limburg University, P.O. Box 616,
6200 MD Maastricht, The Netherlands

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Summary—Several authors have pointed to similarities between eating disorders and addictive behaviors. In earlier studies, addicts were found to score high on the Sensation Seeking Scale (SSS) and to habituate rapidly to neutral stimuli. In this study, we found experimental support for an addiction model of eating disorders: restrained eaters also scored significantly higher on the Sensation Seeking Scale and also habituated significantly quicker to a series of neutral stimuli than unrestrained eaters. No clear evidence was found for the hypothesis that restrained eaters score lower on measurements of anxiety. It is hypothesized that rapid habituation promotes sensation seeking, which may be manifested in excessive consumption of either drugs or food. Restraint may be a strategy to prevent negative consequences of sensation seeking and excessive consumption. The model is related to earlier experimental findings.

INTRODUCTION

Several authors have pointed to similarities between eating disorders and addictive behaviors (Scott, 1983; Sinnet, Judd and Olsen, 1983; Dumont and Vamos, 1983; Brisman and Siegel, 1984; Lacey and Evans, 1986; Lacey and Mourel, 1986; Wardle, 1987; Filstead, Parela and Ebbitt, 1988). Both eating disorders and addictions are characterized by craving, preoccupation, and a loss of control over intake. Yet, so far the analogy between eating disorders and addictions is just a theoretical one, which has not been put to direct experimental tests.

Zuckerman (1979) found that individuals who are likely to develop habits of substance abuse are high sensation seekers, where sensation seeking is defined as "a need for varied, novel, and complex sensations and experiences, and the willingness to take physical and social risks for the sake of such experience" (Zuckerman, 1979, p.10). High sensation seekers seek stimulation and stimulation change (Gale and Edwards, 1986). Addicts were found to score higher on the Sensation Seeking Scale (SSS; Zuckerman, 1979) and they also habituated more rapidly to neutral stimuli than normal controls (Zuckerman, 1979). Considering the analogy between addictions and restrained eating (Wardle, 1987) it may be hypothesized that restrained eaters (1) score higher on the SSS, and (2) habituate faster to neutral stimuli than unrestrained eaters.

Indirect support for this hypothesis comes from a study of Calloway, Fonagy and Wakeling (1983). These authors found faster habituation rates of skin conductance responses (SCRs) in bulimic *Ss* than in normal controls and restricting anorexia nervosa (AN) patients. However, the results of this study should be interpreted cautiously; faster habituation rates in patients with bulimia may be induced by a disturbed skin conductance, as a consequence of the electrolyte imbalance resulting from purging activities. Calloway *et al.* (1983) also found less spontaneous fluctuations in skin conductance of bulimics compared to AN-restricters and normal controls. Their finding of less spontaneous fluctuations and faster habituation rates in bulimics, and the fact that anxiety states are often associated with more spontaneous fluctuations (Orr and Pittman, 1987) and slower habituation rates (Lader, 1980; Lelliott, Noshirvani, Marks, Monteiro, Basoglu and Cohen, 1987) provided a third hypothesis: (3) restrained eaters score lower on anxiety measures than unrestrained eaters.

To sum up, the primary aim of this study was to investigate the tenability of the addiction model to restrained eating: do restrained eaters score higher on the sensation seeking scale and do they

*To whom all correspondence should be addressed.

habituate quicker to neutral stimuli than unrestrained eaters? Furthermore, we wanted to elaborate the Calloway study by finding out if their data can be reproduced in a sub-clinical sample, and if indeed restrained eating is inversely related to measures of anxiety.

METHOD

Subjects

296 Female students of the Limburg University were invited to complete the Restraint Scale (Herman, Polivy, Pliner and Threlkeld, 1978). From the 73 students who completed the questionnaire, 11 high restrained and 11 low restrained scorers were asked to participate in the experiment. *S* characteristics are shown in Table 1.

Materials, assessment and measurement

The psychological measurements consisted of Dutch translations of:

- (1) the *Restraint Scale (RS)* (Herman *et al.*, 1978), consisting of 10 items measuring attitudes toward eating, frequency of dieting, and weight fluctuations;
- (2) the *short Sensation-Seeking Scale (SSS)* (Madsen, Das, Bogen and Grosman, 1987). This scale consists of 10 items and has proven to be a reliable and valid measure of sensation seeking. Scores on the short SSS correlate strongly ($r = 0.78$) with those on the total SSS;
- (3) the *Fear Questionnaire (FQ)* (Marks and Mathews, 1979); a 15-item self-rating scale which has proven to give reliable and valid indications of avoidance of certain situations;
- (4) the *Spielberger State-Trait Anxiety Inventory* (Van der Ploeg, Defares and Spielberger, 1980), a 40-item inventory measuring self-reported state anxiety and trait anxiety. State anxiety measures momentary anxiety: a transitory emotional condition which varies in intensity and fluctuates in time as a reaction on a certain situation. Trait anxiety refers to a stable and enduring individual difference in anxiety proneness.

The physiological measurement consisted of skin conductance responses (SCRs) to a series of auditory stimuli. Skin conductance level (SCL) and SCRs were picked up from Beckman Ag-AgCl electrodes (diameter 8 mm), attached to the medial phalanges of the second and third finger of *Ss*' right hand. Electrodes were connected to a Beckman Skin Conductance Coupler (Type 9844), using the method of constant voltage (0.5 V). Respiration rate was recorded with a Beckman Respiration Belt, connected to a Beckman Pressure/Pulse/Voltage Coupler (Type 9884), SCR and respiration were monitored on a Beckman R711 polygraph.

The auditory stimuli were $14 \times 1000\text{Hz}$, 85 dB tones binaurally presented through Beyer dynamic earphones (type DT109) connected to a Sony cassette recorder (TCK 88). Signals were held on the exact dB level by a Dual Signal Gate (Symetrics, type SG 200). The tones were presented at random intervals. Intertrial intervals varied between 15 and 30 s, with an average of 20 s. Duration of the tone was 6 s. A prefabricated tape was used to generate the tones.

Response definition and analysis

SCLs were measured during the pre-experimental baseline period. SCRs were defined as the maximal deflections occurring between 1 and 5 s after stimulus onset. SCLs and SCRs were measured in micromho and square-root transformed.

Two indices of habituation of the SCRs were used. Response decline over trials were analyzed by subjecting the SCRs magnitudes for each trial to a 2 (Group: Restrained vs Unrestrained) $\times$ 14

Table 1. *S* characteristics

	Restrained <i>Ss</i> M (SD)	Unrestrained <i>Ss</i> M (SD)	<i>t</i>	<i>P</i> *
Age	22.4 (3.4)	20.5 (2.6)	1.48	NS
BMI	23.2 (2.3)	20.4 (1.7)	3.29	0.002
RSS	14.5 (3.5)	4.5 (1.4)	8.83	0.001

*One-tailed.

BMI = Body Mass Index-weight/height³.

RSS = Restraint Scale Score (Herman *et al.*, 1978).

(Trials) Analysis of Variance (ANOVA), with the last factor being a repeated measure. Furthermore, a trials-to-criterion measure of habituation was used: habituation was said to have occurred when *Ss* failed to respond (SCRs smaller than 0.05 micromho) on two successive trials. Group differences in mean-trials-to-criterion were evaluated with a *t*-test.

Additionally, spontaneous fluctuations (SFs) were counted. SFs were defined as SCRs which exceeded 0.05 micromho, occurring before stimulus onset or at least 5 s after stimulus offset.

Respiration was used as a control variable: trials with respiratory irregularities were excluded from the data analysis. SCR values for these trials were estimated on the basis of the mean SCRs on adjacent trials.

Procedure

After entering the laboratory, the *S* was asked to complete the short SSS and FQ. The *S* sat in a comfortable chair which was placed in a dimly lit, sound-attenuated chamber. The tape recorder and recording apparatus were in an adjacent room. After cleaning *S*'s fingers with distilled water, electrodes were attached. Before baseline measurement started, the *S* was told that during the experiment she would hear something through the earphones. Baseline measurement started and SCL was registered. After a 4 min period of baseline measurement the series of tones was presented. Next, the *S* completed the State-Trait Anxiety Inventory. Then her weight and height were measured, and finally, she was debriefed and paid for participation.

RESULTS

The results are shown in Table 2.

Table 2. Test scores and skin conductance measures for 11 restrained and 11 unrestrained eaters

	Restrained <i>Ss</i> M (SD)	Unrestrained <i>Ss</i> M (SD)	<i>t</i>	<i>P</i> *
Sensation Seeking	7.09 (1.45)	5.55 (1.5)	2.45	0.01
Fear Questionnaire	25.0 (6.39)	31.6 (11.9)	1.63	0.06
State Anxiety	33.18 (9.2)	34.55 (8.7)	0.36	NS
Trait Anxiety	41.27 (13.1)	37.82 (6.9)	0.77	NS
Mean Trials to Criterion	9.09 (2.95)	11.55 (2.98)	1.94	0.03
Spontaneous Fluctuations	7.9 (6.5)	10.6 (6.98)	0.95	NS

*One-tailed.

As is evident from Table 2, hypothesis 1 was supported: Restrained *Ss* scored significantly higher on the short SSS than Unrestrained *Ss*. The picture was less clear as the hypothesis of Restrained *Ss* being unanxious was concerned: Restrained and Unrestrained *Ss* did not differ in State and Trait Anxiety. On the Fear Questionnaire Restrained *Ss* tended to score lower; this difference was marginally significant. Closer inspection of the data revealed no differences between Restrained and Unrestrained *Ss* on the three subscales (agoraphobia, social anxiety, and blood-injury) of the FQ.

Restrained and unrestrained *Ss* did not differ in mean SCL at the start of the experiment, the means (micromho) being 7.4 (SD = 3.4) and 7.0 (SD = 2.9) respectively [$t(20) = 0.23$, NS]. Also, there was no difference in Orientation Response, defined as the SCR to the first stimulus, between Restrained and Unrestrained *Ss*: means were 2.2 (SD = 1.2) and 1.8 (SD = 1.7) respectively [$t(20) = 0.63$, NS].

In line with the hypothesis, Restrained *Ss* habituated significantly faster to the series of tones than unrestrained *Ss*. The group differences in habituation were evident in both indices of habituation rate: (a) restrained *Ss* reached the criterion of non-responding much earlier than unrestrained *Ss* (see Table 2), and (b) SCRs of restrained *Ss* declined more rapidly over trials than did SCRs of unrestrained *Ss* [Group $\times$ Trial interaction: $F(1, 20) = 2.1$, $P = 0.01$]. To control for BMI differences, the ANOVA was repeated with BMI as covariate. The ANCOVA showed significant main effects for Trials [$F(1, 20) = 28.56$, $P = 0.00$] and for Groups [$F(1, 20) = 5.38$, $P = 0.032$], and again a significant Group $\times$ Trials interaction [$F(1, 20) = 2.1$, $P = 0.01$] emerged.

However, contrary to what was predicted, we observed no between group differences in the number of Spontaneous Fluctuations (see Table 2).

DISCUSSION

The first and second hypothesis, stating that restrained eaters score higher on the short SSS and habituate quicker to a series of neutral stimuli than unrestrained eaters, were confirmed. The third hypothesis, of restrained eaters scoring low on measurements of anxiety, was partly supported: restrained eaters tended to score lower on the FQ than unrestrained eaters, but no difference emerged between both groups in State and Trait Anxiety. Also, the number of Spontaneous Fluctuations, the frequency of which was found to be higher in anxiety states (Lader, 1980; Orr and Pitman, 1987), did not differ between restrained and unrestrained eaters.

The findings of our study fit with the addiction model of restrained eating. Zuckerman (1979) showed that addicts are rapidly habituating sensation seekers, and in line with this we showed that restrained eaters are rapidly habituating sensation seekers. Since especially high scorers on the "disinhibition" subscale of the total SSS showed fast habituation of the SCR (see Gale and Edwards, 1986)—while this "disinhibition" subscale measures the hedonistic pursuit of pleasure through extraverted activities such as social drinking, sex and parties—it may be of interest to test whether restrained eaters are particularly high "disinhibition" scorers.

The fact that we could reproduce Calloway's findings in a subclinical sample of restrained eaters strongly supports that Calloway's findings did not result from purging activities of their bulimics, but that they are associated with addiction-like behavior. Calloway's observation of fewer SFs in bulimics is in sharp contrast with our findings: we found only marginal differences in anxiety measurements between groups. Calloway *et al.* (1983) relate bulimia to psychopathy because of their faster habituation rates and fewer SFs in Skin Conductance. However, it should be noted that the psychophysiology of psychopathy is different. Psychopaths are characterized by faster habituation rates, but also by tonic lower levels of SC and smaller ORs (Zahn, 1986). In our study and in the study of Calloway *et al.* (1983) no differences in OR and SC levels were found between restrained eaters and bulimics respectively, and normal controls. A resemblance of the electrodermal activity of bulimics and psychopaths may thus be restricted to rapid habituation, it does not include ORs and SCLs.

Research on overeating after a preload may help elucidate the present findings. Analogous to a binge of bulimics, which may be triggered by eating a small amount of food, restrained eaters in the laboratory were found to lose control over their food intake after a small preload (Herman and Mack, 1975; Hibscher and Herman, 1977; Jansen, Merckelbach, Oosterlaan, Tuiten and van den Hout, 1988a; Jansen, Oosterlaan, Merckelbach and van den Hout, 1988b). This uncontrollable of food intake after a preload is usually thought to be a consequence of the preload breaking the restraint. The common line of reasoning runs as follows. Binges of bulimics, and sub-clinical over-eating in laboratory situations, are typically preceded by periods of dieting (Wardle, 1980; Polivy and Herman, 1985; Ruderman, 1985, 1986). The explanation given for overeating is that dieting suppresses the body weight below a biological set-point for body weight. Breaking the psychological restraint, e.g. by eating a preload, triggers a biological urge to eat (Herman and Mack, 1975). However, the concept of set-point is rather untestable, and analogous to the overeating/bingeing response in Ss preoccupied with eating, is the drinking response of restrained alcoholists after a preload of alcohol. After drinking a small amount of alcohol the alcoholist engages in a "drinking binge" (Scott, 1983). And it is questionable whether this has anything to do with an inborn, naturally predetermined, set-point.

Considering that (1) rapid habituation to a series of neutral stimuli is characteristic of addicts as well as restrained eaters, (2) addicts and restrained eaters are high sensation seekers, and (3) overconsumption after breaking their restraint is characteristic of both addicts and restrained eaters, the following speculation is made. People who are rapidly habituating to stimuli need a lot of stimulation to reach an optimal level of stimulation: they are high sensation seekers. When high sensation seekers give in to their need of much stimulation, they run the risk of being excessive consumers. This excessive consumption may have negative consequences, e.g. being fat or having a hangover. In order to prevent these negative consequences, the rapidly habituating sensation seekers need to restrain themselves. In this view, restraint is considered to be a consequence of the rapid habituation: rapidly habituating individuals seek stimulation and thus are prone to excessive intake. Then restraint is a preventive means for not suffering from aversive results following

excessive consumption. Breaking the restraint leads to overconsumption, and has thus much more to do with the rapid habituation rates and need for stimulation than with a biologically predetermined set-point for body weight. Schematically:

There are various lines of evidence to support this view.

First, there is the often reported cross-over and co-existence in addictive behavior. Eating-disordered patients often report excessive alcohol drinking or drug abuse in the past or present (Brisman and Siegel, 1984; Jonas and Gold, 1988; Beary, Lacey and Merry, 1986; Lacey and Mourel, 1986), and several studies indicate that certain groups of drug abusers may have (had) elevated rates of eating disorders (Jonas and Gold, 1988). Clearly, being excessive in one modality may often simply be replaced by/or be accompanied with excessiveness in another modality in order to be stimulated one way or the other. It would be of interest to test whether the sub-clinically restrained eaters also show restraint in other behaviors, like drinking or drug abuse.

Second, from this theory it can be predicted that slowing down the habituation rate may reduce the need for external stimulation and thus excessive consumption. If someone is exposed to high levels of environmental stimulation, habituation rate will be reduced (Zahn, 1986). Thus, it may be hypothesized that restrained eaters (1) will habituate normally when they are exposed to high levels of environmental stimulation, and (2) will regulate normally for a preload when they are optimally stimulated. Indirect support for this hypothesis is given by a recent study of Herman, Polivy, Lank and Heatherton (1987). As mentioned before, laboratory studies on food intake stress the importance of dieting status and degree of repletion, i.e. being preloaded or not, as main determinants for food intake: restrained eaters do not regulate their food intake after a preload, while unrestrained eaters do (Herman and Mack, 1975; Hibscher and Herman, 1977; Herman and Polivy, 1980; Ruderman and Christensen, 1983; Jansen *et al.* 1988a, b). Herman *et al.* (1987) stressed the importance of a third factor: level of anxiety. In particular, the interaction between dieting status, degree of hunger/repletion and level of anxiety is supposed to determine food intake. In a recent study, Herman and co-workers (1987) found that only preloaded dieters in a low anxiety condition counter-regulated. Dieters in a high anxiety condition regulated: they ate less after a preload than without one.

Highly intriguing in the study of Herman *et al.* (1987) is that highly anxious restrained eaters regulated their food intake after a preload, just like lowly anxious unrestrained eaters do. Post hoc analysis of our own data on food intake of restrained and unrestrained eaters after a preload or no-preload (Jansen *et al.*, 1988a) revealed the same pattern of regulation in highly anxious dieters after a preload. To explain the disappearance of counterregulation in anxious dieters, Herman *et al.* (1987) suggest that only a first disinhibitor (anxiety or a preload) induces overeating in restrained subjects. After a first disinhibitor the diet is no more intact and, as a consequence, a second disinhibitor will have no more effect; "Anxiety cannot disinhibit a dieter who is already disinhibited" (p. 266). Thus, when restrained eaters are *either* preloaded *or* anxious, they will counterregulate, but when they are preloaded *and* anxious, only the first disinhibitor will be an effective disinhibitor. However, if that is true, one should not expect highly anxious restrained eaters to regulate their food intake after a preload. This explanation does not cover the available data. It should be noted that either the preload or the anxiety should have disinhibited the restrained eater and should thus have resulted in excessive food intake. This was not the case. With the explanation given by Herman *et al.* (1987) the crucial issue remains unsettled: what causes lowly anxious restrained eaters not to regulate their food intake after a preload, while highly anxious restrained eaters do regulate for a preload just like normally unrestrained eaters? From the present model it appears that the introduction of anxiety slowed down the habituation rates of restrained eaters. The slower habituation rate as a consequence of increased anxiety diminishes the need for external stimulation and thus excessive consumption, leading to normal regulatory behavior. This post-hoc explanation of the Herman *et al.* data has to be subject to experimental tests: do restrained eaters (a) habituate normally when they are exposed to high levels of environmental stimulation, and (b) will they regulate normally for a preload when they are optimally stimulated?

The model given earlier in this discussion was suggested by the present data and it covers some existing findings quite well. Meanwhile, a model is as good as the predictions that flow from it. Some of the most straightforward implications are given below. As mentioned, restrained eaters (1) should especially be high "disinhibition" scorers, (2) should also show restraint in other behaviors, like drinking or drug use, (3) should habituate normally when they are exposed to high levels of environmental stimulation, and (4) should regulate normally for a preload when they are optimally stimulated. Furthermore, (5) it is postulated that the sensation seeking, excessiveness, and restraint result from rapid habituation. The model predicts that rapidly habituating people (a) score higher on sensation seeking scales, (b) score higher on restraint scales, and (c) show more excessive behaviors than slowly habituating people. Finally, (6) it is theorized that sensation seeking leads to excessive consumption and ends up in restraint: are high sensation seekers more often either excessive consumers or restrained *Ss* than low sensation seekers? Clarification of these issues may help understand the intriguing relationship between eating disorders and substance abuse; it may prove a rewarding enterprise.

5.8 on being led into temptation: 'counterregulation' of dieters after smelling a 'preload'

Anita Jansen¹ & Marcel van den Hout¹

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abstract

In the present study, it was found that dieters lost control over their food intake and 'counterregulated' after merely smelling a 'preload', while dieters without a 'preload' were able to maintain control over their food intake. Non-dieters, however, even ate marginally less after smelling a 'preload' than they did in a 'no-preload' condition. Apparently, actually breaking one's diet and such subsequent thoughts as *"I've blown my diet, I might as well continue to eat"* is not necessary for counterregulation to occur. The observation that merely smelling a 'preload' is sufficient to produce 'counterregulation' in dieters but not in non-dieters challenges the explanatory power of the widely held cognitive explanation of experimental counterregulation in preloaded dieters. With reference to earlier findings on counterregulation and to findings in the field of addictive behaviors, the authors suggest that overeating may result from a preparatory hyperinsulinemia/hypoglycemia response which can be elicited by several disinhibitors.

introduction

A well-documented finding in laboratory research on food intake is that dieters eat more after forced consumption of a preload than when they are not given a preload. This phenomenon is called counterregulation and is usually explained in cognitive terms. The general idea is that it is the awareness of a broken diet that disinhibits the dieter. Dieters tend to respond to a violation of their diet with thoughts such as *"I've blown my diet, I may as well continue to eat"*, and, thus, are led to temporarily abandon their restraint (Polivy, 1976; Ruderman and Wilson, 1979; Polivy

¹ Dept. of Mental Health Sciences / Experimental Psychopathology, Limburg University, P.O. Box 616, 6200 MD Maastricht, the Netherlands.

and Herman, 1985; Ruderman, Belzer and Halperin, 1985; Ruderman, 1985, 1986). However, there is reason to doubt about the explanatory power of the assumed disinhibitive thoughts. Until now, no unequivocal support for the "cognitive explanation" of counterregulation has been documented. And what's more, an experimental study of dieters' self-talk during overeating provided no confirmation of the very widely accepted view that dieters are disinhibited by thoughts; non-regulating dieters were *not* characterized by a different set of cognitions or self-talk during food intake than regulating non-dieters (Jansen et al., 1988).

It should be noted that the measurement of cognitions poses serious methodological problems, and it is questionable whether it is ever possible to precisely analyze the stream of consciousness that accompanies certain behaviors of people. It may be more fruitful to deduce *testable* hypotheses from the cognitive perspective on counterregulation. A direct implication of the idea that dieters in the usual preloadparadigm only are disinhibited by the very awareness of a broken diet is that not breaking the diet will logically not result in a counterregulatory response. In the present study, the diet was not broken; subjects merely concentrated intensely on the smell of a 'preload'. Despite the fact that no real preload was given and thus no real counterregulation could take place, the authors have chosen to use these terms to stress the similarity of design of the present study with the usual preloadparadigm. Because no evidence of disinhibitive elements in the stream of consciousness of non-regulating dieters could be found in the Jansen et al. (1988) study, it was reasoned that not only may disinhibitive thinking be unnecessary for counterregulation but actually preloading dieters may not be a *sine qua non* for counterregulation. It was, therefore, hypothesized that dieters would lose control over their food intake and 'counterregulate' after merely smelling the 'preload', while dieters who do not smell the 'preload' would not lose control over food intake and, thus, be able to keep to their diet. It was further hypothesized that non-dieters in both conditions would maintain control over their food intake and eat similar amounts whether or not they had smelled the 'preload' intensively.

method

subjects

Fifty-one female students and employees of the University of Limburg volunteered to complete a questionnaire on food preferences. The hypotheses under review concern differences between restrained and unrestrained subjects. To increase chances of finding the hypothesized effect, it was decided beforehand to compare relatively extreme groups. Given the fact that the median split score of our subject sample on the RS in an earlier study was 12 (Jansen et al., 1988), it was decided to classify subjects scoring a quarter higher (≥ 15) as high restrained eaters, i.e.

dieters, and subjects scoring a quarter lower (≤ 9) as low restrained eaters, i.e. non-dieters. Sixteen subjects scored between 9 and 15 on the RS and they were not included in the analysis of the data. We thus ended up with 36 subjects. Subject characteristics are shown in Table 1.

table 1: subject characteristics

	dieters (n=19)	non-dieters (n=17)	t
age	24.4 (6.7)	20.9 (2.0)	2.0*
BMI [#]	25.2 (3.5)	19.9 (1.3)	5.8**
RS ^{##}	19.6 (3.3)	6.9 (2.1)	13.6**

* $p=0.05$; ** $p=0.000$; [#] BMI = Body Mass Index (weight/height²); ^{##} RS = Restraint Score

assessment

The following instruments were used:

The Restraint Scale (RS) (Polivy et al., 1978), measuring attitudes toward eating, frequency of dieting, and weight fluctuations.

The Perceived Control List (PCL). A list of six statements concerning perceived control over food intake was composed by the authors (see Table 2). Each statement was scored on a 100 mm visual analogue scale (VAS), with scores ranging from 0 ("not at all") to 100 ("extremely").

The dependent variable was the total **caloric intake**.

table 2: the Perceived Control List (PCL)

1	I couldn't control my eating (0-100).
2	I found it difficult to stay away from the food (0-100).
3	I felt compelled to go on eating (0-100).
4	I couldn't stop eating (0-100).
5	I ate more than I actually meant to eat (0-100).
6	I had complete control over my eating behavior (100-0).

procedure

Ss were asked to have breakfast, lunch, or dinner 1-3 hours before participating in the study. Food intake thus served as a caloric overload and could not be influenced by deprivation. Ss were randomly assigned to the 'preload' or 'no-preload' condition. All Ss were run individually between 9.00 a.m. and 5.00 p.m. Care was taken to balance the number of Ss run in each condition at various times of the day. After entering the

laboratory, Ss in the 'no-preload' condition spent 12 minutes completing a questionnaire on sensation seeking which, for this study, was irrelevant. They did this in a neutral place. Subjects in the 'preload' condition were directly presented with 750 grams of Dutch liquorice, 900 grams of English licorice, 500 grams of cake, 500 grams of smarties, 400 grams of nuts, 400 grams of spiced biscuits, 350 grams of shortbreads and 400 grams of soft sweets. The food was presented in large amounts and, when necessary, broken into small pieces so that later in the experiment subjects could inconspicuously eat a lot. Subjects in the 'preload' condition were stimulated by the experimenter, who was in the same room as the subject, to hold the dishes with the food directly under her nose and to concentrate on the smell of the food. Subjects completed a questionnaire on the smell of the different food items. After 12 minutes of exposure, subjects were given a questionnaire on preferences concerning the food items they had been presented with. Subjects in the 'no-preload' condition entered the room with food items after their 12 minutes in the no-exposure, neutral place. They were also given the food preference questionnaire. Subjects in both conditions were instructed to fill in the questionnaire and they were invited to eat as much as they wanted to, but they were not obliged to do so. The food preference questionnaire was constructed in such a way that tasting the food was not necessary in order to answer the questions; this way the subject had the option of eating nothing at all. Each subject was left alone for 15 minutes to complete the food preference questionnaire (in a pilot study it was found that subjects needed 7 to 8 minutes to complete the questionnaire). After their 15 minutes alone in the room with the food items, the experimenter returned and the subject was asked to answer the questions of the PCL with regard to their behaviour in the previous task, i.e. when completing the food preference questionnaire. The subject was then taken to a second experimenter in another room where she filled in the RS. Her weight and height were determined and, after debriefing, she was paid for her participation. The food that remained was then reweighed by the first experimenter and, after conversion into calories, the subjects' total caloric intake was determined.

results

analysis

Because dieters and non-dieters differed significantly in BMI and age (see Table 2), analyses of covariance (ANCOVA) were carried out on the data. The main factors were Group (dieters vs non-dieters) and Condition ('preload' vs 'no-preload'), with BMI and age as covariates. If hypotheses 1-3 were true, the Group x Condition ANCOVA would show specific interaction effects, namely, 'counterregulation' and a perceived loss of control after a 'preload' in the case of dieters, and no effect of the 'preload'

figure 1: caloric intake and loss of control of dieters and non-dieters after a 'preload' and without a 'preload'.

in the case of non-dieters. The interactions were tested by means of pre-planned contrasts, and the specific *within*-group effects were tested by means of t-Tests. The results are shown in Fig.1.

caloric intake

The ANCOVA showed a significant main effect for Group (pre-planned contrasts: $t(1,35)=2.0$, $p=0.03$, one-tailed), meaning that dieters' caloric intake was higher than non-dieters' caloric intake. As hypothesized, the Group x Condition interaction was significant ($t(1,35)=1.85$, $p=0.04$, one-tailed); thus, the 'preload' was related to a higher caloric intake in dieters, whereas it was related to marginally less intake in non-dieters. Closer inspection of the data by t-Tests showed that dieters ate significantly more after smelling the 'preload' intensively than they did when not smelling a 'preload' ($t(17)=1.95$, $p=0.03$, one-tailed), and although non-dieters ate less after smelling the 'preload' than when not smelling one, the difference in food intake was not significant ($t(15)=1.13$, NS).

perceived control

Ss retrospectively rated their perceived control over food intake on the 6-item PCL. For each subject, the scores on the six PCL items were added up, with a higher total score meaning a heightened perceived loss of control. The ANCOVA showed a significant main effect for Group ($t(1,35)=1.93$, $p=0.03$, one-tailed), meaning that dieters retrospectively perceived more loss of control than non-dieters. Also, as hypothesized, the interaction Group x Condition was found to be significant ($t(1,35)=1.9$, $p=0.03$, one-tailed). As is clear from Figure 1, no differences in perceived control emerged between dieters and non-dieters in the 'no-preload' condition. However, in the 'preload' condition, i.e. after smelling the food intensively, dieters lost control while non-dieters even managed to increase control.

discussion

In the present study, the typical pattern of 'counterregulation' was replicated. In accordance with the retrospectively reported loss of control over food intake, dieters in the 'preload' condition 'counterregulated' after merely smelling the 'preload'. In contrast, dieters in the 'no-preload' condition did not report a loss of control over food intake; they were, indeed, able to keep to their diets. Surprisingly, but in line with their subjectively experienced heightened control, non-dieters even ate less after the exposure ritual.

It should be noted that the present data were derived from subject samples selected on the basis of extreme scores on the RS. When data *post-hoc* were re-analyzed using a median split categorization, the observed interaction dropped to a non-significant level. In most of the relevant studies on counterregulation after eating a preload, dieters and non-dieters are selected by a median split procedure. Possibly, a strong manipulation (*eating* a preload) requires less stringent selection criteria (median split), whereas a weaker manipulation (*smelling* a preload) requires stringent selected subject samples (extreme scorers) to find the effect, i.e. counterregulation.

The fairly exact replication of 'counterregulation' in dieters who were merely exposed to, and intensively smelled, the 'preload' but whose diets were not actually broken, raises a problem for the cognitive explanation that dieters counterregulate after eating a preload because their diet has already been broken. The present study shows that, in the preload-paradigm, breaking a diet is not necessary for counterregulation to occur; merely smelling a 'preload' is enough to make high restrained subjects lose control and overeat. Of course, the present findings do not exclude the disinhibitive power of certain cognitive manipulations. A most intriguing finding is that manipulation of the dieters' beliefs concerning the caloric content of the preload influences subsequent consumption (Polivy, 1976; Spencer and Fremouw, 1979). In a very elaborate study, Spencer and Fremouw (1979) found that dieters who were just told that they had consumed a preload rich in calories counterregulated, while dieters who believed their preload was low in calories did not overeat.

How should we account for these apparently conflicting findings? Counterregulation of dieters has been found to occur on several occasions. Generally speaking, dieters have been found to lose control over food intake after forced consumption of a preload (Herman & Mack, 1975; Hibscher & Herman, 1977; Jansen et al., 1988), when they are in a strong emotional state, as when depressed or anxious (Baucom & Aiken, 1981; Frost, Goolkasian, Ely & Blanchard, 1982), after manipulation of cognitive beliefs (Polivy, 1976; Spencer & Fremouw, 1979) and, as the present study shows, after intensive smelling of food. Despite the fact that various disinhibitors lead to the identical phenomenon of overeating, different processes are proposed to explain how a particular disinhibitor leads the

dieter to overeat. For example, the perception of having overeaten is supposed to be the reason why a preload disinhibits the dieter, while a loss of motivation to diet is thought to be the disinhibitive factor in depressed dieters. Yet, one should not rule out the possibility that one and the same mechanism underlies all of the aforementioned counter-regulatory challenges. For reasons of parsimony, a unifying theory seems preferable.

The analogy between addictive behaviors and overeating may be of relevance here. In the past decade several authors have pointed to similarities between substance abuse disorders and overeating (Bemis, 1985; Wardle, 1987; Jansen et al., 1989). Both addicts and individuals who overeat are characterized by a preoccupation with a particular substance, a craving for the substance, and a loss of control over intake (Bemis, 1985; Ruderman et al., 1985; Wardle, 1987; Jansen et al., 1988). The results of recent animal studies provide empirical support for a classical conditioning model of craving and drug tolerance. After repeated association of cues specifically related to the substance taken, these cues (*conditioned stimuli*; CS) come to elicit a response (*conditioned compensatory response*; CCR) which is opposite in direction to the unconditioned response of the substance (Siegel, 1983). Over the course of repeated substance administration in the presence of perceivable cues, the CCR moderates the unconditioned substance response and tolerance is acquired. The CCR, which is triggered by contextual cues (CS), may be considered a "preparatory" response, that is, the CR anticipates and compensates for the effects of the substance in order to maintain a homeostatic balance. It is this CCR that may cognitively be labeled as "craving" (for reviews, see Siegel, 1983 and Macrae et al., 1987). Conceptualizing dieters' overeating response as addictive behavior, the cues normally associated with excessive food intake would be considered CS. Interestingly, exteroceptive as well as interoceptive stimuli may act as CS's (Ludwig and Wikler, 1974). Exteroceptive stimuli comprise a variety of environmental stimuli associated with prior food-taking behavior, such as the smell of food, seeing other people eating, or being in an environment where overeating often takes place. Interoceptive stimuli refer to the internal physiological effects of a preload as well as to emotional dysphoria and cognitive processes (Ludwig and Wikler, 1974).

With respect to the CCR, insulin release and blood sugar level may play a role. Normally, eating elevates the blood sugar level, and insulin is then released in order to bring the blood sugar level back down again. Several authors have pointed to environmental and cognitive influences on insulin secretion (see Rodin, 1985). Tasting, smelling, or even merely seeing or thinking of food is found to trigger an insulin response (Rodin, 1985). The hyperinsulinemia response has been associated with increased hunger, heightened perceived pleasantness of sweet taste, and increased food intake (Rodin, 1985). This anticipatory hyperinsulinemia response may be considered a "preparatory" response: with heightened insulin secre-

tion blood sugar level declines, thereby anticipating and compensating for the effects of subsequent food intake. It is the increase in insulin and subsequent decline in blood sugar level (CCR) that may subjectively be experienced as "craving".

Indirect support for the idea of learned responses being responsible for binge eating/counterregulation comes from salivation studies. Dieters on a highly invariable diet repeatedly were found to salivate significantly less to food cues than dieters on a highly variable diet (Legoff, Leichner and Spigelman, 1988; Klajner et al., 1981; Herman et al., 1981; Wooley & Wooley, 1981). If certain cues (olfactory, taste, visual, cognitive etc.) are sometimes followed by particularly strong UCSs (high calorie intake) it can be expected that 'unsuccessful' dieters (i.e. high scorers on the Restraint Scale, see Jansen et al., 1988) show strong CRs to any cues that typically predict food intake. It follows that counterregulation of dieters should occur not only if actual diet breaking is expected, as cognitive theory would predict. The clinical implication that follows from the assumption that binges follow exaggerated CRs that are due to the probabilistic CS (*cue*) UCS (*extreme food intake*) contingencies. Then it follows that breaking the bond between CS and UCS is broken, the exaggerated CRs will extinguish (see also Jansen et al., 1989).

The conditioning model specifies a way in which the overeating response of dieters after a variety of disinhibitors may take place. Although the model *post hoc* covers the range of existing findings from overeating to insulin levels quite well, the predictive validity of the compensatory response model with respect to the overeating response of dieters still has to be proven. Future research should focus on the predictions that follow directly from the model. They are (1) that classical conditioning of food intake (UCS) to exteroceptive and interoceptive stimuli (CS) will, after repeated association, result in an anticipatory hyperinsulinemia and a compensatory hypoglycemia response (CCR); (2) that after a variety of disinhibitors (cognitive manipulation, eating a preload, smelling a preload, and emotional agitation) individuals who overeat will show the anticipatory hyperinsulinemia response and the compensatory decline in blood sugar level; (3) that this hyperinsulinemia/hypoglycemia response will be subjectively experienced as craving; and (4) that inducing a hyperinsulinemia/hypoglycemia response in non-dieters will trigger craving and an overeating response.

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A CASE OF BULIMIA SUCCESSFULLY TREATED BY CUE EXPOSURE

A. JANSEN*, M. A. VAN DEN HOUT*, C. DE LOOF†,
J. ZANDBERGEN† and E. GRIEZ†

Limburg University, Maastricht, The Netherlands

Summary — In the present case-study, a 22-year-old female patient with a 7-year history of bulimia nervosa was treated by cue-exposure. During cue-exposure sessions the relationship between CS (stimuli associated with binge-eating behaviour) and UCS (the sensations related to the intake of binge food) was broken. As predicted, the patients' craving for food declined within as well as between cue exposure sessions and a radical decrease in the frequency of binge-eating persisted for at least nine months. Although during the therapy no explicit attention was paid to the patient's low mood and irrational self-talk, mood was improved and beliefs were less irrational after cue-exposure and at the follow-ups. The data suggest that cue exposure may be an effective part of the treatment of bulimia nervosa.

Bulimia nervosa is characterized by recurrent episodes of uncontrollable binge-eating, in which large amounts of food are consumed rapidly (APA, 1987). Recently, several authors have pointed to the similarities between bulimia and addictive behaviors (Scott, 1983; Dumont & Vamos, 1983; Brisman & Siegel, 1984; Jonas & Gold, 1988; Wardle, 1987; Filstead et al., 1988; Jansen et al., 1988, 1989). Both bulimia and addictions are characterized by craving, preoccupation with and loss of control of intake and by relapse. Also, experimental data supports the addictive interpretation of bulimia: restrained eaters were found to be rapidly habituating sensation seekers, just as addicts are (Jansen et al., 1989). The analogy between addictions and bulimia may have therapeutic implications.

Considerations Regarding Treatment Of Addictive Behaviors Relevant To The Management Of Bulimia

Recent developments with respect to "cue exposure" may be of relevance here. Based on

an impressive data base primarily from animal work, Siegel and co-workers stressed the importance of contextual cues and preparatory responses in the persistence of addictive behaviors (Siegel, 1983; Macrae et al., 1987; Niaura et al., 1988). In short, after repeated drug administration, the set of stimuli (CS) specifically associated with the drug taking behavior (UCS) will elicit physiological responses that are the biological basis of craving. The CS (cues) will elicit craving as long as the CS remains a specific predictor of the UCS (drug). Therefore, according to this paradigm, effective treatment of substance abuse should be directed at decreasing cue reactivity. Extinction of the craving may be achieved by prolonged exposure to the CS (cues) without UCS (drug) reinforcement. This so-called "cue exposure" appears to hold promise for the behavioural treatment of drug addicts (Siegel, 1983) and alcoholics (Rankin et al., 1983).

If bulimics' bingeing is conceptualised as behavior similar to that of addicts, cue exposure may also be effective in the treatment of bulimia. The intake of binge food may be

Requests for reprints should be addressed to A. Jansen, *Dept. of Mental Health Sciences/Experimental Psychopathology, Limburg University, P.O. Box 616, 6200 MD Maastricht, the Netherlands.

†Dept. of Clinical Psychiatry/Experimental Psychopathology, Limburg University, the Netherlands.

considered as the UCS, while the CS consists of the stimuli specifically associated with the binge-eating behavior. In the present case study, a bulimic patient was treated with cue exposure. The aim of the study was to test whether breaking the probabilistic relationship between CS and UCS by cue exposure is effective in diminishing the bulimic's craving for food and the frequency of binge-eating. Attention was also directed at changes in mood and specific thinking patterns.

Method

Subject

The subject was a 22-year-old single female, who met the DSM-III-R criteria for bulimia nervosa (APA, 1987). She had suffered from uncontrollable binge eating episodes for 7 years. Although she was of normal weight, her weight fluctuated extremely, e.g., about 10 kg in four weeks. After bingeing, the patient vomited. Her eating pattern was chaotic; binge eating episodes alternated with strict dieting episodes in which she merely ate "healthy" food, e.g. no sweets and much fruit. A strong preoccupation with weight and appearance was evident, as well as dichotomous all-or-nothing reasoning and depressed mood. The patient agreed to being subjected to an experimental new treatment.

Assessment

Several self-report instruments were employed before and after treatment and at the 2-month and 9-month follow-ups. They included (a) an eating diary from which the *frequency of self-reported binge-eating* was calculated, (b) the *Beck Depression Inventory*, range 0–63, and (c) a *Self-Talk List*, composed by the first author for the present study, and containing 10 statements concerning all-or-nothing reasoning and irrational thoughts about weight, appearance, and eating behaviour. Some statements

were derived from clinical interviews with the patient. Each statement was rated by the patient on a 0 ("I don't agree at all") to 100 mm ("I fully agree") Visual Analogue Scale (see later).

During cue-exposure sessions the patient rated her *craving* every 5 minutes on a 0–100 mm VAS ("How strong is your urge to eat?" from "none at all" to "extreme").

Measurement Periods

The first goal of the treatment was to reduce the frequency of binge-eating episodes and, the second was to enable the patient to keep food without eating it all. To this end, a list of food items was made. The patient was instructed to buy all these food items, and, when running out of the relevant food, to restock the supply directly. In the continuous presence of this food, she monitored her eating behavior for exactly 21 days, which forms the pre-treatment measurement (PRE). The patient also completed the Self-Talk List and the Beck Depression Inventory at the start of this pre-treatment measurement.

The patient continued registering her food intake during and between cue exposure sessions. Promptly after the last cue exposure session, the patient completed the inventories again and registered her food intake for 25 days (POST).

Two and nine months after treatment she monitored her food intake for another 32 days, these being first and second follow-ups (FU1 and FU2). During both the POST and the FU registration periods, the patient kept the same food items as during the pre-treatment registration period. On the last day of the FU1 and FU2 the patient completed both inventories again.

Identifying The CS

During pre-treatment monitoring, 4 sessions of behavior analysis took place. The therapist (the first author) and the patient exactly

identified the contextual cues (CS) in which the patient's binges occurred. Contact with the patient focussed on future *in vivo* exposure treatment, and aspects of such exposure were discussed. Care was taken not to engage in cognitive restructuring. The patient nearly always binged on specific food items (Malaga ice cream, chocolate, cookies, shortbread, liquorice all-sorts, Dutch liquorice, bananas, currant buns, milk, 7-up light, and tea). The binge was really a ritual event; first, the patient put on comfortable clothes (jogging trousers). Then, she arranged all the binge food on a tray, after which she would sit down on her couch, pick up a particular magazine and start to "read". While "reading", she stuffed herself with all the food in about a quarter of an hour. The magazine was considered a specific binge cue. The patient reported that she had forbidden herself to read this kind of magazine except during binges. Buying the magazine heralded a binge-eating episode. The binge-eating ritual depended on the contextual binge cues which constituted its CS. The UCS was defined as the consumption of binge food — food sweet and rich in calories which the patient had forbidden herself to eat. (The UCS is actually a sequence of events — confronting the food, eating it, and the consequent pleasurable sensations.)

Treatment Procedure

After the monitoring period of 21 days, *in vivo* cue exposure was started, and continued for 16 successive days. Breaking the CS-UCS probability relationship was aimed at in two ways. First, the patient was exposed to the contextual cues predicting a binge (CS), while she was prevented from eating (UCS). During the cue exposure sessions, the patient was asked to touch the food, to raise it to her mouth and nose, to smell it and to act as if she was going to eat it. Exposure sessions lasted at least 1 hr *each day* and the patient rated her craving every 5 min. Second, before each exposure session, the patient was forced to eat,

together with the therapist, a preload of binge food (UCS) in a different place not predicting a binge, e.g., to eat an ice cream in an ice-cream parlor. After having eaten binge food in a non-binge environment, the patient went home and confronted herself with the binge food and contextual binge cues without eating anything. In continuation of these strategies for breaking the CS-UCS probability relationship, she confronted binge food all day. She took along large amounts of binge food every day and ate small bites of one of the binge food items, without eating all of it, two or three times a day. Except for the first session, the patient did the exposure sessions on her own.

Results

Craving

Figure 1 shows that the first five cue exposure sessions were very effective as to the extinction of the urge to eat within sessions. Also, the starting level of the patient's craving decreased considerably between the sessions. After the fifth exposure session she reported that she no longer experienced any craving. The therapist and the patient then agreed to make the exposure more difficult by introducing more food items. This is clearly reflected in a raised starting level of craving at the sixth session. However, habituation again took place within and between the sessions.

Frequency of Binge Eating

As can be seen from Figure 2, the frequency of binge-eating decreased substantially after treatment. The patient monitored 16 binges during the pretreatment registration period (averaging over 21 days, 0.76 binges a day), 0 binges during cue-exposure, 1 binge during the post treatment registration period (averaging over 25 days, 0.04 a day), 4 binges during the 2-month follow-up (averaging over 32 days, 0.13 a day) and 0 binges during the 9-month follow-up.

Figure 1. Habituation of craving within and between exposure sessions. The dots in Fig. 2 indicate the average of three ratings of craving in each session. In this way, the dot over A represents the average of the ratings after 5, 10, and 15 minutes, the dot over B after 20, 25, and 30 mins., C: 35, 40, 45 mins., D: 50, 55, 60 mins., E: 65, 70, 75 mins., and F: 80, 85, and 90 mins. The lines connecting the dots indicate the average of the ratings of craving over two or three sessions.

Figure 2. Frequency of binge-eating before, during, and after cue-exposure, and at two- and nine-month follow-ups.

Depression and Self-talk

Before treatment, the patient's mood was low (BDI = 22). Promptly after treatment, the patients' mood had improved (BDI = 6). At the first follow-up, mood was lowered again (FU1; BDI = 15), but at the second follow-up mood had again improved (FU2; BDI = 9).

Surprisingly, the patient thought in a less dichotomous manner and less irrationally after cue exposure without specific cognitive inter-

ventions. To a lesser degree, this improvement continued during the post and follow-up measurements (see Figure 3).

Discussion

This case-study shows that cue exposure may be an effective form of treatment for bulimia nervosa. As predicted, craving declined both within and between cue exposure sessions, and a radical decrease in the frequency of binge-eating persisted for at least nine months. The changes in mood and self-talk after cue exposure therapy are interesting. Although before and during the therapy, no explicit attention was paid to the patient's low mood and dichotomous/irrational self-talk, her mood had perceptibly improved after cue exposure. Also, without any specific cognitive intervention, the patient thought in a less dichotomous manner and less irrationally, as measured by the Self-Talk List. At the follow-up her self-talk seemed to have worsened a bit. Despite the fact that some improvement on the cognitive items was noticeable after cue-exposure, dysfunctional thinking patterns were still present after cue-exposure, and cognitive therapy might have been indicated.

Figure 3. Scores on the Beck Depression Inventory and the Self-talk List before, during, and after cue-exposure and at the 2- and 9-month follow-ups. The irrational belief score represents the mean scores on 10 irrational belief statements ("What others think of me is most important", "I am not pleased with my looks", "Sweet things are the beginning of all misery", "I must not eat sweet things", "If I do not have complete control of myself, everything will go wrong", "I am worthless if I am fat", "It is all or nothing", "If I eat over 1000 cal. a day, everything is lost", "If I weigh less, everything will be better" and "The way I look determines my self-confidence"). The patient rated agreement with the statements on a 0 ("I don't agree at all") to 100 mm ("I fully agree") VAS.

The notion that exposure may be helpful in the treatment of bulimia is not new. By analogy with the rituals of obsessive-compulsive disorders, Wilson and co-workers (1986) argue that the vomiting in bulimia reduces anxiety and underline the importance of exposure and response prevention of vomiting in the treatment of bulimia. However, Schmidt and Marks (1988) noted that that is very aversive and that the control of binges rather than of vomiting may be more acceptable. Therefore, Schmidt and Marks (1988) treated four bulimics with prolonged cue-exposure. Their results were "moderately encouraging" (p. 671); two subjects showed considerable improvement of frequency of bingeing and vomiting, while one dropped out of treatment, and one improved mainly after, but not during, the treatment. However, the most crucial aspect of the cue-exposure rationale is breaking the CS-UCS bond. Therefore, among other things, cue-exposure should take place in the usual binge-environment. Schmidt and Marks (1988) exposed their patients to binge food in the laboratory/clinic and not in the binge-environment (UCS). This may be the difference that explains the more positive results in our case than in the four cases of Schmidt and Marks.

All in all, the data suggest that cue exposure

is an effective treatment of bulimia. However, the limitations of data from a single case are well known.

There should be replication in controlled outcome studies. Currently, stimulus-control techniques often form part of the treatment of bulimia. However, their effects are opposite to those of the CS-UCS model. To learn stimulus-control techniques is, in fact, to learn *avoidance*; i.e. to avoid the stimuli associated with binge-eating. In contrast, exposure to the CS extinguishes the craving. Since food can hardly be avoided entirely, much more relapse is likely after interventions based on avoidance, than after those based on exposure.

If cue-exposure is indeed effective in bulimia nervosa, we need to know how many exposure sessions are needed for the craving to extinguish, how the sessions should be spaced, whether it is necessary for the patient to abstain completely between cue exposure sessions, and how broad the range of cues should be for maximizing the effects.

6 discussion

6.1 general discussion

The starting point of the present dissertation was the observation that certain 'disinhibitors' produce counterregulation in dieters as well as binge eating in bulimics. The question was: "*why?*".

Some answers were given and dismissed. They appeared to be untenable on theoretical and/or empirical grounds. *Set point theory* is repeatedly put forward by researchers of counterregulation. But the introduction of a 'set point' is merely an easy way out when one is completely at a loss. The theory is inapplicable to the lab data on counterregulation. Set point theory requires rather successful dieting for someone's body weight to drop below its alleged 'set point'. Dieters counterregulating in the lab are especially unsuccessful dieters: it is unlikely and, what is more important, still untestable that unsuccessful dieters' weight is below set point. Apart from that, it is useless to take the 'set point' for granted as so many researchers of counterregulation do. With the use of 'set point' as a self-evident explanation, the discussion about the etiology of counterregulation is closed too easily. Such premature, unjustified consensus is counter-productive when it comes to the development of new theories (e.g. that the maintenance of overeating has nothing to do with food restriction and hunger, but everything with impulsive, addictionlike behavior).

Besides the set-point theory, the *5-HT hypothesis* was also put forward. Originally, the 5-HT theory focussed on the clinical binge. Bingeing was thought to serve a self-medical purpose: by eating relatively large amounts of carbohydrates bingers were thought to restore their otherwise low 5-HT levels. To make 5-HT theory convincing, it is a prerequisite that binges are relatively rich in carbohydrates, but research has proved that they are not (cf.5.3).

Moreover, both the set point theory and the 5-HT theory do not explain satisfactorily why 'disinhibited' dieters counterregulate whereas 'non-disinhibited' dieters do not. 'Disinhibited' as well as 'non-disinhibited' dieters should, according to both theories, be characterized by a biological deficit. For explaining the counterintuitive behavior of dieters in the lab, auxiliary hypotheses about the intermediate mechanism which has been put into operation in 'disinhibited' dieters but not in 'non-disinhibited' subjects, will always be needed.

Cognitive theory can explain the mechanism. It makes it clear why 'disinhibited' dieters overeat whereas 'non-disinhibited' dieters do not. The

underlying idea is that the different disinhibitors lead to overeating via certain 'disinhibitive' cognitions which are only present in 'disinhibited' dieters. After dieters perceive their diet as broken, they think thoughts such as "*I've blown my diet, I might as well continue to eat*" and they suit the action to the word. This explanation may sound plausible. However, there is not one single controlled indication that disinhibitors will lead to 'disinhibitive' thoughts (cf. 5.5). Moreover, the theory fails when it comes to explaining why dieters overeat after non-food related disinhibitors, such as mood induction.

In sum, somehow or other existing theories fail when it comes to explaining why dieters counterregulate after various challenges. The main contribution of the present dissertation is the presentation of an alternative explanation which covers the range of challenges quite well. In the course of the present research, similarities between addicts and bulimics caught the attention. The loss of control that is repeatedly reported by dieters (cf. 5.1 & 5.5) and the rapid habituation rates (cf. 5.8) support the alleged addiction analogy. With the use of experimental data on classically conditioned compensatory responses in addictive behaviors, an analogous model for counterregulation/binge eating can be construed. The idea is that binge eating and counterregulation are *cue-dependent*. They are supposed to result from learned compensatory responses (hypoglycemia) which prepare the organism for the intake of food. The compensatory response model, which is outlined in section 3.5, seems to be promising. The model specifies the way in which dieters' counterregulation or binge eating after a variety of disinhibitors may take place. An important difference between the currently especially popular cognitive explanation of counterregulation and the present model lies in the exchange of stimulus and process (see below). The cognitive model states that a 'disinhibitor' (stimulus) leads to counterregulation (response) via disinhibitive cognitions (process in the organism). The compensatory model states that a 'cue' (stimulus) leads to counterregulation (response) via physiological compensatory responses (process in the organism):

Cognitive model

Conditioning model

According to the cognitive model, the process that accounts for counter-regulation is formed by cognitions. It should be noted that, according to the compensatory model, cognitions may act as cues (stimuli), but are not included in the *process* according to which counterregulation takes place. The cognitive 'disinhibitor' is, in fact, a cue, whereas the real 'disinhibitor' is a classically conditioned compensatory response (CCR).

Post hoc, the model covers earlier findings quite well, and some predictions following from it could also be corroborated (cf. 5.8 & 5.9). It was, for example, predicted that if certain CSs are sometimes followed by strong UCSs, unsuccessful dieters will also counterregulate after any of these cues that typically predict food intake. Dieters did counterregulate after they had merely smelled a 'preload'. This is in flat contradiction with cognitive theory, which states that dieters overeat because they perceive their diet as broken, whereas, in this case, it was not.

Future research should further concentrate on this model; mine certainly will. First, attention should be paid to fundamental predictions. Later on, research on clinical issues will possibly also be warranted.

6.2 fundamental research

According to the model, dieters counterregulate because of a classically conditioned anticipative compensatory response. It was argued that in response to a cue, an insulin-induced hypoglycemia has to be undone by eating, that is, the hypoglycemia has to be compensated for by large food intake to restore blood sugar level.

The following hypotheses can be formulated:

- 1 after repeated association of a specific set of cues (CS) with large food intake (UCS), presentation of merely the CS will elicit a compensatory hypoglycemic response (CCR). See figure.

- 2 the decline in blood sugar level (CCR) is mediated by an anticipatory insulin response (CPR).
- 3 the hyperinsulinemia/hypoglycemia is subjectively experienced as craving.
- 4 the classically conditioned compensatory response, i.e. the hypoglycemia, will be positively correlated to the amount of *ad lib* food intake.
- 5 counterregulation of dieters in the lab will be accompanied by an anticipative hyperinsulinemia and a compensatory hypoglycemia response.
- 6 insulin-induced hypoglycemia will trigger craving and an overeating response in unrestrained subjects after a preload.

6.3 clinical issues

Insofar as counterregulation/bingeing actually follows from classical conditioned cue responses, there is a straightforward clinical implication (see also section 3.5). The model states that cue responding is maintained because certain CSs are often reinforced by large intake. Cues elicit CCRs because they are predictors of UCSs (large food intake). Pavlovian learning theory predicts that if the CS-UCS relationship is broken, CCRs will be extinguished and counterregulation/bingeing should be extinguished as well.

Interestingly, the type of therapy that follows from this will be highly similar to the behavioral techniques that rank among the most effective therapies for curing compulsive disorders and phobias: *in vivo* exposure (Emmelkamp, 1982). Compulsive behavior, phobic anxiety and avoidance are typically cue controlled. *In vivo* exposure boils down to confronting the patient with the stimulus for prolonged periods, without avoidance behavior (response prevention). Success percentages are approximately 70% for severe clinical phobias and compulsions, while results in long-term follow-up are very good (Emmelkamp, 1982). The CCR theory likewise states: exposure & response prevention. More specifically: long exposure to cues (smell, sight, touch etc.) and response prevention (i.e. not eating). In this way extinction is expected. The very fact that dozens of clinical trials established *in vivo* exposure as highly effective in the area of anxiety disorders promises well for the management of eating disorders.

In 5.9 a case history was described of a woman who was successfully treated with cue-exposure. The present author is fully aware of the limitations of this case history (cf. 3.5). However, a case study is useful in generating hypotheses. Of course, controlled trials are necessary for determining whether cue-exposure is really as effective as it is supposed to be.

Though CCR theory implies that certain factors are important in maintenance and therapy, we do not have any insight into parameter values.

Such insights may be required before clinical trials will be conducted. More specifically, explicit attention should be paid to the following questions:

- 1 how many exposure sessions are needed for craving to be extinguished completely?
- 2 how should sessions be spaced?
- 3 is it necessary for the subject to abstain completely from binge eating between the cue exposure sessions?
- 4 how broad should the range of cues be?

Finally, it should be noted that the exposure strategy is radically different from the current treatments of eating disorders by *self-control* and *stimulus-control* techniques. These techniques, in fact, mean learning avoidance strategies; the patient learns to avoid stimuli associated with overeating (see e.g. Mizes & Lohr, 1983; Hsu & Holder, 1986; Laessle et al., 1987). In this way craving will never be extinguished. The model that has been presented states that it is only exposure to the cues associated with binge eating and response prevention (no binges) that will lead to the extinction of craving. The empirical implication is that, in the long term, much more relapse is expected after learning self-control techniques than after exposure. Research will tell.

summary

Excessive eating and self-induced vomiting are timeless phenomena. Whereas the old Roman's stuffing and vomiting is considered to be an *aristocratical custom*, in the 17th, 18th, and 19th century the stuffing-vomit habit was registered as a typical medical disease. In chapter 1 it is reported that nowadays there are also individuals who periodically experience recurrent episodes of uncontrolled and excessive eating. When this is attended by persistent overconcern with body shape and weight, the Diagnostic and Statistical manual of *Mental Disorders* speaks of bulimia nervosa. The episodes of excessive eating are followed either by self-induced vomiting, or use of laxatives, or strict dieting or fasting, or vigorous exercise in order to prevent weight gain.

In chapter 2 a laboratory model of bulimia nervosa is discussed. Subjects selected on the criterium of the intention to restrict food intake (i.e. dieters) are considered to be a subclinical variant of bulimia nervosa. Dieters respond in a different way to various experimental manipulations than non-dieters do. Non-dieters regulate their food intake: they eat little after a food preload and they eat a large amount without a preload. Non-dieters are less conventional. They eat little without a preload and a large amount after a preload: they counterregulate. A preload is not the only stimulus which induces excessive eating in dieters. Emotions such as depressed mood or anxiety, the anticipation of a high-calorie meal, and the idea that one ate a high-calorie preload are also stimuli which lead dieters to overeat. *Why do dieters not regulate their food intake?*

Various explanations are offered. Two biological theories (set point theory and the serotonin hypothesis) and a psychological explanation (dysfunctional thoughts) are critically discussed in chapter 3. The three theories cannot satisfactorily explain the typical behaviour of dieters. Auxiliary hypotheses are needed. If the set point theory, the serotonin hypothesis, and the cognitive theory cannot offer a satisfying explanation on theoretical and empirical grounds, how then can it be explained that dieters and bulimic patients experience recurrent episodes of uncontrolled and excessive eating?

Based on the experimental findings reported in the present dissertation and shortly summarized below, a new model was developed to explain counterregulation of dieters. This model is described in section 3.5.

In chapter 5, empirical studies by the present author are presented. An overview of the studies is given in chapter 4.

In summary, the main findings are:

- 1 Clinical binges (of bulimic subjects) differ in severity from non-clinical binges (of non-bulimic subjects). The DSM-III-R criteria discriminate clinical binges quite well from normal eating episodes. Hunger is of no importance in the development of binges (cf. 5.1), which is not easily compatible with the set point explanation of BN.
- 2 Non-regulation of food intake is characteristic for people preoccupied with caloric intake, but independent from being characterized as a successful or an unsuccessful dieter (cf. 5.2). This finding of successful and unsuccessful dieters non-regulating their food intake casts doubt on the validity of the set point explanation.
- 3 Bulimic's binges were not characterized by a higher carbohydrate/protein ratio than their normal eating episodes and eating episodes of a normal control group (cf. 5.3). This is contrary to the 5-HT hypothesis.
- 4 Dieters' counterregulation is mood-dependent: happy dieters regulated their food intake whereas depressed dieters did not. The unusual behaviour of non-dieters (they did not regulate their food intake, neither when happy, nor when depressed) warrants caution in generalizing the findings (cf. 5.4).
- 5 Dieters who did not regulate their food intake did not show more disinhibitive thoughts than other dieters and non-dieters. Also, dieters did not think in a more irrational way than non-dieters (cf. 5.5). This is contrary to the cognitive hypothesis.
- 6 The overeating response of the dieters took place in the lab. After participation in a laboratory experiment disinhibited dieters did not overeat outside the laboratory. Also, pre-experimental macronutrient intake did not differ from post-experimental macronutrient intake (cf. 5.6). This finding suggests that overeating is cue-dependent.
- 7 The galvanic skin response of dieters habituated significantly more rapidly than the galvanic skin response of non-dieters to a series of neutral auditory stimuli. Also, dieters scored significantly higher on the Sensation Seeking Scale (cf. 5.7). Addicts also habituate more rapidly and score higher on the Sensation Seeking Scale than normal subjects. The data support the addiction model of Bulimia Nervosa.
- 8 Dieters 'counterregulated' after intensive smelling of a 'preload' whereas non-dieters did not. Dieters also reported a subjective loss of control over eating after smelling the 'preload' whereas non-dieters actually reported more control (cf. 5.8). The experiment was designed to allow for a crucial test of two theories. The data falsify the cognitive hypothesis. It corroborates the notion that overeating in dieters can be provoked by exposure to cues that bear a probabilistic relation to actual food intake.
- 9 In a $n=1$ study breaking the probabilistic relation between CS and UCS ('cue-exposure') seemed to be effective in the treatment of bulimia nervosa.

The model as presented in section 3.5 is related to modern Pavlovian explanations of addictive behaviour. It is postulated that binges are cue-dependent. Certain stimuli, which have become reliable predictors of excessive food intake, are supposed to elicit an anticipative compensatory response. One could speculate about an anticipative decline in blood sugar. The model explains *post facto* why disinhibitors elicit counterregulation in dieters and do not in non-dieters. The various stimuli are all potential conditioned responses. Of more importance is the model's prediction that olfactory stimulation is enough to elicit counterregulation in dieters. This was indeed the case (see above). In chapter 6, a series of clinical and experimental implications are formulated.

samenvatting

Onmatig eetgedrag en zelfopgewekt braken komt al sinds jaar en dag voor: het is een tijdloos fenomeen. Pleegde men het bunkeren en braken van de oude Romeinen als een *aristocratische gewoonte* te karakteriseren, in de 17^e, 18^e en 19^e eeuw stond het e.e.a. te boek als een typisch *medische kwaal*. In hoofdstuk 1 wordt gerapporteerd dat ook tegenwoordig individuen zich bij tijd en wijle te buiten gaan aan ongecontroleerde eetbuien. Indien dat gepaard gaat met een overmatige bezorgdheid over uiterlijk en gewicht, dan spreekt men, overeenkomstig het Diagnostic and Statistical manual of *Mental Disorders*, van Bulimia Nervosa. Het overmatig eten wordt dan gecompenseerd door bijvoorbeeld te braken, te lijnen en/of excessief te sporten.

Psychopathologische fenomenen kunnen in het laboratorium bestudeerd worden. Dat heeft het voordeel dat condities optimaal gecontroleerd kunnen worden. In hoofdstuk 2 wordt melding gemaakt van een analogon van Bulimia Nervosa zoals dat al vanaf de zeventiger jaren in het gedragslaboratorium wordt onderzocht. Gevonden werd dat lijners anders reageren op verschillende experimentele manipulaties dan niet-lijners. Niet-lijners reguleren steeds hun voedselinname: zij eten weinig nadat zij vooraf iets gegeten hebben (= *preload*) en veel zonder preload. Lijners daarentegen gedragen zich minder konventioneel. Zij eten weinig zonder preload en veel na een preload: lijners *counterreguleren*. Een preload is niet de enige stimulus die overmatig eten in lijners ontlokt. Andere stimuli waarvan regelmatig werd gedocumenteerd dat zij lijners tot overmatig eten aanzetten zijn emoties als somberheid en angst, anticipatie op het eten van een hoogkalorische maaltijd, en de idee een hoogkalorische preload te hebben gegeten. Hamvraag is: *hoe komt het dat lijners hun eetgedrag niet normaal reguleren?*

Verschillende verklaringen zijn geopperd. Twee biologische verklaringen (de set-point theorie en de serotonine hypothese) en een psychologische verklaring (dysfunktionele gedachten) worden in hoofdstuk 3 kritisch besproken. De drie theorieën kunnen het typische gedrag van lijners niet op bevredigende wijze verklaren. Steeds zijn hulphypothesen nodig. Als de set-point theorie, de serotonine hypothese en de kognitieve theorie op grond van theoretische en empirische overwegingen geen bevredigende verklaring bieden, hoe kunnen de periodiek optredende eetbuien van 'lijners' en BN patiënten dan verklaard worden?

N.a.v. de experimentele bevindingen die in dit proefschrift worden gerapporteerd en die hieronder uiterst summier zijn samengevat, werd een

nieuw model ontwikkeld dat de counterregulatie van lijners verklaart. Dit model vindt de lezer beschreven in paragraaf 3.5.

In hoofdstuk 5 worden eigen empirische- en experimentele studies gepresenteerd. Voor een overzicht van de studies raadplege men hoofdstuk 4.

De belangrijkste bevindingen zijn kort samengevat de volgende:

- 1 Klinische eetbuien verschilden in ernst van niet-klinische eetbuien. De DSM III criteria diskrimineerden klinische eetbuien goed van normale eetepisodes. Honger speelde geen rol van betekenis in het ontstaan van eetbuien (cf. 5.1), hetgeen in strijd is met de set-point hypothese.
- 2 Het niet reguleren van de voedselinname bleek kenmerkend voor mensen die gepreokkupeerd zijn met calorische inname, doch onafhankelijk van karakterisering als succesvol of onsuccesvol lijner (cf. 5.2). De bevinding, dat zowel succesvolle als niet succesvolle lijners hun voedselinname niet reguleren plaatst vraagtekens bij de set-point theorie.
- 3 Eetbuien van BN-patiënten werden niet gekenmerkt door een hogere koolhydraat/proteïne ratio dan gewone eetepisodes van BN-patiënten en eetepisodes van een normale controlegroep (cf. 5.3). Dit is in strijd met wat de 5-HT hypothese doet vermoeden.
- 4 Counterregulatie van lijners bleek afhankelijk van stemming: vrolijke lijners reguleerden hun voedselinname in tegenstelling tot sombere lijners. I.v.m. het ongebruikelijke gedrag van niet-lijners (niet-lijners reguleerden hun voedselinname niet, onafhankelijk van stemming) is voorzichtigheid geboden bij het generaliseren van de bevindingen (cf. 5.4).
- 5 Lijners die hun voedselinname niet reguleerden werden niet gekenmerkt door meer disinhibitieve gedachten dan andere proefpersonen. Ook dachten lijners niet irrationeler dan niet-lijners (cf. 5.5). Dit is in strijd met de cognitieve hypothese.
- 6 Het overeten van lijners bleek enkel binnen het laboratorium op te treden: na deelname aan een experiment in het laboratorium overaten experimenteel 'gedisinhibeerde' lijners zich niet nog eens buiten het laboratorium. Ook de inname van macronutriënten verschilde pre- en post-experimenteel niet (cf. 5.6). Het suggereert dat overeten stimulus-specifiek is.
- 7 De huidgeleidingsreactie van lijners habitueerde significant sneller aan een serie neutrale auditieve stimuli dan die van niet-lijners. Ook scoorden lijners significant hoger op de 'Sensation Seeking Scale' dan niet-lijners (cf. 5.7). Daarin stemmen zij overeen met verslaafden. De bevindingen verlenen enige steun aan het verslavingsmodel van BN.

- 8 Lijners 'counterreguleerden' nadat zij intensief aan een 'preload' geroken hadden. Voor niet-lijners geldt dit niet. Ook rapporteerden lijners na het ruiken een subjectieve beleving van controleverlies over eetgedrag terwijl niet-lijners juist een verhoogd gevoel van controle rapporteerden (cf. 5.8). Dit falsifieert de kognitieve hypothese en ondersteunt de theorie dat counterregulatie afhankelijk is van een stimulus (cue).
- 9 Doorbreking van de probabilistische relatie tussen CS en UCS ('cue-exposure') lijkt effectief in de behandeling van BN.

Het model zoals weergegeven in paragraaf 3.5 is verwant aan moderne pavlovianse verklaringen van verslavingsgedrag. Gepostuleerd wordt dat eetbuien stimulus specifiek zijn. De idee is dat bepaalde stimuli, zodra zij betrouwbare voorspellers worden van overmatige voedselinname, op den duur een voorbereidende kompensatoire respons in het organisme zullen ontlokken. Gedacht wordt aan een anticipatieve bloedsuikerdaling. Het model verklaart *post facto* waarom de in de literatuur gerapporteerde stimuli ('disinhibitors') bij lijners wel en bij anderen niet leiden tot counterregulatie. De verschillende stimuli zijn allen potentieel gekonditioneerde stimuli. Belangrijker wellicht is dat het model leidt tot de (juist gebleken) voorspelling dat olfactoire stimulatie een voldoende voorwaarde is voor het provoceren van counterregulatie in lijners. In hoofdstuk 6 werd een serie klinische en experimentele implicaties geformuleerd.

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