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EATING BEHAVIORS

Cognitive-behavioral therapy and nutritional counseling in the treatment of bulimia nervosa and binge eating

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Abstract

The goals of manual-based cognitive-behavioral therapy (CBT) and nutritional counseling for eating disorders are similar, namely, eliminating dysfunctional patterns of eating. Modifying these behaviors requires specific therapeutic expertise in the principles and procedures of behavior change that is not typically part of the training of nutritionists and dieticians or mental health professionals without specific expertise. We discuss ways in which principles of behavior change can be applied to eating disorders by non-CBT experts. Specific nutritional rehabilitation programs have the potential to augment CBT in addressing the array of appetitive abnormalities present in eating disorder patients. The dysfunctional appetitive, hedonic, and metabolic characteristics of patients with bulimia nervosa (BN) and binge eating disorder are reviewed. These abnormalities constitute potential target areas that might be more fully addressed by nutritional interventions designed to restore normal appetitive function. © 2000 Elsevier Science Ltd. All rights reserved.

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Cognitive-behavioral therapy (CBT) is the most intensively investigated and best empirically supported treatment for bulimia nervosa (BN) (American Psychiatric Association, 2000). CBT is quick-acting; produces a clinically significant degree of improvement across all four of the specific features of BN, namely, binge eating, purging, dietary restraint and abnormal attitudes about body shape and weight; reduces associated psychopathology (e.g., depressed mood); and is associated with good maintenance of change at 1-year follow-up (Agras, Walsh, Fairburn, Wilson, & Kraemer, 2000; Fairburn et al., 1995). CBT is significantly more effective than either pharmacological or alternative psychological treat-

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ments with which it has been compared (Wilfley & Cohen, 1997; Wilson & Fairburn, 1998). As such, it is the first-line treatment of choice for BN (Walsh et al., 1997).

CBT is based on a cognitive model of what maintains BN (Fairburn, 1997). Social pressures on women to be thin results in overvaluation of body weight and shape. This leads them to restrict their food intake in rigid and unrealistic ways, a process that leaves them physiologically and psychologically susceptible to periodic loss of control over eating, namely binge eating. Purging and other extreme forms of weight control are the person's attempt to compensate for the effects of binge eating. Purging helps maintain binge eating by reducing the individual's anxiety about potential weight gain and disrupting learned satiety that regulates food intake. In turn, binge eating and purging cause distress and lower self-esteem, thereby reciprocally fostering the conditions that will inevitably lead to more dietary restraint and binge eating. Treatment is directed at reducing dietary restraint in favor of more normal eating patterns, developing cognitive and behavioral skills for coping with high risk situations that trigger binge eating and purging, and modifying dysfunctional thoughts and feelings about the personal significance of body weight and shape (Fairburn, Marcus, & Wilson, 1993).

Manual-based CBT has also been shown to be effective in treating BED, although, unlike the case of BN, it is not superior to alternative treatments such as interpersonal psychotherapy (IPT) or traditional behavioral weight loss control programs (Marcus, Wing, & Fairburn, 1995; Wilfley, 1999).

1. CBT and dietary restriction

Manual-based CBT for BN is designed to overcome three forms of dysfunctional dieting: skipping meals, avoiding entire classes of foods thought to be fattening (''forbidden foods''), and limiting overall number of calories consumed. The course of treatment is deliberately sequenced. Developing a regular, more normal pattern of eating (three meals plus two planned snacks per day) is the first dietary target. In this stage, the focus is on when patients eat, not on what they eat. Modification of the two other forms of dysfunctional dieting follow later in the treatment.

The introduction of forbidden foods into patients' meal patterns is designed to empower them — to prove that eating normally will not result in an automatic loss of control. The change is made in a planned and deliberate manner. The goal is not to incorporate high fat or unhealthy foods into the daily diet. In BN, a disorder characterized by excessive dietary restriction, treatment is also designed to increase the overall amount of food consumed. This is obviously also the case in AN.

The strategies used to make these major changes in eating behavior and attitudes include self-monitoring, education, the use of alternative behavior to binge eating, self-control strategies, the exposure principle, problem-solving, cognitive restructuring, and relapse prevention (see Fairburn et al., 1993 & Wilson, Fairburn, & Agras, 1997 for details). Controlled studies document that CBT has been more effective than alternative treatments in reducing dietary restraint (Agras et al., 2000; Wilson & Fairburn, 1998). This is not surprising, given that specific CBT strategies are used to effect such changes. We also know

that the success of CBT in reducing binge eating and purging is partly mediated by reduction in dietary restraint (Wilson, 1999).

Patients with BED differ from BN in showing significantly less dietary restraint (Howard & Porzelius, 1999; Wilfley, Schwartz, Spurrell, & Fairburn, in press). BED is characterized, however, by disorganized eating patterns and overeating even between binge episodes (Castonguay, Eldredge, & Agras, 1995). CBT is designed to overcome chaotic patterns of overconsumption by developing flexible but moderate food intake within a structured framework (Fairburn et al., 1993).

The focus on directly modifying eating behavior and the associated attitudes and affect is one of the distinguishing features of CBT for eating disorders. It sets CBT apart from other forms of psychotherapy. For example, in traditional insight-oriented and psychodynamic psychotherapies, therapists relegate modification of eating behavior to nutritionists while focusing their efforts on putative underlying causes of the eating disorder. A distinguishing feature of this approach is that nutrition intervention sessions are the arena for discussions about food and its content, while psychotherapy is devoted to psychological issues (Reiff & Reiff, 1992; Rock & Curran-Celentano, 1996; Willard, Anding, & Winstead, 1983).

The American Dietetic Association (1988) has recommended a team approach combining the skills of a medical, psychiatric, dental, psychological, and nutritional specialist. The ''team'' or ''multidisciplinary'' approach to treating eating disorders is widespread in the U.S. The recent *Practice Guideline for the Treatment of Eating Disorders* from the American Psychiatric Association (2000) is consistent with this general model.

2. CBT vs. nutritional counseling

The stated goals of CBT and nutritional counseling programs are often similar, if not identical. For example, in its section on ''Nutritional Rehabilitation and Counseling,'' the *Practice Guideline for the Treatment of Eating Disorders* from the American Psychiatric Association (2000) emphasizes the importance of changing behavior such as consuming too few calories, skipping meals, and eating only rigidly restricted types of food in BN and AN. These are precisely the three forms of dysfunctional dieting that manual-based CBT is designed to change (Fairburn et al., 1993). The challenge is how to do it. The question is whether it is useful to recommend that these essential treatment goals be addressed by employing ''nutritional rehabilitation/counseling.''

Overcoming these forms of dysfunctional dieting can sometimes be straightforward, but more often than not, it is a challenging task — one that involves modification of patterns of eating behavior, but also the alteration of key cognitive and affective processes governing dieting (Fairburn, 1997). Modifying these behaviors inevitably demands addressing patients' fear and conflict about weight gain, negative self-evaluation, overconcern with body shape and weight, inability to tolerate negative affect, and ambivalence about change. More than simple education about sound nutrition is required. Besides, both AN and BN patients are already more knowledgeable about the caloric and macronutrient content of common foods than normal young women (Laessle et al., 1988; Sunday, Einhorn, & Halmi, 1992). Much of their frustration derives from ''knowing'' when and what they should eat. But this knowledge

alone is no match for the raw emotion of their disorder — guilt over eating forbidden foods, fear of gaining weight, intense dissatisfaction with body shape and weight — that motivates their dysfunctional behavior.[1]

Achieving behavior change of the sort described above demands expertise in psychological treatment such as CBT. This requires formal professional training and specific therapeutic expertise in the principles and procedures of behavior change (Wilson, 1998). It would be misleading to believe that nutritionists and dieticians can routinely provide such therapy unless they have had specialized training in CBT, which is rarely the case. Indeed, the same can be said of mental health professionals more generally. In similar fashion, it is misleading to include the task of helping patients "deal with their concerns about weight gain and body image changes" in a section on "nutritional rehabilitation" (American Psychiatric Association, 2000, p. 4). Modifying dysfunctional concerns about body shape and weight is arguably the most challenging task in treating AN and BN patients, and requires the most sophisticated cognitive-behavioral interventions (Rosen, 1997; Vitousek, Watson, & Wilson, 1998). The Guideline also states that "nutritional rehabilitation programs" should also "provide ongoing support to patients and their families" (p. 2). Yet coping with family conflicts over a daughter with an eating disorder requires specialized therapy skills that are not normally part of nutrition training programs.

Some proponents of nutritional counseling would seem to concur with this analysis. Beumont, Beumont, Touyz, and Williams (1997, p. 181) pithily point out that the "main obstacles to a return to normal eating are behavioral and psychological. It is not so much the management of the patient's diet that is difficult, but rather the management of the patient taking the diet." Yet these same investigators recommend that dieticians should be responsible for this aspect of treatment. They advance two reasons: first, unlike psychiatrists and psychologists, dieticians have expert knowledge about dietary matters; and second, they are less threatening to patients. But Beumont et al. appear to contradict themselves. They emphasize that effective treatment requires little special dietary knowledge, and prudently recommend against the use of modified ("safe") foods. Whether dieticians are perceived as less threatening is unknown. Suffice it to say that whether any health care provider is threatening or not depends on the skill of the therapist.

2.1. CBT for non-specialists

There are ways in which cognitive-behavioral principles of behavior change can be applied to eating disorders by non-CBT experts, including dieticians and nutritionists (Wilson, Vitousek, & Loeb, 2000). One is the use of self-help strategies. Preliminary studies have suggested that at least a subset of BN and BED patients can be effectively treated using brief and cost-effective intervention such as guided self-help (Fairburn & Carter, 1997). This approach is illustrated in a well-controlled study of the treatment of women with BED (Carter & Fairburn, 1998). Participants received 12 weeks of either guided use of the Fairburn (1995)

[1] We agree with Beumont et al. (1997), however, that some patients may have "highly selective" and possibly inaccurate information that may need correcting.

manual, pure self-help with the book, or no treatment (a wait-list control condition). A primary aim of this study was to evaluate self-help as it would be used in primary care settings or in the general community. Thus, the guided self-help treatment was conducted by non-specialist therapists (labeled ''facilitators'') with no formal clinical qualifications. These facilitators had worked in primary care and received only a limited amount of training and supervision on how to conduct the treatment. As would happen in normal clinical practice, the facilitators were not required to adhere rigidly to the treatment manual. Wait-list control patients were randomized at 12 weeks to one of the two treatment conditions, and were included in the longer-term comparisons of the two self-help groups. The two active interventions produced significant improvements in binge eating that were maintained at the 6-month follow-up. Both treatment conditions were superior to the control group, but similar to one another in reducing binge eating frequency and general psychopathology over the 12 weeks. Binge eating results for the full sample across the 9 months of the study, however, favored guided self-help.

A second approach that allows the use of non-specialists given appropriate training is psychoeducation. The goal of psychoeducation is the normalization of eating patterns and body shape/weight concern through didactic instruction; the content comprises education and cognitive-behavioral change strategies. For example, Davis, Olmsted, and Rockert (1990) developed a psychoeducation program comprising five 90-min group sessions that produces significantly greater improvement than a wait-list control. Compared with a longer course of 19 sessions of individual CBT, the program was less effective overall. But for the subset of patients with less severe specific eating disorder symptoms and associated psychopathology, the group psychoeducational program was comparably effective (Olmsted et al., 1991).

A third approach involves simplifying manual-based CBT. Illustrating such an approach, Waller et al. (1996) devised an abbreviated version of manual-based CBT, consisting of eight 20-min sessions, that could be administered in primary care settings non-specialist therapists. Components of CBT that require more specialist training (e.g., cognitive restructuring) were omitted. This scaled-down treatment proved effective with some patients. Non-responders, who in Waller et al.'s example suffered from other psychiatric problems, could be referred for more intensive treatment delivered by specialists.

2.2. A rose by any other name

Some treatments labeled as nutritional counseling or management seem virtually identical to elements of manual-based CBT. For example, Laessle et al. (1991) compared what they called a nutritional management (NM) treatment with stress management. The former closely approximated the behavioral components (Stage 1) of manual-based CBT (Fairburn et al., 1993). The senior therapists who administered NM in the Laessle et al. study were three doctoral level clinical psychologists and one dietician. Arguably, this is not a typical nutritional intervention. The results showed marginally significant differences in favor of NM both at post-treatment and 1-year follow-up. There is evidence that full manual-based CBT is superior to behavioral versions of the treatment that omit cognitive restructuring and the focus on modifying attitudes towards body shape and weight (Fairburn et al., 1993).

3. Distinctive features of nutritional interventions

Nutritional interventions make a potentially distinctive contribution to treatment that goes beyond what is done in manual-based CBT when the primary focus is on the micro- and macronutrient content of recommended diets. Moreover, the principal providers of these interventions will feature dieticians and nutritionists because of their specialized knowledge, expertise that the typical mental health provider of CBT lacks.

3.1. Beyond CBT

Various nutritional counseling programs for BN have differed from manual-based CBT even though they overlap in many respects, such as prescribing a regular pattern of flexible eating. For example, in an early description of nutritional counseling, Willard et al. (1983) suggested that patients follow a diet based on the American Dietetic Association exchange list. Patients are discouraged from weighing themselves daily, and weekly weighing is considered unnecessary for BN patients. In CBT, an important component of treatment is that patients weigh themselves weekly. Willard et al. also recommend a specific macronutrient distribution (45% to 50% carbohydrates, 25% protein, and 25% fats) that is thought to reduce anxiety and the temptation to binge, and patients are instructed to supplement their diet with a multivitamin. Finally, food models are used to illustrate suggested meal and snack contents. Story's (1986) description of NM of BN more closely corresponds to the components of CBT, with self-monitoring, planned and structured meals and snacks, and introduction of forbidden foods as its central strategies. Here the two areas of departure from CBT are the use of exchange lists for meal planning, and the instruction to patients not to weigh themselves. In Story's program, however, patients are weighed weekly by their nutritional counselor.

Hsu, Holben, and West (1992, p. 57) described a nutritional protocol focusing on establishing regular eating and nutritional education. Part of this education included a substantial reading list on nutritional information. This program also made use of a food exchange method and teaching patients how to use a scale to weigh food portions so that she can better monitor her intake. Other nutritional programs have added the use exchange lists and instruction on cooking and meal preparation to the core behavioral components of CBT (O'Connor, Touyz, & Beumont, 1988). Rock and Curran-Celentano (1996) have also suggested pre-portioning and labeling previously avoided foods with their caloric and nutrient content when these foods are being reintroduced back into the diet. Weighing food portions and counting calories or fat grams is proscribed in CBT for BN, as we detail below.

Although inconsistent with CBT for BN, the programs described by Hsu et al. (1992) and Rock and Curran-Celentano (1996) seem well-suited to helping overweight or obese patients control their weight, and hence, to the treatment of BED. Indeed, the use of exchange lists and calorie counting are standard elements of comprehensive dietary/behavioral treatment programs that have been shown to be effective, at least in the short-term, in the treatment of obese patients with BED (Marcus et al., 1995; Wilson & Fairburn, 1998).

In an elaboration of nutritional counseling and CBT, Ventura and Bauer (1999) described a program of psychobiological nutritional rehabilitation (PNR) whose goal was to resynchronize the appetite system, previously undermined by dieting. The PNR program involved

teaching patients about the psychobiological mechanisms controlling appetite and encouraging them to recognize feelings of hunger and satiety and to experiment with different foods. This rehabilitation program accompanied standard CBT, administered simultaneously by different therapists. A control group consisted of patients treated with CBT plus a traditional nutritional rehabilitation (TNR) program that did not provide psychoeducation on appetite, hunger, or satiety. The PNR program was reported to lead to significantly lower rates of binge eating and purging at treatment termination and at a 9- and 12-month follow-up. Long-term lipid intake also increased significantly more in the PNR group. This treatment approach differed from traditional nutritional counseling programs in that it attempted to take into account and address the dysfunctional expression of appetite in patients with BN. Ventura and Bauer (1999) made clear that patients received psychoeducation on some of the biological factors controlling appetite. Patients were encouraged to try out "new ways of eating" in relation to these factors, to decide on meal plans based on foods patients found personally satiating, and to try to recognize hunger signals. However, these are the only described aspects of the PNR program that distinguish it from TNR, and it was not made clear how patients were expected to eat in relation to the psychoeducational principles presented or how they were to learn to recognize hunger or satiety. It is possible that behavioral or cognitive interventions were used other than those described.

3.1.1. The addiction model

Proponents of the addiction model of eating disorders at times prescribe extreme dietary measures such as eliminating all refined carbohydrates. For example, a popular treatment program is the organization Overeaters Anonymous (OA), which uses exactly the same 12-step program of recovery as is used in Alcoholics Anonymous (AA), substituting the words "food" and "compulsive overeating" for AA's "alcohol" and "alcoholism" (Anonymous, 1984). Abstinence is the goal of this program (Weiner, 1998), and this often involves the complete elimination of certain foods from the diet such as refined sugar or flour. This strategy is based in part on OA's belief that "compulsive overeaters" are completely powerless over food (Anonymous, 1981).

There are theoretical and practical problems with this approach. BN does not fit the defining clinical features of the addiction-as-disease model: tolerance, physical dependence, withdrawal, loss of control, and craving (Wilson, 1991). Furthermore, while the treatment of addiction to psychoactive substances is based upon abstinence, successful treatment of BN calls for the opposite approach (Wilson, 1993). Treatment methods based on the gradual reintroduction of forbidden (binge-triggering) foods have demonstrated effectiveness, whereas there is no evidence for the effectiveness of abstinence-based treatment. On the contrary, symptomatic BN patients already display excessive and severe restriction of food intake, a pattern that leads patients to episodes of compensatory binge eating.

4. Appetitive abnormalities

The nutritional programs summarized above were mainly derived from interventions originally designed to produce weight loss in overweight and obese individuals. An

alternative approach would be to identify specific appetitive abnormalities in the respective eating disorders. Ideally, treatments could then be tailored to remedying these abnormalities in a conceptually coherent and empirically based manner.

We first summarize what is currently known about appetitive abnormalities in BN and BED.

4.1. Hunger and satiety

Disturbed eating behavior is the primary diagnostic criterion for BN and BED in the DSM-IV (American Psychiatric Association, 1994). Laboratory studies confirm that BN patients consume almost three times as much food as control subjects during binge episodes (Walsh, 1993) and over a 24-h period (Weltzin, Hsu, Pollice, & Kaye, 1991), and that they eat faster and for longer amounts of time. Patients may also consume less food than controls during non-binge episodes (Walsh, 1993).

Patient's self-reports of hunger and fullness are also affected. Food intake may fail to reduce hunger or increase satiety over the course of meals in BN patients. Over the course of an experimental liquid meal, bulimic patients had little change in their satiety scores (Halmi, Sunday, Puglisi, & Marchi, 1989). Geracioti and Liddle (1989) have found lower self-reported satiety as compared with controls at both 5 and 45 min after eating. BN patients also fail to exhibit sensory-specific satiety, the normal decrease in liking for a particular food after it has been eaten (Hetherington & Rolls, 1989).

The amount of food ingested by BN patients appears to have little correlation with their subsequent reports of satiety. Large test meals consumed by patients have been found to produce satiety levels similar to those produced by small test meals consumed by control subjects (Geliebter et al., 1992). Regardless of whether a soup that patients had consumed was high or low in calories, the reports of fullness of BN patients at a subsequent meal did not vary. When asked to binge eat, patients consumed nearly twice as much food as controls, and yet subsequently reported similar hunger and fullness levels (Hadigan, Walsh, Devlin, LaChausse, & Kissileff, 1992). Walsh, Kissileff, Cassidy, and Dantzic (1989) also found that even though the food intake of patients was larger than that of controls, patients reported to feel hungrier than controls after the end of a meal or a binge.

Recent research has also examined satiety disturbances in BED patients. Relative to days when they did not binge eat, BED patients consumed significantly more calories and engaged in more feeding bouts on binge days. They also ate at a substantially faster rate during binge episodes than during non-binge episodes (Rossiter, Agras, Telch, & Bruce, 1992). Yanovski et al. (1992) found that during binge episodes, energy intake in BED patients exceeded both their own intake during non-binge episodes and the intake of weight matched controls instructed to binge eat. Patients also consumed more than controls during normal meals.

Disturbances in satiety manifest themselves in patients' self-reports, failure to stop eating, and inability to base their feelings of fullness on the amount of food consumed. It is difficult to disentangle whether disruptions in satiety are a cause or consequence of binge eating or purging. Satiety disturbances and the other appetitive abnormalities present in eating disorders may be a function of disturbed eating behavior and purging. For example, caloric restriction might trigger psychological or physiological processes that disrupt and distort satiety and thus promote binge eating (Walsh & Devlin, 1998). Binge eating may further alter

physiological and/or cognitive perceptions of hunger and satiety and lead to confusion of the perceptions of hunger and satiety (Halmi & Sunday, 1991). Once established, deficits in satiety and other appetitive disturbances might help to maintain problematic eating patterns (Walsh & Devlin, 1998).

4.2. Dietary selection

Macronutrients differ in their satiating power, their capacity to reduce later food intake. Protein has been shown to be more satiating than carbohydrates over the short-term (Booth, Chase, & Campbell, 1970) and long-term (Johnstone, Harbron, & Stubbs, 1996), and carbohydrates in turn are more satiating than fat (Blundell, Burley, Cotton, & Lawton, 1993). Therefore, although the most outstanding difference between the intake of patients and controls is in the overall amount of food consumed (Walsh et al., 1989), it may also be useful to examine the pattern of dietary and macronutrient selection of BN patients.

"Binge foods" often consist of dessert and snack foods (Rosen, Leitenberg, Fisher, & Khazam, 1986), high in fat and low in protein. Weltzin et al. (1991) have found that while controls showed a significant positive correlation between meal size and the percentage of protein in the meal, bulimic patients showed no increase in the percentage of protein as the size of the meal increased.

Patients have been found to begin binge episodes by consuming dessert and snack foods and delaying the intake of fish and meat, whereas controls began with meat consumption (Hadigan, Kissileff, & Walsh, 1989). Hadigan et al. concluded that the early consumption of high-protein foods by control subjects may lead to the development of satiety later on in the course of the meal. During binge episodes, the proportion of protein consumed by patients is slightly but significantly smaller than that of control subjects, while proportions of carbohydrate and fat are similar (Walsh, Hadigan, Kissileff, & LaChausse, 1992; Walsh et al., 1989). Compared with the non-binge episodes of BN patients, patients' binge episodes contain a significantly lower proportion of calories from protein during binge meals and a higher proportion of calories from fat (Van der Ster Wallin, Noring, & Holmgren, 1994; Walsh et al., 1992). Walsh et al. proposed that if the problem were purely one of a disturbance in caloric regulation, one would not expect such differences in macronutrient composition. They speculate that because protein is satiating, the increase in the proportion of calories from protein during non-binge episodes may help patients in limiting their intake during these episodes. Further experimental research is needed, however, to test the assumption that patients with BN are responsive to the satiating properties of protein (Walsh et al., 1992).

Though a large proportion of total dietary intake comes from binges, it is difficult to draw conclusions about nourishment from and the impact of food intake during binges since up to 50% of intake from purged meals and binges is not absorbed due to self-induced vomiting (Kaye, Weltzin, Hsu, McConaha, & Bolton, 1993).

In the overall diet, the proportion of energy intake from protein intake has been found to be somewhat lower in BN patients than in controls (Hetherington, Altemus, Nelson, Bemat, & Gold, 1994). As meal size and 24-h caloric intake increase in BN patients, the percentage of consumed fat (the least satiating of the macronutrients, Blundell et al., 1993) increases

(Weltzin et al., 1991). In non-bulimic dieters, however, lower dietary fat intake and energy intake was associated with a greater degree of eating pathology (Rock, Demitrack, & Drewnowski, 1996). A high rate of overlap has been reported between eating disorders and vegetarianism in female runners (Gadpaille, Sanborn, & Wagner, 1987). Adolescent females who abstained from eating meat were found to engage in self-induced vomiting and laxative abuse several times as often as those who ate meat (Neumark-Sztainer, Story, Resnick, & Blum, 1997).

Similar findings for BED patients have emerged. Yanovski et al. (1992) found that BED patients consumed a greater percentage of energy from fat and a lower percentage from protein than weight-matched controls when instructed to binge eat, as well as more dessert and snack foods. BED patients have also been found to consume a higher percentage of calories from protein on non-binge days than during binge episodes, which were typically high in carbohydrates (Rossiter et al., 1992).

It is possible that the intake of a higher proportion of protein by patients during non-binge episodes (Walsh et al., 1992) and by control subjects at the beginning of meals (e.g., Hadigan et al., 1989) and across daily intake (Hetherington et al., 1994) may be a protective factor preventing overconsumption. Patients may not ingest sufficient amounts of protein, or they may need amounts greater than those needed by normal subjects, for this protective effect to occur. High levels of fat consumed during binge episodes may also promote passive overconsumption (e.g., Blundell & MacDiarmid, 1997).

4.2.1. Carbohydrate craving

A popular theory of proponents of the addiction model of treatment is that BN patients selectively crave carbohydrate-rich foods and that these foods trigger a biological reaction that causes patients to binge eat. There is no evidence for higher-than-normal craving of carbohydrates or intake of carbohydrate-rich foods. Although patients have been found to consume slightly higher levels of fat and lower levels of protein than control subjects, proportions of carbohydrate have been consistently found to be similar for patients and non-patients during binge episodes (Walsh et al., 1992, 1989) and throughout the day (Hetherington et al., 1994).

4.3. Hedonic differences

Eating disturbances in bulimic patients may be associated with disturbances in taste and taste receptors. Patients differ from control subjects in their greater sensory preferences for sweetness, and prefer the sweetest stimulus offered (Drewnowski, Halmi, Pierce, Gibbs, & Smith, 1987). A reduction in palatability of a food is normally reported following intake of that food (“negative allisthesia”). This effect is involved in the satiety process — an individual will be less likely to continue eating a substance if its taste becomes less pleasant (Rodin, Bartoshuk, Peterson, & Schank, 1990). Patients with BN fail to exhibit the normal decrease in liking for a repeatedly presented sweet taste stimulus (Rodin et al., 1990). The basis for this finding may be physiological: unlike control subjects, BN patients fail to demonstrate salivary habituation after repeated presentations of a palatable food (Wisniewski, Epstein, Marcus, & Kaye, 1997). Wisniewski et al. (1997) also found that patients’ initial

salivary response to the food stimulus was blunted compared with controls. These findings could result from bingeing and purging behaviors, which alter salivary gland physiology, and could also serve to perpetuate these behaviors.

In addition, BN subjects have reported, to a significantly larger degree than control or anorexic subjects, that they find liquid experimental meals more palatable (Halmi et al., 1989). Food choices during binges often consist of soft, milky, fluid foods that may connote high caloric density (Drewnowski, 1989). Drewnowski et al. (1987) have suggested that identifying factors responsible for sensory preferences and aversions should influence the nutritional treatment of eating disorders.

For all four taste stimuli (sweet, salty, sour, and bitter), intensities on the palate have been found to be lower in bulimic subjects than in control subjects (Rodin et al., 1990). This reduced taste sensitivity affected only the palate and not the whole mouth, suggesting that the disturbance was a result of symptoms of BN such as purging. Specifically, taste receptors located on the palate may become damaged because vomit is directed toward the roof of the mouth during purging (Drewnowski, 1989; Rodin et al., 1990).

The hedonic properties of binge eating may differ between BN and BED patients. Both BN subjects and BED subjects have reported that after binge eating episodes, they experience feeling physically ill or uncomfortable, and depressed. However, BED patients are more likely to report that they have enjoyed the food, its taste, smell, and texture while binge eating (Mitchell et al., 1999). Mitchell et al. suggested that these findings may have implications for the treatment of BED, where the more positive valence associated with binge eating might make it harder for patients to focus on this as a problem behavior. More research is needed among both BED patients and BN patients to determine whether the hedonic characteristics reviewed here are a cause or consequence of the disorders and whether the characteristics persist after recovery.

4.4. Metabolic abnormalities

A blunted release of the satiety agent CCK after meals, especially larger meals, has been found in patients with BN (Devlin et al., 1997; Geracioti & Liddle, 1989). Fasting levels of CCK present in the central nervous system are also significantly lower in BN patients than in controls (Lydiard et al., 1993). This may represent a satiety deficit that could either be a result or cause of binge eating. This deficit may be reversible, as treatment with tricyclic antidepressants, which reduced binge eating frequency, increased patients' CCK responses to levels similar to those of control subjects (Geracioti & Liddle, 1989).

BN patients may have a lower resting metabolic rate (Devlin et al., 1990) and reduced energy requirements for weight maintenance compared with normal controls (Gwirtsman et al., 1989). These differences, in addition to a decrease in fasting glucose levels compared with controls, may suggest that these patients show physiological and metabolic evidence of semi-starvation (Devlin et al., 1990). Altemus and Gold (1992) have suggested that binge eating and vomiting may keep patients' resting metabolic rate from falling lower as it does during enforced abstinence from these behaviors; binge eating and vomiting may therefore be reinforcing for a person who has a low metabolic rate or is struggling to remain below an undesirable body weight set point.

Other metabolic disturbances in BN may also affect eating and the expression of satiety. An increased insulin release after binge eating and vomiting (Johnson, Jarrell, Chupurdia, & Williamson, 1994) and after smaller meals may increase appetite, thus causing the urge to eat to be perceived as uncontrollable (Russell, Storlien, & Beumont, 1987). Elevated insulin secretion has been found in BN patients after a test meal (Schweiger et al., 1987), after binge eating and vomiting as compared with control women ingesting a large meal (Kaye, Gwirtsman, & George, 1989), and after binge eating and vomiting as compared with after normal eating (Russell et al., 1987). Russell et al. suggested that these findings may indicate reduced glucose tolerance and possible insulin resistance. Altemus and Gold (1992) hypothesized that exaggerated insulin release, which leads to sympathetic nervous system activation, may be the mechanism for the elevation of resting metabolic rate during binge eating and vomiting. Other researchers have not found exaggerated insulin responses to glucose tolerance tests (Hohlstein, Gwirtsman, Whalen, & Enns, 1986) or meals (Johnson et al., 1994). Johnson et al. found that self-induced vomiting leads to hypoglycemia. They suggested that this hypoglycemia may increase appetite and contribute to repeated cycles of binge eating and purging.

Finally, gastric capacity may be disturbed in normal-weight bulimic patients. These patients exhibited a larger-than-normal gastric capacity, even though they did not differ from control subjects in body weight, body fat, or muscle (Geliebter et al., 1992). Distention of the stomach is important in inducing feelings of satiety, and large stomach capacity (which may be increased by repeated binge eating) may help perpetuate binge eating. Slower gastric emptying rates in bulimic subjects than in normal subjects were also observed. This finding may result from patients' larger gastric capacity and may be associated with their reduced release of CCK (Geliebter et al., 1992).

On the whole, patients with BED have exhibited few biologic or metabolic correlates of binge eating. Yanovski (1995), in a review of the biological correlates of binge eating in individuals who do not purge, reported that neuroendocrine function and metabolic rate are similar to those of weight-matched controls. Several of the abnormalities discussed above may therefore be unique to BN patients and may stem from imbalances in electrolytes, hormones, or nutrient intake (Yanovski, 1995). However, the metabolic responses of BED patients may be similar to those of obese individuals, considering the high prevalence of obesity in BED samples. In one study, decreased CCK levels were found in a sample of obese patients. These levels were restored to normal after an 8-week period on a low-calorie, protein-rich diet directed at long-term satiety (Raptis, Zoupas, Rosenthal, Karaiskos, & Dimitriadis, 1980).

5. Implications for treatment

5.1. Possible advantages of an increased focus on nutrition

Whether the appetitive abnormalities in BN patients are the cause or consequence of the eating disorder remains to be shown (Walsh & Devlin, 1998). Kaye et al. (1998) performed one of the only studies identifying neurotransmitter disturbances that persist after recovery

from BN, suggesting that disturbances existed prior to the disorder. An important line of future research will be to examine the persistence of other physiological abnormalities in recovered patients.

5.1.1. Hunger and satiety

Several implications for the treatment of BN follow from the research on appetitive abnormalities. For the most part, however, these implications may already be addressed by current manual-based CBT for BN. For example, establishing normal eating and eliminating the three types of dieting (limiting overall calorie intake, leaving excessive time between bouts of eating, and restricting whole categories of food; Fairburn et al., 1993) is designed to increase satiety and restore normal hunger and fullness. Patients are made aware that their hunger and satiety signals are disturbed due to their eating disorder, and that they are to follow a prescribed pattern of eating regardless of hunger levels, until these signals are reestablished. This pattern of eating, along with stimulus control measures, is also designed to prevent the intake of excessive amounts of food accompanied by a loss of control.

The hedonic idiosyncrasies of BN patients may be addressed through the reintroduction of forbidden foods, where patients are encouraged to stop depriving themselves of their favorite food choices and to plan to eat them in controlled portions under controlled circumstances. These strategies may prevent patients from overeating on a specific type of food due to a failure to develop sensory-specific satiety (e.g., Hetherington & Rolls, 1989).

It is also possible that CBT targets some of the metabolic disturbances present in BN patients. A more consistent pattern of food intake in the form of frequent, small meals may help to regulate the metabolic rate of patients and eliminate their physiological and metabolic signs of semi-starvation (e.g., Devlin et al., 1990). The elimination of self-induced vomiting may prevent hypoglycemia and a resulting increase in appetite (e.g., Johnson et al., 1994).

Future research should explore the value of combining current CBT with innovative nutritional strategies. One additional approach could be to expand the boundaries of the psychoeducation provided to patients to cover the appetitive and physiological abnormalities discussed here. Ventura and Bauer (1999) used psychoeducation as a primary strategy in their nutritional rehabilitation program. Although they reported increased success after using this program, future research needs to evaluate whether psychoeducation alone may account for these results or whether specific behavioral interventions were responsible for "resynchroniz[ing] the appetite system" (p. 57) of the patients they evaluated.

Another implication for treatment that follows from the research on appetitive abnormalities is the possibility of increasing the level of satiety produced by meals and snacks. Rock and Curran-Celentano (1996) have emphasized the importance of adequate fat and fiber intake, with the goal of promoting satiety (fiber is also recommended to improve gastrointestinal function). The recommendation of fiber intake to increase satiety is consistent with reports that fiber content has been found to correlate positively with indices of satiety (Holt, Brand-Miller, Petocz, & Farmakalidis, 1995). The recommendation to increase fat intake to increase satiety, however, is inconsistent with extensive evidence that fat is less satiating than other macronutrients and promotes overconsumption (Blundell & MacDiarmid, 1997). Rather, studies of macronutrient selection in control and bulimic subjects during binge and non-binge eating suggest that in order to increase satiety from meals and snacks, individuals

should decrease their level of fat and increase their level of protein. Further research is needed to experimentally validate this hypothesis.

Increasing protein and decreasing fat, and other strategies to increase satiety, may be useful with obese BED patients as well. Behavioral weight loss manuals have often emphasized increasing the intake of certain carbohydrates in the diet (Brownell, 1994), but it may also be worth pointing out to patients that increasing protein intake can increase satiety.

5.1.2. Nutritional status

There are limited data on the nutritional status of recovered BN patients who are medically stable and have ceased binge eating and purging. Kirkley, Agras, and Weiss (1985) examined the food records of a sample of patients who had ceased binge eating and purging after cognitive-behavioral treatment. Records from the week after treatment termination revealed a prevalence of dietary inadequacies. Common inadequacies included the insufficient intake of fiber, fat, potassium, calcium, magnesium, iron, and thiamin. Significantly, this study did not use a non-bulimic control group for nutritional comparison. Kirkley et al. concluded that intervention must focus on all aspects of dietary intake so that BN is replaced by a healthy eating style.

Rock and Curran-Celentano (1996) proposed that whereas the emphasis in CBT is on the regularity of eating rather than the composition of meals or snacks, many patients benefit from more detailed assistance in planning meals. CBT and nutritional intervention do recommend expanding patients' diet to incorporate previously forbidden foods. However, the goal of this flexibility is primarily to prevent restriction-induced binge eating (e.g., Beumont et al., 1997). Any effects of this strategy on increasing vitamin and micronutrient intake are, in most cases, probably coincidental. One of the recommendations of nutritional counseling programs that differs from CBT is that patients use exchange lists to monitor dietary intake (O'Connor et al., 1988; Story, 1986; Willard et al., 1983). Rock and Curran-Celentano even suggested that foods be pre-portioned and labeled with caloric and nutrient content, partly as a means of reassuring patients when re-introducing forbidden foods into the diet.

5.2. Potential problems with an increased focus on nutrition

It may be counterproductive for BN patients to focus too much attention on calories, exchanges, and the composition of food. In CBT they are discouraged from counting calories or fat grams, or precisely measuring or weighing food (Fairburn et al., 1993). Quantities should be described in approximate terms (e.g., one large slice of pizza). Counting calories gives patients a false sense of security, since energy absorption varies among individuals even when calorie levels are held constant (Vitousek, 1999). Patients are better served by allowing a certain margin of error in choosing their food intake. BN patients' over-vigilant attempts to control caloric intake and expenditure typically lead to increasingly restrictive food choices, misleading notions about weight control, unnecessary anxiety and fear, and a sense of defeat and lost control when a rule is broken or a mistake is made (Wilson & Vitousek, 1999). The challenge to nutritional approaches will be to address specific appetitive abnormalities without exacerbating the dysfunctional vigilance and rigid concerns that characterize BN patients.

The treatment of BED poses a different problem. Obesity is a major comorbidity in BED patients (Wilfley & Cohen, 1997). Chaotic eating patterns with little or no restriction between binge eating episodes are characteristic of these patients, and binge eating frequently precedes dieting in the development of the disorder (Howard & Porzelius, 1999; Yanovski, 1993). Given the importance of weight loss or at least preventing additional weight gain, self-monitoring of calories, fat grams, or exchange units is essential. It is now also clear that behavioral weight loss programs aimed at reducing caloric intake in obese patients with or without BED have positive effects in reducing binge eating (National Task Force on the Prevention of Obesity, in press). There is less need to be concerned about the problems of increasing dietary restraint as in BN.

6. Concluding comments

Effective treatment of BN and BED requires modification of eating behavior, attitudes governing food intake, and dysfunctional concerns about body shape and weight. Currently, manual-based CBT is the treatment with the most empirical support (Wilson & Fairburn, 1998). CBT is designed to restore a regular pattern of moderate yet flexible eating (Fairburn et al., 1993). As Beumont et al. (1997) observed, the key to treatment is changing the psychology of the individual with the eating disorder rather than modifying the micro- and macronutrient specifics of the person's diet.

In general, manual-based CBT requires formal professional training and specific clinical expertise. Within a stepped-care approach to treatment, however, simpler and more cost-effective forms of CBT lend themselves to use by a wide range of health providers including nutritionists and dieticians.

A better understanding of the appetitive abnormalities that have been identified in BN and BED may result in additional interventions that augment the efficacy of CBT (Ventura & Bauer, 1999). In introducing a specific focus on the role of micro- and macronutrients in the treatment of BN, care must be taken to avoid undermining the effects of CBT in reducing rigid and dysfunctional preoccupation with food.

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