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Review

Cognitive behavior therapy for eating disorders versus normalization of eating behavior

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HIGHLIGHTS

- Cognitive behavior therapy (CBT) assumes that cognitions maintain eating disorders.
- The rate of remission and relapse after CBT is <50% and >30%, respectively.
- These measures improve to 75% and 10% by treating eating behavior directly.
- Normalizing eating behavior resolves the cognitive effects of eating disorders.

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ABSTRACT

We examine the science and evidence supporting cognitive behavior therapy (CBT) for the treatment of bulimia nervosa and other eating disorders. Recent trials focusing on the abnormal cognitive and emotional aspects of bulimia have reported a remission rate of about 45%, and a relapse rate of about 30% within one year. However, an early CBT trial that emphasized the normalization of eating behavior had a better outcome than treatment that focused on cognitive intervention. In support of this finding, another treatment, that restores a normal eating behavior using mealtime feedback, has an estimated remission rate of about 75% and a relapse rate of about 10% over five years. Moreover, when eating behavior was normalized, cognitive and emotional abnormalities were resolved at remission without cognitive therapy. The critical aspect of the CBT treatment of bulimia nervosa therefore may actually have been the normalization of eating behavior.

Contents

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1. Introduction

Reviews of current treatments for eating disorders invariably conclude that there is need for improvement in the outcomes of these therapies [1–10]. It was recently suggested that rather than undertaking all the randomized controlled trials (RCT) necessary to evaluate which of the many psychotherapeutic interventions are the most effective in behavioral disorders, the field should aim at interventions that are based on an understanding of the mechanisms that mediate the abnormal behavior of the patients [11,12]. Surprisingly, "… there is a plethora of behavioral intervention development research that does not explicitly and directly relate to or integrate any basic science at all" [12].

The use of cognitive behavior therapy (CBT) for the treatment of bulimia nervosa is supported by clinical evidence from RCTs. Because CBT is one of the few treatments recommended for bulimia in treatment guidelines (e.g., [13]), clinicians may take for granted that such recommendations have a solid scientific basis [14]. However, neither the theoretical status of CBT for bulimia, nor the data supporting its clinical effectiveness have been carefully evaluated. Therefore, we will first analyze the assumptions on which CBT rests. We will then examine the success of CBT in the clinic. After that, we will contrast the science and clinical effects of CBT with those of a treatment that is based on normalizing eating behavior, rather than normalizing the cognitive changes associated with eating disorders [15]. We will end by suggesting that normalization of eating behavior is the critical aspect of any effective treatment for eating disorders.

2. Science of CBT for bulimia

When the diagnosis of bulimia nervosa was introduced, this eating disorder was considered to be a variant of anorexia nervosa [16], and from the early descriptions of CBT for bulimia, it was again pointed out that bulimia is quite similar to anorexia [17,18]. For example, the bingeing seen in bulimia occurs after a fast, and it is long known that occasional bingeing and purging also occur in anorexia, a state characterized by chronically fasting [19].

2.1. Introduction of CBT for bulimia: eating habits cause psychological change

In a report describing 11 women with bulimia nervosa, CBT was used to interrupt overeating and vomiting and to "modify her abnormal attitudes to food, eating, body weight and shape" [20]. This outcome was achieved by informing the patients about the importance of self-control, the dangers of self-induced vomiting, asking them to record the food they ate, increasing their self-control by reducing the availability of food, and encouraging activities that were incompatible with overeating. The circumstances in which vomiting occurred were then identified and these were often associated with changes in mood, including feelings of guilt. To change these feelings, the patients were trained in problem-solving, as well as being trained to recognize irrational thoughts about their body weight and shape. Additionally, the patients were gradually exposed to foods that they had been avoiding [20].

Thus, CBT for bulimia originally intended to normalize eating behavior, along with normalizing some psychological responses to food. The finding that these "specific psychopathologies" decreased when eating habits were normalized, however, suggests that the psychopathology is secondary to the disordered eating behavior, rather than their cause [21,22].

2.2. Cognitive behavioral theory: psychological change maintains eating disorders

A cognitive behavioral theory was then developed to support the use of CBT for the treatment of anorexia nervosa [23]. Central to this theory is a "need for self-control in general, which is likely to be a product of these individuals´ well-recognized and characteristic sense of ineffectiveness and perfectionism, and which interacts with longstanding low self-esteem" [23].

The concept of self has long been part of psychological theory and self-control has been defined as the capacity to regulate thought and behavior in the face of conflict [24–26]. Accordingly, something done effectively or perfectly is done in a controlled manner. Effectiveness and perfectionism are therefore not independent variables, and the notion that self-control is a function of effectiveness and perfectionism is a definition, rather than a hypothesis. Self-esteem has been defined as the sense of self-worth, which is the result of professional, scholastic, and other social achievements [26]. Since self-esteem, i.e., the sense of self-worth, is a function of self-control, these two aspects of self are also not independent. Therefore, the theory should predict that anorexia is a function of self-esteem. Self-esteem increases as someone learns to do things the right way and many studies have concluded that self-

esteem influences many aspects of life [24–26]. The cognitive behavioral theory for the basis of anorexia, therefore, lacks specificity.

A scientific theory is "a well-substantiated explanation of some aspect of the natural world that can incorporate facts, laws, inferences, and tested hypotheses" [27]. Hence, rather than proposing a theory, the cognitive behavioral theory was first formalized as a hypothesis, which is "A tentative statement about the natural world leading to deductions that can be tested" [27].

2.3. Transdiagnostic theory

The cognitive behavioral theory was subsequently expanded into a "transdiagnostic theory" that addresses the psychopathological processes involved in the maintenance of all eating disorders. The central notion put forward involved four such processes: perfectionism, self-esteem, mood intolerance, and interpersonal difficulties interacting with "a dysfunctional system of self-worth", which were regarded as the "core eating disorder maintaining mechanism" [28–30].

While perfectionism and self-worth may be more similar than different, the addition of "mood intolerance" to the list of emotional issues was suggested to reflect "a more complex relationship between emotional states and binge eating" [28,30]. Several sub-processes were then introduced to explain the variety of behavioral responses observed in eating disorders, which were regarded to be "habitual means of mood modulation". The interpersonal difficulties that can be associated with bulimia were also analyzed with the introduction of four extra substressors, including family tensions, interpersonal environments, adverse interpersonal events, and long-term interpersonal difficulties [28].

A long recognized problem with cognitive theories, however, is that their paradigm of one cognitive process causing or maintaining another generates an endless circular regress, making it "… impossible to break into the circle" [31]. This problem was identified by Aquinas already 750 years ago [32]. Also, the increasing complexity of this approach is the opposite of the more common scientific approach, "… to make the irreducible basic elements as simple and as few as possible" [33].

When the clinical interview aiming at identifying the cognitive processes or attitudes maintaining eating disorders was developed, it was noted that these processes and attitudes are not specific, but similar to "widely held views" [34]. While the interview and the subsequently developed questionnaire have been reported to have good validity and reliability [35–37], this finding has not been consistently replicated [38–41]. The inconsistencies in the data may be the result of the use of factor analytical methods, which are based on the assumption that the data that are analyzed are normally distributed. However, answers obtained from questionnaires do not yield such data, but application of the methods of item response theory may resolve these inconsistencies [42].

There have been only a few attempts to test the cognitive behavioral theory of eating disorders. One such effort failed [43], and the other study demonstrated correlations between the processes that are postulated in the theory, but noted that it was not possible to establish causality [44]. Exaggerated attention towards eating and body shape and weight related stimuli, an important aspect of the cognitive behavioral theory, has been demonstrated in women with anorexia nervosa, but the authors pointed out that there were no controls for the effect of starvation and that the effect, therefore, might not be specific for anorexia [45–48]. Moreover, the effects on attention found in other eating disorders were not consistently replicated (*ibid*).

2.4. Causation

While the analyses of causation and reductionism are complex [24,49], the concept of mechanistic cause used in the cognitive behavioral theory departs from the common concept of mechanism, which is a reference to a more basic level of analysis in a reductionistic hierarchy, for example the neuroscience level [50]. Darwin [51] pointed out long ago, however, that in normal conditions, the causal relationship between brain and behavior is bidirectional, a suggestion that has been confirmed and extended in the context of eating behavior [52,53]. Out of many possibilities [54], a mechanism could involve the release of serotonin in the brain that inhibits eating [55]. This mechanism would be of interest in the context of bulimia, as binge eating may involve loss of control over eating. The neural engagement in response inhibition is presently analyzed in considerable detail in other contexts [56], with serotonin playing a significant role [55]. However, any theory of eating disorders should take into consideration that a healthy, stable, relatively low body weight is maintained only when the physical price of food is high and that, in these normal circumstances, humans are able to consume large amounts of food [57]. This behavioral aspect of the human homeostatic phenotype, and perhaps overeating in bulimia as well, should therefore be cautiously interpreted and not necessarily as a sign of loss of control, or a deficient response inhibitory mechanism, but as a normal, evolutionarily conserved capacity [57].

2.5. Summary

The most recent version of the cognitive behavioral theory allows for many combinations of thoughts and moods that are regarded as important for the maintenance of eating disorders [28,30,58]. It would therefore be difficult to test the efficacy of a treatment based on all of these emotional interactions. Thus, it is easy to identify processes that are the same, but are proposed as being different, such as self-esteem and self-worth, or effectiveness and perfectionism. However, at the heart of the theory is the hypothesis that a low self-esteem maintains eating disorders, but as self-esteem is engaged in many aspects of life, the hypothesis lacks specificity. Interestingly, however, although the cognitive behavioral theory does not address the cause of eating disorders, it recognizes that anorexia starts with "the onset of dietary restriction" [30] and that dieting is a risk factor for bulimia as well [59].

3. Outcomes of CBT for the treatment of bulimia

Theories of eating disorders should be evaluated by their clinical success. We will therefore discuss the effect of CBT in clinical practice. When it was introduced, CBT was given as 20 weekly, approximately one-hour out-patient sessions, over about five months, and its effect was compared with that of interpersonal therapy (IPT) and behavior therapy (BT) [18,60]. The IPT was developed to treat depression and focused on solving problems associated with interpersonal relations [61]. BT endeavors to normalize eating behavior. While it is not possible to determine precisely how long the patients had abstained from bingeing when they were in remission in the various studies, we will use the primary criterion for remission adopted in these studies to be the abstinence of bingeing and purging for 28 days. This criterion has also been used in meta-analyses of the outcome of RCTs in which CBT has been compared with other treatments [62]. No other criteria for remission will be considered. Differences in the proportions of patients who met this criterion will be evaluated with chi square tests.

3.1. First evaluation of CBT: combination of patients from two RCTs

In the first evaluation, the results from an RCT with 25 patients treated with CBT were combined with the results from another RCT with 10 similarly treated patients. Thus, 35 patients treated with CBT were reported, including a follow-up period of approximately six years [18,60,63,64]. The comparison group of 32 patients was also a combination of the patients treated with IPT in these two RCTs. Another comparison group included patients treated with BT, but because this therapy had a minor positive effect which was followed by rapid relapse, it will only be reconsidered at the end of this review (see "5.1. Behavioral techniques are more effective than cognitive techniques" below).

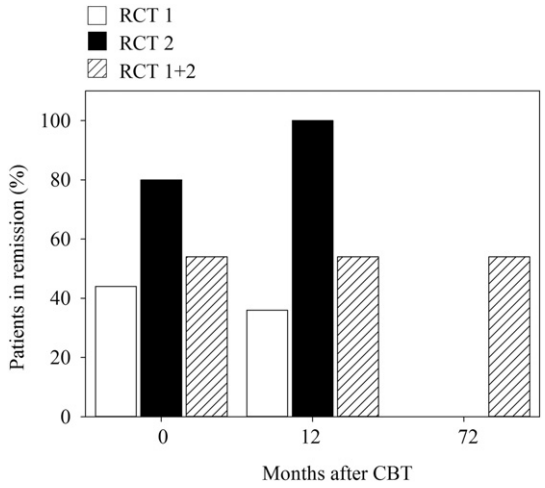

Fig. 1. Percentage of bulimic patients in remission 0, 12, and 72 months after treatment with cognitive behavior therapy (CBT) in two randomized controlled trials (RCT). There were 25 patients in RCT 1 (open bars) and 10 patients in RCT 2 (black bars). The outcomes in RCT 1 and RCT 2 were combined (striped bars) in the report [64].

While in the first RCT, 44% and 36% of the patients were in remission after treatment and at 12 months of follow-up, 80% and 100% of the patients in the second RCT were in remission at these points in time (Fig. 1). The probability that these differences in outcome occurred by chance is $p = 0.052$ and $p < 0.01$, respectively. Thus, two patients relapsed over the first year of follow-up in the first RCT, two remitted in the second RCT, and 54% of the patients were reported to be in remission at all times of follow-up (Fig. 1) [64]. This is the only study in which patients in remission from bulimia after CBT were followed for a prolonged period of time.

In the comparison group treated with IPT, outcomes were similar in the two RTCs that were combined but fewer of the patients were in remission after treatment compared with the patients treated with CBT. However, the rate of remission increased from <30% to 50%, and finally to 70% at 0, 12, and 72 months of follow-up, with no relapse [64].

It should be noted that while absence of binge eating was reported in one study (Figure in [60]), the absence of an eating disorder diagnosis was reported in the other (Figure in [64]). Although the complete absence of binge eating is not a necessary criterion for the absence of bulimia nervosa, it is a necessary criterion for the absence of the diagnosis of an eating disorder not otherwise specified, according to the diagnostic manual that was used in these studies [65]. Hence, we assume that the patients reported as not having an eating disorder diagnosis did not display bulimic behavior, although their report is not clear on this point [64].

3.2. Replication of the effect of CBT and IPT

The 54% rate of remission after CBT for bulimia, however, has not been replicated [66–72]. According to the most recent meta-analysis, the combined rate of remission as assessed by the abstinence of binge eating averages 37% [62,73]. There are two interesting exceptions to these findings. One is a report of a 63% rate of remission from bulimic behavior in a group of patients treated with CBT in combination with support via the Internet over 12 weeks [74]. In a similar recent study, the patients improved without web-based CBT [75]. However, a minority of patients can benefit from online interventions [76]. For example, in another recent study, the rate of remission was about 15% with online CBT [72]. It should be noted that not all of the patients in these studies were diagnosed with bulimia nervosa, some had a binge eating disorder diagnosis. The other successful study reported the absence of bulimic behavior after a mere nine weekly sessions of an individualized version, as opposed to the manualized version of CBT, although the number of patients in remission was not reported [77].

On the other hand, the delayed effect of IPT has been replicated, i.e., patients keep going into remission well after treatment, with no relapse [62]. However, 44% of all CBT and IPT patients continued to be diagnosed with an eating disorder after treatment [64], outcomes that have also been replicated [62,73].

There is only one other report of the long term effect of CBT on bulimia nervosa [78]. Although the patients in this study improved markedly, they continued bingeing about once a week and purging about 2–4 times every other week for more than four years. After they eventually stopped bingeing, they continued purging at a low rate throughout the five years of follow-up [78]. These patients were not in remission, using the absence of bulimic behavior for 28 days as the standard for remission.

3.3. Relapse after CBT

The original, long-term evaluation of CBT for bulimia was a combination of two studies [64]. While two patients relapsed over the first year of follow-up in one of these studies, two remitted over the same period of time in the other study and 19 (54%) of 35 patients were in remission over the six years of follow-up (Fig. 1) [64]. Thus, the reported data give the impression that there was no relapse after CBT for bulimia (Fig. 1).

The absence of relapse after CBT also has not been replicated. For example, only 21 of 32 patients who recovered with CBT remained recovered after 12 months [66]. Thus, 34% of the patients relapsed. It was subsequently pointed out that in this study, "the rate of relapse [was] matched by an equivalent rate of remission among those participants who were not yet fully asymptomatic at the end of treatment" [79]. Using 28 days without bulimic behavior as the criterion for remission, another study found a 44% rate of relapse within four months after CBT, although this rate was subsequently lowered by relaxing the criterion for remission [80,81]. However, one of the two studies mentioned above that reported better outcomes for bulimics after CBT also reported no relapse over six months of follow-up [77]. No relapse after 12 months of follow-up was also reported in six out of 13 patients treated to remission [82]. Finally, a recent study reported an increase in the rate of remission from 20% at discharge to 32% 12 month later in a group of 12–18 year old patients. However, 59% of these patients received treatment during follow-up [83].

These results bring into question the stability of remission achieved by CBT. This problem is evident in a recent RCT comparing CBT with psychoanalytic psychotherapy. While 15 (42%) of 36 patients were in remission after CBT in this study, five (33%) of these 15 patients had relapsed 19 months later. At the same time, six other patients went into remission 5–19 months after CBT, thus 16 (44%) of 36 patients were in remission 19 months after CBT. Once again, patients who relapsed after CBT were matched by an equivalent rate of remission among the other patients. The data presented in their report [84], however, give the impression that there is no relapse after CBT for bulimia. This impression was also maintained when the paper was podcasted by the journal that published the paper [85]. The problem of reporting no relapse, while, in fact, patients do relapse, was recently pointed out by the authors of another overview of the effect of CBT for the treatment of bulimia [86].

3.4. Post-treatment remission

The 18–44% rate of relapse within one year of CBT is apparently compensated for by an equal rate of post-treatment remission among patients who did not remit at discharge from the treatment [64,66,79–81,84].

Additional treatment during follow-up is one obvious reason why patients may go into remission after their initial treatment. Indeed, 38% of the patients reported in the first evaluation of CBT and 31% in the recent study received such additional treatment [64,84].

Unsurprisingly, more patients who were not in remission compared to patients who were in remission received treatment during follow-up [64]. Paradoxically, however, treatment of patients who relapse after CBT has been reported to have no effect [87].

Similarly, a potential reason for the increase in the rate of remission up to six years after IPT may be because these patients were also treated during follow-up [64]. Considering that fewer than 30% of the patients were in remission after treatment with IPT, it is not surprising that they sought out additional treatment. No groups in these studies were included to control for the effect of the post-treatment remission after CBT or after IPT.

3.5. Natural course of bulimia

A study of the natural course of bulimia nervosa over 7.5 years reported that 74% out of 95 patients had achieved a "full recovery" at about six years of follow-up [88]. This percentage of recovered patients is higher than the 54% of the patients who were in remission at the same time of follow-up after CBT (Fig. 1, [64]) and twice a high as the 37% rate of remission reported in meta-analyses [62]. In another report, an average of about one-third of 95 patients had remitted at each of five yearly follow-up examinations [89]. Thus, naturalistic sample reports have obtained rates of remission that are as high or higher than those reported in patients treated with CBT. While about a third of the patients had relapsed in the naturalistic studies [88,89], this rate of relapse is not unlike the 18–44% relapse rates within one year after CBT [64,66,80,84].

Even though the rates of remission and relapse in these studies are about the same as those reported with CBT, the outcomes are not directly comparable. In one study on the natural course of bulimia, 10% of the patients received treatment and 16% had previously been treated at the time of recruitment, but the probability that the patients received additional treatments increased as the duration of follow-up increased ([88] and see above).

It is noteworthy that the 34% and 53% spontaneous rate of remission at a two-year examination of the patients in naturalistic studies [88,89] is >2–3 times as high as the 15% rate of remission after two years of psychoanalytic psychotherapy [84] ($p = 0.062$ and $p < 0.01$, respectively). These data suggest that psychoanalytic therapy not only has no positive effect on bulimia, but that this therapy actually makes the patients worse. Therefore, it is quite important that all RCTs use an untreated control group when the effect of a treatment is unknown [14,90]. While it is often considered unethical to use untreated control groups in clinical trials, this ethical problem is the same as that which arises when one intervention turns out to be superior to another. The solution to the problem is to apply the standard stop rules in clinical trials [14,90].

The reported superiority of CBT for bulimia over psychoanalytic psychotherapy in the absence of an untreated control group has, therefore, limited value [84]. The authors correctly pointed out that patients waiting for treatment do not remit in the short term. However, the studies on the naturalistic course of bulimia suggest that patients may remit over the course of each year, although it must be kept in mind that patients are also likely to receive more treatment in the long term [62,88,89].

It has not been clearly demonstrated, therefore, that patients who remit many years after CBT or IPT remit as a result of these original treatment approaches. Yet according to guideline recommendations, ITP is as effective as CBT, but takes longer to work [62]. However, it is not possible to explain how remission is achieved years after the initial IPT has been completed [64]. Considering that relapse is an important problem in the management of bulimia nervosa, ranging between 25 and 63% with most patients relapsing within six months [91], the high rate of post-treatment remission long after the completion of ITP is enigmatic.

3.6. CBT for bulimia in clinical practice

In an attempt to use CBT for bulimia in primary care, fewer than 30% of the patients completed the treatment [92]. Another effort at using CBT within the U.K. health-care system started with 683 referrals, 451 patients were then assessed, 396 were deemed suitable for treatment, 272 started treatment and 135 of these patients completed treatment [93]. Although there was a significant improvement in this group, patients who completed the treatment continued to binge. A better outcome was achieved in a sample of 78 patients out of whom 65% were bulimic [94]. In a group of 203, including all eating disorder diagnoses, 100 (49%) patients completed the treatment and 31% of these were in remission after treatment [95]. The outcomes for CBT for bulimia in primary care, therefore, are not impressive.

3.7. CBT for anorexia and eating disorders not otherwise specified

The transdiagnostic theory has been promoted to explain the maintenance of all eating disorders and CBT has therefore been used to treat other eating disorders as well as bulimia. The results of these studies were recently listed and briefly discussed, but not analyzed [96].

Thus, patients diagnosed with anorexia nervosa including an average Body Mass Index (BMI, weight/height squared, kg/m^2) of about 16–17 have been treated with CBT as out-patients. The rate of drop-out has been up to 37% and those who have completed the treatment have gained weight, but not to normal levels [97–100]. Unsurprisingly, the outcome was better in young patients who had anorexia for a short time than in older patients [101]. In a long-term follow up study, 12 of 19 patients with a relatively high average BMI = 17.3, completed CBT [102,103]. The patients were reported to have normal BMIs on average 6.7 years later, but since this outcome included five patients who did not complete the treatment, the results are difficult to interpret. A review of the results of CBT in anorexia concluded that there is "inconsistent information on the effectiveness of CBT for AN" [104] and another review suggested that the supporting "evidence is tentative as CBT-E is still in its early phases of empirical testing" [105].

Interestingly, CBT has been reported to have a better effect in inpatient treatment of anorexia. Thus, the BMI increased from an average of 14.3 to 19.2 in a group of patients treated with CBT, out of whom only one of 27 dropped-out [106–108]. Taking changes in energy expenditure, metabolic rate, glycogen storage, muscle and fat mass, and physical activity into consideration, it can be estimated that these patients must have consumed ≥2500 kcal/day [109–114]. This rapid increase to a normal body weight in low weight patients is remarkable and will be further discussed in the final part of this review (see "5.2. The CBT - BT paradox" below).

One study on patients, 62% of whom had an eating disorder not otherwise specified, reported some success, although 40% of those who were eligible did not take part [115]. A better outcome was obtained in patients who were not underweight [116]. In another study, including patients with any eating disorders, 53% completed CBT and 56% of these patients went into remission [117]. A 22% rate of drop-out and a 66% rate of remission were reported among a group of patients, most of whom had an unspecified eating disorder [118]. A better outcome of CBT has been reported for the treatment of obese patients with a binge eating disorder (BED), without a decrease in BMI in two studies [119,120], but with a significant decrease in BMI in the other [121]. Finally, a recent review concluded that there is "… strong support for therapist-led CBT … in helping patients with BED reduce binge-eating frequency and achieve abstinence" [122]. However, this conclusion was based on five studies, none of which used the standard criterion "the abstinence of bingeing and purging for 28 days" [123]. Nevertheless, CBT has demonstrated some efficacy for eating disorders other than bulimia.

3.8. Summary

A recent review pointed out that there has been no improvement of the effect of CBT for bulimia over time, despite shifting the focus of the treatment to additional personality issues and away from targeting

eating behavior [86]. To the contrary, the reported rate of remission after CBT decreased from 54%, reported in the first evaluation of CBT [64] to about 37% today [62,73]. While it is often the case that an improvement of the rate of remission with a new treatment cannot be consistently replicated (see discussion in [90]), it is also important to recall that the 54% rate of remission was obtained by combining two groups of patients, one in which 36% were in remission with another group in which 100% were in remission 12 months after CBT. No explanation was given for the difference in outcome, which brings into question the interpretation of their results. This study is the only one in which patients treated to remission with CBT have been followed for a prolonged period of time. Although this study reported that only a few patients relapsed, the rate of relapse has been >30% within a year in other studies. Moreover, these reported rates of remission and relapse are similar to those reported in naturalistic studies on the long-term outcome of bulimia. Because patients receive additional treatment over prolonged periods of follow-up, reports on remission, relapse, and long-term effects of CBT are inconclusive.

The more than doubling of the rate of remission in the years following IPT has not been explained. Attempts at using CBT within the public-health care system reached only a minority of the patients and preliminary findings of the effect of CBT in anorexia and other eating disorders are uncertain.

While "CBT has retained and extended its status as first-line therapy for bulimia nervosa" [73], it is obvious that there is room for improving the treatment of bulimia and other eating disorders. We will therefore suggest how an improved outcome can be achieved.

4. A treatment that addresses the disordered eating behavior in eating disorders

It is generally recognized that the various forms of eating disorders are more similar than different, and, in most accounts, they are regarded as "chronic, relapsing illnesses with a substantial medical morbidity and high mortality" [7,8,124], a description that has not changed much for decades [125]. The reason for this lack of significant progress, we suggest, is that most explanations regarding the cause and maintenance of eating disorders rest on weak or no scientific evidence. In fact, it was recently concluded that many psychotherapeutic interventions do not "… relate to or integrate any basic science at all" [12]. In order to improve the situation, it has been suggested that research on behavioral interventions should be related to realistic neurobiological mechanisms [126].

4.1. Mechanisms mediating onset and maintenance of eating disorders

We have suggested that dieting is the major cause of anorexia, the prototypical eating disorder from which the other disorders emerge [127]. This suggestion is in line with the original conclusions reached in CBT, that: "Dieting is the most prominent characteristic of people with eating disorders" [30]. We went on to suggest that dieting increases the release of dopamine from the ventral striatal terminals of the dopamine cell bodies in the mesencephalon, which are engaged in "reward", encouraging a patient to continue eating less food. At the time, there was a substantial amount of data in experimental animals supporting this model and the model was subsequently supported by evidence in humans, showing that ventral striatal activity was higher when the patients were exposed to stimuli associated with being underweight than when exposed to normal-weight stimuli, while normal-weight women had the opposite response [128]. The role of dopamine in eating and the development of anorexia nervosa is now generally acknowledged [129–131].

We further suggested that the disordered eating behavior is maintained by conditioning to the situations that provided the reward, because the brain's system for attention (the noradrenaline cell bodies in the locus coeruleus and their forebrain projections) is also engaged by continued dieting [127]. Simultaneously, conditioning to initially neutral cues was also suggested to play an important role in the development of disordered eating [132]. We have reviewed the extensive body of facts taken from basic neuroscience that supports this view and additional evidence has since accumulated to solidify this framework [15,57,131,133–135]. Our framework is further strengthened by the recent demonstration that the locus coeruleus noradrenaline neurons, which are engaged in cue conditioning and attention, respond in proportion to the incentive power of the cues [136].

A word of caution seems appropriate. Thus, it should be recognized that changes in behavior, however they are achieved, are ultimately represented in the brain. Therefore, changes in neurotransmitter systems should be carefully interpreted. Relating disordered eating and recovery to changes in the brain that usefully informs interventions requires more sophisticated data and analyses than provided in the present review. The point we wish to make is that our framework complies with the request for neural plausibility [126]. The meaning of the term "framework" in behavioral and cognitive neuroscience was explained by Crick and Koch [137] in an analysis that we have recapitulated in the context of eating disorders [138]. The majority of the interventions used in behavioral treatments do not take basic science into consideration [12] and we have suggested that this is also the case with most treatment for eating disorders, a possible reason for their limited effectiveness [133].

At the core of the cognitive behavioral theory, however, eating disorders are "essentially cognitive disorders" [30], but as we will discuss below, the abnormal emotional and cognitive symptoms in patients with anorexia and bulimia are *caused* by their disordered eating behavior.

4.2. Intervention: targeting eating behavior

Eating behavior, which has changed in eating disorders, is the main target in a treatment, first tested on patients with anorexia nervosa [139]. Patients practice normal eating behavior, assisted by feedback on a small computer screen that shows how much food there is to eat and how quickly they should eat it (Fig. 2). The patients eat very little food very slowly, and they are trained to increase the amount of food that they eat, along with increasing their rate of eating. The eventual goal is to eat 300–350 g of food in 10–15 min; the amount of food and the rate at which the food is eaten were obtained in tests of healthy subjects of normal weight [140]. Ordinary Swedish food has been used and a typical meal might be mixed vegetables and grilled chicken cubes (385 kJ; 9.3 g protein, 8.0 g carbohydrates, 2.8 g fat/100 g, Findus AB, Bjuv, Sweden).

Patients also learn to feel satiated during meals in a similar manner (Fig. 2). It is noteworthy in this context that behavioral interventions can be more effective if delivered by computer than if delivered by people [142].

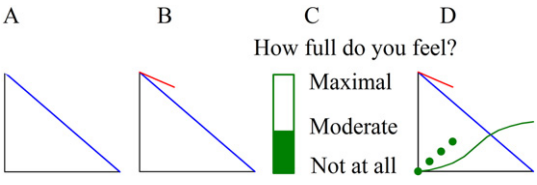

Fig. 2. Computer assisted eating. A. Patient should follow blue training line while eating. B Patient eating too slowly as indicated by the red line, the computer encourages the patient to eat more quickly to follow the blue training line. C. Patient is asked to rate feeling of fullness (satiety) at regular intervals during the meal. D. Satiety ratings (green dots) are plotted on computer screen and the patient learns to associate feeling of fullness with green S-shaped line. No numerical values are displayed on axes during treatment. Eating slowly and rating satiety high as in this case indicate an anorexic pattern of eating. Reproduced from [141] with permission.

At admission, patients with a low BMI displayed an eating behavior similar to that of patients with a higher BMI, but both groups of patients had the same high level of anxiety, determined by a self-rating scale [143]. As food intake and the rate of eating increased from admission to remission the level of anxiety normalized and they were maintained over five years of follow-up (Fig. 3) [138]. Thus, anxiety in these patients was related to their eating behavior rather than their BMI.

In addition to managing eating behavior, this treatment also has other features, including the provision of external heat, a reduction of physical activity, and the restoration of social activities (see [15,133] for the rationale of these aspects of our treatment). The treatment has been demonstrated to be effective in an RCT, with an estimated rate of remission of 75% in an average of one year of treatment and a rate of relapse of 10% over five years of follow-up [15,133]. There was no mortality either during treatment or during follow-up.

4.3. Decelerated versus linear eaters

The change in the rate of eating over the course of the meal is an important characteristic of eating behavior. Thus, food intake in research animals and humans of normal weight gradually decreases over the course of the meal, thereby describing a decelerated curve [140, 144–148]. Westerterp-Plantenga et al. [147] first referred to this behavior as decelerated eating.

In a model that describes the slowing of food intake over the course of a meal, $y = kx^2 + lx$, y = food intake, k = the change in the rate of eating over the course of the meal, and l = initial rate of eating [140, 145,146]. As the negativity of k increases, l increases, and the pattern of eating of a decelerated eater emerges. Conversely as $k \rightarrow 0$, l decreases, and a pattern of eating, which Westerterp-Plantenga et al. [147] first referred to as linear eating, emerges. A positive k-value is rarely seen, although individual patients, diagnosed with bulimia nervosa, ingesting a yoghurt shake at an accelerating rate in laboratory conditions have been reported [149,150].

4.4. Behavioral risk for eating disorders

95% of eating disorders patients are women and the immediate cause of eating disorders is dieting [57,127,134,135]. It is noteworthy, therefore, that men and women respond differently to a brief fast, such as skipping dinner. While men eat more food the following day, women eat less food the following day [151,152]. These food-deprived, but healthy women also adopt a linear pattern of eating behavior that is similar to that seen in anorexia nervosa [135,152,153]. Linear eating was subsequently shown to be associated with loss of control of food intake when the rate of eating was experimentally increased or decreased [140]. For example, as opposed to decelerated eaters, female linear eaters eat less food, yet they perceive a high level of satiety after the meal when eating at a reduced rate, i.e., their behavior comes close to that of patients with anorexia nervosa (Fig. 4 [140]).

Dieting is thus followed in women by a linear pattern of eating behavior that is similar to that of underweight patients with anorexia. At the same time, women who skipped dinner the day before testing are able to eat a normal-size meal if assisted by mealtime feedback [135, 152]. While available information on the neurobiological and metabolic changes associated with short-term or long-term fasting [154] does not permit an explanation of the changes in eating behavior, the fact that the behavioral changes are reversible suggests that whatever the biological changes are, they do not prevent the display of the normal pattern of eating behavior [152]. This finding is not unlike what is seen in women with anorexia nervosa, who typically eat very little food very slowly, yet have a capacity to eat a normal-size meal at the proper rate [57]. By contrast, women with bulimia nervosa, who are of normal weight, eat normal-sized meals at a normal rate at admission [133]. Their eating behavior is similar to that of healthy women when tested in laboratories, although their intake of an excessive amount of food in a short period of time, i.e., binge eating, can be elicited in laboratory conditions by asking them to binge [149,150]. However, the amount of food that they consume in these conditions may have been exaggerated in some accounts [155], and intake of excessive amounts of food in a short period of time can occur in healthy people and has been used in the entertainment industry for a long time (e.g., [156]). It is also long known that people normally ingest large amounts of calories when the need for physical activity is high [157].

4.5. A note on the evolution of the behavioral response to fasting

The decrease in the rate of eating and the increase in the linearity of eating after fasting were noted long ago in both experimental animals [158] and humans [159]. Remarkably, it was demonstrated that *Drosophila* responds in a similar manner when food is in short supply, including having an increased release of dopamine in their brain [148], the postulated initial response in the neuroendocrine chain of events which leads to anorexia [127]. The behavioral, metabolic, and physiological responses to fasting and starvation appear conserved across phyla, and it has therefore been suggested that Drosophila can be used as a model for the study of metabolic homeostasis, including human metabolic disorders [160]. It has also been suggested that "nothing in biology makes sense unless viewed in the light of evolution" [161]. The way women respond when the supply of food is limited should therefore not be considered as a sign of an emerging disease, but as a normal response that has evolved to meet the challenge of starvation, the main threat to the maintenance of human life over the millennia [57]. Because women with anorexia nervosa can eat normal meals even at a BMI =

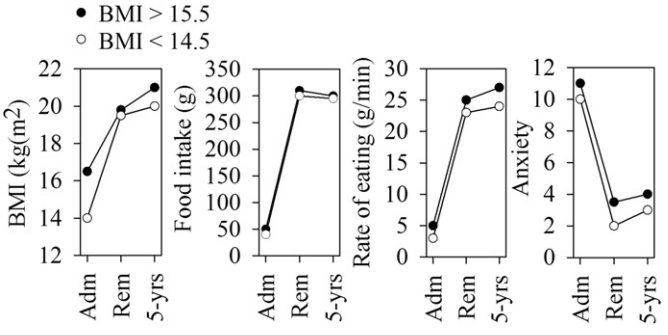

Fig. 3. Food intake, rate of eating, and level of anxiety in 18 anorexic patients selected with a BMI > 15.5 and in 27 anorexic patients selected with a BMI < 14.5. The patients were treated to remission and followed-up over five years. Values are means at admission (Adm), remission (Rem), and at five years of follow-up (5-yrs). Measures of variability have been omitted to facilitate visual inspection of the data. Reproduced from [138] with permission.

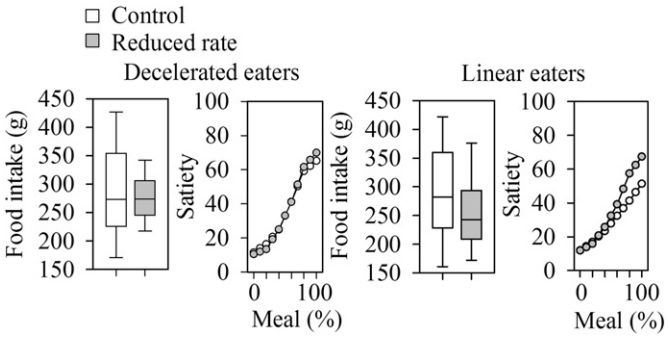

Fig. 4. Food intake and rating of fullness (Satiety) in 17 women selected for eating at a decelerated rate (Decelerated eaters) and in 30 women selected for eating at a linear rate (Linear eaters). The women ate with no time constraints (Control) and at an experimentally reduced rate (Reduced rate). Satiety ratings are expressed in relation to the percentage of the meal. Vales are box plots. Reproduced from [140] with permission.

15, they provide an example of an ancient human homeostasis, in which body weight was maintained at a constant low level, physical activity was permanently high, and there were no constraints on the capacity to eat large meals [57]. Although a detailed description of this phenotype, including its evolutionary origin, was provided some time ago [57] and had been discussed even earlier [162], the role of starvation in human evolution is not taken into consideration in analyses of the biology and clinical management of anorexia nervosa [7,8,130,163].

4.6. Eating behavior: the cause of mental change

The behavioral treatment for both anorexia and bulimia is based on the assumption that the psychiatric symptoms of eating disorders patients are the consequences of starvation and that normalization of eating behavior will reverse these emotional problems [15,57,133–135]. In support of this hypothesis, the psychiatric symptoms, including anxiety, depression and obsessions, normalize as eating behavior normalizes (Fig. 3) [138]. Indeed to be considered to be in remission with this treatment, there can be no persisting psychiatric symptoms [15,133]. It is important to note that patients diagnosed with bulimia nervosa starve themselves before bingeing and purging and that normalization of their eating behavior also reverses their symptoms [15,133]. This is in line with CBT for bulimia (see "5. CBT appears to act by normalizing eating behavior" below).

In further support of this hypothesis is evidence that healthy people develop all of the symptoms of eating disorders, including the mental symptoms, following starvation [57,159]. By contrast, the evidence supporting the hypothesis that a pre-existing mental condition is a cause of eating disorders [124] has been repeatedly falsified [15].

4.7. Linear eating: the cause of cognitive change

The suggestion of the cognitive behavioral theory that a cognitive process of "dietary restraint" stems directly from a "core psychopathology" [28] has been challenged experimentally. Instead, this abnormal cognitive status has been shown to be a consequence of the linearity of eating.

As the k-value of the curve of cumulative intake approaches 0, the value of dietary restraint increases in women. Thus, women with an average k = −1.8 had a score on a questionnaire for dietary restraint = 1.5 [164], while women with an average k = −0.3 had a significantly higher average score = 3 [165]. However, when women who were selected as linear eaters practiced eating assisted by mealtime feedback on the computer screen that was set to show an average of k = −1.7 over eight weeks, they ate with an average k = −1.5 when subsequently tested without feedback (Fig. 5). While the amount of food eaten did not change significantly, the initial rate of eating increased, as is normally the case among decelerated eaters (Fig. 5) [140,145–147]. As a consequence of the change in eating behavior, their score of dietary restraint

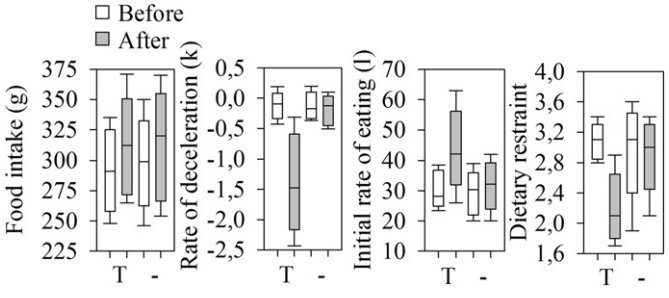

Fig. 5. Food intake, rate of deceleration (k), initial rate of eating (I), and dietary restraint in two groups of 17 women selected for eating at a linear rate. One group of women practiced eating at a decelerated rate, k = −1.7, over eight weeks of training (T), the other group did not practice (−). The women were tested before and after the training. Vales are box plots. Reproduced from [165] with permission.

decreased significantly (Fig. 5) [165]. Hence, rather than being a cause of eating behavior [166], the cognitive process of dietary restraint is a consequence of the pattern of eating in healthy women, just as anxiety, obsessions, and depression are consequences of the abnormal eating pattern in anorexia nervosa. Interestingly, a recent review of the effects of CBT makes the same point: "CBT attempts to eliminate dietary restraint in the early stages of treatment via the 'regular eating' strategy" [10]. Further support that fasting, rather than dietary restraint, is the causal factor in bulimia was presented by Stice et al. [167]. These results verify the hypothesis that dieting and the associated change in eating behavior, rather than cognitive processes, are important for the establishment of eating disorders.

Recent evidence provides additional support for this hypothesis. Thus, cue conditioned eating is not significantly affected by differences in emotions, including emotional eating and impulsivity [168,169]. Instead, it has been suggested that some of these emotions, including dietary restraint, are the consequence of previous failed attempts at weight control [170].

4.8. The effect of normalization of eating behavior

The effectiveness of the normalization of eating behavior has now been validated in the treatment of 571 patients with anorexia nervosa, 246 patients with bulimia nervosa, and 611 patients with an eating disorder not otherwise specified, i.e., for a total of 1428 patients [15]. Mortality was eliminated with this treatment, as were any lingering "comorbid" behavioral and physiological abnormalities in those patients who were in remission. Because psychoactive drugs, which may be successfully used to treat mental symptoms in patients with other disorders, have no effect on patients with eating disorders [6], these drugs are withdrawn as part of the treatment.

4.9. Summary

The outcomes of the normalization of eating behavior are encouraging in comparison with the outcomes of other treatments for bulimia and anorexia, such as CBT. We will end this review by suggesting that the outcome of CBT can be improved by shifting focus from cognitive to behavioral intervention. Evidence that this is the case was presented long ago.

5. CBT appears to act by normalizing eating behavior

A now forgotten RCT indicated that treating eating behavior is more effective than treating the cognitive, emotional issues associated with bulimia with CBT [171]. And it turns out that the positive impact CBT has on eating disorders may be due to the fact that this treatment also addresses the eating behavior of the patients. Thus, "*the prescription of a pattern of regular eating* (italics in original) *is probably the single most effective procedure in the treatment*" (italics added) [172]. The importance of "regular eating" is repeatedly stressed and "*in the great majority of cases [this] results in a marked reduction in the frequency of binge eating and purging and an associated decrease in the level of general psychiatric symptoms*" (italics added) [172]. In the more recent accounts of CBT, the importance of regular eating is pointed out once again [58,173].

5.1. Behavioral techniques are more effective than cognitive techniques

Rather than focusing on the cognitive aspects of CBT, it is possible that the BT used in the first evaluation of CBT should have been expanded [60,64]. Indeed evidence established in the forgotten RCT indicated that re-establishing a normal pattern of eating, using behavioral techniques, was more effective than cognitive techniques, focusing on the patients' dysfunctional beliefs, in reducing bulimic behavior, i.e., BT had a better effect than CBT [171]. For example, fewer of the women given BT dropped-out of treatment than those given CBT and they had

lower scores on body dissatisfaction and interpersonal distrust than the women given CBT. The BT also tended to modify symptoms earlier than CBT and there seemed to be no advantage in adding cognitive elements to their treatment. When the BT patients in this study began to get their bulimic behavior under control, they felt better, their self-esteem increased, and their distorted view of themselves in relation to food began to alter [171]. While these observations support the suggestion that cognitive changes occur as a consequence of a change in eating behavior rather than vice versa [171], the data were disregarded because no "satisfactory measure of the 'core psychopathology' of bulimia" was used [18]. This response is surprising, because outcomes of CBT could have been improved by the focusing on behavioral approaches. By contrast, expanding the concept of CBT to include other psychological dimensions (mood intolerance, perfectionism, interpersonal problems and low self-esteem) has not improved the outcomes of this approach [62,64,86].

Additional evidence against the possibility that cognitive training exerts an effect beyond the effect of behavior training was provided by a study on patients with anorexia nervosa. In this study, targeting dysfunctional thoughts associated with eating, body weight and size was not superior to a behavioral method by which the patients were gradually exposed to feared foods and situations [174]. There were no differences in outcome between these treatments and the authors suggested that the improvements simply reflected spontaneous remission rates, a possibility that was discussed above in the context of bulimia.

5.2. The CBT - BT paradox

The first stage of CBT aims at normalizing eating behavior in all eating disorders [58,172,173]. This is the BT part of CBT and one wonders why this intervention was effective in one study [171], but not in the other [64]. It is possible that the difference in the outcome of BT in the two studies was caused by differences in behavioral technique.

In CBT, a schedule with three meals and two snacks is introduced and the patients are asked to engage in activities that are incompatible with disordered eating to displace these undesirable behaviors [63,173]. The effective BT introduced the same meal schedule "… consisting of three meals a day with snacks between meals, with no self-induced vomiting, purgative abuse, or dieting. Only behavioural techniques were used, which included self monitoring, systematic modification of eating behaviour, using graded tasks, and the teaching of alternative coping strategies …" [171].

Although the BT of CBT leads to "… *an associated decrease in the level of general psychiatric symptoms*" (italics added) [172], patients are considered to be in need of cognitive therapy to regain self-esteem, overcome interpersonal problems, and solve problems in order to eventually be able to resume their normal social activities [172,173]. However, these interventions were not necessary to achieve cognitive change in the other study, targeting eating behavior alone was effective [171].

It appears that the two treatments used similar behavioral interventions. It is surprising, therefore, that while the outcome of CBT was similar in these two studies, the outcome of BT was better than that of CBT in one study [171] and worse in the other [64]. The information in these two publications does not permit a resolution of this apparent paradox.

However, the reports on the rapid weight increase by in-patient CBT treatment of patients with anorexia nervosa mentioned above [107,108] support the view that the effect was achieved by treating eating behavior. Thus: "The first week the energy intake is set at 1,500 kcal per day, and then it is increased at 2,000 kcal per day in the second week, and to 2,500 kcal per day in the third week. Subsequently, the energy intake is adjusted based on the body weight variations to maintain a weight gain comprised between 1 to 1.5 kg per week. Over the 2,500 kcal per day, patients have the option to use normal food or high-energy drinks" [175]. To comply with this schedule, the patients ate three meals, and even three snacks between meals, each day assisted by dieticians. By contrast, cognitive support was provided for one hour only and only during two days each week [58]. It is clear from this description, that treatment of eating behavior was the main intervention. The increase of BMI thus achieved was accompanied by a marked decrease in eating disorders symptoms and general psychopathology [107]. While this schedule normalized body weight from a relatively low initial level, the BMI decreased to about 18 within a year after treatment, despite the fact that most patients received further treatment during this year of follow-up [107,108]. These effects, which have been replicated [176,177], suggest that the treatment of eating behavior should be maintained as the patients lost weight after treatment.

5.3. The "cognitive" support of the treatment that normalizes eating behavior

We suggest that while the introduction of a variety of cognitive interventions has not improved the outcome of CBT, normalizing eating behavior is the main reason for the success of any treatment of eating disorders. As a matter of fact, "cognitive" support is also used in the treatment that aims at normalization of eating behavior, e.g., setting short-term social goals, modifying these goal as they are reached, and informing the patients that normal eating will facilitate reaching these goals [133]. However, these goals, including getting a hair-cut and starting school, are not specific for patients with eating disorders but are rather more like good advice for anyone in need of a haircut or schooling. Negotiating goal setting in CBT for eating disorders is done using the Socratic approach [30], i.e., eliminating contradictions [178], but it is difficult to question the advice, considering that the aims are self-evident, e.g., establishing a good relationship with the patient, her friends and relatives, informing her of the adverse effects of dieting and the physical consequences of starvation, improving her problem solving abilities, etc. [30,172]. No one would question such reasonable suggestions, which are more like common-sense than based on scientific considerations.

5.4. The role of patient characteristics

Differences in patient characteristics at admission may affect the outcomes of any treatment, although this is unlikely in the present case. For example, in a recent study on anorexia nervosa, the average BMI of 99 patients was 16.1 when CBT was initiated and it remained below normal after treatment [98]. Two other similar recent studies reported that the average BMI at admission of 242 and 284 patients, respectively, was about 16.7 and that these patients did not achieve a normal BMI after treatment [179,180]. By contrast, the average BMI of the 571 patients with anorexia nervosa who had their eating behavior normalized was 14.9 at admission and a normal BMI is a criterion for remission [15,133]. The treatment included 251 severely ill patients, diagnosed with either anorexia nervosa or bulimia nervosa, who were treated as in-patients because they had a BMI ≤ 13.5, and/or a body temperature of ≤36 °C, bradycardia (<40 bpm), prolonged QTc-time, at risk of cardiac arrhythmia, dehydration 5–10%, <90/60 mmHg (hypotension), hypokalemia (≤3.2 nmol/l), binge-eating, vomiting several times each day, or suicidal tendencies. Although these severely ill patients take longer to treat, they do go into remission [15]. It is therefore noteworthy that normalization of eating behavior is more effective than CBT for the treatment of eating disorders, even with severely affected patients. The recent successful normalization of body weight by in-patient treatment of patients with a BMI = 14.3 was also the result of the treatment of eating behavior [107,108].

5.5. A comparison between standard of care and the normalization of eating behavior

Just as the study demonstrating the superiority of behavioral techniques over cognitive techniques [171] has not been taken into

consideration in subsequent accounts, the more recent demonstration of the effectiveness normalization of eating behavior [15,133] is also at risk of being disregarded because of a study that aimed at comparing the effect of normalization of eating behavior with the effects of the standard of care for anorexia [181]. While that study claimed that the standard of care had a somewhat better outcome that normalization of eating behavior, the study had some limitations. Thus, the patients were not randomly assigned to the two treatments, inclusion and exclusion criteria were not made explicit, several patients assigned to standard care fulfilled remission criteria for body weight at admission and other patients dropped-out and/or were inappropriately excluded from the statistical analysis. Moreover, standard care was the first treatment for only a minority of the patients, most of them had already failed in the same treatment. Furthermore, it would be impossible to replicate the standard care, which included "psychomotor therapy", "creative therapy", and "additional treatments", which were "assigned to patients according to their needs" [181]. The effect of such treatments has never been established; these are treatments that rely on the "… behavioral intervention research that does not explicitly and directly relate to or integrate any basic science at all", mentioned in the introductory paragraph [12]. Yet, the authors reported that their standard care had an outcome that was twice better than they had themselves previously reported using the same treatment (reviewed in [15]).

5.6. A note on statistical methods

Another reason why the effect of normalization of eating behavior has been disregarded is because the RCT that demonstrated its effectiveness is thought to be a "small" RCT [181]. This argument is based on misunderstanding of the relationship between sample size and effect size. Thus, a power calculation, based on preliminary results [139] and estimates of the rate of drop-out, was made to determine the sample size needed. Ethical committees do not, and should not, permit studying more patients than needed. Because the size of the effect, 14 out of 16 treated patients went into remission, compared to one out of 16 patients who went into remission in the control group, normalization of eating behavior was demonstrated to be very effective [133]. As mentioned above ("4.2. Intervention: targeting eating behavior"), the study reported a 75% rate of remission and a 10% rate of relapse and no mortality [133]. As also mentioned above ("4.10. The effect of normalizing eating behavior"), these results have been obtained in 1428 patients [15]. The outcome of each individual patient was described at three months intervals in the study [15]. It is important to note that no patient was excluded from the analysis.

5.7. Summary

Normalization of eating behavior was found to be more effective than CBT in the treatment of bulimia nervosa in an RCT published 29 years ago [171] and this behavioral intervention is "probably the single most effective procedure" also in most versions of CBT [30,58,60]. However, rather than enhancing its behavioral methods, CBT has expanded its cognitive procedures without significantly improving their outcomes. By contrast, outcomes for eating disorders patients, even seriously affected ones, have been improved by treating eating behavior directly. The cognitive procedures that are part of CBT probably play a minor role in producing positive outcomes. Indeed, the cognitive therapy can be considered to be good advice similar to that given to patients whose eating behavior is being normalized.

6. Conclusions

Although the cognitive behavioral theory pioneered the development of the treatment of bulimia nervosa, it focused on normalizing cognitive and mental effects, which are more likely the consequences than the causes of eating disorders. This decision is probably the reason for the fact that treatment outcomes have not improved over the years and the positive effects that CBT can have is more reasonably attributed to the normalization of eating behavior that is part of the therapy. By contrast, a method that treats eating behavior directly has increased the rate of remission and decreased the rate of relapse across all eating disorders. Because of the marked difference in outcomes and because several hundred patients have been treated to remission and remained in remission over five years of follow-up, an RCT comparing CBT with normalization of eating behavior is redundant. While patient characteristics remain to be more carefully examined, results in the literature suggest that the difference in outcomes is caused by the differences in treatment. Needless to say, however, the scientific basis of this treatment needs to be validated and further developed and the method to teach patients how to eat needs to be optimized. It seems likely that if the method of normalizing eating behavior is added, not only to CBT, but to other standard treatments of eating disorders as well, outcomes will improve.

Acknowledgements

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