

New and emerging treatment options for adolescent bulimia nervosa

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While bulimia nervosa (BN) was first described more than 25 years ago, and a substantial body of literature has been accumulated describing both psychosocial and pharmacological treatments for adults with this disorder, little is known about effective interventions for adolescents with BN. The scope of this article is to review recent developments in the treatment of adolescent BN. We highlight our current knowledge of the epidemiology of this disorder among adolescents, emphasize the importance of early recognition of this disorder, and summarize the existing treatment literature for adolescent BN. The gaps in our knowledge regarding adolescents are accentuated by referring, in contrast, to the efficacious treatment for adults with BN. We conclude by underscoring the need to conduct large-scale randomized trials testing efficacious treatments for adolescent BN by borrowing from what is known in the treatment of adults with BN, as well as adolescents with anorexia nervosa.

Bulimia nervosa (BN) was first described by Gerald Russell more than 25 years ago [1]. Since then, a substantial body of literature has been established describing the treatment of adults with BN. By comparison, very little has been accomplished in terms of either the clinical description of adolescents with BN or how to treat adolescents with BN. The purpose of this article is to succinctly summarize what is known about the epidemiology of BN in adolescents, and to review new and emerging treatment options for this clinical population. As substantially more is known about treatment for adults with BN [2], we will make reference to this body of literature in order to put the state of our knowledge for adolescent BN in perspective.

Epidemiology of adolescent bulimia nervosa

The key characteristics of BN are binge-eating episodes, followed by inappropriate compensatory behaviors, for example, self-induced vomiting, laxative or diuretic misuse, fasting and excessive exercise. Feelings of loss of control, guilt and remorse usually accompany these episodes of overeating [3]. As is the case with anorexia nervosa (AN), patients with BN overvalue shape and weight, and often present with repeated attempts to lose weight [4,5]. Premenarchal onset of BN is relatively rare [6], while peak age of onset among adolescents is between 16 and 18 years [7–9], with a point prevalence of approximately 1–2% [10,11]. Partial BN cases are clinically common among

adolescents. Some studies have indicated that between 2 and 3% of adolescents present with significant bulimic symptoms, even though these do not meet full threshold criteria [10,11]. Moreover, a recent study demonstrated that almost half (48%) of the adolescent patients who presented with BN symptoms at a tertiary treatment program did not meet full criteria for BN [7]. The only difference between those who met full versus those who met partial criteria for BN was that patients with BN reported significantly higher frequencies of objectively large binge eating and purging.

BN among adolescents can be a source of psychiatric morbidity and result in impaired functioning in many areas of life. The most common psychiatric comorbidities include depression with suicidality, anxiety disorders and substance abuse [12,13]. While physical health is often maintained despite binge eating and purging behaviors, medical morbidities are not uncommon. These include, but are not limited to, complications such as electrolyte disturbances, parotid gland swelling, gastrointestinal (GI) symptoms such as GI perforation and loss of dental enamel [14,15].

Outcome among adolescents with BN remains unknown. However, among adults, approximately half of those with partial BN go on to develop a full syndrome disorder. For instance, Tozzi and colleagues found that among patients with an initial diagnosis of BN, 27% crossed over to AN within 5 years of their first diagnosis, posing additional medical and

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psychiatric risks [15]. In terms of prognosis for adolescent BN, we will have to turn to the adult data, as no information is available for the younger age group. Relatively little is known about the natural course of eating disorders among women in the community [16]. In a meta-analysis, Keel and Mitchell examined 88 outcome studies and demonstrated that 50% of patients achieved full recovery, while another 20% continued to meet diagnostic criteria for BN 5–10 years after the onset of the illness [17]. The remaining 30% of patients relapsed, in that their bulimic symptoms reappeared. While treatment appears to be effective in the short term, the long-term impact remains rather limited [18], and with substantial relapse rates in some studies, the enduring benefits of evidence-based treatments for adults with BN should still be viewed as tentative.

Detrimental impact of bulimia nervosa

Eating disorders, and BN is no exception, are associated with significant burdens on several domains, for example, personal, familial and societal. BN in adolescents, especially if it is untreated, may persist into adulthood [19–21]. The risk for the development of secondary physical and mental disorders increases with increased duration of illness [22]. In a longitudinal birth cohort study of 15 psychological disorders in young adults aged 21 years, Lewinsohn *et al.* found that BN was among the disorders with the highest levels of impairment [20]; 46% sought treatment, 23% attempted suicide and 23% reported use of psychotropic medication. The onset of illness was during adolescence for more than 90% of these cases. Similarly, Striegel-Moore and colleagues argue that psychosocial adjustment for young adult women who had BN as adolescents is compromised and associated with impairments at various levels, for example, health, self-image and social functioning [23]. Moreover, the burden associated with BN extends beyond the sufferer to the care provider, – that is, the parents or partners of patients. The care of an individual with BN is likely to be as challenging and stressful as it is for the carers of individuals with AN. In a large cohort of adolescents with BN, parents and partners report significant practical, emotional and interactional difficulties in relation to their young family member with BN [24,25]. Focusing on the mental health and caregiving experience of relatives of adolescents with BN, Winn and colleagues found that more than 50% reported mental health difficulties [26].

Early identification & early treatment

Physicians who see children and adolescents do not always consider a diagnosis of an eating disorder [27]. Consequently, there is often a delay from the time that a child or adolescent presents with clinically significant symptoms and is referred to a specialty clinic. The detrimental impact of such a delay on the outcome in AN has been argued quite persuasively [28], as it has been shown that there is little difference in severity between prepubertal and postpubertal onset AN [29]. In fact, Gowers *et al.* have argued that prepubertal onset AN may represent a particularly pernicious form of the disorder, heightening the argument for early intervention [30]. The same argument also applies to subthreshold presentations of AN. Partial AN can be associated with morbidities such as growth retardation, pubertal delay and deficiencies in bone mineral acquisition [31]. Psychological distress and impairment can often be as significant and equivalent to levels seen in AN [32].

Information about early-onset BN and subthreshold presentations of BN is not available to the same degree that it is for AN. BN may be more difficult to identify than early-onset AN as bulimic symptoms are easier to hide, especially given the guilt and shame associated with these symptoms. Notwithstanding, two recent cross-sectional studies of adolescent eating disorders have demonstrated that approximately half of the referrals to a specialist adolescent service are subthreshold cases of BN. The general and eating disorder psychopathology profiles of the subthreshold cases were similar to their full-syndrome BN counterparts [7,33]. In keeping with expectations, the difference between the two groups was limited to significantly higher frequencies of binge eating and purging in the full syndrome cases. This finding is somewhat misleading. While BN patients reported significantly more objectively large binge-eating episodes than subthreshold cases, the latter reported almost double the amount of subjectively large binge-eating episodes as compared with their BN counterparts. Consequently, the purge frequency for both groups matches the combined binge frequency (objective plus subjective binge episodes); this suggests that the salient binge feature may not be frequency but a sense of loss of control. The latter seems equally prevalent in these diagnostic groups. Eating psychopathology was also similar for BN and subthreshold cases, suggesting that distress and impairment might be equivalent between these two groups.

The uncertainty about eating disorder diagnoses has been addressed by the American Academy of Pediatrics. The Academy has provided diagnostic codes for clinically significant eating disorder presentations in children and adolescents that do not meet Diagnostic and Statistical Manual of Mental Disorders 4th Edition criteria [34]. The Academy has also issued a policy statement on the identification and treatment of eating disorders. This recent report urges pediatricians to be aware of the clinical and prognostic significance of eating disorder-not otherwise specified (ED-NOS) in adolescence [14]. While it seems appropriate not to differentiate between full syndrome and ED-NOS BN cases, it remains far from certain as to what the appropriate treatment for this clinical population should be. In the following section, we will attempt to review recent advances that have been made towards establishing efficacious treatments for adolescent BN.

As we have indicated in our review so far, adolescent BN has the potential for serious physical, emotional and social developmental consequences, including a chronic and severe course [35], and might constitute the majority of those presenting for treatment [36]. It is also clear that treatment is probably quite complex in that it requires attention to several key aspects of the disorder, such as psychiatric, medical, nutritional and developmental aspects [37]. By far the majority of treatment inquiry has focused on adults with BN. In fact, more than 70 controlled treatment trials have been conducted in adults with BN. By contrast, only two randomized trials for adolescents with BN have been concluded. An obvious dilemma is that findings from adult studies are not necessarily generalizable to children and adolescents. Consequently, little is known about efficacious treatments for adolescents with BN. Therefore, to provide a context for the limited adolescent data, our review will include the most seminal work among the treatment trials for adults with BN.

Treatments for adolescent bulimia nervosa

Clinicians have relatively few clinical treatment guidelines for eating disorders at their disposal. It was therefore particularly timely when the National Institute for Clinical Excellence (NICE) in the UK recently took the meaningful step to summarize guidelines for eating disorders based on a comprehensive review of the literature [38]. Upon completion of their review,

NICE recommended that treatment modalities be graded A–C. Grade A implies strong empirical support from several well-conducted randomized trials, while grade C implies expert consensus. By far the majority of the more than 100 recommendations that were made received only a grade C. The only treatment for adult BN to receive grade A was cognitive-behavioral therapy (CBT). CBT for adult BN has a relatively solid body of research demonstrating its superiority to any other psychological or pharmacological treatment. More specifically, the NICE guidelines recommend that the majority of adults with BN should receive 16–20 sessions of CBT. As an alternative, especially when patients do not respond to CBT, a course of interpersonal psychotherapy (IPT) may be considered. However, patients should be informed that this treatment might require more time in order to achieve similar outcomes as CBT. The fact that the NICE guidelines made no specific recommendation for the treatment of adolescents with BN reflects the fact that, to date, systematic research in the treatment of BN has focused almost exclusively on adults. This is true despite the fact that both binge eating and purging begin during adolescence, and that many cases of BN start in adolescence [39,40].

Significant progress has been made in understanding a range of efficacious treatments for adults with BN, including CBT, IPT and antidepressant medications. By contrast, other than case series data on CBT and two randomized, controlled trials (RCTs) that were recently completed [41,42], these treatments have not been studied with an adolescent population. The average age of participation in research studies of these treatment approaches was 28.4 years, the duration of the disorder approximately 10 years [43–45], and the cutoff age for entry 18 years. In the largest clinical trial of psychological treatments for BN to date ($n = 220$) [43], the mean age of participants was 28.1 years (standard deviation: 7.2) years. In the absence of substantial published data of established treatments for adolescent BN, the main focus of our review is on treatments for adults with BN [43,46–49].

Psychotherapies

Cognitive-behavioral therapy

The cognitive-behavioral model of BN assumes that the maintenance of the disorder is based on dysfunctional attitudes toward body shape and weight. These beliefs lead to overvalued ideas of

thinness and increased body dissatisfaction, and are typically followed by attempts to control shape and weight by excessive dieting. This excessive dieting causes a sense of both psychological and physiological deprivation, and sometimes increases depressed moods. In addition, because of dietary restriction, hunger is increased and this, in turn, leads to an increased probability of binge eating. Binge eating, because of the fears of weight gain as a result of eating a large amount of usually calorie-dense food, is eventually followed by purging as an attempt to allay these anxieties [50]. CBT has been tested in numerous controlled studies and has been found to be the most effective psychotherapeutic approach to the treatment of BN. CBT has been found to be more effective than other treatments, including no therapy, nondirective therapy, pill placebo, manualized psychodynamic therapy (supportive–expressive), stress management and antidepressant treatment [43,46–49]. Of those completing CBT, approximately 40% of patients with BN are abstinent, while another 20% are much improved. Fairburn and colleagues at the University of Oxford (Oxford, UK) followed bulimic patients treated with CBT for 5 years post-treatment [51]. Nearly 60% of patients studied had no eating disorder, and a further 20% had a subclinical disorder. The remainder were unrecovered, with a small percentage diagnosed as having AN. Hence, relapse rates for the successfully treated patients appear to be low, and the benefits are long-lasting. In a meta-analysis of nine double-blind, placebo-controlled medication trials (870 subjects) and 26 randomized psychosocial studies (460 subjects), CBT for BN was found to produce significantly larger-weighted pooled effect sizes for binge eating (95% CI: 1.09–1.47), purge frequency (95% CI: 1.06–1.39), depression (95% CI: 1.10–1.51) and eating attitudes (95% CI: 1.12–1.58) than comparison treatments [52], strongly supporting the view that CBT is the treatment of choice for BN in adults. In addition, a recent study directly evaluated the CBT model using structural equation modeling and found that the factors of self-esteem, overconcern with weight and shape, and dietary restraint accounted for 97% of the variance in outcome of binge eating and purging [53]. These results provide support for the CBT model of BN.

CBT has been compared with IPT, modified for BN [54], which focuses on the interpersonal context within which the eating disorder developed and is maintained, with the principal aim of

helping the patient make specific changes in identified ‘problem areas’. CBT was compared with IPT in a large multisite trial of adult patients with BN [43]. In this trial, CBT was superior to IPT at the end of treatment, but at 1 year follow-up, no differences were found between the two treatments. These studies suggested that BN was responsive to IPT as well as CBT, but that the improvements associated with IPT were slower to develop. IPT for adolescent BN is unexamined.

CBT for adolescent BN

There have been two published case series of adolescent subjects with BN treated with CBT designed for adolescent use (CBT-A), both at Stanford University in California, USA [55,56]. These modifications included:

- Increased contact between the therapist and the adolescent in early treatment to promote therapeutic alliance;
- Involvement of parents in supporting treatment;
- Use of concrete examples to illustrate points;
- Exploration of adolescent developmental issues (e.g., autonomy concerns) in the context of BN.

The results of these two case series provide preliminary evidence that CBT-A is acceptable and feasible as a treatment for adolescents with BN. In addition, there is one RCT that was conducted in London, UK, which compared cognitive–behavioral guided self-care (CBT-GSC) with family therapy [42]. CBT-GSC was manualized with an accompanying workbook for the patient. This treatment comprised of ten weekly sessions, three monthly follow-up sessions and two optional meetings with a close other. The initial focus of the treatment is on the function that bulimic symptoms may have on the individual’s life. Self-monitoring is introduced followed by problem-solving exercises with behavioral experimentation. As is the case in regular CBT, homework is a central part of this treatment. Results suggest that CBT-GSC was an acceptable and feasible treatment for adolescent BN, with a treatment drop-out rate of 29% and an abstinence rate (from both binge eating and purging) at 6-month follow-up of 36%. These abstinent rates were comparable with those found in more recent adult studies of CBT [2]. It is noteworthy that CBT-A used in the Stanford studies includes direct parental involvement, and is therefore a different model from the therapist-led guided self-care model that was studied by the London group. It is instead more similar to typical CBT used in treatment studies of adults with BN.

Family-based treatment

Family therapy was first implemented for adolescents with BN in a small case series [57]. This study demonstrated significant reductions in bulimic behaviors through educating the family about the eating disorder and helping the parents to disrupt binge-eating and purging episodes. More recently, le Grange and colleagues at the University of Chicago (IL, USA) [58] described the progression of an adolescent in family-based treatment for BN (FBT-BN). FBT-BN is derived from the approach found to be effective for adolescents with AN originally developed at the Maudsley Hospital in London [59–61], and subsequently manualized [62] and examined in treatment studies in the USA [63,64], although modified from the approach employed with adolescent AN, this treatment shares many characteristics with the original Maudsley family treatment model and has recently been manualized [65]. For example, FBT-BN assumes that the secrecy, shame and dysfunctional eating patterns associated with BN have negatively affected adolescent development in the bulimic patient and have confused and disempowered parents and other family members. In addition, parental guilt about having possibly caused the illness and anxiety about how best to proceed have further disabled them. FBT-BN is agnostic as to the cause of BN, but assumes that the usual progress through adolescence is negatively affected by the disorder. To target these problems, FBT-BN employs a three-stage treatment. In the first stage, treatment aims at empowering parents to disrupt binge eating, purging, restrictive dieting and any other pathologic weight-control behaviors. It also aims to externalize and separate the disordered behaviors from the affected adolescent to promote parental action and decrease adolescent resistance to their assistance. Once abstinence from disordered eating and related behaviors has been achieved, the second stage of treatment begins wherein parents transition control over eating and weight-related issues back to the adolescent under their supervision. The third stage is focused on the ways the family can help to address the effects of BN on adolescent developmental processes, both on the adolescent and the family as a whole.

Taken together, the FBT-BN approach differs from that in adolescent AN in some significant ways. First, the emphasis is not on weight restoration, but rather on regulating eating and curtailing purging. Second, whereas in AN the parents take charge of restoring the adolescent's

weight, treatment in BN is more collaborative between the adolescent and her/his parents. Third, the secretive nature of BN, as well as the guilt and shame that are caused by these symptoms, make it more probable that the illness, and its severity, can be overlooked by the parents. In AN, on the other hand, the patient's emaciated state is usually more obvious, and allows for the therapist to help the parents remain focused on the seriousness of her/his condition. Finally, treatment in BN is more often complicated by comorbid illnesses, which are probably more prevalent in BN than in AN.

The first two RCTs for adolescents with BN and partial BN have recently been concluded. In the first of these, and as alluded to earlier in this review, the Maudsley group in London compared family therapy with CBT-GSC among participants aged 12–20 years (mean: 7.6 years; standard deviation: 0.3) [41]. At 6-month follow-up, no statistical differences on abstinence rates were found between family therapy and individual CBT-GSC.

At around the same time, a controlled study was conducted by a group at the University of Chicago. Participants, aged 12–19 years (mean: 16.1 years; standard deviation: 1.6), were allocated to either manualized FBT-BN; or individual supportive psychotherapy (SPT) [42]. At post-treatment and 6-month follow-up, significantly more patients in FBT-BN were binge/purge abstinent compared with SPT. Family therapy as described by the London group resembles FBT-BN, however, one key difference between FBT-BN and family therapy is that 'family' is defined as any 'close other', rather than just a parent. This definition of family occurred in approximately a quarter of cases, and was likely utilized, in part, because the mean age of the subjects in this study was almost 18 years. This is close to adulthood, especially in the UK, where the age of consent is 16 years. While defining family as a close other may fit well with this older age group, this might not be the most effective way to approach FBT-BN with younger adolescents, where parental authority is key to the success of FBT-BN. This point is emphasized further by the rate of treatment uptake for the UK study, wherein 25% of eligible participants refused the study because they did not want their families involved in treatment [24]. By contrast, in the younger adolescents studied by the Chicago group, 11% dropped out of treatment and none of these reported involving the family as the

reason for discontinuing treatment [42]. Nonetheless, the abstinence rates (41%) for family therapy in the Schmidt *et al.* [41] study were comparable with those achieved during FBT-BN in the Chicago study. Definition of abstinence was equally strictly defined for both these studies, that is, no binge eating and purging for the 4 weeks preceding the assessment.

Schmidt and colleagues acknowledge that a limitation of their study was the sample size, which was likely too small to detect differences between two active treatments for some of their outcomes [41]. In addition, the authors acknowledge that the absence of a waiting list or attention placebo-control group prevent them from ruling out that improvement was simply due to passage of time or nonspecific effects. The Chicago study, which had a similar overall sample size to the London study, was adequately powered to demonstrate the potential benefits of an active treatment (i.e., FBT-BN) over a non-specific control treatment (i.e., SPT), and to demonstrate comparable benefits that were not due to time effects.

Pharmacotherapy

Antidepressant medications in the treatment of BN have received intense research attention in adults. A series of double-blind, placebo-controlled trials of antidepressant medications among adults with BN have been conducted [44,66–68]. In almost all of these controlled trials, most types of antidepressants have proven superior to placebo in reducing binge frequency. Generally, mood disturbance and preoccupation

with shape and weight also show greater improvement with medication than with placebo [69]. Several controlled studies have directly evaluated the relative and combined effectiveness of CBT and antidepressant drug treatment [44,68–70]. Although antidepressant medications have been shown to be more effective than a placebo in reducing symptoms of BN [48,50], when added to psychological treatments (e.g., CBT or IPT), medications did not improve the outcomes of core eating-related symptoms. One small open-label study of ten adolescents, aged 12–18 years, found that 8 weeks of fluoxetine at 60 mg/day was well tolerated in the context of supportive psychotherapy. While the medication was well tolerated and the findings were encouraging (approximately 70% were rated as either much improved or improved), the effectiveness of this medication in this age group is still unknown [71]. A familiar dilemma is that we are bound to extrapolate from adult data to adolescents, who may in fact have a different clinical presentation [72]. Taken together, the data suggests that the use of antidepressants in adults with BN, although useful, offers only a marginal advantage over CBT alone. The available treatment studies for adolescent BN are summarized in Table 1.

Conclusion

There are no evidence-based treatments for adolescent BN. Two emerging treatments for this condition are family treatment (family therapy or FBT-BN), refined from the adolescent AN treatment model of the same name,

Table 1. Psychosocial and medication studies for adolescent bulimia nervosa.					
Study	Study type	n	Age* (years)	Type of treatment	Ref.
Dodge <i>et al.</i> (1995)	Case series	8	16.5 (1.2)	FT	[57]
le Grange <i>et al.</i> (2003)	Case study	1	17	FBT-BN	[58]
Kotler <i>et al.</i> (2003)	Open trial	10	12–18	Fluoxetine 60 mg	[71]
Lock (2005)	Case series	34	15.8	CBT-A	[63]
Schapman <i>et al.</i> (2006)	Case series	7	16.3 (1.3)	CBT-A	[54]
Schmidt <i>et al.</i> (2007)	RCT	85	17.6 (0.3)	FT vs CBT-GSC	[41]
Le Grange <i>et al.</i> (2007)	RCT	80	16.1 (1.6)	FBT-BN vs SPT	

*Mean (standard deviation), actual years, age range or mean age with no SD.
CBT-A: Cognitive-behavioral therapy for adolescents; CBT-GSC: Cognitive-behavioral guided self-care; FT: Family therapy; FBT-BN: Family-based treatment for bulimia nervosa; RCT: Randomized, controlled trial; SPT: Supportive psychotherapy.

and CBT (self-guided or adjusted for an adolescent age group, which includes a parental component, i.e., CBT-A). These treatments share a developmental perspective on the needs of adolescents. For instance, in FBT-BN, treatment is modified to account for the usually older age of adolescents and the egodystonic nature of BN, characteristics that typically differentiate adolescents with BN from those with AN. In CBT-A, adjustments are made to account for variation in motivation, cognitive ability and special needs for therapeutic alliance building. Little evidence suggests that existing medications will play a primary role in the treatment of adolescents with BN, especially given concerns about impulsivity or other forms of self-harm in this age group [73], and the potential for increased suicidal ideation when treated with selective serotonin re-uptake inhibitors.

The need to develop an evidence base for treatments for adolescents with BN is clear, given the medical, psychological, social and societal costs of this disorder over the lifetime of a person who develops the disorder early in life.

In the near future, more definitive studies of the most promising treatments described here (FBT-BN and CBT-A) are needed as a first step in advancing treatments for adolescent BN.

Future perspective

A large RCT will establish the treatment of choice for both younger and older adolescents with BN. In addition, the first combination trial of fluoxetine and psychotherapy might indicate whether this selective serotonin re-uptake inhibitor is as helpful in the treatment of adolescents with BN as it has been in the treatment for adults with BN.

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Executive summary

- Treatment studies for adolescent bulimia nervosa (BN) are limited, with no established evidence base for this disorder in younger patients.
- Applying the findings from the substantial evidence base for adults with BN to a younger patient population is inappropriate.
- Family-based treatment and cognitive-behavioral therapy are promising treatments for adolescent BN.
- The role of antidepressant medication in the treatment of adolescent BN is unknown.
- Determining the relative efficacy of family-based treatment for BN and cognitive-behavioral therapy for adolescents is a priority.
- The American Psychiatric Association offers a thorough review of the treatment guidelines for eating disorders at [101].

Bibliography

1. Russell GFM: Bulimia nervosa: An ominous variant of anorexia nervosa. *Psychol. Med.* 9, 429–448 (1979).
2. Mitchell J, Agras WS, Wonderlich S: Treatment of bulimia nervosa: where are we and where are we going. *Int. J. Eat. Disord.* 39, 95–101 (2006).
3. American Psychiatric Association: *Diagnostic and Statistical Manual of Mental Disorders (4th Edition)*. American Psychiatric Association, Washington, USA (2000).
4. Fairburn CG, Harrison PJ: Eating disorders. *Lancet* 361, 407–416 (2003).
5. Steiger H, Séguin JR: Eating disorders: anorexia nervosa and bulimia nervosa. In: *Oxford Textbook of Psychopathology*. Millon T, Blaney PH (Eds). Oxford University Press, NY, USA, 365–389 (1999).
6. Kent A, Lacey H, McClusky SE: Pre-menarchal bulimia nervosa. *J. Psychosom. Res.* 36, 205–210 (1992).
7. le Grange D, Loeb KL, Orman S, Jellar C: Bulimia nervosa: a disorder in evolution? *Arch. Ped. Adolesc. Med.* 158, 478–482 (2004).
8. Fairburn CG, Cooper Z, Doll H *et al.*: The natural course of bulimia nervosa and binge eating disorder in young women. *Arch. Gen. Psychiatry* 57, 659–665 (2000).
9. Flament M, Ledoux S, Jeammet P *et al.*: A population study of bulimia nervosa and subclinical eating disorders in adolescence. In: *Eating Disorders in Adolescence: Anorexia and Bulimia Nervosa*. Steinhausen H (Ed.). Brunner/Mazel, NY, USA, 21–36 (1995).
10. Kotler LA, Walsh BT: Eating disorders in children and adolescents: pharmacological therapies. *Eur. Child and Adolesc. Psychiatry* 9, 108–116 (2000).
11. van Hoeken D, Seidell J, Hoek HW: Epidemiology. In: *Handbook of Eating Disorders, (2nd Edition)*. Treasure J, Schmidt U, van Furth E (Eds). John Wiley & Sons, Chichester, USA, 11–34 (2004).
12. Herzog DB, Keller MB, Sacks NR *et al.*: Psychiatric comorbidity in treatment-seeking anorexics and bulimics. *J. Am. Acad. Child Adolesc. Psychiatry* 31(5), 810–818 (1992).
13. Herzog DB, Nussbaum KM, Marmor AK: Comorbidity and outcome in eating disorders. *Psychiatr. Clin. North Am.* 19(4), 843–859 (1996).

14. Golden NH, Katzman DK, Kreipe RE *et al.*: Eating disorders in adolescents: position paper of the Society for Adolescent Medicine. *J. Adolesc. Health* 33, 496–503 (2003).
15. Tozzi F, Thornton LM, Klump KL *et al.*: Symptom fluctuation in eating disorders: correlate of diagnostic crossover. *Am. J. Psychiatry* 162, 732–740 (2005).
16. Newman DL, Moffitt TE, Caspi A *et al.*: Psychiatric disorder in a birth cohort of young adults: prevalence, comorbidity, clinical significance, and new case incidence from ages 11 to 21. *J. Consult. Clin. Psychol.* 64, 552–562 (1996).
17. Keel PK, Mitchell JE: Outcome in bulimia nervosa. *Am. J. Psychiatry* 154, 313–321 (1997).
18. Klein DA, Walsh BT: Eating disorders. *Int. Rev. Psychiatry* 15, 205–216 (2003).
19. Striegel-Moore RH, Leslie D, Petrelli SA *et al.*: One-year use and cost of inpatient and outpatient services among female and male patients with an eating disorder: evidence from a national database of health insurance claims. *Int. J. Eat. Disord.* 27, 381–389 (2000).
20. Lewinsohn PM, Striegel-Moore, Seeley JR: Epidemiology and natural course of eating disorders in young women from adolescence to young adulthood. *J. Am. Acad. Child Adolesc. Psychiatry* 39, 1284–1292 (2000).
21. Kotler L, Cohen P, Davies M *et al.*: Longitudinal relationships between childhood, adolescent and adult eating disorders. *J. Am. Acad. Child Adolesc. Psychiatry* 40, 1434–1440 (2001).
22. Johnson JG, Cohen P, Kotler L, Kasen S, Brook JS: Psychiatric disorders associated with risk for the development of eating disorders during adolescence and early adulthood. *J. Consult. Clin. Psychol.* 70, 1119–1128 (2002).
23. Striegel-Moore RH, Seeley JR, Lewinsohn PM: Psychosocial adjustment in young adulthood of women who experienced an eating disorder during adolescence. *Am. Acad. Child Adolesc. Psychiatry* 42, 587–593 (2003).
24. Perkins S, Winn S, Murray J, Murphy R, Schmidt U: A qualitative study of the experience of caring for a person with bulimia nervosa. Part 1: The emotional impact of caring. *Int. J. Eat. Disord.* 36, 256–268 (2004).
25. Winn S, Perkins S, Murray J, Murphy R, Schmidt U: A qualitative study of the experience of caring for a person with bulimia nervosa. Part 2: Carers' needs and experiences of services and other support. *Int. J. Eat. Disord.* 36, 269–279 (2004).
26. Winn S, Perkins S, Walwyn R *et al.*: Predictors of mental health problems and negative caregiving experiences in careers of individuals with bulimia nervosa. *Int. J. Eat. Disord.* 40, 171–178 (2007).
27. Bryant-Waugh RJ, Lask BD, Shafran RL, Fossion AR: Do doctors recognise eating disorders in children? *Arch. Dis. Child.* 67, 103–105 (1992).
28. le Grange D, Loeb KL: Early identification and treatment of eating disorders: prodrome to syndrome. *Early Interv. Psychiatry* 1, 27–39 (2007).
29. Cooper PJ, Watkins B, Bryant-Waugh R, Lask B: The nosological status of early onset anorexia nervosa. *Psychol. Med.* 32, 873–880 (2002).
30. Gowers SG, Crisp AH, Joughin N, Bhat A: Premenarcheal anorexia nervosa. *J. Child Psychol. Psychiatry* 32, 515–524 (1991).
31. Bachrach LK, Gudo D, Katzman DK: Decreased bone density in adolescent girls with anorexia nervosa. *Pediatrics* 86, 440–447 (1990).
32. Bunnell DW, Shenker IR, Nussbaum MP, Jacobson MS: Subclinical versus formal eating disorders: differentiating psychological features. *Int. J. Eat. Disord.* 9, 357–362 (1990).
33. Binford R, le Grange D: Adolescents with bulimia nervosa and eating disorder not otherwise specified-purging only. *Int. J. Eat. Disord.* 38, 157–161 (2005).
34. Wolraich ML, Felice ME, Drotar D: *The Classification of Child and Adolescent Mental Diagnoses in Primary Care: Diagnostic and Statistical Manual for Primary Care (DSM-PC) Child and Adolescent Version*. American Academy of Pediatrics, IL, USA (1996).
35. Steiner H, Lock J: Anorexia and bulimia nervosa in children and adolescents: a review of the past 10 years. *Child Adolesc. Psychiatry* 37, 352–359 (1998).
36. Currin L, Schmidt U, Jick H, Treasure J: Time trends in the incidence of bulimia nervosa. *Br. J. Psychiatry* 186, 132–135 (2005).
37. Yager J, Andersen A, Devlin M, Mitchell J, Powers P, Yates A: American Psychiatric Association practice guidelines for eating disorders. *Am. J. Psychiatry* 15, 201–228 (1993).
38. National Collaborating Centre for Mental Health: *Eating Disorders: Core Interventions in the Treatment and Management of Anorexia Nervosa, Bulimia Nervosa and Related Eating Disorders*. British Psychological Society and Gaskell, Leicester & London, UK (2004).
39. Lock J, Reisel B, Steiner H: Associated health risks of adolescents with disordered eating: how different are they from their peers? Results from a high school survey. *Child Psychiatry Hum. Dev.* 31, 249–265 (2001).
40. Stice E, Agras WS: Predicting onset and cessation of bulimic behaviors during adolescence. *Behav. Ther.* 29, 257–276 (1998).
41. Schmidt U, Lee S, Beecham J *et al.*: A randomized controlled trial of family therapy and cognitive behavioral guided self-help for adolescents with bulimia nervosa and related conditions. *Am. J. Psychiatry* 164, 591–598 (2007).
42. le Grange D, Crosby R, Rathouz P, Leventhal B: A randomized controlled comparison of family-based treatment and supportive psychotherapy for adolescent bulimia nervosa. *Arch. Gen. Psychiatry* 64, 1049–1056 (2007).
43. Agras WS, Walsh BT, Fairburn CG *et al.*: A multicenter comparison of cognitive-behavioral therapy and interpersonal psychotherapy for bulimia nervosa. *Arch. Gen. Psychiatry* 57, 459–466 (2000).
44. Agras WS, Rossiter EM, Arnow B *et al.*: Pharmacologic and cognitive-behavioral treatment for bulimia nervosa: a controlled comparison. *Am. J. Psychiatry* 149, 82–87 (1992).
45. Wilson GT, Eldredge KL, Smith D, Niles B: Cognitive-behavioral treatment with and without response prevention for bulimia. *Behav. Res. Therapy* 29, 575–583 (1991).
46. Agras WS, Telch CF, Arnow B *et al.*: One-year follow-up of cognitive-behavioral therapy for obese individuals with binge eating disorder. *J. Cons. Clin. Psychology* 65, 343–347 (1997).
47. Fairburn CG, Jones R, Peveler RC *et al.*: Psychotherapy and bulimia nervosa. Longer-term effects of interpersonal psychotherapy, behavior therapy, and cognitive behavior therapy. *Arch. Gen. Psychiatry* 50, 419–428 (1993).
48. Pope HG, Hudson JL, Jonas JM, Yurgelin-Todd D: Bulimia treated with imipramine: a placebo-controlled, double-blind study. *Am. J. Psychiatry* 140, 554–558 (1993).
49. Wilfley DE, Agras WS, Telch CF *et al.*: Group cognitive-behavioral therapy and group interpersonal psychotherapy for the nonpurging bulimic: a controlled comparison. *J. Cons. Clin. Psychology* 61, 296–305 (1993).

50. Apple RE, Agras WS: *Overcoming Eating Disorders: A Cognitive-Behavioral Treatment for Bulimia Nervosa and Binge Eating Disorder. Client Workbook*. The Psychological Corporation, TX, USA (1997).
51. Fairburn CG, Norman PA, Welch SL *et al.*: A prospective study of outcome in bulimia nervosa and the long-term effects of three psychological treatments. *Arch. Gen. Psychiatry* 52, 304–312 (1995).
52. Whittal ML, Agras WS, Gould RA: Bulimia nervosa: a meta-analysis of psychosocial and pharmacological treatments. *Behav. Ther.* 30, 117–135 (1999).
53. Byrne S, McLean N: The cognitive-behavioral model of bulimia nervosa: a direct evaluation. *Int. J. Eat. Disord.* 31, 17–31 (2002).
54. Lock J: Adjusting cognitive behavioral therapy for adolescent bulimia nervosa: results of a case series. *Am. J. Psychother.* 59, 267–281 (2005).
55. Fairburn CG: Interpersonal psychotherapy for bulimia nervosa. In: *Handbook of Treatment for Eating Disorders (2nd Edition)*. Garner DM, Garfinkel P (Eds). Guilford Press, NY, USA (1997).
56. Schapman A, Lock J, Couturier J: Cognitive-behavioral therapy for adolescents with binge eating syndromes: a case series. *Int. J. Eat. Disord.* 39, 252–255 (2006).
57. Dodge E, Hodes M, Eisler I, Dare C: Family therapy for bulimia nervosa in adolescents: an exploratory study. *Am. J. Fam. Ther.* 17, 59–77 (1995).
58. le Grange D, Lock J, Dymek M: Family-based therapy for adolescent with bulimia nervosa. *Am. J. Psychother.* 67, 237–251 (2003).
59. Russell GF, Szmukler GI, Dare C, Eisler I: An evaluation of family therapy in anorexia nervosa and bulimia nervosa. *Arch. Gen. Psychiatry* 44, 1047–1056 (1987).
60. Eisler I, Dare C, Hodes M *et al.*: Family therapy for adolescent anorexia nervosa: the results of a controlled comparison of two family interventions. *J. Child Psychol. Psychiatry* 41, 727–736 (2000).
61. le Grange D, Eisler I, Dare C, Russell G: Evaluation of family treatments in adolescent anorexia nervosa: a pilot study. *Int. J. Eat. Disord.* 12(4), 347–357 (1992).
62. Lock J, le Grange D, Agras WS, Dare C: *Treatment Manual For Anorexia Nervosa: A Family-based Approach*. Guilford Press, NY, USA (2001).
63. Lock J, Agras WS, Bryson S, Kraemer H: A comparison of short- and long-term family therapy for adolescent anorexia nervosa. *J. Am. Acad. Child Adolesc. Psychiatry* 44, 632–639 (2005).
64. le Grange D, Binford R, Loeb KL: Manualized family-based treatment for anorexia nervosa: a case series. *J. Am. Acad. Child Adolesc. Psychiatry* 44, 41–46 (2005).
65. le Grange D, Lock J: *Treating Bulimia in Adolescents: A Family-Based Approach*. Guilford Press, NY, USA (2007).
66. Agras WS, McCann U: The efficacy and role of antidepressants in the treatment of bulimia. *Ann. Behav. Med.* 9, 18–22 (1987).
67. Walsh BT, Hadigan CM, Devlin MJ *et al.*: Long-term outcome of antidepressant treatment for bulimia nervosa. *Am. J. Psychiatry* 148(9), 1206–1212 (1991).
68. Walsh BT, Wilson GT, Loeb KL *et al.*: Medication and psychotherapy in the treatment of bulimia nervosa. *Am. J. Psychiatry* 154, 523–531 (1997).
69. Mitchell JE, Raymond N, Specker S: A review of the controlled trials of pharmacotherapy and psychotherapy in the treatment of bulimia nervosa. *Int. J. Eat. Disord.* 14(3), 229–247 (1993).
70. Leitenberg H, Rosen JC, Wolf J *et al.*: Comparison of cognitive-behavior therapy and desipramine in the treatment of bulimia nervosa. *Behav. Res. Therapy* 32(1), 37–45 (1994).
71. Kotler L, Devlin B, Davies M, Walsh BT: An open trial of fluoxetine in adolescents with bulimia nervosa. *J. Child Adolesc. Psychopharmacology* 13, 329–325 (2003).
72. le Grange D, Schmidt U: The treatment of adolescents with bulimia nervosa. *J. Mental Health* 14, 587–597 (2005).
73. Fischer S, le Grange D: Co-morbidity and high-risk behaviors in treatment seeking adolescents with bulimia nervosa. *Int. J. Eat. Disord.* (2007) (In Press).

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