ARTICLE

Bulimia Nervosa in Adolescents

A Disorder in Evolution?

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Background: There are few reports that describe the manifestation of bulimia nervosa (BN) among adolescents. Moreover, none make reference to the comparative clinical manifestation of adolescent BN and adolescent anorexia nervosa. Nor are any reports available of how distinct partial-syndrome BN cases are from those that meet full diagnostic criteria for BN.

Objectives: To describe 3 groups of adolescents, those with a full-syndrome eating disorder (BN and anorexia nervosa) or partial-syndrome BN, and to compare these groups along demographic, general psychopathology, and eating disorder variables.

Design: The study population included 120 adolescents with eating disorders who were initially seen at The University of Chicago Eating Disorders Program, Chicago, Ill, for treatment. All participants completed an assessment prior to treatment.

Measures: Weight and height were obtained from all participants. Participants also completed a baseline demographic questionnaire (eg, menstrual status, ethnicity, family status), Beck Depression Inventory, Rosenberg Self-Esteem Scale, and the Eating Disorder Examination.

Results: Partial-syndrome BN cases are clinically quite similar to their full-syndrome counterparts. Only objective binge eating episodes and purge frequency distinguished BN and partial-syndrome BN cases. Anorexia nervosa cases, on the other hand, were quite distinct from BN and partial-syndrome BN cases on almost all variables.

Conclusion: Early recognition and swift treatment of eating disorders in adolescents, regardless of whether a diagnostic threshold is met, are imperative because they will lead to early intervention thereby potentially improving eating disorder recovery rates.

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BULIMIA NERVOSA (BN) AFFECTS as many as 3% of young women[1] and usually arises in adolescence with the peak age of onset reported to occur between 15.7 years[2] and 18.1 years.[3] Key features are binge eating followed by inappropriate compensatory behaviors such as self-induced vomiting, laxative or diuretic misuse, fasting, and excessive exercise. Episodes of overeating are accompanied by feelings of loss of control, guilt, and remorse. Patients overvalue shape and weight and, as in anorexia nervosa (AN), often make repeated attempts to lose weight.[4,5]

Recent reports have described alarmingly high numbers of adolescents with BN.[6,7] The relative frequency of premenarchal BN in children is particularly disconcerting.[8] Partial-syndrome eating disorders are even more common. Early studies of dieting and binge eating behaviors in community samples have shown that 10% to 50% of adolescent girls and boys frequently engage in binge eating behavior.[9,10] Applying stringent diagnostic criteria to community samples, studies have found that only 1% to 5% of adolescent girls surveyed qualify for a diagnosis of BN.[11] This prompts the argument that children and adolescents may have partial syndromes that do not fall as neatly into extant *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition* (*DSM-IV*) eating disorder categories as adults.[12] This poses a diagnostic dilemma for conducting treatment outcome investigations in this younger age population especially because partial-syndrome cases may go on to develop the full syndrome in time.

Research comparing the demographics of adolescent BN to adult BN has been limited. Recent findings suggest that both patient populations are represented across racial and socioeconomic groups.[13] While

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boys represent approximately one fifth of adolescents with BN,[14] about one tenth of adults with BN are men.[5]

There are also few reports available specifically describing the clinical manifestation of BN among adolescents. Full-syndromal BN is more common among young adults than among adolescents.[12] A recent comparison of adolescents and young adults first seen for eating disorder treatment revealed that adults report more binge eating and laxative use than their adolescent counterparts.[15] There are no published reports available examining how distinct partial-syndrome BN (PBN) cases might be from those that meet full diagnostic criteria. In addition, no reports make specific reference to how the clinical manifestation of BN in adolescents compares with that of adolescents with AN.

Therefore, the purpose of the present investigation was to describe and compare the demographic and clinical manifestations of adolescents with BN, PBN, and AN. This description could better inform the physician in making diagnostic and treatment decisions.

METHODS

PARTICIPANTS

Participants were selected from 142 consecutive adolescent referrals to an eating disorder research–based treatment service at The University of Chicago, Chicago, Ill. Of these 142 patients, 42 (29.6%) met interview-based *DSM-IV*[16] criteria for AN, 38 (26.8%) met *DSM-IV* criteria for BN, and 62 (43.7%) were eating disorder not otherwise specified (EDNOS). Of the EDNOS cases, 40 met study criteria for PBN (ie, not meeting full *DSM-IV* criteria for BN [which requires binge eating and purging twice per week for 3 months], but binge eating or purging at a frequency of at least once per week for 6 months). The remaining 22 EDNOS cases were excluded (15 with partial AN, 4 with PBN who did not satisfy the above-mentioned criteria, and 3 with binge eating disorder). Appropriate institutional review board approval for this project was obtained and corresponding consent procedures followed.

MEASURES

Each participant completed a comprehensive assessment battery at the time of initial examination and was offered a course of treatment on completing this evaluation.

Demographic Variables

These variables include weight, height, duration of illness, race/ethnicity (white, African American, Hispanic, and Asian), and family status (intact, divorced, single, reconstituted). Menstrual status was categorized in 3 groups: absent (primary or secondary amenorrhea), irregular (1-2 cycles in the past 3 months), or regular (3 cycles in the past 3 months).

General Psychopathology

Depression was assessed with the Beck Depression Inventory,[17] a 21-question scale that has been used in numerous studies of adolescent depression.[18,19] Self-esteem was assessed with the Rosenberg Self-Esteem Scale,[20] a widely used self-report instrument of 10 items. The Rosenberg Self-Esteem Scale has been used in previous BN treatment studies and has been found to be a robust predictor of outcome.[21]

Table 1. Patient Characteristics for Bulimia Nervosa (BN), Partial-Syndrome Bulimia Nervosa (PBN), and Anorexia Nervosa (AN)*

	BN (n = 38)	PBN (n = 40)	AN (n = 42)	F Value	*P* Value
Age, y	16.6 (1.5)	15.8 (1.8)	14.8 (2.6)	8.75	<.001
Duration ill, mo	22.5 (19.5)	20.0 (15.8)	15.1 (12.8)	2.23	.11
Weight, kg (BMI)	23.4 (4.1)	21.9 (2.6)	16.3 (1.7)	65.18	<.001

Abbreviation: BMI, body mass index (calculated as the weight in kilograms divided by the height in meters squared).
*Values are expressed as mean (SD) unless otherwise indicated.

Eating Disorder Psychopathology

Participants completed the Eating Disorder Examination (EDE) (C. G. Fairburn, MD, and Z. Cooper, PhD, unpublished data, 2000), a standardized, psychometrically sound, well-tested semistructured clinical interview that measures the severity of the characteristic psychopathology of eating disorders and generates operational (*DSM-IV*–based) definitions of eating disorder diagnoses.[22] Recently, the EDE has been used with adolescents.[23]

With the EDE, the trained interviewer assesses the severity of psychopathology along 4 subscales: restraint, eating concern, shape concern, and weight concern. In addition, the EDE assesses the frequency of key eating disorder behaviors including objective bulimic episodes (OBEs) (These are defined as eating an unusually large amount of food in a circumscribed period with an accompanying feeling of loss of control over eating. The interviewer determines whether an eating episode is objectively large using established guidelines [C. G. Fairburn, MD, and Z. Cooper, PhD, unpublished data, 2000] that incorporate the amount eaten given the context and what others in a similar situation [eg, holidays] would consume. If the amount presented in the interview is ambiguous, a consensus may be reached among trained interviewers to determine whether it meets operational criteria for "large".); subjective bulimic episodes (SBEs) (These are defined as not objectively large in terms of food quantity as would be defined for OBE but characterized by patients thinking that they have overeaten accompanied by feeling out of control.); and purging (eg, self-induced vomiting). The OBE is equivalent to the *DSM-IV* definition of a binge episode in the diagnostic criteria for BN. The EDE was primarily used to generate the diagnoses of AN, BN, and PBN.

DATA ANALYSIS

Differences in categorical demographic variables were examined using χ^2 statistics. Race/ethnicity and family status were each collapsed into 2 groups (eg, non-Hispanic white vs nonwhite and intact family vs nonintact family). Continuous variables were examined using a series of 1-way analyses of variance. To control for type I error in multiple comparisons, a Bonferroni correction was applied to this set of analyses.

RESULTS

DEMOGRAPHIC VARIABLES

The mean (SD) age of the participants (N=120) was 15.7 (2.2) years (range, 9-19) and the mean (SD) illness duration was 19.1 (16.3) months (range, 2-78). The majority of the sample was female (113 [94.2%]), non-Hispanic white (91 [75.8%]), and from intact families (87 [72.9%]). Results are summarized in **Table 1**. Post hoc

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Table 2. General Psychopathology for Patients With Bulimia Nervosa (BN), Partial-Syndrome Bulimia Nervosa (PBN), and Anorexia Nervosa (AN)*

	BN (n = 33)	PBN (n = 38)	AN (n = 28)	F Value	*P* Value
RSE score†	27.5 (5.5)	25.8 (8.1)	20.1 (6.2)	9.49	<.001
BDI score†	24.1 (10.0)	24.4 (16.2)	10.4 (8.1)	12.20	<.001

Abbreviations: BDI, Beck Depression Inventory; RSE, Rosenberg Self-Esteem Scale.

*Values are expressed as mean (SD) unless otherwise indicated.

†Higher RSE scores indicate lower self-esteem. Higher BDI scores indicate greater degree of depression.

Table 3. Eating Disorder Examination (EDE) Symptoms*

	BN (n = 30)	PBN (n = 39)	AN (n = 27)	F Value	*P* Value
OBE frequency†	31.4 (23.6)	3.5 (5.1)	0.7 (3.5)	46.49	<.001
SBE frequency‡	11.1 (18.7)	21.1 (28.1)	1.7 (4.5)	6.97	.002
Vomit frequency	45.5 (30.4)	17.8 (28.1)	0.6 (2.3)	24.18	<.001
Restraint	3.87 (1.1)	3.91 (1.6)	2.02 (1.7)	16.12	<.001
Weight concern	4.24 (1.4)	3.48 (1.9)	1.81 (1.7)	15.65	<.001
Shape concern	4.46 (1.3)	3.76 (1.8)	1.84 (1.8)	18.89	<.001
Eating concern	3.23 (1.2)	2.55 (1.4)	1.30 (1.3)	16.32	<.001

Abbreviations: AN, anorexia nervosa; BN, bulimia nervosa; OBE, objective bulimic episode; PBN, partial-syndrome bulimia nervosa; SBE, subjective bulimic episode.

*Values are expressed as mean (SD) unless otherwise indicated.

†An OBE is equivalent to a *DSM-IV*(*Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition*)–defined binge (ie, the consumption of an objectively large amount of food accompanied by a sense of loss of control).

‡An SBE is the consumption of a normal to small amount of food accompanied by a sense of loss of control. For the EDE, higher scores imply greater psychopathology.

analyses showed that there were no differences in age between patients with BN and patients with PBN or between patients with PBN and patients with AN. However, patients with BN were significantly older than those with AN ($P<.001$). There were no significant differences in duration of illness. Patients with AN had significantly lower body mass indexes than both patients with BN and patients with PBN ($P<.001$ for both comparisons). There was no difference in body mass index between patients with BN and patients with PBN.

Across groups, most patients were female: BN, 36 (94.7%); PBN, 39 (97.5%); and AN, 38 (90.5%), ($\chi^2_2=1.87$; $P<.39$). Ethnicity analyses revealed that the majority of patients were non-Hispanic white (BN, 36 [71.1%]; PBN, 39 [72.5%]; and AN, 38 [83.3%]) and that there were no differences between the 3 groups ($\chi^2_2=2.006$; $P<.37$). In examining family status, some differences did emerge. Most patients with AN were from intact families (27 [90.5%]), whereas 29 [68.4%] patients with BN and 35 [57.9%] patients with PBN were from intact families. This difference was statistically significant ($\chi^2_2=11.28$; $P<.004$). By definition, significantly more girls with AN (38 [97.1%]) compared with girls with BN (26 [35.3%]) and girls with PBN (23 [47.2%]) self-reported absent or irregular menses ($\chi^2_2=31.22$; $P<.001$).

GENERAL PSYCHOPATHOLOGY

Significant differences between patients with BN, PBN, and AN were reported for self-esteem as assessed with the Rosenberg Self-Esteem Scale ($P<.001$) and depression as assessed with the Beck Depression Inventory ($P<.001$) (**Table 2**). Post hoc analyses indicated that patients with AN reported higher self-esteem than either patients with BN ($P<.001$) or patients with PBN ($P<.004$). There were no differences between patients with BN and patients with PBN. For depression, patients with AN scored lower on the Beck Depression Inventory than either the patients with BN ($P<.001$) or patients with PBN ($P<.000$). Again, there were no differences between patients with BN and patients with PBN.

EATING DISORDER SYMPTOMS

Results are summarized in **Table 3**. Significant differences between the groups were reported across all variables examined ($P<.002$ for all variables). Post hoc analyses showed that patients with BN reported significantly higher binge eating frequency compared with patients with PBN ($P<.001$) and patients with AN ($P<.001$). There was no difference between patients with PBN and patients with AN in OBE frequency. For SBEs, only the difference between the PBN and AN groups was statistically significant ($P<.001$). Patients with BN reported a higher purge frequency than patients with PBN ($P<.001$), and patients with PBN in turn reported higher purge frequencies than patients with AN ($P<.003$). Patients with BN and patients with PBN were similar on all 4 EDE subscales (restraint eating, weight concerns, shape concerns, eating concerns), and both BN groups scored higher than the AN group on these subscales (all *P* values <.001).

COMMENT

This study aimed to provide a description of adolescents initially seen with symptoms of BN. Almost half (58 [48%]) of the patients who were initially seen at our clinic with BN symptoms did not meet full criteria for BN. Our findings revealed that patients with BN and patients with PBN showed more similarities than differences, with the only difference being the 2 diagnostic items; patients with BN reported significantly higher frequencies of binge eating and purging. This echoes the findings of a recent study of adult BN and PBN.[24]

Contrary to the cognitive model of BN,[25] which postulates that binge eating is usually followed by purging, patients in our study who were initially seen with PBN purged in the absence of objectively large binge eating episodes. That is, patients with PBN reported a relatively low average of 0.9 OBE per week but 4.5 purge episodes during the same period. However, examining OBEs plus SBEs, patients in the PBN group purged every time they perceived food intake as a binge (OBEs plus SBEs equals, on average, 6.1 binge eating episodes per week followed by 4.5 purge episodes during the same period). The same was true for patients with BN (OBEs plus SBEs equals, on average, 10.6 binge eating episodes per week

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followed by 11.3 purge episodes during the same period). This finding is consistent with 2 previous reports[26,27] questioning the validity of making a distinction between objective and subjective binge eating and suggests that *DSM* diagnostic criteria should emphasize purging more than binge eating, especially for younger patients.

While patients with BN reported significantly more objectively large binge episodes than patients with PBN, the latter reported almost double the amount of subjectively large binge episodes compared with their counterparts with BN (21 vs 11 episodes in 28 days; $P<.097$). Since purge frequency for both patients with BN and patients with PBN matches binge frequency when OBEs and SBEs are combined (ie, patients purged every time they perceived food intake to constitute a binge regardless of quantity), it would appear the salient binge feature is not the size of the binge but rather the patient's experience of loss of control during the particular eating episode. Also, in the present investigation, patients with BN and patients with PBN did not differ on measures of general psychopathology, a finding similar to that of a prior study examining OBEs and SBEs.[27] Taken together, these findings indicate that clinical management should not distinguish between BN and PBN.

In addition to binge and purge frequencies, menstrual irregularities in this patient population also are of concern. As expected, the majority of AN cases reported an absence of menses, but more than a third of patients with BN and nearly half of patients with PBN also reported menstrual irregularities. This is in keeping with the rate that has been previously reported in the literature.[28] While menstrual irregularities are common in immediately postmenarchal adolescents, the mean age of patients with BN and patients with PBN with menstrual disruption or irregularity was 15.9 years, a point in adolescent development where regular menstrual cycles should have been established for many teenagers.[29] There was no age difference between those adolescents with BN and adolescents with PBN with and those without menstrual abnormalities (15.9 years vs 16.4 years).

Patients with AN differed from those with BN and PBN on almost every variable examined. Patients with AN were significantly younger and had lower body mass indexes than patients with BN. Patients with AN more frequently came from intact families as opposed to patients with BN or patients with PBN. This finding has potential clinical implications in that the few available treatment studies for adolescent AN,[30-32] one small case series,[33] and one case study of family-based treatment for adolescent BN[34] all include parents. Consequently, we do not know whether family-based strategies will be appropriate for this clinical population, given that relatively large numbers of patients with BN are not embedded in intact family structures and single-parent families are significantly more represented.

These findings have significant nosologic implications. Given that there are more similarities than differences between patients with BN and patients with PBN, it seems reasonable not to view PBN as a separate diagnostic entity. Moreover, as these partial syndrome cases are initially seen with similar general and eating disorder psychopathologies as their full syndrome counterparts, except in the cases of OBEs and purge frequency, it makes little sense for treatment to be any less aggressive for patients with PBN than for patients with BN. There are clear indications that early recognition and treatment for adolescent AN[34,35] bodes favorably in terms of treatment outcome. While there are no published treatment trials available for adolescent BN, it is clinically feasible to advocate the same degree of urgency for both BN and PBN. For adolescents with AN, it has been shown that involving parents in weight restoration can be fruitful in recovery.[30-32] Future research will reveal whether similar family-based treatment strategies can also be helpful for adolescents with BN or whether interventions originally designed for adults with BN (eg, cognitive behavior therapy) will be more useful.

Results from this study are based on a clinical sample and may not generalize to community samples of adolescent eating disorders. This limitation may be especially pertinent to the patients with BN and the patients with PBN; while AN, a visible disease, may be more readily brought to clinical attention, BN behaviors often remain secretive and undetected for many years. Nevertheless, these data demonstrate that, at least within a clinical population, BN onsets for many cases during midadolescence and, similar to AN, pose substantial psychiatric and medical morbidity. Bulimia nervosa in adolescents seems to be a disorder in evolution (ie, there is considerable fluidity within and between diagnostic groups). The likelihood that someone with a subthreshold manifestation may go on to develop a full-blown syndrome remains a pertinent research question.

What This Study Adds

There are few reports that describe the clinical manifestation of BN in adolescents. None of these reports make reference to the comparative manifestation of adolescents with AN or those who do not meet full criteria for BN. This study therefore adds to the sparse data on adolescents with BN.

This study provides a description of a large cohort of adolescents with eating disorders. There are few differences between adolescents with BN and those who do not meet full criteria for this disorder. Adolescents with BN are quite distinct from their counterparts with AN both in terms of weight and binge/purge symptoms as well as demographic variables. Early recognition and swift treatment of eating disorders in adolescents, regardless of whether a diagnostic threshold is met, is imperative because they will lead to early intervention thereby potentially improving eating disorder recovery rates.

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REFERENCES

1. Kotler LA, Walsh BT. Eating disorders in children and adolescents: pharmacological therapies. *Eur Child Adolesc Psychiatry.* 2000;9(suppl 1):I108-I116.
2. Fairburn CG, Cooper Z, Doll HA, Norman P, O'Connor M. The natural course of bulimia nervosa and binge eating disorder in young women. *Arch Gen Psychiatry.* 2000;57:659-665.
3. Herzog DB, Keller MB, Sacks NR, Yeh CR, Lavori PW. Psychiatric comorbidity in treatment-seeking anorexics and bulimics. *J Am Acad Child Adolesc Psychiatry.* 1992;31:810-818.
4. Fairburn CG, Harrison PJ. Eating disorders. *Lancet.* 2003;361:407-416.
5. Steiger H, Séguin JR. Eating disorders: anorexia nervosa and bulimia nervosa. In: Millon T, Blaney PH, eds. *Oxford Textbook of Psychopathology.* New York, NY: Oxford University Press; 1999:365-389.
6. Schmidt U, Tiller J, Hodes M, Treasure J. Risk factors for the development of early onset bulimia nervosa in eating disorders in adolescence: anorexia and bulimia nervosa. In: Steinhausen HC, ed. *Eating Disorders in Adolescence: Anorexia and Bulimia Nervosa.* Berlin, Germany: Walter de Gruyter; 1995:83-93.
7. Stein S, Chalhoub N, Hodes M. Very early onset bulimia nervosa: report of two cases. *Int J Eat Disord.* 1998;24:323-327.
8. Kent A, Lacey H, McClusky SE. Pre-menarchal bulimia nervosa. *J Psychosom Res.* 1992;36:205-210.
9. Jones J, Bennett S, Olmsted MP, Lawson ML, Rodin G. Disordered eating attitudes and behaviours in teenaged girls: a school-based study. *CMAJ.* 2001;165:547-552.
10. Keel PK, Mitchell JE. Outcome in bulimia nervosa. *Am J Psychiatry.* 1997;154:313-321.
11. American Academy of Pediatrics Committee on Adolescence. Identifying and treating eating disorders. *Pediatrics.* 2003;111:204-211.
12. Nicholls D, Chater R, Lask B. Children into *DSM* don't go: a comparison of classification systems for eating disorders in childhood and early adolescence. *Int J Eat Disord.* 2000;28:317-324.
13. Rhea D. Eating disorder behaviors of ethnically diverse urban female adolescent athletes and non-athletes. *J Adolesc.* 1999;22:379-388.
14. Steiner H, Lock J. Anorexia and bulimia nervosa in children and adolescents: a review of the past 10 years. *J Am Acad Child Adolesc Psychiatry.* 1998;37:352-359.
15. Fisher M, Schneider M, Burns J, Symons H, Mandel F. Differences between adolescents and young adults at presentation to an eating disorders program. *J Adolesc Health.* 2001;28:222-227.
16. American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition.* Washington, DC: American Psychiatric Association; 1994.
17. Beck AT. *Beck Depression Inventory.* San Antonio, Tex: The Psychological Corp; 1987.
18. Brent D, Holder D, Kolko D, et al. A clinical psychotherapy trial for adolescent depression comparing cognitive, family, and supportive therapy. *Arch Gen Psychiatry.* 1997;54:877-885.
19. Lewinsohn P, Clarke GN, Hops H, Andrews J. Cognitive-behavioral group treatment for depressed adolescents. *Behav Ther.* 1990;21:385-401.
20. Rosenberg M. *Conceiving the Self.* New York, NY: Basic Books; 1979.
21. Agras WS, Fairburn CG, Wilson GT, Kraemer HC. A multicenter comparison of cognitive-behavioral therapy and interpersonal psychotherapy for bulimia nervosa. *Arch Gen Psychiatry.* 2000;57:459-466.
22. Fairburn CG, Cooper Z. The eating disorders examination. 12th ed. In: Fairburn CG, Wilson GT, eds. *Binge Eating: Nature, Assessment, and Treatment.* New York, NY: Guilford Press; 1993:317-360.
23. Passi V, Bryson S, Lock J. Assessment of eating disorders in adolescents with anorexia nervosa: self-report versus interview. *Int J Eat Disord.* 2003;33:45-54.
24. Crow SJ, Agras WS, Halmi K, Mitchell JE, Kraemer HC. Full syndromal versus subthreshold anorexia nervosa, bulimia nervosa, and binge eating disorder: a multicenter study. *Int J Eat Disord.* 2002;32:309-318.
25. Fairburn CG, Marcus MD, Wilson GT. Cognitive-behavioral therapy for binge eating and bulimia nervosa: a comprehensive treatment manual. In: Fairburn CG, Wilson GT, eds. *Binge Eating: Nature, Assessment, and Treatment.* New York, NY: Guilford Press; 1993:361-404.
26. Niego SH, Pratt EM, Agras WS. Subjective or objective binge: is the distinction valid? *Int J Eat Disord.* 1997;22:291-298.
27. Pratt EM, Niego SH, Agras WS. Does the size of a binge matter? *Int J Eat Disord.* 1998;24:307-312.
28. Sandler DP, Wilcox AJ, Homey LF. Age at menarche and subsequent reproductive events. *Am J Epidemiol.* 1984;119:765-774.
29. Fisher M, Golden N, Katzman DK, et al. Eating disorders in adolescents: a background paper. *J Adolesc Health.* 1995;16:420-437.
30. Le Grange D, Eisler I, Dare C, Russell GFM. Evaluation of family treatments in adolescent anorexia nervosa: a pilot study. *Int J Eat Disord.* 1992;12:347-357.
31. Russell GFM, Szmukler GI, Dare C, Eisler I. An evaluation of family therapy in anorexia and bulimia nervosa. *Arch Gen Psychiatry.* 1987;44:1047-1056.
32. Eisler I, Dare C, Russell GFM, Hodes M, Dodge E, Le Grange D. Family therapy for adolescent anorexia nervosa: the results of a controlled comparison of two family interventions. *J Child Psychol Psychiatry.* 2000;41:1025-1030.
33. Dodge E, Hodes M, Eisler I, Dare C. Family therapy for bulimia nervosa in adolescents: an exploratory study. *J Fam Ther.* 1995;17:59-77.
34. Le Grange D, Dymek M, Lock J. Family-based therapy for adolescents with bulimia nervosa. *Am J Psychother.* 2003;57:237-251.
35. Eisler I, Dare C, Russell GFM, Szmukler GI, Le Grange D, Dodge E. Family and individual therapy in anorexia nervosa. *Arch Gen Psychiatry.* 1997;54:1025-1030.

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