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REVIEW

Atypical eating disorders: a review

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Introduction: Atypical eating disorders (AEDs), also known as eating disorders not otherwise specified (EDNOS), are established eating disorders (EDs) presenting a significant clinical severity that do not fulfill the diagnostic criteria for typical anorexia nervosa (AN) or bulimia nervosa (BN).

Methods: Current literature on AEDs was reviewed in MedLine and EBSCO databases using the following keywords: "atypical eating disorders", "eating disorders not otherwise specified", "EDNOS", "diagnosis", "treatment", and "prevalence". All articles published between 1980 and 2010 were considered.

Results: There is currently no consensus regarding the definition of AEDs. Current proposals for the classification of EDs in the upcoming fifth edition of the *Diagnostic and Statistical Manual of Mental Disorders* are marked by less strict criteria for typical EDs, which should lead to limiting the field of AEDs. AEDs are among the most prevalent EDs in the general population and clinical setting. AEDs affect primarily adolescents and young adult women, causing an important psychological, somatic, and social burden. The incidence of AEDs is much higher than that reported for typical EDs. AEDs are frequently underdiagnosed because of limitations in patient disclosure and the lack of specific clinical symptoms. AED remission occurs in 75% of cases, and transition to typical EDs may also occur; nevertheless, the transition from a typical ED to an AED seems to be associated with a good prognosis.

Conclusion: As described in this review, AEDs are more typical and severe than was generally thought. Treatment should therefore be started as soon as possible. AEDs may be a transitional form of typical ED that could be more sensitive to prevention strategies.

Keywords: atypical eating disorders, anorexia nervosa, bulimia nervosa, EDNOS, diagnosis, prognosis, treatment, review

Introduction

Atypical eating disorders (AEDs), or eating disorders (EDs) not otherwise specified (EDNOS), are the most prevalent type of EDs.[1–3] Although AED is a common diagnosis in clinical practice, it has been largely neglected by researchers, and few studies regarding its treatment are available.[4] One of the main reasons for disregarding research on AEDs is probably the lack of clear positive diagnostic criteria to delineate these disorders.[5]

Since the beginning of the update of the *Diagnostic and Statistical Manual of Mental Disorders* for its fifth edition (DSM-V), much attention has been given to AEDs/EDNOS. Many authors have attempted to better characterize the different phenotypes of AEDs in order to "legitimize" the existence of this category of EDs.

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EDs are the third largest cause of morbidity among teenagers and young people.[6] Much of the interest in learning more about AEDs is related to the clinical course of these disorders and the possibility of early detection and prevention of EDs.[7]

The authors' aims were to review and clarify the definitions, epidemiology, and clinical aspects of AEDs, opening a discussion on the new DSM-V criteria, and to highlight the importance of early diagnosis and treatment of AEDs.

Method

An extensive review of the current literature on AEDs was carried out by the authors on MedLine and EBSCO databases using the following keywords: "atypical eating disorders", "eating disorders not otherwise specified", "EDNOS", "diagnosis", "treatment", and "prevalence". All articles published between 1980 and 2010 were considered. In addition, some of main websites on the subject were reviewed. Articles' bibliographies and book chapters on the topic were examined for relevant information and references. According to cited literature, the terms "AED" and "EDNOS" will be used as synonyms in this article, albeit they belong to different nosological classifications.

Definition and nosologic classification

An ED could be defined as "a persistent disturbance of eating behavior or behavior intended to control weight, which significantly impairs physical health or psychosocial functioning. This disturbance should not be secondary to any recognized general medical disorder".[8] In the 10th edition of the *International Classification of Diseases* (ICD-10)[9] and the fourth edition of the DSM (DSM-IV),[10] the leading nosologic systems in psychiatry, recognize two main typical EDs: anorexia nervosa (AN) and bulimia nervosa (BN). AN is characterized by volitional self-starvation[11,12] and BN's primary characteristics are binge eating and purge behaviors.[13] Moreover, overestimation of eating, shape, and weight and obsessional control are hallmarks of the two typical EDs.[14,15] Both DSM-IV and ICD criteria use nonempirically established minimal weight and binge/purging frequency for AN and BN, respectively.

Although the concept of an ED tends to be equated with AN and BN in the medical setting, both cited classification systems recognize a third residual category of ED: "atypical eating disorder" or "eating disorder not otherwise specified". The wording of the categories reflects the clinical severity included in the definition of an ED that does not fulfill the criteria for AN or BN. AED/EDNOS are therefore exclusion diagnoses. ICD-10 and DSM-IV and criteria for AEDs are listed in Tables 1 and 2, respectively.

In most cases of AEDs/EDNOS, clinical features resemble those of AN and BN but are slightly different in intensity or combination.[5,16,17] The cases of EDs not reaching diagnostic thresholds (eg, weight or binge/purging frequency or duration) are often called "subthreshold" EDs.[5] Some authors criticize the use of the word "atypical" because the prevalence of AEDs is higher than that of typical AN and BN together.[8] Another criticism is addressed to the term "subclinical eating disorders", which implies a mild severity. Indeed, by definition, AEDs are associated with a significant level of clinical impairment and severity.[8,18] The heterogeneity of the AED category is a drawback for clinical practice and research because it provides little information about patients' symptoms, outcome, prognosis, or therapeutic strategy; this underlines the need for more precise homogeneous subtypes.[18]

Many efforts have been made to better characterize AEDs/EDNOS. The most successful resulted in the creation of a third "typical" ED, named binge eating disorder (BED), which has already been added as a provisional diagnosis in the DSM-IV. BED was first recognized among individuals

Table 1 International Classification of Diseases, 10th Edition, criteria of atypical eating disorders[9]

F50.1 Atypical anorexia nervosa: Disorders that fulfill some of the features of anorexia nervosa but in which the overall clinical picture does not justify that diagnosis. For instance, one of the key symptoms, such as amenorrhea or marked dread of being fat, may be absent in the presence of marked weight loss and weight-reducing behavior. This diagnosis should not be made in the presence of known physical disorders associated with weight loss.

F50.3 Atypical bulimia nervosa: Disorders that fulfill some of the features of bulimia nervosa but in which the overall clinical picture does not justify that diagnosis. For instance, there may be recurrent bouts of overeating and overuse of purgatives without significant weight change, or the typical overconcern about body shape and weight may be absent.

F50.4 Overeating associated with other psychological disturbances: Overeating due to stressful events, such as bereavement, accident, or childbirth.

F50.5 Vomiting associated with other psychological disturbances: Repeated vomiting that occurs in dissociative disorders and hypochondriacal disorder and that is not solely due to conditions classified outside this chapter. This subcategory may also be used in addition to O21 (excessive vomiting in pregnancy) when emotional factors are predominant in the causation of recurrent nausea and vomiting in pregnancy.

F50.8 Other eating disorders: Pica in adults, psychogenic loss of appetite.

F50.9 Eating disorder, unspecified

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Table 2 *Diagnosis and Statistical Manual of Mental Disorders, Fourth Edition, Text Revision* (DSM-IV-TR) criteria for eating disorders not otherwise specified (EDNOS)[10] (Adapted with permission)

The EDNOS category is for disorders of eating that do not meet the criteria for any specific eating disorder. Examples include:

1. For females, all of the criteria for anorexia nervosa are met except that the individual has regular menses
2. All of the criteria for anorexia nervosa are met except that, despite significant weight loss, the individual's current weight is in the normal range
3. All of the criteria for bulimia nervosa are met except that the binge eating and inappropriate compensatory mechanisms occur at a frequency of less than twice a week or for a duration of less than 3 months
4. The regular use of inappropriate compensatory behavior by an individual of normal body weight after eating small amounts of food (eg, self-induced vomiting after the consumption of two cookies)
5. Repeatedly chewing and spitting out, but not swallowing, large amounts of food
6. Binge eating disorder: recurrent episodes of binge eating in the absence of the regular use of inappropriate compensatory behaviors characteristic of bulimia nervosa (see Appendix B in DSM-IV-TR for suggested research criteria).

involved in weight control programs and presenting episodes of binge eating without engaging in purging or inappropriate compensatory behaviors and consequently becoming obese.[19–21] The recognition of this subgroup was confirmed by a multicenter study concerning 1984 participants attending weight loss clinics.[22] In this study, 30% of patients attending weight loss clinics met the criteria for BED, whereas only 2% of individuals randomly selected from the general population met these criteria. Another study concerning 1785 individuals from 18 weight control programs confirmed these findings. In addition, BED was more frequent in females than in males (32% versus 21%) and was often associated with severe obesity (body mass index [BMI] above 35) and a history of marked weight fluctuations.[23]

Other types of AED previously described in prospective studies are atypical AN,[24] subthreshold BN, night eating syndrome[25,26] and purging disorder.[27]

Because there is a lack of consensus on the definition of AED and because the criteria of ICD-10 and DSM-IV are unsatisfactory to classify these disorders, some researchers have adopted their own definitions of AED.[28]

In a meta-analysis evaluating 125 studies, the clinical severity of AEDs/EDNOS was highlighted.[18] Moreover, few differences in eating behaviors, general psychopathology, and physical health were found between individuals with AED/EDNOS and typical ED according to the DSM-IV. The authors suggested that refining current criteria of DSM-IV and delineating homogeneous AED subtypes would be the best option.[18]

DSM-V proposals for EDNOS

Since 1999, the American Psychiatric Association proposed modifications to the DSM criteria for most of the disorders. The Eating Disorders Work Group has recently renamed the ED category to "feeding and eating disorders" including feeding disorders first diagnosed in childhood or adolescence. EDNOS has now been renamed "feeding and eating conditions not elsewhere classified", and this category comprises "several conditions of potential clinical significance" and "feeding or eating problems not meeting criteria for currently recognized disorders".[29] Five types of feeding and eating conditions not elsewhere classified have been proposed: atypical AN, subthreshold BN, subthreshold BED, purging disorder, night eating syndrome and residual conditions named other "feeding or eating condition not elsewhere classified". The work group recognizes that currently available data are insufficient to consider these conditions as a true disorder, although these conditions may be associated with levels of distress or impairment comparable with those of well-recognized feeding disorders and EDs. It may be questioned how this rather complex subclassification of former EDNOS can be handled in the everyday primary care practice where EDNOS should be recognized at an early stage. In addition, assessment of severity is lacking. In the case of AN and BN, current criteria have been attenuated by reducing thresholds of weight and binge crises and eliminating amenorrhea as a mandatory symptom for the diagnosis of AN.

Prevalence of AEDs

AEDs are by far the most prevalent type of ED. According to different studies in the general population (Table 3), the lifetime prevalence of AEDs is 6.3%–17.5% and point prevalence is 3%–14.5% in women aged 12–35 years. Controversial results have been found between studies evaluating AEDs in both men and women. One study found a total point prevalence of 0.32%,[30] and another reported 9.4%.[31] The wide range of lifetime and point prevalence is probably caused by differences in evaluation methods and criteria used to classify AEDs. Longitudinal studies suggest that the prevalence of EDNOS relative to AN and BN has increased over time.[32]

In a clinical setting, the prevalence of AEDs is even greater, reaching 40%–60% in epidemiological studies

Table 3 Lifetime and point prevalence of typical and atypical eating disorders (AEDs)

Study	n	Sample type	Prevalence	AN	BN	BED	AED	AN-NOS	BN-NOS
Isomaa et al[3]	595	Female adolescents	Lifetime	2.6%	0.4%		17.5	7.7%	1.3%
Wade et al[33]	1002	Female adult twins	Lifetime	1.9%	2.9%	2.9%	7.7%	2.4%	5.3%
Favaro et al[34]	934	Female adults aged 18–25 years	Lifetime	2.2%	4.6%	0.6%	6.3%	2.6%	3.1%
Favaro et al[34]	934	Female adults aged 18–25 years	Lifetime	2.2%	4.6%	0.6%	6.3%	2.6%	3.1%
			Point	0.3%	1.8%	0.1%	3.2%	0.7%	2.4%
Machado et al[35]	2028	Female students aged 12–23 years	Point	0.52%	0.39%	0.13%	3.13%	0.53%	2.47%
Zini et al[31]	788	Male and female adolescents	Point				9.4%		
Pelaez Fernandez et al[36]	1545	Males and females aged 12–21 years	Point	0.33	2.45		3.2		
Faravelli et al[30]	2355	Males and females aged >14 years	Point	0.42%	0.32%		0.32%		
Cotrufo et al[37]	919	Female students aged 13–19 years	Point	0.2%	2.3%		14.5%		
Vetrone et al[38]	300	Female schoolgirls	Point		1.7%		6.4%		
Gotestam and Agras[39]	2500	Females	Point	0.43%	1.62%	3.24%	3.03%		

Abbreviations: AN, anorexia nervosa; AN-NOS, atypical anorexia nervosa; BED, binge eating disorder; BN, bulimia nervosa; BN-NOS, atypical bulimia nervosa.

evaluating patients seeking treatment in ED specialized clinics,[1,2,4,40–48] 75% of cases among young women with EDs in a community prevalence study[35] and 90% in outpatient psychiatric practice.[49]

Incidence of AEDs

The incidence of AEDs is estimated at 964–2800 cases/100,000/year. These values vary according to age range and duration of follow-up. Two studies with a longer duration and concerning younger women reported the highest values[50,51] and one study presenting a shorter follow-up in older women reported the lowest value.[52] Indeed, these values are higher than those obtained for AN plus BN in most of the studies evaluating the prevalence of EDs.

Clinical course of AEDs and prognostic factors

At least four clinical courses should be considered for AEDs: i) as a completely new disorder, evolving in a specific clinical course; ii) as a disorder preceding the development of a typical ED; iii) as an outcome of a previous typical ED; and iv) as a way station from one typical ED to another typical ED.

Outcomes were mainly studied for AEDs with bulimic symptoms (AED-BN).[53] It seems that in studies with a follow-up lasting less than 5 years, remission rates were higher (67%–69%) for AED-BN than those reported for typical BN (28%–37%),[54–56] but this difference disappears with a longer follow-up. One study comparing AED-BN and BN reported a remission rate of 75% for both disorders.[57] This result was replicated in a second study after 20 years of follow-up.[58] It may thus be inferred that AED is a chronic disorder in about 25% of the patients. Moreover, remission rates for other AEDs do not diverge from those found for AED-BN.[3,59] However, AED-AN patients are less prone to lose weight after discharge, they recover faster and they have lower cumulative risk for developing binge/purging behavior.[60]

Early studies on AEDs described a continuum between AEDs and full syndromal EDs,[61] suggesting that AEDs are an intermediate, less intense form of ED. In fact, follow-up studies mainly describe the migration from a typical ED to an AED[62,63] and the contrary seems to be less frequent,[58,59] occurring in 26% of AED cases in one study mainly through a typical BN[64] and among adolescent series.[50,55] Agras et al suggest that AEDs seem to be experienced by individuals who are mainly in the pathway to remission and less frequently relapse.[59] AED patients seem to improve over time. It seems that individuals who have never presented a typical ED are unlikely to reach a severity of symptoms allowing the diagnosis of AN or BN.[53] Overall, it seems that AEDs are an intermediate stage between a full ED and remission. Although AEDs are prone to remission, 25% of patients might undergo a chronic course.

Quality of life is also impaired by AEDs, as demonstrated by one study evaluating 87 young women, 68% of them with AED, who presented a lower quality of life and higher psychological distress and immature coping style.[65] Mortality of AED patients was reported only in one study, but the mortality rate was not specifically assessed.[66]

Among the main prognostic factors of AED are low BMI, the presence of AN symptoms[54,62] and the absence of close friends, probably reflecting greater interpersonal problems.[67]

Clinical aspects

In clinical terms AEDs may resemble AN or BN with a lower intensity of symptoms or may be completely different from typical EDs. Some AEDs may involve chronic dietary restriction, overexercising, laxatives, diuretics, anorexigenic drug misuse, and inappropriate eating behaviors like pica, chewing and spitting out, binging, or only purging without binging. Night binging disorder was also described and is characterized by binging episodes during the night.[25]

Both typical ED and AED patients seeking treatment present psychopathological traits that are much alike.[1,16,40,61,68–70] Perfectionism, a widely acknowledged factor involved in the onset and maintenance of EDs, is also frequently found in AED patients.[71]

Differential diagnosis should be performed with some organic disorders causing either hypophagia (eg, depression, dementia, hyperthyroidism, personality disorders), hyperphagia (eg, hypothalamic tumors, Prader–Willi and Kleine–Levin syndromes, anxiety disorders, or depression), gastrointestinal disorders (eg, dysphagia, achalasia, diarrhea, esophageal cancer dyspepsia, irritable bowel syndrome or rumination),[72,73] voluntary food eviction (eg, obsessive-compulsive disorder, delusional disorders)[74,75] or eating symptoms caused by drugs or medicine consumption. Increased awareness of EDs has been suggested as a potential misleading diagnosis in the case of digestive disorder pathology mainly in children and adolescents.[72] Consequently, an ED diagnosis should be made only after the exclusion of all other organic pathology.

Psychiatric comorbidities are equally present in AEDs and full EDs. In one study a higher lifetime prevalence of depression and obsessive-compulsive disorder was observed in AED-BN patients than in BN patients.[55] Some authors reported a higher rate of suicide attempts[24,76] and self-harm in AED patients than in typical ED patients. Similar to typical ED patients, substance abuse is also highly prevalent in AED patients, particularly AED-BN patients.[77–80] One study analyzing the association between AEDs and substance abuse in adolescents found that dieting severity was positively correlated to the prevalence of alcohol, cigarette and cannabis use.[81] In adults, alcohol use disorder was strongly associated with BN, followed by BEDs and AEDs.[82] In another study on substance use in five European countries, the prevalence of alcohol abuse was higher in AED patients than in typical ED patients.[80] Tobacco exerts effects on weight and appetite and is probably used by many patients with EDs to achieve restriction and weight loss.[83] These data emphasize the importance of assessing substance use in individuals with disturbed eating behavior and vice versa. Treatment focusing on both EDs and addictive disorders should be considered in cases of comorbidity.[84,85]

Incidences of somatic consequences of AEDs have barely been studied because AEDs have been considered "mild" or "subclinic" disorders.[28] Nevertheless, AED physical damage and psychosocial impairments closely resemble those of AN and BN. In line with BN, AED patients presenting purging behaviors like vomiting or laxative or diuretic misuse are more susceptible to hydroelectrolytic disturbances, fatigue, hypotension, and electrocardiographic (ECG) disturbances. Gastrointestinal disorders like chronic constipation, regurgitation, dyspeptic symptoms, nausea and rumination are among the most frequently described in AED patients.[86–90] Cycle abnormalities and infertility are equally present in AED patients. Amenorrhea has been described in AED patients in two studies[91,92] and dysmenorrheal antecedents have been described among 93% of students with AED.[93] AED was also described in 16.7% of women consulting an infertility clinic.[94] Bone demineralization and precocious osteoporosis are also important burdens of AEDs to young patients. Bone disturbances may cause significant impairment in young patients with AEDs[95] and are caused by hypogonadism but also by calcium and vitamin D deficiencies. Decreased bone density osteoporosis was present in an AED patient in one study[96] and there was an increased risk of fractures in an AED patient in another study indicating permanent skeletal damage secondary to this disorder.[97] Moreover, patients with BED are obese and at risk for physical, psychological and social disabilities associated with obesity (eg, metabolic syndrome, diabetes, heart disease, low self-esteem, prejudice).[98]

Treatment of AEDs

Because of their significant psychological, somatic and social burdens, treatment should be proposed for patients with AEDs. In spite of the elevated prevalence and the burden of AEDs, to date, no evidence-based therapy has been developed specifically for these disorders, excepted for BED.[99] In the absence of established pathophysiological mechanisms and clear diagnostic criteria the development and evaluation of therapeutic strategies for AEDs have been markedly hampered. Some authors have combined clinical outcome data for AEDs with those for AN and BN[100] or BN with subthreshold binging episodes.[101,102] In the absence of validated treatment, clinicians are encouraged to use currently available treatments for typical EDs adapted to the most significant symptoms presented by AED patients.[103]

Current ED treatment is based on four approaches that should be adapted for each clinical situation according to AED symptoms, patient preferences and local availability: i) early restoration of a normal nutritional and physiological state, ii) establishment of a therapeutic alliance, iii) a multidisciplinary team approach[104] and iv) pharmacologic treatment. Outpatient treatment should be used whenever possible and based on in-depth clinical evaluation, psychoeducation, weight and malnutrition follow-up, nutritional complements, comorbidity treatment and psychotherapy. Inpatient treatment is necessary when a significant somatic risk is present (eg, hypokalemia, ECG disorder, hypotension, bradycardia, hypothermia, renal or hepatic failure, BMI < 14 kg/m^2), when deterioration occurs during outpatient treatment, or when outpatient treatment is insufficient to stabilize a patient.[105] Inpatient treatment should be organized in an integrated model employing internal medicine, psychiatric clinics and ED clinics according to patients' needs and availability. In the AN type of AED, weight restoration should be addressed by renutrition using dietary education, nutritional complements, and nasogastric tube feeding[106] whenever required. Antidepressant drugs like fluoxetine can reduce the frequency of binge eating as described in three systematic reviews.[107–109] However, moderate improvement (50% or more reduction of binge eating) and a remission rate of 20% (similar to placebo) were reported in a meta-analysis study that evaluated eight trials concerning 901 patients.[109] Psychotherapy could be delivered individually or with family participation. Cognitive behavior therapy (CBT) has strong evidence-based support to be used as first-line treatment for BN.[110] Thirty-one trials using CBT in BN were assessed in a systematic review, but, in fact, only eight trials included AED patients and nine evaluated BED patients alone. CBT achieved a 37% cumulative binge eating abstinence when compared with a rate of almost zero in the cumulative wait-list trials. A recent study evaluated a newly refined 20-week CBT program in a sample of 154 patients with BN or BN-type AED.[111] At the end of the 60-week follow-up 51.3% of the sample had ED symptoms less than one standard deviation above the community mean based on the Eating Disorders Examination.[112] No evidence supporting one specific type of psychotherapy in AN has been described in a systematic review,[113] although a specialist approach is more effective than support therapy delivered by a nonspecialist.

Prevention

Similar to typical EDs, AEDs affect adolescents and young adults. Related to the discrete nature of symptoms and disclosure difficulties AEDs may remain undetected[114] and consequently result in severe somatic or psychological consequences, including malnutrition. It is thus necessary to implement screening strategies for EDs in primary care and prevention settings.[115] Primary care givers, such as general practitioners and preventive medicine professionals, should play a crucial role in the early diagnosis and management of patients with EDs. For this purpose screening questionnaires like the SCOFF questionnaire are fundamental.[7,116,117] It is well established that a shorter duration of illness prior to initiation of care or hospitalization and a higher BMI, in the case of AN, are significant prognostic indicators.[118] Some studies evaluating treatment indicate that outcomes are better when detection is early and treatment of EDs occurs soon after symptom onset.[119] Patients with EDs may consult nutrition specialists aiming to lose weight or asking for specific diets, including exclusion diets for reported food intolerance or digestive discomfort. Signs of malnutrition without an organic etiology in adolescents or young adults should be regarded as suggestive of an underlying ED. ED diagnosis is often neglected or is made at a stage of severe malnutrition or metabolic disorders, including hypokalemia and/or osteoporosis. Recently, "early intervention" programs for EDs have been proposed in primary care aiming at enhanced prevention of EDs. These programs are based on early-stage detection[120] and nutritional care, including nasogastric tube feeding like in typical EDs.[106] Prevention programs for EDs have been conducted in school and community settings and have focused on body image, unhealthy eating behavior and eating attitudes. Although some of them have produced encouraging results, there is no consensus regarding the most effective content, setting, and target group.[121]

Conclusion

As described in this review, AEDs are more typical and severe than was previously thought; therefore, treatment should be started as soon as they are diagnosed. AEDs should not be considered a milder form of ED but rather represent a transitional form of typical ED that could lead to significant opportunities for prevention. New perspectives on simple and practical diagnostic criteria of AEDs are needed to promote new epidemiological and therapeutic research on AEDs.

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Disclosure

The authors declare that they have no conflicts of interest.

References

1. Ricca V, Mannucci E, Mezzani B, et al. Psychopathological and clinical features of outpatients with an eating disorder not otherwise specified. *Eat Weight Disord*. 2001;6(3):157–165.
2. Turner H, Bryant-Waugh R. Eating disorders not otherwise specified (EDNOS): profiles of clients presenting at a community eating disorder service. *Eur Eat Disord Rev*. 2004;12:18–26.
3. Isomaa R, Isomaa AL, Marttunen M, et al. The prevalence, incidence and development of eating disorders in Finnish adolescents: a two-step 3-year follow-up study. *Eur Eat Disord Rev*. 2009;17(3):199–207.
4. Dalle Grave R, Calugi S. Eating disorder not otherwise specified in an inpatient unit: the impact of altering the DSM-IV criteria for anorexia and bulimia nervosa. *Eur Eat Disord Rev*. 2007;15(5):340–349.
5. Fairburn CG, Bohn K. Eating disorder NOS (EDNOS): an example of the troublesome "not otherwise specified" (NOS) category in DSM-IV. *Behav Res Ther*. 2005;43(6):691–701.
6. Lucas A, Beard C, O'Fallon W, Kurland L. 50-year trends in the incidence of anorexia nervosa in Rochester, Minnesota, in a population based study. *Am J Psychiatry*. 1991;148:917–922.
7. Garcia FD, Grigioni S, Chelali S, et al. Validation of the French version of SCOFF questionnaire for screening of eating disorders among adults. *World J Biol Psychiatry*. 2010;11(7):888–893.
8. Fairburn CG, Walsh BT. Atypical eating disorders (eating disorders not otherwise specified). In: Fairburn CG, Brownell KD, editors. *Eating Disorders and Obesity: A Comprehensive Handbook*. New York, NY: Guilford Press; 2002.
9. World Health Organization. *The International Statistical Classification of Diseases and Health Related Problems ICD-10*. 10th ed. Geneva: World Health Organization; 1992.
10. American Psychiatric Association. Task Force on DSM-IV. *Diagnostic and Statistical Manual of Mental Disorders: DSM-IV-TR*. 4th ed. Washington, DC: American Psychiatric Association; 2000.
11. Gull W. Anorexia nervosa (apepsia hysterica, anorexia hysterica). *Transactions of the Clinical Society of London*. 1874;7:22–28.
12. Laségue C. De l'anorexie hysterique. *Archives Generales de Médecine*. 1873:386–403.
13. Russell GFM. Bulimia nervosa: an ominous variant of anorexia nervosa. *Psych Med*. 1979;9:429–448.
14. American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders – DSM-IV*. 4th ed. Washington, DC; 1994.
15. Cooper M, Turner H. Underlying assumptions and core beliefs in anorexia nervosa and dieting. *Br J Clin Psychol*. 2000;39(2):215–218.
16. Crow SJ, Stewart Agras W, Halmi K, et al. Full syndromal versus subthreshold anorexia nervosa, bulimia nervosa, and binge eating disorder: a multicenter study. *Int J Eat Disord*. 2002;32(3): 309–318.
17. Waller G. Why do we diagnose different types of eating disorder? Arguments for a change in research and clinical practice. *Eat Disord Rev*. 1993;1:74–89.
18. Thomas JJ, Vartanian LR, Brownell KD. The relationship between eating disorder not otherwise specified (EDNOS) and officially recognized eating disorders: meta-analysis and implications for DSM. *Psychol Bull*. 2009;135(3):407–433.
19. Stunkard AJ. Eating patterns and obesity. *Psychiatr Q*. 1959;33: 284–295.
20. Gormally J, Black S, Daston S, Rardin D. The assessment of binge eating severity among obese persons. *Addict Behav*. 1982;7(1): 47–55.
21. Marcus MD, Wing RR, Lamparski DM. Binge eating and dietary restraint in obese patients. *Addict Behav*. 1985;10(2):163–168.
22. Spitzer RL, Devlin M, Walsh BT, et al. Binge eating disorder: a multisite field trial of the diagnostic criteria. *Int J Eat Disord*. 1992;11: 191–203.
23. Spitzer RL, Yanovski S, Wadden T, et al. Binge eating disorder: its further validation in a multisite study. *Int J Eat Disord*. 1993;13(2): 137–153.
24. Santonastaso P, Bosello R, Schiavone P, et al. Typical and atypical restrictive anorexia nervosa: weight history, body image, psychiatric symptoms, and response to outpatient treatment. *Int J Eat Disord*. 2009; 42(5):464–470.
25. Striegel-Moore RH, Franko DL, Garcia J. The validity and clinical utility of night eating syndrome. *Int J Eat Disord*. 2009;42(8):720–738.
26. Stunkard AJ, Grace WJ, Wolff HG. The night-eating syndrome: a pattern of food intake among certain obese patients. *Am J Med*. 1955;19(1):78–86.
27. Keel PK, Striegel-Moore RH. The validity and clinical utility of purging disorder. *Int J Eat Disord*. 2009;42(8):706–719.
28. Chamay-Weber C, Narring F, Michaud PA. Partial eating disorders among adolescents: a review. *J Adolesc Health*. 2005;37(5): 417–427.
29. American Psychiatric Association. DSM-5 Development: Proposed Revisions: Eating Disorders. http://www.dsm5.org/ProposedRevisions/Pages/EatingDisorders.aspx. Accessed December 19, 2010.
30. Faravelli C, Ravaldi C, Truglia E, et al. Clinical epidemiology of eating disorders: results from the Sesto Fiorentino study. *Psychother Psychosom*. 2006;75(6):376–383.
31. Zini A, Siani R, Sandri M, et al. Partial syndromes in eating disorders: a prevalence study on a sample of Italian adolescents. *Eat Weight Disord*. 2007;12(3):125–131.
32. Ash JB, Piazza E. Changing symptomatology in eating disorders. *Int J Eat Disord*. 1995;18(1):27–38.
33. Wade TD, Bergin JL, Tiggemann M, et al. Prevalence and long-term course of lifetime eating disorders in an adult Australian twin cohort. *Aust N Z J Psychiatry*. 2006;40(2):121–128.
34. Favaro A, Ferrara S, Santonastaso P. The spectrum of eating disorders in young women: a prevalence study in a general population sample. *Psychosom Med*. 2003;65(4):701–708.
35. Machado PP, Machado BC, Goncalves S, Hoek HW. The prevalence of eating disorders not otherwise specified. *Int J Eat Disord*. 2007; 40(3):212–217.
36. Pelaez Fernandez MA, Labrador FJ, Raich RM. Prevalence of eating disorders among adolescent and young adult scholastic population in the region of Madrid (Spain). *J Psychosom Res*. 2007;62(6):681–690.
37. Cotrufo P, Barretta V, Monteleone P, Maj M. Full-syndrome, partial-syndrome and subclinical eating disorders: an epidemiological study of female students in Southern Italy. *Acta Psychiatr Scand*. 1998;98(2): 112–115.
38. Vetrone G, Cuzzolaro M, Antonozzi I. Clinical and subthreshold eating disorders: case detection in adolescent schoolgirls. *Eat Weight Disord*. 1997;2(1):24–33.
39. Gotestam KG, Agras WS. General population-based epidemiological study of eating disorders in Norway. *Int J Eat Disord*. 1995;18(2): 119–126.
40. Rockert W, Kaplan AS, Olmsted MP. Eating disorder not otherwise specified: the view from a tertiary care treatment center. *Int J Eat Disord*. 2007;40 Suppl:S99–S103.
41. Button EJ, Benson EN, Nollet C, Palmer RL. Don't forget EDNOS (eating disorders not otherwise specified): patterns of service use in an eating disorders service. *Psych Bull*. 2005;29:134–136.
42. Fairburn CG, Cooper Z, Bohn K, et al. The severity and status of eating disorder NOS: implications for DSM-V. *Behav Res Ther*. 2007;45(8): 1705–1715.
43. Fairburn CG, Cooper Z. Thinking afresh about the classification of eating disorders. *Int J Eat Disord*. 2007;40 Suppl:S107–S110.
44. Martin CK, Williamson DA, Thaw JM. Criterion validity of the multiaxial assessment of eating disorders symptoms. *Int J Eat Disord*. 2000;28(3):303–310.
45. Williamson DA, Womble LG, Smeets MA, et al. Latent structure of eating disorder symptoms: a factor analytic and taxometric investigation. *Am J Psychiatry*. 2002;159(3):412–418.

46. Nollett CL, Button EJ. Questionnaire measures of psychopathology in eating disorders: comparisons between clinical groups. *Eur Eat Disord Rev*. 2005;13:211–215.
47. Eddy KT, Celio Doyle A, Hoste RR, et al. Eating disorder not otherwise specified in adolescents. *J Am Acad Child Adolesc Psychiatry*. 2008; 47(2):156–164.
48. Millar HR. New eating disorder service. *Psych Bull*. 1998;22: 751–754.
49. Zimmerman M, Francione-Witt C, Chelminski I, et al. Problems applying the DSM-IV eating disorders diagnostic criteria in a general psychiatric outpatient practice. *J Clin Psychiatry*. 2008;69(3):381–384.
50. Lahortiga-Ramos F, De Irala-Estevez J, Cano-Prous A, et al. Incidence of eating disorders in Navarra (Spain). *Eur Psychiatry*. 2005;20(2): 179–185.
51. Patton GC, Selzer R, Coffey C, et al. Onset of adolescent eating disorders: population based cohort study over 3 years. *BMJ*. 1999; 318(7186):765–768.
52. Ghaderi A, Scott B. Prevalence, incidence and prospective risk factors for eating disorders. *Acta Psychiatr Scand*. 2001;104(2):122–130.
53. Keel PK, Brown TA. Update on course and outcome in eating disorders. *Int J Eat Disord*. 2010;43(3):195–204.
54. Clausen L. Time to remission for eating disorder patients: a 2(1/2)-year follow-up study of outcome and predictors. *Nord J Psychiatry*. 2008; 62(2):151–159.
55. Schmidt U, Lee S, Perkins S, et al. Do adolescents with eating disorder not otherwise specified or full-syndrome bulimia nervosa differ in clinical severity, comorbidity, risk factors, treatment outcome or cost? *Int J Eat Disord*. 2008;41(6):498–504.
56. Ben-Tovim DI, Walker K, Gilchrist P, et al. Outcome in patients with eating disorders: a 5-year study. *Lancet*. 2001;357(9264):1254–1257.
57. Grilo CM, Pagano ME, Skodol AE, et al. Natural course of bulimia nervosa and of eating disorder not otherwise specified: 5-year prospective study of remissions, relapses, and the effects of personality disorder psychopathology. *J Clin Psychiatry*. 2007;68(5):738–746.
58. Keel PK, Gravener JA, Joiner TE Jr, Haedt AA. Twenty-year follow-up of bulimia nervosa and related eating disorders not otherwise specified. *Int J Eat Disord*. 2010;43(6):492–497.
59. Agras WS, Crow S, Mitchell JE, et al. A 4-year prospective study of eating disorder NOS compared with full eating disorder syndromes. *Int J Eat Disord*. 2009;42(6):565–570.
60. Strober M, Freeman R, Morrell W. Atypical anorexia nervosa: separation from typical cases in course and outcome in a long-term prospective study. *Int J Eat Disord*. 1999;25(2):135–142.
61. Dancyger IF, Garfinkel PE. The relationship of partial syndrome eating disorders to anorexia nervosa and bulimia nervosa. *Psychol Med*. 1995; 25(5):1019–1025.
62. Ro O, Martinsen EW, Hoffart A, et al. Two-year follow-up after inpatient treatment. *Eur Eat Disord Rev*. 2005;13:255–263.
63. Vrabel KR, Rosenvinge JH, Hoffart A, et al. The course of illness following inpatient treatment of adults with longstanding eating disorders: a 5-year follow-up. *Int J Eat Disord*. 2008;41(3):224–232.
64. Milos G, Spindler A, Schnyder U, Fairburn CG. Instability of eating disorder diagnoses: prospective study. *Br J Psychiatry*. 2005;187: 573–578.
65. Hay P, Buttner P, Mond J, et al. Quality of life, course and predictors of outcomes in community women with EDNOS and common eating disorders. *Eur Eat Disord Rev*. 2010;18(4):281–295.
66. Helverskov JL, Clausen L, Mors O, et al. Trans-diagnostic outcome of eating disorders: a 30-month follow-up study of 629 patients. *Eur Eat Disord Rev*. 2010;18(6):453–463.
67. Herzog DB, Dorer DJ, Keel PK, et al. Recovery and relapse in anorexia and bulimia nervosa: a 7.5-year follow-up study. *J Am Acad Child Adolesc Psychiatry*. 1999;38(7):829–837.
68. Herzog DB, Hopkins JD, Burns CD. A follow-up study of 33 subdiagnostic eating disordered women. *Int J Eat Disord*. 1993;14(3):261–267.
69. Keel PK. Purging disorder: subthreshold variant or full-threshold eating disorder? *Int J Eat Disord*. 2007;40 Suppl:S89–S94.
70. Bunnell DW. Subclinical versus formal eating disorders: differentiating psychological features. *Int J Eat Disord*. 1990;9:357–362.
71. Woodside DB, Bulik CM, Halmi KA, et al. Personality, perfectionism, and attitudes toward eating in parents of individuals with eating disorders. *Int J Eat Disord*. 2002;31(3):290–299.
72. Dabritz J, Domagk D, Monninger M, Foell D. Achalasia mistaken as eating disorders: report of two children and review of the literature. *Eur J Gastroenterol Hepatol*. 2010;22(7):775–778.
73. Gourcerol G, Dechelotte P, Ducrotte P, Leroi AM. Rumination syndrome: when the lower esophageal sphincter rises. *Dig Liver Dis*. In press.
74. Garcia FD, Houy-Durand E, Thibaut F, Dechelotte P. Obsessive compulsive disorder as a cause of atypical eating disorder: a case report. *Eur Eat Disord Rev*. 2009;17(6):444–447.
75. Jesus P, Garcia FD, Dechelotte P. Derrière la pseudo-demence à 40 ans, l'anorexie mentale. *Nutrition Infos*. 2010;3:32–33.
76. Neumark-Sztainer D, Story M, French SA. Covariations of unhealthy weight loss behaviors and other high-risk behaviors among adolescents. *Arch Pediatr Adolesc Med*. 1996;150(3):304–308.
77. Striegel-Moore RH, Garvin V, Dohm FA, Rosenheck RA. Psychiatric comorbidity of eating disorders in men: a national study of hospitalized veterans. *Int J Eat Disord*. 1999;25(4):399–404.
78. Wiseman CV, Sunday SR, Halligan P, et al. Substance dependence and eating disorders: impact of sequence on comorbidity. *Compr Psychiatry*. 1999;40(5):332–336.
79. le Grange D, Binford RB, Peterson CB, et al. DSM-IV threshold versus subthreshold bulimia nervosa. *Int J Eat Disord*. 2006;39(6):462–467.
80. Krug I, Treasure J, Anderluh M, et al. Present and lifetime comorbidity of tobacco, alcohol and drug use in eating disorders: a European multicenter study. *Drug Alcohol Depend*. 2008;97(1–2):169–179.
81. Krahn D, Piper D, King M, et al. Dieting in sixth grade predicts alcohol use in ninth grade. *J Subst Abuse*. 1996;8(3):293–301.
82. Gadalla T, Piran N. Co-occurrence of eating disorders and alcohol use disorders in women: a meta analysis. *Arch Womens Ment Health*. 2007; 10(4):133–140.
83. Grigioni S, Lemercier A, Beaucreux M, et al. Tabac, poids et comportement alimentaire chez 1216 étudiantes. *Nutr clin metabol*. 2008;22 Suppl 1:46–47.
84. Benjamin L, Wulfert E. Dispositional correlates of addictive behaviors in college women: binge eating and heavy drinking. *Eat Behav*. 2005; 6(3):197–209.
85. Sinha R, O'Malley SS. Alcohol and eating disorders: implications for alcohol treatment and health services research. *Alcohol Clin Exp Res*. 2000;24(8):1312–1319.
86. Dechelotte P, Grigioni S, Fetissov S. Gastrointestinal disorders in anorexia nervosa. *Nutr Clin Metabol*. 2007;21:166–171.
87. Killen JD, Taylor CB, Telch MJ, et al. Self-induced vomiting and laxative and diuretic use among teenagers. Precursors of the binge-purge syndrome? *JAMA*. 1986;255(11):1447–1449.
88. Buddeberg-Fischer B, Bernet R, Schmid J, Buddeberg C. Relationship between disturbed eating behavior and other psychosomatic symptoms in adolescents. *Psychother Psychosom*. 1996;65(6):319–326.
89. Lau B, Alsaker FD. Dieting behavior in Norwegian adolescents. *Scand J Psychol*. 2001;42(1):25–32.
90. Winstead NS, Willard SG. Gastrointestinal complaints in patients with eating disorders. *J Clin Gastroenterol*. 2006;40(8):678–682.
91. Johnson J, Whitaker AH. Adolescent smoking, weight changes, and binge-purge behavior: associations with secondary amenorrhea. *Am J Public Health*. 1992;82(1):47–54.
92. Selzer R, Caust J, Hibbert M, et al. The association between secondary amenorrhea and common eating disordered weight control practices in an adolescent population. *J Adolesc Health*. 1996;19(1): 56–61.
93. Kreipe RE, Strauss J, Hodgman CH, Ryan RM. Menstrual cycle abnormalities and subclinical eating disorders: a preliminary report. *Psychosom Med*. 1989;51(1):81–86.
94. Stewart DE, Robinson E, Goldbloom DS, Wright C. Infertility and eating disorders. *Am J Obstet Gynecol*. 1990;163(4 Pt 1): 1196–1199.

95. Powers PS. Osteoporosis and eating disorders. *J Pediatr Adolesc Gynecol*. 1997;12(2):51–57.
96. Joyce JM, Warren DL, Humphries LL, et al. Osteoporosis in women with eating disorders: comparison of physical parameters, exercise, and menstrual status with SPA and DPA evaluation. *J Nucl Med*. 1990; 31(3):325–331.
97. Vestergaard P, Emborg C, Stoving RK, et al. Fractures in patients with anorexia nervosa, bulimia nervosa, and other eating disorders–a nationwide register study. *Int J Eat Disord*. 2002;32(3):301–308.
98. National Collaborating Centre for Mental Health Royal College of Psychiatrists' Research Unit. *Eating Disorders: Core Interventions in the Treatment and Management of Anorexia Nervosa, Bulimia Nervosa and Related Eating Disorders*. London: The British Psychological Society, Gaskell, The Royal College of Psychiatrists; 2004.
99. Vocks S, Tuschen-Caffier B, Pietrowsky R, et al. Meta-analysis of the effectiveness of psychological and pharmacological treatments for binge eating disorder. *Int J Eat Disord*. 2009;43(3):205–217.
100. Walsh B, Kaplan AS, Atia E, et al. Fluoxetine after weight restoration in anorexia nervosa: a randomized controlled trial. *JAMA*. 2006;295:2605–2612.
101. Schmidt U, Landau S, Pombo-Carril MG, et al. Does personalized feedback improve the outcome of cognitive-behavioural guided self-care in bulimia nervosa? A preliminary randomized controlled trial. *Br J Clin Psychol*. 2006;45(Pt 1):111–121.
102. Bara-Carril N, Williams CJ, Pombo-Carril MG, et al. A preliminary investigation into the feasibility and efficacy of a CD-ROM-based cognitive-behavioral self-help intervention for bulimia nervosa. *Int J Eat Disord*. 2004;35(4):538–548.
103. Fairburn CG, Harrison PJ. Eating disorders. *Lancet*. 2003;361: 407–416.
104. Comerci GD. Eating disorders in adolescents. *Pediatr Rev*. 1988;10(2): 37–47.
105. Treasure J, Claudino AM, Zucker N. Eating disorders. *Lancet*. 2009;375(9714):583–593.
106. Rigaud D, Brondel L, Poupard AT, et al. A randomized trial on the efficacy of a 2-month tube feeding regimen in anorexia nervosa: A 1-year follow-up study. *Clin Nutr*. 2007;26(4):421–429.
107. Shapiro JR, Berkman ND, Brownley KA, et al. Bulimia nervosa treatment: a systematic review of randomized controlled trials. *Int J Eat Disord*. 2007;40(4):321–336.
108. Hay PJ, Bacaltchuk J. Bulimia nervosa. *Clin Evid (Online)*. 2008:1009.
109. Bacaltchuk J, Hay P. Antidepressants versus placebo for people with bulimia nervosa. *Cochrane Database Syst Rev*. 2003;4:CD003391.
110. Hay PP, Bacaltchuk J, Stefano S, Kashyap P. Psychological treatments for bulimia nervosa and binging. *Cochrane Database Syst Rev*. 2009;4:CD000562.
111. Fairburn CG, Cooper Z, Doll HA, et al. Transdiagnostic cognitive-behavioral therapy for patients with eating disorders: a two-site trial with 60-week follow-up. *Am J Psychiatry*. 2009;166(3): 311–319.
112. Fairburn CG. *Cognitive Behavior Therapy and Eating Disorders*. New York: Guilford Press; 2008.
113. Hay P, Bacaltchuk J, Claudino A, et al. Individual psychotherapy in the outpatient treatment of adults with anorexia nervosa. *Cochrane Database Syst Rev*. 2003;4:CD003909.
114. Whitehouse AM, Cooper PJ, Vize CV, et al. Prevalence of eating disorders in three Cambridge general practices: hidden and conspicuous morbidity. *Br J Gen Pract*. 1992;42(355):57–60.
115. Ogg EC, Millar HR, Pusztai EE, Thom AS. General practice consultation patterns preceding diagnosis of eating disorders. *Int J Eat Disord*. 1997;22(1):89–93.
116. Morgan JF, Reid F, Lacey JH. The SCOFF questionnaire: assessment of a new screening tool for eating disorders. *BMJ*. 1999;319:1467–1468.
117. Garcia FD, Grigioni S, Allais E, et al. Detection of eating disorders in patients: validity and reliability of the French version of the SCOFF questionnaire. *Clin Nutr*. 2011; 30(2): 178–181.
118. Jarman FC, Rickards WS, Hudson IL. Late adolescent outcome of early onset anorexia nervosa. *J Paediatr Child Health*. 1991;27(4): 221–227.
119. Ratnasuriya RH, Eisler I, Szmukler GI, Russell GF. Anorexia nervosa: outcome and prognostic factors after 20 years. *Br J Psychiatry*. 1991; 158:495–502.
120. Currin L, Schmidt U. A critical analysis of the utility of an early intervention approach in the eating disorders. *J Ment Health*. 2005; 14(6):611–624.
121. Pratt BM, Woolfenden SR. Interventions for preventing eating disorders in children and adolescents. *Cochrane Database Syst Rev*. 2002;2:CD002891.