

Editorial: Eating Disorders: Update, Controversies, and the Israeli Perspective

Eating disorders (EDs) are currently considered among the most prevalent public health problems, reaching in many Western countries an epidemic proportion. Nevertheless, symptoms and syndromes of self-starvation are not a new, modern phenomenon, first appearing in the clinical descriptions of anorexia by Gull and Lasegue in the 19th century; but they have been observed for more than 1,500 years, being interpreted in each era according to prevailing beliefs and cultural norms (1). EDs, traditionally conceptualized as of psychosomatic origin, include anorexia nervosa (AN), bulimia nervosa (BN), binge eating disorders (BED), and eating disorders not otherwise specified (ED-NOS), also termed partial ED syndromes. The latter include a variety of maladaptive eating-related attitudes and behaviors that do not reach the severity of full-blown EDs (2), but are distinct from mere dieting in having the potential to induce significant morbidity. Furthermore, almost half of individuals with ED-NOS may progress to the full syndrome within several years (3).

Diagnostic Criteria

Diagnostic criteria of AN include refusal to maintain weight at or above a minimally normal weight for age and height, or failure to reach expected weight; fear of gaining weight although being underweight; disturbances in body perception, or undue influence of weight and shape on self-evaluation, or denial of seriousness of low weight; and amenorrhea in postmenarcheal females (in 33% AN appears before menarche) (DSM-IV, 1994). AN appears primarily during adolescence, although in recent years there is increasing evidence of first appearance of AN both in younger children and at an older age. AN usually starts as a restricting subtype, with 30-60% of restricting patients progressing to purging/ bingeing-purging AN or BN.

Diagnostic criteria of BN include recurrent episodes of uncontrolled binge eating; recurrent inappropriate compensatory behaviors to prevent weight gain, including in most cases purging behaviors such as self-induced vomiting (which represents around 85% of purging behaviors), and misuse of medications, particularly laxatives, but also non-purging behaviors such as fasting and excessive exercise; and undue influence of weight and shape on self-evaluation. The bingeing and purging behaviors in BN are not associated with low weight (i.e., they do not occur exclusively during AN episodes).

BED, a provisional diagnosis in the DSM-IV, is defined by bingeing behaviors similar to those occurring in BN, but with no compensatory behaviors to prevent weight gain. Around 30% of overweight individuals are diagnosed with BED. BN and BED appear primarily in young adults, and all EDs occur primarily in women, with 5-10% of the patients being male (4).

Prevalence, Comorbidity, Mortality Rates, and Outcome

The prevalence of AN in young females is currently estimated as 0.3%, and that of BN as 1% (4). Whereas the prevalence of BED is not yet well determined, it is likely higher than that of BN (4). The prevalence of partial ED (ED-NOS) syndromes is in the range of 3 to 22% (2). The wide ranges reflect lack of positive diagnostic criteria for the diagnosis of ED-NOS, so that there is no agreed way of determining what constitutes a "case," as well as differences in assessment tools (interviews vs. questionnaires) (3). Both AN and BN have shown an increase in incidence in recent years, although at least some studies (5) argue that what has risen is actually the frequency of treatment use, rather than real incidence.

AN and BN are associated with a high rate of

DSM-IV Axis I comorbidities. Between 40-70% of these patients have lifetime affective (mainly depressive) and anxiety (mainly obsessive compulsive and social phobia) disorders, and similar rates of patients with bingeing/purging AN and BN have lifetime substance use disorders (SUDs) (6, 7). These comorbid disorders are also found to a greater extent in first-degree relatives of AN and BN patients compared with healthy controls. SUDs usually appear after the onset of the ED (7), and affective disorders may appear before, during or after the onset of the ED, namely they do not necessarily reflect the influence of starvation. By contrast, the majority of anxiety disorders appear before the onset of AN and BN, so that they may potentially predispose to the development of these disorders (6).

The lifetime mortality rate in AN is between 5-20% (8), being higher in the case of bingeing-purging type AN, and considerably higher than that reported for most psychiatric disturbances. By contrast, low crude mortality rates in the range of 0.3-3% have been reported in BN (8).

AN and BN represent chronic disorders with recovery occurring mostly after 4-10 years from the start of the illness (9). Recovery — defined in terms of achieving normal weight, regular menstrual cycles and normal eating patterns with no evidence of restricting, bingeing or purging for a period of no less than one year — occurs in 40-50% of AN (9) and BN patients (10), and a subclinical course in 30-35% of these patients. Despite treatment, around 20% of AN (9) and BN (10) patients show a chronic non-remitting course over time. Furthermore, between 30-50% of BN patients may relapse within a few months to several years (10).

Less favorable outcome in AN is associated with longer duration of illness until receiving treatment, refusal to accept and maintain treatment, severe disturbances in body image, obsessional-ritualistic eating and physical exercise, presence of purging/bingeing and comorbid disorders, maladaptive relations with family members, dysfunctional social skills, and childhood sexual abuse (9). In BN, a less favorable outcome is associated with a longer duration of illness until receiving treatment, elevated obsessiveness (11) and particularly with multi-impulsivity, namely BN characterized by both vomiting and laxative abuse, elevated risk of self-injuri-

ous and suicidal behaviors, comorbid substance use disorders, impulse control disorders, and borderline personality disorder and childhood sexual abuse (12).

Etiology

EDs likely reflect complex, inter-dependent, multi-dimensional causalities (13). Dieting behaviors may be propelled into a full-blown disorder by an interaction of antecedent genetic, biological, psychological, familial and social conditions with various environmental influences. The study of ED etiology is highly problematic, primarily because of their relative rarity, which necessitates extremely large cohorts, the confounding influences of malnutrition and erratic consummatory patterns, the low rate of people who meet stringent diagnostic criteria for EDs than are seen in mental health care, and the influence of sociocultural and political concerns on the study of EDs.

EDs have been traditionally conceptualized as socio-cultural dependent syndromes, related primarily to the thin body ideal, and being of relevance predominantly in wealthier Western countries (1). Many factors may potentially support such a contention, including the different rates and presentations of EDs in different cultures (Western vs. non-Western cultures) and populations (females vs. males), or the different internalization of norms related to weight and shape in young women at risk for developing EDs, compared with healthy girls (1). More recent theories regard EDs as a cultural byproduct of modernity that cuts across geographic and economic lines, rather than being a strictly Western phenomenon (14). EDs are, thus, attributed to a combination of socioeconomic development, changing roles of women, a sociocultural emphasis on thinness, and a shift in eating patterns (14). Still other studies emphasize that the relative infrequency of full-blown EDs in comparison to the high rate of females who are dissatisfied with their weight and preoccupied with dieting signifies that sociocultural parameters may have only a limited role in the predisposition to EDs (5).

Epidemiological, family and twin studies show a major role of genetic factors in EDs, with the heritability estimates for AN and BN being in the

range of 0.54-0.80 (15). These studies show elevated rates of decreased/elevated weight, AN, BN and ED-NOS in first-degree relatives of patients with AN and BN, and elevated concordance rates of AN, BN, and/or ED-NOS disorders in monozygotic twins in whom the afflicted twin has AN or BN, compared to dizygotic twins. Candidate gene approaches and genome wide scans show particular relevance of specific polymorphisms of serotonin (5HT) transporter, dopamine receptor, melanocortin receptor, and catechol-O-methyl transferase genes in some forms of EDs.

Central nervous system (CNS) neuropeptides, including neurosteroids, opioids, neuropeptide-Y, peptide Y, vasopressin, oxytocin, cholecystokinin, leptin and adiponectin, and CNS neurotransmitters (serotonin, dopamine and norepinephrine), play an important role in the regulation of feeding behavior. However, as most of the neuropeptide alterations apparent during symptomatic episodes of AN and BN tend to normalize upon physical recovery, they likely represent consequences of malnutrition and maladaptive consummatory patterns, rather than their causes (16).

Recent neuroimaging studies have shown serotonin alterations in cingulate limbic structures, and in frontal, temporal and parietal cortical regions (17), and dopamine alterations in the antero-ventral striatum and dorsal caudate areas of the basal ganglia (18), that persist also in patients recovered from AN, and in some recovered cases of BN. Findings persisting in recovered patients may be a consequence of prior illness and explain why these patients still suffer from some physiological, behavioral and psychological handicaps. Nevertheless, the finding of neurotransmitter alterations in the absence of confounding nutritional influences raises the likelihood of them representing long-standing, premorbid traits (19). The elevated dopamine receptor activity and serotonin activity in recovered patients may potentially account not only for feeding-behavior dysfunctions such as extremes of appetitive behaviors, but also for personality traits found elevated both in recovered AN and BN patients, i.e., potentially predisposing to these disorders. These include elevated harm-avoidance (reflecting resistance to changes), behavioral inhibition, perfectionism, obsessiveness and negative emotionality, particu-

larly anxiety (11,16,19). These findings are in keeping with the elevated rate of Cluster III (avoidant, dependent, obsessive-compulsive) personality disorders in AN (20). In the case of BN, patients in the acute stage of the disorder demonstrate elevated rates of Cluster II personality disorders (particularly borderline personality disorder) and elevated impulsivity and novelty seeking (20). Recovered patients show significant attenuation of these traits, reflecting the influence of the erratic consummatory and emotional patterns in symptomatic BN patients — in which restraint and disinhibition often alternate — on personality presentation.

Shared environmental influences, namely experiences shared by all family members, have only a limited influence in the development of an ED. By contrast, according to twin studies, non-shared environmental influences, namely those experiences that are unique to one sibling and not shared by other siblings reared in the same family, account for 0.17-0.46 of the variance in the development of both AN and BN (21). Initial data indicate that differential parental relationships, body weight teasing, peer group experiences, and adverse life events may account for the development of eating pathology in one sibling versus another. Whereas the “psychosomatic family” — characterized by enmeshment, over-protectiveness, rigidity and lack of conflict resolution — was traditionally associated with the development of AN, most studies have found no evidence supporting a specific family constellation in the predisposition to an ED.

Treatment

The treatment of AN and BN requires multi-modal interventions, with nutritional rehabilitation being a necessary, albeit insufficient, prerequisite for recovery (13). Whereas many psychotherapeutic interventions have been applied in the treatment of AN, a specific structured family-based intervention appears to be the only evidence-based effective treatment for adolescents with short-term AN (22). Cognitive behavioral treatment (CBT) has established itself as the only evidence-based effective psychotherapeutic intervention for BN and BED (3). Specific serotonin reuptake inhibitors (SSRIs) are effective in the treatment of BN and BED, although

prolonged use may be accompanied by relapse (23). Combining CBT and SSRIs may be more effective than each intervention. Preliminary findings support the effectiveness of the 5-HT₃ antagonist ondansetron, and of topiramate in some patients with BN, and of sibutramine and topiramate in some overweight BED patients (23). There is as yet no established medication with the potential to improve weight gain in AN. A few studies support the role of SSRIs in reducing the risk of relapse in weight-restored AN patients. Atypical antipsychotics, most often olanzapine, have shown promise in a number of uncontrolled studies in chronic AN patients (23).

EDs in Israel

Israel is an important site for the study of EDs, particularly in relation to sociocultural aspects. This is because Israeli society encompasses various ethnic and religious groups, characterized by very old traditions on the one hand, yet being on the cutting-edge of the newest technology (24).

Whereas Israeli researchers have contributed significantly to the study of genetic, biological, medical, psychiatric, psychological, familial and treatment-related aspects of EDs, it is unfortunate that no systemized epidemiological studies have been conducted in Israel. Male and female Israeli adolescents have been found to be more dissatisfied with their weight and preoccupied to a greater extent with dieting and weight loss than adolescents in 34 other Western countries (25).

Disordered Eating in Diverse Sociocultural and Ethnic Groups in Israel

Judaism does not encourage asceticism, but supports enjoyment and pleasure with food as part of worshipping God. This may perhaps account for the more positive attitudes towards food and the lower rate of eating-related psychological and behavioral disturbances found in Jewish religious compared with secular adolescents and young adults, both in the U.S.A. and Israel (1).

Israel has received a wave of immigration from the former U.S.S.R. and Ethiopia in the last fifteen years. Youngsters emigrating from these countries to Israel face a sociocultural phase of transition (26), to

which they might react by adopting perceived standards of the Israeli youth subculture, such as the thin body ideal (25). Interestingly, a recent study conducted in an outpatient clinic has shown that the number of adolescents and young adult immigrants who seek treatment for disturbed eating is considerably lower in comparison to their percentage in the Israeli population. This finding, however, likely reflects maladaptive help-seeking characteristics, rather than the actual prevalence of eating disturbances in young immigrants.

Female Arab adolescents and young adults generally show low incidences of EDs compared to Western populations (26). Whereas a similar trend has been shown in Israeli-Arab girls, as reflected by a low representation rate among referrals to ED clinics, different findings emerge when relating to attitudes to eating, weight and physical appearance, in that young Arab girls are found to be as disturbed as Jewish Israeli girls (24). Studies in different Israeli-Arab sub-populations have found that Circassian adolescents have the lowest scores for eating-related pathology, whereas Bedouin adolescents have the highest (27). The authors relate these findings to the fact that Circassian adolescents live in small, relatively self-contained communities, while Bedouin women, formerly isolated, are currently entering mainstream Israeli society, being in this respect a highly vulnerable group in transition.

In the early years of the kibbutz, until 1965, AN was considered rare. Over a 25-year period, its annual incidence has increased by 800% (28). Whereas in the 1980s, kibbutz adolescents were found to have the highest scores for pathological eating-related attitudes and behaviors, more recent studies have shown kibbutz adolescents to endorse the lowest scores compared to any other Israeli sub-population, despite having the highest representation rate among referrals to outpatient ED clinics (24, 27, 29). These changes may reflect various processes that female kibbutz members have encountered throughout the years concerning female gender identity and gender role, and/or high awareness and usage of available treatment options.

References

1. Witztum E, Stein D, Latzer Y. Anorexia nervosa as a

- culture bound phenomenon in the era of globalization. In: Wiesel-Lev R, Zwickel J, and Barak N, editors. *Mental health of Israeli women*. Jerusalem: The Brookdale Institute, 2005: pp. 205-228 (in Hebrew).
2. Shisslak CM, Crayo M, Estes LS. The spectrum of eating disturbances. *Int J Eat Disord* 1995;18:209-219.
 3. Fairburn CG, Harrison PJ. Eating disorders. *Lancet* 2003;361:407-416.
 4. Hoek HW, van Hoeken D. Review of the prevalence and incidence of eating disorders. *Int J Eat Disord* 2003;34:383-396.
 5. Keel PK, Klump KL. Are eating disorders culture-bound syndromes? Implications for conceptualizing their etiology. *Psychol Bull* 2003;129:747-769.
 6. Kaye WH, Bulik CM, Thornton L, Barbarich N, Masters K. Comorbidity of anxiety disorders with anorexia and bulimia nervosa. *Am J Psychiatry* 2004;161:2215-2221.
 7. Bulik CM, Klump KL, et al. Alcohol use disorder comorbidity in eating disorders: A multicenter study. *J Clin Psychiatry* 2004;65:1000-1006.
 8. Keel PK, Dorer DJ, Eddy KT, Franko D, Charatan DL, Herzog DB. Predictors of mortality in eating disorders. *Arch Gen Psychiatry* 2003;60:179-183.
 9. Steinhausen HC. The outcome of anorexia nervosa in the 20th century. *Am J Psychiatry* 2002;159:1284-1293.
 10. Keel PK, Mitchell JE, Miller KB, Davis TL, Crow SJ. Long-term outcome of bulimia nervosa. *Arch Gen Psychiatry* 1999;56:63-69.
 11. von Ranson KM, Kaye WH, Weltzin TE, Rao R, Matsunaga H. Obsessive-compulsive disorder symptoms before and after recovery from bulimia nervosa. *Am J Psychiatry* 1999;156:1703-1708.
 12. Lacey JH. Self-damaging and addictive behavior in bulimia nervosa: A catchment area study. *Br J Psychiatry* 1993;163:190-194.
 13. Halmi KA. Eating disorders in females: Genetics, pathophysiology, and treatment. *J Pediatr Endocrinol Metab* 2002;15 Suppl 5:1379-1386.
 14. Nasser M, Katzman MA, Gordon RA, editors. *Eating disorders and cultures in transition*. London: Brunner-Routledge, 2001.
 15. Bulik CM, Tozzi F. The genetics of bulimia nervosa. *Drugs Today* 2004;40:741-749.
 16. Kaye W, Strober M, Jimerson D. The neurobiology of eating disorders. In: Charney DS, Nestler EJ, editors. *The neurobiology of mental illness*. New York: Oxford, 2004: pp. 1112-1128.
 17. Kaye WH, Frank GK, Bailer UF, Henry SE, Meltzer CC, Price JC, Mathis CA, Wagner A. Serotonin alterations in anorexia and bulimia nervosa: New insights from imaging studies. *Physiol Behav* 2005 19;85:73-81.
 18. Frank GK, Bailer UF, et al. Increased dopamine D2/D3 receptor binding after recovery from anorexia nervosa measured by positron emission tomography and [(11)C]Raclopride. *Biol Psychiatry* 2005; June 28.
 19. Klump KL, Strober M, Bulik CM, et al. Personality characteristics of women before and after recovery from an eating disorder. *Psychol Med* 2004;34:1407-1418.
 20. Vitousek K, Manke F. Personality variables and disorders in anorexia nervosa and bulimia nervosa. *J Abnorm Psychol* 1994;103:137-147.
 21. Klump KL, Wonderlich S, Lehoux P, Lilienfeld LR, Bulik CM. Does environment matter? A review of nonshared environment and eating disorders. *Int J Eat Disord* 2002;3:118-135.
 22. Lock J, le Grange D. Family-based treatment of eating disorders. *Int J Eat Disord* 2005;37 Suppl:S64-67, S87-89.
 23. Pederson KJ, Roerig JL, Mitchell JE. Towards the pharmacotherapy of eating disorders. *Expert Opin Pharmacother* 2003;4:1659-1678.
 24. Latzer Y. Disordered eating behaviors and attitudes in diverse groups in Israel. In: Ruggiero GM, editor. *Eating disorders in the Mediterranean area: An exploration in transcultural psychology*. Hauppauge, New York: Nova Science, 2003: pp. 159-181.
 25. Harel Y, Ellenbogen-Frankovits S, Molcho M, Abu-Ashas K, Habib J. *Youth in Israel*. Jerusalem: Brookdale Institute, 2002.
 26. Nasser M. Eating disorders: The cultural dimension. *Soc Psychiatry Psychiatr Epidemiol* 1988;23:184-187.
 27. Apter A, Abu Shah M, Iancu I, Abramovitch H, Weizman A, Tyano S. Cultural effects on eating attitudes in Israeli subpopulations and hospitalized anorectics. *Genet Soc Gen Psychol Monogr* 1994;120: 83-99.
 28. Kaffman M, Sadeh T. Anorexia nervosa in the kibbutz: Influencing the development of monoideistic fixation. *Int J Eat Disord* 1989;8:33-35.
 29. Latzer Y, Tzischinsky O. Eating attitudes in a varied group of Israeli adolescent females: A comparison study. *J Adolesc* 2005;28:317-323.

Yael Latzer, Rambam Medical Center,
Haifa

Eitan Gur, Chaim Sheba Medical Center,
Tel Hashomer

Daniel Stein, Chaim Sheba Medical Center,
Tel Hashomer
Guest Editors