

Psychiatric Comorbidity and Eating Disorder Inventory (EDI) Profiles in Eating Disorder Patients

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Objective: This study examines potential overlaps between psychiatric comorbidity (Axis I and II) and scores on the subscales of the Eating Disorder Inventory (EDI) in women with eating disorders (EDs).

Method: In a sample of 248 women (72 with anorexia nervosa, 140 with bulimia nervosa, and 36 with eating disorders not otherwise specified), we determined psychiatric comorbidity using the Structured Clinical Interview for DSM-IV. Behavioural and psychological characteristics of EDs were quantified with the EDI.

Results: Psychiatric comorbidity was high in both axes (74% for Axis I and 68% for Axis II). While most EDI subscales pertaining to psychological traits showed significant associations with Axis I and II disorders, the subscales concerning eating and perception of weight and shape were much less associated with psychiatric comorbidity. Affective and anxiety disorders, as well as personality disorders of clusters A and C, showed a similar pattern with links to most psychological subscales. The profile for substance-related disorders was different, showing associations with the Ineffectiveness and Interoceptive Awareness scales. Personality disorders of cluster B were related only to the Bulimia subscale and not to any of the psychological subscales.

Conclusions: The EDI appears to primarily reflect Axis I and II disorders related to affective and anxiety problems. Clinicians and researchers employing the EDI should be aware that it is not sensitive for all forms of comorbidity prevalent in ED patients.

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Clinical Implications

- In clinical work with eating disorder (ED) patients it is crucial to conduct a comprehensive assessment of DSM-IV comorbid disorders.
- The Eating Disorder Inventory (EDI) subscales are generally sensitive to Axis I and II disorders but not to cluster B personality disorders.
- The EDI primarily reflects Axis I and II disorders related to affective and anxiety problems.

Limitations

- Because the sample was selective and not randomized, it may not be representative of all subjects with EDs.
- Participants may have several comorbid disorders in one or both axes, and certain patterns of accumulated disorders may be more frequent than others.
- To limit the overall number of comparisons, we did not examine associations between EDI subscales and comorbidity for differences among ED subtypes.

Key Words: eating disorders, psychiatric comorbidity, Axis I, Axis II, Eating Disorder Inventory, anorexia nervosa, bulimia nervosa, eating disorders not otherwise specified

Most persons with the clinical diagnosis of eating disorder (ED) anorexia nervosa (AN), bulimia nervosa (BN), and eating disorders not otherwise specified (EDNOS) have additional psychiatric diagnoses (1,2). The accompany-

ing Axis I and II diagnoses play an important role in the planning of specific therapeutic strategies. Various outcome studies have shown that comorbid disorders could be seen as an aggravating factor for the course of EDs (3–6).

In clinical practice and in ED research, a multidimensional self-rating instrument called the Eating Disorder Inventory (EDI) is widely used. The development of this instrument was based on the multifactorial etiology of EDs. The 8 subscales of the EDI assess cognitive-behavioural symptoms commonly found in persons with EDs, as well as psychological correlates or personality characteristics (7) present in, but not exclusive to, EDs (8,9). The EDI has been used as a prognostic indicator in outcome studies (10,11), as an outcome measurement (12–14), and as a measurement of symptom evolution during treatment (15,16).

Associations between comorbidity and constructs measured by the EDI may exist. Several studies suggest that some EDI subscales may be influenced by the presence of other psychiatric disorders, such as obsessive-compulsive disorders, depression, or personality disorders (17–19). Because both comorbidity and EDI scores are frequently used as predictors in studies examining the course of EDs, associations between these constructs would represent important mediating mechanisms that should be considered in analyzing and evaluating results. However, to the best of our knowledge, no studies have analyzed the interplay of the whole range of Axis I and II comorbidity and the EDI subscales. This study examines associations between psychiatric comorbidity of Axis I and II and the EDI subscales and identifies possible overlaps between these different diagnostic approaches.

Material and Methods

Participants

This study was approved by the Research Ethics Commission of the Psychiatric University Hospital of Zurich. Participants were enrolled during a 24-month period (from March 1997 to March 1999). Criteria for inclusion in the study were a current DSM-IV diagnosis of ED, a minimum age of 17 years, and the ability to speak German with adequate fluency. All participants received detailed information about the procedures and aims of the study, and all gave written informed consent. Overall, we evaluated 300 persons with EDs. We consecutively recruited a total of 67 participants from the ED inpatient unit of the University Hospital in Zurich; we consecutively recruited 66 participants as outpatients of the psychiatric department of the University Hospital in Zurich; and we recruited 31 participants via contacts with ED self-help groups in the Zurich area. We enrolled an additional 136 participants with a clinical diagnosis of ED via advertisements in local newspapers, to which 280 persons responded.

To allow comparisons with ED literature, we excluded 11 women over age 50 years and 12 men. We excluded an additional 29 women because of missing data (that is, questionnaires were incomplete or not returned). The sample we

investigated comprised 248 participants; specifically, 72 (29%) AN participants (mean Body Mass Index [BMI] 15.2, SD 1.5), 140 (56%) BN participants (mean BMI 21.5, SD 3.4), and 36 (15%) EDNOS participants (mean BMI 22.2, SD 6.1). Thirty-four (47%) AN cases were of the restrictive type and 38 (53%) were of the binge-purging type. Of the BN cases, 129 (92%) were of the binge-purging type, and 11 (8%) were of the nonpurging type.

Mean age at the time of interview was 28.1 years (SD 7.4 years), with AN subjects (mean 26.0 years, SD 6.5) being younger than both BN subjects (mean 28.5 years, SD 7.5) ($z = 2.2, P = 0.03$) and EDNOS subjects (mean 30.7 years, SD 8.0) ($z = 3.0, P = 0.003$). The average age of ED onset (as reported by participants) was 17.2 years (SD 3.9 years) and did not vary significantly among ED diagnoses groups. Thus, the average ED duration of AN (mean 8.4 years, SD 6.8) was shorter than that of both BN (mean 11.2 years, SD 7.9) and EDNOS (mean 13.7 years, SD 9.3).

Measures and Procedure

We collected the data during the baseline phase of a mixed retrospective-prospective survey examining the course of EDs. We diagnosed ED and psychiatric comorbidity using the German version of the Structured Clinical Interview for Axis I and II of the DSM-IV (20), which was conducted by 4 psychologists who did not meet with the participants outside the study interviews (interrater reliability 0.8). All diagnoses were discussed and finalized with the first author. As part of a comprehensive questionnaire package the participants completed the EDI (21). The EDI is a widely used self-reporting measure of ED symptoms and consists of 8 standardized subscales representing dimensions that are clinically relevant to ED. The first 3 dimensions deal with attitudes and behaviour concerning eating, weight, and body shape, namely, Drive for Thinness (DT), Bulimia (B), and Body Dissatisfaction (BD). The remaining 5 dimensions tap more general psychological and relational constructs relevant to ED, namely, Ineffectiveness (I), Perfectionism (P), Interpersonal Distrust (ID), Interoceptive Awareness (IA), and Maturity Fears (MF).

Statistical Analysis

Because the distribution of age, the age of ED onset, and the distribution of several EDI scales was skewed, we conducted group comparisons in these variables using nonparametric tests (the Kruskal-Wallis [KW] and Mann-Whitney *U*). All tests were 2-tailed, and alpha levels were set at 5%. For post hoc tests, we set the minimum alpha level at 1% to reduce the risk of inflating the probability of type 1 errors. In addition, because of the large number of comparisons to make when testing associations between comorbidity and EDI scales, we assumed a minimum alpha level of 1%.

Table 1 EDI scores for total sample and by Axis I comorbidity

EDI scales	Total n = 248		Any Axis I disorder n = 184		Affective disorder n = 129		Substance-related disorder n = 186		Anxiety disorder n = 134	
	Mean (SD)	P	No n = 64	Yes n = 184	No n = 119	Yes n = 129	No n = 186	Yes n = 62	No n = 114	Yes n = 134
Drive for thinness (DT)	11.1 (5.7)	0.007	9.4 (5.5)	11.6 (5.7)	10.2 (5.6)	11.9 (5.8)	10.5 (5.7)	12.6 (5.5)	10.4 (5.8)	11.6 (5.7)
Bulimia (B)	7.8 (5.8)	—	6.4 (5.2)	8.3 (6.0)	7.3 (5.8)	8.3 (5.8)	7.2 (5.5)	9.8 (6.3)	7.0 (5.6)	8.6 (5.9)
Body dissatisfaction (BD)	13.6 (8.2)	—	12.9 (8.6)	13.8 (8.0)	13.7 (8.5)	13.5 (7.9)	13.0 (8.2)	15.2 (7.8)	13.5 (8.6)	13.6 (7.8)
Ineffectiveness (I)	8.9 (6.8)	<0.001	4.7 (5.1)	10.3 (6.7)	5.9 (5.7)	11.6 (6.6)	8.0 (6.5)	11.5 (7.0)	7.0 (6.5)	10.4 (6.6)
Perfectionism (P)	7.6 (4.5)	—	6.4 (4.1)	8.0 (4.6)	6.6 (4.2)	8.5 (4.7)	7.2 (4.4)	8.7 (4.8)	6.7 (4.2)	8.3 (4.7)
Interpersonal distrust (ID)	4.6 (4.2)	<0.001	3.0 (3.3)	5.2 (4.3)	3.4 (3.6)	5.6 (4.4)	4.3 (4.0)	5.3 (4.7)	3.9 (4.0)	5.1 (4.2)
Intercept awareness (IA)	9.3 (6.4)	<0.001	6.6 (4.8)	10.3 (6.6)	7.8 (5.5)	10.7 (6.8)	8.7 (6.1)	11.3 (6.7)	7.7 (5.5)	10.7 (6.8)
Maturity fears (MF)	5.1 (4.8)	0.004	3.6 (3.6)	5.7 (5.1)	4.5 (4.9)	5.7 (4.8)	5.2 (5.0)	5.0 (4.4)	4.5 (4.5)	5.6 (5.1)

Data are presented as mean (SD); P values are based on Mann Whitney U tests, only P values <0.01 are noted

Results

The 3 diagnostic subgroups (AN, BN, and EDNOS) were compared regarding their scores in the EDI subscales. Significant overall differences emerged for EDI scales B (KW $\chi^2 = 42.0$, df 2, $P < 0.001$), I (KW $\chi^2 = 6.4$, df 2, $P = 0.04$), and MF (KW $\chi^2 = 6.7$, df 2, $P = 0.034$). Post hoc tests showed that BN subjects had higher B scores (mean 9.8, SD 5.3) than did AN subjects (mean 4.8, SD 5.7) and EDNOS subjects (mean 6.2, SD 4.8) (both $P < 0.001$). None of the post hoc tests for I and MF were significant at the 1% level.

In addition to the ED, 74% ($n = 184$) of patients had another lifetime Axis I disorder (that is, a current or remitted disorder). Most common were anxiety disorders (54%, $n = 134$), affective disorders (52%, $n = 129$) and substance-related disorders (25%, $n = 75$). Other types of Axis I disorders were rare (< 3%). The rate of Axis II disorders was also high (68%, $n = 169$). The most commonly observed Axis II disorders belonged to cluster C (anxious-fearful; 53%, $n = 131$). The second largest group was constituted by disorders of cluster B (dramatic-emotional-erratic; 21%, $n = 51$). Cluster A disorders (odd-eccentric) were diagnosed in 8% ($n = 20$). Depressive personality disorders were diagnosed in 25% ($n = 63$) and negativistic personality disorders in 5% ($n = 13$) (see DSM-IV appendix B). We grouped these last 2 together in the category “depressive or negativistic personality disorders” (28%, $n = 70$). Only 16% ($n = 39$) of subjects had a pure ED with no psychiatric comorbidity, 16% ($n = 40$) had only Axis I comorbidity, and 10% ($n = 25$) had only Axis II comorbidity. Most patients (58%, $n = 144$) had both Axis I and Axis II comorbidity.

Groups with and without Axis I comorbidity were compared regarding EDI scores (Table 1). Of the 8 EDI subscales, 5 showed strong associations with Axis I comorbidity in general (DT, I, ID, IA, MF). Substance-related disorders and anxiety disorders were all associated with higher levels of I and IA. Affective disorders were linked with higher levels of P and ID, and anxiety disorders were linked with higher levels of P.

Table 2 compares participants with and without Axis II comorbidity regarding EDI. All 5 of the EDI scales tapping into more general psychological constructs were related to Axis II

Table 2 EDI scores for total sample and by Axis II comorbidity

EDI scales	Any Axis II disorder			Cluster A			Cluster B			Cluster C			Depressive or negativistic PD		
	No n = 79	Yes n = 169	P	No n = 228	Yes n = 20	P	No n = 197	Yes n = 51	P	No n = 197	Yes n = 51	P	No n = 117	Yes n = 131	P
Drive for thinness (DT)	9.6 (5.7)	11.8 (5.6)	0.006	10.8 (5.7)	14.1 (5.4)	—	11.0 (5.8)	11.4 (5.5)	—	10.3 (5.8)	11.8 (5.7)	—	10.4 (5.7)	12.8 (5.5)	0.003
Bulimia (B)	7.3 (5.6)	8.1 (5.9)	—	7.6 (5.7)	10.3 (6.5)	—	7.3 (5.7)	9.7 (5.8)	0.006	8.0 (5.9)	7.6 (5.8)	—	7.6 (5.6)	8.5 (6.4)	—
Body dissatisfaction (BD)	12.5 (8.5)	14.1 (8.0)	—	13.3 (8.1)	16.7 (8.5)	—	13.4 (8.1)	14.2 (8.5)	—	13.7 (8.3)	13.5 (8.1)	—	12.8 (8.1)	15.5 (8.0)	—
Ineffectiveness (I)	5.1 (4.6)	10.6 (6.9)	< 0.001	8.4 (6.5)	14.1 (7.3)	0.001	8.6 (6.7)	10.0 (6.9)	—	6.8 (6.1)	10.7 (6.8)	< 0.001	7.2 (6.0)	13.1 (6.8)	< 0.001
Perfectionism (P)	5.6 (3.9)	8.5 (4.5)	< 0.001	7.3 (4.5)	11.0 (4.1)	0.001	7.4 (4.4)	8.3 (4.9)	—	6.1 (4.1)	8.9 (4.5)	< 0.001	6.9 (4.4)	9.2 (4.6)	0.001
Interpersonal distrust (ID)	3.2 (3.6)	5.2 (4.3)	< 0.001	4.3 (4.1)	7.9 (4.2)	< 0.001	4.5 (4.2)	5.0 (3.9)	—	3.7 (3.8)	5.4 (4.3)	0.001	4.1 (3.9)	5.8 (4.5)	0.004
Intercept awareness (IA)	6.5 (5.0)	10.6 (6.5)	< 0.001	8.8 (6.1)	14.6 (6.6)	< 0.001	8.8 (6.2)	11.1 (6.7)	—	7.4 (5.4)	11.0 (6.7)	< 0.001	8.3 (6.0)	11.9 (6.6)	< 0.001
Maturity fears (MF)	3.5 (3.5)	5.9 (5.2)	0.001	5.0 (4.7)	6.9 (5.7)	—	5.0 (4.9)	5.6 (4.8)	—	4.1 (4.3)	6.0 (5.1)	0.002	4.6 (4.6)	6.5 (5.3)	0.005

Data are presented as mean (SD). P values are based on Mann Whitney U tests, only P values < 0.01 are noted

comorbidity in general (I, P, ID, IA, MF). Of the scales relating to ED symptoms, only DT was related to general Axis II comorbidity. When examining specific clusters, the scales I, P, ID, and IA were all associated with cluster A disorders, cluster C disorders, and depressive or negativistic personality disorders. In addition, depressive or negativistic disorders were related to a higher DT and greater MF, cluster C disorders were related to greater MF, and cluster B disorders showed no association with any of the EDI scales, except the B scale.

Discussion

This study determines the psychiatric comorbidity of Axis I and II in a large sample of persons suffering from EDs. We also examine associations between comorbidity and EDI subscales, which, to our knowledge, has not been done before.

As expected, participants with BN had higher scores in the EDI B subscale than did both AN and EDNOS subjects. Consistent with reports by Garner (9), differences between the diagnostic subgroups in the other EDI subscales were not detectable.

The comorbidity of Axis I and II was high. The most common Axis I disorders were affective, anxiety, and substance-related disorders; for Axis II, most common were personality disorders of cluster C. Consistent with existing research on ED, the results of this study show that comorbidity in both axes was predominantly characterized by disorders of an anxious and depressive nature (1,3,22).

Although the EDI should not be considered an exhaustive sampling of psychopathological characteristics (9), our results show that most of the psychological EDI subscales were sensitive to the presence of additional DSM-IV psychiatric disorders. The analysis of Axis I disorders shows that participants with affective and anxiety disorders presented similar EDI profiles. Participants with such disorders had significantly higher scores in nearly all psychological subscales than did participants without such disorders. In line with these findings, clinical practice shows that inadequacy, insecurity, and poor self-esteem (I); reluctance to form close relationships (ID); and confusion and apprehension in recognizing and accurately responding to emotional states (IA) are typical impairments in persons with depression and anxiety disorders. Perfectionism (P) in participants with anxiety disorders was higher than in those without. This result is not surprising and likely reflects a construct overlap between obsessive-compulsive disorders and P.

EDI profiles associated with substance-related disorders show a different pattern from EDI profiles associated with affective and anxiety disorders. Of the 3 EDI subscales relating to ED symptomatology, only the B scale differentiated significantly between participants with substance-related disorders and those without. This finding is consistent with observations that BN and addiction problems are closely related (1,23–26). Of the 5 psychological subscales, only I and IA were associated with substance-related disorders. Indeed, the psychopathology of patients with substance-related disorders is often characterized by a lack of

control over their own lives, insecurity, and confusion and apprehension in recognizing and accurately responding to emotional states.

Associations between the MF subscale and additional Axis I psychiatric disorders were overall less pronounced than for the other EDI psychological subscales. This may indicate that this construct is relatively specific to the ED syndrome and less sensitive for other forms of additional psychiatric problems.

When examining personality disorder clusters, similar patterns emerged for cluster A, cluster C, and depressive-negativistic personality disorders, all showing associations with most EDI psychological subscales. However, the subscales related to attitudes and behaviours concerning eating, weight, and body shape showed no significant results. The 5 psychological subscales assess impairments in interpersonal relationships and intrapersonal life, which are frequently considered typical difficulties of patients with personality disorders (27,28). Our findings were partially comparable with the results of Yates and colleagues (29). These authors found that in BN patients, personality disorders were linked with higher scores in 2 EDI psychological subscales (I and ID) but also in 2 of the ED symptomatology subscales (DT and BD). However, comparability of results is somewhat curtailed because the study by Yates and colleague did not differentiate between specific types or clusters of personality disorders.

An interesting finding of this study was the difference in EDI profiles between participants with and without cluster B personality disorders. Only subscale B was associated with the presence of cluster B disorders. Indeed, overlaps between BN symptoms and symptoms of personality disorders of cluster B (including borderline personality disorders) have been pointed out (30). None of the psychological EDI subscales were sensitive to this cluster. These results are consistent with findings by Sunday and colleagues, who reported that borderline personality characteristics had little influence on EDI profiles in patients with BN (19). Several mechanisms might explain this pattern of results. The central clinical characteristics of persons with cluster B disorders include impulsiveness, emotional instability with rapid mood changes, and a tendency to self-aggrandize. This symptomatology often impairs patients' ability to perceive and recognize their own emotional states and processes, which may in turn decrease the probability that they will endorse items describing psychopathology. In fact, it is conceivable that patients with cluster B disorders have difficulties in completing any instruments that require self-observation and self-reflection. In addition, the pronounced impulsiveness of these patients could interfere with the self-regulatory processes necessary for completing questionnaires (for example, concentration, reading items carefully, and deciding on the most appropriate answer). However, participants with cluster B disorders likely also have high levels of affective and anxiety comorbidity, which may confound specific associations with EDI subscales.

Several methodological issues should be considered in evaluating our results. As in most clinical research on ED, the representativeness of our sample is curtailed because of the

selective recruitment sources and lack of randomization. To limit the overall number of comparisons, we did not distinguish between ED subtypes in our analyses. When interpreting the results, one should consider that participants might have several comorbid disorders in one or both axes. Some patterns of accumulated comorbid disorders may be more frequent than others. Thus, the comorbid disorders cannot be assumed to be independent of each other. Further, the assessment of Axis I and II comorbidity can be subject to a state-trait confusion in patients in an acute dysphoric or anxious Axis I state (1,31). When comparing results to other studies, it should be noted that the assessment of Axis II disorders has been shown to be (to some degree) dependent on the diagnostic instrument used (31,32). Because of the study's cross-sectional design, none of the identified associations can be interpreted as causal.

In longitudinal ED research, both psychiatric comorbidity and the EDI have been used as outcome measurements, and both have been implemented as predictors of outcome (5,11,19,33,34). The associations between comorbidity and EDI scales identified in this study could represent important mediating mechanisms that should be considered in prospective research employing comorbidity or the EDI as measures. Although the EDI was not explicitly intended to be a measure of ED severity (9), it is frequently employed in this manner (16,35,36). The results of our study caution against such use of the instrument, since associations with comorbidity indicate that the EDI may reflect the severity of a person's global psychopathology. The psychological subscales of the EDI appear to capture difficulties in intrapersonal and interpersonal relationships that can result from additional disorders of Axis I and Axis II, particularly disorders of an anxious and affective nature. Thus, the question of whether ED severity, when quantified on a behavioural level (for instance, frequency of binge-purge attacks), is associated with comorbidity remains open. Our findings indicate that clinicians and researchers employing the EDI ought to be aware that it is not sensitive for all forms of comorbidity prevalent in ED patients. Further, patients with cluster B disorders may provide biased responses in the EDI and other self-report measures.

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References

1. Braun DL, Sunday SR, Halmi KA. Psychiatric comorbidity in patients with eating disorders. *Psychol Med* 1994;24:859-67.
2. Milos GF, Spindler AM, Buddeberg C, Cramer A. Axis I and II comorbidity and treatment experiences in eating disorder subjects. *Psychother Psychosom* 2003;72:276-85.
3. Herzog DB, Nussbaum KM, Marmor AK. Comorbidity and outcome in eating disorders. *Psychiatr Clin North Am* 1996;19:843-59.
4. Herpertz-Dahlmann BM, Wewetzer C, Schulz E, Remschmidt H. Course and outcome in adolescent anorexia nervosa. *Int J Eat Disord* 1996;19:335-45.
5. Herpertz-Dahlmann B, Muller B, Herpertz S, Heussen N, Hebebrand J, Remschmidt H. Prospective 10-year follow-up in adolescent anorexia

- nervosa—course, outcome, psychiatric comorbidity, and psychosocial adaptation. *J Child Psychol Psychiatry* 2001;42:603–12.
6. Saccomani L, Savoini M, Cirrincione M, Vercellino F, Ravera G. Long-term outcome of children and adolescents with anorexia nervosa: study of comorbidity. *J Psychosom Res* 1998;44:565–71.
 7. Nevoen L, Broberg AG. Validating the Eating Disorder Inventory-2 (EDI-2) in Sweden. *Eat Weight Disord* 2001;6:59–67.
 8. Garner DM. Development and validation of a multidimensional eating disorder inventory for anorexia nervosa and bulimia nervosa. *Int J Eat Disord* 1983;2:15–34.
 9. Garner DM. *Eating Disorder Inventory-2: professional manual* 1991, Odessa (FL): Psychological Assessment Resources; 1991.
 10. Bizeul C, Sadowsky N, Rigaud D. The prognostic value of initial EDI scores in anorexia nervosa patients: a prospective follow-up study of 5 to 10 years. *Eating Disorder Inventory. Eur Psychiatry* 2001;16:232–8.
 11. Bulik CM, Sullivan PF, Joyce PR, Carter FA, McIntosh VV. Predictors of 1-year treatment outcome in bulimia nervosa. *Compr Psychiatry* 1998;39:206–14.
 12. Sohlberg S, Norring C. Co-occurrence of ego function change and symptomatic change in bulimia nervosa: a six-year interview-based study. *Int J Eat Disord* 1995;18:13–26.
 13. Kordy H, Percevic R, Martinovich Z. Norms, normality, and clinical significant change: implications for the evaluation of treatment outcomes for eating disorders. *Int J Eat Disord* 2001;30:176–86.
 14. Zubiate JK, Demitrack MA, Fenick A, Krahn DD. Obsessionality in eating-disorder patients: relationship to clinical presentation and two-year outcome. *J Psychiatr Res* 1995;29:333–42.
 15. Blouin JH, Carter J, Blouin AG, Tener L, Schnare-Hayes K, Zuro C, and others. Prognostic indicators in bulimia nervosa treated with cognitive-behavioral group therapy. *Int J Eat Disord* 1994;15:113–23.
 16. Bean P, Weltzin T. Evolution of symptom severity during residential treatment of females with eating disorders. *Eat Weight Disord* 2001;6:197–204.
 17. Thiel A, Broocks A, Ohlmeier M, Jacoby GE, Schussler G. Obsessive-compulsive disorder among patients with anorexia nervosa and bulimia nervosa. *Am J Psychiatry* 1995;152:72–5.
 18. North C, Gowers S. Anorexia nervosa, psychopathology, and outcome. *Int J Eat Disord* 1999;26:386–91.
 19. Sunday SR, Levey CM, Halmi KA. Effects of depression and borderline personality traits on psychological state and eating disorder symptomatology. *Compr Psychiatry* 1993;34:70–4.
 20. American Psychiatric Association. *Diagnostic and statistical manual of mental disorders*. 4th ed. Washington (DC): American Psychiatric Association; 1994.
 21. Garner D, Olmsted MP, Polivy J. The development and validation of a multidimensional eating disorders inventory for anorexia nervosa and bulimia nervosa. *Int J Eat Disord* 1983;2:15–34.
 22. Halmi KA, Eckert E, Marchi P, Sampugnaro V, Apple R, Cohen J. Comorbidity of psychiatric diagnoses in anorexia nervosa. *Arch Gen Psychiatry* 1991;48:712–8.
 23. Laessle RG, Wittchen HU, Fichter M, Pirke KM. The significance of subgroups of bulimia nervosa and anorexia nervosa: lifetime frequency of psychiatric disorders. *Int J Eat Disord* 1989;8:569–74.
 24. Schuckit MA, Tipp JE, Anthenelli RM, Bucholz KK, Hesselbrock VM, Nurnberger JI Jr. Anorexia nervosa and bulimia nervosa in alcohol-dependent men and women and their relatives. *Am J Psychiatry* 1996;153:74–82.
 25. Holderness CC, Brooks-Gunn J, Warren MP. Co-morbidity of eating disorders and substance abuse review of the literature. *Int J Eat Disord* 1994;16:1–34.
 26. Kaye WH, Wisniewski L. Vulnerability to substance abuse in eating disorders. *NIDA Res Monogr* 1996;159:269–321.
 27. Westen D, Harnden-Fischer J. Personality profiles in eating disorders: rethinking the distinction between axis I and axis II. *Am J Psychiatry* 2001;158:547–62.
 28. Wonderlich S, Mitchell JE. The role of personality in the onset of eating disorders and treatment implications. *Psychiatr Clin North Am* 2001;24:249–58.
 29. Yates WR, Sieleni B, Bowers WA. Clinical correlates of personality disorders in bulimia nervosa. *Int J Eat Disord* 1989;8:473–77.
 30. Wonderlich S, Swift W. Borderline versus other personality disorders in the eating disorders: clinical description. *Int J Eat Disord* 1990;9:629–38.
 31. Westen D, Arkowitz-Western L. Limitations of axis II in diagnosing personality pathology in clinical practice. *Am J Psychiatry* 1998;155:1767–71.
 32. Perry JC. Problems and considerations in the valid assessment of personality disorders. *Am J Psychiatry* 1992;149:1645–53.
 33. Bulik CM, Sullivan PF, Fear JL, Pickering A. Outcome of anorexia nervosa: eating attitudes, personality, and parental bonding. *Int J Eat Disord* 2000;28:139–47.
 34. Fichter MM, Quadflieg N. Six-year course and outcome of anorexia nervosa. *Int J Eat Disord* 1999;26:359–85.
 35. Speranza M, Corcos M, Levi G, Jeammot P. Obsessive-compulsive symptoms as a correlate of severity in the clinical presentation of eating disorders: measuring the effects of depression. *Eat Weight Disord* 1999;4:121–7.
 36. Lennkh C, Strnad A, Bailer U, Biener D, Fodor G, de Zwaan M. Comorbidity of obsessive-compulsive disorder in patients with eating disorders. *Eat Weight Disord* 1998;3:37–41.

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Résumé : Profils de la comorbidité psychiatrique et de l'inventaire des troubles alimentaires (EDI) chez les sujets souffrant de troubles alimentaires

Objectif : Cette étude examine les chevauchements éventuels entre la comorbidité psychiatrique (axe I et II) et les scores aux sous-échelles de l'inventaire des troubles alimentaires (EDI) chez les femmes souffrant de troubles alimentaires.

Méthode : Dans un échantillon de 248 femmes (72 souffrant d'anorexie mentale, 140 de boulimie et 36 de troubles alimentaires non spécifiés), nous avons déterminé la comorbidité psychiatrique à l'aide de l'entrevue clinique structurée pour le DSM-IV. Les caractéristiques comportementales et psychologiques des troubles alimentaires ont été quantifiées au moyen de l'EDI.

Résultats : La comorbidité psychiatrique était élevée dans les deux axes (74 % pour l'axe I et 68 % pour l'axe II). Même si la plupart des sous-échelles de l'EDI relevant des traits psychologiques indiquaient des associations significatives avec les troubles de l'axe I et II, les sous-échelles concernant l'alimentation et la perception du poids et de la silhouette étaient beaucoup moins associées à la comorbidité psychiatrique. Les troubles affectifs et anxieux, de même que les troubles de la personnalité des groupes A et C indiquaient un modèle semblable avec des liens à la plupart des sous-échelles psychologiques. Le profil des troubles liés à une substance était différent; il affichait des associations avec les échelles de perception intéroceptive de l'inefficacité. Les troubles de la personnalité du groupe B étaient reliés seulement à la sous-échelle de la boulimie et à aucune des sous-échelles psychologiques.

Conclusions : L'EDI semble refléter principalement les troubles de l'axe I et II reliés aux problèmes affectifs et anxieux. Les cliniciens et les chercheurs qui utilisent l'EDI doivent savoir qu'il n'est pas sensible à toutes les formes de comorbidité prévalentes chez les patients souffrant de troubles alimentaires.