Predicting the Outcome of Eating Disorders Using Structural Equation Modeling

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Abstract: **Objective:** *There is a need for models that predict accurately the course of mental disorders.* **Method:** *Eating-disordered female inpatients were assessed longitudinally at the beginning of treatment (t_1), at the end of treatment (t_2), at 2 or 3-year follow-up (t_3), and at 6-year follow-up (t_4). The sample consisted of 196 women with bulimia nervosa (BN) purging type, 103 women with anorexia nervosa (AN), and 68 women with binge eating disorder (BED; N = 367). Confirmatory factor analysis and path analysis were used to predict the women's status at 6-year follow-up.* **Results:** *The results for BN and BED show that the specific eating disorder pathology was influenced mainly by specific eating disorder pathology at earlier time points and not by non–eating-specific (general) psychopathology. Similarly, general psychopathology was influenced mainly by general psychopathology at earlier time points. For AN patients, both categories of psychopathology (eating specific and general) were relevant for the 6-year outcome. The potential impact of 14 factors on the level of pathology was estimated (a) at baseline (at the beginning of treatment), (b) during the course of illness (baseline controlled), and (c) on the 6-year outcome of eating disorders (baseline and course controlled). Although there were many correlations between potential factors and baseline pathology, there was only a limited number of significant correlations with the 6-year outcome. This effect was mediated largely by the level of general psychopathology.* **Discussion:** *The models for outcome prediction based on structural equation modeling techniques were very similar for BN and BED. For both BN and BED, there were almost entirely separate predictions for the specific eating disorder on the one hand and non–eating-related (general) psychopathology on the other hand. This was true to a lesser degree for AN.* **Conclusions:** *The use of refined path analytic methods in follow-up studies on larger general populations will be helpful to increase our understanding of the course of illness of psychiatric disorders. © 2003 by Wiley Periodicals, Inc.* Int J Eat Disord 34: 292–313, 2003.

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INTRODUCTION

Accurate prediction of the course of illness is relevant for several reasons. First, it can pinpoint the risk factors for an unfavorable course and identify individuals with a high risk of relapse. Second, it can find and design the right kind and intensity of treatment for individuals at risk of relapse. Third, an accurate prediction can avoid unnecessary treatment in individuals with a low risk of relapse. Finally, it will further help our understanding of the nature of the illness, its etiologic factors, and its nosology. A number of longitudinal studies about anorexia nervosa (AN) have been published and several of them have addressed the issue of risk factors for an unfavorable course. A low body weight at the beginning of treatment, psychiatric comorbidity, childhood obesity, late onset of menarche, and family problems have been described as possible risk factors for an unfavorable course for AN individuals. Since the clinical description and definition of bulimia nervosa (BN) in the late 1970s (Russell, 1979), an increasing number of studies (although fewer than the number of AN studies) have presented longitudinal data and data on risk factors. Very little data exist on the course, outcome, and risk factors for an adverse outcome among individuals with binge eating disorder (BED), which has only been defined preliminarily as a research category in the 4th ed. of the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV; American Psychiatric Association [APA], 1994).

Strober and Humphrey (1987) reviewed the potential effects of family factors on the onset and course of AN and BN. Family studies point to an increased frequency of eating-disordered or affectively disturbed family members. Parker (1984) identified a family climate of affectionless control as a potential risk factor for mental disorders. Pole, Waller, Stewart, and Parkin-Feigenbaum (1988) discovered that bulimic patients and normal controls differ in their perception of parental educational style, whereas Palmer, Oppenheimer, and Marshall (1988) did not find any differences between anorexic and bulimic patients and normal controls regarding this variable.

Several studies documented a high rate of psychiatric comorbidity among eating-disordered patients (Halmi et al., 1991), especially with depressive symptoms or personality disorders (Herzog, Keller, Lavori, Kenny, & Sacks, 1992; Ratnasuriya, Eisler, Szmukler, & Russell, 1991). Friedlander and Siegel (1990) described the relationship between personality development and eating disorder-specific distortions of perceptions. Affective disturbances and personality disorders had a negative impact on the course of eating disorders (Brotman, Herzog, & Hamburg, 1988; Bryant-Waugh, Knibbs, Fosson, Kaminski, & Lask, 1988; Herzog, Hartmann, Sandholz, & Stammer, 1991). Multiimpulsive bulimic patients have a less favorable course than bulimics without other impulsive symptoms (Fichter, Quadflieg, & Rief, 1994).

Fairburn, Welch, Doll, Davies, and O'Connor (1997) and Fairburn, Cooper, Doll, and Welch (1999) compared AN and BN patients with general psychiatric controls. They explored comprehensively the risk factors for AN and BN. Compared with BN subjects, AN subjects experienced less parental disturbance (depression, substance abuse) and parental obesity. Subjects with AN, compared with general psychiatric controls, had a higher negative self-evaluation and were more perfectionistic. In addition, they had no

close friends, parental contact was minimal, parental expectations were higher and parental involvement lower, their parents had a chronic illness, they moved frequently, they had a family member dieting for any reason, their shape, weight, and eating habits were criticized by family members, they were obese during childhood, and were younger at menarche. AN subjects did not have a higher risk for premorbid psychiatric disorder (major depression, substance abuse), parental depression, or substance abuse, nor did they have more low care/high overprotection, parental obesity, and an earlier age at menarche (Fairburn et al., 1999).

Compared with general psychiatric controls, BN individuals had a higher risk for negative self-evaluation, perfectionism, and premorbid major depression. In addition, they had more conduct problems and school absence, less parental contact, more parental arguments, more criticism, higher expectations and underinvolvement, and a higher incidence of parental psychiatric disorder (depression and substance abuse). They experienced repeated severe sexual abuse, had a family member who was dieting for any reason, their shape, weight, and eating were criticized by family members, their parents were more likely to be obese, they were obese during childhood, and were younger at menarche. Premorbid substance abuse and the combination of low care and high overprotection were not significantly different between these groups (Fairburn et al., 1997).

Keel and Mitchell (1997) reviewed the literature on the outcome of BN and prognostic factors. They found that depression, personality disturbance, and the severity and duration of symptoms were prognostic factors for the course of BN. Quadflieg and Fichter (2003) added low self-esteem and substance abuse to this list.

It is beyond the scope of this study to review the complete literature on outcome, predictors of outcome, and risk factors for AN, BN, and BED. Predictors of unfavorable course reported in some studies have not been confirmed by others. The studies differ with respect to sampling, length of follow-up period, and assessment procedures. In addition, most studies did not take into consideration the complex causal pathways at more than one time point (Byrne & McLean, 2002; Huon et al., 1999). The current study uses a different approach by assessing relatively large samples of treated patients with AN, BN, and BED ($N = 367$). Our main intention was to predict eating disorder outcome by using structural equation modeling (SEM) techniques. These methods have been refined in recent years to include dichotomous variables and to deal with multiple indicators (McLachlan & Peel, 2000). We intended to develop a comprehensive model for predicting the outcome of each specific eating disorder. In developing these models, we focused on the interactions between specific eating disorder pathology (such as binging or purging) and non–eating-related (general) psychopathology (e.g., depression and anxieties) over time. We also explored the relevance of these interactions for the prediction of longer-term outcome of AN, BN, and BED. To account for potential influences not covered by eating-specific and general psychopathogy, we introduced additional variables into the models.

METHODS

Sample Characteristics

Of 592 consecutively admitted female eating-disordered patients, 103 females met the DSM-IV diagnostic criteria for AN (APA, 1994), 196 females met DSM-IV criteria for BN purging type (BN), and 68 females met criteria for BED. Other patients had an eating

disorder not otherwise specified (EDNOS) or were obese without other eating disorder symptoms (data not provided). All patients were treated between September 1985 and June 1988 at Klinik Roseneck (Prien, Germany), a 350-bed hospital for behavioral medicine.

AN inpatient treatment averaged 118.6 ± 49 days and the mean age of patients at admission was 24.9 ± 6.7 years. BN inpatient treatment averaged 95.5 ± 43 days ($M \pm SD$) and the mean age of patients at admission was 25.6 ± 6.7 years. BED inpatient treatment averaged 76.7 ± 40 days and the mean age of patients at admission was 29.3 ± 8.4 years. Upon admission, all patients completed a series of questionnaires that covered all diagnostically relevant symptoms. A detailed description of the diagnosis and the course of the eating disorder were published in Fichter and Quadflieg (1997, 1999) and in Fichter, Quadflieg and Gnutzmann (1998). Therefore, this study provides a summary of the diagnostic outcome at the 6-year follow-up.

Patients were assessed at the following four points of time: at the beginning of treatment (admission to inpatient treatment; t_1), at the end of treatment (discharge; t_2), at 2 (AN and BN) or 3-year follow-up (BED; t_3), and at 6-year follow-up (t_4). The 2-year and 6-year follow-ups of the AN and BN patients were done in two steps. All patients received a comprehensive questionnaire.[1] After patients had returned their completed questionnaire, a detailed interview covering similar areas as the questionnaire was conducted by specially trained clinical psychologists. The interviewers at both follow-ups (t_3 and t_4) were supplied with the main information about each patient from earlier assessments to allow them to explore each patient in more detail. The procedure at the 6-year follow-up (first questionnaire, then interview) was identical for all three groups.

Of the AN patients, 26.8% either still had AN at the 6-year follow-up or had relapsed, 9.9% had developed BN, and 2.0% were classified as EDNOS. No major eating disorder was found in 55.4% and 5.9% were deceased. The percentages for the BN patients at the 6-year follow-up were 21.4% (BN), 3.7% (AN), 1.1% (BED), 1.6% (EDNOS), 71.1% (no major eating disorder), and 1.1% (deceased). The percentages for BED patients were 5.9% (BED), 7.4% (BN), 7.4% (EDNOS), 77.9% (no major eating disorder), and 1.4% (deceased).

Measures and Instruments

Data concerning sociodemography, family of origin, and other areas (e.g., duration of illness, childhood obesity, or impulsiveness) were collected at the beginning of treatment (t_1). The Parental Bonding Instrument (PBI; Parker, 1983; Parker, Tupling, & Brown, 1979) measured patients' perceptions of parents' care (Care subscale) and overprotection/control (Overprotection/Control subscale). Patients also completed the Freiburger Personality Inventory, revised edition (FPI-R; Fahrenberg, Hampel, & Selg, 1994), a self-rated personality measure that is used widely in Germany. Psychosocial stressors were rated at the beginning of treatment (t_1) on the Axis IV scale of the 3rd ed. of the Diagnostic and Statistical Manual of Mental Disorders (DSM-III; APA, 1980).

Sexual problems were assessed at the beginning of treatment using a 16-item self-rating scale.[2] Six items addressed the avoidance of body contact and uneasiness with

[1]All BED patients at 3-year follow-up. Only self-rating data were assessed. Due to restrictions in funding at the first follow-up, BED patients were assessed 1 year later than patients with AN and BN.

[2]A copy of this 16-item scale is available from the first author. The sexual problems scale is rated by the patient on a 3-point scale. Cronbach's alpha of the subscales ranged from .79 to .85 (Cronbach, 1951).

sexual arousal; seven items addressed sexual problems with male partners (avoidance of dating, petting, sexual activity, sexual anxieties); and three items addressed sexual taboos in the family of origin.

At all four time points (t_1–t_4), eating disturbance and general psychopathology were measured as described below.

Eating Disturbance

The Structured Inventory for Anorexic and Bulimic Syndromes (SIAB-EX; Fichter, Elton, Engel, Meyer, Mall, & Poustka, 1991; Fichter, Herpertz, Quadflieg, & Herpertz-Dahlmann, 1998) was used at the 2-year and 6-year follow-ups. A self-rating version of the SIAB (SIAB-S; Fichter & Quadflieg, 2000) was used at both follow-ups. The SIAB-EX assesses six factors (body image and slimness ideal, social integration and sexuality, depression, anxieties and obsessions, bulimic behavior, laxative abuse). Items and average sum scores are scaled from 0 (symptom not present) to 4 (very severe).

The Eating Disorder Inventory (EDI; Garner, Olmstead, & Polivy, 1983) and the Anorexia Nervosa Inventory for Self-Rating (ANIS; Fichter & Keeser, 1980) are self-rating instruments. After the SIAB-EX was completed at the 6-year follow-up, the Psychiatric Status Rating Scale (PSR; Herzog, 1990; Herzog, Keller, Lavori, Bradburn, & Ott, 1990) was used to assess the overall status of the eating disturbance at the 6-year follow-up. The PSR was modified for AN or BED as appropriate. It comprises a 6-point scale that is operationalized and anchored to DSM-IV criteria. Keller et al. (1987) reported intraclass coefficients of .95 for the PSR for mood disorders and .81 for other psychopathology. The PSR also assessed body weight, treatments following discharge, and the presence of other symptoms and disorders.

General Psychopathology

General psychopathology was measured using the Hopkins Symptom Checklist (SCL-90; Derogatis, Liberman, Rickels, Uhlenhutz, & Cori, 1974) and the Beck Depression Inventory (BDI; Beck, Ward, Mendelson, Mock, & Erbaugh, 1961). Therapists documented inpatient psychiatric diagnoses according to DSM-III and somatic diagnoses according to ICD 9 (WHO, 1979). The Munich Diagnostic Checklists for DSM-III-R (MDCL; Hiller & Zaudig, 1992; Hiller, Zaudig, & Mombour, 1990) were used by the interviewers at the 2-year follow-up of AN and BN patients to assess lifetime comorbidity.

Factors potentially influencing the course of illness were derived from the measures described above and defined as follows:

1. Duration of eating disorder: Time (years) between the onset of the eating disorder and the date of admission to index inpatient treatment.
2. Psychiatric lifetime comorbidity: Psychiatric lifetime comorbidity was present if the patient had suffered from any of the following mental disorders: mood disorder, anxiety disorder, substance abuse, personality disorder, and other disorders (e.g., adjustment disorders) according to DSM-III criteria.
3. Childhood obesity: Childhood obesity was diagnosed if patients indicated at admission that they had been overweight between the ages of 6 and 12 years old according to their own understanding of the term *overweight*.
4. Late menarche: Age at menarche was dichotomized. Late menarche occurred at or after the age of 12 years and early menarche occurred before the age of 12 years.
5. Introversion and emotional lability: Introversion and Emotional Lability are standardized scales of the FPI-R.

6. Perfectionism: Perfectionism was derived from the EDI Perfectionism subscale and the ANIS Anancasm scale. Both scales were standardized into a *z*-distribution. Two *z*-scores were added into one perfectionism score.
7. Achievement orientation: Achievement orientation was computed as the sum of the standardized values of two SIAB-S items covering this area.
8. Impulsiveness: Impulsiveness was present if the patient reported that at least one of three impulsive behaviors (autoaggression, shoplifting, or promiscuity) had occurred in her lifetime.
9. Interpersonal distrust and sensitivity: Interpersonal distrust and sensitivity were derived from EDI scores on the Interpersonal Distrust subscale and from SCL-90 scores on Interpersonal Sensitivity. Scores were standardized and then added together.
10. Low self-esteem: Low self-esteem consisted of ANIS Feelings of Insufficiency and the SIAB-S items reduction of self-esteem, feelings of insufficiency, and reduced self-confidence. All scores were standardized and totaled as one score.
11. Sexual problems and psychosocial stressors: This variable was derived from four items described above: sexual problems, sexual problems with male partners, sexual taboos in the family of origin, and the DSM-III Axis IV rating (psychosocial stressors). Scores were standardized and added.
12. Affectionless control: Affectionless control was derived from the PBI Care and Overprotection/Control scores. A score below 23 for Care and a score of 19 or higher for Overprotection/Control indicated affectionless control for mothers. A score below 20 for Care and a score of 17 or higher for Overprotection/Control indicated affectionless control for fathers. The variable entered into the model was scored 0 (no parent showed affectionless control), 1 (either mother or father showed affectionless control), or 2 (both parents showed affectionless control).
13. Parental substance use problems: Parental alcohol problems were scored as 0 if neither father nor mother had any problems with alcohol according to the patient's report. A score of 1 was given if either father or mother had alcohol problems and a score of 2 if both father and mother had alcohol problems. Illicit drug use and dependence were defined in an analogous way. Parental alcoholism and drug abuse scores were standardized and added. Assessment of this variable was based on information provided by the patients.

All factors except one (psychiatric comorbidity) were assessed at the beginning of the index treatment. During therapy, psychiatric comorbidity was assessed by the therapist. At the 2-year and 6-year follow-ups, comorbidity for the follow-up periods was assessed by research psychologists using the MDCL.

Statistical Analysis

SEM techniques were used to test the a priori hypotheses. This statistical technique allows the modeling of complex causal pathways to one or several outcomes. These pathways are a priori specified, taking into consideration temporal structure and theoretic knowledge about which relationships are considered causal and which are just correlational. The pathways are then transformed into a set of structural equations, which are solved simultaneously (see description of the cross-lagged panel model below).

Due to the relatively small sample and the complexity of the model, a stepwise procedure was selected. First, the postulated latent constructs were identified and tested for unidimensionality using confirmatory factor analysis (Jöreskog, 1971, 1979). Second, the relationships between constructs were tested using path analytic procedures. As measure of goodness of fit, the root mean square error adjusted (RMSEA) recommended by Kline (1998) was computed.

Identifying and Testing Latent Constructs

Latent constructs were constructed using confirmatory factor analysis. For instance, the drive for thinness construct was operationalized using the following indicators: EDI Drive for Thinness, ANIS Figure Consciousness, and SIAB-S item slimness ideal. As all three indicators theoretically measure aspects of an individual's drive for thinness, their respective values should correlate. All three should load very highly on the same factor, which was indeed the case (see below: measurement models of eating specific pathology). Therefore, the loadings of the measured indicators on a construct can be used to identify the theoretically postulated relationships. This procedure was used for all constructs reported.

Because indicators for some of the constructs were dichotomous or ordinal, Pearson correlations could not model the relationships with the usual maximum likelihood estimation. This would have led to distorted parameter estimates and incorrect goodness-of-fit measures and standard errors (Jöreskog & Sörbom, 1993b, 1993c). Therefore, tetrachoric correlations in the case of 2×2 tables (Muthén, 1989), polychoric correlations in the case of ordinal variables with more than two categories (Olsson, 1979), and polyserial correlations for mixtures of ordinal and interval variables (Jöreskog & Sörbom, 1993a) were the bases for all models described, together with the respective asymptotic covariance matrices. The choice of correlation measures can be justified by Monte Carlo studies where only the mixture of tetrachoric, polychoric, and polyserial correlations did not lead to an underestimation of the true relationships (Jöreskog & Sörbom, 1988). All input matrices were estimated with the program PRELIS 2 (Jöreskog & Sörbom, 1993a). A nonnormal theory-weighted least squares approach to estimate the models yields asymptomatically correct standard errors (estimation by LISREL 8.20, see Jöreskog & Sörbom, 1993b, 1993c).

The path analytic model and the effects of other factors were estimated with the program Mplus (Muthén & Muthén, 1998). This program is an extension of the classical path analysis. The extension concerns the introduction of latent variables into the system, the development of new estimation techniques to include dichotomous or ordinal variables as dependent, and the inclusion of the time perspective. Traditionally, estimation has been done by maximum likelihood in preceding programs. However, Muthén, du Toit, and Spisic (in press) developed a robust estimation technique based on weighted least squares and quadratic estimation techniques, which produce better results for models including dichotomous and ordinal variables. To include dichotomous outcome variables, probit regression is used to solve the simultaneous equations.

For each time point in the basic cross-lagged panel model, the influence of factors from preceding time points was estimated. Therefore, the level of eating-specific pathology at t_2 was modeled by introducing influences from both the level of eating disorder-specific pathology at t_1 and from general psychopathology at t_1. In addition, eating disorder-specific pathology and general psychopathology were assumed to be correlated. Finally, the level of pathology at time t_x was postulated to influence the level of pathology not

only at time t_{x+1}, but also at time t_{x+2} for the same category. For example, eating-specific pathology at t_1 influences eating-specific pathology at t_3 and general psychopathology at t_1 influences general psychopathology at t_3.

Figures 1–4 provide an overview of the cross-lagged panel model. For the final empirical model, all nonsignificant relationships were deleted from the model. For instance, if the level of eating disorder-specific pathology at t_1 did not influence significantly the level of general pathology at t_2, the arrow will not appear with quantification (Figures 1–4), but only as a gray-shaded arrow. To keep the figures more readable, all direct paths from t_1 to t_4 for eating-specific and non–eating-specific pathology were deleted. None of these relationships was significant. To assess the influence of the variables, paths from each of the factors were estimated to all variables in the basic cross-lagged panel model (e.g., to the seven variables included in Figure 1). One factor was introduced at a time, so 14 additional models were estimated to evaluate all factors. Due to the limited sample size, it was not possible to enter all factors into one model. As a result, this approach had to be used.

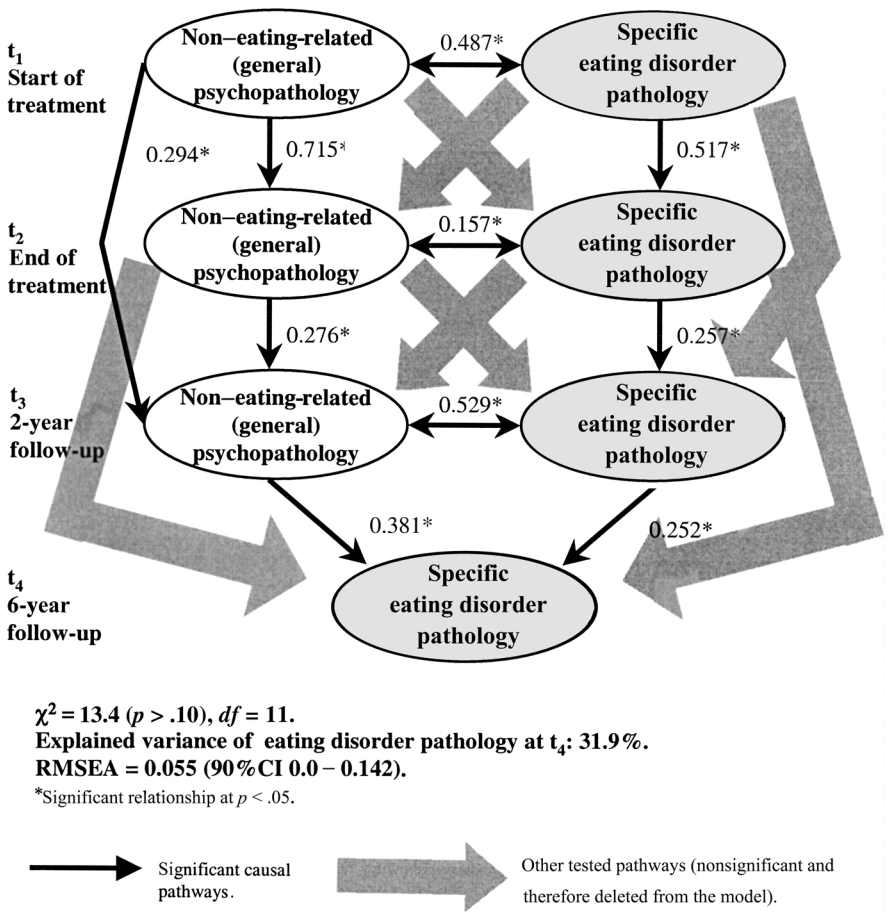

Figure 1. Final model on the course of anorexia nervosa: Eating disorder pathology as outcome ($N = 72$). CI = confidence interval; RMSEA = root mean square error adjusted.

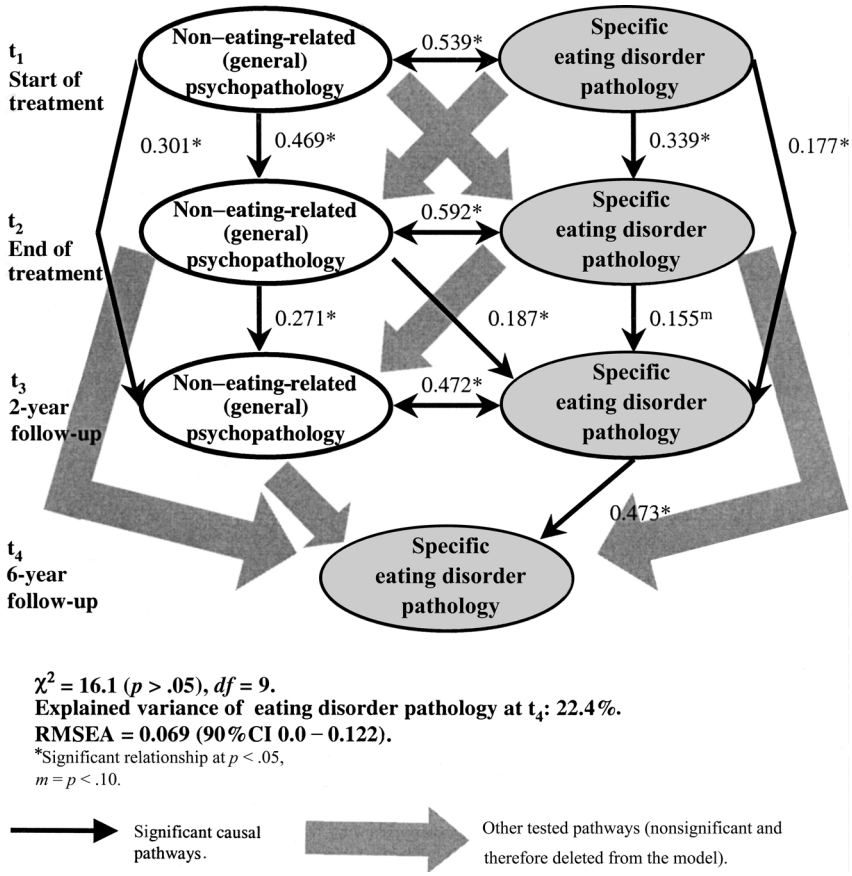

Figure 2. Final model on the course of bulimia nervosa: Eating disorder pathology as outcome ($N = 166$). CI = confidence interval; RMSEA = root mean square error adjusted.

RESULTS

Measurement Models of AN, BN, and BED

The model distinguished between two basic sets of latent constructs (i.e., general psychopathology and eating-specific pathology) at different time points.

General Psychopathology

General psychopathology for all time points was based on two latent variables. These were depression and psychopathology other than depression. Depression had two indicators: the BDI (at the 2-year and 3-year follow-ups, the SCL-90 Depression subscale was substituted for the BDI) and the SIAB-S item depression. Psychopathology other than depression had six indicators: SCL-90 Somatization, SCL-90 Obsessive-Compulsive Behavior, SCL-90 Anger/Hostility, SCL-90 Phobic Anxiety, SCL-90 Anxiety, and the SIAB-S items anxieties and obsessions. As all indicators had significant loadings greater than 0.7 on the postulated latent factors, these factors were construed with equal weights of the indicators (after z-standardizing all indicators). The factors of general psychopathology at the different

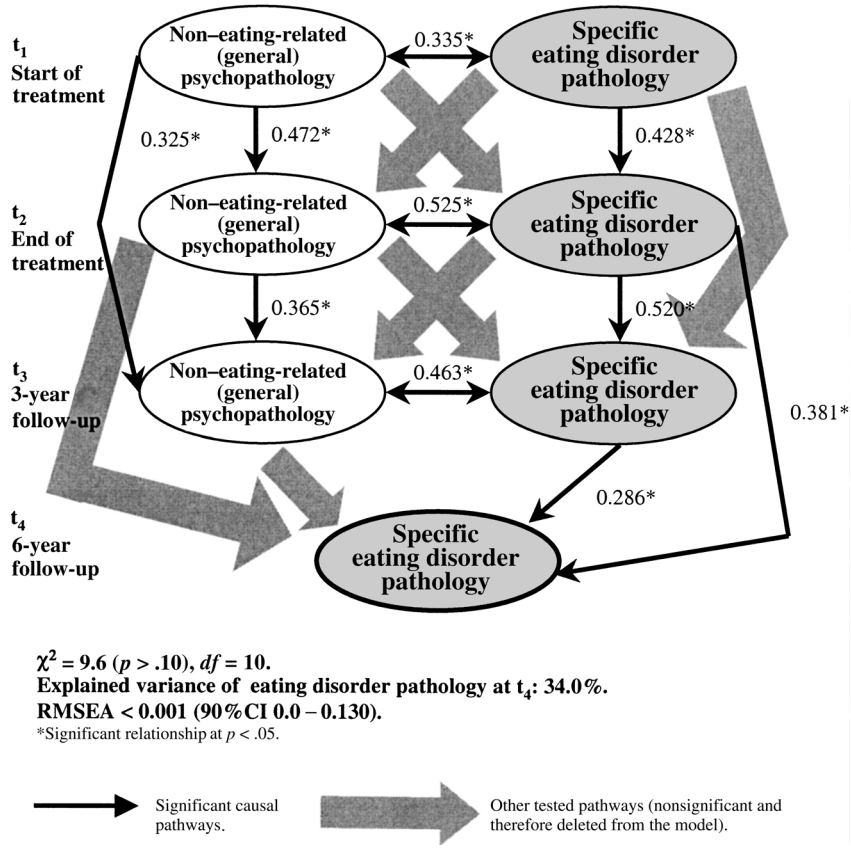

Figure 3. Final model on the course of binge eating disorder: Eating disorder pathology as outcome ($N = 62$). CI = confidence interval; RMSEA = root mean square error adjusted.

time points were built by adding depression and other psychopathology, taking the respective average weighting from the path analytic model as 1.0 for depression and 0.8 for psychopathology other than depression. For the first follow-up, social integration was added with a weight of 0.6. This variable was composed of the SIAB-S scores for leisure activities, social contacts, and the existence of a confidant. The same measurement model of general psychopathology was defined for all diagnostic groups.

Eating-Specific Pathology

Eating-specific pathology for AN was derived from three latent variables: body weight as the body mass index (BMI); drive for thinness with three indicators (EDI Drive for Thinness, ANIS Figure Consciousness, and SIAB-S slimness ideal); and the number of different compensatory behaviors used (vomiting, laxative abuse, abuse of diuretics, abuse of appetite suppressants, excessive dieting, and excessive exercising). Of these constructs, compensatory behaviors was the most central (loading 0.8), followed by drive for thinness (0.7), and BMI (−0.7 at the end of treatment and at both follow-ups; at beginning of therapy, −0.5).

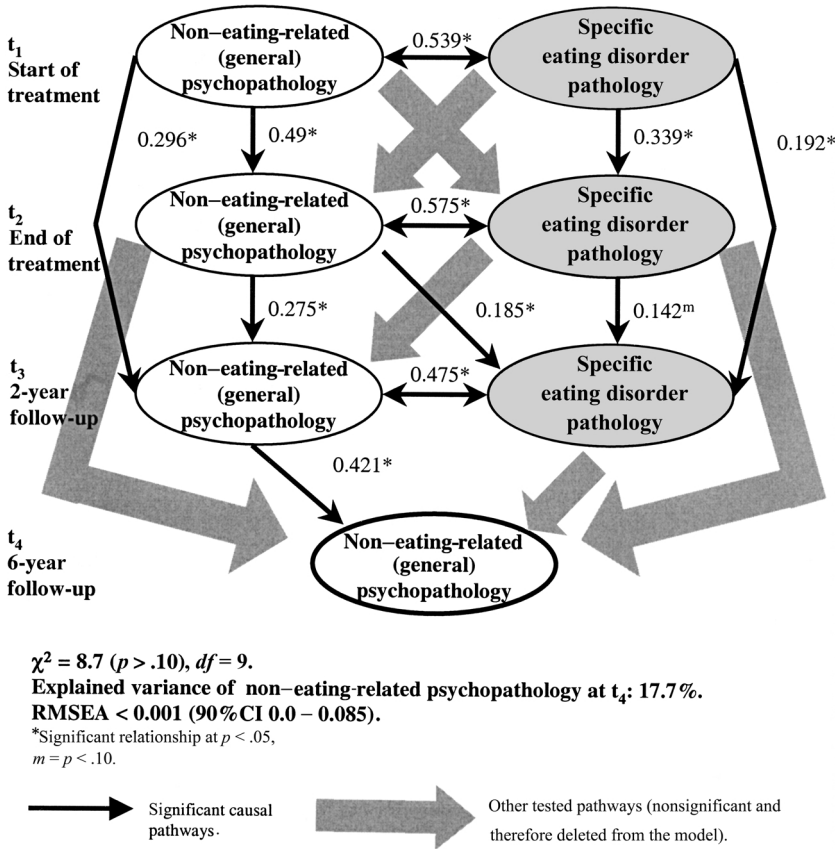

Figure 4. Final model on the course of bulimia nervosa: Non–eating-related psychopathology as outcome ($N = 166$). CI = confidence interval; RMSEA = root mean square error adjusted.

Eating-specific pathology for BN was composed of three latent variables: binging with EDI Bulimia (modified by deleting Item 53 [vomiting] from computation) and ANIS; drive for thinness with three indicators (EDI Drive for Thinness, ANIS Figure Consciousness, and SIAB-S slimness ideal); and the number of different compensatory behaviors used (vomiting, laxative abuse, abuse of diuretics, abuse of appetite suppressants, excessive dieting, and excessive exercising). Of these constructs, binging proved to be most central (average loading over time 0.9), followed by drive for thinness (0.7), and the number of different compensatory behaviors (0.5).

Eating-specific pathology for BED was composed of two latent variables: binging with EDI Bulimia (modified by deleting Item 53 [vomiting] from computation) and ANIS; drive for thinness with three indicators (EDI Drive for Thinness, ANIS Figure Consciousness, and SIAB-S slimness ideal). Both constructs had loadings of 0.9. Overall, the models fit very well, with no significant deviation of the data from the model as measured by a chi-square test.

The 6-year outcome of eating disorder pathology for AN was derived from four latent variables: the PSR after the comprehensive data collecting with the SIAB-EX; BMI; drive for thinness with three indicators (EDI Drive for Thinness, ANIS Figure Consciousness, and SIAB-S slimness ideal); and the number of different compensatory behaviors used

(vomiting, laxative abuse, abuse of diuretics, abuse of appetite suppressants, excessive dieting, and excessive exercising). These constructs were included with weights of 1.0 for the PSR, 0.8 for compensatory behaviors, 0.7 for drive for thinness, and −0.7 for the BMI.

The 6-year outcome of eating disorder pathology for BN was built from four latent variables: the PSR after the comprehensive data collecting with the SIAB-EX; binging with EDI Bulimia (modified by deleting Item 53 [vomiting] from computation) and ANIS; drive for thinness with three indicators (EDI Drive for Thinness, ANIS Figure Consciousness, and SIAB-S slimness ideal); and the number of different compensatory behaviors used (vomiting, laxative abuse, abuse of diuretics, abuse of appetite suppressants, excessive dieting, and excessive exercising). These constructs were included with weights of 1.0 for the PSR, 0.9 for binging, 0.7 for drive for thinness, and 0.5 for compensatory behaviors.

The 6-year outcome of eating disorder pathology for BED was composed from three latent variables: the PSR after the comprehensive data collecting with the SIAB-EX; binging with EDI Bulimia (modified by deleting Item 53 [vomiting] from computation) and ANIS; and drive for thinness with three indicators (EDI Drive for Thinness, ANIS Figure Consciousness, and SIAB-S slimness ideal). These constructs were included with weights of 1.0 for the PSR, 0.9 for binging, and 0.9 for drive for thinness.

Models on the Course of AN, BN, and BED

For each eating disorder, a distinct model on the course of eating disorder symptoms was tested. Seventy-two AN patients (Figure 1), 166 BN patients (Figures 2 and 4), and 62 BED patients (Figure 3) had sufficient data points across time to be included in the final models. The variables were put into the cross-lagged panel analysis. All three empirical models did not differ significantly from the expected model. In Figures 1–4, the arrows show the significant paths. The black and gray-shaded arrows show the underlying general concepts of the cross-lagged panel analyses (excluding the gray-shaded arrows from t_1 to t_4 because of insignificance as noted above).

The final model on the course of AN is shown in Figure 1. At t_1 (admission) and t_2 (discharge), there were no causal pathways from specific eating disorder pathology to non–eating-related (general) psychopathology at later points of time and vice versa. The non–eating-related (general) psychopathology at t_1 (admission) influenced the non–eating-related psychopathology at t_3 (2 years after the end of therapy). At each time point, general psychopathology and specific eating disorder pathology were correlated, but there was almost no prediction between the two developments across time. However, different from BN and BED (Figures 2 and 3), eating disorder outcome at t_4 (6 years after inpatient treatment) was influenced by eating disorder pathology at t_3 (0.252) and non–eating-related pathology at t_3 (0.381).

Correlations between t_1 and t_2 (start to end of treatment) were higher than correlations between t_2 and t_3 or between t_3 and t_4. This may be because the interval between t_1 and t_2 was much shorter than the other intervals. RMSEA was 0.055 with the 90% confidence interval (CI; 0.0–0.142), indicating an acceptable goodness of fit.

Table 1 shows the estimated effects of the factors on AN. Significant relationships between the factors and the level of pathology were found at t_1 (admission). Psychiatric comorbidity before or at t_1 (0.275 at admission) and late onset of menarche (0.200) were related independently to the 6-year outcome of the eating disorder (baseline pathology and pathology during course of the eating disorder were controlled). Late onset of

Table 1. Effects of potential variables on the course of eating disorders (separately for AN, BN, and BED; standardized path coefficients)

	Baseline Pathology						Pathology during Course (Measured at First Follow-up[a]) (Baseline Controlled)						Six-Year Outcome of Eating Disorder		
	Eating Specific			Non–Eating-Related (General)			Eating Specific			Non–Eating-Related (General)			(Baseline and Course-Controlled)		
Influencing Factor $N =$	AN 72	BN 166	BED 62	AN 72	BN 166	BED 62	AN 72	BN 166	BED 62	AN 72	BN 166	BED 62	AN 72	BN 166	BED 62
Personal characteristics															
Introversion	—	—	—	—	.26	—	—	—	—	—	—	—	—	—	—
Emotional lability	—	.26	—	.46	.45	.69	—	—	—	—	—	—	—	—	—
Impulsiveness	—	.22	.30	.46	—	—	—	—	—	—	—	—	—	—	—
Low self-esteem	.45	.54	.37	.88	.87	.86	—	—	—	—	—	—	—	—	—
Perfectionism	.41	.41	.47	.49	.27	—	—	—	—	—	—	—	—	—	—
Achievement orientation	.32	.39	.28	.58	.52	.60	—	—	—	—	—	—	—	—	—
Interpersonal distrust and sensitivity	.40	.40	.28	.73	.74	.76	.28	—	—	—	—	—	—	—	—
Sexual problems and psychosocial stressors	—	—	—	—	.22	.27	—	—	—	.20	—	.25	—	—	—
Family problems															
Parental style of affectionless control	—	—	—	.33	.26	—	—	—	—	—	—	—	—	—	—
Parental substance use problems	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
Other															
Duration of eating disorder before start of treatment	—	—	—	.25	—	—	—	—	—	—	—	—	—	—	—
Psychiatric comorbidity before or at admission	—	—	—	.30	.26	.28	—	.15	—	—	.14	—	.28	—	—
Late menarche	—	—	—	—	—	—	—	—	—	—	—	—	.20	—	—
Child obesity	—	—	—	—	.19	—	—	—	—	—	—	—	—	—	—

Note: Analyses were computed separately for AN ($N = 72$), BN ($N = 166$), and BED ($N = 62$). Numbers indicate significant ($p < .05$) standardized path coefficients. AN = anorexia nervosa; BN = bulimia nervosa; BED = binge eating disorder; — = nonsignificant.

[a]Two years after end of treatment for AN and BN and 3 years after end of treatment for BED.

menarche (at or after 12 years old) was associated with a worse outcome. Two variables were related independently to pathology during the course of illness. The variable interpersonal distrust and sensitivity was related to eating-specific pathology and the variable sexual problems and psychosocial stressors was related to non–eating-related pathology. Introversion, parental substance use problems, and obesity during childhood had no independent influence on either the state of illness at admission, the course of illness, and the 6-year outcome of the eating disorder.

Figure 2 shows that there were no causal pathways from eating disorder pathology to non–eating-related (general) psychopathology at later time points in BN patients. General psychopathology at t_2 (end of therapy) had an effect on eating disorder pathology 2 years later (standardized loading of 0.19). Eating disorder pathology at t_1 had a direct influence on eating disorder pathology at t_2 and t_3. Accordingly, non–eating-related (general) psychopathology had a direct influence on non–eating-related (general) psychopathology at t_2 and t_3. The main course of illness in BN is specific by category. Therefore, the level

of specific eating disorder pathology influenced the level of specific eating disorder pathology at later time points, whereas the level of general psychopathology influenced the level of general psychopathology at later time points. At each time point, general psychopathology and specific eating disorder pathology showed correlations around 0.6, but there was almost no prediction between the two developments across time. Eating disorder outcome 6 years after the start of treatment was influenced only by specific eating disorder pathology at an earlier time point (0.473).

The relationship between specific eating disorder pathology at the start and at the end of treatment was lower than the correlation for the much longer time period between the 2-year and 6-year follow-up. RMSEA for this model was 0.069 (90% CI 0.0–0.122).

The estimated effect of the factors on BN is summarized in Table 1. Similar to the findings for AN, most significant relationships reached the level of pathology at admission. None of the factors was related independently to the 6-year outcome of the eating disorder when baseline and pathology during course were controlled. Only one variable (psychiatric comorbidity [to specific eating disorder pathology] and non–eating-related [general] pathology) was related independently to the pathology during the course of illness (baseline controlled). Parental substance use problems, duration of eating disorder before the beginning of treatment, and late onset of menstruation had no independent influence on baseline pathology, the pathology during the course of illness, and the 6-year outcome of the eating disorder.

There were no causal pathways for BED from specific eating disorder pathology to non–eating-related (general) psychopathology at later time points and vice versa (Figure 3). Non–eating-related (general) psychopathology at the beginning of treatment (t_1) influenced the general psychopathology 3 years after the end of treatment (t_3). The main course of illness was specific by category. Therefore, the level of specific eating disorder pathology influenced the level of specific eating disorder pathology at later time points.

At each time point, general psychopathology and specific eating disorder pathology were correlated (around 0.5), but there were no predictions between the two developments (general psychopathology and specific eating disorder pathology) across time. However, the 6-year outcome of eating disorder was influenced by specific eating disorder pathology at the 3-year follow-up (0.286 at t_3) and by specific eating disorder pathology that existed at the end of treatment (0.381). As for BN, but not for AN, specific eating disorder outcome at the 6-year follow-up was not influenced significantly by non–eating-related (general) psychopathology at earlier time points. In BED, relationships between the beginning and the end of treatment were not higher than the relationships between the end of treatment and the 3-year follow-up. Goodness of fit was very good with RMSEA $< .001$ (90% CI, 0.0–0.13).

Table 1 shows the estimated effect of the factors on BED patients. As for AN and BN patients, most significant relationships reached the level of pathology at the beginning of treatment. None of the factors was related independently to the 6-year outcome. Only one variable (sexual problems and psychosocial stressors) was related independently to the pathology during the course of illness. This variable was related to non–eating-related (general) psychopathology. Introversion, parental style of affectionless control, parental substance use problems, duration of eating disorder before the beginning of treatment, and obesity during childhood had no independent influence on either the baseline pathology, the pathology during the course of illness, and the 6-year outcome of the eating disorder.

We also addressed how the 6-year outcome of non–eating-related (general) psychopathology was influenced by the variables assessed at earlier time points (t_1, t_2, and t_3). We performed analogous analyses of models, substituting non–eating-related (general) psychopathology at the 6-year follow-up for eating-specific pathology at the 6-year outcome

(Figure 4). Because of space limitation, we present only the final model on the course of BN with non–eating-related (general) psychopathology as the outcome variable. For BN patients, the non–eating-related (general) psychopathology was predicted only by the non–eating-related (general) psychopathology at earlier time points, but not by the specific eating disorder pathology (RMSEA $< .001$; 90% CI, 0.0–0.085). The findings for BED patients were similar to those for BN patients. The 6-year outcome was predicted only by non–eating-related (general) psychopathology at earlier time points but not by specific eating disorder pathology. In contrast to BN and BED patients, both categories of psychopathology (eating-specific and general) for AN patients showed a significant relationship to the 6-year outcome. Therefore, the main course of illness was specific by category (eating-specific or general) for bulimic disorders (BN and BED), but not for AN.

DISCUSSION

We evaluated the impact of eating-specific and non–eating-specific psychopathology on the course and outcome of eating disorders. Confirmatory factor analysis to reduce the factors assessed by multiple measures and cross-lagged panel analysis (to estimate causal pathways over time) were used to predict the outcome in a design with repeated assessment points. The model fit was very high. No significant deviation of the empirical data from the postulated model was found in the four models reported. Therefore, the postulated theoretic relationships explained the structure of the empirical data. The explained variance of the eating-specific outcome variables was meaningful. The models predicted one fifth to one third of the variance, suggesting that a substantial variation of outcome could be predicted.

In AN, the eating disorder-specific and nonspecific pathology showed an independent course over time. Both areas, however, have great relevance for the eating disorder outcome 6 years after treatment. Non–eating-related symptoms showed an even higher relationship (0.38) than eating-specific symptoms (0.25). The final model on the course of AN differs considerably from that of BN and BED for the prediction of eating disorder pathology at the 6-year follow-up. Our data indicate that there are differences between the AN prediction model and the models of BN and BED. A large number of studies have described the course and outcome of AN (e.g., Bryant-Waugh et al., 1988; Eckert, Halmi, Marchi, Grove, & Crosby, 1995; Fairburn et al., 1999; Herzog, Dorer, Keel, Selwyn, Ekeblad, Flores, Greenwood, Burwell, & Keller, 1999; Herzog, Schellberg, & Deter, 1997; Ratnasuriya et al., 1991; Strober, Freeman, & Morrell, 1997; Theander, 1992; Tolstrup et al., 1985; Willi, Limacher, Helbling, & Nussbaum, 1989). The course of AN is protracted (Steinhausen, Rauss-Mason, & Seidel, 1991). Steinhausen (1999, 2002) has extensively reviewed follow-up studies on AN. He reported the following favorable prognostic indicators: an early age at onset, a hysterical personality, a conflict-free parent-child relationship, a short interval between the onset of symptoms and the beginning of therapy, a short duration of inpatient treatment, no readmissions, and a high socioeconomic status/educational level. He also described the following unfavorable prognostic factors: vomiting, bulimic symptoms and purgative abuse, extreme weight loss, chronicity/compulsivity, and premorbid development/clinical abnormalities (including childhood eating disorders). The current study does not refer to several of these factors (e.g., the early age at onset, a hysterical personality, and the short interval between the onset of symptoms and treatment). Our data revealed that the two main

prognostic indicators for a negative outcome for AN patients at 6-year follow-up were psychiatric comorbidity and late onset of menarche. Late menarche is likely to be associated with subclinically reduced body weight that protracts menarche. Therefore, late menarche may be an early sign of AN. However, according to our data, a long duration of the eating disorder was not a predictor for negative outcome. A possible explanation for this finding is that our patients were a relatively homogeneous group of chronic cases who required inpatient treatment. Childhood obesity was not associated significantly with any variable of our prognostic model, confirming the conclusion of Steinhausen (1999, 2002). This finding is supported by Strober et al. (1997) who found childhood obesity to be of no prognostic value for several measures of outcome in AN.

A number of personal characteristics (e.g., emotional lability, impulsiveness, low self-esteem, perfectionism, high achievement orientation, and interpersonal distrust/sensitivity) are associated with a high level of pathology (eating-specific as well as general psychopathology) at the beginning of treatment for AN patients. Some of these characteristics were also correlated with high symptom expression in BN and BED patients. However, these personal characteristics had no impact on the 6-year outcome of eating disorders when baseline pathology and pathology during the course of illness were controlled.

In BN patients, specific eating disorder pathology, such as binging and purging at 6-year follow-up, was predicted not by general psychopathology but only by specific eating disorder pathology at the 2-year follow-up. Eating disorders show patterns of interaction between eating disorder-specific psychopathology and general psychopathology, which are unique for AN, BN, or BED. In BN, both categories of psychopathology (eating-disorder specific vs. general psychopathology) run a course quite separate from each other. There was only one exception of this rule, that is, general psychopathology at the end of treatment affected the specific eating disorder pathology at the 2-year follow-up. Other than that, each remained within its category. For example, specific eating disorder pathology affected specific eating disorder pathology at later time points (but not general psychopathology). Conversely, general psychopathology affected general psychopathology at later time points. Therefore, if the final outcome is defined in terms of specific eating disorder pathology, the predictors at earlier time points are specific eating disorder pathologies. However, if the final outcome is defined in terms of (general) psychopathology, the predictors at earlier time points are measures of general psychopathology. Our results confirm the importance of the distinction between specific eating disorder pathology and non–eating-related (general) psychopathology. Specific eating disorder pathology at the 2-year follow-up was the only significant factor that influenced the final (6-year) outcome of BN. Specific eating disorder pathology at t_1 (beginning of treatment) predicted specific eating disorder pathology at t_2 (end of treatment) and at t_3 (2-year follow-up). The predictions of general psychopathology at the beginning of treatment were accordingly for that category. Long-term (6-year) predictions of specific eating disorder pathology based on data gathered at the beginning or end of treatment were not possible according to our data for BN. These findings are disappointing. Neither the specific eating disorder pathology nor the general psychopathology assessed at the start or end of treatment predicted specific eating disorder pathology at the 6-year follow-up.

Keel and Mitchell (1997) and Quadflieg and Fichter (2003) reviewed the literature on the prognostic variables for BN. They found evidence for a prognostic value of Axis 1 disorders (depression, substance abuse), personality disturbance, severity of symptoms, duration of symptoms, and treatment of BN. Because all the patients in our study received treatment, the latter cannot serve as a predictor. Having controlled baseline pathology and pathology during the course of illness, none of the predictors for BN listed

by Keel and Mitchell and by Quadflieg & Fichter were confirmed in our study. Our findings on the distinction between eating-specific and general psychopathology are consistent with the results of Keel, Mitchell, Miller, Davis, and Crow (1999). These authors found that depression and anxiety at baseline assessment and lifetime non–eating-specific psychopathology were not associated with the 11.5-year outcome of BN.

The final model on the course of BED was similar to that of BN. It was even more prototypic in the sense that only specific eating disorder pathology at an earlier time point predicted specific eating disorder pathology at a later time point. In addition, only general psychopathology at an earlier time point predicted general psychopathology at a later time point. In no instance did the specific eating disorder pathology at an earlier time point predict general psychopathology at a later time point, nor did general psychopathology at an earlier time point predict specific eating disorder pathology at a later time point. Depending on the definition of the final outcome indicator (specific eating disorder pathology or general psychopathology), the predictor of outcome at a later time point was either specific eating disorder pathology or general psychopathology. This may have implications for treating BED patients. If the major aim of treatment is the reduction of eating pathology, treatment should focus primarily on specific eating disorder pathology. The same is true for treating BN patients. However, effective treatment of AN patients should focus on both specific eating disorder pathology and general psychopathology. For BED and largely for BN specific eating disorder pathology 'breeds true' and so does general psychopathology. According to these data, there is no evidence for a symptom shift from disordered eating to anxieties or vice versa.

There are few data on the longer-term outcome of BED patients (Fichter, Quadflieg, Gnutzmann, 1998). Cachelin et al. (1997) described the natural course of illness in a community sample of women with BED over a period of only 6 months. However, Fairburn, Cooper, Doll, Norman, and O'Connor (2000) described the natural course of BN and BED in community-based cohorts over a 5-year period. They reported that only a few participants switched from one diagnostic category to the other. The outcome of the BED cohort was better than that of the BN cohort. In our study, there were no prognostic factors for the 6-year outcome of BED.

We also calculated models using general psychopathology as the 6-year outcome variable. Results are like a mirror image of the analysis using eating disorder pathology as the 6-year outcome variable for BN and BED. In almost all instances, general psychopathology was predicted by general psychopathology at an earlier time point, not by eating disorder pathology. For AN, the 6-year outcome of general psychopathology was predicted mainly by general psychopathology at the 2-year follow-up ($p < .05$) and (marginally) by eating disorder pathology at the 2-year follow-up ($p < .10$). Based on the models used, there were no indications for a symptom shift to depression, anxiety, or obsessive-compulsive disorder in BN and BED patients.

One major finding of our study was that the best predictor of a set of behaviors over a 6-year course was the same set of behaviors at an earlier time point for BN and BED patients. For AN patients, however, non–eating-specific psychopathology was a slightly better predictor than eating-specific psychopathology for eating disorder outcome. This finding supports the usefulness of distinguishing between AN and BN/BED. Our results are consistent with the results of other studies. A history of major depression predicted partial remission of AN (Herzog et al., 1999). In BN, no association between recovery and general psychopathology (Herzog et al., 1999) or between recovery from BN and depression or anxiety (Keel et al., 1999) was found. To set our results in context, we also applied a more conventional approach and computed logistic regression analyses. We

entered all pretreatment variables that were used in the SEM procedures to predict recovery at the 6-year follow-up. Recovery was defined as the patient having her ''usual self'' or only minimal residual symptoms. Lower self-esteem, less perfectionism, and higher achievement orientation at the beginning of treatment were significant predictors of recovery from AN ($R^2 = .53$). All significant predictors correlated in our SEM model with eating and non–eating-specific baseline pathology.

In BN, a lower expression of drive for thinness at the beginning of treatment significantly predicted recovery 6 years later ($R^2 = .16$). This predictor was from the definition of eating-specific baseline pathology in our SEM model. A higher severity of depression, childhood obesity, and a shorter duration of eating disorders before the beginning of treatment were significant predictors of recovery at the 6-year follow-up in BED patients ($R^2 = .46$). Depression was taken from the definition of non–eating-related baseline pathology in our SEM model. The other two predictors did not have a significant influence on the SEM model.

Breaking down comorbidity into distinct categories (mood disorders, anxiety disorders, substance use disorders) and entering these predictors instead of comorbidity into logistic regression analysis did not result in any of the diagnostic categories being a significant predictor. This is contrary to the findings of Keel et al. (1999). They found that a history of substance abuse predicted a worse outcome in BN. Breaking down psychiatric comorbidity into various illnesses reduces the number of cases in the (sub) categories, thereby reducing the statistical power. This may explain, in part, the discrepancy. The finding by Keel et al. needs further replication.

Only 2 of 14 factors tested were related significantly and independently to the 6-year outcome in AN patients. In BN and BED patients, none of the 14 factors related significantly to the 6-year outcome. This contradicts the results of studies using more conventional approaches to the prediction of outcome in eating disorders (Quadflieg & Fichter, 2003). Most of these studies centered on direct relations between patients' characteristics assessed at an earlier time and the eating disorder outcome at some later time. Our approach was unique because we took into account the influence of the predictors on the level of pathology at time points earlier than the assessment of outcome.

Our results do not suggest that the postulated factors were not related to the outcome. Most of this influence was mediated by the level of pathology at the beginning of treatment (e.g., for BN patients, the average absolute Pearson correlation scores were 0.21 and 0.32 for specific eating disorder pathology and non–eating-related [general] psychopathology, respectively). Most of the postulated factors influenced the level of pathology at the beginning of treatment, but had no independent effect on course of illness or the 6-year outcome.

Most of the predictive power of the variables was absorbed by the degree of severity of eating disorders and non–eating-related psychopathology at baseline assessment and at the 2-year follow-up. This pattern of association would be hard to detect in the usual design of predictive studies. Therefore, differing degrees of symptom severity in the samples may account for some of the contradictory results on the significance of predictors found in these studies.

Our data provide general conclusions about the predictors of outcome for eating disorder patients. Personal characteristics do not have an independent influence on the course and outcome of eating disorders but are correlated to the severity of eating-specific and general psychopathology. It is noteworthy that impulsiveness in AN patients is correlated with non–eating-specific pathology, whereas impulsiveness in BN and BED patients is related to eating-specific symptom severity.

Parental substance use problems was not a predictor for either AN, BN, or BED. This variable was based on patients' self-report and they may not have known about all relevant parental behavior. A more important predictor of the course and outcome of AN and BN was psychiatric comorbidity. This variable correlated with eating and non–eating-related pathology at the 2-year follow-up for BN patients and at the 6-year outcome for AN patients. Expectedly, late menarche was relevant only in AN patients.

The duration of inpatient treatment in this study was 3–4 months. In Germany, the length of inpatient treatment has shortened considerably in recent years. At the time of admission to the study (1985–1988), long inpatient treatments for psychotherapy were the norm. Unfortunately, we were unable to evaluate the influence of length or type of treatment because we did not have a sample of outpatients nor did we have a comparison group of patients who received a shorter duration of treatment. However, it was the aim of the study to use an SEM to identify possible predictors for the psychopathology of eating disorders in patients who received intensive treatment. It was not our aim to analyze effects of different treatments.

The variables used to estimate eating-specific pathology in AN, BN, and BED were identical. This may have led to similar models in BN and BED. Considering the results of the variables, we believe there are enough dissimilarities between the models to warrant the computation of separate models on BN and BED. The reader should keep in mind that we included exclusively patients with the purging type of BN. Therefore, our data may be indicative of more similarities between BN and BED, suggesting that BED may be a variant of the nonpurging type of BN. However, we do not have enough data from our samples to research this possibility.

This study has several limitations. First, the sample sizes are not large enough for simultaneous modeling measurement (e.g., the relationship between different indicators and the construct and substantive relations could not be determined). Second, our sample was restricted to inpatients only. To reduce possible selection factors, we included consecutively admitted patients. The natural course of eating disorders in an unselected community sample would have been more appropriate. However, this approach would be very costly (Fairburn et al., 1997). Third, some of our measures were retrospective (e.g., child obesity, age at menarche, comorbidity before admission) and one depended on information that the patient may not have had (e.g., the severity of parental substance use problems). However, a major strength of our study is that we used a prospective design with several (four) cross-sectional assessments. This helped to minimize the bias of retrospective assessment.

Our results should be treated cautiously, pending independent cross-validation. To gain further insights into the etiology and course of BN and other eating disorders, a longitudinal cohort study is needed. This study should include individuals with and without BN at baseline who are followed up at several cross-sectional assessments. Such a design would allow the identification of risk factors for the onset of BN and other eating disorders and of maintaining factors of the illness. The sample size of such a study should be large enough to allow the simultaneous estimation of the factors based on several indicators and the pathways between such factors. To reduce the waste of valuable resources in assessment, the sample should be limited to 15–30-year-olds in the general population because this is the age group that is most affected by eating disorders. In addition, screening tools may limit the number of persons for whom a full interview is required (see Rehm & Fichter, 1995, for a more general discussion of designs for studying the course of mental disorders). The resulting data would allow unique

insights into the onset and course of eating disorders and would enable better prevention strategies as well as more focused therapeutic procedures.

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