Depressive symptoms and weight change in inpatients with anorexia nervosa: A cross-lagged panel model

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ABSTRACT

Objective: Anorexia nervosa (AN) is marked by a high rate of comorbid depression, which raises the question whether depressive symptoms may adversely affect treatment outcome. Thus, we examined whether depressive symptoms at admission would predict weight change from admission to discharge in a large sample of inpatients with AN. In addition, we also explored the reverse direction, that is, whether body mass index (BMI) at admission would predict changes in depressive symptoms.

Methods: A sample of 3011 adolescents and adults with AN (4% male) who received inpatient treatment at four Schön Clinics was analyzed. Depressive symptoms were measured with the Patient Health Questionnaire–9.

Results: BMI significantly increased and depressive symptoms significantly decreased from admission to discharge. BMI and depressive symptoms were unrelated at admission and discharge. Higher BMI at admission predicted smaller decreases in depressive symptoms and higher depressive symptoms at admission predicted larger weight gain. The latter effect, however, was mediated by longer length of stay.

Conclusion: Results indicate that depressive symptoms do not adversely affect weight gain during inpatient treatment in persons with AN. Instead, higher BMI at admission is predictive of smaller improvements in depressive symptoms but this effect seems to be negligible in terms of clinical relevance.

1. Introduction

Anorexia nervosa (AN) is marked by a significantly low body weight, which often leads to medical complications and, thus, the primary objective of AN treatment is weight restoration [8,29]. There is also a high rate of comorbid mental disorders in AN, the most common of which are affective disorders [13,27]. In fact, it seems that inpatients with AN show similar levels of depressive symptom severity to inpatients with affective disorders [30]. As there are considerable individual differences in treatment response, psychiatric comorbidity may be one key variable that moderates treatment outcome in AN [12,19].

A recent review article that examined effects of comorbid depression on weight gain during AN treatment concluded that studies were heterogeneous in design, purpose, and outcome [6]. Similarly, findings were inconsistent and only one study that was included in the review suggested that depression negatively affects weight gain in patients with AN (cf. [21]). Specifically, a study in which patients were categorized into three groups based on body weight data during treatment found that those with a slow weight gain had higher rates of comorbid affective disorders than those with moderate or rapid weight gain [5]. However, the slow weight gain group also had the highest body mass index (BMI) at admission and, therefore, the slow weight gain may simply be explained by the fact that these patients already started with a higher body weight at admission, independent of comorbid affective disorders or depressive symptoms.

A recent study in 87 inpatients with AN (22 of which were diagnosed...
with comorbid major depressive disorder) corroborated that weight gain from admission to discharge was smaller in patients with comorbid major depressive disorder than in patients without comorbid major depressive disorder [22]. However, this finding does not hold when applying appropriate corrections for multiple testing and similar to the study by Berona et al. [5], those with comorbid major depressive disorder had a higher BMI at admission than those without comorbid major depressive disorder (although this difference was not statistically significant, likely due to the small group sizes).

Most studies that investigated effects of comorbid depression on weight gain during AN treatment were conducted at the construct level, for example investigating whether individuals with AN who also fulfilled DSM-5 criteria for major depressive disorder showed a different weight gain outcome compared to those without comorbid major depressive disorder (e.g., [22]). In addition, most studies only investigated one potential causal direction, namely that depression might influence weight gain. This is problematic for three reasons: First, depression is an extremely heterogeneous syndrome: Two individuals with major depressive disorder are likely to present distinct symptom patterns even when showing the same depression severity on dimensional measurements [7]. Second, inpatients with AN are comparable to inpatients with primary diagnoses of depressive disorders regarding the severity of their depressive symptoms [30]. This might explain why studies investigating DSM-5 major depressive disorder as a potential moderator of weight gain during AN treatment showed inconsistent findings, given that many inpatients with AN but without a diagnosed comorbid depressive disorder also show high depressive symptom severity. Third, it is also possible to assume that weight gain during inpatient treatment might also affect depressive symptoms. For example, a small effect of fast weight gain on later depressive symptoms was found in a randomized controlled study investigating family-based treatment for adolescents with AN [1].

Thus, it is currently unclear whether higher depressive symptomatology predicts smaller weight gain during AN treatment and, if so, whether this effect may simply be explained by higher body weight at admission. To clarify the role of depressive symptoms on weight gain during AN treatment, we analyzed BMI and depressive symptom severity of more than 3000 inpatients with AN at admission and discharge. Specifically, we used structural equation modeling to examine whether depressive symptom severity at admission predicted changes in BMI from admission to discharge (i.e., BMI at discharge while controlling for BMI at admission). In addition, we also explored the reverse direction, that is, whether BMI at admission predicted changes in depressive symptoms from admission to discharge (i.e., depressive symptoms at discharge while controlling for depressive symptoms at admission). Structural equation modeling allows to test and compare such effects in one model with at least two assessment time points—a cross-lagged panel model.

2. Methods

2.1. Sample description

Clinical records of patients with AN (N = 3011) were analyzed who received treatment at four Schoen Clinics in Germany (Roseneck/Prien am Chiemsee [n = 1427], Bad Arolsen [n = 785], Bad Bramstedt [n = 358], Bad Staffelstein [n = 441]) between 2015 and 2020. Inclusion criteria were (1) a diagnosis of either full syndrome or atypical AN, (2) BMI at admission <18.5 kg/m², (3) admission for inpatient treatment, and (4) complete information available about BMI at admission and discharge, depressive symptom scores at admission, and medication during the inpatient stay (Fig. 1).

In the hospitals, data from the routine diagnostic assessment (e.g., age, sex, diagnoses, body weight, questionnaire scores) are automatically transferred to a database from which they can be exported without any identifying information (e.g., name, date of birth, place of residence) by authorized employees. Thus, accessing individual patient charts is unnecessary. According to the guidelines by the institutional review board of the LMU Munich, retrospective studies conducted on already available, anonymized data are exempt from requiring ethics approval and do not require signed informed consent from the patients.

The inpatient treatment offered in the hospitals adheres to the German S3-guidelines for the treatment of AN [3,24] in terms of admission criteria, treatment elements, and therapy goals. Thus, patients received a cognitive-behavioral therapy-oriented, multimodal AN treatment that included several treatment elements such as individual psychotherapy sessions, group therapy sessions, exercise therapy, meal preparation classes, body image exposure, nutrition counseling, and food intake protocols as well as clinical management of medical complications. The treatment includes a high-calorie refueling schedule (starting on the first day of treatment) that aims at a weight gain of 0.7–1.0 kg per week for all underweight AN patients. This schedule includes three meals per day, each having approximately 700 kcal and, thus, totaling to a daily caloric intake of approximately 2100 kcal. Meals are supervised by therapeutic staff members (nurses, psychotherapists, or physicians) in earlier treatment stages. The schedule is individually tailored if patients do not finish their meals or do not show the expected weight gain by increasing portion size, adding snacks between meals, or offering sip feeds. As normalization of eating behavior is one of the therapeutic goals, patients do not receive nasogastric feeding. Patients can choose between vegetarian and non-vegetarian menus; vegan menus are not offered.

2.2. Measures

2.2.1. Patient Health Questionnaire–depressive symptom severity scale (PHQ–9)

Depressive symptoms at admission and discharge were measured with the German version [16] of the PHQ–9 [14,15], which is part of the routine diagnostic assessment in the hospitals. The PHQ–9 has nine items that are answered on a four-point scale (0 = not at all to 3 = nearly every day). It comprises nine common depressive symptoms of different symptom domains, including affective, cognitive, and somatic symptoms. Higher sum scores indicate higher depressive symptom severity. Internal reliability was α = 0.854 at admission and α = 0.876 at discharge.

2.2.2. Other information

Patients’ height and weight were measured at the hospitals and used to calculate BMI (kg/m²) at admission and discharge. All other information was also taken from the clinical records of the hospitals (age, sex, comorbid mental disorders, type of AN [full syndrome vs. atypical], length of stay, medication).

2.3. Data analyses

All analyses were run with JASP version 0.16.3 [10] and RStudio version 2022.07.1 [26] using R version 4.2.1 [23]. Changes in BMI and depressive symptoms from admission to discharge were tested with paired-samples t-tests. Cross-sectional associations between BMI and depressive symptoms at admission and at discharge were tested with Pearson’s correlation coefficients.

To examine the predictive effects of BMI at admission on changes in depressive symptoms and of depressive symptoms at admission on changes in BMI, we specified a cross-lagged panel model with the R package lavaan version 0.6–12 [25]. Specifically, exogenous variables were BMI and depressive symptoms at admission and endogenous variables were BMI and depressive symptoms at discharge. The model included both autoregressive paths and both cross-lagged paths as well as the covariances between BMI and depressive symptoms at admission and between residual variances of BMI and depressive symptoms at discharge (Fig. 2). Because of incomplete data (PHQ–9 scores were
Fig. 1. Flowchart of patients included in the analyses. PHQ–9 = Patient Health Questionnaire–depressive symptom severity scale.
missing for \( n = 615 \) patients), full information maximum likelihood estimation with robust (Huber–White) standard errors was used. In additional models, we examined whether adding paths of length of stay, age, sex, type of AN, comorbid depression, or antidepressant medication on the endogenous variables would change the estimates of the original model and we examined whether these variables moderated the cross-lagged paths by including interaction terms with the exogenous variables. Because of the large sample size and numerous inferential tests, we considered effects as significant when \( p < .005 \), as has been suggested by others [4]. The R code for the cross-lagged panel models as well as the data with which the findings reported in this article can be reproduced are available at https://osf.io/dy9fn.

3. Results

3.1. Sample characteristics

Sample characteristics are reported in Table 1. The majority of patients were female, were adults, had full syndrome AN, had comorbid depression, and did not receive any medication or medication other than antidepressants (e.g., nutritional supplementation, antipsychotics, tranquilizers, analgesics, antiphlogistics) during the inpatient stay (Table 1).

Fig. 2. Standardized estimates of the cross-lagged panel model. Asterisks indicate \( p < .005 \). Coefficients for intercepts and (residual) variances are not displayed for the sake of simplicity and clarity. BMI = Body mass index, PHQ–9 = Patient Health Questionnaire–depressive symptom severity scale.

3.2. Changes from admission to discharge

BMI significantly increased from admission to discharge with a large effect size (\( t_{(3010)} = 73.9, p < .001, d = 1.35 \); Table 1). Depressive symptoms significantly decreased from admission to discharge with a large effect size (\( t_{(2295)} = 49.8, p < .001, d = 1.02 \); Table 1).

3.3. Cross-sectional correlations

BMI and depressive symptoms were uncorrelated at admission (\( r_{(n=3011)} = -0.011, p = .552 \)) and discharge (\( r_{(n=2396)} = -0.048, p = .181 \)).

3.4. Cross-lagged panel model

Standardized estimates of the cross-lagged panel model are displayed in Fig. 2. The covariance between BMI and depressive symptoms at admission was not significant and the covariance between residual variances of BMI and depressive symptoms at discharge was significant. Both autoregressive paths were significant, indicating that BMI at admission predicted BMI at discharge and depressive symptoms at admission predicted depressive symptoms at discharge. Both cross-lagged paths were also significant with positive coefficients. This indicates that higher BMI at admission predicted smaller decreases in depressive symptoms and more depressive symptoms at admission predicted larger weight gain. To test whether the size of the two cross-lagged paths differed, we compared the original model with a restricted model, in which the paths were fixed to be equal. The restricted model had a significantly worse model fit (\( \Delta \chi^2 = 21.0, p < .001, \Delta \text{AIC} = 20, \Delta \text{BIC} = 14 \)), indicating that the size of cross-lagged path estimates in the original model were different, that is, the effect of BMI at admission on changes in depressive symptoms was larger than the effect of depressive symptoms at admission on changes in BMI. However, albeit being statistically significant, these cross-lagged effects were clinically irrelevant: When comparing two patients with a BMI at admission either one SD above or below the mean BMI (i.e., 13.2 kg/m\(^2\) or 16.8 kg/m\(^2\)) at mean levels of depressive symptoms at admission, these participants differed by \( \Delta = 1.01 \) points in the PHQ–9 scores.
Similarly, when comparing two patients with depressive symptoms either one SD above or below mean PHQ–9 scores at admission (i.e., a score of 8.8 or 21.2) at mean levels of BMI at admission, their BMI differed by only 0.17 kg/m².

When including pathways of either age, sex, type of AN, comorbid depression, or antidepressant medication on the endogenous variables in our models, the size of the cross-lagged paths did not change substantially as the standardized estimates for BMI on depressive symptoms ranged between 0.04 and 0.05 and for depressive symptoms on BMI between 0.07 and 0.09. However, including length of stay substantially changed the cross-lagged path of depressive symptoms on changes in BMI (standardized estimate = −0.001, p = .914), suggesting that the small effect of higher depressive symptoms at admission on larger weight gain might be mediated by a longer length of stay. We formally tested this by including an indirect effect of depressive symptoms on changes in BMI through length of stay in the model, which was significant (standardized estimate = 0.04, p < .001). That is, higher depressive symptoms at admission lead to longer length of stay, which in turn lead to larger weight gain. Note, however, that this indirect effect was also very small in size, suggesting that it is a negligible effect in terms of clinical relevance. No interaction effects were significant (all standardized estimates <0.34, ps > 0.024), indicating that length of stay, age, sex, type of AN, comorbid depression, and antidepressant medication did not moderate the cross-lagged paths.

As sample characteristics were quite heterogeneous across the four hospitals (Table 1), we also ran the original model for each hospital separately. As sample sizes differed between hospitals, some paths were not statistically significant in the smaller subsets. However, the size of the coefficients were largely comparable across hospitals. For example, the standardized estimates for the cross-lagged path of BMI on depressive symptoms ranged between 0.03 and 0.10 and for the cross-lagged path of depressive symptoms on BMI between 0.01 and 0.08, which is consistent with the analyses of the total sample in that both cross-lagged paths were positive with very small effect sizes. A graphical depiction of the cross-lagged panel model for each hospital separately can be found at https://osf.io/dy9fn.

### 4. Discussion

The current study examined relationships between BMI and depressive symptoms in inpatients with AN cross-sectionally and longitudinally. On average, BMI increased and depressive symptoms decreased from admission to discharge. Cross-sectionally, BMI and depressive symptoms were unrelated at admission and discharge. Longitudinally, both BMI and depressive symptoms predicted changes of the other respective variable. Although findings about the role of depressive symptoms in AN treatment have been inconsistent, it has been previously suggested that higher depressive symptomatology might attenuate weight gain [5,6,22]. However, our findings clearly indicate that this does not seem to be the case. First, higher depressive symptoms at admission actually predicted larger increases in BMI, suggesting—at first glance—that depressive symptoms might foster weight gain. However, the size of this effect was very small and could be explained by the fact that patients with higher depressive symptoms had a longer treatment duration, which in turn related to higher weight gain. Similarly, BMI at admission predicted changes in depressive symptoms with a larger effect size such that a higher BMI at admission related to smaller decreases in depressive symptoms. Of note, this was a robust effect that also held when controlling for length of stay, age, sex, type of AN, comorbid depression, or antidepressant medication and was

### Table 1

<table>
<thead>
<tr>
<th>Sample characteristics.</th>
<th>Total sample (n = 3011)</th>
<th>Rosenbeck/Prien am Chiemsee (n = 1427)</th>
<th>Bad Aroslen (n = 785)</th>
<th>Bad Bramstedt (n = 358)</th>
<th>Bad Staffelstein (n = 441)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>M = 24.3 (SD = 10.5)</td>
<td>M = 22.4 (SD = 9.77)</td>
<td>M = 23.1 (SD = 9.79)</td>
<td>M = 29.8 (SD = 12.0)</td>
<td>M = 28.1 (SD = 9.83)</td>
</tr>
<tr>
<td>Adolescents</td>
<td>n = 820 (27.2%)</td>
<td>n = 569 (39.9%)</td>
<td>n = 226 (28.8%)</td>
<td>n = 24 (6.7%)</td>
<td>n = 1 (0.2%)</td>
</tr>
<tr>
<td>Adults</td>
<td>n = 2191 (72.8%)</td>
<td>n = 858 (60.1%)</td>
<td>n = 559 (71.2%)</td>
<td>n = 334 (93.3%)</td>
<td>n = 440 (99.8%)</td>
</tr>
<tr>
<td>Sex</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>n = 119 (4.0%)</td>
<td>n = 49 (3.4%)</td>
<td>n = 42 (5.4%)</td>
<td>n = 28 (7.8%)</td>
<td>n = 0 (0%)</td>
</tr>
<tr>
<td>Female</td>
<td>n = 2892 (96.0%)</td>
<td>n = 1378 (96.6%)</td>
<td>n = 743 (64.6%)</td>
<td>n = 330 (92.2%)</td>
<td>n = 441 (100%)</td>
</tr>
<tr>
<td>Type of anorexia nervosa</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Full (ICD–10 code F50.0)</td>
<td>n = 2698 (89.6%)</td>
<td>n = 1334 (93.5%)</td>
<td>n = 648 (82.5%)</td>
<td>n = 290 (81.0%)</td>
<td>n = 426 (96.6%)</td>
</tr>
<tr>
<td>Atypical (ICD–10 code F50.1)</td>
<td>n = 313 (10.4%)</td>
<td>n = 93 (6.5%)</td>
<td>n = 137 (17.5%)</td>
<td>n = 68 (19.0%)</td>
<td>n = 15 (3.4%)</td>
</tr>
<tr>
<td>Comorbid depression (ICD–10 code F32 or F33)</td>
<td>n = 1879 (62.4%)</td>
<td>n = 751 (52.6%)</td>
<td>n = 604 (76.9%)</td>
<td>n = 269 (75.1%)</td>
<td>n = 255 (57.8%)</td>
</tr>
<tr>
<td>No</td>
<td>n = 2132 (37.6%)</td>
<td>n = 676 (74.4%)</td>
<td>n = 181 (23.1%)</td>
<td>n = 89 (24.9%)</td>
<td>n = 186 (42.2%)</td>
</tr>
<tr>
<td>Medication</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Antidepressants (with or without medication)</td>
<td>n = 840 (27.9%)</td>
<td>n = 346 (24.2%)</td>
<td>n = 201 (25.6%)</td>
<td>n = 162 (45.3%)</td>
<td>n = 131 (29.7%)</td>
</tr>
<tr>
<td>No medication or medication other than antidepressants</td>
<td>n = 2171 (72.1%)</td>
<td>n = 1081 (75.8%)</td>
<td>n = 584 (74.4%)</td>
<td>n = 196 (54.7%)</td>
<td>n = 310 (70.3%)</td>
</tr>
<tr>
<td>Length of stay</td>
<td>M = 81.0 (SD = 45.9)</td>
<td>M = 97.4 (SD = 52.3)</td>
<td>M = 67.1 (SD = 32.7)</td>
<td>M = 71.0 (SD = 34.9)</td>
<td>M = 60.6 (SD = 30.5)</td>
</tr>
<tr>
<td>Body mass index (kg/m²)</td>
<td>M = 15.0 (SD = 1.81)</td>
<td>M = 14.7 (SD = 1.96)</td>
<td>M = 15.3 (SD = 1.55)</td>
<td>M = 15.4 (SD = 1.62)</td>
<td>M = 15.2 (SD = 1.72)</td>
</tr>
<tr>
<td>Admission</td>
<td>M = 17.4 (SD = 1.97)</td>
<td>M = 17.6 (SD = 2.08)</td>
<td>M = 17.3 (SD = 1.86)</td>
<td>M = 16.8 (SD = 1.85)</td>
<td>M = 17.2 (SD = 1.71)</td>
</tr>
<tr>
<td>Discharge</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Patient Health Questionnaire–9</td>
<td>M = 15.0 (SD = 6.21)</td>
<td>M = 14.6 (SD = 6.29)</td>
<td>M = 15.1 (SD = 5.97)</td>
<td>M = 15.6 (SD = 6.22)</td>
<td>M = 15.5 (SD = 6.28)</td>
</tr>
<tr>
<td>Admission</td>
<td>M = 8.95 (SD = 5.71)</td>
<td>M = 8.21 (SD = 5.79)</td>
<td>M = 9.09 (SD = 5.37)</td>
<td>M = 10.2 (SD = 5.93)</td>
<td>M = 10.0 (SD = 5.48)</td>
</tr>
</tbody>
</table>

*Note that for those with full syndrome AN, AN subtype was not coded by the therapists for n = 1434 patients (53.2%). For the minority of patients with available subtype specification (n = 1264, 46.8%), n = 836 (66.1%) had restrictive-type AN (ICD–10 code F50.00), n = 423 (33.5%) had binge/purge-type AN (ICD–10 code F50.01), and n = 5 (0.4%) had unspecified AN subtype (ICD–10 code F50.08). Due to the large amount of missing data, however, this information was not used in further analyses.

*1 Data based on n = 2396 due to missing values.
also not moderated by these variables. However, both cross-legged effects were very small and, despite being statistically significant, border at clinical irrelevance. Thus, on average, neither do comorbid depressive symptoms affect weight gain nor does BMI at admission affect changes in depressive symptoms during inpatient treatment for AN on a meaningful level.

Interpretation of these findings is limited by the use of clinical diagnoses, which may be less precise compared to structured clinical interviews, and self-reported depressive symptoms, which may be influenced by social desirability, simulation, or recall bias. Thus, we cannot exclude the possibility that findings may be different when using diagnoses of comorbid depression derived from a structured clinical interview. Relatively, information on AN subtype was only available for a subset of patients, which is why moderating effects of this variable could not be tested. As weight gain trajectories during treatment seem to be different in patients with restrictive vs. binge/purge-type AN [18] and the prevalence and type of comorbid mental disorders also differ between these two subtypes, it may be that relationships between depressive symptoms and weight change might differ as well. Similarly, information on lifetime eating disorder and other diagnoses as well as weight history were not available due to the retrospective nature of this study but may be relevant variables to consider when analyzing individual differences in weight gain.

Interpretation is further limited to inpatients with AN in Germany and, thus, findings may not translate to other countries, for example, as inpatient treatment for AN in Germany is longer than most of the structured treatments in other parts of the world [2]. However, the average treatment duration in the current study (81 days) was close to the worldwide average treatment duration (76 days) and lower than the average treatment duration in Europe (106 days) that has been reported in a recent meta-analysis of inpatient and daypatient treatments [11], indicating that treatment duration was not substantially longer than in most other studies.

Another limitation is that only BMI at admission and discharge was available, with which only linear changes in body weight can be modeled. However, weight change during inpatient treatment of AN actually is non-linear, with the most common weight curve representing average treatment duration in Europe (106 days) that has been reported -20, 28]. Thus, examining these non-linear changes in body weight and maybe also dynamic changes in depressive symptoms during treatment may indeed reveal an interplay between these variables in different treatment stages. Finally, from a methodological standpoint, cross-legged panel models came under increased scrutiny in recent years as, despite their intention, they cannot fully disentangle between-person and within-person effects and causal claims based on these models may be unfounded [9, 17]. Several of suggested alternatives such as the random-intercept cross-legged panel model, however, need at least three assessment waves and were, therefore, not suitable for analyzing our data. Future studies should thus consider investigating whether our findings hold true when accounting for stable trait components examining longitudinal data with at least one follow-up assessment.

In conclusion, our findings suggest that depressive symptoms do not have a negative impact on short-term treatment outcome in terms of weight gain in inpatients with AN. That is, eating disorder-specific inpatient treatment leads to a significant and similar increase in body weight in patients with AN, independent of their depressive symptomatology. Therefore, patients reporting with elevated depressive symptoms do not require a specially tailored type of treatment to achieve weight restoration.

Declaration of Competing Interest

None.
