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Non-specific predictors of weight gain in the early stages of outpatient cognitive behavioral therapy for adults with anorexia nervosa: Replication and extension

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Running head: WEIGHT GAIN EARLY IN CBT FOR ANOREXIA NERVOSA

Non-specific predictors of weight gain in the early stages of outpatient cognitive behavioral therapy for adults with anorexia nervosa: Replication and extension

Abstract

Objective: This study extends previous work, examining psychological factors that influence the level of weight gain across the first 20 sessions of cognitive behavioral therapy (CBT) for anorexia nervosa.

Method: Thirty-two patients with anorexia nervosa (mean BMI = 16.0; <u>SD</u> = 1.11) completed measures of eating attitudes and comorbid axis 1 pathology at the outset of CBT, and their weight gain was measured at the sixth and twentieth sessions of the therapy.

Results: Unhealthy eating attitudes at the start of therapy were associated with slower weight gain across the first 20 sessions of CBT. In contrast, higher levels of pre-treatment axis 1 pathology were associated with slower weight gain only after the sixth session. The axis 1 features that moderated weight gain over 20 sessions of CBT were broader than those that had previously been shown to predict weight gain over the first ten CBT sessions.

Discussion: During CBT for anorexia nervosa, weight gain might be enhanced by addressing a range of aspects of axis 1 pathology (e.g., depression, hostility, features of anxiety). However, that approach is likely to be less important at first than directly addressing eating pathology and overvalued ideas about eating, shape and weight.

Key words: cognitive behavioral therapy; anorexia nervosa; weight gain

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Non-specific predictors of weight gain in the early stages of outpatient cognitive behavioral therapy for adults with anorexia nervosa: Replication and extension

There is little empirical evidence regarding treatment outcomes for adults with anorexia nervosa, with most therapies having relatively weak effects, with little difference in their impact (1-3). Furthermore, retention and treatment adherence are poor (3). While there is some preliminary evidence that cognitive-behavioral therapy (CBT) is an effective approach in some adult cases of anorexia nervosa (4-6) and that it might reduce relapse risk in such patients (7), those studies have been limited in terms of the range of patients' weights. A range of factors have been shown to impact on the prognosis and treatment outcomes for anorexia nervosa, including personality traits, neuropsychological characteristics and mood (8-10). However, as weight gain is the key clinical outcome in the treatment of anorexia nervosa (1), it is clearly important to consider the impact of factors that might predict higher or lower levels of weight gain in anorexia nervosa.

Studying the early impact of CBT in a routine clinical sample of anorexia nervosa sufferers (mean initial BMI = 16.4; range = 12.8-18.4), Lockwood and colleagues (11) have shown that eating-disordered attitudes and anxiety impact on weight gain over the first ten sessions of outpatient treatment. Those patients with more severe eating attitudes were slower to gain weight over the sixth to tenth session, and those with more severe levels of general and phobic anxiety were slower to gain weight across the whole ten sessions. This impact of anxiety can be conceptualised by seeing restriction of intake as a safety behavior, offering short-term relief from anxiety but contributing to long-term anxiety enhancement. However, this is a preliminary finding, which needs to be replicated and extended further into the timeframe of CBT for anorexia nervosa.

The current study aimed to replicate and extend previous work (11), examining predictors of weight gain over the first 20 sessions of outpatient CBT for anorexia nervosa. Eating attitudes, body mass index and levels of axis 1 comorbidity at the start of treatment were used to predict the amount of weight gain over this period. It was hypothesised that

anorexia nervosa sufferers with higher initial levels of eating and comorbid pathology would show slower weight gain across treatment.

Method

Participants

The participants consisted of 37 women with a diagnosis of anorexia nervosa, who entered outpatient cognitive behavioral therapy for their eating disorder. This sample is entirely different to that reported by the previous study (11). Of the total sample, 20 had anorexia nervosa of the restrictive subtype, 15 had the binge/purge subtype, and 2 had atypical anorexia nervosa (12). Participants were consecutive outpatients and participation was entirely voluntary, as stated in the information and consent sheets of the study. An a priori power calculation showed that a sample size of 34 patients would be necessary to have 80% power to detect a clinically significant effect in terms of predicting weight changes (one-tailed alpha = .05). In order to allow for drop-out rates comparable to the previous study (11), a total sample of 40 patients was sought. During the data collection phase (20 sessions of CBT), five patients disengaged (two dropped out after erratic attendance, one left due to travel plans, one ended treatment after her therapist left the service, and one moved out of the clinic's catchment area and opted to restart treatment in her new area). No patients left the trial or were withdrawn for any other reason.

Measures and procedure

Ethical approval for the study was granted by the South East London Research Ethics Committee (equivalent of an IRB). At the start of treatment, patients were measured to calculate their body mass index. All met DSM-IV criteria, as assessed by clinicians trained in diagnosis. The participants also completed the following self-report measures at this point. Patients' objective weights were taken by their clinicians at each therapy session, including the sixth and twentieth sessions.

Eating Disorder Examination-Questionnaire (EDE-Q – v.6 [13]). The EDE-Q is a selfreport measure of eating psychopathology, addressing cognitions regarding restriction and eating, weight and shape concerns. It has been widely validated in clinical and non-clinical groups (14-15). Items are rated on Likert scales (0 to 6) that capture frequency or severity of eating disorder attitudes and behaviours. Individual subscales scores can be produced, and those subscales can be used to yield a global EDE-Q score (as used in this study). Higher scores indicate a greater level of eating pathology.

<u>Brief Symptom Inventory (BSI [16]).</u> The BSI was used to explore comorbid pathology. It addresses: somatisation; obsessive-compulsive features; interpersonal sensitivity; depression; anxiety; hostility; phobic anxiety; paranoid ideation; and psychoticism. The BSI has satisfactory psychometric properties (17). Items are rated on a Likert scale, ranging from 0 (not at all) to 4 (extremely). The scale can be used to yield a global severity score by summing all 53 items, or individual subscale scores can be produced, reflecting the separate elements of comorbid pathology (as was done in this study). Higher scores suggest greater psychological symptom severity.

CBT for anorexia nervosa

The cognitive-behavioral treatment used in this study is described in detail elsewhere (18-19). While it resembles CBT as developed by Fairburn (13), this approach places a greater emphasis on dietary changes at the beginning of treatment, with the aim of weight gain from early sessions, in order to facilitate greater use of behavioral experimentation. The approach also differs in that it entails a more explicitly Socratic approach towards behavioral and cognitive change. Sessions were usually weekly, and the aim is for 40 sessions of treatment. In the early part of treatment (sessions 1-6), the emphasis is on achieving behavioral changes via exposure - increasing structure and amount of food intake (19). This is supported with psychoeducation regarding starvation effects and patients' nutritional and energy needs. Motivational work is used where necessary, but with a behavioral focus (20). After the sixth session, the ongoing behavioral and dietary changes are supplemented with more behavioral experiments (e.g., testing specific beliefs about food intake) and cognitive restructuring (addressing beliefs about eating, shape and weight). There might also be a focus on body image and comorbid difficulties (e.g., low self-esteem, perfectionism,

depression and anxiety), although that would normally be after the first 20 sessions of CBT (as covered by this study), alongside relapse prevention.

Data analysis

Data were first analysed for normality of distribution using Kolmogorov-Smirnov tests. As they were not normally distributed in the great majority of cases, non-parametric analyses were carried out. Weights at different time points were compared using Friedman's test and post-hoc Wilcoxon tests. The weight change scores were then correlated (Spearman's <u>rho</u>) with patients' start of therapy levels of eating and comorbid pathology.

Results

Levels of pathology among anorexia nervosa patients who did or did not drop out of CBT

Table 1 shows the mean start of treatment scores and other characteristics for the whole sample of patients, divided into those who reached the 20th session of CBT ($\underline{N} = 32$) and those who left treatment before that point ($\underline{N} = 5$) for whatever reason (e.g., dropping out, moving out of area). There were no differences between those who did or did not remain in treatment for this time period. Therefore, it can be concluded that loss to treatment is unlikely to have skewed the findings below.

Insert Table 1 about here

Weight changes across the first 20 sessions of CBT for anorexia nervosa

Considering the 32 patients who reached the 20th session, their mean weight at the outset of treatment was 43.1 kg (<u>SD</u> = 4.44). By session six, this had risen to 44.1 kg (<u>SD</u> = 4.48). At session 10, the mean weight was 44.7 kg (<u>SD</u> = 4.60). Finally, at session 20, their mean weight was 45.6 kg (<u>SD</u> = 5.04). This pattern demonstrates a relatively smooth rise in weight across this phase of treatment. A Friedman test showed that the increase in weight was significant ($X^2 = 52.3$; <u>P</u> < .001). Post hoc Wilcoxon tests showed that the differences

between each pair of time points were also significant ($\underline{Z} > 3.30$, $\underline{P} < .001$ in all cases), confirming that relatively smooth rise in weight.

Associations of initial eating and comorbid pathology with weight change

Patients' weight changes across the whole of this period of treatment (sessions 1-20) and during the early and late parts of this period (sessions 1-6; sessions 6-20) were correlated with their initial levels of eating and comorbid pathology. Change was calculated as the later score minus the earlier score, so that positive scores indicate weight gain, while negative scores indicate weight loss. Table 2 shows the correlation coefficients (Spearman's <u>rho</u>). Negative correlations indicate that patients with higher levels of that initial element of psychopathology were slower to gain weight (or even lost weight) over the relevant time period in treatment.

Insert Table 2 about here

Across the whole 20 sessions, weight gain was slower among patients with higher initial levels of eating pathology, somatisation, interpersonal sensitivity, depression, hostility and phobic anxiety. However, the pattern of associations was different across the early and later parts of this time period. During the first six sessions, higher initial EDE-Q scores predicted lower levels of weight gain, but lower initial BMI was associated with greater weight gain over this time period. In contrast, during the later sessions, initial comorbid pathology played a much stronger role in predicting change in weight. Lower weight gain in this period was still associated with higher initial EDE-Q scores (but not BMI), but was also associated with higher levels of obsessive-compulsive pathology, interpersonal sensitivity, depression, hostility and psychoticism.

Discussion

While it is not yet clear what constitutes the optimal treatment for anorexia nervosa, it

remains important to understand what predicts the impact of treatments on the key outcome variable – weight gain (1). This study has examined the factors that might predict weight gain in the first 20 sessions of CBT for anorexia nervosa, building on previous research (11). This form of CBT led to significant weight gains among those patients who completed all 20 treatment sessions, having started with a mean BMI of 16.0 (mean weight = 43.1kg, mean height = 1.64m). In order to achieve a mean BMI of 19-20 (as suggested elsewhere [13]), the present group would need to reach a mean weight of 51.0-53.7kg. The 32 anorexia nervosa patients achieved an initial weight gain of 2.50 kg during the first 20 sessions of treatment 0.125 kg/week). This is lower than the maximum rate suggested in existing guidelines (3), and suggests that treatment might require approximately 60 sessions, unless the rate of weight gain were increased in later sessions. Levels of eating and comorbid pathology were associated with weight gain over the whole period of 20 treatment sessions. However, while more negative eating cognitions (EDE-Q score) were associated with slower weight gain only in the latter part of this phase of treatment (sessions 6-20).

The rate of weight gain in this study was slower than that shown by the previous study (11), despite a comparable therapeutic approach. Therefore, it is possible that the lower initial BMI of the sample in this study indicates that the time required for treatment of anorexia nervosa using CBT might need to be extended disproportionately for patients of a lower BMI. In a similar vein, given that early weight gain predicts better outcome for anorexia nervosa patients (21), the current results suggest that there should be a more intensive therapeutic focus on eating pathology early in CBT for patients who have high EDE-Q scores at entry to treatment. It is also noteworthy that a wider range of comorbid pathologies were associated with slower weight gain than in the previous study (11), suggesting that factors such as depression, hostility and psychotic tendencies have their impact on weight gain after the first ten sessions but before the 20th session. However, existing cognitive-behavioural approaches tend to focus on such comorbid pathology in the later part of treatment (13,18).

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The present findings indicate that there should be a stronger emphasis on comorbid pathology (e.g., depression) from earlier than session 20, in order to facilitate weight gain.

This study has confirmed the need to consider the role of initial eating pathology and comorbid psychopathology when understanding rates of weight gain during CBT for anorexia nervosa. Unlike previous findings (11), core anxiety was not a significant predictor of weight gain in the current study. Further research is needed to clarify whether or not anxiety plays a role in influencing weight gain. In anorexia nervosa, and at what stage in treatment. It will also be necessary to explain the mechanisms underlying other elements of pathology that predict weight gain. For example, levels of depression and hostility might have their impact through reduction in willingness to consider change as treatment progresses. This research also needs to be extended in other ways – particularly to describe and explain rates of weight gain across the whole course of therapy, and to determine whether these early predictors of weight gain apply in other therapies (allowing for more effective treatment matching).

In summary, both eating and comorbid pathology need to be assessed prior to CBT for anorexia nervosa, and their potential relevance to treatment should be formulated. Clinicians should be ready to address a wide range of comorbid pathology (e.g., elements of anxiety and depression) in order to facilitate weight gain within that treatment. However, those issues need to be addressed after initially addressing eating-related cognitions (13, 18).

Note

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Table 1

Mean levels of eating and comorbid pathology at the start of treatment among anorexia nervosa sufferers who did and did not complete the first 20 sessions of CBT for their eating disorder.

	Completers		Drop-outs		Mann-Whitney	
	(<u>N</u> = 32)		(<u>N</u> = 5)		tests	
Mean scores at start of treatment	M	(<u>SD</u>)	<u>M</u>	(<u>SD</u>)	<u>U</u>	<u>P</u>
Age	26.1	(8.74)	26.2	(4.92)	64.5	<u>NS</u>
Duration of illness	6.25	(4.71)	6	(4.30)	78	<u>NS</u>
BMI	16.0	(1.11)	16.9	(2.65)	53	<u>NS</u>
EDE-Q Global Score	2.92	(1.49)	3.62	(0.59)	59	<u>NS</u>
BSI subscales						
Somatisation	1.12	(0.77)	1.83	(1.57)	61	<u>NS</u>
Obsession-compulsive	1.53	(1.04)	1.30	(1.21)	65	<u>NS</u>
Interpersonal sensitivity	1.61	(1.31)	1.45	(1.27)	74	<u>NS</u>
Depression	1.56	(1.32)	1.77	(1.54)	70.5	<u>NS</u>
Anxiety	1.21	(1.03)	1.10	(1.37)	62.5	<u>NS</u>
Hostility	0.98	(0.97)	0.60	(0.85)	53	<u>NS</u>
Phobic anxiety	0.66	(0.84)	0.76	(0.98)	78.5	<u>NS</u>
Paranoid ideation	0.89	(0.88)	0.88	(1.06)	74	<u>NS</u>
Psychoticism	1.31	(1.03)	1.32	(1.40)	76.5	<u>NS</u>

 $\underline{NS} = not significant$

Table 2

Correlations (Spearman's <u>rho</u>) of weight changes with initial eating pathology (EDE-Q scores; BMI) and comorbid pathology (BSI scores) among 32 patients completing 20 sessions of cognitive-behavior therapy for anorexia nervosa

	Correlations with weight changes across different					
	parts of treatment					
	Sessions 1-20	Sessions 1-6	Sessions 6-20			
BMI	216	378*	037			
EDE-Q Global score	479**	431*	391*			
BSI subscales						
Somatisation	418*	213	347			
Obsession-compulsive	304	078	385*			
Interpersonal sensitivity	508**	268	514**			
Depression	428*	131	493**			
Anxiety	326	189	272			
Hostility	419*	177	416*			
Phobic anxiety	355*	187	296			
Paranoid ideation	304	386*	157			
Psychoticism	309	091	374*			

* <u>P</u> < .05, ** <u>P</u> < .01