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Psychotherapy for transdiagnostic binge eating: A randomized controlled trial of cognitive-behavioural therapy, appetite-focused cognitive-behavioural therapy, and schema therapy

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ABSTRACT

Cognitive-behavioural therapy (CBT) is the recommended treatment for binge eating, yet many individuals do not recover, and innovative new treatments have been called for. The current study compares traditional CBT with two augmented versions of CBT; schema therapy, which focuses on early life experiences as pivotal in the history of the eating disorder; and appetite-focused CBT, which emphasises the role of recognising and responding to appetite in binge eating. 112 women with transdiagnostic DSM-IV binge eating were randomized to the three therapies. Therapy consisted of weekly sessions for six months, followed by monthly sessions for six months. Primary outcome was the frequency of binge eating. Secondary and tertiary outcomes were other behavioural and psychological aspects of the eating disorder, and other areas of functioning. No differences among the three therapy groups were found on primary or other outcomes. Across groups, large effect sizes were found for improvement in binge eating, other eating disorder symptoms and overall functioning. Schema therapy and appetite-focused CBT are likely to be suitable alternative treatments to traditional CBT for binge eating.

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1. Introduction

Cognitive-behavioural therapy (CBT) is the treatment of choice for adults with binge eating, including both bulimia nervosa (BN) and binge eating disorder (BED), yet many individuals do not recover with CBT (Smink et al., 2013), or relapse following successful treatment, and novel and more effective treatments have been called for (Hay, 2013). Continued research is needed to examine ways to augment and improve upon CBT. This could be achieved by increasing the therapeutic focus on specific domains of psychopathology in individuals with BN and BED, by addressing domains of therapy that are not currently addressed, or by importing elements of treatments that have demonstrated efficacy for other related disorders (for example, schema therapy for mood disorders (Carter et al., 2013)). It has been suggested that additive designs, in which an additional element or focus is added to an existing

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evidence-based treatment to test whether outcome is improved, maximize the amount of basic knowledge and specific conclusions generated from randomized controlled trials (RCTs) (Borkovec and Sibrava, 2005). Study designs comparing an existing effective treatment with augmented or novel elements can yield useful conclusions either if outcomes improve relative to the original treatment or if alternative versions of treatments perform comparably, giving additional treatment options to clinicians and patients.

Two promising directions for the augmentation of CBT, one targeting appetite, and the other targeting underlying schema, were used in the current study. Appetite-focused CBT (CBT-A), developed for the present study, was based on an etiological model in which diminished hunger recognition and insensitivity to satiety cues are instrumental in initiating and perpetuating binge eating (Hetherington and Rolls, 1989). Neuroendocrine and metabolic systems involved in the regulation of appetite, satiety, and weight can be disturbed in individuals who binge (Jimerson et al., 2000; Tanaka et al., 2003; Yanovski, 1995). Appetite-focused CBT emphasises how disregarding appetite is important in the

development and maintenance of disordered eating, and focuses on recognising and responding to hunger and satiety in eliminating binge eating. Evidence exists that focusing on appetite in treatment can be efficacious in reducing binge eating, and that patients may prefer monitoring appetite over traditional monitoring of food and fluid intake (Dicker and Craighead, 2004). Appetite-focused CBT has not been the subject of clinical trials, although a small trial of appetite-focused dialectical behaviour therapy has shown some promise for bulimia nervosa (Hill et al., 2011).

Schema therapy was adapted for eating disorders based on the observed link between early life experiences and the development and maintenance of eating problems (Waller et al., 2007). The association between childhood experiences and psychological disorders, including eating disorders (Steiger et al., 2010), has been shown to be mediated by the development of maladaptive schemas (Wright et al., 2009). It has been suggested that improved treatment efficacy may be obtained with models that better incorporate past experiences in the etiology of these disorders (Waller et al., 2007). Schema therapy is efficacious in treating psychological disturbances, including borderline personality disorder (Farrell et al., 2009; Giesen-Bloo et al., 2006), depression (Carter et al., 2013), substance abuse (Ball, 1998), agoraphobia (Bamber, 2004), and posttraumatic stress disorder (Young, 2005). There is evidence of an association between schemas and eating disorder behaviours (Waller, 2003); and clinical improvement has been reported in single case studies using video therapy schema therapy (Simpson and Slowey, 2011) and imagery rescripting (Ohanian, 2002) for eating disorders, and in a case series of group schema modes therapy (Simpson et al., 2010). Therapy aims first to increase awareness of maladaptive schemas or schema modes and the early experiences from which they developed, and then to treat the maladaptive schemas, thereby reducing the current drive for eating disordered behaviours.

The present study reports a randomized controlled trial of traditional CBT for transdiagnostic DSM-IV binge eating, and two versions of CBT, one that augments the appetite-focus, CBT-A, and one that augments the cognitive component, schema therapy. It was hypothesized that schema therapy and appetite-focused CBT would result in better eating disorder and general outcome, as measured by the frequency of binge eating and purging, the severity of eating disorder attitudes, and overall functioning, as measured by the Global Assessment of Functioning, Axis V of DSM-IV.

2. Method

2.1. Participants

Participants were recruited by referrals from general practitioners or other health professionals and by advertisements. Inclusion criteria were female gender, age 16–65, and a primary DSM-IV binge eating diagnosis, with objective binge episodes, the consumption of an abnormally large quantity of food within a discrete time period, the subjective experience of dyscontrol, and not currently underweight. Exclusion criteria were other conditions requiring treatment – severe major depression or serious suicidal intent, severe psychoactive substance dependence, bipolar I disorder, schizophrenia, severe physical illness including severe medical complications of the eating disorder, cognitive impairment, psychotropic medication, and an adequate trial of CBT or schema therapy in the past year.

2.2. Procedure

The trial received ethical approval from the Upper South A Regional Ethics Committee, and was conducted in Christchurch, New Zealand, recruiting between May 2005 and October 2010.¹ Initial telephone screening included describing the

research nature of the programme, determining the likely presence of binge eating, and likely absence of exclusion criteria. A clinical assessment with a clinical psychologist was scheduled for potentially eligible and interested individuals. At this assessment, eligibility was determined, and after complete description of the study to participants, written informed consent was obtained. Baseline assessments confirmed study inclusion and exclusion criteria, including structured clinical interviews (SCID-I and II, EDE-12) and completion of self-report questionnaires. The study had a three-arm parallel group design with participants randomized in a 1:1:1 ratio based on a randomization sequence of permuted blocks of 30. The randomization sequence and allocation to treatment were performed by someone independent of the study and blind to baseline assessment information. Treatment allocation was made available to the therapist and patient in sealed envelopes after baseline assessments.

2.3. Therapy

Therapy consisted of six months of weekly individual psychotherapy sessions, followed by six months of approximately monthly sessions. Four clinical psychologists, experienced in all therapies, and in treating eating disorders, were trained in the delivery of the three therapy conditions. Prior to the commencement of the clinical trial, therapists treated training cases in each modality. This involved close review of audio-recorded therapy sessions by the clinical supervisor, a clinical psychologist with experience in supervising the therapies, manual-based eating disorders treatment, and rating treatment competence (Carter et al., 2013). Therapists received weekly clinical supervision from the above clinical psychologist, including close attention to adherence to the three models of therapy, therapy manuals, and the quality of treatment. Training and trial cases were rated for competence using the Cognitive Therapy Rating Scale for CBT (Dobson et al., 1985), and modified forms of the Cognitive Therapy Rating Scale for schema therapy and appetite-focused CBT. Adequate competency on the Cognitive Therapy Rating Scale is defined as a score of 40 or more. Mean ratings over the course of the study were 52.0 (8.2) for CBT, 51.1 (5.7) for schema therapy, and 45.9 (5.7) for appetite-focused CBT for randomly selected sessions, which were not significantly different for the three therapies ($F(2)=2.94, p=0.07$).

2.3.1. Cognitive-behavioural therapy

Cognitive-behavioural therapy for binge eating is based on the premise that dysfunctional thinking about food, eating, body shape and weight is central to the onset and maintenance of disordered eating behaviour. CBT for binge eating aims to correct the disturbed pattern of eating by identifying and evaluating unhelpful thinking, understanding and managing cues for binge eating, and through education and advice about resuming normal eating. CBT was manual-based, adapted from previous CBT manuals for bulimia nervosa and anorexia nervosa, based on traditional CBT (Fairburn et al., 1993), and supported in prior randomized controlled trials (Bulik et al., 1998; McIntosh et al., 2005). Therapy was divided into three overlapping phases. Phase one introduces CBT and its rationale, and the core techniques of self-monitoring and homework. Motivation to change is recognized and addressed. Increased regularity and variety of eating is prescribed, and appropriate portion sizes are successively approximated. Patients are coached to use strategies to resist the urge to binge. In phase two further CBT skills are taught – challenging dysfunctional thoughts, thought restructuring, techniques for avoiding binge eating, and identifying cue-behaviour-consequence sequences. Written psychoeducational materials are provided. Phase three prepares the patient for termination, providing information on the relapse and recovery process, and teaching strategies to reduce risk of relapse.

2.3.2. Appetite-focused CBT

Appetite-focused CBT augments standard CBT with strategies to assist individuals to recognize and respond to hunger and satiety cues in the return to normal eating (McIntosh et al., 2007). All aspects of self-monitoring, including forms, instructions and the rationale of self-monitoring emphasize awareness of hunger and satiety, encouraging eating in response to moderate hunger, and cessation of eating in response to moderate satiety. Education and advice about food choices are informed by principles of eating for satiety (Latner et al., 2009; Ludwig, 2000), choosing foods with greater volume and lower energy density, greater satiating potential, such as the inclusion of protein throughout the day, and choosing longer lasting carbohydrates with lower glycemic indices. Individuals learn to identify non-appetite-related emotional or situational cues for eating, and are encouraged to use appropriate non-food responses in these situations.

2.3.3. Schema therapy

Schema therapy is a development of CBT that focuses on modifying maladaptive schemas in order to enable core psychological needs to be met (Young et al., 2003), and to bring about change in the eating disorder. Schema therapy relies heavily on imagery and other experiential techniques to bring about cognitive, emotional and behaviour change. Imagery rescripting aims to modify unhealthy core beliefs by allowing the patient's healthy adult self to bring a more mature perspective, including a greater capacity for rational thought and emotional

¹ The trial protocol is available from the corresponding author on request.

responsivity to the understanding of material that had been encoded during childhood and thereafter (Ohanian, 2002). Schema therapy for eating disorders involves identifying, challenging and restructuring schemas rather than addressing negative automatic thoughts or dysfunctional assumptions relating to food, eating, body shape, and weight. Bingeing and other impulsive behaviours serve the function of avoiding affect associated with maladaptive schemas. For example, being excluded from a social occasion may trigger abandonment and mistrust-abuse schemas, which lead to feeling angry and isolated. Binge eating then serves to avoid the schemas, and is reinforced by the reduction of negative affect. Complex combinations of schemas led to theory of schema modes, enduring facets of the self, not fully integrated with other facets. This theory has led to the development of mode work within schema therapy, involving the identification and integration of the modes under the overseeing health adult mode. Limited reparenting involves the establishment of a secure attachment with the therapist, in order to meet the patient's unmet core needs. Helping the patient find experiences that were missed in early childhood heals damaging experiences that led to maladaptive schemas or modes and in turn to destructive eating disorder behaviours.

2.4. Assessments

2.4.1. Eating disorder

The Eating Disorder Examination-12 (EDE-12; Fairburn and Cooper, 1993) is a clinician-administered structured interview measuring the severity of eating disorder psychopathology. From this interview, conducted in full or part at baseline, weeks 4, 8, 26, end of treatment, and 12 month follow-up, measures were derived of the frequency of objective binge eating (eating an objectively large quantity of food in a discrete period of time accompanied by loss of control), frequency of purging (self-induced vomiting, laxative and diuretic abuse), dietary restraint, eating, weight and shape concerns, dietary restriction, and pattern of eating (regularity). The Eating Disorder Inventory-2 (EDI-2; Garner et al., 1983) is a self-report questionnaire, the first three subscales of which, drive for thinness, body dissatisfaction, and bulimia, are reported. A global eating disorder rating was completed at the end of the clinical interview (McIntosh et al., 2005). Participants were rated as 1=having no features of eating disorder; 2=having a few features of eating disorder; 3=not meeting full diagnostic criteria for an eating disorder, but having a number of features; 4=meeting full criteria for an eating disorder.

2.4.2. General psychopathology

Psychiatric comorbidity was assessed using the Structured Clinical Interview for DSM-IV (SCID; First et al., 1996). The Symptom Checklist-90-Revised (SCL-90-R; Derogatis et al., 1973), a self-report measure of general psychiatric symptoms, was administered at baseline, end of treatment, and at 12 month follow-up. The global severity index measured overall psychopathology. The SCL-90-R depression subscale measured severity of depression.

2.5. Perceived credibility of therapy

Participants rated the credibility of therapy on a 7-point Likert-type scale at the beginning and end of treatment, answering the questions "How logical does this therapy seem?", "How useful does this therapy seem?", "How confident are you that this therapy will be successful?", and "How confident would you be in recommending this therapy to a friend with similar difficulties?".

2.6. Outcome measures

Outcome measures were determined a priori. Primary outcome was the frequency of objective binge episodes. Secondary outcome comprised other features of the eating disorder; abstinence from binge eating, frequency of purging, eating disorder remission (abstinence from binge eating or purging, and no eating disorder diagnosis in the past 28 days), EDE-12 dietary restraint, eating, weight and shape concerns, and global eating disorder severity, EDI-2 drive for thinness, body dissatisfaction and bulimia subscales, and global assessment of functioning (GAF, Axis V of DSM-IV). Tertiary outcome comprised mood and general distress, measured using the SCL-90-R depression subscale and Global Severity Index, respectively. Primary outcome, the frequency of binge eating episodes, was rated by an assessor who was unaware of treatment allocation. Other outcome measures were assessed by trained interviewers who may not have been blind to treatment allocation, as some interviews were conducted by non-treating clinicians who were involved in group supervision.

2.7. Statistical analyses

The sample size calculation for the study was based on results of previous controlled trials of CBT for bulimia nervosa and binge eating disorder (Agras et al., 2000; Wilfley et al., 2002). For the primary outcome variable, it was calculated that for 80% power and a 2-tailed $\alpha=0.05$, it would be possible to detect an average difference of two episodes of binge eating per 28 days among treatments with a

group size of 67 participants. On the basis of these power calculations, it was planned to recruit 200 participants for this study to allow for attrition; however, recruitment was slower than expected, and recruitment closed due to funding ending with 112 participants giving approximately 80% power (2-tailed $\alpha=0.05$) to show an effect size of 0.65 equating to a difference of approximately 2.7 binge episodes per 28 days.

Change from baseline to end of treatment was examined using paired *t*-tests for continuous variables and the χ^2 test for dichotomous outcome variables. Effect sizes for these changes were calculated using Cohen's *d*, using the formula $mean-diff/sddiff$. Analysis of variance was used to compare the numbers of individuals completing treatment, participants' perceptions of treatment, the number of sessions attended, and the total therapy time among the three groups. The primary analysis of outcome at end of treatment was by intention-to-treat. Where there were missing end of treatment data, the last observation was carried forward to characterize a participant's response. The primary analysis of outcome at 12 month follow-up (twelve months after treatment ended, two years after baseline assessment) included all participants for whom data were available without imputation of the last observation. Change variables were calculated from baseline to end of treatment and from baseline to 12 month follow-up for the three treatment groups. Analysis of covariance was conducted for primary, secondary, and tertiary outcome change from baseline variables by therapy group, with the baseline value included as the covariate. Effect sizes (Cohen's *d*) were calculated using the difference between means for CBT and either schema therapy or appetite-focused CBT, whichever difference was larger. A secondary analysis was conducted for those who completed treatment using analysis of covariance for all outcome change from baseline variables by therapy group, with the baseline value included as the covariate.

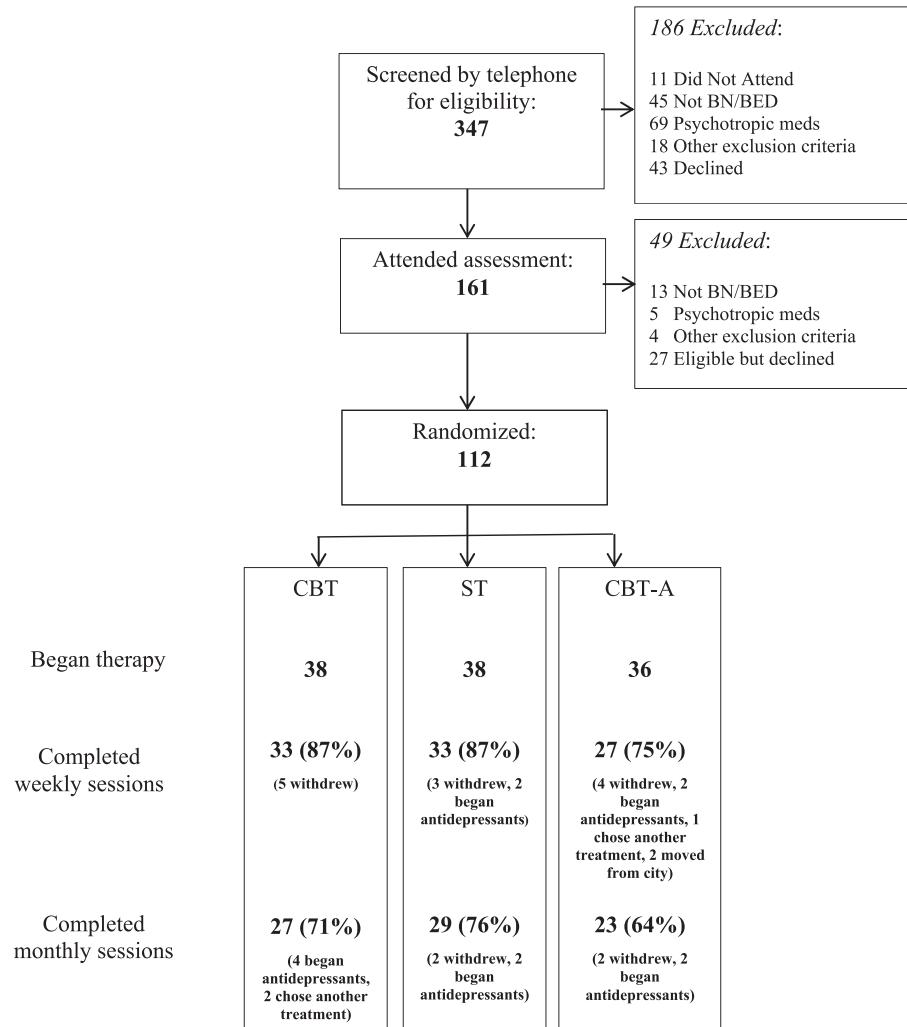
3. Results

Fig. 1 shows the flow of participants through the study. Three hundred and forty-seven participants were screened for the study, 112 women with transdiagnostic binge eating were randomized to therapy, 38 to CBT, 38 to schema therapy, and 36 to CBT-A.

Table 1 shows the pre-treatment demographic and psychiatric comorbidity data for the total sample, and for the three treatment groups. Mean age of participants was 35.3 years, with a body mass index (kg/m^2) of 29.9. Mean duration of the eating disorder was 15.2 years. Almost 40% of the sample had never married, and 11% were currently unemployed. Mean length of education was 15.4 years. Ethnicity was predominantly New Zealand Caucasian (67%); other ethnicities were New Zealand Maori (10%), non-New Zealand born Caucasian (17%, European, North and South American, African), and Asian (4%).

Fifty two percent of the sample engaged in purging or non-purging behaviours designed to compensate for binge eating (including vomiting, laxative or diuretic use, excessive exercise); 48% did not engage in compensatory behaviours (Jordan et al., 2014). Seven percent of participants had previously met criteria for anorexia nervosa. Approximately two-thirds had a lifetime history of major depression, with 26% meeting criteria currently. One third of participants had a lifetime alcohol use disorder and 22% a substance use disorder. Over one quarter of the sample had a lifetime history of social phobia, with the majority of these currently meeting criteria for social phobia. Other lifetime anxiety disorders included posttraumatic stress disorder (19%), generalized anxiety disorder (21%), and obsessive compulsive disorder (8%). The lifetime rate of intentional non-suicidal self-harm was 29%, and 18% of participants had made a suicide attempt. No differences were found among the three treatment groups on demographic or psychiatric comorbidity variables at baseline.

No differences were found among the three therapy groups in the proportion completing treatment ($\chi^2=1.38$, $p=0.50$), nor in the number of therapy sessions attended (CBT 17.5 (5.5); schema therapy 18.0 (4.7); CBT-A 16.4 (5.1); $F(2,110)=0.91$, $p=0.40$). The three therapy groups did not differ on measures of perceived treatment credibility at the time of randomization, how logical (CBT 6.4 (0.9); schema therapy 6.2 (0.9); CBT-A 6.3 (0.8); $F(2,110)=0.74$, $p=0.48$), useful (CBT 6.3 (1.0), schema therapy 6.2 (1.0), CBT-



CBT=cognitive-behavioural therapy; ST=schema therapy; CBT-A=appetite-focused CBT; BN=bulimia nervosa; BED=binge eating disorder

Fig. 1. Flow chart showing participants screened, eligible, randomized and treated with cognitive-behavioural therapy (CBT), schema therapy (ST), and appetite-focused CBT (CBT-A).

A 6.3 (0.9), $F(2,110)=0.52$, $p=0.59$), or successful (CBT 5.4 (1.1), schema therapy 5.3 (1.2), CBT-A 4.8 (1.2), $F(2,110)=2.76$, $p=0.07$) therapy was expected to be, nor whether participants would recommend the treatment to someone else (CBT 5.9 (1.3), schema therapy 5.6 (1.5), CBT-A 5.7 (1.2), $F(2,110)=0.70$, $p=0.50$).

Primary, secondary and tertiary outcome variables at pre-treatment, end of treatment and 12 month follow-up are shown in Table 2, with the last observation carried forward when end of treatment data were not available. Across the three combined treatment groups, a large effect was found for the change from pre-treatment to end of treatment for the primary outcome variable, frequency of binge eating ($d=1.06$). Large effects were also found for most secondary outcome variables, including each of the EDE-12 subscales, the EDI-2 bulimia subscale, and the GAF; a medium effect for EDI-2 drive for thinness; and small effects for the EDI-2 body dissatisfaction subscale, and the frequency of purging variable. Purging values were calculated for those individuals who reported purging behaviours at one or more time point – baseline, end of treatment, or 12 month follow-up. Fig. 2 shows the percentage of the total sample and of the three treatment groups within one standard deviation of the community mean for the EDE-12 global, considered an approximation of the

population norm, and indicative of clinically significant change, which was not significantly different among the three groups (End of treatment: Total sample 60%, CBT 58%, schema therapy 60%, CBT-A 61%, $\chi^2(2)=0.91$, $p=0.95$; 12 month follow-up: Total sample 64%, CBT 67%, schema therapy 59%, CBT-A 68%, $\chi^2(2)=0.63$, $p=0.73$). Fig. 3 shows the percentage of the total sample and the three treatment groups abstinent over the course of treatment and 12 month follow-up.

No difference was found among the three therapy groups in change from baseline to end of treatment for the primary outcome variable, binge frequency ($F(2,110)=0.20$; $p=0.82$, $d=0.14$, 95% CI –6.63 to 3.55). No differences were found among the groups in any secondary or tertiary outcome measures (Table 3). Due to the smaller than planned sample size, in order to consider whether insufficient statistical power could account for the absence of differences among the three groups, effect sizes for between group one-way ANCOVAs were calculated for change from baseline to end of treatment values for all primary, secondary and tertiary outcome measures. Small between group effect sizes were found for the primary outcome measure, and for the secondary outcome measures of eating disorder attitudes and behaviours. Medium effect sizes were found for weight and BMI, and for social

Table 1
Means and standard deviations, or numbers and percentages, and statistics for baseline characteristics, demographics, and psychiatric diagnoses for the total sample and for the three therapy groups.

Demographics	Total sample <i>n</i> =112		CBT <i>n</i> =38		ST <i>n</i> =38		CBT-A <i>n</i> =36		<i>f</i> / χ^2	<i>p</i>
	<i>x</i> / <i>n</i>	<i>s.d</i> / <i>%</i>	<i>x</i> / <i>n</i>	<i>s.d</i> / <i>%</i>	<i>x</i> / <i>n</i>	<i>s.d</i> / <i>%</i>	<i>x</i> / <i>n</i>	<i>s.d</i> / <i>%</i>		
Age	35.3	12.6	34.4	13.0	37.1	12.9	34.3	11.9	0.57	0.56
Never married	44	39%	17	45%	15	39%	12	33%	1.01	0.60
Education (years)	15.4	2.9	15.7	2.8	15.1	2.5	15.3	3.4	0.38	0.68
Unemployed	12	11%	1	3%	6	16%	5	14%	4.00	0.14
Weight (kg s)	83.2	22.4	83.0	22.4	82.0	21.5	84.7	23.8	0.14	0.87
BMI	29.9	7.8	30.0	7.9	30.0	7.9	29.8	7.8	0.01	0.99
Lowest weight	61.0	11.9	62.4	14.0	58.9	10.6	61.8	10.9	0.95	0.39
Highest weight	91.5	25.1	89.7	24.3	88.7	21.4	96.9	29.3	1.03	0.36
Length of ED (years)	15.2	12.7	14.6	13.2	15.7	11.4	15.4	13.9	0.07	0.93
Psychiatric history										
Bulimia nervosa										
Lifetime	71	63%	24	63%	25	66%	22	61%	0.18	0.92
Past month	58	52%	20	52%	20	53%	19	50%	0.07	0.97
Binge eating disorder										
Lifetime	68	61%	24	63%	22	60%	22	61%	0.22	0.89
Past month	54	48%	18	47%	18	47%	18	50%	0.07	0.97
Anorexia nervosa										
Lifetime	8	7%	4	10%	2	5%	2	5%	0.99	0.61
Major Depression										
Lifetime	74	66%	21	55%	26	68%	27	75%	3.35	0.19
Past month	30	26%	12	31%	10	26%	8	22%	0.83	0.66
Bipolar II disorder										
Lifetime	4	4%	2	5%	2	5%	0	0	1.96	0.37
Past month	0	0	1	3%	0	0	0	0	1.96	0.37
OCD lifetime										
Lifetime	9	8%	3	8%	2	5%	4	11%	0.86	0.65
Past month	7	6%	3	8%	0	0	4	11%	4.16	0.12
PTSD										
Lifetime	21	19%	9	24%	5	13%	7	19%	1.40	0.50
Past month	10	9%	4	11%	2	5%	4	11%	0.96	0.62
GAD										
Past month	23	21%	8	21%	5	13%	10	28%	2.43	0.30
Social phobia										
Lifetime	30	27%	10	26%	13	34%	7	19%	2.06	0.36
Past month	26	23%	8	21%	12	32%	6	17%	2.46	0.29
Alcohol use disorder										
Lifetime	38	34%	10	26%	13	34%	15	42%	1.94	0.38
Past month	5	4%	1	3%	1	3%	3	8%	1.86	0.39
Substance disorder										
Lifetime	25	22%	12	32%	5	13%	8	22%	3.72	0.16
Past month	3	3%	1	3%	0	0	2	6%	2.19	0.33
Suicide attempt (Y/N)	20	18%	7	18%	5	13%	8	22%	1.05	0.59
Deliberate self-harm (Y/N)	33	29%	10	26%	9	24%	14	39%	2.33	0.31

CBT=cognitive-behavioural therapy; ST=schema therapy; CBT-A=appetite-focused CBT; ED=Eating disorder; BMI=body mass index (kg/m²); OCD=obsessive compulsive disorder; PTSD=posttraumatic stress disorder; GAD=generalized anxiety disorder.

adjustment. Taken together, these results suggest sample size is unlikely to account for the absence of differences among therapies.

Secondary analysis of covariance for those 79 individuals who completed therapy showed the same pattern of results, with no differences among the three therapy groups for any outcome measures.

3.1. Eating disorder diagnosis and treatment response

Study hypotheses did not include predictions of outcome relative to diagnosis; however, subgroup analyses are included here due to previous research showing differential treatment outcome for BN and BED. Participants with BN and BED entered the study with similar psychopathology, as described previously (Jordan et al., 2014). The percentage within one standard deviation of the community mean for the EDE-12 global, considered an approximation of the population norm, was not significantly different at baseline (BN 1.7%; BED 7.4%; $\chi^2(1)=2.12$, $p=0.15$), nor was the frequency of binge eating episodes (BN 20.76 (12.87); BED 17.70

(11.08); $t(1)=1.80$, $p=0.18$). The proportion of BN vs. BED in each therapy group did not differ (CBT BN=51.8% BED=48.2%; schema therapy BN=52.6% BED=47.4%; CBT-A BN=50.0% BED=50%; $\chi^2(2)=0.07$, $p=0.97$). Change in the frequency of binge eating at the end of treatment was greater for the BED group than the BN group (mean reduction of 14.80 (SD=10.09) and 11.39 (SD=14.03), $t(1)=5.0$, $p < 0.05$) binges per month from baseline to end of treatment, for BED and BN, respectively, covarying for baseline frequency of binge eating. At 12 month follow-up no difference was found between the groups in the change in binge frequency (BN=13.42 (SD=13.99); BED=15.14 (SD=16.47); $t(1)=0.53$, $p=0.47$). At the end of treatment, and at 12 month follow-up no differences were found in the percentage of the two groups within one standard deviation of the community mean for the EDE-12 global (end of treatment: BN=53.4%; BED=64.9%; $\chi^2(1)=1.49$, $p=0.22$; 12 month follow-up: BN=54.5%; BED=64.1%; $\chi^2(1)=0.78$, $p=0.38$).

Table 2

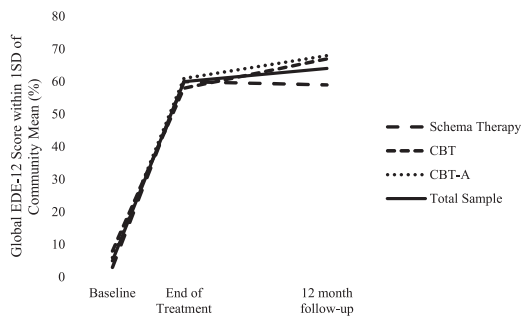
Means and standard deviations for primary, secondary and tertiary outcome measures at pre-treatment, end of treatment, and 12 month follow-up for the three therapy groups.

Outcome measure	Pre-treatment						End of treatment ^a						Effect size (pre- to end-treatment)	12 month follow-up						Effect size (pre- to 12 mth f-up)
	CBT n=38		ST n=38		CBT-A n=36		CBT n=38		ST n=38		CBT-A n=36			CBT n=30		ST n=29		CBT-A n=28		
	x	sd	x	sd	x	sd	x	sd	x	sd	x	sd		x	sd	x	sd	x	sd	
Primary outcome																				
Binge frequency (28 days)	19.6	10.7	18.6	13.0	19.6	12.7	7.3	13.9	5.0	13.2	6.5	13.0	1.06	7.3	18.9	4.3	10.4	3.6	9.5	0.94
Secondary outcomes																				
Purge frequency (28 days) ^b	17.0	18.8	21.0	26.7	12.6	18.8	10.1	14.4	7.9	20.0	13.2	29.2	0.32	9.1	12.8	7.1	15.7	14.6	26.3	0.60
EDE-12 Restraint	3.0	1.3	3.0	1.4	2.8	1.7	1.2	1.5	1.4	1.5	1.5	1.9	0.99	1.1	1.4	1.3	1.2	1.1	1.6	0.99
EDE-12 Eating Concerns	3.2	1.2	3.2	1.1	3.1	1.3	1.2	1.4	1.2	1.3	1.6	1.9	1.22	0.7	1.1	0.8	0.9	0.6	0.9	1.99
EDE-12 wt Concerns	3.6	1.3	3.8	1.3	3.7	1.3	2.0	1.5	2.4	1.5	2.1	1.7	1.03	1.7	1.6	2.1	1.3	1.8	1.3	1.29
EDE-12 Shape Concerns	4.0	1.4	4.3	1.2	4.2	1.2	2.4	1.7	2.8	1.5	2.5	1.8	1.06	1.6	1.5	2.3	1.5	2.0	1.4	1.46
EDE-12 Global	3.5	1.0	3.6	1.0	3.5	1.1	1.7	1.3	2.0	1.3	1.9	1.7	1.29	1.3	1.2	1.6	1.0	1.4	1.2	1.80
EDI-2 Drive for Thinness	12.2	5.4	13.3	5.2	11.8	5.8	8.6	6.2	9.9	6.1	8.1	6.5	0.52	5.9	5.6	7.7	6.4	5.5	6.2	0.91
EDI-2 Body Dissatisfaction	19.6	7.7	19.8	7.9	18.8	7.9	15.2	9.4	18.3	8.1	15.4	9.3	0.35	13.4	9.9	17.0	9.0	14.8	7.9	0.49
EDI-2 Bulimia	10.4	3.6	10.4	4.6	9.6	4.5	3.9	5.4	3.7	5.2	2.9	4.3	1.23	2.1	2.9	2.8	4.4	1.4	3.1	1.58
Weight	83.0	22.4	81.5	21.6	84.7	23.8	84.4	24.2	83.6	22.8	83.7	22.3	0.12	83.9	21.9	83.7	21.3	86.4	22.4	0.05
BMI	30.0	7.9	30.0	7.9	29.8	7.8	30.5	8.6	30.5	8.2	29.5	7.3	0.13	30.3	8.3	30.4	7.7	30.4	7.6	0.00
Social Adjustment Scale	2.3	0.4	2.2	0.4	2.2	0.4	2.0	0.4	2.0	0.5	2.1	0.5	0.68	1.8	0.5	1.9	0.4	1.9	0.4	1.03
GAF	54.7	5.0	55.7	4.2	54.8	4.8	67.2	14.2	71.2	12.5	69.9	16.3	1.06	72.3	16.4	71.5	14.8	70.5	16.1	1.10
Tertiary outcome																				
SCL-90-R GSI	1.1	0.6	1.0	0.6	1.0	0.5	0.6	0.6	0.6	0.6	0.7	0.7	0.78	0.4	0.5	0.6	0.5	0.6	0.6	0.89
SCL-90-R Depression	1.5	0.9	1.4	0.8	1.4	0.8	0.9	0.9	0.8	0.9	1.0	1.0	0.62	0.6	0.6	0.8	0.7	0.8	0.7	0.85

CBT=cognitive-behavioural therapy; ST=schema therapy; CBT-A=appetite-focused CBT; EDE-12=Eating Disorder Examination; EDI-2=Eating Disorder Inventory-2; BMI=body mass index (kg/m²); GAF=Global Assessment of Functioning; SCL-90-R=Symptom Checklist-90-Revised; GSI=Global Severity Index.

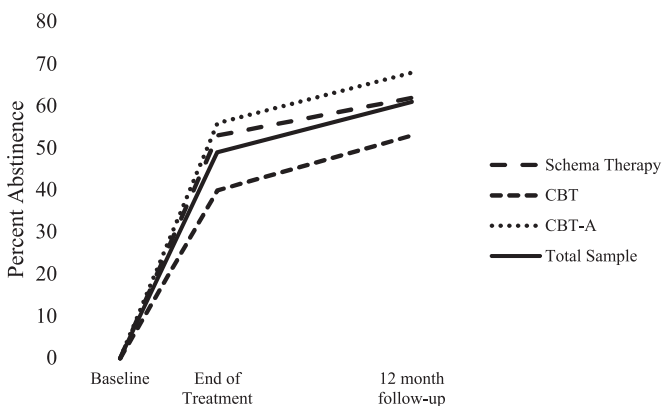
^a Last observation carried forward where no end of treatment value available.

^b Analyses excluded individuals with no purging at any of baseline, end of treatment, or 12 month follow-up.



CBT=cognitive-behavioural therapy; CBT-A=appetite-focused CBT; EDE-12=Eating Disorder Examination

Fig. 2. Percentage of each therapy group and the total sample with Global EDE-12 scores within one standard deviation of community mean, showing the effects of cognitive behavioural therapy (CBT), schema therapy, and appetite-focused CBT (CBT-A) over treatment and 12 month follow-up.



CBT=cognitive-behavioural therapy; CBT-A=appetite-focused CBT

Fig. 3. Percent abstinence from binge eating over treatment and 12-month follow-up for the total sample, schema therapy, cognitive-behavioural therapy (CBT), and appetite focused CBT (CBT-A).

4. Discussion

The current study found the augmented focus on appetite and on schemas within a cognitive behavioural approach to binge eating did not result in improved outcomes compared with CBT at the end of treatment or at 12 month follow-up. At the end of treatment 49% of the total sample was abstinent from binge eating, and 47% of the sample was abstinent from binge eating and purging and met no eating disorder diagnoses in the past month. Overall, 60% scored within one standard deviation of the community mean for adult females on the EDE-12 global score, an indicator of meeting community levels of eating disorder attitudes and behaviours, and indicating clinically significant change (Jacobson and Truax, 1991). This pattern was maintained over the following twelve months, with 64% of the total sample within one standard deviation of community norms. These results are consistent with or superior to outcome of CBT in other studies of binge eating, with both traditional CBT (Fairburn et al., 1993) and enhanced CBT (CBT-E) (Agras et al., 2000; Fairburn et al., 2015; Fairburn et al., 2009).

Although no RCT to date has directly compared the outcome of CBT and CBT-E, the percentage of patients within one standard deviation of EDE global community norms after CBT or CBT-E treatment can be examined across existing studies. The percentage varies from 41% for traditional CBT (Agras et al., 2000), 52% and 54% for focused and broad CBT-E, respectively (Fairburn et al., 2009), 58%, 60% and 61% for traditional CBT, schema therapy and CBT-A in the current study, and 65% for CBT-E (Fairburn et al., 2015) at the end of treatment. In follow-up studies from these

RCTs, 44–56% was found for CBT-E, focused and broad across 20–60 weeks (Fairburn et al., 2009), 59% for schema therapy, 67% for traditional CBT, and 68% for CBT-A at 12 month follow-up in the current study, and 69% for CBT-E at 60 weeks (Fairburn et al., 2015).

Across diagnostic categories, change in binge frequency was greater for BED than BN from baseline to end of treatment in the current study, but was comparable for the two diagnoses at 12 month follow-up. The proportion of BN and BED who met community norms on the EDE global score did not differ at the end of treatment or at 12 month follow-up. The three therapies did not differ in rates of treatment completion, or in the perceived suitability of treatment. Study results are consistent with other well-conducted RCTs, which show comparable outcome among two or more active treatments (Bulik et al., 1998; Carter et al., 2013; Wampold et al., 2002). Of note, outcome of both schema therapy and CBT-A in the current study are markedly superior to outcome of IPT in existing RCTs (Agras et al., 2000; Fairburn et al., 2015), although IPT is currently considered a leading alternative treatment to CBT (Fairburn et al., 2015).

A number of factors may limit generalisability of these findings. The study was conducted in a clinical research unit, and considerable effort was made to limit exclusion criteria (only 6% of those excluded were due to comorbidity or other exclusion criteria), however, participants may not be representative of the transdiagnostic binge eating population in general. The length of treatment (six months of weekly, and six months of monthly sessions) was longer than is usual for CBT, and shorter than is usual for schema therapy, and was standardized across therapies to ensure that dose was not confounded with therapy. Optimal dose of therapy has not been determined for transdiagnostic binge eating or the eating disorders in general. Although 19–20 sessions of CBT has been adopted as standard, an earlier dismantling study of CBT for bulimia nervosa found good outcome with eight sessions of a non-behavioural cognitive form of CBT (Bulik et al., 1998). Further research is warranted to ascertain the required dose of CBT. Ethnicity of the sample is somewhat different than in the New Zealand population (Statistics New Zealand, 2014), with fewer participants identifying as Maori (10% compared with 15% in the 2013 census) and Asian (4% compared with 12% in the population), and no Pacific peoples (compared with 7% in the population), and differs from ethnicity of other populations, which may limit generalisability. The smaller than planned sample size could limit the study's power to detect group differences. However calculation of effect sizes for change scores across time indicated large effects for primary and most secondary outcome measures for the total sample, indicating the effectiveness of all three treatments, although the study did not include a no-treatment control group.

Strengths of the study are the inclusion of two innovative and potentially effective approaches for binge eating, use of standardized assessment instruments, very good retention of participants in the trial over the weekly phase of therapy (83% retained in the first six months of weekly therapy sessions), and good retention over the full twelve months of treatment (over 70% retained for the total sample). All therapists conducted the three therapies, reducing the possible confounding effect of therapists with therapies.

Although study hypotheses that focused on improving outcome above that achieved with CBT were not supported, the present findings that the three therapy approaches do not differ in outcome in treating transdiagnostic binge eating are promising and potentially important. Schema therapy is conceptually and practically different from CBT, and with no difference in outcome compared with traditional CBT found in the current study, has the potential to be a good alternative to CBT that may be preferred by

Table 3

Change score means and standard errors from analyses of covariance with baseline values as the covariate, or frequency and percentages and Chi square statistic for all outcome measures at end of treatment, and 12 month follow-up for the three therapy groups.

Outcome measure	End of treatment ^a						12 month follow-up												
	CBT n=38		ST n=38		CBT-A n=36		Effect size ^b			CBT n=30		ST n=29		CBT-A n=28		f/χ ² p			
	\bar{x}/n	s.d./%	\bar{x}/n	s.d./%	\bar{x}/n	s.d./%				\bar{x}/n	s.d./%	\bar{x}/n	s.d./%	\bar{x}/n	s.d./%				
Primary outcome																			
Binge frequency (28 days) ^c	12.2	1.8	13.9	1.8	13.0	1.9	0.14	0.20	0.82	12.6	2.4	14.8	2.5	15.4	2.5	0.3	0.71		
Secondary outcomes																			
Abstinence from bingeing	15	39.5	20	52.6	20	55.6	0.24	2.20	0.33	16	53.3	18	62.1	19	67.9	1.3	0.52		
Purge frequency (28 days) ^c	3.3	12.2	6.5	11.9	-0.5	13.3	0.33	2.17	0.12	4.1	1.6	5.3	1.6	2.3	1.6	0.8	0.43		
Remission (no binge, purge, ED)	13	34.2	20	52.6	20	55.6	0.32	4.03	0.13	16	53.3	17	58.6	19	67.9	1.3	0.52		
Global ED status ^c	1.3	0.2	1.8	0.2	1.7	0.2	0.34	1.27	0.28	1.9	0.2	2.1	0.2	2.3	0.2	0.95	0.39		
EDE Restraint ^c	1.8	0.2	1.5	0.2	1.3	0.2	0.35	1.01	0.97	1.8	0.2	1.6	0.2	1.8	0.2	0.35	0.71		
EDE Eating Concerns ^c	2.0	0.2	2.0	0.2	1.6	0.2	0.31	1.19	0.31	2.5	0.2	2.5	0.2	2.6	0.2	0.07	0.94		
EDE Weight Concerns ^c	1.6	0.2	1.3	0.2	1.6	0.2	0.20	0.46	0.63	2.0	0.2	1.7	0.2	2.0	0.2	0.40	0.67		
EDE Shape Concerns ^c	1.7	0.2	1.5	0.2	1.7	0.2	0.19	0.35	0.71	2.6	0.3	2.0	0.3	2.2	0.3	0.69	0.34		
EDE Global ^c	1.7	0.2	1.6	0.2	1.6	0.2	0.20	0.08	0.93	2.2	0.2	1.9	0.2	2.1	0.2	0.40	0.67		
EDI Drive for Thinness ^c	3.7	1.0	2.8	1.0	4.1	1.0	0.15	0.43	0.65	6.8	1.1	5.1	1.2	7.1	1.2	0.8	0.45		
EDI Body Dissatisfaction ^c	4.3	1.3	1.3	1.3	3.7	1.3	0.38	1.5	0.23	6.5	1.6	3.1	1.8	4.8	1.8	1.0	0.37		
EDI Bulimia ^c	6.4	0.8	6.6	0.8	7.0	0.8	0.14	0.18	0.83	7.9	0.7	7.2	0.7	8.5	0.7	0.8	0.44		
Weight ^c	-1.4	0.9	-1.5	0.9	1.0	0.9	0.43	2.53	0.08	0.79	1.4	0.80	1.4	-0.056	1.4	0.31	0.73		
BMI ^c	0.55	0.3	0.51	0.3	-0.34	0.3	0.46	2.41	0.09	-0.19	0.5	-0.22	0.5	0.42	0.5	0.49	0.62		
Social Adjustment ^c	0.31	0.0	0.23	0.0	0.16	0.0	0.45	1.76	0.18	0.47	0.1	0.33	0.1	0.29	0.1	1.9	0.16		
GAF ^c	12.5	2.2	15.5	2.2	15.1	2.3	0.22	0.53	0.59	18.2	2.8	15.7	2.8	15.7	2.9	0.2	0.78		
Tertiary outcome																			
SCL-90-R GSI ^c	0.49	0.1	0.39	0.1	0.33	0.1	0.33	1.01	0.37	0.64	0.1	0.42	0.1	0.43	0.1	1.8	0.17		
SCL-90-R Depression ^c	0.57	0.1	0.59	0.1	0.47	0.1	0.12	0.23	0.79	0.91	0.1	0.60	0.1	0.61	0.1	1.8	0.17		

ED=Eating disorder; EDE=Eating Disorder Examination; EDI=Eating Disorder Inventory; BMI=body mass index (kg/m²); GAF=Global Assessment of Functioning; SCL-90-R=Symptom Checklist Revised; GSI=Global Severity Index.

^a Last observation carried forward where no end of treatment value available.

^b Calculated using the difference between means for CBT and either schema therapy or CBT-A, whichever difference was larger.

^c Values represent change from baseline scores.

some individuals with an eating disorder. Schema therapy focuses on early childhood experiences, unmet psychological needs, and the development of maladaptive schemas, which in turn are theorized to lead to the onset and maintenance of disordered eating. The experiential treatment strategies are appealing to some patients and some therapists. For some patients, the inclusion of early life factors in the treatment rationale is consistent with their conceptualization of their illness. Similarly, some practitioners may find this treatment strategy appealing, such as those trained in therapeutic orientations that prioritize the importance of childhood experiences yet who wish to use evidence-based strategies for treating eating disorders.

Appetite-focused CBT's rationale of increasing awareness of, and responsiveness to internal cues for eating may also appeal to many individuals who binge eat, and to many therapists, and may be preferred over traditional food monitoring and prescription of meals and snacks.

There is some evidence that patient expectations or the "fit" of the therapy model can influence treatment outcome (Carter et al., 2011; Joyce and Piper, 1998). This has not been studied in the eating disorders, but there have been calls for tailored or stepped treatment (Kaplan et al., 2001; Kordy et al., 2006; Tasca et al., 2013). Offering patients a choice of known effective treatments may improve treatment outcome for psychological problems, by way of more effective engagement in therapy sessions, lower dropout, and increased motivation (Meyer et al., 2002; Sotsky et al., 1991; Treasure et al., 1999). Patients are more likely to drop out from treatment when there is lack of congruence between patients' and therapists' expectations of potential treatment interventions. In a large study of treatments for depression, treatment expectancy predicted response across both psychotherapy

and pharmacotherapy conditions (Sotsky et al., 1991). In further analyses of these data, it was found that patients who expected treatment to be effective tended to engage more constructively in therapy sessions, which helped bring about greater symptom change (Meyer et al., 2002). Future research might examine the efficacy of different eating disorder treatments when matching patients to their preferred treatment.

This is the first randomized controlled trial comparing CBT with schema therapy, and with appetite-focused CBT, and therefore requires replication. The trial was not designed as an equivalency trial; however, results suggest schema therapy and appetite-focused CBT may be potential alternative treatments with comparable outcome that may be offered alongside traditional CBT for binge eating. Further research is warranted with these therapy approaches.

Conflict of interest

Authors have no conflict of interest to declare.

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Ethical standards

The authors assert that all procedures contributing to this work comply with the ethical standards of the relevant national and institutional committees on human experimentation and with the Helsinki Declaration of 1975, as revised in 2008.

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