OUTPATIENT COGNITIVE BEHAVIOUR THERAPY FOR ANOREXIA NERVOSA: WHICH PATIENTS SHOW EARLY WEIGHT GAIN?

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Overview

There are three parts to this thesis. The first section is the literature review, which examines the main cognitive behavioural models for anorexia nervosa and which reviews research trials using this treatment (in comparison to other treatments and control groups). Numerous weaknesses are identified in those studies, and their limitations are considered when treating anorexia nervosa. Future work on this topic is suggested.

The second section is a research project. This project examines the cognitive and emotional factors present at the start of CBT for anorexia nervosa that predict a key treatment CBT target in anorexia nervosa – change in weight over the first ten sessions of treatment. The study also examines predictors of behaviour change across this period of therapy. Finally, it considers factors that predict changes in dysfunctional assumptions in this group.

The final section is a critical appraisal of the research process. Following from the discussion of the literature review and empirical paper, it considers in more detail methodological and conceptual issues of conducting this work, and considers the cognitive behavioural model in more detail. The clinical implications of the work are also considered in more detail, and a personal reflection on the whole process is given.
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PART 1: LITERATURE REVIEW

COGNITIVE BEHAVIOUR THERAPY FOR ANOREXIA NERVOSA: A REVIEW OF THE EVIDENCE
Part 1 literature Review: CBT for Anorexia Nervosa: A review of the evidence

1. Abstract

This review initially discusses the most prominent models of Cognitive Behaviour Therapy (CBT) for the eating disorders currently and considers the aims of the various treatments. The paper then reviews research trials examining the efficacy of Behaviour Therapy (BT) or CBT in comparison to other treatment types or control groups. There are numerous weaknesses identified across the trials, and few conclusions can be drawn because of this and the lack of consistent results. The NICE (National Institute for Clinical Excellence) guidelines for the treatment of Bulimia Nervosa (BN) and binge eating disorder in contrast consistently point to CBT. Within Anorexia Nervosa (AN) there is some suggestion for using CBT in those who are weight recovered. Dropout was highlighted as a recurrent problem across trials and this was considered further within the AN population. The clinical limitations of the research in the area of treatment trials and dropout were considered, and future directions suggested.

2. Introduction

A number of recent reviews (Bulik et al., 2007; Cochrane Review, Hay et al., 2007) have highlighted the poor standards of research trials across the eating disorders, but they talk very broadly of difficulties and do not focus on the specific details of studies. There is also a lack of the exploration of the CBT theory and models utilised in these studies in the treatment of AN. Dropout within the AN population in terms of its impact on treatment trials has not specifically been examined in previous literature. The current paper will address in more details some of the highlighted
weaknesses of research, and will also address some of the unaddressed areas identified. The paper is divided into sections covering CBT models; treatment trials which include CBT or BT; the importance of dropout; and finally limitations and future research suggestions.

3. Questions to be addressed
   - What CBT models of treatment are there?
   - What conclusions can be drawn from research studies examining treatment of AN utilising CBT and BT?
   - What are the limitations of the research trials?
   - What influence does drop out have on treatment trials and what conclusions can be drawn?
   - Limitations and future directions

4. Search strategy

Psychinfo, Pubmed, Web of Science and The Cochrane Library were searched using the terms ‘anorexia nervosa’, ‘treatment’, ‘CBT’, ‘weight gain’, ‘outcome predictors’, ‘therapy’, ‘drop-out’, and ‘attrition’. There was no publication date time limitation on the articles selected. Further publications were identified through the references sections of the original sourced articles. Within 2007, two review papers (Bulik et al., 2007 and Cochrane Review, Hay et al., 2007) that utilised sound research methods were used in the current study as guidance on the treatment trial papers that should be evaluated within the research. Nine papers were identified and five of these were identified as meeting the criteria of the literature review. It was not seen as productive to re-review papers that had already been deemed
methodologically flawed by two large research papers, or were inappropriate given the specification of the current literature review, hence the following papers were omitted: Ball & Mitchell, 2004 (adolescent population), Brambilla, 1995 (drug treatment included as comparison group); Eckert, 1979 (behaviour modification different from BT and only 5 sessions); Hall & Crisp, 1987 (adolescent population).

5. Background

Although AN is a relatively rare psychiatric disorder (Agras et al., 2004), because it is associated with high medical and psychiatric risk, proportionally the condition takes a lot of time to manage, involves substantial costs in care and demands on resources. There are no firm conclusions from research trials examining the efficacy of CBT for AN, suggesting that what is needed is a more close exploration of a smaller number of higher quality research trials examining the specific area of CBT for AN.

6. Models

6.1 The development of cognitive behavioural models of disorders

Cognitive behavioural models provide a means for understanding the development and maintenance of the most important cognitive and behavioural aspects of many psychological disorders. Such models also lead to strategies for treatment.

Beck's (1976) cognitive model of emotional disorders was originally developed to account for depression, but it provides a general framework for understanding the development and maintenance of a number of emotional disorders. Beck (1976) gives a central role to cognitions, proposing that one's emotions are influenced by
one’s thoughts (and vice versa), and that emotional disorders result from particular interpretations of events. Consequently, changing a thought that arises in a situation is hypothesised to directly change mood (and also behaviour). Beck (1976) proposes that different types of thinking (negative automatic thoughts, dysfunctional assumptions, and core beliefs/schemas) are characterised by cognitive distortions and during therapy these can be identified and corrected.

Recent models integrate cognitive and behavioural elements, (e.g., Fairburn et al., 2003) as well as addressing specific disorders (e.g. Clark’s model of panic – Clark, 1988). In the treatment approaches based on cognitive behavioural models, behavioural experiments are commonly used to gather evidence to examine the validity of thoughts and assumptions (Bennett-Levy et al., 2004; Greenberg & Padesky, 1995) in addition to cognitive techniques such as cognitive restructuring and the use of thought records. With time more deeply rooted beliefs about the self, world and others (known as schema or core beliefs) may also be challenged within therapy (Young, 1993).

6.2 Cognitive Behaviour Therapy (CBT) models of Bulimia Nervosa (BN)

CBT for BN is based on a cognitive model of what maintains the disorder (Fairburn, 1981; Fairburn et al, 1993). Control in eating is lost in response to efforts at extreme dieting and consequently a chaotic eating pattern develops involving binge eating and compensatory purging. Treatment begins with reducing dietary restraint and introducing a regular eating pattern. Later stages involve developing and utilising cognitive and behavioural skills for coping with situations that are likely to trigger binge eating and purging, and challenging dysfunctional thoughts and feelings about
body, weight and shape (Fairburn et al., 1993). CBT for BN works in a number of ways. Initially, once a regular eating pattern is adopted there is less likelihood of bingeing and hence consequential purging because bingeing is reinforced by both restriction and by purging. Secondly self-efficacy for coping with situations that trigger binge eating and purging is increased, which is likely to cause a further shift in bingeing and purging behaviour. The third aspect is the modification of dysfunctional attitudes about body weight and shape, in order to decrease the likelihood of dieting.

CBT has been a well-established treatment for BN since the 1980s. Studies reveal that it is as effective as, or superior to antidepressant drugs and more effective than alternative psychological treatments (Wilson & Fairburn, 1998). Meta- (e.g. Shapiro et al, 2007) also support the efficacy of CBT in the treatment of BN in analysis comparison to no treatment controls and alternative active treatments.

In terms of time course, Wilson et al. (2002) and Agras et al. (2000) both found that CBT had more rapid effects than interpersonal therapy (IPT, a comparison therapy which addresses interpersonal difficulties but does not focus on weight or eating). However, by the time patients were followed up, differences between the two treatments were no longer significant. Wilson (1999) found that, for CBT, 76% of total improvement in the frequency of binge eating and 69% of total improvement in the frequency of vomiting were evident by session 5. It appears that the rapid action of CBT differs from IPT in which a more gradual dose-response relationship is described. What mediates this early response to CBT is important to determine. It is possible that the actions of behavioural homework assignments are a reason for the
rapid effect of CBT. Homework assignments may validate the specific CBT rationale and also enhance self-efficacy by enabling a sense of control over behaviour previously experienced as out of control. Self-monitoring may also work in a similar vein, as it is likely to prompt an increased awareness of the connection between the problem behaviour and factors that influence the behaviour.

The superiority of CBT over other treatments for BN, in terms of both behavioural change and speed of change, seems to be relatively well established. However, the picture is less promising for AN. The development of CBT models for AN and the resultant treatment approaches will be considered in the following sections.

6.3 Early CBT models of Anorexia Nervosa (AN)

Initially Garner & Bemis (1982) applied the principles of Beck et al’s. (1979) cognitive theory and therapy of depression to the treatment of AN. In an extension of Bruch’s (1973) approach, they emphasized the importance of the patient’s thinking style and their individual interpretation of the meaning of events, as well as the role of self-esteem. Vitousek advanced this approach, adding the theme of ‘over evaluation of body shape and weight’ and the role of ‘schema’ in core cognitive disturbance (Vitousek, 1996). Slade (1982) suggested that a ‘need for control’ is central to the development and maintenance of AN. He proposed that general dissatisfaction with oneself and life develops in response to a combination of interpersonal problems and adolescent conflict. This dissatisfaction is proposed to interact with premorbid perfectionism, so that a need to control and achieve success in particular aspects of life becomes focused around weight loss. On an individual basis, the models discussed here all contain components of models used currently.
However, on their own, previous models suffer a lack of specificity, which means that it is difficult to derive a treatment approach from them. In recent years, Garner, Vitousek & Pike (1997) have extended the models discussed by creating a treatment protocol involving combined elements from the models.

A new CBT based theory of AN was developed by Fairburn et al. (1999). Following on from Slade’s (1982) work, this theory maintains that the principal focus of treatment should be on the issue of self-control. Within this context, issues addressed include the use of eating, shape and weight as indices of self-control and self-worth, disturbed eating and the associated extreme weight-control behaviour, body checking and low body weight. Fairburn et al. (1999) suggest that cognitive therapy should focus on the excessive need for control over weight and shape. Part of the treatment also involves focusing on the patient’s need for self-control in general, with the focus being shifted away from eating, by helping patients derive satisfaction and a sense of achievement from other activities, and by demonstrating that control over eating does not provide satisfaction. As always, the aim is to encourage patients to change their behaviour. Behavioural therapy applies behavioural strategies such as keeping a diary of eating patterns, exposure to “normal” eating and techniques to help distract from weight control behaviours such as vomiting. Despite this appealing and clearly specified model, there are as yet no published trials using this particular CBT model for AN.

6.4 A transdiagnostic model of all eating disorders

Perhaps in response to difficulties establishing the efficacy of CBT for AN, but also in reflection of a growing view that the eating disorders have more commonalities
than differences, a new model was developed by Fairburn et al. (2003). The theory, developed in the light of increasing evidence, suggests considerable overlap between the different eating disorders, with fluid movement between diagnoses (Sullivan et al., 1998). It also reflects suggestions that the largest diagnostic group in the eating disorders is ‘Eating Disorder Not Otherwise Specified’ (EDNOS), rather than AN or BN (Fairburn & Walsh, 2002; Turner & Bryant-Waugh, 2004) - a diagnosis that had previously received scant attention in the literature.

The model is based on Fairburn’s original model of CBT for BN (Fairburn & Wilson, 1993) but proposes that one or more of four additional maintaining processes interact with the core eating disorder, presenting obstacles to change. The additional four maintaining mechanisms involve: the influence of severe perfectionism (clinical perfectionism); the impact of unconditional and pervasive low self-esteem (core low self esteem); difficulty coping with intense mood states (mood intolerance); and interpersonal difficulties. Treatment should be based on an individualised formulation which focuses on addressing any of the 4 mechanisms which are relevant, as well as the core eating disorder symptoms.

6.5 A ‘Culture free’ Cognitive Interpersonal Maintenance Model of AN

In recent times Schmidt & Treasure (2006) have developed a model that departs from other models of AN, in that it does not give a central role to weight and shape related factors in the maintenance of AN. The model proposes that anorectic symptoms are maintained intrapersonally by beliefs about the positive function of the illness for the person (Serpell et al., 1999; Serpell et al., 2004) and interpersonally by both positive and negative responses elicited from close others (in response to the physical
presentation and behaviours associated with AN). The disorder develops in individuals who are vulnerable because of avoidant (Gilberg et al., 1995) and/or obsessive-compulsive personality traits (Brecelj-Anderluh et al., 2003; Serpell et al., 2004) and unknown biological factors. The avoidance centres mainly on a need to avoid the experience and expression of intense negative emotions and the need to avoid close relationships (which trigger these emotions). The obsessive-compulsive traits are manifested in perfectionism and rigidity, which make these individuals prone to rigid thinking styles, meticulous attention to details and a fear of making mistakes (Bulik et al., 1995; 2003, Tchanturia et al, 2004). These vulnerability traits are intensified further by the state of starvation (Keys et al., 1950).

6.6 Wallers’ model of eating disorders

Waller et al’s (2007) propose a new schema-based cognitive-behavioural model of eating disorders. The treatment protocol takes elements from all the existing models incorporating a package that includes: engagement of the patient and where appropriate, family and carers; assessment; explanation of treatment and boundaries; comprehensive formulation; planning of treatment with the patient; motivational enhancement; psychoeducation; introduction of structure to dietary intake; addressing of central targets using CBT techniques; weight gain/stabilisation; working with comorbidity and other problems; what to do when CBT is not working; relapse prevention; endings.

The model is predicated on the assumption that important differences between bulimic and restrictive psychopathology exist at the level of schema processing. Proposed are two levels of avoidance; primary avoidance of affect (strategies to
avoid the activation of emotion), and secondary avoidance of affect (strategies to reduce the experience of emotion once it has been triggered). Primary and secondary avoidance of affect are found in anorexia, where anorexics of the binge/purge subtype display the highest levels. In contrast, secondary avoidance of affect is found to characterise bulimia nervosa, and this avoidance is confined to the behavioural/somatic domain. These findings cannot be explained by existing models of eating disorders, but offer some support for the proposed schema-based model.

### 6.7 Summary of the models (Table 1)

<table>
<thead>
<tr>
<th>Previous models</th>
<th>Bruch 1973; Garner &amp; Bemis 1982; Vitousek 1996; Slade 1982</th>
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<tr>
<td>Cognitive</td>
<td>Interpretation of events</td>
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<td></td>
<td>Over-evaluation of weight/shape</td>
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<tr>
<td>Behavioural</td>
<td>Perfectionism</td>
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<td></td>
<td>Control</td>
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<tr>
<td>Emotional</td>
<td>Self-esteem</td>
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<td></td>
<td>Schemas</td>
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<td>Interpersonal problems</td>
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<td>Lack of specific treatment targets</td>
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<td>Fairburn et al., 1999</td>
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<tr>
<td>Cognitive</td>
<td>Over-evaluation of eating/shape/weight</td>
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<tr>
<td>Behavioural</td>
<td>Self control</td>
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<td></td>
<td>Body checking</td>
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<td></td>
<td>Restricting</td>
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<td></td>
<td>Compensatory behaviours</td>
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<tr>
<td>Emotional</td>
<td>Self worth</td>
</tr>
<tr>
<td>Treatment targets</td>
<td>-Self-control, eating/weight/shape as indices of self-control/self worth</td>
</tr>
<tr>
<td></td>
<td>-Weight control behaviour/body checking/low body weight</td>
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<tr>
<td></td>
<td>-Cognitive restructuring (control)</td>
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</table>
Fairburn et al., 2003

Cognitive
- Over-evaluation of eating/weight/shape
- Perfectionism
- Schema-self evaluation

Behavioural
- Restriction
- Compensatory behaviours

Emotional
- Low self esteem
- Mood intolerance
- Interpersonal difficulties

Treatment targets
- Formulation
- Barriers to change
- 4 maintaining mechanisms (clinical perfectionism, core low self esteem, mood intolerance, interpersonal difficulties)
- Focus on eating disorder psychopathology

Schmidt & Treasure, 2006

Cognitive
- Perfectionism
- Thinking styles

Behavioural
- Avoidance

Emotional
- Avoidance of relationships

Treatment targets
- Motivational interviewing
- Exploration of function of AN in persons life
- Identification of pro anorexic beliefs (underlying core beliefs, values and rules)
- Self monitoring
- Letters rather than diagrammatic formulations
- Family and individual factors considered

6.8 Consideration of these models

When considering the most influential models, there has been a clear shift over time in areas of focus in the role of maintenance of AN (see Figure 1). The roles of ‘perfectionism’ and ‘control’ have been considered in more detail within a behavioural, emotional and cognitive framework. Cognitions have been explored and
understood both in terms of their relation to weight, shape and eating, but also in regard to the function of control and perfectionism. Behaviours have been explored and understood in relation to the functions they serve. Self esteem and self worth (relating to the ‘feeling’ aspect of the CBT model) have been reconceptualised in relation to the function of perfectionistic traits associated with AN (e.g., behaviourally manifested as restricting). In the latter models, the relation of these behaviours (e.g., restricting and bingeing) to ‘mood intolerance’ has been considered and formulated as part of the maintaining factors in the disorder. In this way, the influence and impact of interpersonal difficulties has been explored and considered in much more detail in recent models.

![Diagram of CBT model]

**COGNITIONS**
- WEIGHT/SHAPE/EATING RELATED
- CONTROL
- PERFECTIONISM
- SCHEMAS

**EMOTIONS**
- SELF-ESTEEM
- SELF-WORTH
- MOOD INTOLERANCE
- INTERPERSONAL PROBLEMS

**BEHAVIOUR**
- Restricting
- Bingeing
- Body Checking
- Emotion Blocking

Primary cognitive, emotional and behavioural areas considered within CBT model (Figure 1)

6.9 Aims of CBT for AN

As can be seen when considering the most recent models as discussed above, there are many different perspectives on AN, including socio-cultural views, family views, cognitive behavioural accounts and neurobiological ideas. There are many different approaches to treatment - some accounts of the disorder focus on its development
and others on its maintenance. Cognitive behavioural accounts of the maintenance of other disorders have led to important advances in treatment (Fairburn et al., 2003; Waller et al., 2007). The key behaviour is dietary restriction (although bingeing and compensatory behaviours may also need to be addressed). The principal aims of treatment for AN are the restoration of the patient’s weight to a level that is healthy for their height and age, the amelioration of their extreme weight and shape concerns, remission from abnormal eating behaviours such as purging, improvement in depressive and other co-morbidity, and most importantly improvement in quality of life (Garner, 1997).

7. Review of clinical trials of CBT for AN

7.1 Selection criteria

In the following section, the evidence for the efficacy of CBT or BT will be considered in comparison to other treatment approaches for AN, treatment as usual, dietary advice, or a control. Considering the evidence to date, it is apparent that the trials vary enormously in methodology, quality and results. The studies discussed were identified using systematic review of the literature, but are also those identified by the most recent reviews of the area (Bulik et al., 2007; Cochrane Review, Hay et al., 2007). The quality of research was assessed using Bulik and colleagues’ (2007) adaptation of West and colleagues’ (2002) quality of research scale. This takes account of: research/study aims; study population; randomisation; blinding; interventions; outcomes; statistical analysis; results; discussion; external validity; and funding/sponsorship. Three categories were used (poor, fair, good), and the studies included in the current review were all rated as ‘fair’. None were found to be ‘good’. Those rated as ‘poor’ were left out of the review. Outcome measures included some
or more of the following: MR-Scale change, weight gain (measured BMI), reducing psychological features of AN (as assessed by the Eating Disorders Inventory/examination (EDI/EDE); Dysfunctional Attitudes Scale (DAS) and Locus of Control of Behaviour (LCB), e.g., drive for thinness, and lessening depression and anxiety.

Papers reviewed are from an adult only population (17-45). The majority of trials fail to specify the type and nature of the CBT employed, but it is assumed that the principles will be similar to the previous models discussed.

7.2 Results of trials of treatment for AN

All the studies discussed involve the use of a common set of outcome measure criteria, the Morgan-Russell (MR) criteria, with the exception of Serfaty’s (1999) and McIntosh et al.’s (2005) papers. There are numerous forms of the criteria, which have advanced and changed with time, and are summarised below (MR Composite Score; MR General Scale; Global AN Measure.)
Table 2. Morgan-Russell criteria (MR Composite Score; MR General Scale; Global AN Measure.)

MR COMPOSITE SCORE

Morgan and Russell compromises 5 subscales measuring:

- Nutritional status, aggregating scores on dietary restriction, worries about food and shape and current body weight
- Menstrual scale
- Mental state
- Psychosexual scale (current activities; sexual partner status; interest in sexuality; ambitions in sexuality; wish eventually to have a child) and sexual functioning
- Socio-economic scale (relationship with family, emancipation from family, capacity to confide, capacity for group leisure, capacity to work/study full time)

MR GENERAL SCALE

'Good outcome' – normal body weight (above 85% of average for age, gender and height) with normal menstruation

'Intermediate outcome' – normal or near normal body weight and/or menstruation abnormalities

'Poor outcome' – low weight and absent or scanty menstruation

GLOBAL AN MEASURE

4=full criteria for AN, 3=not full AN but having a number of features of eating disorders,
2=few features of eating disorders, 1=no significant features of eating disorders.
7.3 Treatment trials including CBT/BT

Channon et al.'s. (1989) study included 24 outpatients with a dropout of 13%; the range of ages was from 22 to 26. Treatment involved 18 sessions over 6 months and 6 booster sessions over 6 months follow up and 12 month follow up. The trial was randomised (but the groups were not matched for duration of illness). Channon et al. (1989) compared CBT with BT and a control group (half hour session with psychiatrist, weight restoration aim). At 6 months CBT was associated with better psychosexual functioning than BT. BT was associated with greater improvement in menstrual functioning than CBT. However, these results did not differ significantly from those of the control group. On the M-R Composite Scale there were no differences between groups. At 12 month follow up, CBT and BT combined improved nutritional functioning more than was found in the control group. However the control group showed more improvement in drive for thinness than in the CBT and BT groups combined. There were no significant changes on the other measures, regardless of group membership. When change was analysed regardless of group membership, there were significant improvements in weight, nutritional functioning, menstrual functioning, psychosexual functioning, preferred weight, the EDI subscales drive for thinness, interoceptive awareness and maturity fears, and depressed mood.

Limitations include the fact that the control group were seen by a therapist at the therapist’s discretion and they had fewer therapy sessions than the CBT and BT group. There were also variations across groups in age of onset, duration of illness and previous hospitalisation. The behavioural group had an average of 5 years later onset age, fewer previous hospital admissions, and a shorter duration of illness. In
terms of prognostic factors, these differences might have had an impact on the evaluation of treatment success. The impact of pre-treatment characteristics on therapy outcome will be explored in the study reported in part 2 of this thesis.

Treasure et al’s. (1995) study had 30 patients, aged 18-45. Treatment involved 20 weekly sessions over 5 months and involved outpatients. The trial was randomised. The dropout rate was 33%. Average duration of illness was 4.7 years. Treasure et al. (1995) compared (Cognitive Analytic Therapy (CAT), Ryle & Kerr, 2002) with EBT (educational behavioural therapy, a form of BT). At one year, average weight gain across groups was 6.8kg, and 19 (63%, MR General Scale) had a ‘good’ or ‘intermediate’ recovery in terms of nutritional outcome. The outcome ‘poor’ was found in 37% of participants. CAT was associated with higher self-rating of improvement. There was no advantage of CAT over EBT in eating, mood or weight outcomes. In terms of prognostic factors, only percentage weight loss contributed significantly to the good outcome category.

The size of the study was small and therefore the power to distinguish between the two forms of treatment was limited. The therapists were also relatively inexperienced. Patients with a mixed diagnosis of AN and BN were included. Patients who had made a good recovery from AN in terms of weight and menstruation but who had developed BN were categorised as having a ‘good’ outcome. This highlights having alleviated one condition and having it behaviourally manifested in another form.
The findings suggest that an initial step of outpatient treatment is appropriate even for the older group with a longer history. Only two thirds of the sample completed the full course of treatment and the outcome did not differ between the cohort who completed treatment and those who didn’t, raising the possibility that shorter courses of treatment are appropriate for some cases.

Serfaty et al.’s (1999) study had 35 patients. Patients received cognitive therapy (CT) or a dietary intervention (control group) in an outpatient setting. 23 out of 25 patients who received cognitive therapy were followed up at 6 months. None of those in the control group remained in treatment. Significant improvements in BMI’s, EDI’s, Dysfunctional Attitude Scale (DAS) (Weissman & Beck, 1978) and Locus of Control of Behaviour (LCB) (Craig et al., 1984) were observed. After 6 months of treatment 70% no longer satisfied diagnostic criteria for AN, increases in BMI were observed in 87% of patients and 61% had BMI’s of above 17.5. 70% achieved normal sexual function. Total scores in EDI fell and interestingly scores for body dissatisfaction did not increase with weight gain. Significant falls in BDI scores were found. There was also a reduction in LCB scores, perhaps suggesting that CT had an impact on shifting patient’s sense of personal effectiveness. Only BMI at baseline predicted outcome at 6 months.

It must be acknowledged that non-specific treatment effects may account for improvements in the CT group. Measures of patient’s subjective evaluation of treatment and the therapist may have been of interest. Given that improvements may continue for up to 2 years following the start of therapy, a longer period of follow-up is necessary in future studies.
Pike et al’s. (2003) study included 33 patients, aged 18-45. Treatment was manualised and involved 50 sessions over 12 months, and followed discharge from in-patient treatment. The dropout rate was 9%. The trial was randomised (controlling for duration of illness and illness subtype), with patients receiving either CBT or nutritional counselling. The study was unusual in that patients began CBT only after weight restoration on an inpatient unit. CBT in this study focused on a schema-based approach to address issues around self-esteem, self-schema and interpersonal functioning. Findings suggested that CBT significantly reduced relapse risk and increased the likelihood of ‘good’ outcomes. It also led to a lower percentage of treatment failures. Furthermore, patients who had CBT took a longer time in weeks to relapse, compared with those who received nutritional counselling. Criteria used to define ‘good’ outcome were weight restored, no binge eating or purging, eating attitudes, and weight concerns as measured by the EDE. In the CBT group, 44% had ‘good’ outcomes, whereas this was the case for only 6.7% in the nutritional group (as measured by the Global Outcome Scale). However, only 17% of patients in the CBT group and none in the nutritional counselling group were considered to be fully recovered. There was an added benefit of taking antidepressant medication on ‘good’ outcomes. Out of the 8 CBT patients who had good outcome, 7 were also receiving antidepressant medication compared with only 4 of the 10 who received nutritional counselling. Therefore, the superiority of CBT may have been related to the combination of medication and therapy.

There are a number of limitations to the study. It is unclear as to whether patients had any exposure to therapies while hospitalised for weight restoration. It is possible that
such treatment may have differed between the two groups, thus possibly biasing their response to current treatment (Strober, 1995). The BMI of those in the study had to be at least 17.5, so patients no longer met diagnostic criteria for anorexia nervosa, and are not necessarily representative of those with AN, affecting the utility of the results.

McIntosh et al’s. (2005) study had 56 patients, aged 17-40. Treatment involved 20 sessions of outpatient treatment. The dropout rate was 38%. The trial was randomised. McIntosh et al. (2005) compared CBT with interpersonal therapy (IPT) and non-specific supportive clinical management (NSCM; incorporating elements of nutritional counselling and behavioural weight restoration strategies). IPT focuses on the interpersonal problem areas: interpersonal disputes, role transitions, grief, and interpersonal difficulties but does not focus explicitly on weight or eating. In an intention to treat analysis, NSCM performed significantly better than IPT in producing global ‘good’ outcome ratings. CBT outcomes were in-between and were not significantly different from outcomes in the other two treatments. NSCM was superior to CBT in improving global functioning over 20 weeks, and CBT was superior to IPT in reducing eating restraint over the 20 weeks. NSCM achieved ‘good’ outcomes in 82% of cases, CBT 42% and IPT 17% (MR General Scale). For eating concerns and weight concerns, both NSCM and CBT were superior to IPT. For the whole sample at the end of treatment, 9% of the subjects had a very good outcome and a further 21% had improved considerably; 70% either did not complete treatment or made small or no improvements. For those completing therapy, there was no difference by therapy group on weight outcome. However, comparing the
participants who did and did not complete treatment revealed a significant difference in the mean base-line weight (46.9 kg and 45.6 kg) respectively.

There were a number of limitations. There was a lenient BMI requirement for inclusion in the study, which did not require participants to have amenorrhoea. Using the MR General Scale (which uses amenorrhoea as part of its recovery criteria) is therefore questionable in terms of its utility in evaluating outcome. A substantial proportion of the sample had a higher pre-treatment BMI than many of the patients in other trials have had after treatment, making their outcome difficult to evaluate. It is of note that the NSCM treatment contains elements of BT and therefore may not be an ideal control treatment to compare against CBT. Again, the absence of follow-up data means that the results can only be considered interim. It is possible that IPT has a slower but similar effect to CBT, as has been shown in BN (Wilson, 1999; Fairburn et al., 1993).
### 7.4 Characteristics of included studies (Table 3)

#### Channon 1989

| **Methods** | RCT, treatment 6 months duration, 6 booster session over 6 months, and follow-up at 1-year  
| | Outcome evaluation not blind  
| | Allocation concealment unclear |
| **Participants** | 24 female patients (dropout 13%) with AN (Russell’s criteria 1983)  
| | Mean age 23.8 years  
| | Exclusion rate 29%  
| | Outpatient |
| **Interventions** | CBT vs. BT vs. control group |
| **Outcome measures** | BMI, MR Composite Score, EDI, BDI, Maudsley Obsessive-Compulsive Inventory and preferred weight |
| **Results** | **6 months**  
| | CBT better psychosexual functioning than BT  
| | BT greater improvement in menstrual functioning than CBT. Both these results did not differ significantly from control group  
| | No differences on MR composite scores across groups  
| | **1 year**  
| | CBT/BT better nutritional functioning than control group  
| | Control group more improvement in drive for thinness  
| | No significant changes on other measures regardless of group membership |

#### Treasure 1995

| **Methods** | RCT, treatment 5 months duration, and follow-up at 1-year  
| | Outcome evaluation not blind  
| | Allocation concealment unclear |
| **Participants** | 30 patients (one man) (dropout 33%) with AN (ICD-10)  
| | All over 18 years  
| | Exclusion rate 21%  
| | Outpatient |
| **Interventions** | Educational Behavioural Therapy (EBT) vs. CAT |
| **Outcome measures** | BMI, MR General Scale |
| **Results** | **1 year**  
| | There was no advantage of CAT over educational BT in eating, mood or weight outcomes. |
Serfaty 1999

Methods
RCT, treatment 6 months duration,
Outcome evaluation not blind
Allocation concealment adequate

Participants
35 patients (2 men) (dropout 13%) with AN (DSM-III-R)
Over 16 years
7 patients had a BMI of between 17.5 and 19
None of those screened were excluded
Outpatient

Interventions
CT vs. control group (dietary advice)

Outcome measures
BMI, EDI, Dysfunctional Attitudes Scale (DAS), Locus of Control of
Behaviour (LCB) and BDI

Results
6 months
Significant improvements in BMI's, EDI's, Dysfunctional Attitude Scale
(DAS) (Weissman & Beck, 1978) and Locus of Control of Behaviour (LCB)
(Craig et al., 1984) were observed. After 6 months of treatment 70% no
longer satisfied diagnostic criteria for AN, increases in BMI were observed
in 87% of patients and 61% had BMI's of above 17.5. 70% achieved normal
sexual function
All of dietary advice group dropped out

Pike et al. 2003

Methods
RCT, treatment 1 year duration, 50 sessions
Outcome evaluation not blind
Allocation concealment unclear

Participants
33 patients (dropout 9%) with AN (DSM-IV)
Between ages 18 and 45
10 people excluded
Outpatient, post hospitalisation

Interventions
Cognitive Behavioural Therapy (CBT) vs. nutritional counselling

Outcome measures
EDE, BMI, MR General Scale

Results
1 year
A significantly higher percentage of individuals receiving CBT (44%, 8 of
18) then in the nutritional counselling group (7%, 1 of 15) met MR criteria
for "good" outcome. Reaching "full recovery" were met by 17% (3 of 18) in
the CBT group and none of the patients in the nutritional counselling group.
CBT group had lower dropout and relapse rates also.
McIntosh 2005

Methods
RCT, duration of treatment was 20 sessions over a minimum of 20 weeks
Blinding of outcome evaluation was done and was double blind
Allocation concealment adequate
Follow-up is ongoing at 3, 6, 9, 12 months and 2, 3, 5 years post-treatment

Participants
56 female patients (dropout 38%) with AN (DSM-IV criteria) or EDNOS
AN (lenient weight 17.5-19, DM-IV criteria) without amenorrhoea criteria being imposed
Age 17-40 years
Exclusion rate 42%
Outpatient

Interventions
CBT vs. IPT vs. non-specific clinical management (control group)

Outcome measures
20 weeks
Primary outcome: clinician global rating
Secondary outcomes: self-report EDI-2, height, weight, %body fat, EDE subscale scores, GAF, Hamilton Depression Rating Scale

Results
NSCM achieved ‘good’ outcomes in 82% of cases, CBT 42% and IPT 17%
(Global Outcome Measure). For eating concerns and weight concerns, both NSCM and CBT were superior to IPT

7.5 Conclusions of treatment trials
From the studies reviewed above that examined CBT or BT in comparison to other treatment types or control/non treatment group, tentative conclusions can be made that CBT can enhance certain areas of functioning better, such as in the area of ‘nutritional functioning’ and on the MR General Scale which assess weight gain. This is particularly the case where weight restoration has already been established (Pike et al., 2003). In two studies, the ratings of a ‘good’ outcome for CBT on the MR general scale had been around 40% of the group. However in other areas it has been shown to perform worse than routine treatment or other treatment approaches. In the studies reviewed, no difference has been found between BT and CBT.
8. Problems with treatment trials

8.1 Why are there different conclusions?

Conclusions are hard to draw for a variety of reasons, some of which highlight the
difficulties of conducting research studies with this particular population. The
‘quality of research’ critique has been broken down into sections.

8.2 Different outcome measures and definitions of recovery

The most common criteria used across studies were various forms of the M-R
criteria, utilising a number of different scales. The use of different scales across
studies makes it hard to draw conclusions. In terms of the more dated criteria such as
the MR Composite Scale used in Channon et al.’s. (1989) study, this tool is outdated
and therefore not useful. One of the categories of the scale is heterosexist and flawed
in assumption that a ‘desire to have children’ is an indication of psychological health.
Its social and political status represents the climate of the time.

A limitation of the MR criteria is that they do not cover related psychological and
behavioural variables that are core criteria for measuring AN status. Often the studies
discussed have focused primarily on weight gain and behaviour change and have
neglected the consideration of cognitions. Consequently a patient could meet criteria
for ‘good’ outcome but still be symptomatic in terms of weight concerns, shape
concerns and eating behaviour. Gowers (2006) highlight how after re-feeding
patients complain that weight has changed but psychological shifts fail to be
achieved. Criticisms of the MR scales do not apply to the EDE-Q because this
measure directly assess behaviours and cognitions.
In some of the studies reviewed so far, recovery is also assessed using a range of different questionnaires other than the Morgan Russell scales. These measures address different areas of functioning; such as cognitive, behavioural and physical status and these areas have not been examined consistently or repeatedly across studies, and therefore it is difficult to draw overall conclusions. The development of well validated, reliable, wide ranging and comprehensive outcome measures is a fundamental need for future research into the treatment of AN. There is no consensus as to what constitutes “recovery” in the simplest sense from this condition. In fact, no consensus definitions exist for stage of illness remission, recovery and relapse. There is a necessity to develop standardised definitions of these terms for AN and the focus of this work should be on measuring clinically meaningful differences rather than those which are simply statistically significant.

8.3 Poor specification of treatment approaches

In many of the studies it is unclear as to what model or CBT approach is drawn upon in treatment. Garfinkel & Garner (1982) highlight how inadequate descriptions of psychotherapeutic interventions lead to difficulties reproducing them. As mentioned in the initial section of the review, there are still currently a number of CBT orientations and treatment packages that are being administered by clinicians. It will be necessary for future studies to provide information on the exact treatment approach adopted during individual studies. The effects of differential treatment duration are also consistently ignored across studies.

Further research is needed to identify which patients will respond to a particular first-line treatment. It is unclear whether some therapy models are more suitable for some
individuals or whether individuals who fail to respond to one therapy will also fail to respond to another therapy model (McIntosh et al., 2005). Bulik et al. (2007) highlight how we should adapt further various psychotherapeutic interventions that are tailored to the unique core pathology of AN, and that are both efficacious and acceptable to the patients. Factor analysis can be a useful tool for delimitating the types of dimensions researchers might consider when empirically designing a treatment for eating disorders.

8.4 Small sample sizes

The median sample size of trials was very small, and thus power is a problem for many trials. Sample size across studies is commonly insufficient to draw conclusions in terms of differential efficacy across groups.

8.5 Design

Randomisation procedures were often poorly defined and there was often inadequate allocation concealment across trials. Bias was also possible in most trials due to lack of blind outcome assessments. The exclusion criteria used within some studies (e.g., the absence of comorbidity) means that it may be difficult to generalise these findings into clinical practice (Mitchell et al., 1997).

8.6 Time of measurement

In general studies have varied in terms of whether interventions were targeted at individuals before or after weight restoration. An optimal approach to renutrition has not been established. In terms of clinical considerations, duration of therapy differed across and within studies, which makes conclusions difficult to reach. This is
particularly important given the fact that the process of recovery may well continue for several years after the process of weight gain has been completed, with no apparent end point (Woodside, 2005). Future trials need to measure outcomes at similar time points (e.g., at end of treatment and at least at one year follow-up).

8.7 Demographic factors

There was inconsistency across studies as to BMI at time of treatment, age of participants, dropout rates, previous experience of hospitalisation, stage in illness and medication use. Some studies even failed to report basic demographic and clinical information (e.g., duration of illness; age of onset of illness; initial weight; AN subtype).

8.8 Drop out-non acceptance/completion-predictors of adherence

Halmi et al. (1995) show how high attrition rates are very problematic across studies. Substantial dropout rates create a problem for clinical trials for numerous reasons. The small sample size of completers means that statistical tests have low power and poor precision in effect size estimators. Analysis of completers may be biased, as completers in one group may not be comparable with those in another group. Intention to treat analysis imputation methods may lead to unfair comparisons of treatments because adequate imputation requires some understanding of the mechanisms leading to non-completion in each treatment group. It is quite possible that those who drop out of treatment for AN deteriorate rather than remain stable. Alternatively they may seek treatment elsewhere and make significant improvements. Differential attrition between treatment intervention groups and comparison groups is concerning as it may indicate non-acceptance of treatment,
lack of engagement and poor motivation. The reason for dropout is essential to understanding what may be done to improve success with this population.

8.9 Outpatient versus in patient treatment

There are treatment-, therapist- and patient-related factors that may explain why success in treating AN with CBT is limited and why conclusions are inconsistent. The context of the service not only has an impact on treatment trial evaluation, but also has an impact on the delivery of the treatment. The context of a service will reflect the severity of illness a patient presents with, and consequently the utility that CBT has within this setting. Across studies the variety in inpatient and outpatient treatment is apparent, and the lack of success with inpatient treatment is more understandable as often focus is on weight gain, with a lack of consideration for behavioural and cognitive aspects of change. In the few trials conducted, it has been shown that CBT is less effective for the chronically underweight, and therefore it is likely that trials will reflect poor results within the inpatient population. However the success with outpatient treatment is still well below that of treatments for BN. It is possible that part of this may be attributable to clinician stance and a possible lack of adherence to CBT protocols, due to the difficulty of treating AN.

9. Why are dropout rates so high?

It is important to consider factors involved in causing dropout within treatment as this is a very prevalent reality amongst the AN population. This can help clinicians consider what could be done differently within treatment to promote adherence. It may also help explain why individuals are likely to do poorly within treatment. It is possible that drop out differs across diagnoses, and may not be easily considered
transdiagnostically. Therefore, in the following section, patterns of dropout have been considered across articles examining AN only.

9.1 Patient related factors

Individual factors (demographic and clinical factors)

Vandereycken & Pierloot (1983) reported that risk of drop out was influenced by both demographic factors (age at admission, educational level and social class) and clinical factors (duration of illness). Early dropouts compared to those who completed at least the first phase of treatment of the entire programme indicated that early dropout may be associated with later age of onset of AN (as concluded by Steinhausen, 2002), older age at admission, lower socio-economic status and lower educational achievement. However a large number of studies state that there is no effect of age of onset on dropout (Surgenor et al., 2004; Fassino et al., 2002; Kahn & Pike, 2001; Woodside et al., 2004; Zeeck et al., 2005). Fassino et al. (2002) and Zeeck et al. (2005) found no effect on dropout of level of education. There has been no found effect of gender (Surgenor et al., 2004; Zeeck et al., 2005); marital status (Surgenor et al., 2004; Woodside et al., 2004) living situation or being in a relationship (Zeeck et al., 2005) on drop out. The only firm conclusions that can be made are that older age at admission and lower socio economic status are likely to induce early dropout.

Illness status

Halmi et al. (2005) found treatment utilisation to be significantly related to severity of eating disorder symptoms. Contrary to expectations, Kahn & Pike (2001) found that the most severely and chronically impaired patients were not more likely to drop
out of treatment. In terms of duration of illness, it was found that a longer duration increased the likelihood of dropout in a number of studies (Vandereycken & Pierloot, 1983; Steinhausen, 2002; Kahn & Pike, 2001), however other investigations found no significant association with dropout (Fassino et al., 2002; Surgenor et al., 2004; Woodside et al., 2004; Zeeck et al., 2005). Weight loss also revealed mixed results, with a greater weight loss indicating a greater likelihood of drop out in some studies (Steinhausen, 2002; Vandereycken & Pierloot, 1983) and revealing no effect in another (Vandereycken & Pierloot, 1983; % body weight). It might be expected that a low BMI would increase risk of dropout, however results revealed mixed patterns yet again; lower BMI at admission meant a greater likelihood of dropping out in one study, (Surgenor et al., 2004) a lower likelihood of dropping out in another (Woodside et al., 2004) and no significant association with dropout in a further two studies (Fassino et al., 2002; Kahn & Pike, 2001).

Results were consistent when considering a shorter duration of amenorrhea (Vandereycken & Pierloot, 1983) and an increased restriction of fluids at point of admission, (Surgenor et al., 2004), which both highlighted a greater likelihood of dropout. There was no significant association between lowest ever BMI and drop out (Woodside et al., 2004; Kahn & Pike, 2001) and age of menarche (Vandereycken & Pierloot, 1983).

The only firm conclusions to be drawn are that those with more severe eating disorder symptoms are less likely to drop out and those with a shorter duration of amenorrhea and an increased restriction of fluids at point of admission have a greater
likelihood of dropping out. However each of these findings has only been shown in a single study and therefore the results are more speculative.

**Co morbid behaviours**

In Halmi's (2005) study a low level of self esteem led to a medium completion rate (40%) whereas a high level of self-esteem was associated with a high completion rate (86%). Kahn & Pike (2001) and Surgenor et al. (2004) found no significant effect of self-esteem on dropout. Higher co morbid Depression led to a greater likelihood of dropping out (Woodside et al., 2004), a lower likelihood of dropping out (Zeeck et al., 2005) and no significant effects on dropout across studies (Kahn & Pike, 2001; Surgenor et al., 2004). It is interesting that Obsessive-compulsive features led to a greater likelihood of dropping out (Zeeck et al., 2005; Halmi et al., 2005) but that a full diagnosis of OCD had no effect on dropout (Zeeck et al., 2005). Overall no firm conclusions can be made about variables affecting dropout due to contradictory findings across studies.

**Impulsive behaviours**

In terms of diagnosis, Kahn & Pike’s (2001) and Woodside et al’s. (2004) studies revealed that those who engaged in binge/purging behaviours (AN binge/purging subtype) were significantly more likely to leave treatment prematurely. Again Surgenor et al. (2004) revealed an increased likelihood of dropout in AN purging subtype participants. The finding that binge/purging subtype contributes to the risk of dropout in clinical populations can be explained in many ways. Walsh & Garner (1997) show that purger's show poorer impulse control than those who use restrictive behaviour and this may contribute to initiating dropout in the current study. It is
possible that the disruption of the opportunities to purge in treatment may lead people to leave treatment. In line with this, Fassino et al. (2002) highlight that impulsivity/use of impulsive behaviours, resulted in greater likelihood of drop out. However Zeeck et al. (2005) revealed no effect. Overall the evidence indicates that binge/purging/impulsive behaviours increase the likelihood of dropout of treatment.

When breaking down frequencies of behaviours, a greater frequency of bingeing revealed a greater risk of dropout (Steinhausen, 2002) and no effect on dropout (Fassino et al., 2002; Surgenor et al., 2004; Vandereycken & Pierloot, 1983; Woodside et al., 2004). Similarly higher levels of vomiting increased risk of dropout in one study (Steinhausen, 2002) and was shown to have no effect on drop out in others (Surgenor et al., 2004; Vandereycken & Pierloot, 1983, Fassino et al, 2002; Woodside et al., 2004). Clearly the frequency of these behaviours does not have an effect on dropout, but it is the behaviour itself that is influential. Excessive exercise (Fassino et al., 2002; Surgenor et al., 2004; Woodside et al., 2004) and diuretic misuse (Surgenor et al., 2004) were all found to have no effect on dropout.

**Personality traits**

When considering patients rated with higher levels of ineffectiveness, results were mixed, some revealing a greater likelihood of dropping out (Fassino et al., 2002) and some showing no significant effect (Surgenor et al., 2004). The same is true for maturity fears in patients, as this was found to have an effect on dropout (Woodside et al., 2004; Zeeck et al., 2005) and was also shown to have non-significant relationship with dropout (Surgenor et al., 2004). The characteristic, perfectionism (Surgenor et al); awareness (Surgenor et al., 2004) and a higher level of
interpersonal distrust (Surgenor et al., 2004) were all found to have no effect on dropout rates. No firm conclusion can be made in personality traits indicative of early dropout, but there are a number that have been found to have no influence on dropout.

Anorexic cognitions

When considering lower levels of restrictive behaviour and greater weight concern, it was found that there was a greater likelihood of dropping out of treatment (Woodside et al., 2004). There was no effect of anorexic attitudes (Kahn & Pike, 2001) or greater body shape dissatisfaction/perfectionism (Kahn & Pike, 2001; Surgenor et al., 2004) on attrition. Greater drive for thinness was associated with more likelihood of drop out (Fassino et al., 2002) but was also found to have no effect on drop out (Surgenor et al., 2004). Again despite some patterns across particular cognition styles, results do seem somewhat contradictory, as cognitions that would be expected to be linked are not.

There are obviously a number of reasons why an individual is likely to drop out of treatment. In terms of demographic factors, the age at admission of patients appears to have an impact on dropout as it seems that services need to consider that the earlier the admission age the better the likelihood of remaining in treatment across individuals. In terms of illness status those who are in fact less severe are more likely to drop out of treatment, and therefore from an early stage of engagement this needs to be considered by the clinician as a risk factor for dropout. The reason for this also needs to be explored and understood more. There is little in the way of comorbidity or personality traits that appear to have a meaningful effect on dropout. However
impulsive behaviours and the sub categories of AN binge/purging behaviours do
appear to have an impact on dropout. Clinically these behaviours need to be
addressed early within treatment to prevent possible dropout.

In regard to cognitions related to anorexic attitudes and behaviours the pattern is
contradictory. Greater weight concern did appear to indicate a greater likelihood of
dropout in the one study. Greater drive for thinness was associated with a greater
likelihood of dropout and was shown to have no effect on dropout in another study.
In terms of behaviours interestingly lower levels of restriction were associated with
greater dropout. This does connect with the earlier finding that patients who were in
fact less severe were more likely to drop out of treatment. This suggests that it is
perhaps the cognitions that need to be considered within therapy over and above
behaviours at early stages of treatment. There seem to be no effect of the other
anorexic attitudes and beliefs.

9.2 Treatment and service related factors

In terms of dropout, Kahn & Pike (2001) revealed that patients who dropped out in
the earliest phase of treatment had more previous hospitalisations than patients who
dropped out later in treatment, suggesting that the patients who seek discharge early
in course of treatment may be enacting a pattern of repeated, unsuccessful inpatient
treatment. In contrast to this, Zeeck et al. (2005) found that those with longer
treatment history were less likely to drop out and Vandereycken & Pierloot (1983)
found no significant effect in their study. There was no correlation with adherence in
previous dropout history (Zeeck et al., 2005). In this way it seems that conclusions
cannot be made in terms of the experience of repeated inpatient admissions on the success or lack of success in the treatment of AN.

9.3 Therapist related factors

Findings by Thompson-Brenner & Westen (2005) suggest that when utilising CBT therapists use a broad spectrum of approaches, including psychodynamic principles and this is in response to patient needs. However in this study the survey sample size was relatively small (N=100), and may not be representative of those who specialise in eating disorders specifically. Tobin et al. (2007) conducted a larger, more specific trial. Results revealed that most psychotherapists who have experience of working in the eating disorders use an array of interventions. Some work using CBT manuals, and other clinicians utilise treatments that have no randomised control trial support (e.g., psychodynamic therapies). Using CBT in conjunction with other approaches leaves the vulnerability of "dismbodying CBT methods from the cognitive behavioural model that informs their use" Wilson (2005). Tobin et al. (2007) found that only 13% of the sample claimed to practice from a single theoretical perspective. Interestingly, when breaking down actual activity, 98% of the sample engaged in CBT and psychodynamic practice with patients. 16% of clinicians claimed to have not studied treatment manuals. Other dimensions of treatment included coping skills, training, family history, limits and contracts, therapist disclosure and patient feelings. This was assessed using factor analysis. A major limitation of the study is the failure to specify diagnostic categories. It is therefore unclear how eating disorder diagnosis may affect clinician’s choice of interventions. The findings do however reveal that part of our “apparent” lack of success in using CBT, may in fact not be due to the treatment itself, as it is apparent that very few studies are likely to actually be
assessing consistent, manualised CBT. It may be that in some of the previously discussed treatment trials clinicians may have been utilising CBT in a similar way. This fits with the fact that few of the studies were specific about the particular CBT approach that was used.

In terms of dropout, Clinton (1996) found no significant differences in dropout rate when considering therapist variables: level of training, profession, gender, years of experience, specific therapist, change of therapist between assessments. Gallop et al. (1994) do not reveal a relationship between therapist ratings of alliance and dropout. However they show that patients who dropped out of treatment early were more likely to rate the alliance as worse than those who completed. Kahn & Pike (2001) identify that other factors such as motivation for recovery and expectation of treatment efficacy, strength of therapeutic alliance and psychosocial adjustment to the treatment program may be significant moderating factors in determining responsiveness to inpatient treatment. It is therefore important to conduct trials that assess not only the success of manualised CBT, but also the dropout rate associated with doing therapy in this way, as it would not be helpful performing a therapy that has a high drop out, regardless of the success that it may have with a small number of patients who are retained.

9.4 Methodological considerations and limitations of studies considering drop out

There are a number of studies that consider patient, treatment/service and therapist related factors in the role of dropout of treatment and overall conclusions drawn are quite tentative. This is largely due to the contradiction in results across studies. In addition to this there are methodological considerations. Across the studies discussed
the definition of dropout often varies and therefore drop out rates may differ significantly as a result of the definition used (Wierzbicki & Perarik, 1993). Criteria may be based on therapy timeframe, patients’ weight and completion of treatment packages. Again the characteristics of the populations examined are varied across studies, and include different age groups, a variation in the severity of illness and service setting and different treatment types. Inclusion and exclusion criteria also vary (Mahon, 2000). In terms of the quality of research, some studies were conducted by single research groups, affecting the validity of their findings.

It should be kept in mind that attending treatment does not necessarily equate with compliance with treatment and with following treatment recommendations. Again on the other side of the argument, dropout may reflect recovery. Mahon et al. (2001) conclude that dropout is not necessarily predicted by pre-treatment factors alone, and highlight that it may be due to factors that operate throughout treatment. It will be important to replicate any positive results in order to show that any pattern in drop out rates are due to the factors examined and not other related factors or due to random chance.

10. Conclusions
The development of a transdiagnostic model has enabled AN to be considered in an innovative way, and has also enabled EDNOS to be given the consideration necessary. Waller et al’s. (2007) model advances the understanding of eating disorders generally, and specifically AN in terms of conceptualising this as a process of avoiding negative emotion. Schmidt & Treasure (2006) discuss the interpersonal dimension to the disorder, in terms of the need to avoid the experience and
expression of intense negative emotions. In this way AN can be comparable in functionality to other anxiety based disorders.

From the small amount of trials considered, no firm conclusions can be made in terms of best treatment approach. Most clinicians agree that it is not currently possible to predict which patients will do well in a given treatment and that almost any approach works at least some of the time (Woodside, 2005). However, there is tentative evidence that CBT reduces relapse risk for adults; after weight restoration has been accomplished. What would be useful from this point is to make sure studies can be conducted on CBT that is manualised and used in the form in which it was designed. This would enable the efficacy of individual components of CBT, such as behaviour experiments, dysfunctional thought diaries, and psychoeducation, to be easier to evaluate. Behavioural interventions including exposure and response prevention should be explored individually within trials given their effectiveness in treating phobias and OCD.

It may be that a randomised control trial is not the most useful way to achieve evidence of treatment success (Treasure & Kordy, 1998). This is largely because of the variation across factors, for example a treatment varies in theoretical approach (e.g. CBT versus IPT versus psychodynamic), the stage of treatment examined (underweight versus restoration), in setting (in versus outpatient), dose and duration of treatment. These combinations can also have an effect on the sub population of patients in different diagnostic categories (restrictive or binge-purging AN) and of severity. Again, trials do not necessarily reflect the type of treatment typically delivered in the community. It may be more appropriate that treatment approaches
are evaluated within clinical settings, reflecting a more realistic and natural environment.

Across trials it is necessary to use a standard and consistent measure of physical recovery, such as the MR General Scale or the MR Recovery Scale, to determine weight gain and behavioural change. It is also necessary to utilise a consistent measure that considers patterns and shifts in cognitions, such as the TAQ-ED, to determine recovery, as consistently studies have shown that unless changes are made to these dimensions, relapse is more likely. Outcome measures of clinical trials may benefit in considering not only core measures but also associated symptoms such as quality of life and social adaptation (Agras et al., 2004). Broadening the diagnostic criteria may allow more productive exploration of the nature and variations of AN.

In terms of attrition rates across trials, there are a number of variables that have been examined in terms of their predictability of dropout. Of the significant association found, it is useful to know that age appears to have an influence on dropout, with higher dropout in those with older age at admission; those of higher BMI and less chronically and severely impaired were more likely to dropout of treatment; binge/purging subtype of AN were more likely to dropout; and lower levels of restrictive behaviours lead to a greater likelihood of dropout also. In terms of therapist related factors on dropout what did increase dropout rates was therapeutic alliance as assessed by the patient.
Future considerations

Previous studies (Wilson, 1999) have broken down the effective components of CBT for BN in an attempt to understand success of treatment, however the story is different for the case of AN. Perhaps the variety of models for AN explain why it is difficult to break down individual components of the model. In terms of short-term outpatient treatment there is a variety of models of CBT, which can be administered across services and clinicians, and it is therefore near impossible to evaluate success because of this variation. A starting point would be to consider a CBT based model that is consistently adhered to by clinicians in a clinical setting, taking part on a research trial. Again the participants enrolled in clinical trials need to be followed long enough, so that the duration of any treatment effect observed and its larger clinical and public health significance is apparent (Strober, 2005).

The following empirical paper attempts to learn from previous flaws in research examining weight gain and improvement in AN patients using CBT; identified by the literature review. The influence of comorbid psychopathology on weight gain are considered aswell as eating/weight/shape, personality style and mood related cognitions. The rationale being to further our understanding of emotions and cognitions when using CBT on a AN population within a clinical outpatient setting.
References


PART 2: EMPIRICAL PAPER

OUTPATIENT COGNITIVE BEHAVIOUR THERAPY FOR ANOREXIA NERVOSA: WHICH PATIENTS SHOW EARLY WEIGHT GAIN?
Part 2 Empirical Paper: Outpatient Cognitive Behaviour Therapy for anorexia nervosa: Which patients show early weight gain?

1. Abstract

**Background:** Within cognitive behavioural therapy (CBT) models, there is growing evidence that early behavioural change is key to the outcome of treatments for bulimic disorders, and that treatment needs to be enhanced if such early change does not take place (Agras et al., 2002). Although there is no comparable evidence base in anorexia nervosa, clinical experience suggests that weight gain is a crucial early index of likely treatment success. However, there is no current evidence base that allows the clinician to anticipate who will make the necessary early weight gain. This study examined the factors present (cognitive and emotion related) at the start of CBT for anorexia nervosa that predict change in weight over the first ten sessions of treatment.

**Method:** Forty-five women (mean BMI = 16.8; mean age = 20.8 years) entered outpatient CBT for anorexia nervosa or atypical anorexia nervosa (DSM-IV diagnoses). Of the 45 patients, 38 (84.4%) remained in treatment at session 10. Each woman completed the Eating Disorders Examination-Questionnaire, the Brief Symptom Inventory, and the Young Schema Questionnaire at the start of treatment, and was weighed routinely. The Testable Assumptions Questionnaire (TAQ-ED) was completed at the start of treatment and at session 10. Weight change and behaviour change (bingeing, purging and laxative use) was assessed at the beginning, session six and session ten of CBT.

**Results:** There was a significant level of weight gain over the first ten sessions (mean = 1.70 kg). Over the whole ten-session period, weight gain was slower if the
individual had a lower body mass index (BMI), more pathological eating attitudes, higher levels of anxiety (but not depression), and higher levels of perfectionism. Over the first six sessions, weight gain was slower among those with a lower BMI at outset, but BMI did not affect subsequent weight gain. Weight gain was slower in the later sessions of CBT (6-10) if the patient had higher anxiety levels. Broadly, unhealthy initial eating attitudes and perfectionism levels were related to slower weight gain across the course of this early part of treatment. Higher levels of ‘feeling’-related cognitions were associated with less weight gain in the latter stages of the treatment period examined (sessions 6 – 10).

Conclusions: This research has identified a set of factors that predict poorer weight gain in the course of the early part of CBT for anorexia nervosa. These findings suggest that there may be a potential benefit in modifying CBT to address anxiety and perfectionism earlier in treatment.

2. Introduction

2.1 Background

Identifying which patient characteristics predict the outcome of treatment is a fundamental goal in psychotherapy research. The identification of such predictors should aid in the selection of patients who will or will not respond to treatment, and could lead to hypotheses concerning the reasons for failure of a particular treatment, possibly leading to improvements in these treatments. Such understandings may also lead to the possibility of tailoring treatments to individuals. Such an understanding and improvement would have cost, time and resource benefits for the National
Health Service (NHS). For example, abandoning an ineffective treatment might lessen patient frustration and lower drop out rates amongst patients, and may enable clinicians to create treatment algorithms (e.g., Agras et al., 2000).

In the eating disorders, the outcome of treatments for anorexia nervosa (AN) is particularly poorly understood (e.g., Fairburn & Harrison, 2003). Within a cognitive-behavioural therapy (CBT) model, it is important to understand the relevance of early change (Agras et al., 2000). Given that they are commonly key targets of treatment (Fairburn, 1999), it is necessary to determine the role of early changes in cognitions regarding weight, shape and eating as well as other related beliefs.

2.2 Treatments for anorexia nervosa

2.2.1 The current evidence base

There is still little evidence that CBT (or indeed any psychological treatment) is effective in the treatment of AN (Cooper, 2005). The National Institute of Clinical Excellence (NICE, 2004) recommends psychological treatment that has a focus on eating behaviour and attitudes to weight and shape, as well as wider psychological issues, but do not recommend a particular type of therapy. The lack of relevant outcome studies for AN is perhaps unsurprising, given the many difficulties in conducting treatment with this group of patients, who are difficult to engage in treatment, and who drop out frequently.

Of the evidence that does exist for the efficacy of psychosocial therapies, most comes from case reports or case series (Garner & Garfinkel, 1985). Additional evidence comes from the considerable clinical experience that suggests that a well-
conducted regimen of psychotherapy can play an important role both in ameliorating the symptoms of AN and in preventing relapse (NICE, 2004). A small number of clinical research studies show that treatment can be effective in a proportion of patients with AN. However, although some patients improve symptomatically over time, a substantial proportion continue to have disturbances in body image, disordered eating, and other psychiatric difficulties (Yager, 2006).

2.2.2 Treatment aims

Dieting and weight loss contribute to the development and maintenance of AN, supporting the case that the initial stages of treatment need to focus on weight gain (i.e., aiming eventually to reach a weight at which menses and ovulation occur in females, and where a normal sexual drive and hormone levels are found in males). This weight gain enhances patients’ motivation to cooperate in the restoration of healthy eating patterns and to participate in treatment (Tozzi et al., 2003). The rationale for focusing on initial weight gain in patients is not only to reduce their physical risk, but also to enable patients to reach a point where they are able to function normally again on a cognitive level. The further important target of CBT is to modify patients’ negative ideas and dysfunctional assumptions regarding eating and body shape and weight (Sorrentino et al., 2005), specifically enabling them to understand and change the behaviours and attitudes related to their eating disorder. It is clear that adequate nutrition can facilitate this cognitive change.

In terms of weight gain, there is no absolute weight, body mass index or percentage of body fat that indicates when a patient is ready to begin formal psychotherapy, and there are large individual differences. However, clinical experience shows that
patients often display improved mood, enhanced cognitive functioning, and clear thought processes once they begin to eat at a normal level, even before there is substantial weight gain. Consequently, psychotherapeutic interventions usually begin as soon as the patient is no longer in a medically compromised state (Yager et al., 2006), rather than waiting for the patient to be at a particular weight.

2.3 Characteristics, beliefs and cognitions of responders and non-responders to treatment

It is vital to identify characteristics, beliefs and cognitions in those who do and do not respond to treatment so that clinicians can develop more effective treatment strategies and target treatments more specifically. Past research reveals that there are a number of broad characteristics and treatment variables associated with poor and better outcomes in patients.

2.3.1 Short and long-term predictors of treatment outcome

Predictor studies on AN focus on either the short-term or long-term outcome. No data exist considering differential efficacy of psychotherapeutic treatment for AN by gender, race, ethnicity or cultural group. The following variables have been found to be related to an unfavourable course in the long and short term: chronicity, later age of onset, bulimic subtype, psychiatric comorbidity (depression, anxiety, OCD: Bryant-Waugh et al., 1988; Eckert et al., 1995), low weight at discharge and previous hospitalisation (Kaplan et al., 2001; Steinhausen, 2002), a long interval between onset and hospitalisation or a prolonged episode of the illness (Hall et al., 1984; Fitcher et al., 2006), resistance to change and increased length of stay in residential
treatment (McHugh, 2004), and sexual problems (Fitcher et al., 2006). The studies were carried out across different settings - inpatient, outpatient or mixed.

Clearly, a number of different characteristics have been shown to be associated with good or poor outcome from eating disorder treatment. None of the studies focus on the treatment process, with the majority focusing on demographic patient characteristics. However, it seems likely that cognitions have a key role in the treatment process and their impact on treatment outcome needs to be better understood.

2.3.2 Predictors of outcome from CBT treatment
Agras et al. (2000) examined outcome predictors for cognitive behavioural treatment of bulimia nervosa (BN). They found that participants with poor treatment outcome were characterised by poor social adjustment and a lower BMI, presumably indicating greater dietary restriction. Poor outcome was predicted by a reduction of less than 70% of purging by treatment session 6. Early progress in therapy was the best predictor of outcome. No equivalent research has been conducted on an AN population.

2.4 Weight gain
2.4.1 Rate of weight gain
The first issue in the treatment of AN is to engage and keep patients in treatment and to prevent early dropout. Arguments for a faster rate of weight gain include the desire to keep admissions brief. In favour of a slower rate are considerations of refeeding syndrome (Melchior, 1998) and the risk of engendering overwhelming anxiety and a
reduced ability to cooperate with treatment. The good medium-term results of outpatient treatments with slow weight gain indicate that while weight gain should be focused, it should be slow enough to be ‘assimilated’ both physiologically and psychologically (Herzog et al., 2004).

2.4.2 Patterns of drop-out at initial stages of weight gain and long term shifts

It does appear that in terms of weight gain, a slower rate of change may lead to better long-term outcome. It is of interest that it is generally those of lower weight that appear to gain weight faster, Davies & Jaffa (2005). Hartmann et al. (2007) also showed that patients who were moderately underweight (BMI > 16) had a higher risk of treatment failure than those who were severely underweight. It may be that there was a greater pressure on weight gain in the group initially classified as severely underweight. There does seem to be some link with a poorer prognosis and bulimic symptomology also (Hartmann et al., 2007).

In an inpatient setting the focus is largely physiological (re-feeding), and it is possible that the key to the difference in effect is the focus in outpatient treatment, which is largely psychological and allows the patient a sense of control over weight gain. It is likely that if greater psychological change occurs during treatment, this would increase the chances of long-term maintenance. Therefore, beyond the consideration of predictors of weight gain and responders to treatment, there is a need to consider psychological shifts across treatment in the AN population.
2.5 Cognitive aspects of weight gain

From the few studies conducted, we know that there are many changes in psychological features during weight restoration. The following cognitions and behaviours based around eating, weight and shape have an influence during the period of lowest weight and when weight has been restored - fear of becoming fat, inaccurate perception of one’s own body weight or shape, obsessive preoccupation with food, disturbance of hunger perception, disturbance of satiety perception, maudlin mood, irritated mood, and obsessive pursuit of thinness. Symptoms are likely to be exacerbated by starvation and malnutrition. At the time of lowest weight, almost all the psychological features of AN are present, whereas at a time of weight restoration often these features have lessened or are ameliorated (Inanuma, 2002).

2.5.1. The role of cognitions in eating disorders: Negative Automatic Thoughts, Dysfunctional Assumptions and Core Beliefs

CBT addresses several types and levels of cognitions that are associated with the development and maintenance of disorders. Distinctions are made between how information is organised and processed and its content (Clark & Beck, 1999). One important distinction is that made between automatic thoughts and dysfunctional assumptions. Automatic thoughts are specific to situations and are unplanned thoughts that often reflect dysfunctional content. Dysfunctional assumptions are more stable, cross-situational beliefs that give rise to automatic thoughts and influence the interpretation and response to situations. Dysfunctional assumptions about body appearance, food or eating are believed to be specific to eating disorders (Cooper & Fairburn, 1992). Beliefs that focus on perfectionism are understood to
play an operative secondary role in the development of eating disorders (Fairburn et al., 1999; Shafran, Cooper & Fairburn, 2002).

2.5.2 Cognitive theories

Although research has been conducted on personality characteristics and treatment variables within treatment success, there has been less research examining cognitive styles. We still have a poor understanding of the specific cognitions and cognitive processes that are involved in the maintenance and development of AN. Fairburn et al. (1999) highlight how there has been an absence of studies looking at beliefs and attitudes within eating disorders, despite their significance to cognitive behavioural models. Existing cognitive-behavioural formulations of the eating disorders treat dysfunctional assumptions (immediate beliefs that can often be stated as “if….then…” or “should” statements) about eating, weight and shape as central to the maintenance of these problems (Fairburn, Cooper & Shafran, 2003; Vitousek, 1996). Studies reveal how cognitions pertaining to body size and eating are biased in selectively processing information related to fatness/thinness, dieting and control of food intake or body weight. Understanding information processing biases may assist the clinician in understanding a range of psychopathological features of AN, including denial, resistance to treatment and misinterpretation of therapeutic interventions (Williamson, 1999). These models do not consider the developmental perspective, nor do they incorporate recent findings related to negative self-beliefs and the potential role of schema driven maintenance processes (Cooper, 2003).
2.5.3 Cognitive processes - Negative thoughts and dysfunctional assumptions

To gain a clearer understanding of the cognitions involved in AN, the distinction between this condition and normal dieting has been explored in various studies. It is still not totally clear, in cognitive terms, what distinguishes those with AN from dieters who do not have eating disorders. It may be that such differences are more quantitative (differences in the frequency or number of cognitions) than qualitative. For example, Cooper & Fairburn (1992) found that patients with AN were distinguished from dieters by a greater number of negative thoughts about eating.

As an alternative to the hypothesis that eating disordered individuals are differentiated from dieters simply in terms of the quantity of particular cognitions which they experience, some authors have suggested that the differences pertain to the importance or relevance of such thoughts to the individual (Garner & Bemis, 1982; Geller et al., 1997; Geller et al., 1998). Hinrichsen et al.’s. (2005) measure, the TAQ-ED, considers assumptions based around the perceived consequences of expressing negative feelings; assumptions about the world and assumptions about one’s body. Hinrichsen et al. (2005) suggests that patients are more likely to feel dissatisfied with their body if they believe being a certain weight has negative social consequences (e.g., criticism, humiliation or rejection) suggesting a link between maladaptive assumptions about one’s feelings and social-emotional functioning. It may therefore be helpful to focus on specific contents when attempting to alter self-statements in this group of patients.

Although there has been research on the connection between assumptions and beliefs and eating disorder, there has not been any published research on the relationship
between these factors and weight gain within AN. In the current study, the use of the TAQ-ED will enable exploration of how more global assumptions are related to eating behaviour and weight restoration within treatment.

2.5.4 The role of Core Beliefs and Schemas

"Schemas are organizations of knowledge that are constructed through interaction with the environment and reflect a person’s construct of an object or event" (Stein, 1996). Once formed, they are stored in long-term memory and act as the foundation for the processing of subsequent interactions with the environment (Young et al., 2003). Leung et al. (1999) demonstrate a clear link between core beliefs and eating psychopathology. Stein (1996) posits that “a limited collection of positive self-schemas available in memory, in combination with a chronic and inflexibly accessible body-weight schema lead to the disordered behaviours associated with AN”.

Woolrich et al. (2006) found that participants with AN had significantly more negative core-self beliefs than dieters and controls. These findings support previous theoretical hypotheses (e.g., Wolff & Serpell, 1998) and clinical descriptions (Bruch, 1973). Findings in a normal sample indicate that while underlying assumptions predict the presence of eating disorder symptoms, negative self-beliefs predict symptoms of generic emotional distress (Cooper & Todd, 1997). Cooper et al. (1998) suggest that both underlying assumptions about weight, shape and eating, in combination with negative self-beliefs, may distinguish patients with AN from dieters.
While there is preliminary evidence for the general existence of negative self-beliefs in AN (Turner & Cooper, 2002), it is still not clear whether this specific content is of central importance in the disorder. Existing treatment approaches for the eating disorders largely focus on modifying negative assumptions about eating, shape and weight and core beliefs/schemas (Fairburn, Marcus & Wilson, 1993; Bonifazi et al., 2000; Cooper et al., 1997; Turner & Cooper, 2002; Waller et al., 2007). Hinrichsen et al.'s. (2005) recent research suggests that it is likely to be necessary to challenge dysfunctional assumptions that link these two levels of cognition. In therapy, this is likely to involve both cognitive restructuring and the use of behavioural experiments in the testing of assumptions (Bennett-Levy et al., 2004).

2.6 The Behavioural element of the cognitive behavioural model

It has been suggested that dietary restriction may function to compensate for the cognitive and emotional distress associated with negative self-beliefs (Turner & Cooper, 2002). Participants with AN reported using specific behaviours, particularly "not eating enough" to try to reduce the cognitive and emotional distress (e.g., mood; self esteem) associated with their negative self-beliefs. The results suggest a functional link in AN between 'not eating enough' and negative self-beliefs (Woolrich et al., 2006). Waller et al. (2007) suggest that dietary restriction is associated with 'primary avoidance of affect' in that it is an attempt to prevent emotion being experienced. In this way it is important to consider the role of behaviour change in the process of CBT as well as the role of emotions.
2.7 Comorbidity within the eating disorders

Outcome predictors in the short-term treatment of AN reveal a level of inconsistency in terms of the influence of co-morbidity. A very high percentage of treatment-seeking patients with eating disorders report a lifelong history of unipolar depression (Cooper & Hunt, 1998; Herpeitz-Dahlmann et al., 2007). Nutritional insufficiency and weight loss often predispose patients to symptoms of depression (Keys et al., 1950). Tchanturia et al. (2004) report that with an increase in weight there was a decrease in self-reported anxiety and depression. However, several studies suggest that the presence of co morbid depression at initial presentation has minimal or no predictive value for treatment outcome (Keel & Mitchell, 1997). Depressed individuals with an eating disorder often experience greater levels of anxiety (Kaye et al., 2004). Lifetime prevalence rates for anxiety disorders appear to be higher for patients with both AN and BN, but rates specifically for AN vary (Godart et al., 2002). Although there is no clear evidence that co morbid anxiety disorders impact significantly on eating disorder treatment outcome, such co morbid problems should be considered within treatment (Yager, 2006). The current study considers the role of both depression and anxiety on weight gain within the early stages of treatment.

2.8 Research aims

The course of treatment for patients is not consistently smooth, and it is therefore hard to know when a patient is at risk for failure and whether a change in intervention is required.

The proposed causal relationship between cognition and eating disordered weight gain and behaviours (bingeing, and the compensatory behaviours purging and
laxative use) remains largely untested in patients with eating disorders. Existing treatment studies have not measured any of the specific cognitive constructs, such as negative self-beliefs, highlighted in Fairburn et al’s (2003) paper. The current study aims to answer some of the unaddressed questions of previous research. The main aim is to examine whether various emotional and cognitive variables are clinically useful predictors of weight gain and behaviour change in the initial stages of treatment of AN. A second aim is to examine whether a change in dysfunctional assumptions is associated with weight gain. In theory, cognitive behavioural approaches should be effective in shifting these beliefs and where weight gain is accompanied by improvements in cognitions, this should provide the motivation for further eating and weight gain (Woolrich et al., 2006).

2.8.1 Research questions

1. Do eating related cognitions (at the negative automatic thought, dysfunctional assumption and core belief/schema level) predict weight gain and behaviour change in the initial stages of treatment of AN (first 10 sessions)?

2. Do anxiety and depression predict weight gain in the initial stages of treatment of AN?

3. Is weight gain in the early stages of treatment associated with positive changes in eating-related cognitions over the same time period?

2.8.2 Hypotheses

1. There will be an increase in weight over the 10 sessions and a decrease in behaviours over this time period (bingeing, purging, laxative use).
2. Higher levels of negative eating-related cognitions at the beginning of treatment will predict less weight gain over the first ten sessions of treatment for anorexia nervosa.

3. Higher levels of negative mood (depression, anxiety) at the beginning of treatment will predict less weight gain over the first ten sessions of treatment for anorexia nervosa.

4. Over the initial part of treatment, positive changes in behavioural and biological symptoms (e.g., weight gain) will be associated with reductions in eating-related cognitions.

5. Positive changes in cognitions (dysfunctional assumptions) over time (i.e. a reduction) will be associated with more positive levels of eating related cognitions, core beliefs and mood.

3. Method

3.1 Research Design

The study had a mixed comparative and correlational design using prospective, repeated-measures data, drawn from a clinical cohort of patients with AN.

3.2 Participants

Potential participants included all patients referred to a specialist eating disorder service who had a diagnosis of anorexia nervosa (binge/purging and restrictive subtypes) or atypical anorexia nervosa (e.g. someone who meets all the criteria for AN except that they have not had amenorrhea for all of the last 3 months) at assessment and who were offered cognitive-behavioural therapy (CBT). Other
therapies are offered within the service; however the majority of patients referred receive CBT. The study was carried out within the outpatient department of the service; the service also includes inpatients and a day patient service. Therapy was conducted by 6 clinicians working in the department, 5 were clinical psychologists and one was a nurse with specialist training in CBT. All clinicians were supervised by the leading clinical psychologist in the service, ensuring consistency amongst practice.

Exclusion criteria included patients who were under 18 years, experienced learning disabilities, showed an active psychotic condition or severe physical illness, were detained under the mental health act or were receiving treatment other than CBT.

It was estimated based on existing service figures that 50/60 patients were likely to receive CBT during the study time frame. Overall dropout rate based on existing figures within the service is 20-30%. Existing audit figures show that the mean weight gain in the first 10 sessions (10 weeks) within the AN population in the service is 0.8 BMI points.

In total, 45 patients took part in the study. Drop-out rate was 16% (N = 7). Participants consisted of 22 diagnosed with restrictive subtype AN, 8 with binge-purge subtype and 15 with atypical AN. Considering the transdiagnostic approach to eating disorder behaviours (Fairburn et al., 2003), it was appropriate to group these patients together. Participants had a mean age of 20.8 years (s.d=5.6, minimum=20, maximum=42) and a mean body mass index (BMI) of 16.8 (s.d=1.8, minimum=12.8, maximum=21.6).
3.3 Measures

Three measures were used to assess patients eating attitudes, assumptions around their body, the world and feelings, and mood state (anxiety and depression). The following clinical measures are administered routinely in the clinic, and take a total of approximately 30 minutes to complete on each occasion. Each has been validated clinically and psychometrically.

3.3.1 Eating Disorder Examination – Questionnaire Version EDE-Q (Fairburn & Beglin, 1994)

The EDE-Q (Appendix 1) measures negative self-perceptions of weight, shape and eating. The questionnaire includes 36 items looking at the frequency and severity of eating behaviours and related thought patterns and feelings. These are considered over the last 28 days and are rated by patients on a 7-point rating scale. Higher scores indicate higher levels of eating disorder psychopathology. There are 4 subscales, which provide a profile of the individual’s eating disorder pathology (restraint subscale; weight concern; eating concern; shape concern). Information about behaviours associated with the eating disorders (e.g., episodes of bingeing, vomiting, laxative and diuretic misuse and excessive exercise) is also available.

The EDE-Q is derived from the Eating Disorder Examination (EDE; Fairburn & Cooper, 1993). Comparable results between the EDE and EDE-Q have been found across the eating disorder population, general clinical and non-clinical populations. The only exception is that the EDE-Q tends to yield an over-estimation of the number of objective bulimic episodes that an individual has experienced (Wilfley et
al., 1997). The questionnaire has been shown to have acceptable internal consistency (0.78 – 0.93) (Luce & Crowther, 1999; Peterson et al., 2007) and good test-retest reliability (Luce & Crowther, 1999).

3.3.2 Testable Assumptions Questionnaire – Eating Disorders TAQ-ED revised (Hinrichsen, Garry & Waller, 2005)

The TAQ-ED (Appendix 2) measures testable dysfunctional assumptions that are frequently reported by patients with eating disorders (i.e., beliefs about the impact of eating, weight and shape) and contains 20 items. The dysfunctional assumptions fall into three groups - dysfunctional assumptions about the world, body and feelings. Participants rate each item on a five-point Likert scale, ranging from 1 (‘do not agree’) to 5 (‘totally agree’). Lower scores indicate a lower level of belief in the relevant assumption, whereas higher scores indicate a greater level of belief in the assumption.

The TAQ-ED revised is derived from the TAQ-ED. The 12-item TAQ-ED has been found to have acceptable psychometric properties (Cronbach’s alpha assumptions about the world 0.83; body 0.71; feelings 0.55).

3.3.3 Brief Symptom Inventory BSI (Derogatis, 1993; Derogatis & Melisaratos, 1983)

The BSI (Appendix 3) was developed as a short version of the Symptom Checklist-90 (SCL-90, Derogatis et al., 1974; Derogatis et al., 1976), a well-validated measure of a range of psychiatric symptoms. The BSI consists of 53 items, which make up nine subscales: Somatization, Obsessive-Compulsive Behaviour, Interpersonal
Sensitivity, Depression, Anxiety, Hostility, Phobic Anxiety, Paranoid Ideation, and Psychoticism. The measure can also generate a Global Severity Index, which is a measure of overall symptom severity on all subscales. Participants rate each item on a five-point Likert scale, ranging from 1 (‘Not at all’) to 5 (‘Extremely’). Lower scores indicate a lower level of psychopathology, whereas higher scores indicate a greater level of psychopathology. Cronbach’s alpha levels are very good, ranging from 0.71 to 0.85 (Derogatis, 1993). Test-retest reliabilities for the BSI subscales range from 0.68 to 0.91 (Derogatis, 1993). Convergent validity has been established with the Minnesota Multiphasic Personality Inventory (Derogatis et al., 1976). The study suggests that reducing the length of the SCL-90 to the 53-item BSI has not substantially reduced validity.

3.3.4 Young Schema Questionnaire: Short Form Version 3 (YSQ-S; Young, 1998)
The YSQ-S (Appendix 4) is a 75-item self-report questionnaire measuring levels of 15 core beliefs (subscales). It was developed from the 205-item Young Schema Questionnaire: Long Form (YSQ; Young & Brown, 1994), consisting of 16 subscales.

The questionnaire measures the following schemas:

*Emotional Deprivation*

Expectation that one's desire for a normal degree of emotional support will not be adequately met by others.
Abandonment/instability
The perceived instability or unreliability of those available for support and connection.

Mistrust/abuse
The expectation that others will hurt, abuse, humiliate, cheat, lie, manipulate, or take advantage.

Social isolation/alienation
The feeling that one is isolated from the rest of the world, different from other people, and/or not part of any group or community.

Defectiveness/unlovability
The feeling that one is defective, bad, unwanted, inferior, or invalid in important respects; or that one would be unlovable to significant others if exposed.

Failure to achieve
The belief that one has failed, will inevitably fail, or is fundamentally inadequate relative to one's peers, in areas of achievement (school, career, sports, etc.).

Practical incompetence/dependence
Belief that one is unable to handle one's everyday responsibilities in a competent manner, without considerable help from others (e.g., take care of oneself, solve daily problems, exercise good judgment, tackle new tasks, make good decisions).
**Vulnerability to harm/illness**

Exaggerated fear that imminent catastrophe will strike at any time and that one will be unable to prevent it.

**Enmeshment/underdeveloped self**

Excessive emotional involvement and closeness with one or more significant others (often parents), at the expense of full individuation or normal social development.

**Subjugation**

Excessive surrendering of control to others because one feels coerced - usually to avoid anger, retaliation, or abandonment.

**Self-sacrifice**

Excessive focus on voluntarily meeting the needs of others in daily situations, at the expense of one's own gratification.

**Emotional inhibition**

The excessive inhibition of spontaneous action, feeling, or communication - usually to avoid disapproval by others, feelings of shame, or losing control of one's impulses.

**Unrelenting standards/hypercriticalness**

The underlying belief that one must strive to meet very high-internalized standards of behaviour and performance, usually to avoid criticism.
Entitlement/superiority

The belief that one is superior to other people; entitled to special rights and privileges; or not bound by the rules of reciprocity that guide normal social interaction.

Insufficient self-control/self-discipline

Pervasive difficulty or refusal to exercise sufficient self-control and frustration tolerance to achieve one's personal goals, or to restrain the excessive expression of one's emotions and impulses.

Participants are asked to rate how much each statement describes them, on a six-point Likert scale (‘completely untrue of me’ to ‘describes me perfectly’). Higher scores indicate a greater chance of the core belief in question being active in the individual.

The YSQ and YSQ-S have been found to have good internal reliability, internal consistency and discriminant validity. Waller et al. (2001) found the YSQ-S to have good internal reliability, internal consistency and discriminant validity, but acknowledged the need for test-retest reliability analysis. Stopa et al. (2001) compared the YSQ-L with the YSQ-S and found the latter to be a reasonable alternative to the longer version. Wellburn et al. (2002) found the YSQ-S to have good construct validity and good internal consistency within each of the 15 subscales.
3.4 Procedure

Approval was gained from the local Research and Development department for the study to be considered an audit, as it involves the utilisation of data that are already gathered in routine clinical practice. Therefore, it was not necessary to submit this proposal for LREC approval (Appendix 6). Individuals were given a questionnaire pack including the questionnaires measuring eating pathology, related dysfunctional assumptions and schemas (core beliefs) and mood, as part of routine audit within the service. All four questionnaires were given at the start of therapy (time 1) and the dysfunctional assumptions questionnaire (TAQ-R) was also given at session 10 (time 2). The cognition change scores were calculated by subtracting the scores on the TAQ-R at time 1 (session 1) from the scores at time 2 (session 10). They were informed that the questionnaires might be used for ongoing research and were given the opportunity to opt out if they did not wish their questionnaires to be used in this way (Appendix 7). Their height was measured at assessment and their weight was measured weekly throughout treatment. Information on frequency of binges and purging behaviours is also collected weekly as part of clinical routine. Information on the relevant weight and height at session 1, 6 and 10 was gathered through examination of the clinical notes. Measures of objective binges, vomiting and laxatives were collected in the same manner. Information about diagnosis and demographics was gathered through analysis of clinical notes. The normal duration of treatment for AN is between 20 to 40 sessions. Data collection commenced in March 2007 and finished in April 2008. All data were recorded and stored anonymously.
3.5 Data analysis

Prior to analysis, all data were analysed for skewness and normality by examination of histograms and the use of Kolomogrov-Smirnov tests. Comparisons were made between the clinical data and standardized non-clinical comparison groups for all items on the 4 questionnaires utilised. Patterns in completers versus dropouts in treatment for weight, behaviours, eating related cognitions, comorbid psychopathology, and core beliefs were examined using Mann Whitney tests.

Repeated-measures ANOVA was used to examine change in weight and behaviours (bingeing, purging and laxative use) across time (session 1-10: Hypothesis 1). Pearson’s correlations were used to examine the associations between weight change and cognitions (eating/weight/shape beliefs on the EDE-Q; dysfunctional assumptions on the TAQ-ED and core beliefs/schemas on the YSQ-S: Hypothesis 2) and mood (hypothesis 3). Pearson’s correlations were used to examine the association between weight gain and changes in cognitions (TAQ-ED) over time (Hypothesis 4). Pearson’s correlations were also used to examine the associations between change in cognitions (TAQ-ED) across time and beliefs (EDE-Q), core beliefs (YSQ-S) and mood (BSI: Hypothesis 5).

4. Results

4.1 Missing data

Three of the participants did not complete the YSQ and the EDE-Q. One further participant did not complete the BSI. Overall, 6.3% of the data points were
missing. Missing data points were replaced with case means. This approach was used as it reduces the risk of Type II errors (incorrectly accepting the null hypothesis – false negative) without increasing the likelihood of a Type I error (incorrectly rejecting the null hypothesis – false positive).

4.2 Distribution of data

Kolmogorov-Smirnov tests showed all data were normally distributed apart from one subscale of the TAQ-ED (Table 1), two subscales of the BSI (Table 2) and two subscales of the YSQ-S (Table 3). Some such cases would be expected to depart from normality due to chance. Therefore, given the relatively well-distributed nature of the data as whole, parametric tests were used in all further analysis.

4.3 Comparison to normal population scores on the EDE-Q

Table 1 shows results from the Kolmogorov-Smirnov tests. The table also shows that the mean scores on every subscale of the EDE-Q in the clinical sample were substantially higher than the non-clinical sample (US sample matched for age and gender) (Non clinical sample, restraint: M=1.62; eating concern: M=1.11; shape concern: M=2.27; weight concern: M=1.97, Luce et al., 2008). Results for an Australian comparison non-clinical group are similar to the US group (restraint: M=1.29; eating concern: M=0.87; shape concern: M=2.29; weight concern: M=1.89, Mond et al., 2005). To date, no non-clinical norms are available for the TAQ-ED.

Table 1 also displays the mean weight, height and BMI of all participants. The mean BMI of 16.79 is below the cut-off of 17.5 for an AN diagnosis. The table shows the number of patients (N) whom data were collected from and who filled out
questionnaires at time point 1. Questionnaire data is presented in the relevant subcategories. Numbers who completed the TAQ-ED (N=42) were slightly less than those completing the EDE-Q (N=44).

Table 1: Mean (standard deviation) and Kolmogorov-Smirnov tests showing the distribution of weight, height, BMI, EDE-Q total and TAQ-ED total scores.

<table>
<thead>
<tr>
<th>Eating</th>
<th>N</th>
<th>Mean (s.d.)</th>
<th>KS test</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Weight (time 1)</td>
<td>45</td>
<td>45.41 (5.96)</td>
<td>0.64</td>
<td>NS</td>
</tr>
<tr>
<td>Height</td>
<td>45</td>
<td>1.64 (0.07)</td>
<td>0.78</td>
<td>NS</td>
</tr>
<tr>
<td>BMI</td>
<td>45</td>
<td>16.79 (1.80)</td>
<td>0.72</td>
<td>NS</td>
</tr>
<tr>
<td>EDE-Q</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Restraint</td>
<td>44</td>
<td>3.48 (1.70)</td>
<td>0.94</td>
<td>NS</td>
</tr>
<tr>
<td>Eating Concern</td>
<td>44</td>
<td>3.03 (1.35)</td>
<td>1.04</td>
<td>NS</td>
</tr>
<tr>
<td>Shape Concern</td>
<td>44</td>
<td>3.74 (1.30)</td>
<td>0.68</td>
<td>NS</td>
</tr>
<tr>
<td>Weight Concern</td>
<td>44</td>
<td>3.01 (1.54)</td>
<td>0.84</td>
<td>NS</td>
</tr>
<tr>
<td>TAQ-ED time 1</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Body</td>
<td>42</td>
<td>2.73 (0.91)</td>
<td>0.94</td>
<td>NS</td>
</tr>
<tr>
<td>World</td>
<td>42</td>
<td>1.86 (0.83)</td>
<td>1.50</td>
<td>0.02</td>
</tr>
<tr>
<td>Feelings</td>
<td>42</td>
<td>2.49 (1.19)</td>
<td>1.20</td>
<td>NS</td>
</tr>
</tbody>
</table>

Note: NS=non-significant

4.3.1 Comparison to normal population scores on the BSI.

Table 2 shows results from the Kolmogorov-Smirnov tests. The table also shows that, as expected, the mean scores on the BSI clinical sample were higher than the non clinical norms, matched for age and gender (US population) (Non clinical
sample, Anxiety: M=1.82; Somatization: M=.94; Psychoticism: M=1.24; Paranoid ideation: M=1.21; OCD: M=1.60; Hostility: M=1.23; Phobic anxiety: M=.91; Depression: M=1.90; Interpersonal sensitivity: M=1.66, Derogatis, 1993).

Table 2 also shows the number (N) of people who completed the subcategories of the BSI (42).

Table 2: Mean (standard deviation) and Kolmogorov-Smirnov test showing the distribution of BSI subscales.

<table>
<thead>
<tr>
<th>BSI</th>
<th>N</th>
<th>Mean (s.d.)</th>
<th>KS test</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>Z</td>
<td></td>
</tr>
<tr>
<td>Anxiety</td>
<td>42</td>
<td>2.10 (1.09)</td>
<td>1.03</td>
<td>NS</td>
</tr>
<tr>
<td>Somatization</td>
<td>42</td>
<td>1.62 (0.76)</td>
<td>0.77</td>
<td>NS</td>
</tr>
<tr>
<td>Psychoticism</td>
<td>42</td>
<td>1.51 (1.07)</td>
<td>0.86</td>
<td>NS</td>
</tr>
<tr>
<td>Paranoid ideation</td>
<td>42</td>
<td>1.30 (0.87)</td>
<td>0.86</td>
<td>NS</td>
</tr>
<tr>
<td>OCD</td>
<td>42</td>
<td>2.12 (1.01)</td>
<td>1.28</td>
<td>NS</td>
</tr>
<tr>
<td>Hostility</td>
<td>42</td>
<td>1.34 (0.65)</td>
<td>1.00</td>
<td>NS</td>
</tr>
<tr>
<td>Phobic Anxiety</td>
<td>42</td>
<td>1.61 (1.10)</td>
<td>1.41</td>
<td>0.04</td>
</tr>
<tr>
<td>Depression</td>
<td>42</td>
<td>2.19 (1.16)</td>
<td>1.03</td>
<td>NS</td>
</tr>
<tr>
<td>Interpersonal</td>
<td>42</td>
<td>2.19 (1.14)</td>
<td>1.63</td>
<td>0.01</td>
</tr>
<tr>
<td>Sensitivity</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Note NS=non-significant

4.3.2 Comparison to normal population scores on the YSQ-S.

Table 3 shows results from the Kolmogorov-Smirnov tests. The table also displays the mean scores on the YSQ-S clinical sample which were higher on all
subcategories of the questionnaire than the non-clinical norms, matched for age and
gender (Waller et al., 2001). The table also shows the number (N) of patients who
completed the questionnaire. This number varies across scales because some
individuals did not complete all items on the scale.

Table 3: **Mean (standard deviation) and Kolmogorov-Smirnov test showing the distribution of YSQ-S subscales.**

<table>
<thead>
<tr>
<th>YSQ-S</th>
<th>N</th>
<th>Mean (s.d.)</th>
<th>KS test</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>Z</td>
</tr>
<tr>
<td>Emotional deprivation</td>
<td>41</td>
<td>2.38 (1.37)</td>
<td>1.41</td>
</tr>
<tr>
<td>Abandonment</td>
<td>40</td>
<td>2.60 (1.36)</td>
<td>1.00</td>
</tr>
<tr>
<td>Mistrust</td>
<td>40</td>
<td>2.90 (1.24)</td>
<td>0.57</td>
</tr>
<tr>
<td>Social Isolation/ Alienation</td>
<td>41</td>
<td>3.24 (1.66)</td>
<td>0.81</td>
</tr>
<tr>
<td>Defectiveness/ unlovability</td>
<td>41</td>
<td>2.95 (1.40)</td>
<td>0.76</td>
</tr>
<tr>
<td>Failure to Achieve</td>
<td>41</td>
<td>2.90 (1.58)</td>
<td>1.02</td>
</tr>
<tr>
<td>Practical Incompetence/ Dependence</td>
<td>41</td>
<td>2.35 (0.95)</td>
<td>1.11</td>
</tr>
<tr>
<td>Vulnerability to Harm or Illness</td>
<td>41</td>
<td>2.62 (1.12)</td>
<td>0.93</td>
</tr>
<tr>
<td>Enmeshment</td>
<td>41</td>
<td>2.02 (1.17)</td>
<td>1.37</td>
</tr>
<tr>
<td>Subjugation</td>
<td>41</td>
<td>2.67 (1.34)</td>
<td>0.78</td>
</tr>
<tr>
<td>Self-sacrifice</td>
<td>41</td>
<td>3.40 (1.35)</td>
<td>1.13</td>
</tr>
<tr>
<td>Emotional Inhibition</td>
<td>41</td>
<td>3.11 (1.39)</td>
<td>0.67</td>
</tr>
<tr>
<td>Unrelenting</td>
<td>41</td>
<td>3.92 (1.16)</td>
<td>0.76</td>
</tr>
</tbody>
</table>
Standards

| Entitlement/ 
Superiority |       |     |   | Non-Significat |
<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>41</td>
<td>2.65 (0.73)</td>
<td>0.64</td>
<td>NS</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Insufficient Self-Control/Self-Discipline</th>
<th></th>
<th></th>
<th></th>
<th>Non-Significat</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>40</td>
<td>2.91 (1.11)</td>
<td>1.10</td>
<td>NS</td>
</tr>
</tbody>
</table>

Note: NS = non-significant

4.4 Patterns in completers versus dropouts of treatment for weight, behaviours and eating related cognitions.

Mann Whitney tests were used to examine the difference in weight, ED behaviours, the EDE-Q subscales and TAQ—ED subscales across completers of treatment (completers) and those who dropped out of treatment (dropouts). Table 4 shows that there were no significant differences between the two groups with the exception of weight concern, where there were higher levels of weight concern in the completers group. None of these factors (weight, behaviours or questionnaire responses) were predictive of dropout.
Table 4: **Mean (standard deviation) of weight and behaviour measures for the completers and drop out-group.**  

<table>
<thead>
<tr>
<th>Group</th>
<th>Completers (N=38)</th>
<th>Drop-outs (N=7)</th>
<th>Z score</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>BMI</strong></td>
<td>16.75 (1.85)</td>
<td>17.04 (1.59)</td>
<td>.501</td>
<td>NS</td>
</tr>
<tr>
<td><strong>Weight 1</strong></td>
<td>45.09 (5.74)</td>
<td>47.11 (7.34)</td>
<td>.674</td>
<td>NS</td>
</tr>
<tr>
<td><strong>Binge 1</strong></td>
<td>1.08 (3.25)</td>
<td>1.29 (2.63)</td>
<td>.752</td>
<td>NS</td>
</tr>
<tr>
<td><strong>Vomit 1</strong></td>
<td>1.55 (3.59)</td>
<td>.29 (.76)</td>
<td>.538</td>
<td>NS</td>
</tr>
<tr>
<td><strong>Laxative 1</strong></td>
<td>3.87 (15.81)</td>
<td>.00 (.00)</td>
<td>1.00</td>
<td>NS</td>
</tr>
</tbody>
</table>

**EDE-Q**

| Restraint   | 3.58 (1.72) | 2.97 (1.56) | .838 | NS   |
| Eating concern | 3.13 (1.37) | 2.51 (1.24) | 1.19 | NS   |
| Shape concern | 3.87 (1.33) | 3.02 (.84) | 1.80 | NS   |
| Weight concern | 3.20 (1.57) | 2.00 (.84) | 2.05 | 0.05 |

**TAQ-ED**

| Body       | 2.76 (.93) | 2.44 (.79) | .752 | NS   |
| World      | 1.90 (.86) | 1.50 (.49) | .713 | NS   |
| Feelings   | 2.55 (1.18) | 1.95 (1.38) | 1.16 | NS   |

*Note: NS=non-significant*
4.4.1 Patterns in completers versus dropouts from treatment across comorbid psychopathology.

Table 5 shows that there were no significant differences between the two groups and scores on the BSI, with the exception of interpersonal sensitivity, where there were higher levels of interpersonal concern within the completers group. Again, this result might be due to chance (given the number of comparisons made), rather than highlighting any clinical significance.

**Table 5: Mean (standard deviation) of BSI scores for the completers and drop out-group.**

<table>
<thead>
<tr>
<th>Group</th>
<th>Completers (N=38)</th>
<th>Drop-outs (N=7)</th>
<th>Z score</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>BSI</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Anxiety</td>
<td>2.18 (1.12)</td>
<td>1.33 (.30)</td>
<td>1.22</td>
<td>NS</td>
</tr>
<tr>
<td>Somatization</td>
<td>1.64 (.79)</td>
<td>1.43 (.37)</td>
<td>.62</td>
<td>NS</td>
</tr>
<tr>
<td>Psychoticism</td>
<td>1.54 (1.09)</td>
<td>1.20 (.99)</td>
<td>.67</td>
<td>NS</td>
</tr>
<tr>
<td>Paranoid ideation</td>
<td>1.28 (.89)</td>
<td>1.50 (.66)</td>
<td>.56</td>
<td>NS</td>
</tr>
<tr>
<td>OCD</td>
<td>2.19 (1.03)</td>
<td>1.50 (.58)</td>
<td>1.55</td>
<td>NS</td>
</tr>
<tr>
<td>Hostility</td>
<td>1.36 (.69)</td>
<td>1.20 (.16)</td>
<td>.22</td>
<td>NS</td>
</tr>
<tr>
<td>Phobic anxiety</td>
<td>1.66 (1.13)</td>
<td>1.10 (.12)</td>
<td>.70</td>
<td>NS</td>
</tr>
<tr>
<td>Depression</td>
<td>2.24 (1.17)</td>
<td>1.80 (1.13)</td>
<td>.80</td>
<td>NS</td>
</tr>
<tr>
<td>Interpersonal sensitivity</td>
<td>2.30 (1.15)</td>
<td>1.19 (.24)</td>
<td>1.99</td>
<td>0.05</td>
</tr>
</tbody>
</table>

*Note: NS = non-significant*
4.4.2 Patterns in completers versus dropouts of treatment across cognitions.

Table 6 shows that there were no significant differences between completers and dropouts on any of the scales of the YSQ-S. This means that there were no differences on YSQ subscales between those who did and those who did not remain in treatment.
<table>
<thead>
<tr>
<th></th>
<th>Group</th>
<th>Completers (N=38)</th>
<th>Drop-outs (N=7)</th>
<th>Z score</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>YSQ-S</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Emotional</td>
<td></td>
<td>2.40 (1.39)</td>
<td>2.25 (1.37)</td>
<td>.199</td>
<td>NS</td>
</tr>
<tr>
<td>Abandonment</td>
<td></td>
<td>3.05 (1.40)</td>
<td>2.13 (.39)</td>
<td>1.38</td>
<td>NS</td>
</tr>
<tr>
<td>Mistrust</td>
<td></td>
<td>2.93 (1.28)</td>
<td>2.60 (.91)</td>
<td>.57</td>
<td>NS</td>
</tr>
<tr>
<td>Social alienation</td>
<td></td>
<td>3.35 (1.71)</td>
<td>2.30 (.53)</td>
<td>1.10</td>
<td>NS</td>
</tr>
<tr>
<td>Defectiveness/unlovability</td>
<td></td>
<td>2.97 (1.45)</td>
<td>2.75 (.91)</td>
<td>.18</td>
<td>NS</td>
</tr>
<tr>
<td>Failure to achieve</td>
<td></td>
<td>2.95 (1.63)</td>
<td>2.35 (1.00)</td>
<td>.51</td>
<td>NS</td>
</tr>
<tr>
<td>Practical incompetence/dependence</td>
<td></td>
<td>2.36 (.98)</td>
<td>2.30 (.74)</td>
<td>0.66</td>
<td>NS</td>
</tr>
<tr>
<td>Vulnerability to harm illness</td>
<td></td>
<td>2.32 (1.14)</td>
<td>1.70 (.90)</td>
<td>.84</td>
<td>NS</td>
</tr>
<tr>
<td>Enmeshment</td>
<td></td>
<td>1.95 (1.12)</td>
<td>2.65 (1.58)</td>
<td>1.07</td>
<td>NS</td>
</tr>
<tr>
<td>Subjugation</td>
<td></td>
<td>2.72 (1.36)</td>
<td>2.20 (1.21)</td>
<td>.68</td>
<td>NS</td>
</tr>
<tr>
<td>Self Sacrifice</td>
<td></td>
<td>3.46 (1.36)</td>
<td>2.90 (1.35)</td>
<td>.839</td>
<td>NS</td>
</tr>
<tr>
<td>Emotional Inhibition</td>
<td></td>
<td>3.11 (1.46)</td>
<td>3.05 (.50)</td>
<td>0.066</td>
<td>NS</td>
</tr>
<tr>
<td>Unrelenting Standards</td>
<td></td>
<td>3.98 (1.20)</td>
<td>3.35 (.30)</td>
<td>1.26</td>
<td>NS</td>
</tr>
<tr>
<td>Entitlement/Superiority</td>
<td></td>
<td>2.64 (.73)</td>
<td>2.70 (.77)</td>
<td>.04</td>
<td>NS</td>
</tr>
</tbody>
</table>
4.5 Changes in weight and behavioural patterns across the early part of treatment

Repeated-measures ANOVAs were used to examine the difference in weight and frequency of behaviours across session 1, 6 and 10 of treatment. The ANOVA uses the raw figures at time point 1, 6 and 10 to calculate change figures which are represented in Table 7; the figures show the extent of the change. Table 7 shows that there was a significant change in weight across session 1 to 10 (hypothesis 1). The positive score indicates weight gain. There was more weight gain in the first six sessions (1.01kg) than in the next four (0.63kg). Post hoc tests showed that there were statistically significant weight gains during each time period (LSD test; P < .01 for each time period).

There was no significant change in any of the behaviours (binge ing, purging and laxative use) across sessions 1 – 10 (hypothesis 1). The negative scores indicate a non-significant reduction in behaviours. The number of patients who displayed these behaviour traits in the sample was low, and therefore results need to be interpreted with caution. For the remaining results, weight gain will be the measure of behavioural change examined, and the other behaviours (binge ing, purging and laxative use) will not be considered further.
Table 7: Repeated Measures ANOVA showing change in weight and behaviours across time (session 1, 6 and 10)

<table>
<thead>
<tr>
<th>Change in behaviours</th>
<th>Number</th>
<th>T1-10</th>
<th>T1-6</th>
<th>T6-10</th>
<th>ANOVA F</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Weight</td>
<td>38</td>
<td>1.64</td>
<td>1.01</td>
<td>0.63</td>
<td>8.60</td>
<td>.001</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(2.48)</td>
<td>(1.71)</td>
<td>(1.30)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Binges</td>
<td>13</td>
<td>-2.08</td>
<td>-1.0</td>
<td>-1.38</td>
<td>0.94</td>
<td>NS</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(4.52)</td>
<td>(3.19)</td>
<td>(2.36)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Purges</td>
<td>10</td>
<td>-4.10</td>
<td>-1.70</td>
<td>-2.40</td>
<td>3.97</td>
<td>NS</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(4.38)</td>
<td>(5.54)</td>
<td>(6.38)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Laxatives</td>
<td>5</td>
<td>-18.20</td>
<td>-11.20</td>
<td>-7.00</td>
<td>1.75</td>
<td>NS</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(21.75)</td>
<td>(14.46)</td>
<td>(7.58)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Note: NS = non-significant

4.5.1 Association between initial eating-related cognitions and early weight change

Table 8 outlines the relationship between weight change and cognitions (hypothesis 2). There were significant negative associations between weight gain across the ten sessions and the EDE-Q restraint, eating concern, shape concern and weight concern scales. This association shows that higher levels of initial eating-related cognitions were associated with less weight gain in the first ten sessions. This pattern was more consistent during the latter part of this element of therapy (sessions 6-10), suggesting that attitudes reflecting restraint and weight concern begin to have their impact some sessions into therapy.

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Table 8: **Pearson’s correlations showing associations of weight change with eating-related cognitions (EDE-Q subscales) and additional related cognitions (TAQ-ED subscales)**.

<table>
<thead>
<tr>
<th>EDE-Q</th>
<th>Weight 1-10</th>
<th>Weight 1-6</th>
<th>Weight 6-10</th>
</tr>
</thead>
<tbody>
<tr>
<td>Restraint</td>
<td>-.384*</td>
<td>-.261</td>
<td>-.408*</td>
</tr>
<tr>
<td>Eating Concern</td>
<td>-.412*</td>
<td>-.333*</td>
<td>-.375*</td>
</tr>
<tr>
<td>Shape Concern</td>
<td>-.508**</td>
<td>-.397*</td>
<td>-.475**</td>
</tr>
<tr>
<td>Weight Concern</td>
<td>-.345*</td>
<td>-.228</td>
<td>-.358*</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>TAQ-ED</th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Body</td>
<td>.130</td>
<td>.041</td>
<td>.175</td>
</tr>
<tr>
<td>World</td>
<td>.095</td>
<td>.033</td>
<td>.123</td>
</tr>
<tr>
<td>Feelings</td>
<td>-.420**</td>
<td>-.230</td>
<td>-.536**</td>
</tr>
</tbody>
</table>

*Note: **p<.01, *p<.05*

In contrast, the only TAQ-ED scale that was related to weight gain was the ‘feelings’ scale. Again, it is apparent that testable assumptions about feelings at the start of therapy were associated with lower levels of weight gain, but only in the latter part of this time period.

4.5.2 Association between initial comorbid psychopathology and early weight change

Table 9 outlines the relationship between weight change and initial scores on the BSI scales (hypothesis 3). Three anxiety-related variables (anxiety, phobic anxiety, interpersonal sensitivity) were associated with weight gain. In each case,
higher levels of anxiety were associated with lower levels of weight gain. While phobic anxiety had an impact on weight gain across the ten sessions, anxiety and interpersonal sensitivity were associated with lower levels of weight gain in the latter part of this period only. There were no associations between weight gain in this period and other elements of initial psychopathology (particularly depression).

Table 9: Pearson’s correlations showing associations of weight change with comorbid psychopathology at the start of therapy (BSI subscales).

<table>
<thead>
<tr>
<th>BSI</th>
<th>Weight 1-10</th>
<th>Weight 1-6</th>
<th>Weight 6-10</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anxiety</td>
<td>-.391*</td>
<td>-.256</td>
<td>-.393*</td>
</tr>
<tr>
<td>Somatization</td>
<td>-.165</td>
<td>-.087</td>
<td>-.190</td>
</tr>
<tr>
<td>Psychoticism</td>
<td>-.248</td>
<td>-.163</td>
<td>-.256</td>
</tr>
<tr>
<td>Paranoia ideation</td>
<td>-.163</td>
<td>-.092</td>
<td>-.195</td>
</tr>
<tr>
<td>OCD</td>
<td>-.195</td>
<td>-.084</td>
<td>-.255</td>
</tr>
<tr>
<td>Hostility</td>
<td>-.211</td>
<td>-.195</td>
<td>-.146</td>
</tr>
<tr>
<td>Phobic anxiety</td>
<td>-.513**</td>
<td>-.364*</td>
<td>-.483**</td>
</tr>
<tr>
<td>Depression</td>
<td>-.141</td>
<td>-.039</td>
<td>-.203</td>
</tr>
<tr>
<td>Interpersonal Sensitivity</td>
<td>-.380*</td>
<td>-.173</td>
<td>-.473**</td>
</tr>
</tbody>
</table>

Note: **p<.01, *p<.05

4.5.3 Association of initial core beliefs and early weight change

Table 10 outlines the relationship between initial core beliefs and weight gain (hypothesis 2). Only one core belief (Unrelenting Standards) was found to be
associated with weight gain. Thus, higher levels of unrelenting standards were associated with less weight gain, and this applied across the whole of the ten sessions of CBT.
Table 10: Pearson’s correlations showing associations of weight change with core beliefs (YSQ-S subscales).

<table>
<thead>
<tr>
<th>YSQ-S</th>
<th>Weight 1-10</th>
<th>Weight 1-6</th>
<th>Weight 6-10</th>
</tr>
</thead>
<tbody>
<tr>
<td>Emotional deprivation.</td>
<td>.079</td>
<td>.072</td>
<td>.064</td>
</tr>
<tr>
<td>Abandonment</td>
<td>.270</td>
<td>.297</td>
<td>.111</td>
</tr>
<tr>
<td>Mistrust</td>
<td>.038</td>
<td>.124</td>
<td>-.032</td>
</tr>
<tr>
<td>Social Isolation/ alienation</td>
<td>-.032</td>
<td>.003</td>
<td>-.018</td>
</tr>
<tr>
<td>Defectiveness/ unlovableness</td>
<td>-.080</td>
<td>-.022</td>
<td>-.116</td>
</tr>
<tr>
<td>Failure to Achieve</td>
<td>.027</td>
<td>.083</td>
<td>-.044</td>
</tr>
<tr>
<td>Practical Incompetence/ dependence</td>
<td>.191</td>
<td>.187</td>
<td>.129</td>
</tr>
<tr>
<td>Vulnerability to Harm or Illness</td>
<td>.187</td>
<td>.091</td>
<td>.224</td>
</tr>
<tr>
<td>Enmeshment</td>
<td>.271</td>
<td>.233</td>
<td>.206</td>
</tr>
<tr>
<td>Subjugation</td>
<td>.136</td>
<td>.239</td>
<td>-.046</td>
</tr>
<tr>
<td>Self-sacrifice</td>
<td>.000</td>
<td>.049</td>
<td>-.086</td>
</tr>
<tr>
<td>Emotional Inhibition</td>
<td>-.088</td>
<td>-.013</td>
<td>-.123</td>
</tr>
<tr>
<td>Unrelenting Standards</td>
<td>-.485**</td>
<td>-.361*</td>
<td>-.399**</td>
</tr>
<tr>
<td>Entitlement/superiority</td>
<td>-.009</td>
<td>-.093</td>
<td>.146</td>
</tr>
<tr>
<td>Insufficient self-control/ Self-discipline</td>
<td>.034</td>
<td>.104</td>
<td>-.033</td>
</tr>
</tbody>
</table>

Note: **p<.01, *p<.05
4.6 Descriptive data for TAQ-ED scores at the beginning and end of treatment sessions

Table 11 shows that 9 people dropped out or failed to complete the questionnaire at session 10 (21%). There was a significant decrease across 10 sessions on scores on the 'body' and 'feeling' sub categories of the TAQ-ED. This means that there is a reduction in pathological conditional beliefs about feelings and the body as therapy progresses (over 10 sessions).

Table 11: Repeated measures ANOVA and Mean (standard deviation) of TAQ-ED scores at session 1 and session 10.

<table>
<thead>
<tr>
<th>Eating</th>
<th>Number</th>
<th>Mean (s.d.)</th>
<th>ANOVA</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>F</td>
</tr>
<tr>
<td>TAQ-ED</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Body time 1</td>
<td>42</td>
<td>2.73 (.91)</td>
<td></td>
</tr>
<tr>
<td>World time 1</td>
<td>42</td>
<td>1.86 (.83)</td>
<td></td>
</tr>
<tr>
<td>Feelings time 1</td>
<td>42</td>
<td>2.49 (1.19)</td>
<td></td>
</tr>
<tr>
<td>Body time 10</td>
<td>33</td>
<td>2.50 (.95)</td>
<td>11.3</td>
</tr>
<tr>
<td>World time 10</td>
<td>33</td>
<td>1.90 (.83)</td>
<td>2.51</td>
</tr>
<tr>
<td>Feelings time 10</td>
<td>33</td>
<td>2.06 (1.01)</td>
<td>13.4</td>
</tr>
</tbody>
</table>

Note: NS=non-significant
4.6.1 Association of early weight and weight change with changes on the TAQ-ED subscales.

Table 12 outlines the relationship between change in cognitions over time and weight-related variables (hypothesis 4). Weight at session 1 was unrelated to the level of change in cognitions over the ten sessions. There were significant negative associations between weight change over the ten sessions and the ‘feeling’ subscale of the TAQ-ED, but not with the other scales. This correlation shows that weight gain in the first ten sessions is associated with a greater reduction in pathological conditional beliefs about feelings. Obviously, it is not possible to assess the direction of causality.

<table>
<thead>
<tr>
<th>Weight</th>
<th>TAQ-ED body 1-10</th>
<th>TAQ-ED world 1-10</th>
<th>TAQ-ED feelings 1-10</th>
</tr>
</thead>
<tbody>
<tr>
<td>Weight 1</td>
<td>-.037</td>
<td>.290</td>
<td>.240</td>
</tr>
<tr>
<td>Weight 1-6</td>
<td>-.288</td>
<td>-.064</td>
<td>-.474**</td>
</tr>
<tr>
<td>Weight 6-10</td>
<td>.150</td>
<td>.022</td>
<td>-.616**</td>
</tr>
<tr>
<td>Weight 1-10</td>
<td>-.172</td>
<td>-.078</td>
<td>-.660**</td>
</tr>
</tbody>
</table>

Note: **p<0.01, *p<0.05

4.6.2 Association of eating related cognitions and change in cognitions (TAQ-ED subscales)

Table 13 outlines the relationship between eating related cognitions (EDE-Q scales) and cognitive change over time (TAQ-ED subscales) (hypothesis 5). There
was a significant association between shape and weight concerns and cognitions and the ‘feeling’ subscale of the TAQ-ED. This shows that higher levels of initial weight and shape concerns were associated with an increase in concerns about feelings over the 10 sessions.

Table 13: Pearson’s correlations showing associations of TAQ-ED change with eating related cognitions (EDE-Q subscales).

<table>
<thead>
<tr>
<th>Weight</th>
<th>TAQ-ED body 1-10</th>
<th>TAQ-ED world 1-10</th>
<th>TAQ-ED feelings 1-10</th>
</tr>
</thead>
<tbody>
<tr>
<td>EDE-Q</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Restraint</td>
<td>.266</td>
<td>.113</td>
<td>.324</td>
</tr>
<tr>
<td>Eating concern</td>
<td>.255</td>
<td>.089</td>
<td>.307</td>
</tr>
<tr>
<td>Shape concern</td>
<td>.160</td>
<td>.245</td>
<td>.455**</td>
</tr>
<tr>
<td>Weight concern</td>
<td>.276</td>
<td>-.015</td>
<td>.386*</td>
</tr>
</tbody>
</table>

Note: **p<.01, *p<.05

4.6.3 Association of initial comorbid psychopathology and change in cognitions (TAQ-ED subscales)

Table 14 outlines the relationship between cognition change and initial scores on the BSI scales (hypothesis 5). There was a significant negative association between somatization and ‘world’ related cognitions of the BSI. This means that higher initial levels of somatization were associated with a decrease in pathological conditional beliefs about the world across time. There was also a significant
association between anxiety and interpersonal sensitivity and ‘feeling’-related cognitions. This shows that higher levels of these pathologies were associated with an increase in concerns about ‘feeling’ related cognitions across time. There were no other associations between comorbid psychopathology and change in cognitions over time.

Table 14: Pearson’s correlations showing associations of TAQ-ED change with comorbidity psychopathology at the start of therapy (BSI subscales).

<table>
<thead>
<tr>
<th>Weight</th>
<th>TAQ-ED body 1-10</th>
<th>TAQ-ED world 1-10</th>
<th>TAQ-ED feelings 1-10</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>.121</td>
<td>.085</td>
<td>.371*</td>
</tr>
<tr>
<td>Anxiety</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Somatization</td>
<td>.056</td>
<td>-.380*</td>
<td>.162</td>
</tr>
<tr>
<td>Psychotism</td>
<td>-.247</td>
<td>.212</td>
<td>.257</td>
</tr>
<tr>
<td>Paranoid ideation</td>
<td>-.156</td>
<td>.110</td>
<td>.219</td>
</tr>
<tr>
<td>OCD</td>
<td>-.121</td>
<td>.068</td>
<td>.225</td>
</tr>
<tr>
<td>Hostility</td>
<td>-.264</td>
<td>.017</td>
<td>.087</td>
</tr>
<tr>
<td>Phobic anxiety</td>
<td>-.054</td>
<td>-.062</td>
<td>.326</td>
</tr>
<tr>
<td>Depression</td>
<td>-.174</td>
<td>.233</td>
<td>.131</td>
</tr>
<tr>
<td>Interpersonal sensitivity</td>
<td>-.281</td>
<td>.244</td>
<td>.421*</td>
</tr>
</tbody>
</table>

Note: **p<.01, *p<.05
4.6.4 Association of core beliefs and change in cognitions (TAQ-ED subscales)

Table 15 outlines the relationship between cognition change and core beliefs (YSQ-S subscales) (Hypothesis 5). There was an association between ‘mistrust’ and ‘body’-related cognitions. This means that higher levels of mistrust were associated with an increase in ‘body’ related cognitions over time. There was a negative association between ‘unrelenting standards’ and cognitions about the ‘world’. This means that more perfectionistic beliefs were associated with a decrease in ‘world’ related cognitions across time. There were no other significant association between core beliefs and cognition change (TAQ-ED subscales).
Table 15: Pearson’s correlations showing associations of TAQ-ED change with core beliefs (YSQ-S subscales).

<table>
<thead>
<tr>
<th>Weight</th>
<th>TAQ-ED body 1-10</th>
<th>TAQ-ED world 1-10</th>
<th>TAQ-ED feelings 1-10</th>
</tr>
</thead>
<tbody>
<tr>
<td>YSQ-S</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Emotional deprivation</td>
<td>.273</td>
<td>.051</td>
<td>-.125</td>
</tr>
<tr>
<td>Abandonment</td>
<td>.351</td>
<td>.148</td>
<td>-.153</td>
</tr>
<tr>
<td>Mistrust</td>
<td>.357*</td>
<td>.071</td>
<td>.170</td>
</tr>
<tr>
<td>Social isolation</td>
<td>.242</td>
<td>.104</td>
<td>.099</td>
</tr>
<tr>
<td>Defectiveness/unlovability</td>
<td>.245</td>
<td>.104</td>
<td>.060</td>
</tr>
<tr>
<td>Failure to achieve</td>
<td>.308</td>
<td>-.078</td>
<td>.115</td>
</tr>
<tr>
<td>Practical Incompetence/dependence</td>
<td>.265</td>
<td>-.140</td>
<td>-.097</td>
</tr>
<tr>
<td>Vulnerability to Harm/illness</td>
<td>.108</td>
<td>-.066</td>
<td>.105</td>
</tr>
<tr>
<td>Enmeshment</td>
<td>.195</td>
<td>-.114</td>
<td>-.037</td>
</tr>
<tr>
<td>Subjugation</td>
<td>.092</td>
<td>.059</td>
<td>-.010</td>
</tr>
<tr>
<td>Self sacrifice</td>
<td>-.003</td>
<td>.181</td>
<td>-.009</td>
</tr>
<tr>
<td>Emotional inhibition</td>
<td>.000</td>
<td>-.014</td>
<td>.113</td>
</tr>
<tr>
<td>Unrelenting standards</td>
<td>-.018</td>
<td>.442*</td>
<td>.283</td>
</tr>
<tr>
<td>Entitlement/superiority</td>
<td>.206</td>
<td>.014</td>
<td>.174</td>
</tr>
</tbody>
</table>

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4.7 Considerations of repeated testing

It must be noted that carrying out a large number of correlations increases the risk of type I errors. Formal statistical correction of p value is not possible, however the results found generally show a patterning that is consistent with hypotheses. The results also tend to group into themes, such as all the anxiety related variables being found as significant. This gives more strength to the findings also.

5. Discussion

5.1 Aim

A cognitive behavioural approach considers the interactions between cognitions, emotions, and behaviours in maintaining a disorder. The current study examined the role of various aspects of that model in the process of recovery. In terms of behaviour change, early weight change is used to challenge beliefs that patients maintain about eating and weight gain (Waller et al, 2007). The aim of the current study was to examine whether particular emotional and cognitive variables are clinically useful predictors of weight gain in the initial stages of treatment of AN. The behaviour measured was weight gain, and change was measured over the first ten sessions of treatment. The behaviours of bingeing, purging and laxative use were also considered separately in the analysis, in relation to weight gain.
In terms of cognitions, the study examined whether a change in dysfunctional assumptions was associated with weight gain. It would be expected that cognitive behavioural approaches should be effective in shifting these beliefs. Where weight gain is accompanied by improvements in cognitions, this should provide the motivation for further eating and weight gain. (Cooper et al., 2005).

Cognitive models suggest that patients with AN use dieting and weight loss behaviours to try to improve mood. It has been suggested that dietary restriction might compensate for the emotional distress associated with negative self-beliefs (Turner & Cooper, 2002). Therefore, the predictive value of mood (particularly anxiety and depression) on weight gain was also examined in the current study.

5.2 Overall findings and link with existing literature

5.2.1 Weight change

*Hypotheses 1. There will be an increase in weight over the 10 sessions and a decrease in behaviours over this time period (bingeing, purging, laxative use).*

In regard to weight change and hypothesis 1, it was found that patients gained more weight in the first 6 sessions (1.01kg) than in the next four sessions (0.63kg). This change in weight over the 10 sessions was significant. Both Hartmann et al. (2007) and Gowers et al. (1994) have concluded that early, steady weight gain is indicative of a good long term prognosis. Therefore, the early weight gain within the current study may be indicative of a better long-term prognosis. The good medium-term results of outpatient treatments with slow weight gain indicate that while weight gain
should be focused, it should be slow enough to be ‘assimilated’ both physiologically and psychologically.

There were no significant changes in behaviour (bingeing, purging and laxative use) across the ten sessions. Bulimic symptoms have been linked with poorer prognosis in other studies also (Hartmann et al., 2007). Due to the small number of patients who displayed the three types of compensatory behaviours, no further analysis could be performed in terms of looking at shifts in cognitions and mood state. Therefore parts of some of the remaining hypotheses could not be explored further.

5.2.2 Weight change and cognitions

Hypothesis 2. Higher levels of negative eating-related cognitions (negative automatic thoughts, EDE-Q; dysfunctional assumptions, TAQ-ED; core beliefs/schemas, YSQ-S) at the beginning of treatment will predict less weight gain over the first ten sessions of treatment for anorexia nervosa.

Hypothesis 2 was supported, as higher levels of eating-related cognitions at the beginning of treatment did predict less weight gain over the first ten sessions of treatment for AN. Specifically, higher levels of the eating-related cognitions, restraint, eating concern, shape concern and weight concern, were associated with less weight gain. This pattern was more consistent during the latter part (sessions 6-10) of this early element of therapy, when ‘restraint’ and ‘weight concern’ attitudes began to have an impact. However, ‘eating concern’ and ‘shape concern’ cognitions had their impact from the first six sessions of therapy. Cooper et al. (1998) suggest that underlying assumptions about weight, shape and eating (in combination with
negative self-beliefs) distinguish patients with AN patients from dieters. Cooper & Fairburn (1992) and Cooper & Turner (2000) found that patients also had a greater number of negative thoughts about eating.

The second part of hypothesis 2 was only partially supported. It was found that higher levels of dysfunctional assumptions about ‘feelings’ only, were associated with lower levels of weight gain in sessions 6-10 of therapy. Hinrichsen et al. (2005) hypothesised that the specific content of beliefs are influential in AN. However, these findings do not map onto any material in the literature, therefore they cannot be compared or contrasted to other findings. They will be discussed further when outlining the clinical implications of the study and ideas for future research.

When considering the relationship between weight gain and core beliefs/schemas, higher levels of ‘unrelenting standards’ were found to be associated with less weight gain over 10 sessions. No other core beliefs were found to relate to the process of weight gain in contrast to the hypotheses. The trait of perfectionism has been consistently associated with AN (Fairburn et al., 1999; Shafran et al., 2002), and the current results suggest that this trait is influential in the process of treatment. It appears that the higher the perfectionistic drive, the poorer the response to treatment.

5.2.3 Weight change and mood

Hypotheses 3. Higher levels of negative mood at the beginning of treatment will predict less weight gain over the first ten sessions of treatment for anorexia nervosa.

This hypothesis was partially supported. Higher levels of three anxiety-related variables (anxiety, phobic anxiety, interpersonal sensitivity) were associated with
lower levels of weight gain. While phobic anxiety had an impact on weight gain across the ten sessions, anxiety and interpersonal sensitivity were associated with lower levels of weight gain in the latter part of this period only. There is existing evidence supporting the link between eating and anxiety disorders (Godart et al., 2002). Prevalence rates of anxiety within AN samples vary from 23% (Laessle et al., 1987) to 75% (Deep et al., 1995). The lifetime prevalence of GAD in AN has been found to be between 13% (Kaye et al., 2004) and 45.6% (Godart et al., 2003). There were no associations in this period between weight gain and other elements of initial psychopathology, such as depression. This is consistent with studies suggesting that the presence of comorbid depression at initial presentation has minimal or no predictive value for treatment outcome (Keel & Mitchell, 1997; Herpeitz-Dahlmann et al., 2007).

5.2.4 Weight gain and shifts in cognitions (dysfunctional assumptions) over time

_Hypothesis 4. Over the initial part of treatment, positive changes in behavioural and biological symptoms (e.g., weight gain) will be associated with reductions in cognitions (dysfunctional assumptions)_

Weight at session 1 was unrelated to the level of change in cognitions over the ten sessions. Hypothesis 4 was partially supported; there were significant negative associations between weight change over the ten sessions and the ‘feeling’ subscale of the TAQ-ED, but not with the other subscales. This correlation shows that weight increase in the first ten sessions is associated with a greater reduction in pathological conditional beliefs about feelings. Obviously, it is not possible to assess the direction of causality. Previous research has commented on the likely link between ‘feeling’-
related dysfunctional assumptions and eating disorders (Hinrichsen et al., 2005) but has made no reference to shift in patterns over time.

Considering this tentatively it may be that there is a reduction in pathological conditional beliefs about feelings with weight gain, as these beliefs have been addressed and somewhat challenged as part of the therapy. Thinking about the weight loss as a safety behaviour, it makes sense that as this safety behaviour is dropped there is a chance to challenge some of the pathological beliefs that have maintained the cycle of pathology.

5.2.5 Shifts in dysfunctional assumptions over time with mood and other cognitions

_Hypothesis 5. Positive changes in cognitions over time (dysfunctional assumptions) will be associated with more positive levels of eating related cognitions (EDE-Q), core belief/schemas (YSQ-S) and mood (BSI)._ 

There was a significant decrease across ten sessions on scores on the ‘body’ and ‘feeling’ subscales of the TAQ-ED. This reflected a reduction in pathological conditional beliefs about feelings and the body as therapy progresses (over 10 sessions).

In terms of the relationship between eating related cognitions (EDE-Q scales) and cognitive change over time (TAQ-ED subscales), there was a significant association between shape and weight concern cognitions and the ‘feeling’ subscale of the TAQ-ED. This shows that higher levels of weight and shape concerns at the beginning of treatment were associated with an increase in concerns about feelings over the 10
sessions. This is consistent with previous findings in the current study that higher levels of ‘feeling’-related dysfunctional assumptions were associated with less weight gain.

When examining the relationship between cognition change and initial scores on the BSI scales, there was a significant negative association between somatization and ‘world’-related cognitions of the TAQ-ED. Higher levels of somatization were associated with a decrease in pathological conditional beliefs about the world across time. There was also a significant association between anxiety and interpersonal sensitivity and ‘feeling’ related cognitions. This shows that higher levels of these pathologies were associated with an increase in concerns about ‘feeling’ related cognitions across time. Again this seems to be a theme - higher levels of ‘feeling’-related cognitions appear to be connected with less weight gain, greater levels of eating related cognitions and greater psychopathology. However, as previously discussed, with time as therapy progresses there does seem to be an association with a reduction in pathological feeling related beliefs in those patients who gain weight. Whether weight gain or cognitive shifts occur first is not possible to assess, but it is possible that addressing eating related cognitions and anxiety may further increase the chance of weight gain and a shift in the area of feeling related cognitions.

When considering the relationship between cognition change and core beliefs/schemas (YSQ-S subscales) there was an association between ‘mistrust’ and ‘body’ related cognitions. Higher levels of mistrust were associated with an increase in ‘body’ related cognitions over time. There was a negative association between ‘unrelenting standards’ and cognitions about the ‘world’. More perfectionistic beliefs were associated with a decrease in ‘world’ related cognitions across time. There were
no other significant association between core beliefs/schemas and cognition change (TAQ-ED subscales). An absence of previous research means that it is not possible to compare these findings to other findings, but in terms of patterns in the current data, ‘world’ related cognitions do appear to be related to mood and core belief/schema related domains (somatization and ‘unrelenting standards’) and that these are associated with an actual decrease in this specific cognition over time.

5.3 Strengths and Limitations

This study has a number of strengths and limitations. One advantage is that the questionnaires utilised were part of a standard service assessment pack and treatment protocol, meaning that the majority of patients in the service completed them. This makes it less likely that it is only the most motivated patients who are engaging in the study. The sample was therefore quite generalisable to referrals in other specialist eating disorder services. Although the sample is representative of an outpatient clinical sample, this in itself is a reflection of those who have reached the specialist services, and does not represent the whole AN population. Therefore, it would be beneficial to repeat this study within samples at other levels of care. The sample is also limited in that it represents a female population only.

A further limitation of the study was the relatively small sample size. Recruitment in the study ideally would have continued until there were enough participants to examine differences across sub-diagnoses, and to examine differences across behaviours (bingeing, purging and laxative use). It would also have been useful to have looked at differences between those who dropped out of treatment and completers (of the 10 sessions). These considerations leave possibilities for future research. Future research might also consider using a control group of dieters to
enable further conclusions to be drawn in the areas that highlighted significant results in the current study.

The use of questionnaires as a self report measure leaves some question as to whether core beliefs and schemas are accurately assessed by these measures. However, evidence does show that participants can be aware of these processes and are able to rate themselves appropriately (Bond et al., 1986). In terms of eating related cognitions, Wolk et al. (2005) revealed that a higher level of disturbance was reported on the EDE-Q in comparison to the EDE. This may be because participants report their symptoms more honestly in a self-report format than in an interview or alternatively that such measures lead to an overestimate of symptoms. Even though it has been suggested that the EDE-Q provides an accurate measure of pathological thinking in AN patients (Mond et al., 2004; Luce et al., 2008), it is not clear whether subscales reflect specific causes of AN or whether they represent the general level of psychological disturbance (Cooper et al., 1985).

Although patients were treated with CBT, the approach was principle- rather than protocol-driven. However, therapists’ supervision was overseen by a specialist cognitive-behavioural therapist who directed the content of their sessions. In terms of the therapy process, it is possible that other factors had an effect on progress (weight gain). Such factors might include the relationship with the therapist, or the social desirability of responding to treatment. These factors are not easily controlled, meaning that it is questionable whether it was solely CBT that was responsible for creating changes in dysfunctional assumptions. Even if treatment effects are demonstrated, interpretation of findings is still problematic in terms of deciphering
whether it was the cognitive or behavioural aspect of treatment that was most
effective. ‘Cognitive restructuring’ may occur in patients treated with behavioural
techniques, but cognitive restructuring may still explain the improvements observed.
Conversely the cognitive aspect may have been influenced by behavioural changes
alone.

It may be that a longer period of time is required to identify further shifts in
cognitions (dysfunctional assumptions) that were identified, as the time period of 10
sessions is obviously relatively brief.

5.4 Future directions

In addition to considering the limitations outlined above and improving on the
methodology accordingly, there are further factors to consider within future research.

It is important to consider adherence in relation to the therapy process, as remaining
in treatment does not necessarily mean that a patient is adhering to treatment.
Adherence might be considered by measuring punctuality, cancellations, DNAs and
homework completion.

The current study is the first of its kind to examine cognitive, emotional and
behavioural predictors of weight gain within a CBT framework for AN. The results
open multiple avenues in terms of future research, one being to examine the patterns
found (in attitudinal shift and mood responses) over an extended period of time
(perhaps the next step would be to examine the process over sessions 10-20). In
addition to examining the link between anxiety and eating disorders further
(particularly across time), it will be important to explore what factors contribute to weight and shape concerns among those who are anxiety prone.

It would have been beneficial to examine the EDE-Q scores at session ten to see whether there had been a shift in pathological attitudes across the eating attitudes. This was not done because previous research has examined negative automatic thoughts in relation to eating, weight and shape, but has not examined dysfunctional assumptions which were the focus of the current study. Increases in weight might be associated with paradoxical increases in EDE-Q scores, because of increasing dissatisfaction with weight. Conversely, CBT might reduce body dissatisfaction and associated distress with increasing weight. This is relevant, taking into account that body dissatisfaction may be the strongest predictor of dietary restraint and therefore needs to be considered within treatment (Rosen et al., 1987).

With a larger number of participants it would have been useful to have carried out multiple regression analysis to gain an understanding of the variation in strength of the predictor variables influence on weight gain. In this way possible models could be proposed for the factors considered most influential on weight gain at the beginning of therapy and the possible process by which they work. In line with this consideration of future research with a larger number of participants, pattern of drop-outs could also be examined further. It would be useful to consider which cognitive and mood related variable are associated with early drop-out and at which time point in the therapy. This would aid clinicians in thinking about what factors need to be considered or addressed early in therapy to reduce the likelihood of dropout.
In terms of diagnostic considerations, there is a difficulty in measuring rates of anxiety disorders within the AN population due to the impact over time in changing diagnostic categories for both eating disorders and anxiety disorders. Therefore, research needs to use standardised measures to identify and examine eating and anxiety disorders. This is relevant as the impact of these findings have significance in terms of the aetiology of disorders as well as therapeutic implications (Swinbourne & Touyz, 2007). However, again in terms of the session length, ten sessions may not be enough time in which to find a diagnosis of anxiety.

5.5 Clinical implications

It has been highlighted by research that additional therapeutic treatments lead to less additional gain, depending on the previous amount of therapy (Howard et al., 1986). Again, it is important to maximise efficacy of treatment on the first occasion.

Individual factors should be taken into account at assessment, and treatment should be tailored to these. Considerations involve both cognitive and emotional avenues. In line with previous research (e.g., Cooper et al., 1997), right from the beginning of treatment ‘shape’ and ‘eating’ relating cognitions need to be considered, and in the latter part (sessions 6-10) it is likely that ‘restraint’ and ‘weight concerns’ also become relevant. Pressure to gain weight at the beginning of therapy tends to hinder weight gain and therefore it is important to consider these cognitions in relation to the focus and goals of therapy. In terms of general cognitions (dysfunctional assumptions), those patients with higher levels of ‘feeling’ related cognitions, in other words, those who fear expressing feelings or use behaviours to block emotions, (e.g. questionnaire items: ‘restricting helps me to control negative thoughts and
feelings’; ‘my friends and family would get angry with me, if I told them how I really feel inside’) also appear to be a greater risk of less weight gain. In this way, clinicians should be aware that after the first six sessions of treatment, unaddressed ‘feeling’ related cognitions are likely to interfere in the treatment programme. In line with cognitive-behavioural principles, clinically these beliefs could be addressed in a behavioural manner, allowing expectations of rejection in social situation (e.g., sharing personal information) to be tested out and disconfirmed by patients themselves. Those of lower weight also need to be targeted early on in treatment.

Those patients with more perfectionistic beliefs (‘unrelenting standards’ core beliefs/schemas) also need to be considered as at increased risk of less weight gain. In this way cognitive work from an early stage of therapy should aim to address these specific belief systems, to prevent them hindering treatment. This links in with Fairburn et al’s. (2003) work suggesting that clinical perfectionism needs to be addressed as an individual component of therapy. Again, in terms of intervention, in line with Corte & Stein’s (2005) suggestion, it may be important to focus on the development and elaboration of new domains of self-definition in this population and to create positive core beliefs and schemas that reflect personal importance, strength and competence, in response to the schemas that have been identified in the current study as related to less weight gain. If non weight-related core beliefs and schemas are activated in memory, then in theory negative emotions may be lower and consequently the necessity to reduce psychological distress through disordered eating may be reduced.
In terms of presenting mood, it is vital that the role of anxiety is considered as predictor of poor weight gain. It appears that therapy processes need to consider tackling anxiety in patients with AN right from the beginning of therapy. CBT methods that are used in the treatment of anxiety may be appropriate, such as; considering meta cognitions involved in the maintenance of anxiety, exposure to feared scenarios experimentally, and considering the typical belief systems associated with generalised and specific anxieties (Wells, 1997; Hawton et al., 2005).

5.6 Diagnostic implications

There is an overlap in the cognitions and behaviour patterns involved in AN and anxiety disorders - specifically the cognitions involved, heightened vigilance and defensive avoidance. Interestingly, anxiety disorders found in those with AN usually begin before signs of weight loss and food preoccupation (Bulik et al., 1997). Sub threshold features of anxiety also often persist after recovery of normal body weight (Casper, 1990).

In terms of genetic factors, twin studies show evidence that there is a genetic correlation between eating disorders and certain anxiety disorders (Wade et al., 2000; Keel et al., 2005; Mangweth et al., 2003). Strober et al. (2007) looked at the role of familial transmission as evidence of the comorbidity between anxiety and AN. They found that relatives of probands with AN were shown to have a threefold greater risk of obsessive compulsive disorder, generalised anxiety disorder and OCPD compared with relatives of ‘never ill controls’, and an increased risk of panic disorder, social phobia, separation anxiety and simple phobia. The findings lend support to the idea that “AN is part of a spectrum of disorders that share in common a transmitted inherited propensity for extreme anxiety and fear learning” (Strober et
al., 2007). In line with this there are suggestions that early onset anxiety disorders may represent a potential genetically mediated pathway toward the development of an eating disorder (Kaye et al., 2004) or leave a predisposition towards developing an eating disorder (Bulik et al., 1996).

Waller (2008) proposes that “Eating disorders may be treated as a specific behavioural and cognitive manifestation of the anxiety disorders”. In this way, Waller (2008) suggested that eating disorders could be part of a category of anxiety disorders in a single ‘transdiagnostic’ group, where sub divisions are based on cognitions and behaviours that distinguish clinical presentations. Anxiety disorders as a general category would be characterised by vulnerability cognitions and the use of safety behaviours that alleviate the vulnerability. The behaviours that characterise eating disorders (e.g., restriction, bingeing, purging) may be viewed as safety behaviours, by lowering the level of anxiety through impulsive or compulsive ‘blocking’ of awareness of that emotional state (Pallister & Waller, 2008). Subdivisions would be defined by different cognitive, behavioural and psychophysiological manifestations.

5.7 Summary
Clinical experience in treating anorexia nervosa suggests that weight gain is a crucial early index of likely treatment success. However, there is no current evidence base that allows the clinician to anticipate who will make the necessary early weight gain. This study examined psychological factors present at the start of CBT for AN that predict change in weight over the first ten sessions of treatment.
Broadly, unhealthy initial eating attitudes, anxiety and perfectionism levels were related to slower weight gain across the course of treatment. Higher levels of ‘feeling’ related cognitions were associated with less weight gain in the latter stages of treatment. This research has identified a set of factors that predict slower weight gain in the course of the early part of CBT for AN. These findings suggest that there may be a potential benefit in modifying CBT to address anxiety and perfectionism earlier in treatment. There is a need to focus on patients identified overtly as anxious and those of low weight. In terms of intervention, the focus of this should be on anxiety, making use of behavioural experiments, exposure and safety behaviours.

Of clinical interest, the implication of part of these and previous findings mean that there may be a need to reformulate the status of AN in the DSM-V. There have been suggestions of grouping AN with a broad group of mood/anxiety disorders. These would be hierarchically structured, at the highest point defined by broad, genetic vulnerability factors (e.g., negative affect) which branch to more narrowly defined clinical phenotypes, where symptom formation had been influenced by genetic, neural and socio-environmental mechanisms.
References


PART 3: CRITICAL REFLECTION
Part 3 Critical Reflection

1. Introduction

The first part of the thesis examined the empirical evidence to date on treatment trials of CBT for AN. The second element, the empirical study, investigated various cognitive and emotional predictors of weight gain, and also examined shifts in cognitions over time. The last part of the dissertation is a critical reflection on the research process as a whole.

2. Considering CBT as a model

This study reflects a very early stage of research looking at elements involved in the difficulty with the treatment of AN. Rather than fall into a similar trap of setting up more unhelpful trials comparing CBT to an alternative treatment in its entirety, the current study has taken a step back and has considered the flaws of previous research. It has attempted to go back to basics and break down the rationale of what we are doing with our therapies and why. In terms of CBT, research into the cognitive pathways and processes involved in eating disorders is fundamental to treatment. Waller at al. (2007) conceptualises eating disorders by focusing on the function of behaviours involved in eating disorders. He maintains that AN can be viewed as an ‘emotion blocking behaviour’, at the initial stage as it prevents emotions from being activated. If viewed from this perspective, the function of cognitive processes within AN may be viewed differently from other disorders. It may be that the content and pathway of cognitions is different, and that emotions are strong drivers in maintaining behaviours also.
In an attempt to understand what type of cognitions affect weight gain in the early stages of therapy, the current study has considered food related negative automatic thoughts; at a deeper level dysfunctional assumptions, which are more broadly related to feelings/body/world, and core beliefs unrelated to food/weight/shape. Even from examining the early stages of a longer-term therapy, the break down of the first and second half of a 10-session period offers useful insight in terms of highlighting cognitive shifts. Feeling related cognitions (dysfunctional assumptions) only become associated with weight gain after the first 5 sessions; and the reality that feeling related cognitions appear to have an impact at this early stage is important to acknowledge. In terms of emotional states, anxiety and interpersonal sensitivity were found to be active in the latter part of the 10 session period, as it appears that anxiety levels do affect weight gain, but that this is not necessarily apparent at the very initial stages of treatment. Therefore emotionality does have an important role, certainly in the early stages of the maintenance of AN. The role of cognitions at the level of negative automatic thoughts is strongly associated with weight, shape and eating and this has been supported by previous research. However in terms of treatment this represents a stumbling block when considered as the sole focus of therapy. An additional interest is the role non-food/weight/shape cognitions play. In line with Waller et al.'s. (2007) work, ‘feeling’ related cognitions examined by the TAQ-ED may highlight how part of the role of the AN is to be an emotion blocking behaviour, e.g. “restricting my eating helps me control negative thoughts and feelings”. It must be taken into account that this is an early stage of research, and the TAQ-ED questionnaire utilised is also relatively new in its use. This leaves numerous possibilities for future research.
In terms of evaluating CBT, formulation-driven CBT involves flexibility and thus is hard to manualise. In this way, comparisons across cases are not easily standardised. It is apparent that a rationalistic approach to CBT is to use a structured approach, however in reality a client's thinking pattern is not fixed and contains a huge amount of variability and requires the model of CBT to be adjusted accordingly. Therefore the applicability of this needs to be considered when reducing CBT to a manualised approach, which randomised control trials require.

3. Personal reflection

Having conducted and published previous research within the field, I already had experience of the research process, data collection, the rationale of statistical testing and the process of writing up research. However the process was different within the current research as I was not able to work within the service where the research was conducted. This meant that although time was spent at the service collecting data and discussing the research, I did not have a clinical connection to the data (people). This brought some difficulties that I had not anticipated. In previous research conducted I had primary control of recruitment and continuous access to both patients and clinicians involved in the research. An absence of this left me with some anxiety about having sufficient control over the project, for example, ensuring that questionnaires were continually being given to and completed by patients, at the appropriate time points. The most effective decision was having a coordinator from UCL who worked at Vincent Square eating disorder service; reinforce the messages I wanted to give clinicians who were involved in carrying out the therapy. This did alleviate my anxieties.
I was able to work at another eating disorder service, and conducted CBT with patients using the model utilised by the service where my research was taking place. I believe this was vital, as I was made to think about the cognitions, emotions and behaviours linked with AN. It also highlighted some of the many difficulties of working with this client group. I was able to think about elements of CBT that relate to the first 10 sessions of therapy and in this way make sense of clinical-research links. I was also able to consider the realities of high attrition rates within the eating disorders.

Within my experience I have learnt the importance of services being equipped with the tools to carry out continuous research. As part of continuous audit, collecting client data (e.g., demographics, beliefs, attitudes) at relevant time points opens up opportunities for continuous meaningful research. Having the tools in place to measure shifts over time in attitudes, beliefs, mood and behaviour is an important aspect that any service should be considering, both specifically in terms of CBT change and on a global service level, and it is surprising that many services continue to fail to do this.

In terms of the research I examined around my literature review, this helped me think about the range of models used for treatment of AN and the individual rational for these different approaches. It made me question and think about my own practice with this client group, and continue to consider evidence base practice. I was surprised by the total absence of methodologically ‘fair’ trials that have assessed CBT in the treatment of AN. The lack of details also made it very difficult to assess trials thoroughly. In terms of future considerations, there is clearly a need to establish
generalised standards, for example, considering that trials state the breakdown of the focus of sessions, so as researchers we are able to evaluate the individual components of success more easily. The manner in which success of treatment is measured, particularly utilising the various forms of the MR-Scales is inconsistent and makes comparisons across trails near to impossible. Research might consider a generalised measure that incorporates the most efficient elements from existing measures. A similar point is true for the measurement of dropout.

Some aspects of dropout were examined in the literature review, particularly patterns of dropout. Despite suffering similar methodological flaws as the papers evaluating treatment success, there is a distinct lack of papers that have examined psychological predictors of dropout. Considering a large reason for dropout might be considered to be both psychological and treatment focused, it is surprising that papers have not considered the cognitive and emotional aspects in individuals who do and do not engage in treatment. If this information was available it would be more likely to be able to develop concepts and even models of the cognitions and belief systems involved in the process of dropout.

Findings from the empirical paper are extremely interesting. They highlight how even thinking on a small scale, such as focusing on a specific element of a therapy over a very short time period, can have much greater clinical and practical implications. Even though it would be naïve to think that more research would not be required, these findings suggest that there is a potential benefit in modifying CBT to address anxiety, feeling related cognitions and perfectionism earlier in treatment, within the AN population. It does seem that anxiety needs to be considered from an
early stage in AN patients. In terms of my own practice, it has made me think about client’s generic presentation, in terms of assessment and treatment; as it is easy to get into the routine of ‘boxing’ clients according to their presenting problem. What it has also highlighted is the validity of considering the function of behaviours that clients present with. The findings also have wider implications in terms of thinking about the categorisation of disorders in the DSM. As a researcher it has made me think and question the way we define disorders, the process of categorisation, and has to some degree changed my perception of this. I have begun to question the function of behaviours, and clinically this has helped me as a therapist. I still adhere to using evidence based practice and treatment protocols, but have become more aware and able to question the individual presentation of behaviours and cognitions.

4. Methodological and conceptual issues

The initial plan for the project was to recruit 50-60 participants from March 2007 to March 2008. Due to various changes in the service structure and hence number of clinicians conducting therapy and involved in the project, this was reduced. This meant that the final number of participants involved was 45, and taking into account the dropout, numbers dropped to 38. This did not affect the validity of the statistical tests performed, but meant that a comparison between restrictive and binge/purging AN was not possible and examining specific behaviours (bingeing, purging, laxative use) was also not possible, as already discussed. This research can be continued within the service as data is collected as part of a continuous clinical audit, so numbers could easily be increased for future studies.
Study findings have been based on questionnaire results, and although all were standardised and showed good psychometric properties, there are many alternative measures that could have been used to measure eating attitudes and mood. It might be that one area of future research could be to replicate the study using other measures, clarifying that the results obtained are repeatable and ‘genuine’. To assess these beliefs and attitudes through interview would have presented a new array of limitations.

Measuring weight gain has associated difficulties, (such as water loading), but since weekly weight change was measured this ensured more control over this. Although weight gain was found to be statistically significant, compared to some other studies the level of weight gain over the equivalent time period was somewhat low. Future research might consider assessing the continuous process of weight change over the remainder of therapy, as the slight decrease in the second half of the first 10 sessions might not represent the continuous pattern of weight change. It was disappointing that it was not possible to use behaviour change (bingeing, purging or laxative use) in terms of there association with cognitions and mood, as the number of patients who were binge/purging subtype, or simply who reported these behaviours, was too low to carry out any further meaningful statistical testing on. It may be that there was under-reporting of these behaviours. Making use of the EDE-Q measures of behaviours in combination with the weekly measurement of binge, purging and laxative use measured by clinicians during the therapy, would have allowed these figures to be cross examined.
5. Future research

A number of questions have been posed by the research, and this is within a very time limited frame of a specific therapy. It is crucial to know the pattern of weight gain, cognitive and emotional states following the next 10 sessions. Keeping with the framework of the current study, various aspects could be explored further. The various beliefs that were found to be significant and associated with weight change could be a focus of further research to examine whether patterns are long lasting of whether other cognitions become active at different time points. It might have been useful to have examined change in core beliefs/schemas over the 10-session time frame, but what we already know from theory is that these are less likely to be relevant in such an early stage of therapy, as these belief systems are not a focus in therapy until later on in therapeutic process.

Considering research suggesting that CBT is as effective as most therapies in terms of treatment for AN, (although it does tend to have more success in those who are less underweight), it might be beneficial for services who utilise other models and treatment approaches, such as IPT or family therapy, to explore components of the therapy over the initial part of treatment, in a similar method to our study. In this way it might help identify what emotional, cognitive and behavioural components appear to be predictive of weight gain or drop out, and will consequently increase the strength of evidence in this area. The current study only considered the adult population. It may also be useful to consider whether the results of this study extend to adolescents with eating disorders. The majority of research carried out in the adolescent population highlights that family therapy presents the best results in terms
of weight gain (Eisler et al., 2005). Therefore this might be a useful area of focus in this population.

6. Clinical implication

Patients with AN and high levels of anxiety, interpersonal sensitivity and phobic anxiety need clinicians to consider this at the very earliest stage of treatment, as it is possible that this will interfere with weight gain if ignored. Again there is a need for all patients starting treatment for AN to have anxiety levels considered at assessment. The role of the core belief perfectionism (‘unrelenting standards’) suggests that this needs to be considered, even at this very early stage of therapy. Perhaps using schema-focused interventions.

Considering some of the techniques that are used to treat anxiety may be helpful in the initial stages of treatment for those with AN. In treating those with high levels of anxiety, this need to be identified and formulations carried out to help patients feel they have been understood and that progress can be made in addressing their relevant cognitions and thinking style, such as the use of safety behaviours. If these are not addressed it is likely that they may maintain a “what if” type of thinking, or “I managed to avoid that happening” which will in avertedly keep the cycle of maintenance in action. Again having a high level of ‘interpersonal sensitivity’ may reduce this type of patient’s ability to articulate their difficulties and this needs to be considered within treatment.
References


Appendix 1: Eating Disorder Examination – Questionnaire
**ED-E-Q**

**Instructions**
The following questions are concerned with the PAST FOUR WEEKS ONLY (28 days). Please read each question carefully and circle the appropriate number on the right. Please answer all the questions.

<table>
<thead>
<tr>
<th>ON HOW MANY DAYS OUT OF THE PAST 28 DAYS ……</th>
<th>No days</th>
<th>1-5 days</th>
<th>6-12 days</th>
<th>13-15 days</th>
<th>16-22 days</th>
<th>23-27 days</th>
<th>Every day</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Have you been deliberately trying to limit the amount of food you eat to influence your shape or weight?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
</tr>
<tr>
<td>2. Have you gone for long periods of time (8 hours or more) without eating anything in order to influence your shape or weight?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
</tr>
<tr>
<td>3. Have you tried to avoid eating any foods which you like in order to influence your shape or weight?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
</tr>
<tr>
<td>4. Have you tried to follow definite rules regarding your eating in order to influence your shape or weight; for example, a calorie limit, a set amount of food, or rules about what or when you should eat?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
</tr>
<tr>
<td>5. Have you wanted your stomach to be empty?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
</tr>
<tr>
<td>6. Has thinking about food or its calorie content made it much more difficult to concentrate on things you are interested in; for example, read, watch TV, or follow a conversation?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
</tr>
<tr>
<td>7. Have you been afraid of losing control over eating?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
</tr>
<tr>
<td>8. Have you had episodes of binge eating?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
</tr>
<tr>
<td>9. Have you eaten in secret? (Do not count binges.)</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
</tr>
<tr>
<td>10. Have you definitely wanted your stomach to be flat?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
</tr>
<tr>
<td>11. Has thinking about shape or weight made it more difficult to concentrate on things you are interested in; for example read, watch TV or follow a conversation?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
</tr>
<tr>
<td>12. Have you had a definite fear that you might gain weight or become fat?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
</tr>
<tr>
<td>13. Have you felt fat?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
</tr>
<tr>
<td>14. Have you had a strong desire to lose weight?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
</tr>
</tbody>
</table>

**OVER THE PAST FOUR WEEKS (28 DAYS)**

<table>
<thead>
<tr>
<th>15. On what proportion of times that you have eaten have you felt guilty because of the effect on your shape or weight? (Do not count binges.)</th>
<th>0 – None of the times</th>
<th>1 – A few of the times</th>
<th>2 – Less than half the times</th>
<th>3 – Half the times</th>
<th>4 – More than half the times</th>
<th>5 – Most of the times</th>
<th>6 – Every time</th>
</tr>
</thead>
<tbody>
<tr>
<td>(Circle the number which applies.)</td>
<td></td>
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</table>


16. Over the past four weeks (28 days), have there been any times when you have felt that you have eaten what other people would regard as an unusually large amount of food given the circumstances? (Please circle YES or NO and put appropriate number in box.)

<table>
<thead>
<tr>
<th></th>
<th>YES</th>
<th>NO</th>
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</thead>
<tbody>
<tr>
<td>17.</td>
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<tr>
<td>18.</td>
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</tbody>
</table>

19. Have you had other episodes of eating in which you have had a sense of having lost control and eaten too much, but have not eaten an unusually large amount of food given the circumstances?

<table>
<thead>
<tr>
<th></th>
<th>YES</th>
<th>NO</th>
</tr>
</thead>
<tbody>
<tr>
<td>20.</td>
<td>( )</td>
<td></td>
</tr>
</tbody>
</table>

21. Over the past four weeks have you made yourself sick (vomit) as a means of controlling your shape or weight?

<table>
<thead>
<tr>
<th></th>
<th>YES</th>
<th>NO</th>
</tr>
</thead>
<tbody>
<tr>
<td>22.</td>
<td>( )</td>
<td></td>
</tr>
</tbody>
</table>

23. Have you taken laxatives as a means of controlling your shape or weight?

<table>
<thead>
<tr>
<th></th>
<th>YES</th>
<th>NO</th>
</tr>
</thead>
<tbody>
<tr>
<td>24.</td>
<td>( )</td>
<td></td>
</tr>
</tbody>
</table>

25. Have you taken diuretics (water tablets) as a means of controlling your shape or weight?

<table>
<thead>
<tr>
<th></th>
<th>YES</th>
<th>NO</th>
</tr>
</thead>
<tbody>
<tr>
<td>26.</td>
<td>( )</td>
<td></td>
</tr>
</tbody>
</table>

27. Have you exercised hard as a means of controlling your shape or weight?

<table>
<thead>
<tr>
<th></th>
<th>YES</th>
<th>NO</th>
</tr>
</thead>
<tbody>
<tr>
<td>28.</td>
<td>( )</td>
<td></td>
</tr>
</tbody>
</table>

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OVER THE PAST FOUR WEEKS (28 DAYS) (PLEASE CIRCLE THE NUMBER WHICH BEST DESCRIBES YOUR BEHAVIOUR.)

<table>
<thead>
<tr>
<th></th>
<th>NOT AT ALL</th>
<th>SLIGHTLY</th>
<th>MODERATELY</th>
<th>MARKEDLY</th>
</tr>
</thead>
<tbody>
<tr>
<td>29. Has your weight influenced how you think about (judge) yourself as a person?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>30. Has your shape influenced how you think about (judge) yourself as a person?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>31. How much would it upset you if you had to weigh yourself once a week for the next four weeks?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>32. How dissatisfied have you felt about your weight?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>33. How dissatisfied have you felt about your shape?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>34. How concerned have you been about other people seeing you eat?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>35. How uncomfortable have you felt seeing your body; for example, in the mirror, in shop window reflections, while undressing or taking a bath or shower?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>36. How uncomfortable have you felt about others seeing your body; for example, in communal changing rooms, when swimming or wearing tight clothes?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
</tbody>
</table>
Appendix 2: Testable Assumptions Questionnaire – Eating Disorders revised
### TAQ-ED Revised

Listed below are a number of beliefs that people with eating difficulties often have. Please read each belief carefully and indicate how much you *generally* agree with each one. Please circle the number that best describes your answer. Please respond to all of the items, and try to be as honest as you can.

**RATING SCALE:**

1 = do not agree  
2 = agree slightly  
3 = agree moderately  
4 = agree very much  
5 = totally agree

<table>
<thead>
<tr>
<th></th>
<th></th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>If I feel bloated or my clothes feel tighter, it means I have put on weight.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>If I don't keep tight control over my eating, I will not be able to stop myself from eating more and more.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>If I were overweight, most people would ridicule or humiliate me.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>Being overweight means you have failed as a person.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>If I change my current eating pattern in any way, I will lose control over my eating.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>If I feel unattractive, it means I look unattractive to others.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>Most men are only interested in women who look slim.</td>
<td></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>8</td>
<td>If I became overweight, nobody would want to be with me.</td>
<td></td>
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<td></td>
<td></td>
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</tr>
<tr>
<td>9</td>
<td>Restricting my eating helps me to control negative thoughts and feelings.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>10</td>
<td>If I tell others how I feel inside they will think I'm odd and reject me.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>11</td>
<td>You can never be successful unless you are physically attractive.</td>
<td></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>12</td>
<td>If I don't weigh myself several times a week, my weight will spiral out of control.</td>
<td></td>
<td></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>13</td>
<td>If I eat in front of others, they will think I'm disgusting.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>14</td>
<td>Vomiting or taking laxatives are effective ways to stop my weight from going up.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>15</td>
<td>If I express my true feelings to others, they will make fun of me and humiliate me.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>16</td>
<td>If I eat three meals and three snacks per day, my weight will increase uncontrollably.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>17</td>
<td>My friends and family would get angry with me, if I told them how I really feel inside.</td>
<td></td>
<td></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>18</td>
<td>If I start eating forbidden foods once, I won't be able to stop myself from eating them all the time.</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>19</td>
<td>If my weight goes up people will stop caring about me.</td>
<td></td>
<td></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>20</td>
<td>I have to use eating in order to cope with negative feelings.</td>
<td></td>
<td></td>
<td></td>
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</tr>
</tbody>
</table>
Appendix 3: Brief Symptom Inventory
**INSTRUCTIONS:**
This is a list of problems people sometimes have. Please read each one carefully, and tick the box that best describes **HOW MUCH THAT PROBLEM HAS DISTRESSED OR BOTHERED YOU DURING THE PAST 7 DAYS, INCLUDING TODAY.** Tick only one box for each problem and do not skip any item. If you change your mind, make an 'X' through the incorrect box and then tick the correct one. Read the example before beginning, and if you have any questions please ask them now.

<table>
<thead>
<tr>
<th>HOW MUCH WERE YOU DISTRESSED BY:</th>
<th>Not at all</th>
<th>A little bit</th>
<th>Moderately</th>
<th>Quite a bit</th>
<th>Extremely</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bodyaches</td>
<td></td>
<td>X</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Please answer the following questions:

<table>
<thead>
<tr>
<th>HOW MUCH WERE YOU DISTRESSED BY:</th>
<th>Not at all</th>
<th>A little bit</th>
<th>Moderately</th>
<th>Quite a bit</th>
<th>Extremely</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Nervousness or shakiness inside.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2. Faintness or dizziness.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3. The idea that someone else can control your thoughts.</td>
<td></td>
<td></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>4. Feeling others are to blame for most of your trouble.</td>
<td></td>
<td></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>5. Trouble remembering things.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6. Feeling easily annoyed or irritated.</td>
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<tr>
<td>7. Pains in heart or chest.</td>
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<tr>
<td>8. Feeling afraid in open spaces or on the streets.</td>
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<tr>
<td>9. Thoughts of ending your life.</td>
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<td></td>
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</tr>
<tr>
<td>10. Feeling that most people cannot be trusted.</td>
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<tr>
<td>11. Poor appetite.</td>
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<td></td>
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</tr>
<tr>
<td>12. Suddenly scared for no reason.</td>
<td></td>
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<tr>
<td>13. Temper outbursts that you could not control.</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>14. Feeling lonely even when you are with people.</td>
<td></td>
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<tr>
<td>18. Feeling no interest in things.</td>
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<tr>
<td>20. Your feelings being easily hurt.</td>
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<tr>
<td>21. Feeling that people are unfriendly or dislike you.</td>
<td></td>
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<tr>
<td>22. Feeling inferior to others.</td>
<td></td>
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</tr>
<tr>
<td>23. Nausea or upset stomach.</td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>HOW MUCH WERE YOU DISTRESSED BY:</td>
<td></td>
<td></td>
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<tr>
<td>-----------------------------------</td>
<td></td>
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</tr>
<tr>
<td>24. Feeling that you are watched or talked about by others.</td>
<td></td>
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<tr>
<td>25. Trouble falling asleep.</td>
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<tr>
<td>26. Having to check and double-check what you do.</td>
<td></td>
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<tr>
<td>27. Difficulty making decisions.</td>
<td></td>
<td></td>
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<tr>
<td>28. Feeling afraid to travel on buses, subways, or trains.</td>
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</tr>
<tr>
<td>29. Trouble getting your breath.</td>
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<td></td>
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</tr>
<tr>
<td>30. Hot or cold spells.</td>
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</tr>
<tr>
<td>31. Having to avoid certain things, places, or activities because they frighten you.</td>
<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>32. Your mind going blank.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>33. Numbness or tingling in parts of your body.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>34. The idea that you should be punished for your sins.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>35. Feeling hopeless about the future.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>36. Trouble concentrating.</td>
<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>37. Feeling weak in parts of your body.</td>
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<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>38. Feeling tense or keyed up.</td>
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<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>39. Thoughts of death or dying.</td>
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<td>40. Having urges to beat, injure, or harm someone.</td>
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<td>41. Having urges to break or smash things.</td>
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<td>42. Feeling very self-conscious with others.</td>
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<td>43. Feeling uneasy in crowds, such as shopping or at a movie.</td>
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<td>44. Never feeling close to another person.</td>
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<td>45. Spells of terror or panic.</td>
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<td>46. Getting into frequent arguments.</td>
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<td>47. Feeling nervous when you are left alone.</td>
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<td>48. Others not giving you proper credit for your achievements.</td>
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<td>49. Feeling so restless you couldn't sit still.</td>
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<td>50. Feelings of worthlessness.</td>
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<td>51. Feeling that people will take advantage of you if you let them.</td>
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<td>52. Feelings of guilt.</td>
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<td>53. The idea that something is wrong with your mind.</td>
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Appendix 4: Young Schema Questionnaire: Short Form Version 3

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INSTRUCTIONS:
Listed below are statements that a person might use to describe himself or herself. Please read each statement and decide how well it describes you using the rating scale below. When you are not sure about how a statement applies to you, base your answer on what you emotionally feel, not on what you think to be true. Using the rating scale below, choose the highest rating from 1 to 6 that applies to you and write the number in the space before the statement.

RATING SCALE:
1 = Completely untrue of me
2 = Mostly untrue of me
3 = Slightly more true than untrue
4 = Moderately true of me
5 = Mostly true of me
6 = Describes me perfectly

1. _______ Most of the time, I haven't had someone to nurture me, share him/herself with me, or care deeply about everything that happens to me.
2. _______ In general, people have not been there to give me warmth, holding, and affection.
3. _______ For much of my life, I haven't felt that I am special to someone.
4. _______ For the most part, I have not had someone who really listens to me, understands me, or is tuned into my true needs and feelings.
5. _______ I have rarely had a strong person to give me sound advice or direction when I’m not sure what to do.
6. _______ I find myself clinging to people I’m close to because I am afraid they’ll leave me.
7. _______ I need other people so much that I worry about losing them.
8. _______ I worry that people I feel close to will leave me or abandon me.
9. _______ When I feel someone I care for pulling away from me, I get desperate.
10. _______ Sometimes I am so worried about people leaving me that I drive them away.
11. _______ I feel that people will take advantage of me.
12. _______ I feel that I cannot let my guard down in the presence of other people, or else they will intentionally hurt me.
13. _______ It is only a matter of time before someone betrays me.
14. _______ I am quite suspicious of other people’s motives
15. _______ I’m usually on the lookout for people’s ulterior motives
16. _______ I don’t fit in.
17. _______ I’m fundamentally different from other people.
18. _______ I don’t belong; I’m a loner.
19. _______ I feel alienated from other people.
20. _______ I always feel on the outside of groups.
21. _______ No man/woman I desire could love me once he/she saw my defects.
22. _______ No one I desire would want to stay close to me if he/she knew the real me.
23. _______ I’m unworthy of the love, attention, and respect of others.
24. _______ I feel that I’m not lovable.
25. _______ I am too unacceptable in very basic ways to reveal myself to people.

Please turn over and continue
26. I almost never finish schoolwork I'm assigned to do.
27. I'm incompetent when it comes to achievement.
28. Most other people are more capable than I am in areas or work and achievement.
29. I'm not as talented as most people are in their work.
30. I'm not as intelligent as most people when it comes to work (or school).
31. I do not feel capable of getting on in everyday life.
32. I think myself a dependent person, when it comes to everyday functioning.
33. I lack common sense.
34. My judgment cannot be relied upon in everyday situations.
35. I don't feel confident about my ability to solve everyday problems that come up.
36. I can't seem to escape the feeling that something bad is about to happen.
37. I feel that disaster (natural, criminal, financial, or medical) could strike at any moment.
38. I worry about being attacked.
39. I worry that I'll lose all my money and become destitute.
40. I worry that I am developing a serious illness, even though nothing serious has been diagnosed by a physician.
41. I have not been able to separate myself from my parent(s), the way other people my age seem to.
42. My parent(s) and I tend to be overinvolved in each other's lives and problems.
43. It is very difficult for my parent(s) and me to keep intimate details from each other, without feeling betrayed or guilty.
44. I often feel as if my parent(s) are living through me - I don't have a life of my own.
45. I often feel that I do not have a separate identity from my parents or partner.
46. I think if I do what I want, I'm only asking for trouble.
47. I feel that I have no choice but to give in to other people's wishes, or else they will retaliate or reject me in some way.
48. In relationships, I let the other person have the upper hand.
49. I've always let others make choices for me, so I really don't know what I want for myself.
50. I have a lot of trouble demanding that my rights be respected and that my feelings be taken into account.
51. I'm the one who usually ends up taking care of people I'm close to.
52. I am a good person because I think of others more than of myself.
53. I'm so busy doing things for the people that I care about that I have little time for myself.
54. I've always been the one who listens to everyone else's problems.
55. Other people see me as doing too much for others and not enough for myself.
56. I am too self-conscious to show positive feelings to others (e.g. affection, showing I care).
57. I find it embarrassing to express my feelings to others.
58. I find it hard to be warm and spontaneous.
59. I control myself so much that people think I am unemotional.
60. People see me as uptight emotionally.
61. I must be the best at most of what I do; I can't accept second best.
62. I try to do my best; I can't settle for "good enough".
63. I must meet all my responsibilities.
64. I feel there is constant pressure for me to achieve and get things done.

Please turn over and continue
65. I can't let myself off the hook easily or make excuses for my mistakes.
66. I have a lot of trouble accepting "no" for an answer when I want something from other people.
67. I'm special and shouldn't have to accept many of the restrictions placed on other people.
68. I hate to be constrained or kept from doing what I want.
69. I feel that I shouldn't have to follow the normal rules and conventions other people do.
70. I feel that what I have to offer is of greater value than the contributions of others.
71. I can't seem to discipline myself to routine or boring tasks.
72. If I can't reach a goal, I become easily frustrated and give up.
73. I have a very difficult time sacrificing immediate gratification to achieve a long-term goal.
74. I can't force myself to do things I don't enjoy, even when I know it's for my own good.
75. I have rarely been able to stick to my resolutions.

Thank you very much for answering these questions.
Appendix 5: Service audit consent form
Consent Form – Potential Research Projects

At some point in the future we may want to use this information for research purposes at this clinic. Your questionnaires will remain confidential and your name will not be used in any database. Your responses to the following questions will not affect your treatment at the Vincent Square Clinic in any way.

Do you give us permission to use the information in these questionnaires for the purposes of future research? Yes / No

Do you give us permission to contact you by letter to see if you would like to participate in additional further research? Yes / No

NAME...........................................................................................................
ADDRESS....................................................................................................
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DATE.............................................................................................................
SIGNATURE.................................................................................................
Appendix 6: Research patient information sheet
Information Sheet

Clinical Audit Project Vincent Square Clinic

The Vincent Square Clinic aims to provide effective treatment for eating disorders. It is important that we assess the outcome of treatment to help us to continue to improve the service we offer and to ensure that the standard of treatment remains high. This process of measuring treatment outcome is called Clinical Audit and the Department of Health encourages all services to conduct Clinical Audit projects.

We measure outcome of treatment by asking you to complete a series of questionnaires that assess the type and severity of eating disorder symptoms as well as related problems such as depressions, anxiety and low self-esteem. We would be grateful if you could complete these questionnaires before you attend for assessment so that we have a baseline measure. We will then ask you to complete the questionnaires again at intervals during treatment, at the end of treatment and at follow-up. This allows us to measure the change in your symptoms over the course of treatment. We are also interested to know how service users find the treatment we offer and so we have a ‘Satisfaction with Treatment Questionnaire’ in all but the initial assessment. The questionnaires are simple to complete and should only take about 15-20 minutes of your time.

The information you give us in these questionnaires will be kept securely and confidentially within the clinic. The information will be entered anonymously in a database. This allows us to track changes in your scores over time and your therapist or key worker may share these results with you in sessions. We aim to use the database to evaluate the effectiveness and acceptability of the treatments we offer and to examine specific research questions to better understand and improve treatment. The outcome of this research will be presented for publication.

We hope very much that you will be willing and able to help us evaluate the treatment that you receive in clinic. Your participation is voluntary. If you change your mind, you are free to withdraw from this project at any point without giving a reason and without it affecting your treatment. If you have any questions, please feel free to ask the member of the clinical team you see for assessment or treatment.

If you would like to take part, please read the attached consent form carefully before signing and returning with the completed questionnaires. Please keep this information sheet for your own information.

The consultants in charge of this project are:

Dr. Frances Connan (Consultant Psychiatrist)
Jane Faulkner (Consultant Psychologist)
Appendix 7: Research informed consent form
Dear Professor Waller

Title of project: Cognitive predictors of early weight change in anorexia nervosa
Clinical supervisor: Glenn Waller
Research student: Rebecca Lockwood, University College, London

This is to confirm that the R&D Department considers this project to be audit rather than research, as it involves the collection of data that are already gathered in routine clinical practice. Therefore, it is not relevant to submit this proposal for LREC approval.

It may be a good idea to inform the Audit Department at CNWL that an audit is going to be carried out.

Regards

Maria Tsappis
Research Governance Co-ordinator