Does Psychotherapy for Binge Eating Remediate Nutritional Intake in People with Bulimia Nervosa and Binge Eating Disorder?

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Abstract

People who binge eat (those with bulimia nervosa (BN) and binge eating disorder (BED)) have a significantly disturbed nutritional intake compared to people who don't binge eat. Moreover, people who binge eat consume excess energy, have altered macronutrient ratios, and have diets that are deficient in many vitamins and minerals. While the nutritional intakes of people who binge eat are well-researched, few studies contrast the nutritional intakes of people with BN and BED. Additionally, while cognitive behavioural therapy (CBT) is the most researched psychotherapy for binge eating-related disorders, little is known about the nutritional outcomes of this treatment. Appetite-focussed cognitive behavioural therapy (CBT-A) is an augmented version of CBT that provides modified nutritional advice and aims to improve response to cues of hunger and satiety. CBT-A has good treatment outcome, similar to that of traditional CBT, however whether there is a nutritional benefit attributable to the nutrition-related augmentations of CBT-A has not been investigated. A sample of 79 women with either BN (n = 38) or BED (n = 41) were randomised to CBT or schema therapy (n = 57), or to CBT-A (n = 22). Participants completed seven-day prospective food records pre-, mid-, and post-treatment and these records were entered into a nutritional software that provided average intakes of total energy, macronutrients and micronutrients. Nutrient intakes were compared to empirically-determined healthy intakes or ratios, and summary measures of macronutrient, micronutrient, and total adequacy were created. Nutritional intake and nutritional adequacy were modelled over time and between groups using linear mixed models analyses. Modelling nutritional variables suggested that psychotherapies for transdiagnostic binge eating reduced the energy, macronutrient, and mineral intakes over weekly sessions of treatment and many of these changes were maintained over monthly sessions. Vitamin intakes did not change despite reductions in food intake, and participants received less of their total energy from sugars, total fats, and

saturated fats, and more from protein. These changes occurred alongside increased macronutrient adequacy over the first half of treatment and reduced micronutrient adequacy over the first half of treatment, which was maintained at the end of treatment. Little separated people with BN and BED nutritionally, and nutritional responses to treatment did not differ between diagnostic groups. Nutritional responses to the two treatment types were indistinguishable. Results support the use of transdiagnostic nutritional advice, however, fail to support the modified nutritional advice of CBT-A. The current study broadens knowledge about the nutritional outcomes of treatments for binge eating and these results likely have implications in understanding the high medical burden associated with BN and BED.

Introduction

Binge eating involves the consumption of a large amount of food in a short period of time, accompanied by the sense of feeling out of control of one's eating behaviour. Those who binge eat have altered nutritional intake compared to people who do not, including disturbances in the intake of various macronutrients, micronutrients, and total energy. While the nutritional disturbances of people who binge eat are well documented, little research has investigated whether this disturbed nutrient intake impacts dietary adequacy. Such research would allow new insights into the association between disturbed nutrient intake and the poor psychological and physical health observed in people who binge eat.

While current first-line treatments for binge eating-related disorders are well established, little is known about the nutritional outcomes of these treatments, especially whether they lead to improved nutritional adequacy, the degree to which food intake meets health needs. While cognitive behavioural therapy (CBT) is the most empirically-validated psychotherapy for binge eating disorders, high drop-out rates (Linardon et al., 2018), and relapse rates (Södersten et al., 2017) are common, suggesting new alternatives are needed to improve treatment outcomes. Appetite-focussed cognitive behavioural therapy (CBT-A), is a variant of traditional CBT with increased focus on food selection and responding appropriately to internal cues of hunger and satiety, processes which are known to be disturbed in people who binge eat (Craighead & Allen, 1995; Dicker & Craighead, 2004; McIntosh et al., 2007). This form of treatment has similar outcomes to traditional CBT (McIntosh et al., 2007), however, while CBT-A places emphasis on selecting more satiating foods, no studies have evaluated the nutritional outcomes of this treatment.

Binge eating episodes characterise two recognised eating disorders: bulimia nervosa (BN) and binge eating disorder (BED; APA, 2013).

Bulimia Nervosa

Bulimia nervosa is characterised by both binge eating episodes and the presence of recurrent compensatory behaviours (APA, 2013). Binge eating behaviour in people with BN is associated with periods of restrictive food intake (Devlin, 1990). Retrospective age-at-onset data suggest that BN typically emerges in adolescence or young adulthood (Nagl et al., 2016), with a female:male gender ratio for lifetime BN diagnoses as high as 16.5:1 (Nagl et al., 2016). Lifetime prevalence of BN in adult females has been reported to be around 1% in the general New Zealand population (Bushnell et al., 1990; Oakley-Browne et al, 2006), and around 2.8% in young females. However, other estimates of lifetime prevalence of BN in women suggest around 1.5% (Hudson et al., 2007).

As well as being associated with many comorbid medical conditions, BN is associated with many additional psychiatric problems. Approximately 80% of those diagnosed with BN also experience another psychiatric illness throughout their lifetime (Fichter & Quadflieg, 1997), including problems with chemical dependency, anxiety disorders, personality disorders, and high rates of affective disorders, including major depression and dysthymia (Mitchell et al., 1991). While the psychiatric aftermath of the recent and ongoing global COVID-19 pandemic is still unknown, some theorise that rates of binge eating may be especially impacted due to the effect that newly enforced practices (e.g. social distancing) have on social networks (Juarascio et al., 2021). One study found a significant positive correlation between social distancing practices and binge eating behaviour (Chang et al., 2021). Binge eating is associated with impaired quality of life (Pollack, 2013), medical morbidity (Masheb & Grilo, 2004), and mortality (Crow et al., 2009; Herzog et al., 2000; Neilson et al., 1998), and these levels are expected to rise in the near future, which highlights the necessity of investigating effective prevention and maximising treatment outcomes for binge eating-related disorders.

Binge Eating Disorder

Binge eating disorder is characterised by recurrent binge eating episodes and by factors such as eating rapidly, eating until uncomfortably full, and eating when not hungry (APA, 2013). Under most circumstances, BED has been found to be the most common DSM-5 eating disorder (Hudson et al., 2007; Smink et al., 2014). On average, men engage in binge eating more often or at similar rates as women, however, men are less likely to experience the distress/loss of control associated with binge eating and therefore are less likely to meet full criteria for BED (Hudson et al., 2007; Ivezaj et al., 2010). Therefore, BED is more frequently seen to be diagnosed in women (3.5%) than in men (2%) and is found have a lifetime prevalence of 2.8% (Hudson et al., 2007). Prevalence rates significantly inflate to around 30% of those participating in weight loss programmes (Spitzer et al., 1993), whilst for those seeking bariatric surgery, prevalence rates have been seen to be as high as 50% (Palavras et al., 2011). There is debate as to whether there are significant differences in prevalence of BED between ethnic groups. Some accounts suggest higher rates of BED within the Caucasian female population (Napolitano & Himes, 2011; Sorbara & Geliebter, 2002), while others suggest that the rates are similar across Caucasian and African American populations (Alegria et al. 2007, Striegel-Moore et al., 2000).

As with BN, high psychiatric comorbidity is common with BED. Welch et al. (2016) found BED to be significantly associated with other eating disorders, major depressive disorder, bipolar disorder, anxiety disorders, post-traumatic stress disorder and elevated risk of suicide attempts.

Comorbid medical conditions are also frequent. Individuals with either BN or BED have demonstrated higher levels of metabolic dysfunction (Barnes et al., 2011; Guerdjiokiva et al., 2007; Roehrig et al., 2009), thyroid dysfunction (Altemus et al., 1996), abnormal lipid levels (Mitchel et al., 2015; Succurro et al., 2015), cardiovascular dysfunction (Mathisen et

al., 2018), menstrual dysfunction (Algars et al., 2014; Gendall et al., 2000; Mitchel et al., 2016), and increased physical measures, namely increased body mass index (BMI), percentage body fat, and weight (Cachelin et al., 2019; Lydecker, et al., 2019; Mathisen et al., 2018).

Nutritional Disturbances in Those Who Binge Eat

Nutrients are comprised of macronutrients and micronutrients (vitamins and minerals), all of which are reported to be disturbed in those who binge eat (Forbush et al., 2014). This consistent finding is important for several reasons. First, nutritional disturbances likely contribute to comorbid physiological disturbances that are common in those with eating disorders. For example, thyroid hormone levels have been associated with macronutrient intake in people who binge eat (Altemus et al., 1996; Spatler et al., 1993). Additionally, chronic pain, diabetes, musculoskeletal disorders, hypertension, and stomach ulcers are all at least 1.6 times more likely to occur in people with BN and BED (Kessler et al., 2013). It is likely that impairments in physical health originating from binge eating are exacerbated by co-occurring psychological and physical conditions (Herman & Bajaka, 2021), and more comorbid psychopathology leads to more severe eating problems and increased binge frequency (Lydecker & Grilo, 2021). Second, observed nutritional disturbances may further perpetuate disordered eating through disruption in the roles that nutrients play in the experience of satiety and hunger (Latner et al., 2008; Latner & Wilson 2004).

Measuring Food Intake

Studies that assess the nutrient intake of people who binge eat vary in their methods of dietary assessment. Many studies use laboratory-based experiments where the food content is predetermined, or retrospective food recalls where participants recall their eating habits, often after considerable delay. Laboratory studies do not represent real-world conditions for

nutritional intake in several ways. Notably, binge eating is context dependent, and a laboratory setting fails to provide a real-world, natural environment within which disordered eating usually occurs (Chami et al., 2021). The typical internal and external triggers for binge eating behaviour, as well as other maintaining mechanisms, may not present themselves within a laboratory setting (Chami et al., 2021; Haedt-Matt & Keel, 2011; Mitchell et al., 1998). Additionally, the presence of experimenters, or the process of being monitored may influence eating behaviour (Segura-García et al., 2014). Retrospective food recall has been found to be susceptible to under-reporting food intake by people who binge eat (Raymond et al., 2012).

Seven-day food records have been found to be the most reliable method of measuring food intake (Edington et al., 1989; Prentice et al., 2011) and are no less accurate than self-report measures lasting up to 16 days (Bingham et al., 1994). As a greater amount of food may be consumed on weekends compared to weekdays (Allison & Timmerman, 2005; Haines et al., 2003), prospective records that span seven consecutive days ensure that weekend days are included within analyses creating a more accurate representation of participant eating behaviours and dietary intake. Therefore, seven-day prospective food records provide a simple, yet accurate method of assessing dietary intake.

Macronutrients

Macronutrients are nutrients that are needed in the body in large amounts which provide energy for major bodily functions. The three main macronutrients ─ proteins, fats and carbohydrates, provide energy to the body and have several methods in which they are metabolised.

Foods Consumed. For people with BN, foods consumed during binge eating episodes consist mostly of breads, pastas, sweets and salty snacks (Allison & Timmerman, 2007), which tend to be high in fats, and simple and complex carbohydrates. Reeves et al. (2001)

found people with BN consume significantly more snacks and deserts and significantly fewer vegetables than people who do not binge eat. People with BED tend to consume more dairy products, even when compared to high weight samples (Raymond et al., 2007), and have been observed to consume more meat compared to weight-matched controls (Cooke et al., 1997). Ultra-processed foods that are highly associated with poor nutrition, have been shown to constitute approximately 70% of their overall diet of those with BN and BED (Ayton et al., 2021). Additionally, binge episodes were found to consist solely of ultra-processed foods that have higher levels of carbohydrates and fats (Ayton et al., 2021). Kales (1990) found that 69% of binge eating episodes consisted of foods that the participants classified as 'forbidden', compared with 15% of eating episodes that were not considered to be binge eating episodes. Forbidden foods were found to be higher in fat content and to have a higher caloric value than non-forbidden food items.

Macronutrient Intake. The total energy intake of people who binge eat has been consistently shown to be larger than the intake of those who do not binge (Cooke et al., 1997; Gendall et al., 1997; Latner et al., 2008; Siega-Riz et al., 2008). Moreover, the ratio of macronutrient intakes as a percentage of total energy have shown to be disturbed in people who binge eat. For example, compared to controls, people with BN have a higher percentage of dietary energy intake from fats and a lower percentage from proteins (Hetherington et al., 1994). Considerable variability is found in the macronutrient ratios of people who binge eat, of particular interest, the macronutrient ratios between binge and non-binge eating episodes, with one study finding higher percentages of energy from fat in binge eating episodes in people with BN (Alpers & Tuschen-Caffier, 2004). For people with BED, on days with binge eating episodes, more proteins, carbohydrates, and fats are eaten than on non-binge days and even more than high weight controls without BED. Intake for people with BN during binge eating episodes is, compared to the general population, significantly higher in percent of

energy from fats (saturated fatty acids and monounsaturated fatty acids) and carbohydrates, and lower in energy from protein (Gendall et al., 1997). Some studies have suggested that there is a low proportion of energy from protein in the diets of people who binge eat, however the findings are mixed (Bartholome et al., 2013; Cooke et al., 1997; Latner & Wilson, 2004; Reeves et al., 2001; Yanovski et al., 1992). Kaye et al. (1992) found that while individuals with BN consumed more energy from carbohydrates than controls, they consumed less energy from fats. Additionally, alterations in polyunsaturated fatty acids, especially a high dietary n-6:n-3 ratio, are more common in people who binge eat (Khoury et al., 2021), an important finding considering a well-balanced dietary n-6:n-3 ratio is essential for the development and functioning of the central nervous system (Bozzatello et al., 2019). The nutrient intake of people who binge eat has also been shown to influence the macronutrient composition and total energy levels of future binges (Gendall et al., 1999), possibly indicating self-perpetuating disordered food intakes. While the observations around disordered macronutrient compositions of binge eating individuals' diets are extensive, some studies find that the macronutrient content of food eaten by individuals who binge eat do not differ from the macronutrient content of non-binge eating controls (Alpers & Tuschen-Caffier, 2004; Horvath et al., 2015; Kaye et al., 1992; Raymond et al., 2003; Weltzin et al., 1991).

Energy/Caloric Intake.

The energy, or caloric, intake of people who binge eat has consistently been found to be disturbed. Within laboratory-based settings, people with BED and BN have been found to have significantly higher energy intake than controls (Bartholome et al., 2013; Bartholome et al., 2006; Raymond et al., 2007; Sysko et al., 2007). Outside of laboratory-based studies, high-weight individuals who binge eat consume significantly more calories during days with at least one binge eating episode than on days without. Additionally, people with BED have

been found to consume more food and energy (Engel et al., 2009; Guss et al., 2002; Rossiter et al., 1992; Yanovski et al., 1992), even when adjusting for body weight (Yanovski & Sebring, 1994). However, on days without binge eating episodes, Raymond et al. (2012) found the caloric intake of women with BED to be similar to that of high-weight individuals who do not binge eat.

In people with BN, caloric intake in a laboratory setting ranged between 7101 and 9260 kcal (kilocalories), and between 3030 to 4479 kcal per binge episode (Mitchel et al., 1998). However, studies that used self-report food records found significantly lower values (Mourilhe et al., 2021). Apers and Tuschen-Caffier (2004) found people with BN to have an average daily caloric intake ranging between 3117 and 4275 kcal and Mitchell et al. (1998) found the average caloric intake of a binge episode to be between 1173 and 2415 kcal. One review found the average caloric value of binge eating episodes across studies to be 2482 kcal for individuals with BN and 2048 kcal for individuals with BED (Forbush et al., 2014). The considerable variability in caloric intake of people who binge eat may be explained by many factors, including family functioning (Jaramillo et al., 2018), sleep quality and quantity (Cerolini et al., 2018), and the presence of night eating (Latzer et al., 2018). People with BN have a total energy intake significantly higher than those in the general population and more frequent binge eating is related to higher total energy intake (Gendall et al., 1997). Similar results have been found for people with BED (Latner et al., 2008). Siega-Riz et al. (2008) reported total energy intake to be higher in pregnant women with BED both before and after pregnancy, compared with women with no eating disorder. Additionally, Cooke et al. (1997) found individuals with BED to have a higher caloric intake than weight-matched controls. Loss of control over one's eating alone, an essential criterion for binge eating, is related to excess energy consumption (Hilbert et al., 2010).

Micronutrient Intake

Micronutrients comprise of both vitamins and minerals, and are essential to many bodily functions but are required in much smaller quantities compared to macronutrients. Compared to macronutrient and energy intake, the micronutrient intake of people who binge eat has not been as extensively researched. The studies available show disturbances in the quantity of many vitamins and minerals. Half of individuals with BN do not meet recommended intakes for vitamins A, B1, B2, B6, B12, C, D, E and folic acid (Phillip et al., 1989; Woell et al., 1989), and half of individuals with BN consume less than two thirds of the recommended daily intake for non-binge calcium, iron and zinc (Gendall et al., 1997). However, these studies are dated and few modern studies have investigated the micronutrient health of people with BN. Additionally, Alvarenga et al. (2003) found lower levels of iron, vitamin E, folate and magnesium compared to controls. Those with BED also consume lower levels of iron, calcium, folate, potassium, and vitamin C (Allen et al., 2013; Horvath et al., 2014; Siega-Riz et al., 2008). Such deficiencies in micronutrient intake persist despite an abnormally high-calorie diet (Woell et al., 1989), indicating that food selection is likely a key factor for explaining the deficits observed in this population.

Implications of Inadequate Eating

Nutritional Adequacy

Most studies that evaluate the nutritional intake of people who binge eat do so by measuring the average intake of each nutrient. Whilst this has some value, results provide little insight into the overall nutritional adequacy of the individual. Comparing the average daily nutritional intake of those who binge eat with empirically determined healthy intakes or ratios of nutrient intakes allows for the assessment of the individual's nutritional health and nutritional adequacy. Additionally, most assessments of nutritional status of people who binge eat are cross-sectional and do not provide multiple measures over time. Comparing

nutritional intakes with recommended intake levels allows for evaluation of different treatments to improve nutritional adequacy, and therefore the nutritional health of people who binge eat, rather than reporting ambiguous increases or decreases in average nutrient intake levels.

Physiological Outcomes of Inadequate Eating

Current popular measures of nutritional adequacy, such as recommended daily intakes and acceptable macronutrient distribution ratios, provide adequacy levels, ranges, or ratios of nutritional intake that relate to an individual's optimal physical health with an absence of negative medical outcomes due to the strong associations between nutritional adequacy (excessive or deficient energy, macronutrient and micronutrient intake) and physical health. Both excessive and deficient energy intakes are associated with poorer outcomes. Excessive energy intake, a phenomenon commonly observed in people who binge eat, is highly associated with the development of weight-related diseases, such as hypertension, abnormal triglyceride and cholesterol levels, type II diabetes, heart and liver disease, and osteoarthritis (Tsai et al., 2004). The considerable research about the association of macronutrient intake and health status predominately relates to the negative effects of excessive intake of specific macronutrients. For example, a meta-analysis concluded that there is a significant positive correlation between total fat intake, BMI, and waist circumference (Hooper et al., 2015). Cholesterol levels, a major predictor of cardiovascular health, are sensitive to the ratio of saturated to unsaturated fat intake (Müller et al., 2003) and are negatively associated with carbohydrate intake (Grundy, 1986). High blood pressure (both diastolic and systolic), an indicator of cardiovascular dysfunction, is predicted by a diet high in fat and by a low polyunsaturated fat to saturated fat ratio (Puska et al., 1983). The same study also showed that remediation of these dietary factors leads to restoration of normal blood pressure. Various micronutrient deficiencies are known to directly cause negative health outcomes.

Iron deficiency, for example, can give rise to a range of issues including paleness, fatigue, dyspnoea and headaches (Lopez et al., 2009). Additionally, negative impacts on immune, lung, cardiac, muscle and metabolic functioning have all been shown to be negatively impacted when vitamin D levels are poor (Berger et al., 2019).

While the relationship between inadequate eating and physiological status of the general population is well established, research concerning the relationship within the binge eating population is much more limited.

Psychological Outcomes of Inadequate Eating

The effects of inadequate diets are not limited to the physical health of an individual. Inadequate diets have been found to be associated with the development of psychological issues, many of which have high comorbidity with BN and BED. Omega-3 fatty acids such as docosahexaenoic acid and eicosapentaenoic acid have been found to be associated with the pathogenesis of many psychiatric disorders such as psychosis, major depression, bipolar disorder, anxiety disorders, obsessive–compulsive disorder, posttraumatic stress disorder, attention deficit hyperactivity disorder, autism spectrum disorders, substance abuse and borderline personality disorder (Bozzatello et al., 2016; Khoury et al., 2021), leading to increased interest in the role of inflammatory responses on mental health conditions. The body's inflammation response is dependent on a range of nutrients, including magnesium and omega fatty acids. One review (Marx et al., 2017) found evidence for the role of nutrients such as magnesium, iron, and zinc in preventing depression. The same review reported the evidence of nutritional interventions remediating mental health conditions, such as depression and anxiety. Although findings are mixed, this research emphasises the link between adequate diets and psychological health. Again, although the relationship between psychological health and nutritional adequacy is topical and growing a sizeable literature

base, the association of nutrition and psychological health is not well-explored within the binge eating population.

Cognitive Behavioural Therapy for Binge Eating

Of the many recognised treatments for binge eating, cognitive behavioural therapy (CBT) has the most empirical evidence for its efficacy in treating binge eating related disorders (Fairburn & Harrison, 2003; Iacovino et al., 2012; Linardon et al., 2017). Most comprehensively described by Fairburn et al.'s (2003) transdiagnostic cognitive behavioural model of eating disorders, disordered eating is brought about by the overvaluation of body shape and weight, as well as the individual's perceived ability to control these. Overvaluation encourages weight-control behaviour, including excessive dieting which may directly (through features of restrictive eating) or indirectly (through emotion regulation) influence binge eating behaviour. Additionally, perfectionism, low self-esteem, mood intolerance and interpersonal difficulties contribute to binge eating behaviour. Cognitive behavioural therapy for binge eating aims to alleviate binge eating by targeting both distorted cognitions and excessive dieting. Cognitive behavioural therapy helps the individual to identify and alter dysfunctional thinking patterns. This comprehensive method for treating binge eating also includes self-monitoring, defining and implementing normal eating behaviour, examining cue sequences, and relapse prevention (Fairburn et al., 1993). Traditional CBT for binge eating leads to complete binge abstinence in approximately 50% of individuals (Wilson et al., 2010), and these results remain two to four years post-treatment (Hilbert et al., 2012; Wilson et al., 2010).

Nutritional Outcomes

Psychotherapies such as CBT have widespread effects and can change eating behaviour, with CBT leading to reductions in fat intake in female athletes (Buffington et al., 2016). However, while treatments for eating disorders result in self-reported changes in food

intake (Rossiter et al., 1988), currently little is known of nutrition-related outcomes of psychotherapy for binge eating. Two studies have assessed the nutrient intake of people who binge eat both pre- and post-treatment. Masheb et al. (2011) found CBT and dietary counselling led to favourable differences in energy density, energy intake, fruit and vegetable intake, fat intake and hunger for high-weight individuals with BED. CBT and dietary counselling has also been shown to result in reduced energy, macronutrient, and sugar intake and increased in fruit intake for people with BED (Masheb et al., 2016). Neither of these studies compared CBT to a non-CBT control group, and the intervention combined CBT with dietary counselling, meaning the effect of CBT alone on dietary patterns in the binge eating population is unknown.

Differences in Treatment Outcomes Between Diagnoses

Much of the existing research about the outcome of CBT for binge eating has focussed either on a single population, BN or BED, or has combined both populations, thereby losing comparative data. Such comparisons would assist clinicians to understand how people with different binge eating disorders respond differentially to treatment and may allow for a more tailored delivery of treatment for binge eating.

Differences in Nutritional Outcomes

Between-diagnosis comparisons of treatment outcome are rare, however, no published studies have directly compared the dietary outcomes of CBT between binge eating diagnoses. Additionally, the two studies that compared dietary outcomes pre- and post-treatment only examined individuals with BED (Masheb et al., 2011; Masheb et al., 2016).

Limitations of Traditional CBT

Despite CBT being the recommended psychotherapy for binge eating, and many studies indicating CBT's superiority in alleviating binge eating symptomatology, 40 – 60% of patients still do not achieve abstinence from binge eating following CBT (Grilo et al., 2011;

Linardon & Wade, 2018). Additionally, high rates of dropout (Linardon et al., 2018), and relapse (Södersten et al., 2017) are common post-treatment but these observations are likely dependent on the quality of treatment (Mulkens et al., 2018). In relation to non-binging measures of treatment success, CBT does not seem to be effective in reducing the weight of high-weight binge eating individuals (Iacovino et al., 2012; Grilo et al., 2012; Wilson et al., 2010). One possible explanation is that CBT for binge eating may reduce distress around eating, without changing eating behaviour for some people (Walsh, 2011). This is important given that weight loss is indicative of caloric restoration in people who binge eat (Yanovski & Sebring, 1993) and current treatments may not be restoring the nutritional health of these individuals. These limitations highlight the need for adaptations of current treatments or the development of new treatments for eating disorders, alongside research that aims to understand nutritional outcomes of treatments for binge eating.

The Role of Appetite in Binge Eating

Individuals who binge eat have impairments in both satiety and hunger (McIntosh et al., 2007). People who binge eat often respond to signals of hunger and fullness when they are at extremes (Craighead & Allen, 1995), eating when hunger cues are absent or extreme and stopping eating when satiety is extreme. People who binge eat experience lower satiety after eating (Nakai et al., 1987; Halmi et al., 1989) and experience a lower-than-normal decrease in the desire for recently eaten foods (Hetherington & Rolls, 1989). The lack of awareness of internal cues of hunger and satiety is hypothesised to be a consequence of a history of both dieting and overeating (Dicker & Craighead, 2004). Dietary restriction and excessive dieting are common patterns among people who binge eat (Fairburn et al., 1993; Stice et al., 2001). Dieting leads to ignoring hunger cues to follow the rules of the current diet, weakening future internal awareness. Overeating involves eating until uncomfortably full, thereby learning to ignore sensations of moderate satiety (Lowe, 1993; Dicker &

Craighead, 2004). This deficit in internal awareness brings about disordered eating in two forms (Dicker & Craighead, 2004). First, the individual may only respond to hunger and satiety cues when they are extreme, rather than low or moderate. Second, due to ignoring internal cues, they may learn to organise their eating by focussing on non-appetite cues such as emotional states.

Binge eating is also hypothesised to be maintained by factors associated with food selection. Foods selected by people who binge eat during both binge and non-binge food consumption are typically less satiating than those chosen by people who do not binge eat (Rosen et al., 1986). During binge eating episodes, individuals with BN and BED consume more foods that they classify as forbidden (Lowe et al., 1990), which tend to be low in proportion of energy from protein (Hetherington et al., 1994; Latner & Wilson, 2004; Gendall et al., 1997), and more energy-dense (Alpers & Tuschen-Caffier, 2004), including foods such as breads and pastas (Allison & Timmerman, 2007), which provide a high glycaemic load, all of which are factors that contribute to either less satiation and less reduction in hunger. Aspects of binge eating, particularly the quantity of food ingested during binge eating episodes, are highly associated with gastric capacity (Geliebter et al., 2004), hypothesised to lead to slowed gastric emptying, which then in turn interferes with cholecystokinin (CCK) functioning, a neuropeptide essential to the experience of satiety (Geliebter & Hashim, 2001; Latner et al., 2008). Similarly, foods eaten during binge episodes typically have lower levels of protein (Gendall et al., 1997; Hetherington et al., 1994), and as protein also stimulates CCK response (Liddle, et al., 1985), foods eaten during binge eating episodes also likely lead to less satiation.

Using Appetite in the Treatment of Binge Eating

Few treatments for binge eating emphasise the role of appetite and hunger in the development and maintenance of binge eating-related disorders. Considering the limitations

of CBT, increasing the focus on disrupted internal cue recognition and responding may be warranted. Observed discrepancies between binge eating and non-binge eating individuals in responding to cues of hunger and fullness, along with differences in food choices that directly affect satiety and hunger, have led to several attempts to supplement traditional CBT with material that addresses these differences.

One study by Craighead and Allen (1995) found that administering a treatment plan that included both typical CBT techniques, and also helped patients to recognise internal hunger and satiety cues, led to a decrease in the frequency of binging behaviour and increased sensitivity to and reliance on internal cues. Supplementing traditional CBT by assisting with recognising hunger and satiety cues has been shown to reduce binge eating more than traditional CBT in individuals with BN (Ventura & Bauer, 1999) and the supplementation of traditional CBT with additional educational material about low-energy-dense diets has been demonstrated to decrease the energy density of participants' diets more than CBT alone (Lowe et al., 2008).

Appetite-Focussed Cognitive Behavioural Therapy

Appetite-focused cognitive behavioural therapy (CBT-A) augments traditional CBT with skills relating to recognising and appropriately responding to internal cues of satiety and hunger, and provides education about choosing more satiating foods. CBT-A aims to retrain cue recognition whereby the individual is able to recognise low to moderate levels of hunger and satiety rather than only eating when hunger is extreme or stopping eating when fullness is extreme. CBT-A also helps people to respond to internal hunger and satiety cues, rather than emotional and external cues for eating (Dicker & Craighead, 2004). Participants are also provided education about food choices that maximise satiety by choosing foods high in protein, low in energy density, and that have a low glycaemic load.

Outcomes of Therapy Focussing on Appetite Cues

Appetite-focussed therapies have been found to significantly reduce or eliminate binge eating in eating disorder populations (Allen & Craighead, 1999; Craighead & Allen, 1995; Craighead et al., 2002; McIntosh et al., 2007). Individuals may also find CBT-A more tolerable than traditional CBT for binge eating, as the food monitoring component in CBT may be viewed as repetitive and similar to dieting (Dicker & Craighead, 2004). In one randomised controlled trial, participants with BN responded favourably to a 12-session course of appetite-focussed therapy (Dicker & Craighead, 2004). Compared to waitlisted controls, participants who engaged in the appetite-focussed therapy had significantly greater reductions in binge eating, purging, and all secondary measures of eating disorder and associated symptomatology. Additionally, 62% of participants fully recovered (77% of participants remitted) and none of the 26 women dropped out of treatment. Furthermore, individuals with BED appear to respond favourably to appetite-focussed therapy (Elder & Craighead, 2003), with reductions in binge frequency, eating disorder symptomatology and general psychopathology, and additional improvement in perceived body image. Although these improvements were maintained at 4-month follow-up, they were not found to be significantly greater than participants who received typical CBT for binge eating. Although CBT-A aims to remediate the dietary patterns and appetite sensations in people who binge eat, and despite the strong link between faulty satiation processes and food intake, there are currently no reports of nutritional or dietary outcomes in either the BN or BED groups.

The Current Study

The current study investigated three questions. First, whether psychotherapeutic treatments (CBT, schema therapy (ST), and CBT-A) for binge eating alter nutritional intake and nutritional adequacy of people who binge eat. It is expected that nutritional adequacy (total nutritional adequacy, macronutrient adequacy, and micronutrient adequacy) will

improve post-treatment. Second, whether there is a difference in the extent to which treatment remediates nutritional intake between diagnostic groups (BN vs. BED). Third, whether CBT-A, an augmented version of CBT that emphasises improving response to hunger and satiety cues and providing additional education about nutritional intake, is more effective in improving nutrition than the more traditional nutritional advice given in CBT and ST.

Method

Participants

Participants in the current study were part of a randomised clinical trial conducted in Christchurch, New Zealand which evaluated psychotherapy treatments in a binge eating sample (McIntosh et al., 2016). Participants were referred from their general practitioners or other health professionals or recruited via advertising. Included in the original study were women aged between 16 and 65, with a primary DSM-IV diagnosis of BN or BED, with objective binge episodes, and a BMI above 17.5. Exclusion criteria were other conditions requiring immediate treatment: severe major depression or serious suicidal intent, severe psychoactive substance dependence, bipolar I disorder, schizophrenia, severe physical illness, severe medical complications of the eating disorder, cognitive impairment, the use of psychotropic medications, or treatment with CBT or schema therapy in the past year. One hundred and twelve women in the original trial completed food records, 79 of whom met the minimum therapy requirements of attending at least 15 therapy sessions, see Figure 1.

Procedure

Initial telephone screening determined the likely presence of binge eating, the presence of inclusion criteria and absence of exclusion criteria. Participants who were thought to be appropriate candidates for the study following telephone screening then completed a clinical assessment, during which eligibility was confirmed and written informed consent was obtained.

Treatment

The clinical trial had a three-arm parallel group design whereby participants were randomly allocated to one of three treatment conditions (CBT, ST or CBT-A) according to a 1:1:1 ratio. Therapy consisted of six months of weekly individual therapy sessions followed by six months of monthly individual sessions. The three therapies were administered by four

clinical psychologists, all experienced in the three forms of therapy. The three therapies included common components targeting core transdiagnostic symptoms of BN and BED.

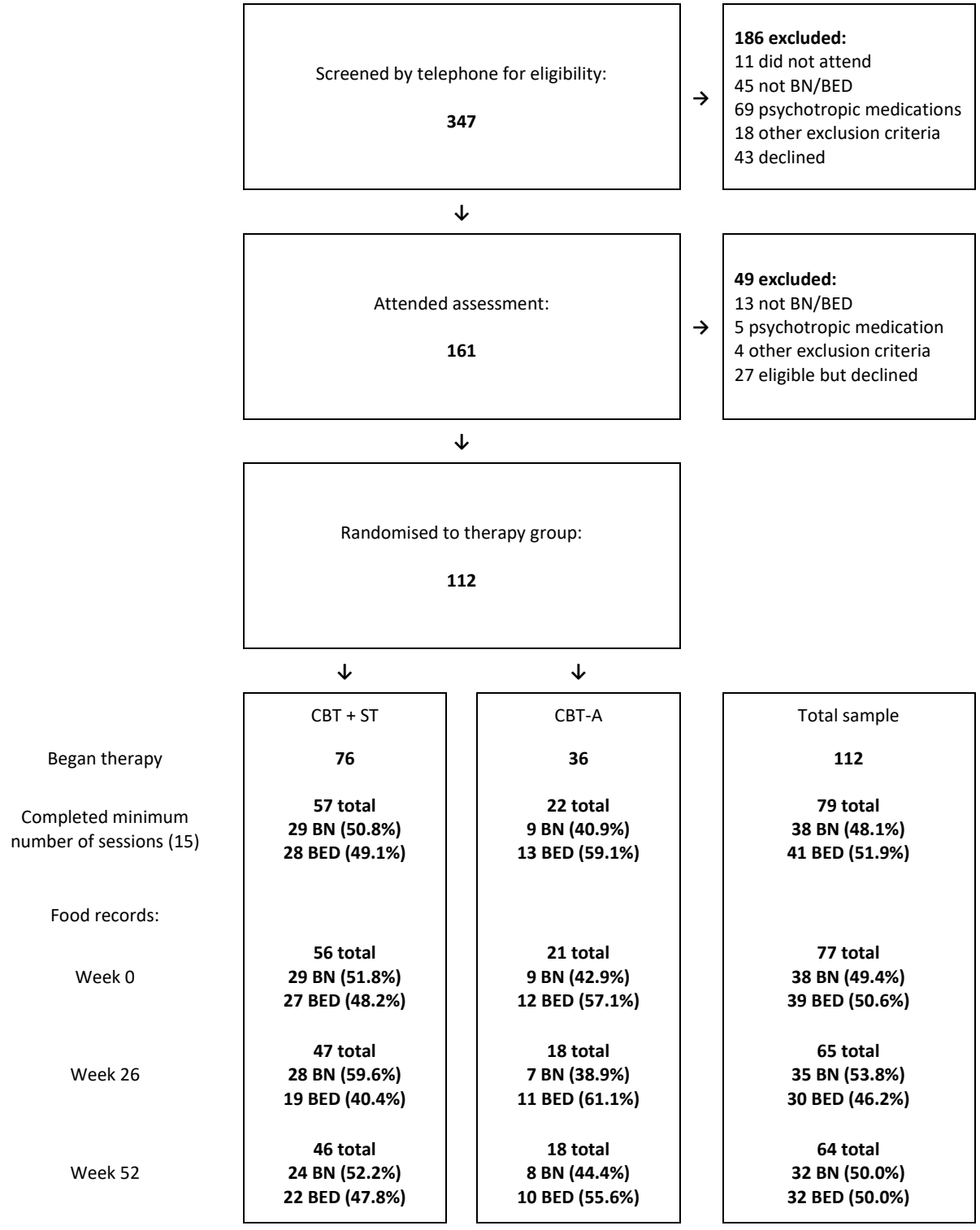
In the current study an important distinction was made between CBT or ST and CBT-A. This was due to the amended nutritional education and the appetite-focussed content of CBT-A, as CBT-A augments traditional CBT in three key ways. First, in CBT-A therapists provide clients with modified nutritional advice, including discussions of food pyramids, education about the role that protein and energy dense foods have on satiety, and education about choosing foods with carbohydrates that have a lower glycaemic index, as these foods provide more long-lasting energy. Patients are encouraged to include more satiating foods in their intake, with the recommendation that they receive at least 20% of their daily energy intake from protein. Second, self-monitoring food and fluid intake is taught with the modification of recording levels of appetite and satiety throughout the day, and before and after eating. Education is provided about the role of hunger and satiety in binge eating, the importance of regular eating, and the importance of starting and stopping eating in response to moderate levels of hunger and fullness. Lastly, patients are provided with protein-rich supplements. These include high-protein snack bars, nuts, cheese, and high-protein drinks. Participants are instructed and encouraged to include these foods as replacements for all or parts of meals and snacks, and are encouraged to slowly transition to incorporating these foods into their daily diet. Therefore, participants who were randomised to either CBT or ST treatment types were combined into a single group and compared to participants who were randomised to CBT-A.

Measuring Food Intake

Within the initial assessment, a research assistant explained and demonstrated how to complete the food records with each participant. Each participant was given a food

Figure 1

Flow Chart Depicting the Number of Participants who were Screened for Eligibility, Attended the Initial Assessment, Initially Engaged in Treatment, and who Completed the Required Amount of Therapy.



	CBT + ST	CBT-A	Total sample
Began therapy	**76**	**36**	**112**
Completed minimum number of sessions (15)	**57 total** **29 BN (50.8%)** **28 BED (49.1%)**	**22 total** **9 BN (40.9%)** **13 BED (59.1%)**	**79 total** **38 BN (48.1%)** **41 BED (51.9%)**
Food records:			
Week 0	**56 total** **29 BN (51.8%)** **27 BED (48.2%)**	**21 total** **9 BN (42.9%)** **12 BED (57.1%)**	**77 total** **38 BN (49.4%)** **39 BED (50.6%)**
Week 26	**47 total** **28 BN (59.6%)** **19 BED (40.4%)**	**18 total** **7 BN (38.9%)** **11 BED (61.1%)**	**65 total** **35 BN (53.8%)** **30 BED (46.2%)**
Week 52	**46 total** **24 BN (52.2%)** **22 BED (47.8%)**	**18 total** **8 BN (44.4%)** **10 BED (55.6%)**	**64 total** **32 BN (50.0%)** **32 BED (50.0%)**

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

monitoring booklet which had been developed for use in an existing study (Edington et al., 1989; Appendix A). The monitoring booklet included instructions about how to accurately measure food and fluid quantities, the descriptive information to be recorded such as brand name and cooking method, and photographs that contained various foods displayed in three different sized portions. Participants were required to report quantities of their meal and ingredients using either conventional measurement units, by volume or weight (either metric or imperial) or with reference to one of the portion sizes from the photographs ─ for example, *photograph 12, portion B.* During this initial session, participants were asked to recall their most recent food and fluid intake as a model of how to complete the food record. Participants were instructed to complete the record of their food and fluid intake for the next seven consecutive days. After approximately 24 hours of recording, participants were phoned by the research assistant and their food record was reviewed, with the research assistant prompting the participant to include additional information to ensure sufficient detail was provided. Opportunities were provided for participants to ask questions. To minimise underreporting of food intake due to shame or fear of judgement, research assistants modelled acceptance of large quantities of food intake such as during binge eating episodes. After a further seven days of recording their food and fluid intake, participants were provided opportunities to ask questions about the process. Food records were completed by participants during assessments at weeks 26 and 52 (at the end of six months of weekly therapy sessions and at the end of a further six months of monthly therapy sessions). Ten participants (12.7%) completed one record, 11 participants (13.9%) completed two, and 58 participants (73.4%) completed records at all three time points.

Dietary information obtained from the prospective food records was entered into the nutrition analysis software, FoodWorks 10 (Xyris Software, 2007), by a team of 6 data enterers. A spreadsheet was created to maintain consistency in the decisions made when

selecting representative foods from the FoodWorks-associated databases and decisions made when selecting appropriate and equivalent serving sizes for each food. This was so that when records did not include enough information about specific foods, or when no representative foods from the FoodWorks-associated databases exactly matched the specified food, decisions would be standardised. These decisions were reviewed regularly in meetings. Nutritional data were imported into Microsoft Excel, and nutritional variables were summed for each day and then averaged per day for each participant. This provided output of average daily macronutrient, micronutrient and energy intake levels. Percentage of energy from each macronutrient was then calculated by multiplying the weighted intake for each macronutrient at each time point by the associated energy density value for each macronutrient (carbohydrates = 16.7kJ/g, protein = 16.7kJ/g, and fats = 37.7 kJ/g). These values were then divided by the total energy intake and the multiplied by 100 to give the percent of total energy from each macronutrient. For each of the time points, weeks 0, 26, and 52, means and standard deviations for each nutritional variable were calculated for the two diagnostic groups (BN and BED), for the two treatment types (CBT + ST and CBT-A) and for the total sample. The 43 nutritional variables used in the current study are listed in Table 1, along with their units of measurement.

Ethical Consultation

The original study from which data for the current study are derived received ethical approval from the Upper South A Regional Health and Disability Ethics Committee (see Appendix B). The original clinical trial approval process included consultation with iwi Māori (see Appendix C). An exemption from further ethical approval was obtained from the University of Canterbury Human Ethics Committee (see Appendix D).

Measures

Demographic and Clinical Measures

During the clinician assessment, participants were administered the Structured Clinical Interview for DSM-IV (SCID; First et al., 1996). This assessment included demographic information (marital status, age, ethnicity, employment status and education).

Table 1

List of Nutrient Variables and their Associated Units of Measurement.

Macronutrients	Unit	Micronutrients	Unit
Average energy intake	kJ	Vitamins	
		Thiamine	mg
Macronutrient weights		Riboflavin	mg
Total protein	g	Niacin	mg
Total carbohydrates	g	Vitamin C	mg
Sugar	g	Vitamin E	mg
Total fat	g	Vitamin B6	mg
Saturated fat	g	Vitamin B12	µg
Trans fat	g	Folate	µg
Monounsaturated	g	Retinol	µg
Polyunsaturated	g		
Omega-6 fatty acids (N6)	g	Minerals	
3 α-Linolenic acid (ALA)	g	Sodium	mg
		Potassium	mg
Macronutrient (% of total kJ)		Magnesium	mg
Total protein	%kJ	Calcium	mg
Total carbohydrates	%kJ	Phosphorus	mg
Sugar	%kJ	Iron	mg
Total fat	%kJ	Zinc	mg
Saturated fat	%kJ	Selenium	µg
Trans fat	%kJ	Iodine	µg
Monounsaturated	%kJ		
Polyunsaturated	%kJ	Adequacy measures	
Omega-6 fatty acids (N6)	%kJ	Total adequacy	Number of
N3 α-Linolenic acid (ALA)	%kJ	Macronutrient adequacy	adequate
		Micronutrient adequacy	nutrients

Note. kJ = Kilojoules; g = grams; %kJ = percent of total energy; mg = milligram; µg = microgram.

The clinician interview also assessed past (lifetime) and current (past month) psychiatric diagnoses, including BN, BED, anorexia nervosa, major depression, bipolar I disorder, bipolar II disorder, anxiety disorders and substance use disorders. The presence of previous suicide attempts and deliberate self-harm was also recorded.

Nutritional Measures

Micronutrient Adequacy. To assess whether individuals' intake of different micronutrients were adequate, the nutritional adequacy ratio (Guthrie & Scheer, 1981) was calculated for each nutrient for each participant at the three time points. The nutritional adequacy ratio is calculated by dividing the average daily intake of a given nutrient by the recommended daily intake and multiplying this number by 100. Recommended daily intake values represent the average daily intake of a given nutrient that is sufficient to meet the requirements in 97-98% of healthy individuals (National Health and Research Council, 2017). Where there are no empirically supported recommended daily intake values for nutritional variables, the adequate intake value was used, which is the average daily intake of a given nutrient in a group of apparently healthy individuals that is assumed to be adequate (National Health and Research Council, 2017). For micronutrients where there is empirical evidence to suggest negative side-effects of excessive intake, and where this point of excessive intake has been experimentally defined, the upper limit value was converted to a nutritional adequacy ratio value. The upper limit represents the highest intake of a nutrient that is likely to pose no adverse health effects on almost all members of the population (National Health and Research Council, 2017). The recommended daily intake was divided by the upper limit, and multiplied by 100 to give a nutritional adequacy ratio value that represents an upper limit.

For micronutrients, adequacy was determined by ascertaining whether the average daily intake is equal to or greater than the nutritional adequacy ratio value of 90, or falls

between 90 and the nutritional adequacy ratio determined by the upper limit (for variables with an upper limit). Ninety was used, as values over this point indicate the participant has consumed at least 90% of the recommended daily intake or adequate intake. This process generated a new set of dichotomous variables that indicated whether participants had adequate average intakes for each nutrient.

Total Energy Adequacy. Adequate energy consumption was determined by assessing whether the individual's average daily energy intake fell within 10% of their required energy intake. The required energy intake was calculated by multiplying the basal metabolic rate, calculated by the Schofield equations (Schofield, 1985), by the participant's physical activity level. Basal metabolic rates were calculated by using an artificial body weight that would give a body mass index value of 22, given the participant's actual height. This was done so that required energy intake values would reflect an energy intake that is required to maintain the weight of a healthy weighted individual. Heavier weights provide higher required energy intakes due to needing a higher energy intake to maintain a heavier weight. However, required energy intakes calculated with unusually high weights may reflect an unhealthy/inadequate diet. Similarly, lower weights would provide lower required energy intakes, which may be inadequate and not correspond to recommended intakes for healthy weight ranges. Therefore, using this manufactured weight provides a required energy intake for an individual with a healthy weight, given their height.

Physical activity levels were generated by transforming a self-report five-point measure of physical activity (1 = none or very little; 2 = less than recommended but some exercise on a regular basis; 3 = meets recommended level (30 to 60 minutes most days per week); 4 = exceeds recommended level but not extremely so; 5 = well in excess of recommended level) into a new five-point scale (1.4 = very sedentary; 1.6 = light activity; 1.8 = moderate activity; 2.0 = heavy activity; 2.2 = vigorous activity). The calculated basal

metabolic rate was then multiplied by the new physical activity level value to provide a required energy intake value (required energy intake = basal metabolic rate × physical activity level). The required energy intake values were then multiplied by 0.9 and 1.1 to form the lower and upper limits of the adequacy range, respectively. Average daily energy intakes within the 90 – 110% range of the individual's required energy intake were considered adequate.

Macronutrient Adequacy. Due to both a lack of data and to the nature of macronutrient needs, which are highly dependent on factors such as age, height, weight, and physical activity level, global empirically validated recommended quantities of macronutrients are limited. Instead, acceptable macronutrient distribution ratios are often used to evaluate the adequacy of macronutrient intake. Acceptable macronutrient distribution ratios are percentile values (minimums or maximums) for a given macronutrient that represent a percentile range of total energy intake from that macronutrient that both maximise general health outcomes for the individual and would allow for the adequate intake of all other nutrients (National Health and Research Council, 2017). These can have only minimum energy requirements, only maximum requirements, or both, providing an adequacy range. Participants were categorised as adequate if they fell within the acceptable macronutrient distribution ratio for that macronutrient and as inadequate if they fall below the minimum or above the maximum.

Overall Adequacy. Three different summations of the adequacy variables were calculated, including total number of macronutrients expressed as a percentage of total energy in the adequate range (ranging from 0 to 10), total number of micronutrients in the adequate range (ranging from 0 to 18), and total number of nutrients in the adequate range (ranging from 0 to 29). Higher numbers on these measures reflect more nutritionally adequate dietary intakes while lower numbers reflect less nutritionally adequate dietary intakes.

Statistical Analyses

Means and standard deviations, and frequencies and percentages were calculated for demographic and psychiatric diagnoses for the total sample, and for the two diagnostic and treatment groups. Comparisons were made between diagnostic groups and treatment types using chi-square tests of independence for categorical variables and independent samples t-tests for continuous variables. Analyses of demographic and psychiatric variables were conducted using SPSS (IBM, 2020).

Linear mixed models were fitted using the 'nlme' package (Pinheiro et al., 2015) within the statistical computing software, R (R Core Team, 2020), for each of the 39 nutrient variables and the three adequacy summary variables (total adequacy, total macronutrient adequacy, and total micronutrient adequacy). Linear mixed models are used to model hierarchical data, a structure of data that is common in psychological studies, such as the current study, where multiple observations are nested within participants, which are nested within groups. These models are 'mixed' models as they allow for the inclusion of both fixed (population-level) and random (individual-level) effects. While repeated measures analyses of variance (rm-ANOVA) allow for within-participants effects, such as time, to be quantified alongside between-subject effects, such as group allocation, linear mixed models are also capable of this with several benefits over methods such as rm-ANOVA. Repeated-measures ANOVA, although capable of modelling variability at both the participant and item levels, are incapable of modelling these simultaneously (Brown, 2021). Additionally, ANOVA deals with missing data by listwise deletion, meaning only participants with observations at every point can be included in the analysis. Linear mixed models can tolerate missing observations meaning sample size is not limited to cases with full sets of observations, increasing the power of analyses with missing data. Additionally, linear mixed models are capable of handling unbalanced designs where groups differ in size, as is the case with the current study.

In the current study, three predetermined linear mixed effects models were fitted for each of the 42 dependent variables (39 nutrient intake variables and three measures of nutritional adequacy, see Table 1) using maximum likelihood estimation. Each macronutrient was modelled in two forms, weighted intake and the percent of total energy from the given macronutrient. For each variable, Model 1 included random intercepts and fixed slopes. Random intercepts allow for variation in initial observations. Ideally, within-participant variables such as time should be modelled as a random effect, however, post-hoc testing can only be conducted with factors that are modelled as fixed effects. Post-hoc testing of these models was conducted to assess whether nutrient intake or nutritional adequacy varied over treatment. Following this, a second set of models (Model 2 for each variable) was created by including diagnostic group (BN vs. BED) and a time × diagnosis interaction effect in the original model, both as fixed effects. Post-hoc testing was conducted with these models to assess whether diagnostic groups differed in their nutrient intake or adequacy at baseline (week 0) or in their trajectories of nutrient intake and adequacy over time. The final set of models (Model 3 for each variable) added treatment group (CBT + ST vs. CBT-A) and a time × treatment interaction effect to Model 1 as fixed effects. Post-hoc testing was conducted with Model 3 for each variable to assess differences between treatment groups in values of the dependent variables at baseline (week 0), and to assess whether treatment types differ in their trajectories over time. Tukey tests were unable to be conducted on interaction effects, therefore Bonferroni corrections (Bonferroni, 1936) were made to the alpha values of these comparisons. Due to having two observations for each interaction effect (differences in groups between weeks 0 and 26, and differences in groups between weeks 0 and 52), the alpha criterion was divided by two meaning that differences between groups in changes over time were considered statistically significant if $p < .01$.

Model residuals were extracted for each of the models and were plotted. Plots were inspected for outliers and non-random patterns of residuals. If patterns of residuals were present, dependant variables were transformed if appropriate and models were re-examined. Outlying observations were inspected and if these values were considered to be likely inaccurate, were removed. Models were then re-run and re-inspected.

Post-hoc tests were completed using the 'estimated marginal means' (emmeans) package in R (Lenth et al., 2021), which computes the estimated marginal means for factors within linear models and compares and contrasts them using Tukey HSD (honest significant difference) tests, which account for multiple comparisons. The emmeans package has variability in how many decimal places are provided in the output (β and *SE*). Results are reported with two decimal places, however, where results are not available with two decimal places, they are reported with as many decimal places as possible.

Results

Demographic Analyses

Means and standard deviations for continuous variables, and frequencies and percentages for categorical variables were calculated for demographic information. No significant differences in either means or frequencies were observed across diagnostic or treatment groups on any demographic measures (see Table 2).

As expected, both lifetime ($\chi^2(1, 79) = 44.83$, $p < .001$, $V = .753$) and past-month ($\chi^2(1, 79) = 79.00$, $p < .001$, $V = 1.00$) diagnoses of BN were significantly higher in the BN group, and both lifetime ($\chi^2(1, 79) = 71.36$, $p < .001$, $V = .950$) and past-month ($\chi^2(1, 79) = 75.10$, $p < .001$, $V = .975$) diagnoses of BED were significantly higher in the BED group. Additionally, lifetime diagnoses of anorexia nervosa were more common in the BN group ($\chi^2(1, 79) = 4.55$, $p = .033$, $V = .240$), see Table 3. On all other psychiatric measures, there were no statistically significant differences between diagnosis or treatment groups.

Nutrient Intakes and Adequacy Measures

Means and standard deviations for total energy intake, weighted macronutrient intakes, and macronutrients expressed as a percentage of total energy were calculated for the total sample at the three time points (see Table 4), for the two diagnostic groups (see Table 6), and for the two treatment types (see Table 8). Means and standard deviations for micronutrients were also calculated for the three time points for the total sample (see Table 5), for the two diagnostic groups (see Table 7), and for the two treatment types (see Table 9). Numbers of individuals and percentages of the total sample with adequate intakes of each nutrient were calculated for each nutrient at each time point (see Table 10). These were also calculated for the two diagnostic groups (see Table 11) and for the two treatment types (see Table 12). Means and standard deviations were calculated for the three adequacy measures at

each time point for the total sample (see Table 13), for the two diagnostic groups (see Table 14), and for the two treatment types (see Table 15).

Linear Mixed Models

Multilevel linear mixed effects models and post-hoc testing were used to characterise all 43 nutritional variables listed in Table 1. Model 1 was created to characterise the individual effect of time (weeks 0, 26, and 52) on these variables. Model 2 was created to determine whether a significant amount of variation in each dependent variable was due to diagnostic group membership (BN vs. BED), and to determine whether groups varied in their change over treatment (time × diagnosis interaction effects). Model 3 was created to determine if a significant amount of variation in the dependent variable was due to treatment type (CBT + ST vs. CBT-A), and to determine whether the two treatment groups varied in their change over time (time × treatment type interaction effects). Residuals plots for each model were inspected. No patterns in residuals were detected for any of the models created. While outliers were observed in some residual plots, when the associated nutrient intakes or adequacy scores were examined, these outlying values were determined to be plausible given the limits of human consumption and were therefore retained.

Total Energy Intake

Model 1 indicates that there is a significant reduction in total energy intake (β = -2525.81, $p < .001$) from week 0 to week 26. This reduction was maintained at week 52, as shown by a significant decrease between weeks 0 and 52 (β =-458.05, $p = .001$), and no significant difference in total energy intake between weeks 26 and 52. The set of models for total energy intake are depicted in Table 16.

Model 2 for total energy intake shows no significant difference in total energy intake between diagnostic groups (BN vs. BED) at week 0, or in how these groups changed over time on total energy intake.

Table 2

Demographic Characteristics of the Sample of 79 Women and t-tests and Chi-square Tests Between Participants with Bulimia Nervosa and Binge Eating Disorder and Between Participants that were Randomised to CBT + ST vs. CBT-A.

	BN (*n* = 38)	BED (*n* = 41)				CBT + ST (*n* = 57)	CBT-A (*n* = 22)				Total sample (*n* = 79)
	M (*SD*)/*n* (%)	*M* (*SD*)/*n* (%)	*t(df)*/$\chi^2(df)$	*p*	Cohen's *d*/ Cramer's *V*	*M* (*SD*)/*n* (%)	*M* (*SD*)/*n* (%)	*t(df)*/$\chi^2(df)$	*p*	Cohen's *d*/ Cramer's *V*	*M* (*SD*)/*n* (%)
Age (years)	32.92 (12.04)	38.20 (12.41)	-1.92 (77)	.059	-0.43	35.82 (12.34)	35.23 (12.98)	0.19 (77)	.850	0.05	35.66 (12.44)
Education (years)	14.99 (2.21)	15.38 (3.49)	-0.60 (68.23)	.551[c]	-0.13	15.21 (2.56)	15.13 (3.81)	0.10 (77)	.921	0.03	15.19 (2.93)
Marital status			1.51 (3)	.680	1.38			0.60 (3)	.883	0.09	
Married[a]	18 (47.4)	18 (43.9)				25 (43.9)	11 (50.0)				36 (45.6)
Separated	4 (10.5)	2 (2.5)				4 (7)	2 (9.1)				6 (7.6)
Divorced	2 (5.3)	4 (9.8)				5 (8.8)	1 (4.5)				6 (7.6)
Never married	14 (36.8)	17 (41.5)				23 (40.4)	8 (36.4)				31 (39.2)
Ethnicity			2.37 (3)	.499	0.17			2.70 (3)	.441	0.19	
NZ European	29 (76.3)	32 (78.0)				42 (73.7)	19 (86.4)				61 (77.2)
Māori	2 (5.3)	2 (4.9)				4 (7.0)	0 (0)				4 (5.1)
Chinese	2 (5.3)	0 (0)				2 (3.5)	0 (0)				2 (2.5)
Other	5 (13.2)	7 (17.1)				9 (15.8)	3 (13.6)				12 (15.2)
Employment			6.91 (4)	.141	0.30			2.90 (4)	.574	0.19	
On benefit[b]	3 (7.9)	4 (9.8)				4 (7.0)	3 (13.6)				7 (8.9)
Unemployed	0 (0)	3 (7.3)				2 (3.5)	1 (4.5)				3 (3.8)
Student	10 (26.3)	6 (14.6)				10 (17.5)	6 (27.3)				16 (20.3)
Housewife	2 (5.3)	7 (17.1)				6 (10.5)	3 (13.6)				9 (11.4)
Employed	23 (60.5)	21 (51.2)				35 (61.4)	9 (40.9)				44 (55.7)

Note: NZ = New Zealand; BN = bulimia nervosa; BED = binge eating disorder; BN = bulimia nervosa; BED = binge eating disorder; CBT = cognitive behavioural therapy; ST = Schema therapy; CBT-A = appetite-focussed cognitive behavioural therapy.

[a]or living in the same house for 1+ years; [b]other than unemployment benefit; [c]equal variances not assumed and adjusted degrees of freedom used.

Table 3

Psychiatric Diagnoses for the Sample of 79 Women, with Numbers and Percentages for the Total Sample, Bulimia Nervosa and Binge Eating Disorder Groups, and CBT + ST and CBT-A Groups, and with χ2 Statistics, Associated p-values, and Effect Sizes for the Difference in Proportions Between Diagnostic and Treatment Groups.

Diagnosis	BN (n = 38) *n* (%)	BED (n = 41) *n* (%)	χ^2(*df* = 1)	*p*	Cramer's *V*	CBT + ST (*n* = 57) *n* (%)	CBT-A (*n* = 22) *n* (%)	χ^2 (*df* = 1)	*p*	Cramer's *V*	Total Sample (*n* = 79) *n* (%)
Bulimia nervosa											
Lifetime	38 (100)	11 (26.8)	44.83	<.001	.753	37 (64.9)	12 (54.5)	0.72	.395	.096	49 (62.0)
Past month	38 (100)	0 (0)	79.00	<.001	1.000	29 (50.9)	9 (40.9)	0.63	.427	.089	38 (48.1)
Binge eating disorder											
Lifetime	2 (5.3)	41 (100)	71.36	<.001	.950	30 (52.6)	13 (59.1)	0.27	.605	.058	43 (54.4)
Past month	0 (0)	40 (97.6)	75.10	<.001	.975	28 (49.1)	12 (54.5)	0.19	.666	.049	40 (50.6
Anorexia nervosa											
Lifetime	4 (10.5)	0 (0)	4.55	.033	.240	4 (7.0)	0 (0)	1.63	.202	.143	4 (5.1)
Past month	0 (0)	0 (0)	-	-	-	0 (0)	0(0)	-	-	-	0 (0)
Major depression											
Lifetime	21 (55.3)	27 (65.9)	0.93	.335	.108	33 (57.9)	15 (68.2)	0.71	.401	.094	48 (60.8)
Past month	9 (23.7)	10 (24.4)	0.01	.942	.008	15 (26.3)	4 (18.2)	0.58	.448	.085	19 (24.1)
Bipolar I disorder											
Lifetime	0 (0)	0 (0)	-	-	-	0 (0)	0 (0)	-	-	-	0 (0)
Past month	0 (0)	0 (0)	-	-	-	0 (0)	0 (0)	-	-	-	0 (0)
Bipolar II disorder											
Lifetime	2 (5.3)	1 (2.4)	0.43	.512	.074	3 (5.3)	0 (0)	1.20	.273	.123	3 (3.8)
Past month	0 (0)	0 (0)	-	-	-	0 (0)	0 (0)	-	-	-	0 (0)
Any Anxiety disorder											
Lifetime	24 (63.2)	22 (53.7)	0.73	.392	.096	33 (57.9)	13 (59.1)	0.01	.923	.011	46 (58.2)
Past month	19 (50.0)	18 (43.9)	0.29	.587	.061	27 (47.4)	10 (45.5)	0.02	.879	.017	37 (46.8)
Substance abuse and/or dependence											
Lifetime	19 (50.0)	14 (34.1)	2.04	.153	.161	25 (43.9)	8 (36.4)	0.37	.545	.068	33 (41.8)
Past month	3 (7.9)	1 (2.4)	1.22	.269	.124	3 (5.3)	1 (4.5)	0.02	.896	.015	4 (5.1)

Note: BN = bulimia nervosa; BED = binge eating disorder; CBT = cognitive behavioural therapy; ST = Schema therapy; CBT-A = appetite-focussed cognitive behavioural therapy

Table 4

Means and Standard Deviations of Total Energy Intake, Intake of Each Macronutrient, and Percentage of Total Energy from Each Macronutrient at Pre- (week 0), Mid- (week 26) and Post-Treatment (week 52) for the Total Sample (n = 79).

Nutrient	Week 0 (*n* = 77)	Week 26 (*n* = 65)	Week 52 (*n* = 64)
	M (*SD*)	*M* (*SD*)	*M* (*SD*)
Average energy intake (kj)	12552.22 (4133.86)	10129.11 (2973.89)	9629.71 (3151.32)
Macronutrient weights			
Total protein (g)	114.00 (32.48)	99.96 (28.44)	100.36 (40.76)
Total carbohydrates (g)	356.27 (129.59)	279.53 (97.29)	273.77 (97.47)
Sugar (g)	174.63 (74.05)	128.56 (53.34)	130.55 (53.21)
Total fat (g)	115.46 (44.75)	87.11 (31.01)	84.44 (34.73)
Saturated fat (g)	46.26 (19.55)	33.27 (13.63)	32.64 (15.86)
Trans fat (g)	1.57 (0.67)	1.35 (0.59)	1.23 (0.63)
Monounsaturated (g)	41.02 (16.87)	31.45 (11.93)	30.19 (12.94)
Polyunsaturated (g)	17.70 (7.27)	14.69 (6.00)	13.66 (6.22)
N6 (g)	13.33 (5.83)	12.00 (4.86)	10.71 (5.25)
ALA (g)	2.26 (1.25)	2.01 (1.18)	1.56 (0.85)
Macronutrients (percent of total kj)			
Total protein (%kJ)	15.56 (3.14)	16.95 (3.58)	17.94 (5.49)
Total carbohydrates (%kJ)	47.34 (6.51)	45.70 (6.12)	47.28 (6.30)
Sugar (%kJ)	23.37 (7.07)	20.95 (5.86)	22.44 (6.07)
Total fat (%kJ)	34.44 (5.68)	32.22 (5.77)	32.75 (6.69)
Saturated fat (%kJ)	13.73 (2.97)	12.17 (2.83)	12.55 (3.56)
Trans fat (%kJ)	0.47 (0.15)	0.50 (0.16)	0.48 (0.20)
Monounsaturated (%kJ)	12.21 (2.52)	11.68 (2.61)	11.74 (2.95)
Polyunsaturated (%kJ)	5.37 (1.64)	5.49 (1.71)	5.31 (1.63)
N6 (%kJ)	4.06 (1.47)	4.48 (1.34)	4.12 (1.22)
ALA (%kJ)	0.68 (0.36)	0.75 (0.39)	0.61 (0.29)

Note: kJ = kilojoules; g = grams; N6 = omega-6 fatty acids; ALA = n-3 alpha-linolenic acid.

Table 5

Means and Standard Deviations of Intakes of Each Micronutrient at Pre- (week 0), Mid- (week 26) and Post-Treatment (week 52) for the Total Sample (n = 79).

Nutrient	Week 0 (*n* = 77)	Week 26 (*n* = 65)	Week 52 (*n* = 64)
	M (*SD*)	*M* (*SD*)	*M* (*SD*)
Vitamins			
Thiamine (mg)	2.68 (3.50)	4.14 (9.84)	2.23 (1.76)
Riboflavin (mg)	3.21 (3.48)	4.44 (9.77)	2.44 (1.33)
Niacin (mg)	28.23 (12.24)	26.85 (12.14)	26.54 (11.35)
Vitamin C (mg)	121.04 (54.77)	125.69 (62.04)	113.14 (58.64)
Vitamin E (mg)	14.57 (6.43)	13.66 (5.86)	12.53 (8.30)
Vitamin B6 (mg)	2.70 (3.75)	4.18 (9.89)	2.10 (1.03)
Vitamin B12 (µg)	4.70 (2.85)	5.44 (6.52)	4.07 (1.85)
Folate (µg)	591.37 (290.39)	553.35 (265.51)	589.76 (285.21)
Retinol (µg)	519.37 (297.59)	347.56 (172.14)	337.98 (171.43)
Minerals			
Sodium (mg)	3667.85 (1543.47)	3066.69 (1175.13)	3116.63 (1168.88)
Potassium (mg)	4122.30 (1245.11)	3791.35(1080.82)	3614.01 (1248.16)
Magnesium (mg)	455.19 (147.04)	401.46 (123.98)	399.02 (133.42)
Calcium (mg)	1230.94 (487.28)	1097.49 (450.99)	1076.82 (421.31)
Phosphorus (mg)	2006.50 (613.03)	1762.17 (521.55)	1711.06 (507.57)
Iron (mg)	16.24 (6.63)	13.97 (5.29)	15.19 (8.90)
Zinc (mg)	13.21 (4.50)	11.92 (3.95)	10.98 (3.69)
Selenium (µg)	89.95 (37.11)	92.36 (46.39)	94.07 (51.54)
Iodine (µg)	193.53 (81.57)	169.53 (75.06)	148.45 (67.58)

Note. mg = milligrams; µg = micrograms.

Table 6

Means and Standard Deviations of Total Energy Intake, Intake of Each Macronutrient, and Percentage of Total Energy from Each Macronutrient at Pre- (week 0), Mid- (week 26) and Post-Treatment (week 52) for Individuals with BN (n = 38) and BED (n = 41).

Nutrient	Bulimia nervosa			Binge eating disorder		
	Week 0 (*n* = 38)	Week 26 (*n* = 35)	Week 52 (*n* = 32)	Week 0 (*n* = 39)	Week 26 (*n* = 30)	Week 52 (*n* = 32)
	M (*SD*)	*M* (*SD*)	*M* (*SD*)	*M* (*SD*)	*M* (*SD*)	*M* (*SD*)
Average energy intake (kj)	13379.07 (4174.60)	10487.87 (3278.94)	10314.32 (3430.93)	11746.58 (3982.34)	9710.56 (2564.40)	8945.11 (2728.21)
Macronutrients						
Total protein (g)	122.33 (31.94)	103.96 (33.67)	113.22 (50.39)	105.90 (31.30)	95.29 (20.33)	87. 50 (22.25)
Total carbohydrates (g)	376.71 (139.01)	288.23 (107.45)	292.84 (104.61)	336.34 (118.09)	269.37 (84.59)	254.71 (87.27)
Sugar (g)	180.85 (72.04)	129.17 (56.59)	137.53 (53.93)	168.57 (76.41)	127.85 (50.24)	123.57 (52.39)
Total fat (g)	120.17 (42.32)	88.62 (32.86)	88.93 (37.50)	110.87 (47.08)	85.34 (29.17)	79.95 (31.67)
Saturated fat (g)	47.99 (16.96)	33.51 (14.46)	34.22 (16.20)	44.59 (21.87)	32.99 (12.83)	31.06 (15.60)
Trans fat (g)	1.66 (0.60)	1.32 (0.58)	1.35 (0.66)	1.47 (0.73)	1.39 (0.61)	1.11 (0.58)
Monounsaturated (g)	43.01 (16.64)	32.30 (12.19)	32.08 (14.60)	39.08 (17.07)	30.45 (11.75)	28.31 (10.94)
Polyunsaturated (g)	18.40 (7.98)	14.93 (6.06)	14.10 (7.08)	17.02 (6.52)	14.40 (6.02)	13.21 (5.31)
N6 (g)	13.39 (6.36)	12.20 (4.98)	11.15 (6.01)	13.27 (5.35)	11.78 (4.81)	10.27 (4.42)
ALA (g)	2.39 (1.23)	2.00 (1.11)	1.64 (0.91)	2.12 (1.27)	2.02 (1.27)	1.48 (0.79)
Macronutrients						
Total protein (%kJ)	15.71 (3.53)	16.88 (3.27)	18.70 (6.35)	15.43 (2.76)	17.04 (3.97)	17.18 (4.44)
Total carbohydrates (%kJ)	46.74 (6.53)	45.43 (6.50)	47.23 (6.70)	47.92 (6.53)	46.01 (5.76)	47.31 (5.99)
Sugar (%kJ)	22.85 (7.66)	20.41 (6.66)	22.19 (6.90)	23.89 (6.52)	21.58 (4.80)	22.68 (5.21)
Total fat (%kJ)	33.82 (5.52)	31.79 (5.75)	32.26 (6.20)	35.04 (5.84)	32.72 (5.84)	33.24 (7.21)
Saturated fat (%kJ)	13.57 (2.52)	11.84 (2.99)	12.40 (3.29)	13.88 (3.37)	12.55 (2.63)	12.71 (3.86)
Trans fat (%kJ)	0.48 (0.15)	0.48 (0.18)	0.50 (0.19)	0.46 (0.16)	0.53 (0.15)	0.46 (0.20)
Monounsaturated (%kJ)	12.08 (2.83)	11.69 (2.49)	11.63 (2.86)	12.34 (2.20)	11.68 (2.79)	11.85 (3.08)
Polyunsaturated (%kJ)	5.11 (1.37)	5.40 (1.52)	5.07 (1.45)	5.61 (1.86)	5.59 (1.92)	5.56 (1.78)
N6 (%kJ)	3.72 (1.22)	4.41 (1.22)	3.98 (1.18)	4.39 (1.62)	4.56 (1.49)	4.27 (1.27)
ALA (%kJ)	0.66 (0.26)	0.73 (0.37)	0.60 (0.33)	0.70 (0.44)	0.77 (0.43)	0.61 (0.26)

Note: kJ = kilojoules; g = grams; N6 = omega-6 fatty acids; ALA = n-3 alpha-linolenic acid.

Table 7

Means and Standard Deviations of Intakes of each Micronutrient at Pre- (week 0), Mid- (week 26) and Post-Treatment (week 52) for Individuals with BN (n = 38) and BED (n = 41).

Nutrient	Bulimia nervosa			Binge eating disorder		
	Week 0 (*n* = 38)	Week 26 (*n* = 35)	Week 52 (*n* = 32)	Week 0 (*n* = 39)	Week 26 (*n* = 30)	Week 52 (*n* = 32)
	M (*SD*)	*M* (*SD*)	*M* (*SD*)	*M* (*SD*)	*M* (*SD*)	*M* (*SD*)
Vitamins						
Thiamine (mg)	3.48 (4.77)	3.67 (7.50)	2.62 (2.30)	1.90 (1.04)	4.68 (12.13)	1.85 (0.83)
Riboflavin (mg)	3.94 (4.71)	4.21 (7.39)	2.62 (1.70)	2.50 (1.28)	4.70 (12.11)	2.25 (0.79)
Niacin (mg)	29.55 (13.38)	28.66 (15.07)	28.84 (13.45)	26.95 (11.03)	24.74 (7.10)	24.24 (8.36)
Vitamin C (mg)	133.56 (57.28)	129.62 (60.33)	115.41 (63.14)	108.84 (49.96)	121.10 (64.70)	110.87 (54.69)
Vitamin E (mg)	15.03 (7.03)	14.19 (5.81)	12.19 (6.62)	14.13 (5.85)	13.04 (5.96)	12.88 (9.80)
Vitamin B6 (mg)	3.22 (4.92)	3.89 (7.41)	2.22 (1.00)	2.20 (2.00)	4.52 (12.29)	1.99 (1.06)
Vitamin B12 (μg)	5.25 (3.57)	5.40 (5.14)	4.30 (2.14)	4.17 (1.81)	5.48 (7.93)	3.85 (1.50)
Folate (μg)	660.69 (360.40)	578.19 (343.62)	630.91 (346.90)	523.82 (180.83)	524.36 (124.60)	548.62 (203.67)
Retinol (μg)	562.10 (284.79)	355.09 (195.29)	350.72 (151.52)	477.36 (307.43)	338.79 (143.29)	325.24 (190.87)
Minerals						
Sodium (mg)	4179.98 (1696.37)	3335.36 (1381.05)	3597.68 (1220.11)	3168.85 (1202.02)	2753.24 (789.09)	2635.58 (900.12)
Potassium (mg)	4463.79 (1377.39)	3993.20 (1226.41)	3952.80 (1476.76)	3789.56 (1011.47)	3555.86 (841.47)	3275.22 (865.04)
Magnesium (mg)	495.63 (162.57)	421.10 (135.01)	430.60 (152.71)	415.78 (119.50)	378.55 (107.42)	367.45 (103.91)
Calcium (mg)	1325.94 (517.87)	1165.27 (500.25)	1133.11 (415.74)	1138.38 (442.65)	1018.42 (378.75)	1020.53 (425.85)
Phosphorus (mg)	2169.53 (651.15)	1876.36 (500.33)	1843.60 (555.38)	1847.65 (535.07)	1628.94 (379.04)	1578.52 (422.91)
Iron (mg)	17.89 (7.51)	14.64 (6.08)	16.76 (10.90)	14.63 (5.26)	13.18 (4.15)	13.63 (6.10)
Zinc (mg)	14.29 (4.45)	12.56 (4.37)	12.13 (4.56)	12.16 (4.34)	11.17 (3.34)	9.84 (2.06)
Selenium (μg)	95.60 (42.03)	91.75 (37.02)	97.61 (45.23)	84.45 (31.17)	93.07 (56.04)	90.53 (57.68)
Iodine (μg)	219.46 (89.93)	181.10 (90.56)	159.97 (73.69)	168.26 (64.09)	156.04 (49.69)	136.93 (59.81)

Note: mg = milligrams; μg = micrograms.

Table 8

Means and Standard Deviations of Total Energy Intake, Intake of Each Macronutrient, and Percentage of Total Energy from Each Macronutrient at Pre- (week 0), Mid- (week 26) and Post-Treatment (week 52) for Individuals Randomised to either CBT + ST (n = 57) and CBT-A (n = 22).

Nutrient	CBT + ST			CBT-A		
	Week 0 (*n* = 56)	Week 26 (*n* = 47)	Week 52 (*n* = 46)	Week 0 (*n* = 21)	Week 26 (*n* = 18)	Week 52 (*n* = 18)
	M (*SD*)	*M* (*SD*)	*M* (*SD*)	*M* (*SD*)	*M* (*SD*)	*M* (*SD*)
Average energy intake (kj)	12611.06 (4064.80)	10471.55 (3245.09)	9930.43 (3342.95)	12395.32 (4411.87)	9234.98 (1905.25)	8861.22 (2520.08)
Macronutrients						
Total protein (g)	115.74 (32.39)	101.58 (31.67)	102.12 (45.09)	109.39 (33.05)	95.74 (17.43)	95.87 (27.28)
Total carbohydrates (g)	357.67 (126.80)	292.92 (105.06)	285.89 (105.54)	352.53 (139.94)	244.55 (63.06)	242.82 (65.68)
Sugar (g)	171.67 (61.40)	134.68 (55.46)	135.32 (56.22)	182.52 (101.89)	112.57 (44.88)	118.37 (43.67)
Total fat (g)	115.02 (44.15)	89.17 (33.40)	86.21 (35.75)	116.62 (47.39)	81.73 (23.65)	79.90 (32.49)
Saturated fat (g)	45.69 (18.57)	34.08 (14.65)	33.48 (15.15)	47.79 (22.36)	31.16 (10.58)	30.50 (17.83)
Trans fat (g)	1.62 (0.67)	1.39 (0.61)	1.32 (0.60)	1.43 (0.67)	1.25 (0.56)	1.01 (0.68)
Monounsaturated (g)	40.89 (17.06)	32.19 (12.78)	30.79 (13.63)	41.38 (16.75)	29.50 (9.42)	28.67 (11.19)
Polyunsaturated (g)	17.57 (7.38)	14.90 (6.33)	13.89 (6.78)	18.04 (7.12)	14.14 (5.17)	13.07 (4.63)
N6 (g)	13.19 (6.02)	12.11 (5.20)	11.11 (5.79)	13.71 (5.44)	11.71 (3.98)	9.70 (3.48)
ALA (g)	2.27 (1.29)	1.99 (1.27)	1.51 (0.84)	2.21 (1.18)	2.05 (0.93)	1.68 (0.89)
Macronutrients						
Total protein (g)	15.74 (3.29)	16.66 (3.57)	17.63 (5.97)	15.10 (2.75)	17.71 (3.61)	18.72 (4.06)
Total carbohydrates (g)	47.42 (6.31)	46.35 (6.13)	47.68 (6.06)	47.12 (7.19)	44.01 (5.93)	46.26 (6.96)
Sugar (g)	23.11 (6.22)	21.34 (5.83)	22.58 (6.66)	24.09 (9.11)	19.93 (5.97)	22.06 (4.36)
Total fat (g)	34.05 (5.82)	31.88 (5.94)	32.50 (6.61)	35.49 (5.24)	33.10 (5.34)	33.38 (7.04)
Saturated fat (g)	13.46 (3.06)	12.04 (2.90)	12.63 (3.23)	14.43 (2.64)	12.51 (2.68)	12.36 (4.40)
Trans fat (g)	0.49 (0.16)	0.51 (0.17)	0.51 (0.19)	0.44 (0.12)	0.50 (0.14)	0.40 (0.19)
Monounsaturated (g)	12.06 (2.54)	11.58 (2.65)	11.62 (2.93)	12.62 (2.48)	11.96 (2.56)	12.04 (3.05)
Polyunsaturated (g)	5.29 (1.62)	5.37 (1.62)	5.17 (1.48)	5.58 (1.72)	5.81 (1.93)	5.69 (1.97)
N6 (g)	3.97 (1.43)	4.35 (1.30)	4.11 (1.26)	4.30 (1.56)	4.82 (1.43)	4.16 (1.16)
ALA (g)	0.67 (0.29)	0.71 (0.40)	0.57 (0.26)	0.71 (0.51)	0.85 (0.38)	0.71 (0.36)

Abbreviations: kJ = kilojoules; g = grams; ALA = n-3 alpha-linolenic acid; ST = Schema therapy; CBT-A = appetite-focussed cognitive behavioural therapy

Table 9

Means and Standard Deviations of Intakes of each Micronutrient at Pre- (week 0), Mid- (week 26) and Post-Treatment (week 52) for Individuals who Underwent CBT + ST (n = 57) and CBT (n = 22).

Nutrient	CBT + ST			CBT-A		
	Week 0 (*n* = 56)	Week 26 (*n* = 47)	Week 52 (*n* = 46)	Week 0 (*n* = 21)	Week 26 (*n* = 18)	Week 52 (*n* = 18)
	M (*SD*)	*M* (*SD*)	*M* (*SD*)	*M* (*SD*)	*M* (*SD*)	*M* (*SD*)
Vitamins						
Thiamine (mg)	2.30 (1.38)	2.50 (2.09)	2.48 (2.01)	3.70 (6.32)	8.41 (18.05)	1.61 (0.42)
Riboflavin (mg)	2.76 (1.43)	2.76 (1.61)	2.54 (1.50)	4.40 (6.20)	8.82 (18.02)	2.17 (0.69)
Niacin (mg)	28.49 (11.86)	27.62 (13.63)	26.12 (12.28)	27.54 (13.47)	24.84 (6.84)	27.63 (8.72)
Vitamin C (mg)	127.34 (56.87)	123.25 (61.20)	113.54 (62.49)	104.24 (45.82)	132.06 (65.53)	112.12 (49.05)
Vitamin E (mg)	14.66 (6.79)	13.77 (6.07)	12.78 (9.15)	14.34 (5.51)	13.36 (5.44)	11.89 (5.77)
Vitamin B6 (mg)	2.12 (1.05)	2.47 (1.55)	2.00 (0.87)	4.26 (6.85)	8.63 (18.23)	2.36 (1.34)
Vitamin B12 (µg)	4.58 (2.19)	4.45 (2.15)	4.03 (2.04)	5.02 (4.19)	8.01 (11.75)	4.20 (1.26)
Folate (µg)	603.73 (322.82)	569.87 (301.15)	596.85 (315.39)	558.40 (179.69)	510.20 (131.39)	571.65 (194.10)
Retinol (µg)	553.29 (308.56)	362.60 (188.85)	335.46 (162.07)	428.20 (250.52)	308.30 (113.08)	344.42 (198.29)
Minerals						
Sodium (mg)	3771.47 (1670.89)	3130.46 (1343.66)	3175.18 (1291.99)	3391.55 (1124.70)	2900.16 (522.40)	2966.99 (782.11)
Potassium (mg)	4228.40 (1257.61)	3948.15 (1129.19)	3691.87 (1338.87)	3839.36 (1194.23)	3381.93 (837.94)	3415.03 (984.92)
Magnesium (mg)	468.61 (152.79)	413.32 (133.05)	406.33 (145.10)	419.40 (126.94)	370.49 (92.49)	380.37 (98.61)
Calcium (mg)	1247.83 (471.06)	1123.20 (475.44)	1083.34 (400.03)	1185.90 (537.71)	1030.36 (383.99)	1060.16 (483.52)
Phosphorus (mg)	2045.98 (610.80)	1813.45 (567.83)	1739.85 (541.28)	1901.22 (621.37)	1628.25 (354.02)	1637.49 (413.80)
Iron (mg)	16.79 (6.90)	14.85 (5.77)	15.91 (10.27)	14.75 (5.75)	11.66 (2.70)	13.37 (3.07)
Zinc (mg)	13.33 (4.55)	12.24 (4.37)	11.13 (4.07)	12.90 (4.46)	11.10 (2.48)	10.59 (2.53)
Selenium (µg)	91.84 (39.45)	89.80 (48.69)	92.63 (52.65)	84.93 (30.30)	99.05 (40.26)	97.75 (49.85)
Iodine (µg)	202.25 (86.86)	171.05 (83.23)	149.49 (73.19)	170.26 (61.31)	165.57 (49.42)	145.80 (52.30)

Note. mg = milligrams; µg = micrograms; ST = Schema therapy; CBT-A = appetite-focussed cognitive behavioural therapy

Table 10

Numbers and Percentages of Participants who were Adequate for each Nutrient at Pre- (week 0), Mid- (week 26) and Post-Treatment (week 52) for the Total Sample (n = 79).

Nutrient	Week 0 (n = 77)	Week 26 (n = 65)	Week 52 (n = 64)
	n (%)	*n* (%)	*n* (%)
Average energy intake	13 (16.5%)	0 (0%)	0 (0%)
Macronutrients			
Total protein	40 (51.9%)	44 (67.7%)	47 (73.4%)
Total carbohydrates	48 (62.3%)	42 (64.6%)	45 (70.3%)
Sugar	51 (66.2%)	47 (72.3%)	44 (68.8%)
Total fat	41 (53.2%)	43 (66.2%)	37 (57.8%)
Saturated fat	7 (9.1%)	13 (20.0%)	18 (28.1%)
Trans fat	77 (100%)	64 (98.5%)	63 (98.4%)
Monounsaturated	77 (100%)	65 (100%)	63 (98.4%)
Polyunsaturated	76 (98.7%)	64 (98.5%)	63 (98.4%)
N6	14 (18.2%)	19 (29.2%)	12 (18.8%)
ALA	33 (42.9%)	37 (56.9%)	20 (31.3%)
Micronutrients			
Vitamins			
Thiamine	73 (94.8%)	58 (86.7%)	58 (90.6%)
Riboflavin	77 (100%)	64 (98.5%)	63 (98.4%)
Niacin	51 (66.2%)	53 (81.5%)	53 (82.8%)
Vitamin C	74 (96.1%)	61 (93.8%)	59 (92.2%)
Vitamin E	76 (98.7%)	63 (96.9%)	58 (90.6%)
Vitamin B6	65 (84.4%)	55 (84.6%)	59 (92.2%)
Vitamin B12	69 (89.6%)	64 (98.5%)	59 (92.2%)
Folate	64 (83.1%)	51 (78.5%)	52 (81.3%)
Retinol	20 (26.0%)	3 (4.6%)	6 (9.4%)
Minerals			
Sodium	14 (18.2%)	0 (0%)	1 (3.1%)
Potassium	75 (97.4%)	57 (87.7%)	55 (85.9%)
Magnesium	73 (94.8%)	52 (80.0%)	56 (87.5%)
Calcium	51 (66.2%)	33 (50.8%)	37 (57.8%)
Phosphorus	74 (96.1%)	65 (100%)	63 (98.4%)
Iron	43 (55.8%)	14 (46.7%)	27 (42.2%)
Zinc	75 (97.4%)	61 (93.8%)	60 (93.8%)
Selenium	68 (88.3%)	59 (90.8%)	54 (84.4%)
Iodine	58 (75.3%)	42 (64.6%)	29 (45.3%)

Note. N6 = N6 = omega-6 fatty acids; ALA = n-3 alpha-linolenic acid.

Table 11

Numbers and Percentages of Participants who were Adequate for each Nutrient at Pre- (week 0), Mid- (week 26) and Post-Treatment (week 52) for Individuals with BN (n = 38) and BED (n = 41).

Nutrient	Bulimia Nervosa			Binge Eating Disorder		
	Week 0 (*n* = 38)	Week 26 (*n* = 35)	Week 52 (*n* = 32)	Week 0 (*n* = 39)	Week 26 (*n* = 30)	Week 52 (*n* = 32)
	n (%)	*n* (%)	*n* (%)	*n* (%)	*n* (%)	*n* (%)
Average energy intake	8 (21.1%)	0 (0%)	0 (0%)	5 (12.8%)	0 (0%)	0 (0%)
Macronutrients						
Total protein	20 (52.6%)	26 (74.3%)	26 (81.3%)	20 (51.3%)	18 (60.0%)	21 (65.6%)
Total carbohydrates	23 (60.5%)	22 (62.9%)	21 (65.6%)	25 (64.1%)	20 (66.7%)	24 (75.0%)
Sugar	26 (68.4%)	26 (74.3%)	23 (71.9%)	25 (64.1%)	21 (70%)	21 (65.6%)
Total fat	22 (57.9%)	23 (65.7%)	19 (59.4%)	19 (48.7%)	20 (66.7%)	18 (56.3%)
Saturated fat	2 (5.3%)	8 (22.9%)	9 (28.1%)	5 (12.8%)	5 (16.7%)	9 (28.1%)
Trans fat	38 (100%)	34 (97.1%)	32 (100%)	39 (100%)	30 (100%)	31 (96.9%)
Monounsaturated	38 (100%)	35 (100%)	32 (100%)	39 (100%)	30 (100%)	31 (96.9%)
Polyunsaturated	38 (100%)	35 (100%)	32 (100%)	38 (97.4%)	29 (96.7%)	31 (96.9%)
N6	4 (10.5%)	11 (31.4%)	5 (15.6%)	10 (25.6%)	8 (26.7%)	7 (21.9%)
ALA	19 (50.0%)	20 (57.1%)	10 (31.3%)	14 (35.9%)	17 (56.7%)	10 (31.3%)
Micronutrients						
Vitamins						
Thiamine	37 (97.4%)	32 (91.4%)	32 (100%)	36 (92.3%)	26 (86.7%)	26 (81.3%)
Riboflavin	38 (100%)	35 (100%)	31 (96.9%)	39 (100%)	29 (96.7%)	32 (100%)
Niacin	23 (60.5%)	27 (77.1%)	25 (78.1%)	28 (71.8%)	26 (86.7%)	28 (87.5%)
Vitamin C	37 (97.4%)	34 (97.1%)	29 (90.6%)	37 (94.9%)	27 (90.0%)	30 (93.8%)
Vitamin E	38 (100%)	34 (97.1%)	29 (90.6%)	38 (97.4%)	29 (96.7%)	29 (90.6%)
Vitamin B6	34 (89.5%)	29 (82.9%)	30 (93.8%)	31 (79.5%)	26 (86.7%)	29 (90.6%)
Vitamin B12	34 (89.5%)	35 (100%)	30 (93.8%)	35 (89.7%)	29 (96.7%)	29 (90.6%)
Folate	32 (84.2%)	25 (71.4%)	25 (78.1%)	32 (82.1%)	26 (86.7%)	27 (84.4%)
Retinol	9 (23.7%)	2 (5.7%)	2 (6.3%)	11 (28.2%)	1 (3.3%)	4 (12.5%)
Minerals						
Sodium	4 (10.5%)	0 (0%)	0 (0%)	10 (25.6%)	0 (0%)	1 (3.1%)
Potassium	37 (97.4%)	31 (88.6%)	29 (90.6%)	38 (97.4%)	26 (86.7%)	26 (81.3%)
Magnesium	37 (97.4%)	28 (80.0%)	29 (90.6%)	36 (92.3%)	24 (80%)	27 (84.4%)
Calcium	26 (68.4%)	19 (54.3%)	21 (65.6%)	25 (64.1%)	14 (46.7%)	16 (50.0%)
Phosphorus	36 (94.7%)	35 (100%)	31 (96.9%)	38 (97.4%)	30 (100%)	32 (100%)
Iron	24 (63.2%)	14 (40.0%)	13 (40.6%)	19 (48.7%)	14 (46.7%)	14 (43.8%)
Zinc	37 (97.4%)	34 (97.1%)	31 (96.9%)	38 (97.4%)	27 (90%)	29 (90.6%)
Selenium	33 (86.8%)	30 (85.7%)	28 (87.5%)	35 (89.7 %)	29 (96.7%)	26 (81.3%)
Iodine	35 (92.1%)	24 (68.6%)	16 (50.0%)	23 (59.0%)	18 (60.0%)	13 (40.6%)

Note. N6 = N6 = omega-6 fatty acids; ALA = n-3 alpha-linolenic acid.

Table 12

Numbers and Percentages of Participants who were Adequate for each Nutrient at Pre- (week 0), Mid- (week 26) and Post-Treatment (week 52) for Individuals who were Randomised to CBT + ST (n = 57) and CBT (n = 22).

Nutrient	CBT + ST			CBT-A		
	Week 0 (*n* = 56)	Week 26 (*n* = 47)	Week 52 (*n* = 46)	Week 0 (*n* = 21)	Week 26 (*n* = 18)	Week 52 (*n* = 18)
	n (%)	*n* (%)	*n* (%)	*n* (%)	*n* (%)	*n* (%)
Average energy intake	9 (16.1%)	0 (0%)	0 (0%)	4 (19.0%)	0 (0%)	0 (0%)
Macronutrients						
Total protein	30 (53.6%)	30 (63.8%)	32 (69.6%)	10 (47.6%)	14 (77.8%)	15 (83.3%)
Total carbohydrates	35 (62.5%)	31 (66.0%)	32 (69.6%)	13 (61.9%)	11 (61.1%)	13 (72.2%)
Sugar	39 (69.6%)	33 (70.2%)	34 (73.9%)	12 (57.1%)	14 (77.8%)	10 (55.6%)
Total fat	31 (55.4%)	31 (66.0%)	24 (52.2%)	10 (47.6%)	12 (66.7%)	13 (72.2%)
Saturated fat	7 (12.5%)	11 (23.4%)	11 (23.9%)	0 (0%)	2 (11.1%)	7 (38.9%)
Trans fat	56 (100%)	46 (97.9%)	45 (97.8%)	21 (100%)	18 (100%)	18 (100%)
Monounsaturated fatty acids	56 (100%)	47 (100%)	46 (100%)	21 (100%)	18 (100%)	17 (94.4%)
Polyunsaturated fatty acids	55 (98.2%)	47 (100%)	46 (100%)	21 (100%)	17 (94.4%)	17 (94.4%)
N6	9 (16.1%)	11 (23.4%)	10 (21.7%)	5 (23.8%)	8 (44.4%)	2 (11.1%)
ALA	25 (44.6%)	26 (55.3%)	12 (26.1%)	8 (38.1%)	11 (61.1%)	8 (44.4%)
Micronutrients						
Vitamins						
Thiamine	53 (94.6%)	42 (89.4%)	41 (89.1%)	20 (95.2%)	16 (88.9%)	17 (94.4%)
Riboflavin	56 (100%)	46 (97.9%)	45 (97.8%)	21 (100%)	18 (100%)	18 (100%)
Niacin	37 (66.1%)	37 (78.7%)	39 (84.8%)	14 (66.7%)	16 (88.9%)	14 (77.8%)
Vitamin C	54 (96.4%)	43 (91.5%)	42 (91.3%)	20 (95.2%)	18 (100%)	17 (94.4%)
Vitamin E	55 (98.2%)	45 (95.7%)	41 (89.1%)	21 (100%)	18 (100%)	17 (94.4%)
Vitamin B6	49 (87.5%)	40 (85.1%)	42 (91.3%)	16 (76.2%)	15 (83.3%)	17 (94.4%)
Vitamin B12	50 (89.3%)	46 (97.9%)	41 (89.1%)	19 (90.5%)	18 (100%)	18 (100%)
Folate	46 (82.1%)	35 (74.5%)	37 (80.4%)	18 (85.7%)	16 (88.9%)	15 (83.3%)
Retinol	18 (32.1%)	3 (6.4%)	3 (6.5%)	2 (9.5%)	0 (0%)	3 (16.7%)
Minerals						
Sodium	10 (17.9%)	0 (0%)	1 (2.2%)	4 (19.0%)	0 (0%)	0 (0%)
Potassium	55 (98.2%)	43 (91.5%)	40 (87.0%)	20 (95.2%)	14 (77.8%)	15 (83.3%)
Magnesium	54 (96.4%)	38 (80.9%)	40 (87.0%)	19 (90.5%)	14 (77.8%)	16 (88.9%)
Calcium	37 (66.1%)	24 (51.1%)	28 (60.9%)	14 (66.7%)	9 (50.0%)	9 (50.0%)
Phosphorus	54 (96.4%)	47 (100%)	45 (97.8%)	20 (95.2%)	18 (100%)	18 (100%)
Iron	33 (58.9%)	24 (51.1%)	20 (43.5%)	10 (47.6%)	4 (22.2%)	7 (38.9%)
Zinc	55 (98.2%)	43 (91.5%)	43 (93.5%)	20 (95.2%)	18 (100%)	17 (94.4%)
Selenium	49 (87.5%)	42 (89.4%)	38 (82.6%)	19 (90.5%)	17 (94.4%)	16 (88.9%)
Iodine	45 (80.4%)	29 (61.7%)	20 (43.5%)	13 (61.9%)	13 (72.2%)	9 (50.0%)

Note. N6 = N6 = omega-6 fatty acids; ALA = n-3 alpha-linolenic acid; ST = Schema therapy; CBT-A = appetite-focussed cognitive behavioural therapy

Table 13

Means and Standard Deviations of Summary Nutritional Adequacy Measures at Pre- (week 0), Mid- (week 26) and Post-Treatment (week 52) for the Total Sample (n = 79).

Adequacy measure	Week 0 (*n* = 77)	Week 26 (*n* = 65)	Week 52 (*n* = 64)
	M (*SD*)	*M* (*SD*)	*M* (*SD*)
Total adequacy	20.31 (2.39)	20.11 (2.52)	19.70 (3.02)
Macronutrient adequacy	6.03 (1.03)	6.74 (1.47)	6.44 (1.27)
Micronutrient adequacy	14.29 (2.15)	13.37 (2.40)	13.27 (2.64)

Table 14

Means and Standard Deviations of Summary Nutritional Adequacy Measures at Pre- (week 0), Mid- (week 26) and Post-Treatment (week 52) for Individuals with BN (n = 38) and BED (n = 41).

Adequacy measure	Bulimia Nervosa			Binge Eating Disorder		
	Week 0 (*n* = 38)	Week 26 (*n* = 35)	Week 52 (*n* = 32)	Week 0 (*n* = 39)	Week 26 (*n* = 30)	Week 52 (*n* = 32)
	M (*SD*)	*M* (*SD*)	*M* (*SD*)	*M* (*SD*)	*M* (*SD*)	*M* (*SD*)
Total adequacy	20.55 (2.44)	20.23 (2.33)	20.00 (2.86)	20.08 (2.36)	19.97 (2.76)	19.41 (3.19)
Macronutrient adequacy	6.05 (0.96)	6.86 (1.68)	6.53 (1.16)	6.00 (1.10)	6.60 (1.19)	6.34 (1.38)
Micronutrient adequacy	14.50 (1.98)	13.37 (2.20)	13.47 (2.44)	14.08 (2.31)	13.37 (2.66)	13.06 (2.86)

Table 15

Means and Standard Deviations of Summary Nutritional Adequacy Measures at Pre- (week 0), Mid- (week 26) and Post-Treatment (week 52) for Individuals who Underwent CBT + ST (n = 57) and CBT (n = 22).

Adequacy measure	CBT + ST			CBT-A		
	Week 0 (*n* = 56)	Week 26 (*n* = 47)	Week 52 (*n* = 46)	Week 0 (*n* = 21)	Week 26 (*n* = 18)	Week 52 (*n* = 18)
	M (*SD*)	*M* (*SD*)	*M* (*SD*)	*M* (*SD*)	*M* (*SD*)	*M* (*SD*)
Total adequacy	20.59 (2.13)	20.00 (2.66)	19.52 (3.10)	19.57 (2.91)	20.39 (2.15)	20.17 (2.85)
Macronutrient adequacy	6.13 (1.05)	6.66 (1.43)	6.35 (1.16)	5.76 (0.94)	6.94 (1.59)	6.67 (1.53)
Micronutrient adequacy	14.46 (1.98)	13.34 (2.56)	13.17 (2.80)	13.81 (2.54)	13.44 (1.85)	13.50 (2.26)

Abbreviations: CBT = cognitive behavioural therapy; CBT-A = appetite-focussed

Additionally, Model 3 for total energy intake showed no significant differences between the two treatment types at week 0, or in how treatment types differed in change over time.

Macronutrients

Weighted Intake. Model 1 indicates that reductions in weighted nutrient intake were observed between weeks 0 and 26 for intakes of total protein (β = -14.44, p = .013), total carbohydrates (β = -79.93, p < .001), sugars (β = -47.37, p < .001), total fats (β = -29.32, p < .001), saturated fats (β = -13.52, p < .001), trans fats (β = -0.23, p = .049), MUFA (β = -9.87, p < .001), and PUFA (β = -3.07, p = .008). All macronutrient weighted intake variables decreased between weeks 0 and 52 (protein (β = -13.08, p = .029) total carbohydrates (β = -80.64, p < .001), sugars (β = -44.25, p < .001), total fats (β = -30.59, p < .001), saturated fats (β = -13.52, p < .001), trans fats (β = -0.33, p = .002), MUFA (β = -10.61, p < .001), PUFA (β = -3.94, p < .001), N6 (β = -2.51, p = .006), and ALA (β = -0.68, p < .001)). There were no changes in weighted intake between weeks 26 and 52, except for ALA (β = -0.44, p = .047), which decreased over this time period. Model sets for macronutrient weighted intakes are depicted in Table 17 – 26.

Model 2 for macronutrient weighted variables depicted no significant differences between diagnostic groups (BN vs. BED) at week 0, and no significant differences in how groups change over time for any of the variables.

Model 3 for macronutrient weighted intake variables indicated no significant differences in any weighted macronutrient intake variables between the two treatment types at week 0, and no significant differences in how the two treatment types changed over time on any of these variables.

Percent of Total Energy. Model 1 indicates that between weeks 0 and 26, the percentage of total energy from sugars (β = -2.39, p = .008), total fats (β = -2.28, p = .015),

and saturated fats (β = -1.61, p < .001) decreased, and the percentage of total energy from N6 increased between weeks 0 and 26 (β = 0.45, p = .047). Between weeks 0 and 52, there was a significant decrease in percentage of total energy from saturated fats (β = -1.17, p = .022), and an increase in percentage of total energy from protein (β = 2.38, p = .002). Only percentage of total energy from ALA changed between weeks 26 and 52 (β = -0.14, p = .031), decreasing over this time. No changes over time were found in the percentage of total energy from total carbohydrates, trans fats, MUFA, and PUFA. Model sets for macronutrients expressed as a percentage of total energy are depicted in Table 27 – 36.

Model 2 indicated that there were no significant differences between diagnostic groups (BN vs. BED) in percentage of total energy from any macronutrient at week 0, and that diagnostic groups did not differ significantly in how they changed over time.

Model 3 indicated that there were no significant difference between the two treatment types in proportion of total energy from any macronutrient at week 0, and no significant differences between treatment types in how they changed over time.

Micronutrients

Vitamins. Model 1 indicated that between weeks 0 and 26, only retinol intake changed (β = -173.56, p < .001), with retinol intake decreasing over this period. Retinol was also the only vitamin to change between weeks 0 and 52, decreasing over this period (β = -181.20, p < .001). No changes in vitamin intakes were observed between weeks 26 and 52. Intakes of thiamine, riboflavin, niacin, vitamin C, vitamin E, vitamin B6, vitamin B12, and folate did not change significantly over time. Model sets for vitamin intakes are depicted in Table 37 – 45.

Model 2 indicated that there were no significant differences between diagnostic groups (BN vs. BED) in intakes of vitamins at week 0, and that diagnostic groups did not differ significantly in how they changed over time.

Model 3 indicated that there were no significant difference between the two treatment types in intakes of vitamins at week 0, and no significant differences between treatment types in how they changed over time.

Minerals. Model 1 indicates that there is a significant decrease in the intakes of most minerals between weeks 0 and 26, including sodium (β = -655.96, $p < .001$), potassium (β = -381.04, $p = .023$), magnesium (β = -58.40, $p = .002$), calcium (β = -144.72, $p = .017$), phosphorus (β = -265.15, $p < .001$), iron (β = -2.51, $p = .026$), and zinc (β = -1.31, $p = .045$). Intake of all minerals decreased with the exception of iron and selenium between weeks 0 and 52 (sodium (β = -497.14, $p = .014$), potassium (β = -512.33, $p = .002$), magnesium (β = -56.17, $p = .003$), calcium (β = -159.89, $p = .008$), phosphorus (β = -289.96, $p < .001$), zinc (β = -2.09, $p < .001$), and iodine (β = -43.3, $p < .001$)). Mineral intake did not change between weeks 26 and 52. Model sets for mineral intakes are depicted in Table 46 – 54.

Model 2 indicated that there were significant differences between diagnostic groups (BN vs. BED) at week 0 in intakes of sodium (β = -1016.9, $p = .009$) and iodine (β = -52.04, $p = .031$), with participants with BN consuming significantly more than participants with BED. Diagnostic groups did not differ significantly in how they changed over time.

Model 3 indicated that there were no significant difference between the two treatment types in mineral intake at week 0, and no significant differences between treatment types in how they changed over time.

Nutritional Adequacy

Total Adequacy. Model 1 indicated that scores of total adequacy did not change over time for the total sample. Model 2 indicated that there were no significant differences between diagnostic groups (BN vs. BED) in total adequacy at week 0, and that diagnostic groups did not differ significantly in how they changed over time. Model 3 indicated that there was no significant difference between the two treatment types in total adequacy at week

0, and no significant difference between treatment types in how they changed over time. Model sets for measures of adequacy are displayed in Tables 55 – 57.

Macronutrient Adequacy. Model 1 indicated that scores of macronutrient adequacy increased between week 0 and week 26 ($\beta = 0.73$, $p < .001$). Scores of macronutrient adequacy did not significantly differ between weeks 0 and 52 and between weeks 26 and 52. Model 2 indicated that there were no differences between diagnostic groups (BN vs. BED) in macronutrient adequacy at week 0, and that diagnostic groups did not differ significantly in how they changed over time. Model 3 indicated that there was no significant difference between the two treatment types in macronutrient adequacy at week 0, and no significant difference between treatment types in how they changed over time.

Micronutrient Adequacy. Model 1 indicated that scores of micronutrient adequacy decreased between weeks 0 and 26 ($\beta = -0.92$, $p = .019$) and between weeks 0 and 52 ($\beta = -0.99$, $p = .011$). Scores of micronutrient adequacy did not significantly differ between weeks 26 and 52. Model 2 indicated that there were no significant differences between diagnostic groups (BN vs. BED) in micronutrient adequacy at week 0, and that diagnostic groups did not differ significantly in how they changed over time. Model 3 indicated that there was no significant difference between the two treatment types in micronutrient adequacy at week 0, and no significant difference between treatment types in how they changed over time.

Discussion

The current study investigated the nutritional intake and nutritional adequacy of women who binge eat and assessed the effect of treatment on these measures. The current study also examined whether women with different binge eating disorders (BN vs. BED) varied in nutritional intake and nutritional adequacy prior to treatment, and whether or not people with these diagnoses displayed different nutritional responses to treatment. Lastly, the current study investigated whether CBT-A, an augmented version of traditional CBT for binge eating with increased focus on appetite and satiety in the recommendations for normalising eating, provided an advantage over the usual dietary recommendations for normalising eating in CBT and ST in restoring nutritional health.

Summary of results

The modelling of nutritional intake provided several areas of interest. First, energy intake decreased between weeks 0 and 26, and this decrease was maintained at week 52. Similarly, the intake of macronutrients, except N6 and ALA, decreased significantly between weeks 0 and 26, with intake of all macronutrients decreasing by week 52. When macronutrients were modelled as a percentage of total energy intake, percentages of energy from sugars, total fats, and saturated fats decreased and percentage of energy from N6 increased between weeks 0 and 26, with only the decrease in energy from saturated fats being maintained at week 52. Percentage of energy from protein increase between weeks 0 and 52. Only percent in energy from ALA changed between weeks 26 and 52, decreasing over this time. Few changes were found in intake of vitamins over treatment with only retinol intake decreasing between weeks 0 and 26, and this decrease being maintained at the cessation of treatment. Minerals, however, appeared to mimic the trajectories of total energy and macronutrient intake, with intakes of all minerals (except selenium) decreasing between weeks 0 and 26, and most of these decreases in intake being maintained at week 52.

Macronutrient adequacy increased between weeks 0 and 26, however, there were no differences in macronutrient adequacy between weeks 0 and 52. Micronutrient adequacy decreased between weeks 0 and 26, and this decrease was maintained at week 52. Total adequacy did not change significantly over treatment.

At week 0, women with BN were only distinguishable from women with BED on intakes of sodium and iodine, with participants with BN consuming more of both. Additionally, diagnostic groups did not differ in any way on how they changed nutritionally over time.

Women who were treated with CBT or ST were indistinguishable from women treated with CBT-A in changes in nutritional intake and nutritional adequacy over time.

Interpretation of Results and Comparisons to Established Findings

Nutritional Intake.

Total Energy Intake. Energy intake decreased over the intensive first half of treatment, and this reduction was maintained at the cessation of treatment. This indicates that weekly sessions of psychotherapy for binge eating are sufficient to reduce the energy intake of women who binge eat, and monthly sessions are sufficient to maintain these lower levels of intake. While it was hypothesised that the modified nutritional advice of CBT-A, which included additional psychoeducation about eating satiating foods, recognising internal cues of hunger and satiety, and the supplementation of protein-rich foods, would lead to additional reductions in energy intake compared to CBT and ST, this was not the case. It should be noted that while only 16.5% of the total sample had an adequate energy intake at the start of treatment, the observed decrease in energy intake was mirrored by this percentage decreasing to 0% at both weeks 26 and 52. This could be due to several factors. First, this may reflect an actual reduction in adequacy, meaning the focus of reducing energy intake may have not been justified. Second, the process that was used to calculate adequate energy intake ranges may

have provided adequacy ranges that were too high for participants' actual needs, or criteria that were too strict. However, whether participants were inadequately low or inadequately high was not measured. Lastly, adequate ranges of energy intake were calculated using an artificial weight that would give a BMI of 22 given their actual height, meaning that these ranges reflect an intake of energy that is needed to maintain a healthy body weight. While not reported in this study, the BMI of people who binge eat has been found to be higher than the general population, especially for those with BED (Cachelin et al., 2019; Lydecker et al., 2019; Mathisen et al., 2018). Therefore, to maintain higher body weights would require a significantly higher amount of energy.

Few studies have investigated the effect of psychotherapy on the nutritional intake of people who binge eat. Masheb et al. (2016) found that participants with BED had significantly decreased total energy intakes, and intakes of macronutrients over a six-month course of CBT and dietary counselling. Masheb et al. found this decrease in energy and macronutrient intake within the context of a decrease in frequency of binge eating episodes over treatment. Similarly, within the current study, the reduction in energy and weighted macronutrient intake is observed alongside a decrease in binge eating episodes (McIntosh et al., 2016). Kales et al. (1990) reported that approximately 70% of foods consumed during binge eating episodes were classified by participants as forbidden foods with high caloric value. Reductions in binge eating episodes, and consequent reductions in forbidden foods might partially explain the significant decrease in energy intake. Additionally, more frequent binge eating episodes are related to higher energy intake (Gendall et al., 1997; Latner et al., 2008), meaning reductions in binge eating episodes may directly account for the reduction in energy intake. However, whether or not the observed reduction in energy intake can be attributed to this reduction in binge eating episodes was not investigated within the current study.

Macronutrient Intake. As expected, the decrease in energy intake was mirrored by decreases in the weighted intakes of almost all macronutrients between weeks 0 and 26, with exception of N6 and ALA intakes. All macronutrient intakes decreased significantly by week 52. Again, CBT-A had no additional effect on macronutrient intake compared to the CBT + ST condition, despite providing extra education about selecting foods with macronutrient compositions that optimise satiety, and supplementation with protein-rich foods.

While modelling macronutrient weighted intake indicated reduced intakes of all macronutrients by week 52, there were fewer significant changes over time in macronutrient intake as a proportion of total energy. During the first six months of weekly therapy sessions, percent of energy from sugars, total fats, and saturated fats decreased and percent of energy from N6 increased, however, only the reduction in percent of total energy from saturated fats was maintained at the end of treatment. Over the full course of treatment, percent of total energy from protein increased, and percent of total energy from ALA increased during the first half of treatment. It was expected that CBT-A would have greater increase in percent of energy from protein due to the advice about increasing percent of energy intake from protein and additional supplementation of protein-rich foods, however this was not the case. Recommendations in CBT and ST included general advice about selecting a range of foods as sources of protein. Therefore, recommendations in CBT and ST may have been sufficient to increase percent of energy from protein, with additional information about the satiating properties of high-protein foods and the supplementation of participants with high-protein foods, such as in CBT-A, providing no added benefit.

There is evidence to suggest that reductions in binge eating episodes may also account for reductions in particular macronutrients. Ayton et al. (2021) found binge eating episodes to consist solely of ultra-processed foods that are high in carbohydrates and fats. Reductions in percent of total energy from sugars, total fats and saturated fats found within the current study

may be explained by reductions in binge eating episodes, and therefore reduction in the intake of ultra-processed foods that are concentrated in these nutrients.

Compared to controls, Hetherington et al. (1994) found the diets of people with BN to have a higher percentage of energy from fats and a lower percentage of energy from protein, and for binge eating episodes to be particularly high in percent of energy from fats and carbohydrates (Alpers & Tuschen-Caffier, 2004; Gendall et al., 1997). Along with findings from the current study, these existing findings suggest that a reduction in binge eating post-treatment may normalise macronutrient ratios. The current study found increased percent of energy from protein across treatment, and decreased percent of energy from fats, saturated fats and sugars, occurring alongside decreases in the frequency of binge eating episodes.

Micronutrient Intake. Few changes were observed in vitamin intake, with only retinol intake changing over treatment. As total energy decreases, it is expected that all nutrients will also decrease due to a reduction in total food. The stability of vitamin intake over treatment may be explained by a change in the concentration of vitamins within participants' diets. While less food was consumed, vitamin concentrations may have increased, possibly explaining why the weighted intake of most vitamins did not change over time. If the lack of change in vitamin intakes over time was due to an increase in concentration, this would suggest that treatment influences decisions about food selection, with participants selecting more vitamin-dense foods such as fruits and vegetables. Increases in the consumption of vitamin-dense foods have been found in previous studies. For example, Masheb et al. (2011) found that participants increased their servings of fruits and vegetables over the course of treatment.

Intake of most minerals (all except selenium) were found to decrease between weeks 0 and 26, and these changes were maintained at week 52, with the exception of iron intake.

As the amount of food eaten decreased, so did mineral intake, possibly indicating that mineral concentrations did not vary across treatment.

Nutritional Adequacy

Macronutrient adequacy was found to increase between weeks 0 and 26. This change was mirrored by a decrease in the intake of most macronutrients between weeks 0 and 26. However, during this time there were significant changes in the percentage of total energy from protein, sugars, total fats, saturated fats, N6, and ALA, indicating that changes in proportions of energy from these macronutrients may have led to more participants meeting the associated acceptable macronutrient distribution ranges. Whether participants were inadequately low or inadequately high, and whether changes in specific nutrient intakes contributed to changes in adequacy were not examined in the current study.

Micronutrient adequacy was found to decrease between weeks 0 and 26, and this decrease was maintained at week 52. This was mirrored by a decrease in the intakes of numerous micronutrients (mostly minerals) during this time. It is likely that such decreases put more participants below the recommended daily intake or acceptable intake levels, decreasing their micronutrient adequacy scores. The contribution of changes in intake of specific nutrients to changes in adequacy measures was not quantified in this study.

While statistically significant changes in micronutrient and macronutrient scores were found, these should not necessarily be considered clinically significant. While macronutrient adequacy scores increased significantly during the first half of treatment, this was due to an estimated average increase of 0.73, meaning on average, participants became increasingly adequate on only 0.73 nutrients over this time. Similarly with micronutrients, a significant estimated drop in micronutrient adequacy during the first half of treatment of -0.92 means that on average, participants became less adequate by only 0.92 micronutrients.

Total adequacy scores were not found to change over treatment, indicating that treatment did not impact the overall nutritional health of participants.

Group Differences

Very little separated diagnostic groups nutritionally. Women with BN were distinguishable from women with BED on intakes of only sodium and iodine, with women with BN consuming more of both nutrients at week 0. Additionally, diagnostic groups did not differ in any way on how they changed nutritionally over time, indicating that the effect of psychotherapy on nutritional intake is not diagnosis-specific. Little research has compared the nutritional intakes of people with BN vs. BED. The current study addresses this gap by describing and comparing the nutritional intakes of women with BN and BED and contrasts their nutritional responses to treatment.

Similarly, the two treatment types did not differ in how they changed in nutritional intake and nutritional adequacy over time, indicating that the augmented recommendations for normalising eating in CBT-A, specifically the amended nutritional information, use of high-protein supplements, and psychoeducation about hunger and satiety cues, did not have additive benefit in bringing about nutritional change.

Lowe et al. (2008) found that supplementing traditional CBT with psychoeducation about diets with a low energy density led to favourable changes in energy intake. While the current study found reductions in energy intake, this reduction was similar between treatment types, indicating that the additional emphasis on psychoeducation about diets with a low energy density in CBT-A may not have provided additional benefit.

Encouraging participants to include more sources of protein in their diet, a component of all therapies included in the study, may have affected participants' diets, as evidenced by an increase in percentage of energy from protein over the course of treatment. This may have contributed to the observed reduction in energy intake, as a higher protein intake has been

associated with more satiety (Astrup, 2005; Westerterp-Plantenga et al., 1999; Westman et al., 2002; Yancy et al., 2004), meaning high-protein meals can reduce caloric consumption. However, supplementing participants with high-protein foods and providing recommendations about increasing proportion of energy intake from protein in CBT-A appeared to have no additive benefit, as there was no differences between treatment types in changes in protein intake and percent of energy from protein over treatment.

There were reductions in total energy from sugars, and increases in total energy from protein between weeks 0 and 52. Foods high in sugars tend to have a high glycaemic index (Miller et al., 1995) and foods with high protein to sugar ratios tend to have a lower glycaemic index (Dorenbos et al., 2020). While CBT-A provides psychoeducation about selecting foods with a low glycaemic index, this did not appear to have any additional effect as there were no significant differences in the way treatment groups changed in percent of energy from protein or sugars over this period. The lack of additional nutritional change in women who were treated with CBT-A indicates that psychoeducation about foods that provide a slower glycaemic response is likely redundant, and the nutritional psychoeducational material within CBT and ST is sufficient to bring about the observed changes.

Implications for Treatment

The results from the current study indicate that psychotherapy for binge eating significantly alters energy, macronutrient, and mineral intakes, and these changes in dietary intake result in changes in nutritional adequacy. The majority of nutritional changes found within the current study occurred within the first half of treatment, where weekly sessions were provided. For many of these changes, monthly sessions were enough to maintain these alterations in dietary intake. These findings suggest that more intensive therapy leads to greater nutritional change.

While a shared goal of psychotherapies for transdiagnostic binge eating is to normalise eating, the reduction in micronutrient adequacy that was observed alongside a decrease in caloric intake, especially reduced mineral intake, is not well-understood. While people who binge eat have previously been shown to have inadequately low mineral intakes (Alvarenga et al., 2003; Gendall et al., 1997; Siega-Riz et al., 2008), further reductions in intake may lead to serious health concerns. For example, while people who binge eat have been shown to have inadequately low iron intake (Gendall et al., 1997), further disturbances in these levels may lead to conditions such as anaemia.

Despite variation in treatment delivery between people with BN and BED, specifically variation in nutritional advice, the current study found that women with BN and BED differ in the intakes of only two nutrients, and found no differences between diagnoses in nutritional response to treatment. These findings suggest that transdiagnostic psychotherapy for different forms of binge eating, can result in nutritionally indistinguishable outcomes for women with BN and BED.

Results from the current study indicate comparable nutritional effects for women given appetite-focussed advice for normalising eating compared with traditional advice within CBT or ST. Therefore, the amended nutritional advice about selecting more satiating foods, nutritional supplementation, and psychoeducation about satiety and hunger cues, does not appear to bring about additional changes when compared with those treated with CBT or ST.

Study Strengths and Limitations

The current study provides both a fine-grain, detailed analysis of the nutritional status of women who binge eat both before and across treatment, and a general overview of nutritional adequacy and how this changed over treatment. Results from the current study fill gaps within existing literature about the nutritional health of people who binge eat. Prior to

this study, no studies have comprehensively compared the diets of people with BN vs. BED. Additionally, few studies have assessed changes in nutritional intake over treatment for binge eating, and no studies had compared treatments' impact on nutritional health. CBT-A appears to have comparable treatment outcomes to well-established psychotherapies such as CBT (McIntosh et al., 2016). However, despite augmenting traditional CBT with material that aims to provide additional benefit in restoring nutritional health for people who binge eat, prior to the current study, the nutritional outcomes of CBT-A were unknown.

It is well known that a weakness of psychotherapies for binge eating is the high rates of treatment attrition (Linardon et al., 2018), therefore sample sizes for studies similar to the current study are limited by the number of participants exposed to an adequate dose of therapy to expect a change in nutrition. Common statistical analyses such as rm-ANOVAs require data points at each possible observation, often restricting sample sizes when there are missing observations. While the current study has a comparatively small sample size, it maximised the sample size available by using a more statistically flexible approach, linear mixed modelling, where data points are not required at each possible observation, maintaining sample size, and therefore, statistical power.

The current study included only women. While eating disorders are more common in the female population, current estimations of male prevalence rates may be underestimations due to diagnostic gender bias (Carey et al., 2017). It is uncertain whether results of the current study are generalizable to males with binge eating disorders.

Another possible limitation is that food monitoring was self-reported. Participants may alter their reporting of food and fluid intake due to feelings of shame or fear of judgement. However, the current study minimised this by modelling acceptance of large quantities of food and fluid intake such as in binge eating episodes. Additionally, seven-day prospective food records are an accurate and simple method of dietary recording (Edington et

al., 1989; Prentice et al., 2011) as they minimise recall errors and are as accurate as food records lasting up to 16 days (Bingham et al., 1994).

Having multiple individuals entering food records into the nutritional software, FoodWorks, had the potential to reduce standardisation when selecting representative foods from the FoodWorks-associated databases, or when selecting appropriate portion sizes. Errors in selecting representative foods and portion sizes were minimised by creating a spreadsheet of the decisions made when selecting representative foods and portion sizes, and this was reviewed regularly.

Further Research

While the current study was large and investigated multiple topics, many questions remain unexplored. To determine whether reductions in energy, macronutrient, and mineral intakes are attributable to a reduction in binge eating episodes, comparisons of the nutritional composition of food between binge eating and non-binge eating episodes are recommended.

Additionally, no comparisons were made to a control sample, or to data from the general 'healthy' population. In the current study, nutritional intake was compared to empirically determined healthy intakes (e.g., recommended daily intakes), providing proportions of participants that were adequate for each nutrient. However, whether these levels of adequacy differ from the general population is unknown. While percentages of people with adequate intake of each nutrient were calculated, nutritional adequacy was analysed across time by comparing composite scores of total, macronutrient, and micronutrient adequacy. While these measures provide information on the overall nutritional adequacy of the sample over time, whether the proportion of participants with adequate intake of each nutrient changed over time is unknown. Additionally, composite measures of adequacy weight all nutrients equally as there is no way to quantify the impact of each nutrient on the health of the individual in relation to one another. Generalised linear mixed

modelling would allow the adequacy of individual nutrients to be modelled over time and between groups.

Conclusion

The current study found that over treatment, for women who binge eat, total energy, macronutrient, and mineral intakes decreased over the intensive first-half of treatment, and most of these decreases were maintained at the cessation of treatment. Vitamin intake appears to be consistent over treatment despite decreases in food intake. When macronutrients were analysed as a percentage of total energy intake, energy from sugars, total fats, and saturated fats decreased and energy from N6 increased over the first half of treatment, energy from ALA decreased over the second half of treatment, and energy from protein increased over the whole of treatment. These findings suggest that treatment affects food selection choices as treatment was associated with decreases in the concentrations of sugars, fats, saturated fats, and ALA, and increases in concentrations of protein, N6, and likely higher concentrations of vitamins.

Macronutrient adequacy increased during the first half of treatment, and micronutrient adequacy decreased over the first half of treatment which was maintained at the cessation of treatment, suggesting that reductions in the intake of most nutrients in the first half of treatment are associated with an increase in macronutrient adequacy and a decrease in micronutrient adequacy. Total adequacy did not change over treatment, suggesting the overall nutritional health of individuals does not change.

Women with BN and BED were distinguishable only on measures of sodium and iodine and did not differ in how they changed over treatment in any measure of nutritional intake or nutritional adequacy. These findings indicate that women with BN and BED have similar nutritional responses to transdiagnostic psychotherapy for binge eating.

There were no differences in how women treated with CBT or ST and CBT-A changed nutritionally over treatment. This indicates that the additional appetite-focus in CBT-A, namely psychoeducation about appetite and satiety, encouraging participants to eat more satiating foods (e.g., foods high in protein and with a relatively lower glycaemic index), and supplementing participants' diets with high-protein foods, had no additive impact on nutritional remediation.

The current study fills gaps within the binge eating literature, particularly the impact of psychotherapy on the nutritional health of people who binge eat, whether women with different binge eating-related disorders differ in nutritional status or nutritional responses to psychotherapy, and whether CBT-A provides additional benefit to remediating the nutritional health of people who binge eat.

As the study made no comparisons to healthy controls or to the general population, future research should compare nutritional intake and nutritional adequacy to that of individuals who do not binge eat. Future efforts should also investigate whether the observed findings are similar for men who binge eat. Additionally, adequacy for individual nutrients should be compared across treatment to determine if the observed changes in nutritional intake are directly related to improvements in nutritional health.

Tables

Table 16

Three Linear Mixed Models Modelling Total Energy Intake with Random Intercepts and Fixed Effects (Time, Group, and Time × Group Interactions).

		Random effects	Fixed effects					
		Intercept	Time		Group		Interaction	
Model		*SD*	Estimate (*SE*)	*t*	Estimate (*SE*)	*t*	Estimate (*SE*)	*t*
1	Intercept	4276.10	12531.57 (399.53)	31.37 ***				
	0 – 26		-2525.81 (507.58)	-4.97***				
	0 – 52		-2873.46 (510.15)	-5.63***				
	26 – 52		-348 (530)	-0.66				
2	Intercept	1745.46	13379.07 (562.72)	23.78***				
	BN – BED				-1662.92 (790)	-2.11		
	0 – 26 × Diag						-893.17 (1018.79)	-0.88
	0 – 52 × Diag						-201.00 (1021.77)	-0.20
3	Intercept	1802.56	12585.31 (468.83)	26.84***				
	CBT – CBT-A				-187.17 (897.07)	-0.21		
	0 – 26 × Treat						970.89 (1146.90)	0.85
	0 – 52 × Treat						766.426 (1144.55)	0.67

Note. Diag = diagnosis group; Treat = treatment type.

* $p < .05$; ** $p < .01$; *** $p < .001$.

Table 17

Three Linear Mixed Models Modelling Protein Intake with Random Intercepts and Fixed Effects (Time, Group, and Time ✕ Group Interactions).

		Random effects	Fixed effects					
		Intercept	Time		Group		Interaction	
Model		*SD*	Estimate (*SE*)	*t*	Estimate (*SE*)	*t*	Estimate (*SE*)	*t*
1	Intercept	17.06	113.85 (3.89)	29.29***				
	0 – 26		-14.44 (5.02)	-2.87*				
	0 – 52		-13.08 (5.05)	-2.59*				
	26 – 52		1.36 (5.25)	0.26				
2	Intercept	15.20	122.33 (5.38)	22.76***				
	BN – BED				-16.60 (7.55)	-2.20		
	0 – 26 ✕ Diag						-8.70 (10.00)	-0.87
	0 – 52 ✕ Diag						9.43 (10.03)	0.94
3	Intercept	16.81	115.54 (4.58)	25.24***				
	CBT – CBT-A				-6.17 (8.76)	-0.70		
	0 – 26 ✕ Treat						-1.16 (11.37)	-0.10
	0 – 52 ✕ Treat						-0.87 (11.35)	-0.08

Note. Diag = diagnosis group; Treat = treatment type.
* $p < .05$; ** $p < .01$; *** $p < .001$.

Table 18

Three Linear Mixed Models Modelling Total Carbohydrate Intake with Random Intercepts and Fixed Effects (Time, Group, and Time ✕ Group Interactions).

		Random effects	Fixed effects					
		Intercept	Time		Group		Interaction	
Model		*SD*	Estimate (*SE*)	*t*	Estimate (*SE*)	*t*	Estimate (*SE*)	*t*
1	Intercept	62.02	355.80 (12.60)	28.23***				
	0 – 26		-79.93 (15.6)	-5.12***				
	0 – 52		-80.64 (15.7)	-5.14***				
	26 – 52		-0.71 (16.3)	-0.04				
2	Intercept	60.12	376.71 (17.88)	21.07***				
	BN – BED				-41.0 (25.1)	-1.64		
	0 – 26 ✕ Diag						-24.65 (31.41)	-0.78
	0 – 52 ✕ Diag						-1.32 (31.50)	-0.04
3	Intercept	60.08	356.90 (14.73)	24.22***				
	CBT – CBT-A				-3.63 (28.2)	-0.13		
	0 – 26 ✕ Treat						40.62 (35.26)	1.15
	0 – 52 ✕ Treat						32.68 (35.17)	0.93

Note. Diag = diagnosis group; Treat = treatment type.

* $p < .05$; ** $p < .01$; *** $p < .001$.

Table 19

Three Linear Mixed Models Modelling Sugar intake with Random Intercepts and Fixed Effects (Time, Group, and Time ✕ Group Interactions).

		Random effects	Fixed effects					
		Intercept	Time		Group		Interaction	
Model		*SD*	Estimate (*SE*)	*t*	Estimate (*SE*)	*t*	Estimate (*SE*)	*t*
1	Intercept	36.13	174.61 (7.03)	24.75***				
	0 – 26		-47.37 (8.58)	-5.52***				
	0 – 52		-44.25 (8.63)	-5.13***				
	26 – 52		3.12 (8.96)	0.35				
2	Intercept	35.92	180.85 (10.09)	17.92***				
	BN – BED				-12.26 (14.2)	-0.87		
	0 – 26 ✕ Diag						-12.33 (17.29)	-0.71
	0 – 52 ✕ Diag						0.74 (17.34)	0.04
3	Intercept	35.73	171.39 (8.28)	20.71***				
	CBT – CBT-A				11.97 15.8	0.76		
	0 – 26 ✕ Treat						29.85 (19.31)	1.55
	0 – 52 ✕ Treat						22.00 (19.25)	1.14

Note. Diag = diagnosis group; Treat = treatment type.

* $p < .05$; ** $p < .01$; *** $p < .001$.

Table 20

Three Linear Mixed Models Modelling Total Fat Intake with Random Intercepts and Fixed Effects (Time, Group, and Time ✕ Group Interactions).

		Random effects	Fixed effects					
		Intercept	Time		Group		Interaction	
Model		*SD*	Estimate (*SE*)	*t*	Estimate (*SE*)	*t*	Estimate (*SE*)	*t*
1	Intercept	18.53	115.21 (4.31)	26.75***				
	0 – 26		-29.32 (5.60)	-5.23***				
	0 – 52		-30.59 (-5.63)	-5.43***				
	26 – 52		-1.28 (5.85)	-0.22				
2	Intercept	18.30	120.17 (6.15)	19.55***				
	BN – BED				-9.73 (8.63)	-1.13		
	0 – 26 ✕ Diag						-5.59 (11.27)	-0.50
	0 – 52 ✕ Diag						0.67 (11.30)	0.06
3	Intercept	18.45	114.76 (5.08)	22.60***				
	CBT – CBT-A				1.71 (9.72)	0.18		
	0 – 26 ✕ Treat						8.85 (12.65)	0.70
	0 – 52 ✕ Treat						7.57 (12.62)	0.60

Note. Diag = diagnosis group; Treat = treatment type.

* $p < .05$; ** $p < .01$; *** $p < .001$.

Table 21

Three Linear Mixed Models Saturated Fat Intake of Food with Random Intercepts and Fixed Effects (Time, Group, and Time ✕ Group Interactions).

		Random effects	Fixed effects					
		Intercept	Time		Group		Interaction	
Model		*SD*	Estimate (*SE*)	*t*	Estimate (*SE*)	*t*	Estimate (*SE*)	*t*
1	Intercept	8.81	46.19 (1.90)	24.30***				
	0 – 26		-13.52 (2.41)	-5.60***				
	0 – 52		-13.52 (2.42)	-5.58***				
	26 – 52		-0.01 (2.54)	-0.00				
2	Intercept	8.74	47.99 (2.72)	17.65***				
	BN – BED				-3.52 (3.82)	-0.92		
	0 – 26 ✕ Diag						-2.71 (4.86)	-0.56
	0 – 52 ✕ Diag						-0.12 (4.88)	-0.02
3	Intercept	8.81	45.58 (2.24)	20.35***				
	CBT – CBT-A				2.25 (4.29)	0.52		
	0 – 26 ✕ Treat						4.94 (5.45)	0.91
	0 – 52 ✕ Treat						4.83 (5.43)	0.89

Note. Diag = diagnosis group; Treat = treatment type.

* $p < .05$; ** $p < .01$; *** $p < .001$.

Table 22

Three Linear Mixed Models Modelling Trans Fat Intake with Random Intercepts and Fixed Effects (Time, Group, and Time ✕ Group Interactions).

		Random effects	Fixed effects					
		Intercept	Time		Group		Interaction	
Model		*SD*	Estimate (*SE*)	*t*	Estimate (*SE*)	*t*	Estimate (*SE*)	*t*
1	Intercept	0.30	1.56 (0.07)	21.64***				
	0 – 26		-0.23 (0.09)	-2.38*				
	0 – 52		-0.33 (0.10)	-3.48*				
	26 – 52		-0.11 (0.10)	-1.07				
2	Intercept	0.30	1.66 (0.10)	16.21***				
	BN – BED				-0.20 (0.14)	-1.36		
	0 – 26 ✕ Diag						-0.26 (0.19)	-1.35
	0 – 52 ✕ Diag						0.07 (0.19)	0.38
3	Intercept	0.29	1.62 (0.08)	19.16***				
	CBT – CBT-A				-0.19 (0.16)	-1.17		
	0 – 26 ✕ Treat						-0.06 (0.21)	-0.27
	0 – 52 ✕ Treat						0.12 (0.21)	0.57

Note. Diag = diagnosis group; Treat = treatment type.

* $p < .05$; ** $p < .01$; *** $p < .001$.

Table 23

Three Linear Mixed Models Modelling Monounsaturated Fatty Acid Intake with Random Intercepts and Fixed Effects (Time, Group, and Time × Group Interactions).

		Random effects	Fixed effects					
		Intercept	Time		Group		Interaction	
Model		*SD*	Estimate (*SE*)	*t*	Estimate (*SE*)	*t*	Estimate (*SE*)	*t*
1	Intercept	6.64	40.92 (1.63)	25.16***				
	0 – 26		-9.87 (2.15)	-4.60***				
	0 – 52		-10.61 (2.16)	-4.92***				
	26 – 52		-0.74 (2.24)	-0.33				
2	Intercept	6.51	43.01 (2.32)	18.57***				
	BN – BED				-4.10 (3.25)	-1.26		
	0 – 26 × Diag						-1.88 (4.32)	-0.44
	0 – 52 × Diag						0.26 (4.33)	0.06
3	Intercept	6.61	40.80 (1.91)	21.27***				
	CBT – CBT-A				0.48 (3.67)	0.13		
	0 – 26 × Treat						3.07 (4.85)	0.63
	0 – 52 × Treat						2.65 (4.84)	0.55

Note. Diag = diagnosis group; Treat = treatment type.

* $p < .05$; ** $p < .01$; *** $p < .001$.

Table 24

Three Linear Mixed Models Modelling Polyunsaturated Fatty Acid Intake with Random Intercepts and Fixed Effects (Time, Group, and Time ✕ Group Interactions).

		Random effects	Fixed effects					
		Intercept	Time		Group		Interaction	
Model		*SD*	Estimate (*SE*)	*t*	Estimate (*SE*)	*t*	Estimate (*SE*)	*t*
1	Intercept	2.89	17.65 (0.75)	23.52***				
	0 – 26		-3.07 (1.00)	-3.06**				
	0 – 52		-3.94 (1.01)	-3.91***				
	26 – 52		-0.88 (1.05)	-0.83				
2	Intercept	2.88	18.40 (1.07)	17.14***				
	BN – BED				-1.47 (1.51)	-0.98		
	0 – 26 ✕ Diag						-0.86 (2.02)	-0.43
	0 – 52 ✕ Diag						-0.24 (2.02)	-0.12
3	Intercept	2.89	17.53 (0.89)	19.79***				
	CBT – CBT-A				0.45 (1.70)	0.27		
	0 – 26 ✕ Treat						1.29 (2.27)	0.57
	0 – 52 ✕ Treat						1.21 (2.27)	0.53

Note. Diag = diagnosis group; Treat = treatment type.

* $p < .05$; ** $p < .01$; *** $p < .001$.

Table 25

Three Linear Mixed Models Modelling Omega-6 Fatty Acid (N6) Intake with Random Intercepts and Fixed Effects (Time, Group, and Time ✕ Group Interactions).

		Random effects	Fixed effects					
		Intercept	Time		Group		Interaction	
Model		*SD*	Estimate (*SE*)	*t*	Estimate (*SE*)	*t*	Estimate (*SE*)	*t*
1	Intercept	2.72	13.29 (0.61)	21.63***				
	0 – 26		-1.35 (0.79)	-1.71				
	0 – 52		-2.51 (0.80)	-3.15**				
	26 – 52		-1.15 (0.83)	-1.40				
2	Intercept	2.75	13.38 (0.88)	15.20***				
	BN – BED				-0.19 (1.24)	-0.16		
	0 – 26 ✕ Diag						0.31 (1.59)	1.19
	0 – 52 ✕ Diag						1.07 (1.60)	0.67
3	Intercept	2.72	13.15 (0.72)	18.16***				
	CBT – CBT-A				0.51 (1.39)	0.37		
	0 – 26 ✕ Treat						0.98 (1.78)	0.55
	0 – 52 ✕ Treat						1.91 (1.78)	1.07

Note. Diag = diagnosis group; Treat = treatment type.

* $p < .05$; ** $p < .01$; *** $p < .001$.

Table 26

Three Linear Mixed Models Modelling N3 α-Linolenic Acid (ALA) Intake with Random Intercepts and Fixed Effects (Time, Group, and Time ✕ Group Interactions).

		Random effects	Fixed effects					
		Intercept	Time		Group		Interaction	
Model		*SD*	Estimate (*SE*)	*t*	Estimate (*SE*)	*t*	Estimate (*SE*)	*t*
1	Intercept	0.43	2.25 (0.13)	17.73***				
	0 – 26		-0.25 (0.18)	-1.46				
	0 – 52		-0.68 (0.18)	-3.94***				
	26 – 52		-0.44 (0.18)	-2.40*				
2	Intercept	0.42	2.39 (0.18)	13.17***				
	BN – BED				-0.28 (0.26)	-1.08		
	0 – 26 ✕ Diag						-0.29 (0.35)	-0.83
	0 – 52 ✕ Diag						-0.09 (0.35)	-0.26
3	Intercept	0.43	2.27 (0.15)	15.14***				
	CBT – CBT-A				-0.07 (0.29)	-0.24		
	0 – 26 ✕ Treat						-0.12 (0.39)	-0.31
	0 – 52 ✕ Treat						-0.23 (0.39)	-0.58

Note. Diag = diagnosis group; Treat = treatment type.

* $p < .05$; ** $p < .01$; *** $p < .001$.

Table 27

Three Linear Mixed Models Modelling Protein Intake as a Percentage of Total Energy with Random Intercepts and Fixed Effects (Time, Group, and Time × Group Interactions).

		Random effects	Fixed effects					
		Intercept	Time		Group		Interaction	
Model		*SD*	Estimate (*SE*)	*t*	Estimate (*SE*)	*t*	Estimate (*SE*)	*t*
1	Intercept	1.12	15.57 (0.47)	33.04***				
	0 – 26		1.41 (0.67)	2.09				
	0 – 52		2.38 (0.68)	3.52**				
	26 – 52		0.97 (0.70)	1.38				
2	Intercept	1.11	15.71 (0.67)	23.37***				
	BN – BED				-0.27 (0.94)	-0.29		
	0 – 26 × Diag						-0.48 (1.35)	-0.35
	0 – 52 × Diag						1.23 (1.35)	0.91
3	Intercept	1.14	15.74 (0.55)	28.43***				
	CBT – CBT-A				-0.65 (1.06)	-0.61		
	0 – 26 × Treat						-1.70 (1.51)	-1.12
	0 – 52 × Treat						-1.75 (1.51)	-1.16

Note. Diag = diagnosis group; Treat = treatment type.
* $p < .05$; ** $p < .01$; *** $p < .001$.

Table 28

Three Linear Mixed Models Modelling Total Carbohydrate Intake as a Percentage of Total Energy with Random Intercepts and Fixed Effects (Time, Group, and Time × Group Interactions).

		Random effects	Fixed effects					
		Intercept	Time		Group		Interaction	
Model		*SD*	Estimate (*SE*)	*t*	Estimate (*SE*)	*t*	Estimate (*SE*)	*t*
1	Intercept	3.98	47.40 (0.72)	66.02***				
	0 – 26		-1.64 (0.84)	-1.97				
	0 – 52		0.00 (0.84)	0.00				
	26 – 52		1.65 (0.87)	1.89				
2	Intercept	3.97	46.74 (1.03)	45.42***				
	BN – BED				1.29 (1.44)	0.90		
	0 – 26 × Diag						0.31 (1.68)	0.18
	0 – 52 × Diag						0.98 (1.69)	0.58
3	Intercept	3.95	47.46 (0.84)	56.21***				
	CBT – CBT-A				-0.18 (1.61)	-0.11		
	0 – 26 × Treat						1.94 (1.89)	1.03
	0 – 52 × Treat						0.69 (1.88)	0.37

Note. Diag = diagnosis group; Treat = treatment type.

* $p < .05$; ** $p < .01$; *** $p < .001$.

Table 29

Three Linear Mixed Models Modelling Sugar Intake as a Percentage of Total Energy with Random Intercepts and Fixed Effects (Time, Group, and Time × Group Interactions).

		Random effects	Fixed effects					
		Intercept	Time		Group		Interaction	
Model		*SD*	Estimate (*SE*)	*t*	Estimate (*SE*)	*t*	Estimate (*SE*)	*t*
1	Intercept	4.51	23.49 (0.73)	32.18***				
	0 – 26		-2.39 (0.78)	-3.06**				
	0 – 52		-1.13 (0.79)	-1.44				
	26 – 52		1.26 (0.82)	1.55				
2	Intercept	4.48	22.85 (1.05)	21.84***				
	BN – BED				1.25 (1.47)	0.85		
	0 – 26 × Diag						-0.22 (1.58)	-0.14
	0 – 52 × Diag						0.40 (1.58)	0.25
3	Intercept	4.51	23.19 (0.86)	26.93***				
	CBT – CBT-A				1.11 (1.65)	0.68		
	0 – 26 × Treat						1.99 (1.76)	1.13
	0 – 52 × Treat						0.70 (1.76)	0.40

Note. Diag = diagnosis group; Treat = treatment type.

* $p < .05$; ** $p < .01$; *** $p < .001$.

Table 30

Three Linear Mixed Models Modelling Total Fat Intake as a Percentage of Total Energy with Random Intercepts and Fixed Effects (Time, Group, and Time × Group Interactions).

		Random effects	Fixed effects					
		Intercept	Time		Group		Interaction	
Model		*SD*	Estimate (*SE*)	*t*	Estimate (*SE*)	*t*	Estimate (*SE*)	*t*
1	Intercept	3.73	34.35 (0.68)	50.22***				
	0 – 26		-2.28 (0.80)	-2.83*				
	0 – 52		-1.65 (0.81)	-2.04				
	26 – 52		0.63 (0.84)	0.75				
2	Intercept	3.71	33.82 (0.98)	34.51***				
	BN – BED				1.04 (1.04)	0.75		
	0 – 26 × Diag						0.61 (1.62)	0.38
	0 – 52 × Diag						0.32 (1.63)	0.19
3	Intercept	3.70	33.96 (0.81)	42.15***				
	CBT – CBT-A				1.42 (1.54)	0.92		
	0 – 26 × Treat						0.27 (1.82)	0.15
	0 – 52 × Treat						0.83 (1.81)	0.46

Note. Diag = diagnosis group; Treat = treatment type.
* $p < .05$; ** $p < .01$; *** $p < .001$.

Table 31

Three Linear Mixed Models Modelling Saturated Fat Intake as a Percentage of Total Energy with Random Intercepts and Fixed Effects (Time, Group, and Time × Group Interactions).

		Random effects	Fixed effects					
		Intercept	Time		Group		Interaction	
Model		*SD*	Estimate (*SE*)	*t*	Estimate (*SE*)	*t*	Estimate (*SE*)	*t*
1	Intercept	1.79	13.72 (0.35)	38.76***				
	0 – 26		-1.61 (0.43)	-3.72***				
	0 – 52		-1.17 (0.44)	-2.70*				
	26 – 52		0.44 (0.45)	0.97				
2	Intercept	1.78	13.57 (0.51)	26.76***				
	BN – BED				0.30 (0.71)	0.42		
	0 – 26 × Diag						-0.31 (0.87)	-0.36
	0 – 52 × Diag						-0.11 (0.88)	-0.13
3	Intercept	1.80	13.45 (0.42)	32.29***				
	CBT – CBT-A				1.01 (0.80)	1.26		
	0 – 26 × Treat						0.47 (0.97)	0.48
	0 – 52 × Treat						1.30 (0.97)	1.34

Note. Diag = diagnosis group; Treat = treatment type.
* $p < .05$; ** $p < .01$; *** $p < .001$.

Table 32

Three Linear Mixed Models Modelling Trans Fat Intake as a Percentage of Total Energy with Random Intercepts and Fixed Effects (Time, Group, and Time × Group Interactions).

		Random effects	Fixed effects					
		Intercept	Time		Group		Interaction	
Model		*SD*	Estimate (*SE*)	*t*	Estimate (*SE*)	*t*	Estimate (*SE*)	*t*
1	Intercept	0.07	0.47 (0.02)	24.30***				
	0 – 26		0.03 (0.03)	1.12				
	0 – 52		0.01 (0.03)	0.20				
	26 – 52		-0.02 (0.03)	-0.88				
2	Intercept	0.07	0.48 (0.03)	17.39***				
	BN – BED				-0.02 (0.04)	-0.51		
	0 – 26 × Diag						-0.06 (0.05)	-1.22
	0 – 52 × Diag						0.02 (0.05)	0.32
3	Intercept	0.07	0.49 (0.02)	21.44***				
	CBT – CBT-A				-0.05 (0.04)	-1.08		
	0 – 26 × Treat						-0.04 (0.06)	-0.63
	0 – 52 × Treat						0.06 (0.06)	1.04

Note. Diag = diagnosis group; Treat = treatment type.

* $p < .05$; ** $p < .01$; *** $p < .001$.

Table 33

Three Linear Mixed Models Modelling Monounsaturated Fatty Acid Intake as a Percentage of Total Energy with Random Intercepts and Fixed Effects (Time, Group, and Time × Group Interactions).

		Random effects	Fixed effects					
		Intercept	Time		Group		Interaction	
Model		*SD*	Estimate (*SE*)	*t*	Estimate (*SE*)	*t*	Estimate (*SE*)	*t*
1	Intercept	1.62	12.16 (0.31)	39.81***				
	0 – 26		-0.53 (0.37)	-1.47				
	0 – 52		-0.43 (0.37)	-1.16				
	26 – 52		0.11 (0.38)	0.28				
2	Intercept	1.62	12.08 (0.44)	27.52***				
	BN – BED				0.17 (0.62)	0.28		
	0 – 26 × Diag						0.041 (0.74)	0.56
	0 – 52 × Diag						0.10 (0.74)	0.14
3	Intercept	1.61	12.02 (0.36)	33.37***				
	CBT – CBT-A				0.52 (0.69)	0.76		
	0 – 26 × Treat						0.18 (0.83)	0.22
	0 – 52 × Treat						0.29 (0.82)	0.35

Note. Diag = diagnosis group; Treat = treatment type.

* $p < .05$; ** $p < .01$; *** $p < .001$.

Table 34

Three Linear Mixed Models Modelling Polyunsaturated Fatty Acid Intake as a Percentage of Total Energy with Random Intercepts and Fixed Effects (Time, Group, and Time × Group Interactions).

		Random effects	Fixed effects					
		Intercept	Time		Group		Interaction	
Model		*SD*	Estimate (*SE*)	*t*	Estimate (*SE*)	*t*	Estimate (*SE*)	*t*
1	Intercept	0.89	5.34 (0.19)	28.23***				
	0 – 26		0.14 (0.24)	0.59				
	0 – 52		-0.04 (0.24)	-0.18				
	26 – 52		-0.18 (0.25)	-0.74				
2	Intercept	0.86	5.11 (0.27)	18.95***				
	BN – BED				0.46 (0.38)	1.21		
	0 – 26 × Diag						0.33 (0.48)	0.69
	0 – 52 × Diag						0.09 (0.48)	0.18
3	Intercept	0.87	5.27 (0.22)	23.70***				
	CBT – CBT-A				0.27 (0.43)	0.64		
	0 – 26 × Treat						-0.08 (0.54)	-0.14
	0 – 52 × Treat						-0.21 (0.54)	-0.38

Note. Diag = diagnosis group; Treat = treatment type.

* $p < .05$; ** $p < .01$; *** $p < .001$.

Table 35

Three Linear Mixed Models Modelling N6 Intake as a Percentage of Total Energy with Random Intercepts and Fixed Effects (Time, Group, and Time × Group Interactions).

		Random effects	Fixed effects					
		Intercept	Time		Group		Interaction	
Model		*SD*	Estimate (*SE*)	*t*	Estimate (*SE*)	*t*	Estimate (*SE*)	*t*
1	Intercept	0.81	4.04 (0.15)	26.11***				
	0 – 26		0.45 (0.19)	2.40*				
	0 – 52		0.08 (0.19)	0.41				
	26 – 52		-0.37 (0.20)	-1.90				
2	Intercept	0.80	3.72 (0.22)	16.93***				
	BN – BED				0.64 (0.31)	2.06		
	0 – 26 × Diag						0.55 (0.37)	1.48
	0 – 52 × Diag						0.48 (0.37)	1.27
3	Intercept	0.80	3.95 (0.18)	21.72***				
	CBT – CBT-A				0.32 (0.35)	0.93		
	0 – 26 × Treat						-0.05 (0.42)	-0.12
	0 – 52 × Treat						0.34 (0.42)	0.80

Note. Diag = diagnosis group; Treat = treatment type.

* $p < .05$; ** $p < .01$; *** $p < .001$.

Table 36

Three Linear Mixed Models Modelling ALA Intake as a Percentage of Total Energy with Random Intercepts and Fixed Effects (Time, Group, and Time ✕ Group Interactions).

		Random effects	Fixed effects					
		Intercept	Time		Group		Interaction	
Model		*SD*	Estimate (*SE*)	*t*	Estimate (*SE*)	*t*	Estimate (*SE*)	*t*
1	Intercept	0.16	0.68 (0.04)	16.92***				
	0 – 26		0.07 (0.05)	1.39				
	0 – 52		-0.07 (0.05)	-1.28				
	26 – 52		-0.14 (0.06)	-2.56*				
2	Intercept	0.16	0.66 (0.06)	11.46***				
	BN – BED				0.04 (0.08)	0.48		
	0 – 26 ✕ Diag						0.00 (0.11)	0.04
	0 – 52 ✕ Diag						0.04 (0.11)	0.35
3	Intercept	0.16	0.67 (0.05)	14.24***				
	CBT – CBT-A				0.04 (0.09)	0.43		
	0 – 26 ✕ Treat						-0.09 (0.12)	-0.75
	0 – 52 ✕ Treat						-0.10 (0.12)	-0.84

Note. Diag = diagnosis group; Treat = treatment type.

* $p < .05$; ** $p < .01$; *** $p < .001$.

Table 37

Three Linear Mixed Models Modelling Thiamine Intake with Random Intercepts and Fixed Effects (Time, Group, and Time × Group Interactions).

		Random effects	Fixed effects					
		Intercept	Time		Group		Interaction	
Model		*SD*	Estimate (*SE*)	*t*	Estimate (*SE*)	*t*	Estimate (*SE*)	*t*
1	Intercept	2.21	2.67 (0.68)	3.91***				
	0 – 26		1.43 (0.94)	1.51				
	0 – 52		-0.48 (0.95)	-0.51				
	26 – 52		-1.92 (0.99)	-1.93				
2	Intercept	2.23	3.48 (0.97)	3.57***				
	BN – BED				-1.59 (1.37)	-1.16		
	0 – 26 × Diag						-2.61 (1.89)	-1.38
	0 – 52 × Diag						-0.87 (1.90)	-0.46
3	Intercept	2.19	2.29 (0.78)	2.94***				
	CBT – CBT-A				1.37 (1.49)	0.92		
	0 – 26 × Treat						-4.51 (2.05)	-2.20
	0 – 52 × Treat						2.36 (2.05)	1.15

Note. Diag = diagnosis group; Treat = treatment type.

* $p < .05$; ** $p < .01$; *** $p < .001$.

Table 38

Three Linear Mixed Models Modelling Riboflavin Intake with Random Intercepts and Fixed Effects (Time, Group, and Time ✕ Group Interactions).

		Random effects	Fixed effects					
		Intercept	Time		Group		Interaction	
Model		*SD*	Estimate (*SE*)	*t*	Estimate (*SE*)	*t*	Estimate (*SE*)	*t*
1	Intercept	2.19	3.20 (0.67)	4.75***				
	0 – 26		1.20 (0.93)	1.29				
	0 – 52		-0.80 (0.94)	-0.86				
	26 – 52		-2.00 (0.97)	-2.06				
2	Intercept	2.20	3.94 (0.96)	4.08***				
	BN – BED				-1.45 (1.36)	-1.07		
	0 – 26 ✕ Diag						-1.95 (1.87)	-1.04
	0 – 52 ✕ Diag						-1.09 (1.88)	-0.58
3	Intercept	2.10	2.77 (0.77)	3.60***				
	CBT – CBT-A				1.58 (1.47)	1.08		
	0 – 26 ✕ Treat						-4.45 (2.03)	-2.19
	0 – 52 ✕ Treat						2.07 (2.03)	1.02

Note. Diag = diagnosis group; Treat = treatment type.

* $p < .05$; ** $p < .01$; *** $p < .001$.

Table 39

Three Linear Mixed Models Modelling Niacin Intake with Random Intercepts and Fixed Effects (Time, Group, and Time × Group Interactions).

		Random effects	Fixed effects					
		Intercept	Time		Group		Interaction	
Model		*SD*	Estimate (*SE*)	*t*	Estimate (*SE*)	*t*	Estimate (*SE*)	*t*
1	Intercept	8.18	28.08 (1.35)	20.75***				
	0 – 26		-19.6 (1.48)	-1.32				
	0 – 52		-1.51 (1.49)	-1.02				
	26 – 52		0.45 (1.54)	1.54				
2	Intercept	8.02	29.55 (1.92)	15.40***				
	BN – BED				-2.83 (2.69)	-1.06		
	0 – 26 × Diag						1.69 (2.96)	0.57
	0 – 52 × Diag						3.15 (2.97)	1.06
3	Intercept	8.16	28.33 (1.60)	17.76***				
	CBT – CBT-A				-0.88 (3.05)	-0.29		
	0 – 26 × Treat						1.87 (3.34)	0.56
	0 – 52 × Treat						-1.76 (3.32)	-0.53

Note. Diag = diagnosis group; Treat = treatment type.

* $p < .05$; ** $p < .01$; *** $p < .001$.

Table 40

Three Linear Mixed Models Modelling Vitamin C Intake with Random Intercepts and Fixed Effects (Time, Group, and Time ✕ Group Interactions).

		Random effects	Fixed effects					
		Intercept	Time		Group		Interaction	
Model		*SD*	Estimate (*SE*)	*t*	Estimate (*SE*)	*t*	Estimate (*SE*)	*t*
1	Intercept	32.60	121.07 (6.64)	18.23***				
	0 – 26		3.71 (8.24)	0.45				
	0 – 52		-8.14 (8.28)	-0.98				
	26 – 52		-11.85 (8.60)	-1.38				
2	Intercept	31.99	133.56 (9.44)	14.15***				
	BN – BED				-24.61 (13.2)	-1.86		
	0 – 26 ✕ Diag						-14.96 (16.53)	-0.91
	0 – 52 ✕ Diag						-19.36 (16.58)	-1.17
3	Intercept	32.85	127.18 (7.80)	16.31***				
	CBT – CBT-A				-22.51 (14.9)	-1.51		
	0 – 26 ✕ Treat						-33.10 (18.40)	-1.80
	0 – 52 ✕ Treat						-20.71 (18.35)	-1.13

Note. Diag = diagnosis group; Treat = treatment type.

* $p < .05$; ** $p < .01$; *** $p < .001$.

Table 41

Three Linear Mixed Models Modelling Vitamin E Intake with Random Intercepts and Fixed Effects (Time, Group, and Time ✕ Group Interactions).

		Random effects	Fixed effects					
		Intercept	Time		Group		Interaction	
Model		*SD*	Estimate (*SE*)	*t*	Estimate (*SE*)	*t*	Estimate (*SE*)	*t*
1	Intercept	3.50	14.53 (0.79)	18.40***				
	0 – 26		-0.92 (1.02)	-0.90				
	0 – 52		-1.89 (1.02)	-1.85				
	26 – 52		-0.97 (1.06)	-0.92				
2	Intercept	3.48	15.03 (1.13)	13.29***				
	BN – BED				-0.98 (1.59)	-0.62		
	0 – 26 ✕ Diag						0.19 (2.05)	0.09
	0 – 52 ✕ Diag						-1.18 (2.06)	-0.58
3	Intercept	3.50	14.63 (0.93)	15.69***				
	CBT – CBT-A				-0.36 (1.79)	-0.20		
	0 – 26 ✕ Treat						0.16 (2.30)	0.07
	0 – 52 ✕ Treat						0.63 (2.30)	0.27

Note. Diag = diagnosis group; Treat = treatment type.

* $p < .05$; ** $p < .01$; *** $p < .001$.

Table 42

Three Linear Mixed Models Modelling Vitamin B6 Intake with Random Intercepts and Fixed Effects (Time, Group, and Time × Group Interactions).

		Random effects	Fixed effects					
		Intercept	Time		Group		Interaction	
Model		*SD*	Estimate (*SE*)	*t*	Estimate (*SE*)	*t*	Estimate (*SE*)	*t*
1	Intercept	2.52	2.70 (0.69)	3.93***				
	0 – 26		1.45 (0.93)	1.57				
	0 – 52		-0.62 (0.93)	-0.67				
	26 – 52		-2.08 (0.97)	-2.15				
2	Intercept	2.53	3.22 (0.98)	3.28**				
	BN – BED				-1.03 (1.38)	-0.75		
	0 – 26 × Diag						-1.65 (1.86)	-0.89
	0 – 52 × Diag						-0.77 (1.87)	-0.41
3	Intercept	2.33	2.12 (0.78)	2.72**				
	CBT – CBT-A				2.08 (1.49)	1.40		
	0 – 26 × Treat						-4.03 (2.03)	-1.99
	0 – 52 × Treat						1.92 (2.03)	0.95

Note. Diag = diagnosis group; Treat = treatment type.

* $p < .05$; ** $p < .01$; *** $p < .001$.

Table 43

Three Linear Mixed Models Modelling Vitamin B12 Intake with Random Intercepts and Fixed Effects (Time, Group, and Time × Group Interactions).

		Random effects	Fixed effects					
		Intercept	Time		Group		Interaction	
Model		*SD*	Estimate (*SE*)	*t*	Estimate (*SE*)	*t*	Estimate (*SE*)	*t*
1	Intercept	1.85	4.70 (0.48)	9.87***				
	0 – 26		0.72 (0.64)	1.14				
	0 – 52		-0.63 (0.64)	-0.98				
	26 – 52		-1.35 (0.66)	-2.03				
2	Intercept	1.85	5.25 (0.68)	7.73***				
	BN – BED				-1.10 (0.96)	-1.15		
	0 – 26 × Diag						-1.28 (1.28)	-1.00
	0 – 52 × Diag						-0.65 (1.28)	-0.51
3	Intercept	1.80	4.58 (0.55)	8.34***				
	CBT – CBT-A				0.40 (1.05)	0.39		
	0 – 26 × Treat						-3.08 (1.40)	-2.20
	0 – 52 × Treat						0.35 (1.40)	0.25

Note. Diag = diagnosis group; Treat = treatment type.

* $p < .05$; ** $p < .01$; *** $p < .001$.

Table 44

Three Linear Mixed Models Modelling Folate Intake with Random Intercepts and Fixed Effects (Time, Group, and Time ✕ Group Interactions).

		Random effects	Fixed effects					
		Intercept	Time		Group		Interaction	
Model		*SD*	Estimate (*SE*)	*t*	Estimate (*SE*)	*t*	Estimate (*SE*)	*t*
1	Intercept	202.91	590.40 (31.67)	18.64***				
	0 – 26		-48.37 (32.7)	-1.48				
	0 – 52		-2.92 (32.9)	-0.09				
	26 – 52		45.45 (34.1)	0.38				
2	Intercept	198.45	660.69 (44.87)	14.73***				
	BN – BED				-137.96 (62.8)	-2.20		
	0 – 26 ✕ Diag						-90.34 (65.49)	-1.38
	0 – 52 ✕ Diag						-60.94 (65.69)	-0.93
3	Intercept	202.37	603.09 (37.39)	16.13***				
	CBT – CBT-A				-45.84 (71.4)	-0.64		
	0 – 26 ✕ Treat						-5.79 (74.05)	-0.08
	0 – 52 ✕ Treat						-34.73 (73.65)	-0.47

Note. Diag = diagnosis group; Treat = treatment type.
* $p < .05$; ** $p < .01$; *** $p < .001$.

Table 45

Three Linear Mixed Models Modelling Retinol Intake with Random Intercepts and Fixed Effects (Time, Group, and Time ✕ Group Interactions).

		Random effects	Fixed effects					
		Intercept	Time		Group		Interaction	
Model		*SD*	Estimate (*SE*)	*t*	Estimate (*SE*)	*t*	Estimate (*SE*)	*t*
1	Intercept	64.01	518.90 (25.89)	20.04***				
	0 – 26		-173.56 (36.8)	-4.71***				
	0 – 52		-181.20 (38.5)	-4.90***				
	26 – 52		-7.63 (38.5)	-0.20				
2	Intercept	61.68	562.10 (36.86)	15.25***				
	BN – BED				-85.21 (51.8)	-1.65		
	0 – 26 ✕ Diag						-66.78 (73.99)	-0.90
	0 – 52 ✕ Diag						-60.92 (74.19)	-0.82
3	Intercept	60.55	553.02 (30.18)	18.32***				
	CBT – CBT-A				-124.90 (57.8)	-2.16		
	0 – 26 ✕ Treat						-72.11 (82.33)	-0.88
	0 – 52 ✕ Treat						-134.75 (82.38)	-1.64

Note. Diag = diagnosis group; Treat = treatment type.

* $p < .05$; ** $p < .01$; *** $p < .001$.

Table 46

Three Linear Mixed Models Modelling Sodium Intake with Random Intercepts and Fixed Effects (Time, Group, and Time ✕ Group Interactions).

		Random effects	Fixed effects					
		Intercept	Time		Group		Interaction	
Model		*SD*	Estimate (*SE*)	*t*	Estimate (*SE*)	*t*	Estimate (*SE*)	*t*
1	Intercept	866.37	3660.03 (151.40)	24.17***				
	0 – 26		-655.96 (173)	-3.80***				
	0 – 52		-497.14 (174)	-2.87*				
	26 – 52		159 (180)	0.88				
2	Intercept	754.36	4179.98 (204.66)	20.42***				
	BN – BED				-1016.9 (287)	-3.54**		
	0 – 26 ✕ Diag						-440.81 (344.11)	-1.28
	0 – 52 ✕ Diag						10.79 (345.14)	0.03
3	Intercept	856.88	3758.20 (178.12)	21.10***				
	CBT – CBT-A				-358.3 (340)	-1.05		
	0 – 26 ✕ Treat						-166.59 (390.59)	-0.43
	0 – 52 ✕ Treat						-101.59 (389.03	-0.26

Note. Diag = diagnosis group; Treat = treatment type.

* $p < .05$; ** $p < .01$; *** $p < .001$.

Table 47

Three Linear Mixed Models Modelling Potassium Intake with Random Intercepts and Fixed Effects (Time, Group, and Time × Group Interactions).

		Random effects	Fixed effects					
		Intercept	Time		Group		Interaction	
Model		*SD*	Estimate (*SE*)	*t*	Estimate (*SE*)	*t*	Estimate (*SE*)	*t*
1	Intercept	851.41	4113.25 (135.44)	30.37***				
	0 – 26		-381 (143)	-2.67*				
	0 – 52		-512 (143)	-3.57**				
	26 – 52		-131 (149)	-0.88				
2	Intercept	800.46	4463.79 (188.19)	23.72***				
	BN – BED				-684.0 (264)	-2.59		
	0 – 26 × Diag						-261.80 (285.64)	-0.92
	0 – 52 × Diag						54.80 (286.50)	0.19
3	Intercept	835.92	4219.79 (158.51)	26.62***				
	CBT – CBT-A				-383.2 (303)	-1.27		
	0 – 26 × Treat						77.22 (322.44)	0.24
	0 – 52 × Treat						-220.82 (320.82)	-0.69

Note. Diag = diagnosis group; Treat = treatment type.

* $p < .05$; ** $p < .01$; *** $p < .001$.

Table 48

Three Linear Mixed Models Modelling Magnesium Intake with Random Intercepts and Fixed Effects (Time, Group, and Time ✕ Group Interactions).

		Random effects	Fixed effects					
		Intercept	Time		Group		Interaction	
Model		*SD*	Estimate (*SE*)	*t*	Estimate (*SE*)	*t*	Estimate (*SE*)	*t*
1	Intercept	95.30	454.75 15.45	29.44***				
	0 – 26		-58.40 (16.6)	-3.53**				
	0 – 52		-56.17 (16.7)	-3.37**				
	26 – 52		2.23 (17.3)	0.13				
2	Intercept	90.50	495.63 (21.58)	22.97***				
	BN – BED				-80.04 (30.2)	-2.65		
	0 – 26 ✕ Diag						-42.65 (33.16)	-1.29
	0 – 52 ✕ Diag						-10.57 (33.27)	-0.32
3	Intercept	94.44	468.66 (18.14)	25.83***				
	CBT – CBT-A				-50.51 (34.7)	-1.46		
	0 – 26 ✕ Treat						-17.59 (37.36)	-0.47
	0 – 52 ✕ Treat						-38.03 (37.17)	-1.02

Note. Diag = diagnosis group; Treat = treatment type.

* $p < .05$; ** $p < .01$; *** $p < .001$.

Table 49

Three Linear Mixed Models Modelling Calcium Intake with Random Intercepts and Fixed Effects (Time, Group, and Time × Group Interactions).

		Random effects	Fixed effects					
		Intercept	Time		Group		Interaction	
Model		*SD*	Estimate (*SE*)	*t*	Estimate (*SE*)	*t*	Estimate (*SE*)	*t*
1	Intercept	336.38	1230.01 (51.48)	23.89***				
	0 – 26		-144.7 (52.0)	-2.78*				
	0 – 52		-159.9 (52.3)	-3.06**				
	26 – 52		-15.2 (54.2)	-0.28				
2	Intercept	329.41	1325.94 (73.16)	18.12***				
	BN – BED				-187.89 (102.4)	-1.83		
	0 – 26 × Diag						-70.50 (104.66)	-0.67
	0 – 52 × Diag						-88.46 (104.97)	-0.84
3	Intercept	337.56	1250.26 (60.87)	20.54***				
	CBT – CBT-A				-72.84 (116.2)	-0.63		
	0 – 26 × Treat						-13.00 (117.25)	-0.11
	0 – 52 × Treat						-107.03 (116.58)	-0.92

Note. Diag = diagnosis group; Treat = treatment type.

* $p < .05$; ** $p < .01$; *** $p < .001$.

Table 50

Three Linear Mixed Models Modelling Phosphorus Intake with Random Intercepts and Fixed Effects (Time, Group, and Time ✕ Group Interactions).

		Random effects	Fixed effects					
		Intercept	Time		Group		Interaction	
Model		*SD*	Estimate (*SE*)	*t*	Estimate (*SE*)	*t*	Estimate (*SE*)	*t*
1	Intercept	386.34	2003.67 (62.69)	31.96***				
	0 – 26		-265.1 (67.3)	-3.94***				
	0 – 52		-290.0 (67.7)	-4.28***				
	26 – 52		-24.8 (70.2)	-0.35				
2	Intercept	362.20	2169.53 (87.28)	24.86***				
	BN – BED				-324.25 (122.3)	-2.65		
	0 – 26 ✕ Diag						-107.09 (135.39)	-0.79
	0 – 52 ✕ Diag						-55.08 (135.79)	-0.41
3	Intercept	382.92	2045.40 (73.72)	27.75***				
	CBT – CBT-A				-150.6 (140.8)	-1.07		
	0 – 26 ✕ Treat						6.49 (152.11)	0.04
	0 – 52 ✕ Treat						-102.71 (151.37)	-0.68

Note. Diag = diagnosis group; Treat = treatment type.

* $p < .05$; ** $p < .01$; *** $p < .001$.

Table 51

Three Linear Mixed Models Modelling Iron Intake with Random Intercepts and Fixed Effects (Time, Group, and Time ✕ Group Interactions).

		Random effects	Fixed effects					
		Intercept	Time		Group		Interaction	
Model		*SD*	Estimate (*SE*)	*t*	Estimate (*SE*)	*t*	Estimate (*SE*)	*t*
1	Intercept	4.31	16.21 (0.80)	20.17***				
	0 – 26		-2.51 (0.96)	-2.63*				
	0 – 52		-1.00 (0.96)	-1.05				
	26 – 52		1.50 (1.00)	1.51				
2	Intercept	4.12	17.89 (1.13)	15.81***				
	BN – BED				-3.29 (1.59)	-2.07		
	0 – 26 ✕ Diag						-1.83 (1.92)	-0.95
	0 – 52 ✕ Diag						0.00 (1.92)	0.00
3	Intercept	4.14	16.77 (0.94)	17.88***				
	CBT – CBT-A				-2.02 (1.79)	-1.13		
	0 – 26 ✕ Treat						0.71 (2.16)	0.33
	0 – 52 ✕ Treat						0.20 (2.16)	0.09

Note. Diag = diagnosis group; Treat = treatment type.

* $p < .05$; ** $p < .01$; *** $p < .001$.

Table 52

Three Linear Mixed Models Modelling Zinc Intake with Random Intercepts and Fixed Effects (Time, Group, and Time × Group Interactions).

		Random effects	Fixed effects					
		Intercept	Time		Group		Interaction	
Model		*SD*	Estimate (*SE*)	*t*	Estimate (*SE*)	*t*	Estimate (*SE*)	*t*
1	Intercept	2.61	13.19 (0.47)	28.10***				
	0 – 26		-1.31 (0.55)	-2.41*				
	0 – 52		-2.09 (0.55)	-3.82***				
	26 – 52		-0.78 (0.57)	-1.37				
2	Intercept	2.44	14.29 (0.65)	21.83***				
	BN – BED				-2.16 (0.92)	-2.35		
	0 – 26 × Diag						-1.04 (1.09)	-0.95
	0 – 52 × Diag						0.13 (1.10)	0.11
3	Intercept	2.59	13.31 (0.55)	24.07***				
	CBT – CBT-A				-0.45 (1.06)	-0.42		
	0 – 26 × Treat						0.37 (1.23)	0.30
	0 – 52 × Treat						-0.11 (1.23)	-0.09

Note. Diag = diagnosis group; Treat = treatment type.

* $p < .05$; ** $p < .01$; *** $p < .001$.

Table 53

Three Linear Mixed Models Modelling Selenium Intake with Random Intercepts and Fixed Effects (Time, Group, and Time × Group Interactions).

		Random effects	Fixed effects					
		Intercept	Time		Group		Interaction	
Model		*SD*	Estimate (*SE*)	*t*	Estimate (*SE*)	*t*	Estimate (*SE*)	*t*
1	Intercept	30.16	89.65 (5.05)	17.75***				
	0 – 26		1.62 (5.58)	0.29				
	0 – 52		4.58 (5.61)	0.82				
	26 – 52		2.97 (5.82)	0.51				
2	Intercept	30.15	95.60 (7.23)	13.21***				
	BN – BED				-11.73 (10.14)	-1.16		
	0 – 26 × Diag						-14.16 (11.18)	-1.27
	0 – 52 × Diag						-3.21 (11.21)	-0.29
3	Intercept	30.14	91.42 (5.96)	15.35***				
	CBT – CBT-A				-6.58 (11.38)	-0.58		
	0 – 26 × Treat						-14.32 (12.57)	-1.14
	0 – 52 × Treat						-11.17 (12.52)	-0.89

Note. Diag = diagnosis group; Treat = treatment type.

* $p < .05$; ** $p < .01$; *** $p < .001$.

Table 54

Three Linear Mixed Models Modelling Iodine Intake with Random Intercepts and Fixed Effects (Time, Group, and Time ✕ Group Interactions).

		Random effects	Fixed effects					
		Intercept	Time		Group		Interaction	
Model		*SD*	Estimate (*SE*)	*t*	Estimate (*SE*)	*t*	Estimate (*SE*)	*t*
1	Intercept	41.49	129.98 (8.56)	22.53***				
	0 – 26		-24.1 (10.7)	-2.25				
	0 – 52		-43.3 (10.7)	-4.03***				
	26 – 52		-19.2 (11.2)	-1.73				
2	Intercept	38.68	219.46 (11.94)	18.38***				
	BN – BED				-52.04 (16.8)	-3.12*		
	0 – 26 ✕ Diag						-30.79 (21.28)	-1.45
	0 – 52 ✕ Diag						-29.50 (21.35)	-1.38
3	Intercept	41.48	201.86 (10.05)	20.08***				
	CBT – CBT-A				-32.57 (19.2)	-1.69		
	0 – 26 ✕ Treat						-29.16 (23.95)	-1.22
	0 – 52 ✕ Treat						-30.97 (23.89)	-1.30

Note. Diag = diagnosis group; Treat = treatment type.

* $p < .05$; ** $p < .01$; *** $p < .001$.

Table 55

Three Linear Mixed Models Modelling Total Adequacy with Random Intercepts and Fixed Effects (Time, Group, and Time ✕ Group Interactions).

		Random effects	Fixed effects					
		Intercept	Time		Group		Interaction	
Model		*SD*	Estimate (*SE*)	*t*	Estimate (*SE*)	*t*	Estimate (*SE*)	*t*
1	Intercept	1.35	20.30 (0.30)	67.48***				
	0 – 26		-0.18 (0.39)	-0.47				
	0 – 52		-0.54 (0.39)	-1.40				
	26 – 52		-0.36 (0.40)	-0.89				
2	Intercept	1.34	20.55 (0.43)	47.76***				
	BN – BED				-0.50 (0.60)	-0.83		
	0 – 26 ✕ Diag						-0.41 (0.78)	-0.52
	0 – 52 ✕ Diag						0.08 (0.78)	0.11
3	Intercept	1.38	20.58 (0.35)	58.35***				
	CBT – CBT-A				-1.02 (0.68)	-1.52		
	0 – 26 ✕ Treat						-1.32 (0.86)	-1.54
	0 – 52 ✕ Treat						-1.77 (0.85)	-2.07

Note. Diag = diagnosis group; Treat = treatment type.

* $p < .05$; ** $p < .01$; *** $p < .001$.

Table 56

Three Linear Mixed Models Modelling Macronutrient Adequacy with Random Intercepts and Fixed Effects (Time, Group, and Time ✕ Group Interactions).

		Random effects	Fixed effects					
		Intercept	Time		Group		Interaction	
Model		*SD*	Estimate (*SE*)	*t*	Estimate (*SE*)	*t*	Estimate (*SE*)	*t*
1	Intercept	0.56	6.03 (0.14)	42.16***				
	0 – 26		0.73 (0.19)	3.86***				
	0 – 52		0.44 (0.19)	2.32*				
	26 – 52		-0.29 (0.20)	-1.46				
2	Intercept	0.56	6.05 (0.20)	29.59***				
	BN – BED				-0.05 (0.29)	-0.18		
	0 – 26 ✕ Diag						0.17 (0.38)	0.44
	0 – 52 ✕ Diag						0.19 (0.38)	0.48
3	Intercept	0.57	6.13 (0.17)	36.54***				
	CBT – CBT-A				-0.38 (0.32)	-1.17		
	0 – 26 ✕ Treat						-0.64 (0.42)	-1.51
	0 – 52 ✕ Treat						-0.69 (0.42)	-1.62

Note. Diag = diagnosis group; Treat = treatment type.

* $p < .05$; ** $p < .01$; *** $p < .001$.

Table 57

Three Linear Mixed Models Modelling Micronutrient adequacy with Random Intercepts and Fixed Effects (Time, Group, and Time ✕ Group Interactions).

		Random effects	Fixed effects					
		Intercept	Time		Group		Interaction	
Model		*SD*	Estimate (*SE*)	*t*	Estimate (*SE*)	*t*	Estimate (*SE*)	*t*
1	Intercept	1.36	14.27 (0.27)	52.46***				
	0 – 26		-0.92 (0.34)	-2.75*				
	0 – 52		-0.99 (0.34)	-2.93*				
	26 – 52		-0.07 (0.35)	-0.19				
2	Intercept	1.36	14.50 (0.39)	37.19***				
	BN – BED				-0.45 (0.55)	-0.83		
	0 – 26 ✕ Diag						-0.60 (0.67)	-0.89
	0 – 52 ✕ Diag						-0.13 (0.68)	-0.19
3	Intercept	1.37	14.45 (0.32)	45.08***				
	CBT – CBT-A				-0.64 (0.61)	-1.05		
	0 – 26 ✕ Treat						-0.67 (0.75)	-0.89
	0 – 52 ✕ Treat						-1.10 (0.75)	-1.47

Note. Diag = diagnosis group; Treat = treatment type.

* $p < .05$; ** $p < .01$; *** $p < .001$.

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Appendix A

Seven-Day Food Monitoring Log (Instructions and Log)

BEP 7-DAY MONITORING

RECORD SHEET

PLEASE READ THESE IMPORTANT INSTRUCTIONS CAREFULLY

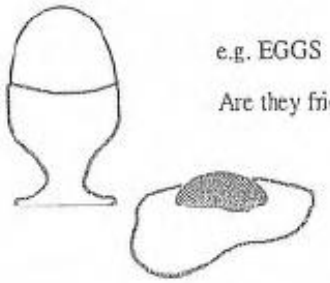
- Please record ALL food and drinks consumed
- Please record the food at the time of eating and NOT from memory at the end of the day
- You should include all meals and snacks, plus sweets, drinks (including water) etc
- Remember to include any additions to food already recorded such as: sauces, dressings, or extras e.g. gravy, salad dressings, stuffings, sugar, honey, syrups etc., butter or margarine (e.g. added to bread, crackers, vegetables)
- Draw a line across the page after each episode of eating/drinking or after a binge.
- Please start a new day on a new page.

ID: ☐☐☐☐☐

DATE: ____/____/____

DESCRIBING FOOD AND DRINK – GUIDELINES

1. Please give details of the method of cooking all foods (e.g. fried, grilled, boiled, roasted, steamed, poached, stewed
2. Give as many details as possible about the **type** of food that you eat e.g. brand name of food where applicable (e.g. Miracle margarine);
   Type of: Breakfast cereal (e.g. Weetbix)
   Milk (e.g. whole milk or 'trim' milk
   Cake or biscuit (e.g. fruit cake or wheatmeal biscuit)
   Fruit (e.g. fresh, canned, dried, stewed)
   Soft drink (e.g. regular or low calorie)
3. Name the type of cheese, fish or meat (e.g. cheddar, cod fillet, loin of pork)



e.g. EGGS

Are they fried, boiled, poached or scrambled?

RECORDING THE AMOUNTS OF FOODS YOU EAT

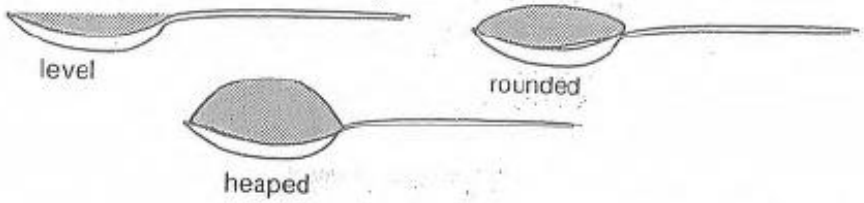
It is also very important to record the quantity of each food and drink you consume.

Here are some suggestions on how to record amounts:

- IN HOUSEHOLD MEASUREMENTS

For many foods such as vegetables, cereals and canned or stewed fruit, a household measurement is adequate.

e.g. STATE THE NUMBER OF TEASPOONS (t), TABLESPOONS (T), CUPS etc. State whether spoons are level, rounded or heaped.



Butter and margarine can be measured in teaspoons or tablespoons if you find this an easy method

- WEIGHTS MARKED ON PACKAGES

All packaged foods have their weight marked on the packaging and this can be quoted e.g. half a 425g can of baked beans.

- BREAD – indicate the size of the slices (e.g. sandwich, medium, toast).

- CHEESE, MEAT AND FISH

If at all possible, please **use the pictures on the attached sheets** to indicate what sort of portion sizes you eat e.g. you might have 1 portions of spaghetti size A, 1 portion of meat size B or 2 slices of cheese size C.

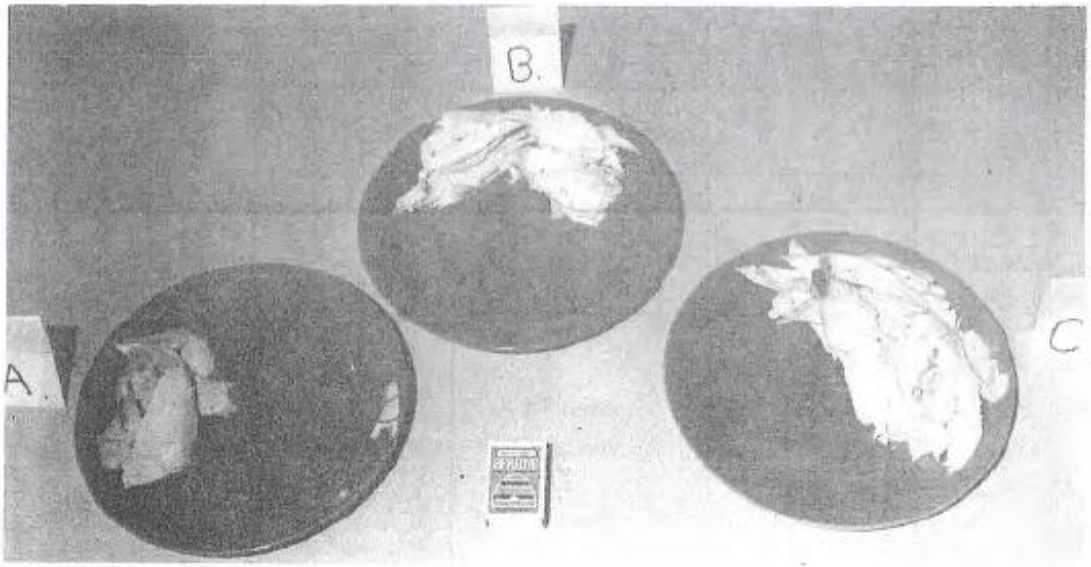
- USE COMPARISONS for describing portion sizes whether this is easier e.g. potato – size of a hen's egg, cheese – size of a matchbox.

IT IS VERY IMPORTANT THAT YOU DO NOT ADJUST WHAT YOU EAT AND DRINK BECAUSE YOU ARE KEEPING A RECORD. THIS IS VERY EASY TO DO, BUT REMBEMBER, WE ARE INTEREDSTED IN YOUR USUAL EATING HABITS.

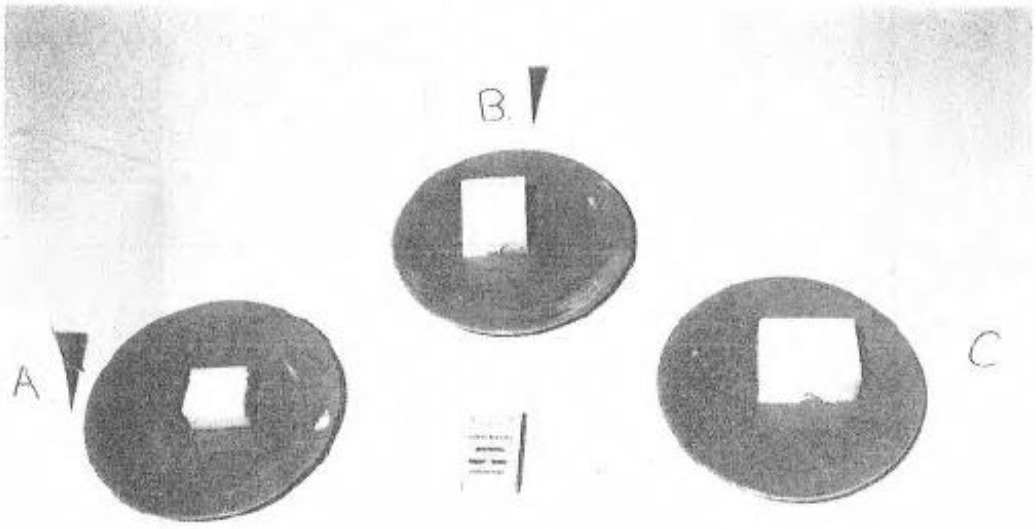
PHOTOGRAPHS FOR ESTIMATING THE SIZE OF THE PORTION OF FOOD THAT YOU EAT.

ALL PHOTOGRAPHS SHOW FOOD ON 22 cm DIAMETER PLATES, UNLESS OTHERWISE STATED.

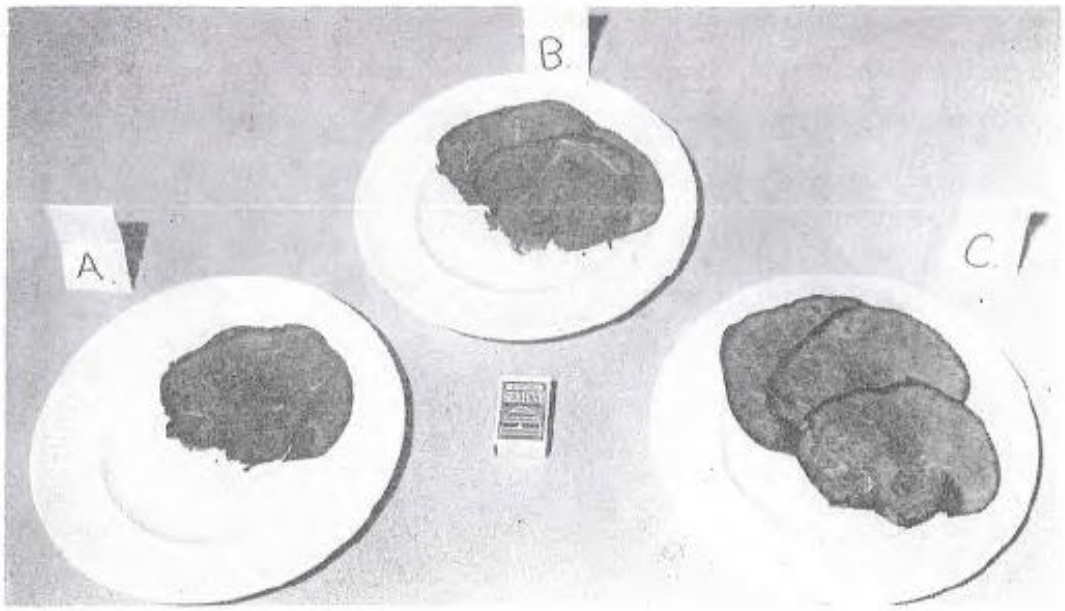
PHOTOGRAPH 1 CHICKEN



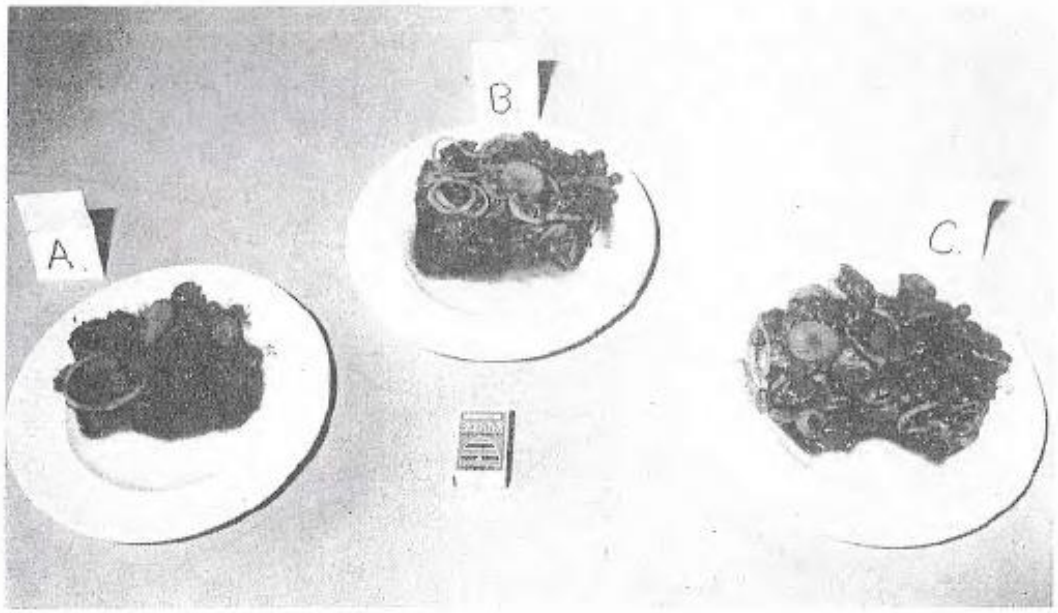
PHOTOGRAPH 2 CHEESE 18 cm diameter plates



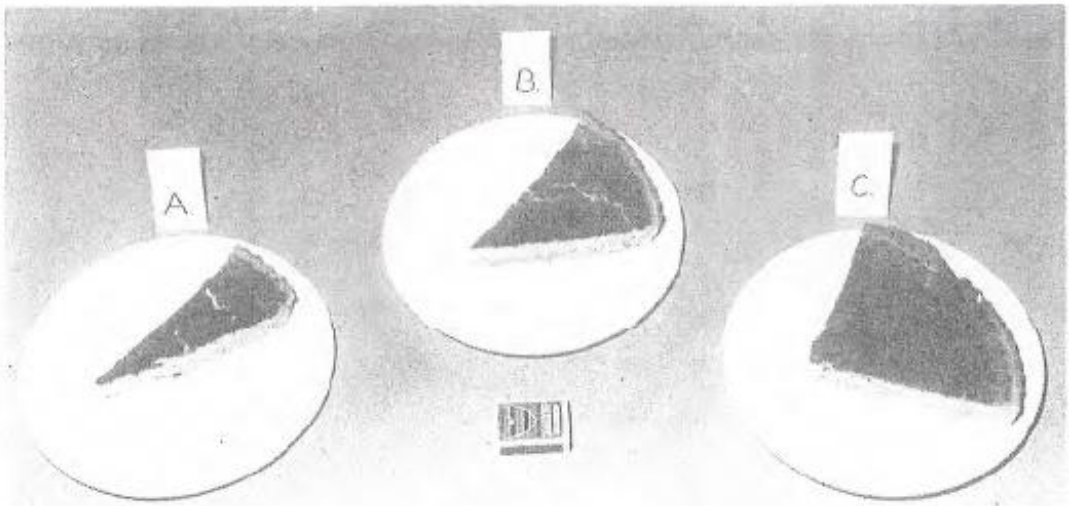
PHOTOGRAPH 3 ROAST MEAT 25 cm diameter plates



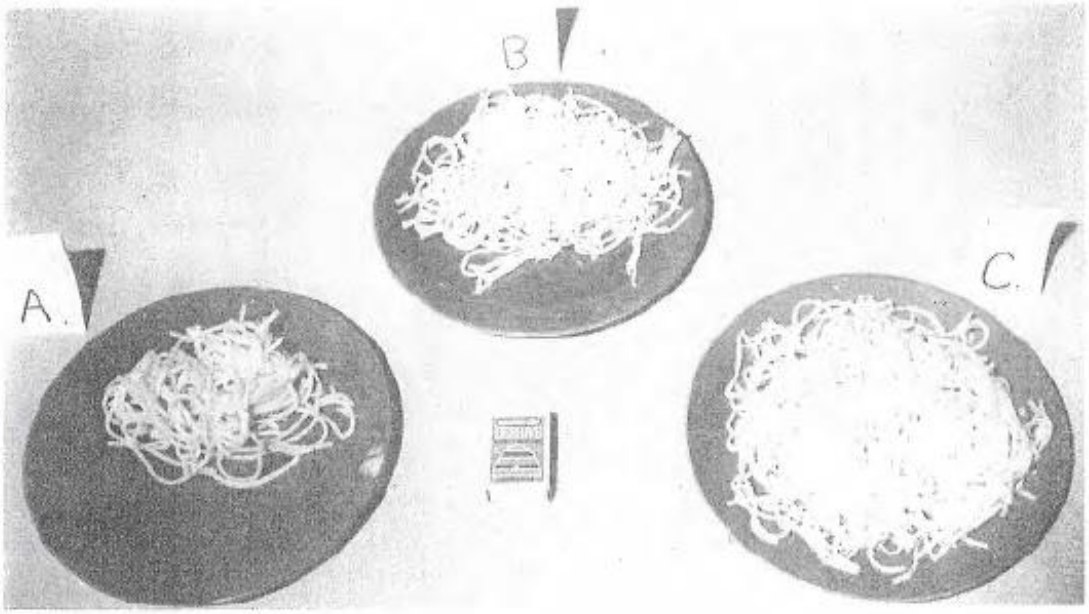
PHOTOGRAPH 4 VEGETABLE OR MEAT STEW



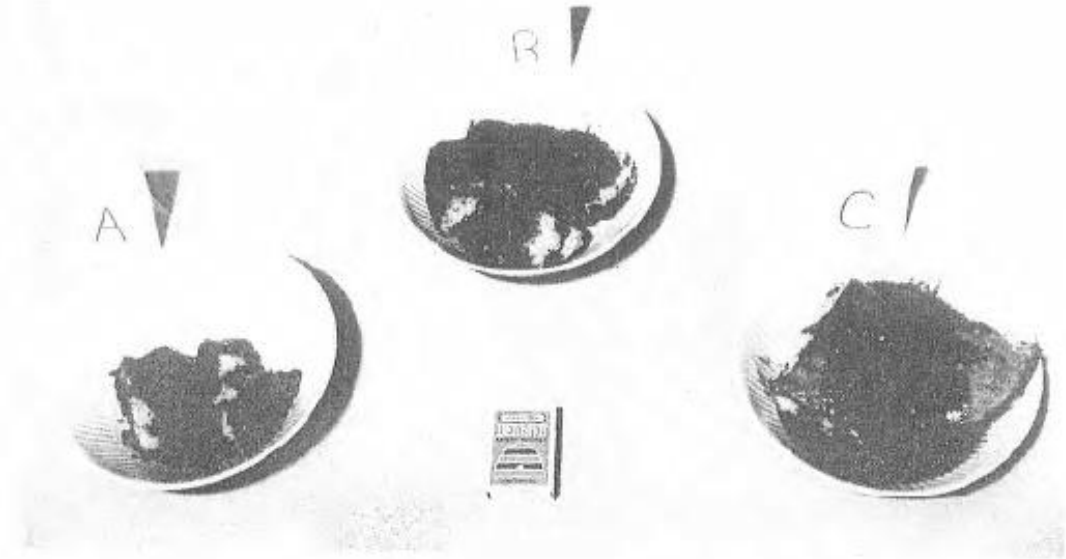
PHOTOGRAPH 5 VEGETABLE PIE



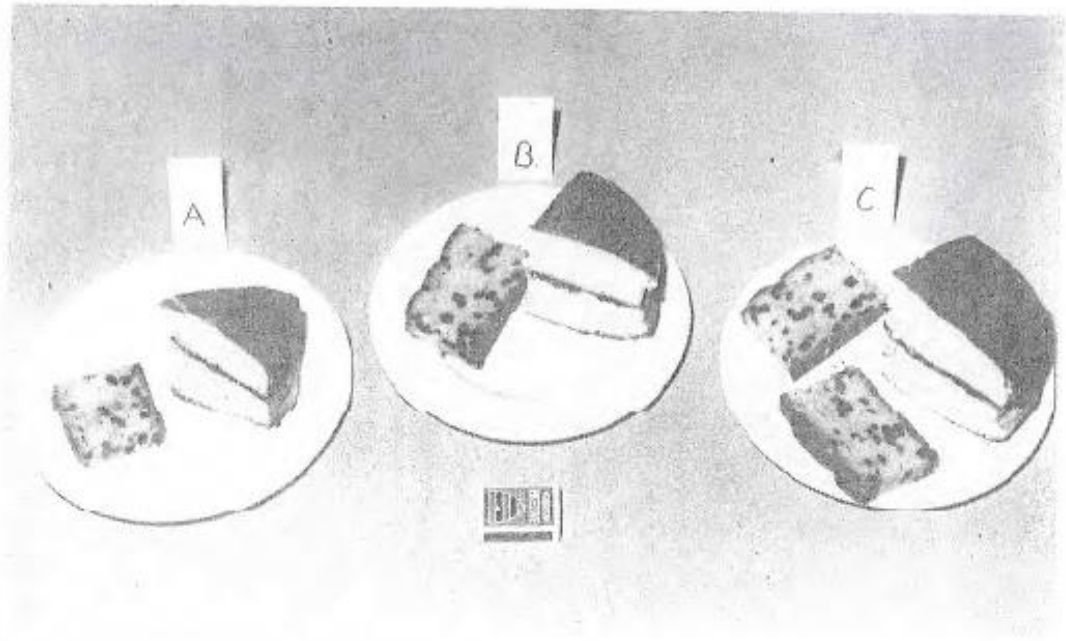
PHOTOGRAPH 6 SPAGHETTI



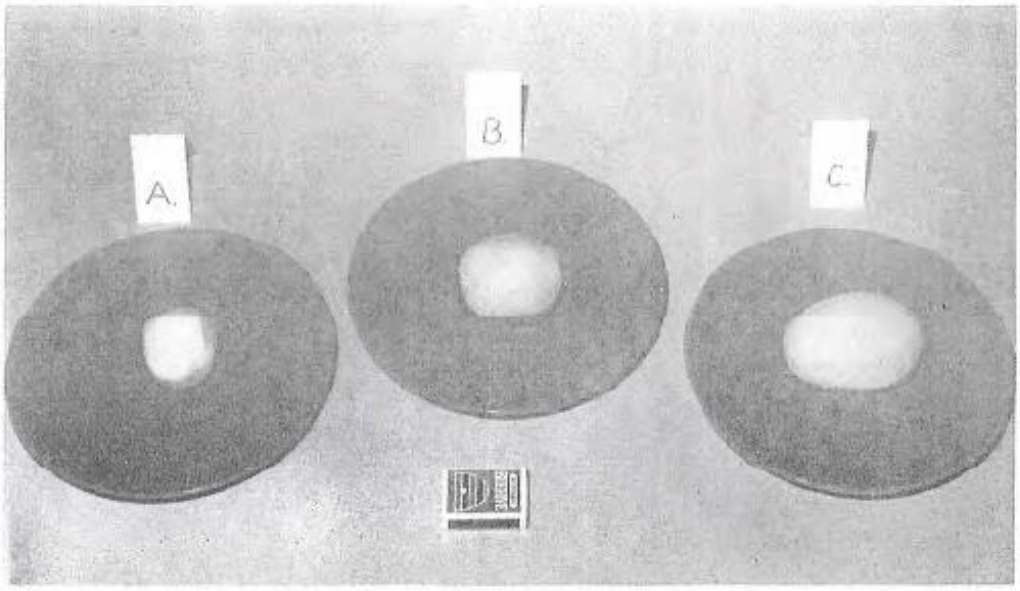
PHOTOGRAPH 7 PUDDING



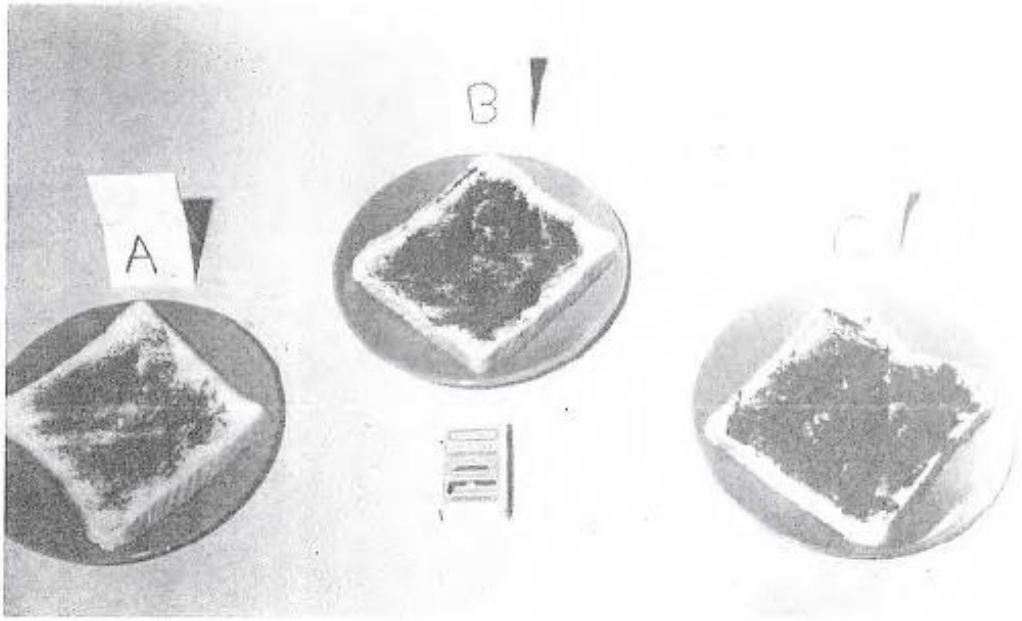
PHOTOGRAPH 8 CAKE



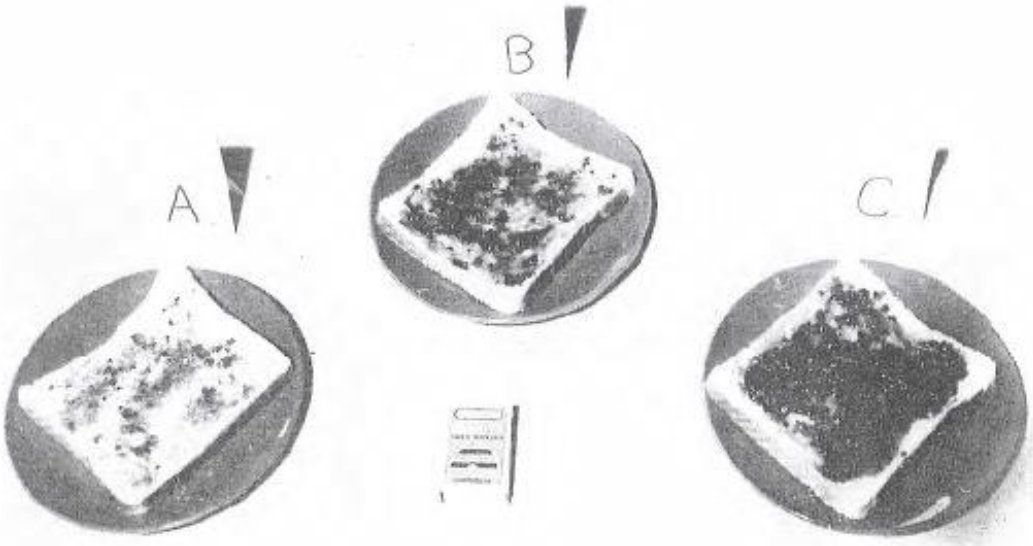
PHOTOGRAPH 9 POTATO



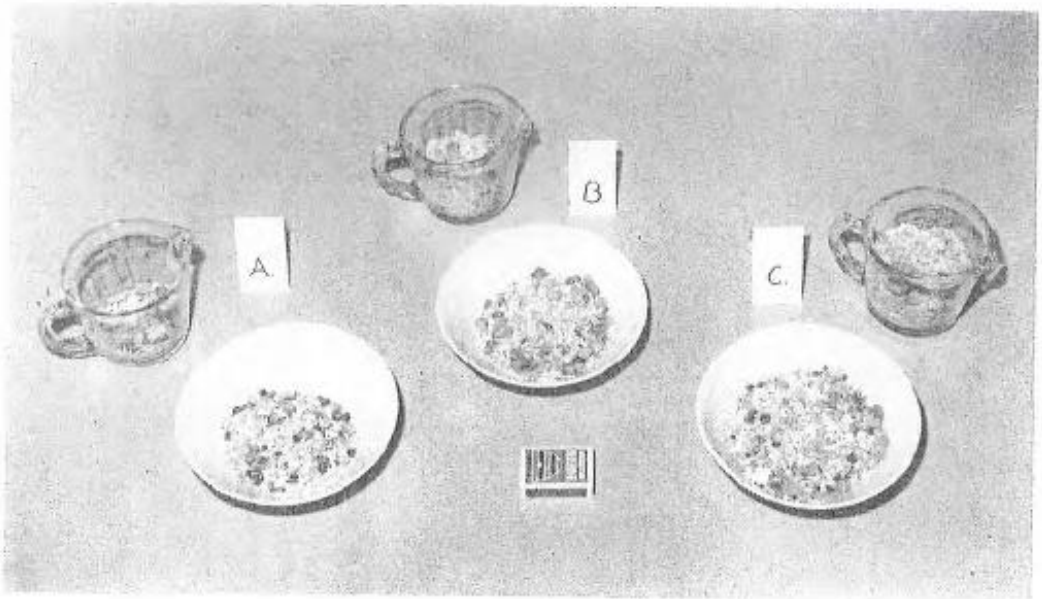
PHOTOGRAPH 10 MARMITE OR VEGEMITE



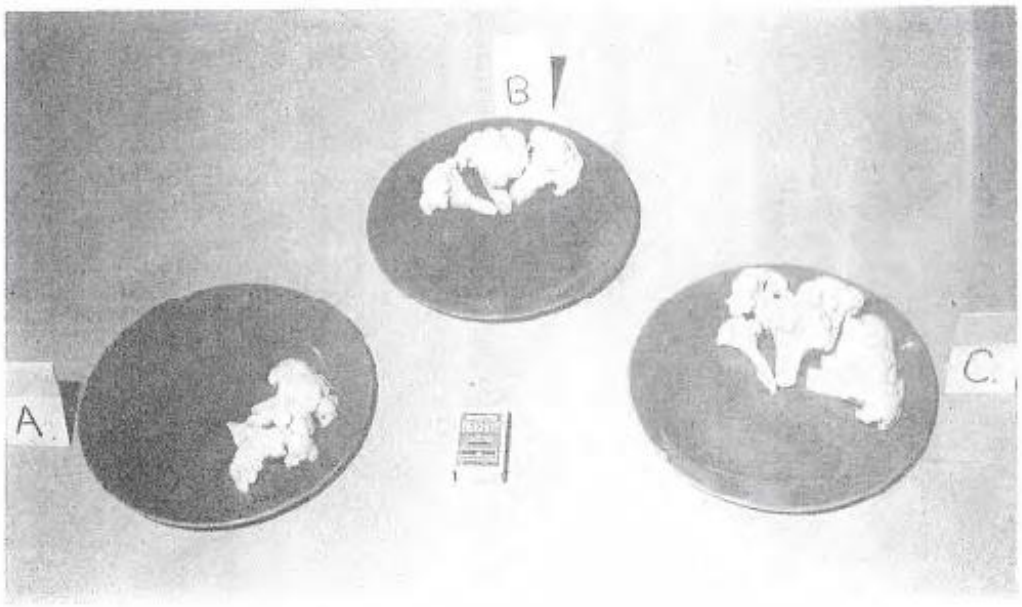
PHOTOGRAPH 11 JAM



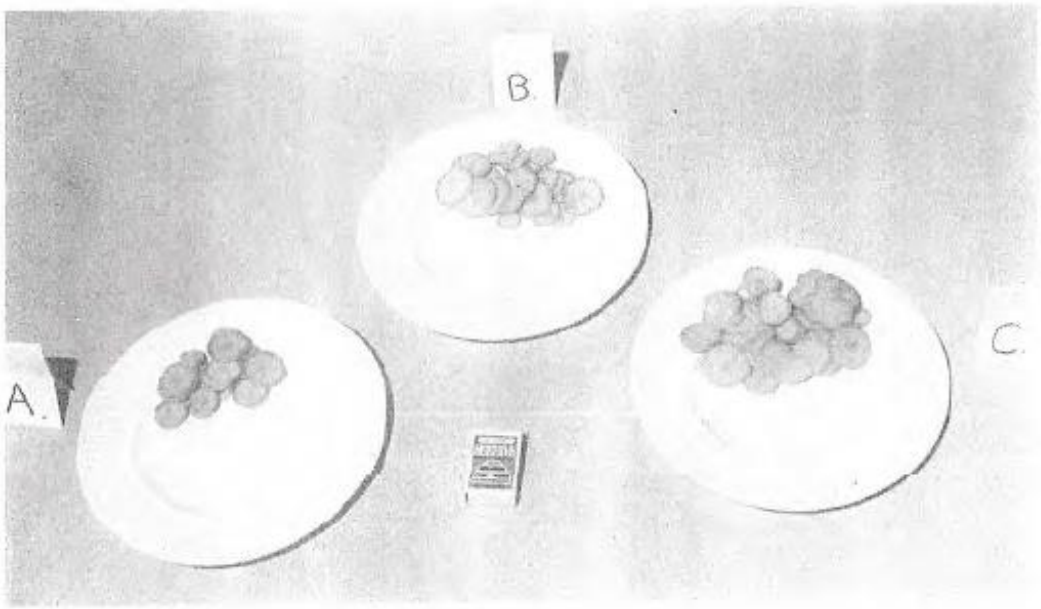
PHOTOGRAPH 12 MUESLI (1/4, 1/2 & 3/4 cup)



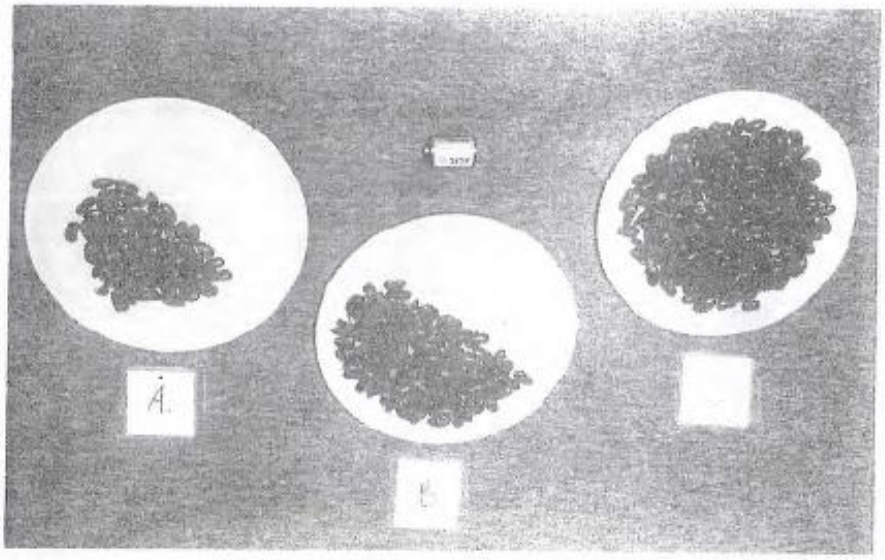
PHOTOGRAPH 13 CAULIFLOWER



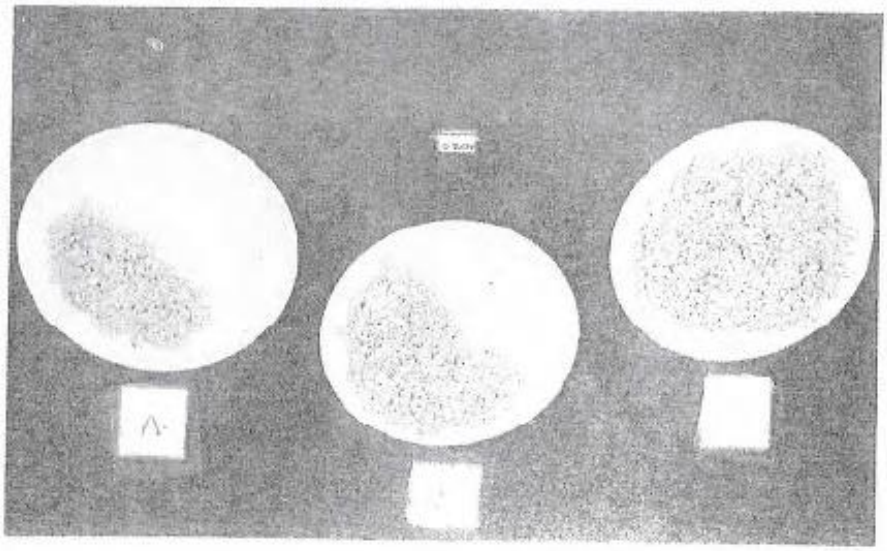
PHOTOGRAPH 14 CARROTS



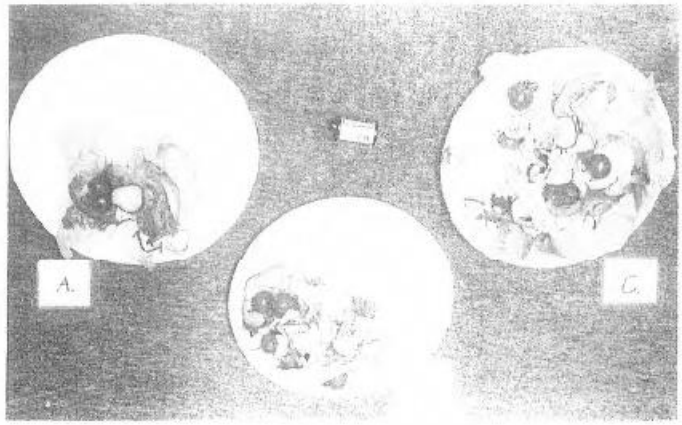
PHOTOGRAPH 15 BEANS



PHOTOGRAPH 16 RICE



PHOTOGRAPH 17 SALAD



PLEASE NOTE: The following record sheets are examples only and may NOT be similar to your own daily intake.

Day Saturday Date 23rd April 2005

- Record ALL food and drink consumed during the day including meals, sweets, snacks, binges, sauces and dressings.
- Please record: METHOD OF COOKING (e.g. *boiled* pasta)
  TYPE OF FOOD (e.g. boiled *wholegrain* pasta)
  QUANTITY OF FOOD (e.g. 6 *heaped T* boiled wholegrain pasta)

EXAMPLE 1:

TIME	QUANTITY EATEN	DETAILS OF FOOD AND DRINK	BINGE/VOMIT
8.30am	1 C 2 T	Coffee, Greggs instant milk, Anchor, Trim	
9.30am	1 C	Cappuccino	
10.30am	3 2 x 10cm 2 3 2 2 t 1 C 1 C	Bacon rashers, fried Chippolata sausages, fried eggs, fried, soft Tomato halves Toast, wholemeal Butter Orange juice, freshly squeezed Cappuccino	
12pm	2 2 250ml (cans) 2 40g	Irvines steak and cheese pies Diet Coke BBQ Kettle Fries Chips	B
3pm	1 C 1 T 2	Tea, Bell Trim milk, Anchor Girl guide biscuits	

Continue over the page or start a new page for each new day

Day Saturday Date 23rd April 2005

TIME	QUANTITY EATEN	DETAILS OF FOOD AND DRINK	BINGE/VOMIT
5pm	2 handles	Tap Canterbury Draught Beer	
	1/2 C	Hot chips	
5.45pm	6	Pascall's wine gums	
7pm	1 40g	Bluebird, ready salted crisps	
	1/2 2L tub	Tip Top Boysenberry ice cream	
	6	Toffee Pop biscuits	B
	1/2 500g block	Chocolate, Cadbury's Energy	
	2 C	Cereal, Coco Pops	
	Size C portion	Pizza, (leftover), (cheese, salami, tomato, red peppers)	
	1/2 packet	Girl guide biscuits	
	1 C	Cashew nuts, salted, roasted	
8.30pm			V
8.40pm	1 C	Cold water	
	1 C	Tea, Bell	
	1T	Trim Milk, Anchor	
	2	girl guide biscuits	

Continue over the page or start a new page for each new day

PLEASE NOTE: The following record sheets are examples only and may NOT be similar to your own daily intake.

Day Sunday Date 24th April 2005

- Record ALL food and drink consumed during the day including meals, sweets, snacks, binges, sauces and dressings.
- Please record: METHOD OF COOKING (e.g. *boiled* pasta)
  TYPE OF FOOD (e.g. boiled *wholegrain* pasta)
  QUANTITY OF FOOD (e.g. 6 *heaped T* boiled wholegrain pasta)

EXAMPLE 2

TIME	QUANTITY EATEN	DETAILS OF FOOD AND DRINK	BINGE/VOMIT
8am	1 C	Black Coffee, Greggs instant	
9am	1C	Black Coffee, Greggs instant	
10am	1	Ryvita cracker, cracked pepper	
10.30am	1C	Black coffee, instant	
12pm	1 150g pottle 1 7cm	Fresh n' Fruity yoghurt, Blueberry and vanilla, lite Red Delicious, apple	
1pm	1C	Black coffee, instant, Greggs	
3pm	1C	Black coffee, Greggs instant	
7pm	3 2	Big Mac Combo's (3 Big mac's, 3 large fries, 3 large drinks - Coke) Chocolate sundaes - McDonalds	B

Continue over the page or start a new page for each new day

END OF EXAMPLES

Day..... Date.....

TIME	QUANTITY EATEN	DETAILS OF FOOD AND DRINK	BINGE/VOMIT

Continue over the page or start a new page for each new day

Appendix B

Letter of Ethical Approval of the Original study from the Canterbury Ethics Committee

Canterbury
Ethics Committees
4th Floor, 250 Oxford Terrace
P.O. Box 3877
Christchurch
Fax (03) 372 1015

10 September 2004

Professor Peter Joyce
Psychological Medicine
University of Otago
PO Box 4345
Christchurch

Dear Professor Joyce

Enhancing Psychotherapy for Bulimia Nervosa and Binge Eating Disorder
Investigators: Prof P Joyce, Dr V McIntosh, J Jordan, Dr J Carter, Dr J McKenzie, Dr J Latner, Dr F Carter, Assoc Prof C Frampton
Ethics Ref: CTB/04/08/139

Thank you for your letter dated 6 September 2004 in response to the Committee's suggestions. The above study has now been given final ethical approval by the Canterbury Ethics Committee.

Approved Documents
Enhancing Psychotherapy for Bulimia Nervosa and Binge Eating Disorder - Study Instruments Part I of II (P.I. Peter Joyce)
Enhancing Psychotherapy for Bulimia Nervosa and Binge Eating Disorder - Study Instruments Part II of II (P.I. Peter Joyce)
Information sheet and consent form version dated 3 September 2004

Certification
The Committee is satisfied that this study is not being conducted principally for the benefit of the manufacturer or distributor of the medicine or item in respect of which the trial is being carried out.

Accreditation
This Committee is accredited by the Health Research Council and is constituted and operates in accordance with the Operational Standard for Ethics Committees, March 2002.

Progress Reports
The study is approved until 30 June 2009. The Committee will review the approved application annually. A progress report is required for this study in June each year. You will be sent a form requesting this information prior to the review date. Please note that failure to complete and return this form may result in the withdrawal of ethical approval. A final report is also required at the conclusion of the study.

Canterbury
Ethics Committees
4th Floor, 250 Oxford Terrace
P.O. Box 3877
Christchurch
Fax (03) 372 1015

Amendments
All amendments to the study must be advised to the Committee prior to their implementation, except in the case where immediate implementation is required for reasons of safety. In such cases the Committee must be notified as soon as possible of the change.

It is also a condition of approval that the Committee is advised of any adverse events, if the study does not commence, or the study is altered in any way, including all documentation eg advertisements, letters to prospective participants. Please quote the above ethics committee reference number in all correspondence.

General
It should be noted that Ethics Committee approval does not imply any resource commitment or administrative facilitation by any healthcare provider within whose facility the research is to be carried out. Where applicable, authority for this must be obtained separately from the appropriate manager within the organisation.

We wish you well with your study.

Yours sincerely

Joanne Hamlyn
Ethics Committee B Administrator
joanne_hamlyn@moh.govt.nz
tel: 03 372 3037

Appendix C

Letter of Approval for the Original Study from Iwi Māori

UNIVERSITY OF OTAGO

Te Whare Wānanga o Otāgo

22 September 2003

Professor P Joyce
Department of Psychological Medicine
Christchurch School of Medicine and Health Sciences
Terrace House

Tena koe, Peter

I met with Jennifer Jordan, Dr Janet Carter and Gini McIntosh at the CSMHS on Friday, 12 September, to discuss your ongoing project entitled *Psychotherapy for eating disorders: Cognitive behaviour therapy, schema focused therapy, and nutrition and appetite enhanced CBT.* I also appreciated receiving your brief summary.

I understand that you received correspondence from Christine Rimene last year (in her capacity as Interim Facilitator, Ngai Tahu Maori Health Research Unit) regarding "the responsiveness to Maori" question. Her comments would still be relevant today. The issues that were raised are the following.

Ethnicity
There is a need to acknowledge the issues pertaining to ethnicity. It was agreed that there would be Maori participants and that there would be a need to consider how ethnicity data is to be collected in your study. Given the poor ethnicity data collection on the hospital databases this information should be collected in demographic information as part of the research. Through our discussions, the Census 2001 ethnicity question would be the preferred tool in recording ethnicity.

Relevance to Maori
From our discussions regarding the research it was recognised that there is information available in Maori health in relation to nutrition, obesity and nutritional education. It was apparent from your summary that you have identified that this research will have impact on Maori health and that is important. Christine Rimene's letter would have also suggested the value of supporting references to assist in the understanding of health issues. There are a number of references available, for example: *Hauora Maori Standards of Health A Study of the Years 1970-1991*, by the Eru Pomare Maori Health Research Unit, Wellington. A special reference in regard to nutrition and Maori is made on page 54.

Ongoing Consultation
It was heartening to know that you have consulted with Maori researchers such as Paul Robertson and Suzanne Pitama who are assisting in recruiting Maori participants for the research project. Cecileah Win, a Maori dietitian working for Pegasus Health, would be an ideal person to consult with as well.

Christchurch School of Medicine and Health Sciences
Research Office, Department of the Dean

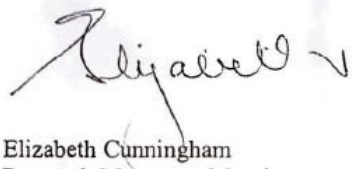
PO Box 4345, Christchurch, New Zealand.
Tel 64 3 364 0237 • Fax 64 3 364 0525 • Email research@chmeds.ac.nz
www.otago.ac.nz
DUNEDIN • CHRISTCHURCH • WELLINGTON • AUCKLAND

Dissemination
Your proposal mentioned that the number of Maori represented in obesity figures is high. Findings from this project may contribute to the development of future hypotheses or projects. It is therefore important that appropriate organisations such as the Maori Women's Welfare League, Maori health professionals and Maori researchers are aware of your findings.

The Research Office of the CSMHS and in particular myself, Research Manager Maori, would be willing to assist with the dissemination once your project has reached a successful conclusion.

I wish you well

Kia manawanui.



Elizabeth Cunningham
Research Manager - Maori

Appendix D

Letter of Approval for the Current study from the University of Canterbury Ethics Committee

HUMAN ETHICS COMMITTEE

Secretary, Rebecca Robinson
Telephone: +64 03 369 4588, Extn 94588
Email: human-ethics@canterbury.ac.nz

UNIVERSITY OF CANTERBURY
Te Whare Wānanga o Waitaha
CHRISTCHURCH NEW ZEALAND

2020/07/EX

6 October 2020

McLeod Robertson
Psychology, Speech and Hearing
University of Canterbury

Dear McLeod,

I can confirm that your request for an exemption for the research project titled "Does Psychotherapy for Binge Eating Remediate Nutrient Intake in People With Bulimia Nervosa and Binge Eating Disorder?" has been reviewed and approved by the Human Ethics Committee.

Yours sincerely

Dr Dean Sutherland
Chair
University of Canterbury Human Ethics Committee