PERSONALITY IN BULIMIA NERVOSA
AND BINGE EATING DISORDER

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A thesis submitted for the degree of

Doctor of Philosophy

University of Otago, Christchurch

NEW ZEALAND

December 2010
ABSTRACT

Background:

Axis I and II disorders are not independent. Understanding the relationship between these groups of disorders requires further exploration. This thesis will explore whether personality disorders, assessed by clinical interview using DSM criteria, or personality traits, assessed by the self-report Temperament and Character Inventory (TCI), impact on the presentation of bulimia nervosa (BN) and binge eating disorder (BED) and on outcome of BN. Examining personality comorbidity in BN and BED using both categorical and dimensional models may help to clarify relationships between personality functioning and these eating disorders. Identifying personality predictors of outcome may have implications for treatments of these eating disorders.

Objectives:

1. To determine the impact of personality functioning on clinical characteristics at pre-treatment and long-term outcome in women with BN

2. To determine the impact of personality functioning on clinical characteristics in women with BED at pre-treatment

3. To compare personality profiles between women with BN and BED at pre-treatment, in comparison to psychiatric control and healthy control groups
Methods:

Participants for this study were drawn from two randomised controlled trials: the Bulimia Treatment Study (BTS) and the Binge Eating Psychotherapy study (BEP). In the BTS study (n=134), separate analyses were used to examine the specific impact of borderline, avoidant and complex personality disorders on clinical characteristics, global functioning and outcome on women with BN. Personality traits and symptoms were examined to determine the ability of these measures to predict long-term outcome of BN.

In the Binge Eating Psychotherapy (BEP) study, 38 women with BN and 41 women with BED participated at pre-treatment. To establish the specificity and the significance of any findings regarding personality profiles, two comparison samples of 39 women with depression and 44 healthy control women were used. The impact of personality disorders on clinical characteristics, general psychiatric functioning and global functioning were compared between BN and BED groups.

Results:

The presence of borderline, avoidant or complex personality disorders had a negative impact on depression severity and global functioning in women with BN however the presence of a personality disorder did not impact on long-term BN outcome. The only personality variable to predict outcome at five-years was self-directedness.

Women with BN and BED had similar personality profiles to depressive controls but were distinguishable from healthy controls. Personality disorders were associated with
increased binge eating in the BED group but not the BN group. Self-directedness and cooperativeness were significantly lower in the BED group than the BN group.

Conclusions:

Although personality disorders are associated with a more severe clinical picture at pre-treatment, they have little predictive validity as they do not influence eating disorder outcome and broader outcomes. Specific dimensional measures of personality had more predictive validity, however the most important of these was self-directedness. Self-directedness may be capturing the essence of personality dysfunction and it appears to be as important in eating disorders as it is in other Axis I disorders. Developing interventions to increase this characteristic may enhance treatment for these eating disorders.
PREFACE

The BEP was funded by the Health and Research Council of New Zealand with Professor Peter Joyce as the principal investigator. Other investigators named on the grant were: Virginia McIntosh, Jennifer Jordan, Janice McKenzie, Janet Carter and Christopher Frampton. The BEP study commenced recruiting in 2005 and completed recruiting in 2010.

I was employed in 2004 as an Assistant Research Fellow prior to the study commencement. I enrolled as a full-time PhD candidate in 2007, examining questions related to how personality contributes to presentation and outcome of BN and presentation of BED in comparison to BN. Whilst the BEP study was recruiting, I focused on participant data from the BTS, a treatment trial previously conducted in this department.

My roles in the BEP study included: coordinating the day-to-day running of the study, recruitment of participants, physical measurements of participants, cognitive testing, creating and maintaining databases, blind rater assessments of binge frequency (the key outcome measure), personality assessments, follow-up/tracing of participants and conducting structured clinical interviews at follow-up.

Finally, I have presented two posters and one paper at international conferences; I have had involvement in four publications related to this thesis and in 2010, I was invited to lecture on comorbidity in eating disorders for a postgraduate paper on eating disorders.
ACKNOWLEDGEMENTS

The nature of a PhD is ‘an individual’s ability to conduct original research, make a significant contribution to knowledge in the particular field and present the findings to a professional standard’, however, this is not achieved alone. Like the majority of research, this thesis is a collaboration with a multidisciplinary team with whom I am indebted. I would like to acknowledge my colleagues and investigators on the BTS and BEP study, Professor Peter Joyce, Dr Jenny Jordan, Dr Gini McIntosh, Dr Frances Carter and Professor Cindy Bulik. Thank you for your generosity in including me as a member of the research team, for training me in clinical assessments, for helping to evolve my writing skills and for the encouragement and support I have received throughout this process – I have learned a great deal from you all. I am immensely grateful to Professor Chris Framptom, who managed to do the unthinkable – demystify statistics. Thank you Chris for your time, patience and encouragement throughout the past three and a half years. I would also like to acknowledge Andrea Bartram, my teacher in managing databases and coordinating research studies. I could not have done this without the knowledge you passed on to me – thank you. I would also like to thank Wendy Sincock for her help in assisting the formatting of this thesis and Dr Kumari Fernando for proof-reading this thesis. You are not only great colleagues but wonderful friends and I appreciate your help and support during this time.

To my supervisor Professor Peter Joyce, thank you for your support, guidance, wisdom and enthusiasm that have taught me so much and have inspired a love of research. The lessons that you have taught me will stay with me throughout my career and I will be forever grateful for the opportunities you have provided me. To my supervisor and
mentor Dr Jenny Jordan, no words can express the gratitude I feel for the hard work, support, encouragement and guidance that you gave me during this process. You are an inspiration and yet, so humble and unaware of it. I feel honored to have you as my supervisor.

I would like to acknowledge the participants, without whom there would be no trials. Thank you for sharing your personal experiences and devoting substantial time to helping us with our research. I hope that this thesis goes some way to showing my appreciation and provides some results and meaning to the many assessments we ask you to complete.

Thank you to my family, particularly my parents (all five of them) – Mum, Dad, Piggy, Ma and Pa, for the work ethic you have instilled in me and for the support, encouragement and hundreds of times you have told me “I am so proud of you”. Your belief in me has always been unwavering and importantly has made me believe in myself.

Thank you to my good friends, particularly Lisa Walker and Rachel Cosgrove, who have tolerated three and a half years of PhD conversation and still not complained once. I am lucky to have surrounded myself with inspirational and strong women such as the both of you.

“Life is either a daring adventure or nothing at all” (Helen Keller). Thank you to my husband Jim, who lives this philosophy with me everyday. You have provided me with the balance that enables me to take on challenges such as this thesis. Your love, support and sense-of-humour helped me get to the end of this chapter and prepare me for a new one, thank you.
LIST OF PUBLICATIONS

Publications directly related to this thesis


LIST OF PUBLICATIONS

Conference presentations related to this thesis


Rowe, S. L. Jordan, J., McIntosh, V. V., Carter, F. A., Frampton, C., Bulik, C. M., Joyce, P. R. (2007). Sorting fact from fiction: Do women with bulimia nervosa and comorbid borderline personality disorder really have poorer outcome over time? Poster at the Australia and New Zealand Academy for Eating Disorders Conference.
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<tbody>
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<td>AN</td>
<td>Anorexia Nervosa</td>
</tr>
<tr>
<td>AN-BP</td>
<td>Anorexia Nervosa – Binge eating/Purging subtype</td>
</tr>
<tr>
<td>AN-R</td>
<td>Anorexia Nervosa – Restricting subtype</td>
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<tr>
<td>ANOVA</td>
<td>Analysis of Variance</td>
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<tr>
<td>BED</td>
<td>Binge Eating Disorder</td>
</tr>
<tr>
<td>BEP</td>
<td>Binge Eating Psychotherapy study</td>
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<tr>
<td>BMI</td>
<td>Body Mass Index</td>
</tr>
<tr>
<td>BN</td>
<td>Bulimia Nervosa</td>
</tr>
<tr>
<td>BTS</td>
<td>Bulimia Treatment Study</td>
</tr>
<tr>
<td>CANTAB</td>
<td>Cambridge Neuropsychological Test Automated Battery</td>
</tr>
<tr>
<td>CBSI</td>
<td>Comprehensive Bulimia Severity Index</td>
</tr>
<tr>
<td>CBT</td>
<td>Cognitive Behaviour Therapy</td>
</tr>
<tr>
<td>CBT-A</td>
<td>Cognitive Behaviour Therapy – Nutrition and Appetite Enhanced</td>
</tr>
<tr>
<td>DBT</td>
<td>Dialetical Behaviour Therapy</td>
</tr>
<tr>
<td>DIPD</td>
<td>Diagnostic Interview for Personality Disorders</td>
</tr>
<tr>
<td>DSM</td>
<td>Diagnostic and Statistical Manual of Mental Disorder</td>
</tr>
<tr>
<td>Abbreviation</td>
<td>Description</td>
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<tr>
<td>DSM-I</td>
<td>Diagnostic and Statistical Manual of Mental Disorder – 1st Edition</td>
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<td>DSM-III</td>
<td>Diagnostic and Statistical Manual of Mental Disorder – 3rd Edition</td>
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<td>DSM-III-R</td>
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<td>DSM-5</td>
<td>Diagnostic and Statistical Manual of Mental Disorder – 5th Edition</td>
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<td>EDE</td>
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<td>EDE-Q</td>
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<tr>
<td>EDI</td>
<td>Eating Disorder Inventory</td>
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<tr>
<td>EDI-2</td>
<td>Eating Disorder Inventory-Revised</td>
</tr>
<tr>
<td>EDNOS</td>
<td>Eating Disorder Not Otherwise Specified</td>
</tr>
<tr>
<td>FDA</td>
<td>Food and Drug Administration</td>
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<tr>
<td>GAF</td>
<td>Global Assessment of Functioning</td>
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<tr>
<td>HDRS</td>
<td>Hamilton Depression Rating Scale</td>
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<tr>
<td>Acronym</td>
<td>Description</td>
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<tr>
<td>IPT</td>
<td>Interpersonal Psychotherapy</td>
</tr>
<tr>
<td>MADRS</td>
<td>Montgomery-Asberg Depression Rating Scale</td>
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<tr>
<td>MDD</td>
<td>Major Depressive Disorder</td>
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<tr>
<td>MMPI</td>
<td>Minnesota Mutiphasic Personality Inventory</td>
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<td>NICE</td>
<td>National Institute for Clinical Excellence</td>
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<td>PDE</td>
<td>Personality Disorder Examination</td>
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<td>Hopkins Symptom Checklist</td>
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<tr>
<td>SCID-I</td>
<td>Structured Clinical Interview for DSM-III-R or DSM-IV – Axis I Disorders</td>
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<td>SCID-II</td>
<td>Structured Clinical Interview for DSM-III-R or DSM-IV – Axis II Disorders</td>
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<tr>
<td>SFT</td>
<td>Schema Focused Therapy</td>
</tr>
<tr>
<td>SPSS</td>
<td>Statistical Package for the Social Sciences</td>
</tr>
<tr>
<td>SSCM</td>
<td>Specialist Supportive Clinical Management</td>
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<tr>
<td>TCI</td>
<td>Temperament and Character Inventory</td>
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<tr>
<td>TCI-R</td>
<td>Temperament and Character Inventory-Revised</td>
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PART 1

Chapter 1: Introduction

1.1 INTRODUCTION

Eating disorders such as bulimia nervosa (BN) and binge eating disorder (BED) are considerably heterogeneous. One source of variance within these diagnostic groups is the presence or absence of a personality disorder. It has been estimated that as many as 60% of women with an eating disorder also have a personality disorder (Loxton & Dawe, 2009; Rosenvinge, Martinussen, & Ostensen, 2000). There are mixed findings about whether the presence of a personality disorder (particularly borderline personality disorder) is associated with a poor eating disorder course and outcome (Fallon, Walsh, Sadik, Saoud, & Lukasik, 1991; Grilo, Sanislow, Shea, Skodol, Stout, Pagano, Yen, & McGlashan, 2003; Johnson, Tobin, & Dennis, 1990; Keel & Mitchell, 1997; Norring, 1993; Steiger & Stotland, 1996; Wonderlich, Fullerton, Swift, & Klein, 1994; Zanarini, Frankenburg, Hennen, & Silk, 2003). Methodological issues have limited conclusions that can be made about the impact of personality and personality disorders on eating disorders. These methodological issues include differences in personality measures used, differences in eating disorder groups and whether normal or abnormal personality is measured.

This thesis aims to address some of the gaps in our knowledge by examining the impact of personality and personality disorders on the symptomatic expression in BN and BED, and the maintenance and outcome of BN. Part 1 provides the context for this thesis by
introducing key concepts of definition, aetiology, course and outcome of eating
disorders (Chapter 1). Part 2, Chapter 2 presents a history of personality and personality
disorders followed by an overview of personality and personality disorders in relation to
Axis I disorders in general. Chapter 3 presents an overview of personality and
personality disorders in eating disorders with a particular focus on BN and BED. Part 3
provides an overview of the methodology in the Bulimia Treatment Study (BTS) and
the Binge Eating Psychotherapy (BEP) study (Chapters 4 and 5 respectively). Part 4
addresses the impact of personality disorder comorbidity in a previously recruited
sample of women with BN (the BTS). Chapter 6 will explore whether the presence of
borderline personality disorder negatively affects the clinical characteristics of BN at
one and three-years post-treatment. Chapter 7 will examine the impact of avoidant
personality disorder on the pre-treatment clinical characteristics and outcome of BN
whilst controlling for social phobia and mood disorder. Chapter 8 will present a new
classification system proposed by Tyrer using overlapping personality disorder
diagnosis to create a complex personality disorder category. This classification system
will be used to examine the impact of multiple Axis II comorbidities on clinical
characteristics in BN. In Chapter 9, the impact of personality dimensions will be
examined against the five-year follow-up for women with BN. Part 5 examines
personality and personality disorders in BN and BED and makes comparisons between
these groups. Chapter 10 examines the impact of personality disorders on the clinical
characteristics in those with BED compared to those with BN in the BEP study. Chapter
11 compares temperament and character profiles within the BN and BED groups, and in
comparison to depressive control and healthy control groups. Part 6 (Chapter 12)
summarises key findings and discusses these in relation to the study goals and
hypotheses, and relevant literature. The implications of these findings will be discussed with regard to eating disorder services and future research.

1.2 THE NATURE OF EATING DISORDERS

Eating disorders are psychological disorders characterised by abnormal eating patterns such as insufficient or excessive food intake. Individuals experiencing an eating disorder generally have a disturbed perception or marked dissatisfaction of their body weight and shape. Those with anorexia nervosa (AN) or BN attempt to control these aspects through behaviours such as excessive dieting, exercising and/or purging (Ministry of Health, 2008). Those with BED do not engage in regular compensatory behaviours. Medical symptoms associated with the eating disorder may result in acute and chronic complications that can be life-threatening or disabling (Buckett, 2002 and Gelder, 2000 in Ministry of Health, 2008).

The typical age-of-onset of an eating disorder is during adolescence or young adulthood and eating disorders such as AN and BN occur disproportionally in females. Eating disorders are often comorbid with other psychiatric disorders such as mood disorders, anxiety disorders, substance use disorders and personality disorders (Hudson, Hiripi, Harrison, Pope & Kessler, 2007; Lewinsohn, Striegel-Moore & Seeley, 2000).

The Diagnostic and Statistical Manual of Mental Disorders – Fourth Edition-Text Revision (DSM-IV-TR) is the most widely used diagnostic system and includes three main categories: AN, BN and ‘eating disorders not otherwise specified’ (EDNOS), which includes the provisional category of BED (American Psychiatric Association, 2000a).
1.2.1  Anorexia nervosa

1.2.1.1 Definition

Anorexia nervosa is characterised by the severe restriction of food intake resulting in significant weight loss or failure to maintain a minimally normal weight. In addition, there is a preoccupation with weight and shape and an intense fear of gaining weight or becoming fat. In the DSM-IV-TR, the presence of amenorrhea in postmenarchal females for three months is also required (American Psychiatric Association, 2000a). There are two subgroups of AN: restricting type (AN-R) and binge eating/purging type (AN-BP).

1.2.1.2 Prevalence and demographics

Lifetime estimates of the prevalence of AN range from 0.5% to 1% in women with the gender ratio being 9:1 of women to men (Attia, 2010). However, the National Comorbidity Survey-Replication (NCS-R) by Hudson and colleagues suggests the gender ratio is much closer at 3:1 (Hudson et al., 2007). The typical age-of-onset range is 15 to 18 years (Kaye, Bulik, Thornton, Barbarich, & Masters, 2004; Morris & Twaddle, 2007).

1.2.1.3 Public health significance

Anorexia nervosa is amongst the top three chronic illnesses in adolescence (Lucas, Beard, O'Fallon, & Kurland, 1991). It has the highest mortality rate of any psychiatric disorders due to medical complications and suicide (Morris & Twaddle, 2007; Papadopoulos, Ekborn, Brandt, & Ekselius, 2009). Standardised mortality ratios range from 1.36 to 17.80 times greater in women with AN compared to the general population.
(Birmingham, Su, Hlynsky, Goldner, & Gao, 2005; Eckert, Halmi, Marchi, Grove, & Crosby, 1995; Nielsen, Moller-Madsen, Isager, Jorgensen, Pagsberg, & Theander, 1998; Papadopoulos et al., 2009). The cost of treating AN is comparable to that of schizophrenia however, the financial burden is less because of the rarity of AN (Striegel-Moore, Leslie, Petrill, Garvin, & Rosenheck, 2000).

1.2.1.4 Aetiology

The aetiology of AN remains unknown, although evidence suggests a biological and/or genetic vulnerability may contribute to the development of this disorder (Attia, 2010). Biological factors such as dysfunction in the serotonergic pathways have been hypothesized as predisposing and maintaining clinical features of AN (Attia, 2010; Treasure & Campbell, 1994). Family and twin studies indicate a significant genetic influence with up to 58% heritability (Holland, Hall, Murray, Russell, & Crisp, 1984; Klump, Miller, Keel, McGue, & Iacono, 2001; Strober, Freeman, Lampert, Diamond, & Kaye, 2000; Wade, Bulik, Neale, & Kendler, 2000). Other features associated with AN such as anxiety, obsessionality and perfectionism, have also been identified as familial traits that are likely to interact with environmental factors to predispose to the development of an eating disorder (Anderluh, Tchanturia, Rabe-Hesketh, & Treasure, 2003a; Wade, Tiggemann, Bulik, Fairburn, Wray, & Martin, 2008). Sociocultural risk factors for AN that have been suggested are gender, socio-economic status, the idealisation of thinness, perinatal and psychosocial factors (Lindberg & Hjern, 2003). Negative family influences such as criticism and overprotectiveness may also contribute to the aetiology of AN (Polivy & Herman, 2002).
1.2.1.5 Comorbidity

Comorbidity in eating disorders is the rule rather than the exception (Braun, Sunday, & Halmi, 1994). High rates of Axis I mood and anxiety disorders have been found in the AN population compared to controls. Levels are similar though, to other eating disorder groups (Kaye et al., 2004; O'Brien & Vincent, 2003). Obsessive-compulsive disorder is specifically elevated in the AN group compared to other eating disorder groups (Godart, Flament, Lecrubier, & Jeammet, 2000; Jordan, Joyce, Carter, Horn, McIntosh, Luty, McKenzie, Frampton, Mulder, & Bulik, 2008; Ro, Martinsen, Hoffart, & Rosenvinge, 2005). Two independent family studies have found AN-R and obsessive-compulsive personality disorder follow a pattern of shared familial transmission, suggesting common underlying aetiological factors (Lilenfeld, Wonderlich, Riso, Cross & Mitchell, 2006; Strober, Freeman, Lampert & Diamond, 2007). Prevalences of substance use disorders have been reported to be lower in AN than BN groups (Wonderlich & Mitchell, 1997). High rates of Axis II Cluster C disorders have been found in AN patients compared to controls however, levels appear to be similar to other eating disorder groups (Karwautz, Rabe-Hesketh, Collier, & Treasure, 2002; O'Brien & Vincent, 2003). Rates of Cluster B disorders are reported to be lower in the AN-R group compared to other eating disorder groups although similar rates have been reported between AN-BP and BN groups (Cassin & von Ranson, 2005; Herzog, Keller, Lavori, Kenny, & Sacks, 1992; Sansone & Levitt, 2005b).

1.2.1.6 Treatment

The success of treatment for AN remains limited and there is a lack of clear empirical support for any specific approach. Specialist inpatient treatment is most likely to be
used in AN compared to other eating disorders because of the need for medical monitoring and refeeding programmes designed for weight restoration (Attia, 2010). Research suggests that family-based therapy for children and adolescents with AN is the most effective intervention (Couturier, Isserlin, & Lock, 2010). Furthermore, there is current debate about whether the Maudsley method of family-based treatment should be recommended as the first-line treatment used in this population (Kohn, Forbes, Harris, & Wallis, 2010). There have been very few psychotherapy trials of adults with AN and there is insufficient evidence of superiority of any single treatment (Hay, Bacaltchuk, Claudion, Ekmejian, & Yong, 2009). Indeed, a clinical trial conducted in this department comparing cognitive-behavioural therapy (CBT), interpersonal therapy (IPT) and specialist supportive clinical management (SSCM) found that SSCM was superior to CBT and IPT at the end of treatment, challenging assumptions about the effective components for successful treatments of AN (McIntosh, Jordan, Carter, Luty, McKenzie, Bulik, Frampton, & Joyce, 2005). Pharmacologic treatment of AN appears to have limited effect (Zhu & Walsh, 2002).

1.2.1.7 Course and outcome

Approximately one-third of AN cases follow a chronic course (Palmer & Treasure, 1999) with reports suggesting an average of five to six years from diagnosis to recovery (Morris & Twaddle, 2007). In a metanalysis using 119 outcome studies of AN, 46.9% of patients were fully recovered, 33.5% of patients improved and 20.8% continued to have a diagnosis of AN at follow-up (Steinhausen, 2002). Across studies, better outcome has been associated with adolescent age-of-onset (Steinhausen, 2002).
1.2.2 Bulimia nervosa

1.2.2.1 Definition

Bulimia nervosa is characterised by binge eating episodes that are defined by the DSM-IV-TR as eating an amount of food that is larger than most individuals would eat, in a discrete period of time and accompanied by a subjective sense of loss of control (American Psychiatric Association, 2000a). The use of inappropriate compensatory behaviours and an over-evaluation of weight and shape is essential for the diagnosis of BN (American Psychiatric Association, 2000a). Compensatory behaviours may include purging (vomiting, laxatives, diuretic or enemas) and non-purging (fasting or excessive exercise) types. Binge eating and compensatory behaviours must occur twice weekly for three months to meet diagnostic criteria (American Psychiatric Association, 2000a).

1.2.2.2 Prevalence and demographics

Lifetime prevalence rates of full-syndrome BN range from 1% to 1.7% among young women however, rates of partial syndrome BN are substantially higher with estimates ranging from 2.3% to 5.4% (Hoek & van Hoeken, 2003; Hudson et al., 2007; Keski-Rahkonen, Hoek, Linna, Raevuori, Sihvola, Bulik, Rissanen, & Kaprio, 2009). Diagnosis of this disorder is relatively rare in men and lifetime prevalence rates range from 0.1% to 0.5% (Hoek & van Hoeken, 2003; Hudson et al., 2007). The typical age range for onset of BN is 16-20 years (Keski-Rahkonen et al., 2009).

1.2.2.3 Public health significance

Eating disorders, including BN, pose a significant public health concern because they are associated with significant physical and psychological morbidity and are often
untreated or under treated (Hudson et al., 2007). Individuals with BN exhibit difficulties with cognitive functioning, emotional functioning and life activities (quality of life, educational and vocational functioning and social adjustment; Klump, Bulik, Kaye, Treasure, & Tyson, 2009). Comorbidity of psychiatric disorders is common within a BN population. A consequence of the comorbidity between BN (and other eating disorders) and additional psychopathology is the potential difficulty in selecting optimal psychological or pharmacological treatments (O'Brien & Vincent, 2003). This can be further complicated by having to modify treatment interventions to accommodate multiple disorders (Vitousek & Stumpf, 2005).

1.2.2.4 Aetiology

The aetiology of BN is thought to be influenced by both environmental and genetic factors. Heritability estimates range from from 28% to 83% with the remaining variance attributed to environmental factors (Bulik, 2005). Non-specific risk factors for BN include: family environmental factors, premorbid psychiatric disorders, personality traits and childhood trauma (Lehoux & Howe, 2007). Specific risk factors associated with BN include: negative self-evaluation, dieting and poor body image (Fairburn, Welch, Doll, Davies, & O'Connor, 1997; Lehoux & Howe, 2007; Stunkard, 1997). Many of these risk factors are included in a review by Jacobi and colleagues (Jacobi, Hayward, de Zwaan, Kraemer & Agras, 2004), along with: pregnancy complications, female gender, ethnicity, childhood obesity, childhood overanxious disorder, adolescent age, pubertal timing, acculturation, adverse family experiences, parental obesity, parental psychopathology and social phobia.
1.2.2.5 Comorbidity

Higher prevalence rates have been found in BN for comorbid Axis I disorders such as mood disorders (Casper, 1998; Godart, Perdereau, Rein, Berthoz, Wallier, Jeammet, & Flament, 2007) and anxiety disorders (Bulik, Sullivan, Fear, & Joyce, 1997; Swinbourne & Touyz, 2007) relative to control groups. There is a similar level of mood and anxiety disorders relative to other eating disorder groups (Johnson, Spitzer, & Williams, 2001; Jordan et al., 2008). One exception may be bipolar II disorder which some studies suggest is associated with elevated levels of BN (Joyce, Luty, McKenzie, Mulder, McIntosh, Carter, Bulik, & Sullivan, 2004; McElroy, Kotwal, Keck, & Akiskal, 2005; Simpson, Almufti, Andersen, & Depaulo, 1992). Substance use disorders have been found to be more common in the BN groups compared to other eating disorder groups (Bulik, Sullivan, Carter, & Joyce, 1997; Wonderlich & Mitchell, 1997). Axis II disorders, particularly borderline personality disorder, are increased in BN (Pearlstein, 2002; Sansone, Levitt, & Sansone, 2005).

1.2.2.6 Treatment

Manual-based CBT (16-20 sessions over four to five months) has been selected by the National Institute for Clinical Excellence (NICE) as the treatment of choice with the best efficacy for patients with BN (Wilson, 2005). Although there is also evidence of efficacy for IPT, it can take 8-12 months to achieve the equivalent efficacy as CBT (Wilson, 2005). Selective serotonin reuptake inhibitor medication has been shown to have a modest effect in the treatment of BN due to a specific anti-binge effect (Zhu & Walsh, 2002). To date, only Fluoxetine has the Food and Drug Administration (FDA) approval for the treatment of BN (Jackson, Cates, & Lorenz, 2010).
1.2.2.7 Course and outcome

The treatment outcome literature indicates that the course of BN may follow a relapsing and remitting pattern with around 50% to 74% of women with BN achieving remission over long-term follow-up (Fichter & Quadflieg, 2004; Grilo, Pagano, Skodol, Sanislow, McGlashan, Gunderson, & Stout, 2007; Keel & Mitchell, 1997). Those taking longer than five years to achieve remission are described as having a chronic course (Keel & Brown, 2010). Predictors of outcome remain unclear with some research reporting poor outcome to be associated with older age-of-onset (Collings & King, 1994; Fichter & Quadflieg, 2004), longer duration of illness (Keel, Mitchell, Miller, Davis, & Crow, 1999; Reas, Williamson, Martin, & Zucker, 2000), history of obesity (Bulik, Sullivan, Joyce, Carter, & McIntosh, 1998b), psychiatric comorbidity such as major depressive disorder (MDD) or personality disorders (Fichter & Quadflieg, 2004; Maddocks & Kaplan, 1991; Rossiter, Agras, Telch, & Schneider, 1993), increased binge or purge frequency (Fahy & Russell, 1993; Garner, Olmsted, Davis, Rockert, Goldbloom, & Eagle, 1990) and specific psychological characteristics (for example, low self-esteem; Fairburn, Peveler, Jones, Hope, & Doll, 1993b). However, other studies have failed to find these associations (Fallon et al., 1991; Grilo et al., 2003; Herzog, Hartmann, Sandholz, & Stammer, 1991; Mitchell, Davis, Goff, & Pyle, 1986).

1.2.3 Eating disorder not otherwise specified

1.2.3.1 Definition

Eating disorder not otherwise specified is the most common eating disorder category representing between 50% and 67% of presentations to eating disorder services (Button, Benson, Nollett, & Palmer, 2005; Turner & Bryant-Waugh, 2004). An EDNOS
diagnosis is used for clinically significant eating disorder behaviours and cognitions that do not meet the specific diagnostic criteria for AN or BN. Research shows that the majority of patients diagnosed with EDNOS at a community eating disorder service have similar levels of psychopathology to those with a full diagnosis of AN or BN (Thomas, Vartanian, & Brownell, 2009; Turner & Bryant-Waugh, 2004). The DSM-IV-TR identifies six EDNOS categories. These are: 1) all the criteria for AN are met except that the individual has regular menses, 2) all the criteria for AN are met except that the individual is in the normal weight range, 3) all the criteria for BN are met except that the binge eating and inappropriate compensatory behaviours are less than the diagnostic frequency of twice weekly for three months, 4) inappropriate compensatory behaviours occur after eating small amounts of food, 5) repeatedly chewing and spitting out, but not swallowing, large amounts of food, 6) binge eating disorder (American Psychiatric Association, 2000a).

1.2.3.2 Prevalence and demographics

Lifetime prevalence rates of EDNOS range from 2.4% to 5.3% (Machado, Machado, Goncalves, & Hoek, 2007; Wade, Bergin, Tiggemann, Bulik, & Fairburn, 2006). Similarly to other eating disorders, EDNOS occurs predominately in females (Button et al., 2005; Mitchell, Crosby, Wonderlich, Hill, le Grange, Powers, & Eddy, 2007). The age-of-onset is difficult to establish due to the variation in EDNOS categories, although for the purging type it has been reported to range from 13 to 34 years (Wade et al., 2006).
1.2.3.3 Public health significance

Research shows no difference between the EDNOS group and full syndrome eating disorders in treatment service consumption (Button et al., 2005). Half of all outpatient appointments were accounted for by the EDNOS group and the labour-intensiveness of the treatment was at the same level as those with full-syndrome eating disorders (Button et al., 2005). This level of service consumption is seldom accounted for when planning specialised eating disorder services, despite the needs of the EDNOS group being similar to those with AN and BN.

1.2.3.4 Aetiology

Due to the large variation and subgroups encompassed within the EDNOS category, there is little or no information about the aetiology for this diagnosis. It is likely that the subthreshold groups would have a similar pattern of aetiology to full-syndrome eating disorders.

1.2.3.5 Comorbidity

Research on comorbidity in EDNOS is limited. High comorbidity of mood disorders, anxiety disorders and substance use disorders have been found in EDNOS groups compared to the general population (Milos, 2009). Comorbidity prevalence rates are similar in EDNOS to those in AN and BN for anxiety and mood disorders, although as noted earlier, BN has higher substance use disorders comorbidity in relation to other eating disorders (Blinder, Cumella, & Sanathara, 2006). Rates of Axis II disorders are similar to AN and BN with avoidant personality disorder the most common in this group (Wonderlich, Mitchell, De Zwaan, & Steiger, 2007).
1.2.3.6 Treatment

Treatment response is unknown as there have been no specific treatment studies conducted for EDNOS, with the exception of the BED category (Fairburn & Bohn, 2005). To date, treatment advice is limited to the NICE (2004) guidelines that suggest “in the absence of evidence to guide the management of eating disorder not otherwise specified, other than BED, it is recommended that the clinician considers following the guidance on the treatment of the eating problem that most closely resembles the individual patient’s eating disorder” (p.71 cited in Wilson, 2005). Wilson (2005) suggests Fairburn’s transdiagnostic model as a treatment option due to its emphasis on specific mechanisms that maintain different eating disorders (Fairburn, Cooper, Doll, O'Connor, Bohn, Hawker, Wales, & Palmer, 2009; Wilson, 2005).

1.2.3.7 Course and outcome

Few studies have examined the course and outcome of EDNOS. The general pattern for the course of the EDNOS group is for a large amount of crossover between subthreshold and full-diagnostic classification (Fairburn & Bohn, 2005). A study by Herzog et al. (1993) showed that almost half of the EDNOS group went on to develop a full-criteria eating disorder and that eating disorder psychopathology in this group is “highly persistent” (p.266). Compared to AN and BN, EDNOS is considered the least stable eating disorder diagnosis (Milos, Spindler, Schnyder, & Fairburn, 2005). Those in partial remission from full AN or BN diagnoses would also fall into this category. A recent study suggests that outcome for this group is similar to BN in which recovery was achieved in 75% of patients at 20-year follow-up (Keel, Gravener, Joiner, & Haedt, 2010).
1.2.4 Binge eating disorder

1.2.4.1 Definition

The diagnostic criteria for BED are recurrent episodes of binge eating that occur on average, at least two days a week for six months and cause marked distress. Binge eating episodes are associated with at least three of the following behaviours: eating more rapidly than usual, eating until uncomfortable full, eating large quantities of food when not feeling physically hungry, eating alone because of embarrassment about how much one is eating, feelings of disgust, depression or guilt after overeating. Unlike AN and BN, it does not include regular compensatory behaviours for weight control (American Psychiatric Association, 2000a).

1.2.4.2 Prevalence and demographics

Prevalence rates for BED are variable ranging from 1.2% to 6.6% (Fairburn, 1993; Ghaderi & Scott, 2000; Grucza, 2006; Hoek & van Hoeken, 2003) depending on the measures used and samples examined (for example, community sample or clinical sample). Demographics for this eating disorder group are more diverse than typically seen in the AN and BN groups. Several studies suggest comparable levels of binge eating in both men and women but differences in feelings of distress and loss of control which are key factors for the diagnosis of BED (Lewinsohn, Seeley, Moerk, & Striegel-Moore, 2002). Inconsistent research findings mean that it is unclear whether rates for full-syndrome BED are similar between men and women (Spitzer, Devlin, Walsh, Hasin, Wing, Marcus, Stunkard, Wadden, Yanovski, Agras, Mitchell, & Nonas, 1992; Striegel-Moore & Franko, 2003). Age-of-onset for BED is typically later than BN,
approximately 23 to 25 years (Grilo & Masheb, 2000; Spurrell, Wilfley, Tanofsky, & Brownell, 1997).

1.2.4.3 Public health significance

Women with BED have been reported as having poorer functioning, higher levels of disability, health problems, insomnia, psychosocial stress and suicidal thoughts than healthy women without BED (Johnson et al., 2001). In addition, binge eating has been associated with obesity (Smith, Marcus, Lewis, Fitzgibbon, & Schreiner, 1998), a major health problem associated with significant physical morbidity. Although those with BED have been shown to have similar levels of impairment to those with BN (Johnson et al., 2001), treatment for this disorder may not be readily available in some settings as BED only has a provisional eating disorder status in the DSM-IV-TR (American Psychiatric Association, 2000a). This may result in a large number of untreated individuals with a clinically significant eating disorder.

1.2.4.4 Aetiology

It has been suggested that genetic factors, ethnicity, temperament and character, and a history of abuse may be involved in the aetiology of BED (Pull, 2004). For example, there appears to be moderate heritability for obesity and binge eating (Bulik, Sullivan, & Kendler, 2003) including a reported gene mutation on the melanocortin 4 receptor that may play a role in the development of binge eating (Branson, Potoczna, Kral, Lentes, Hoche, & Horber, 2003). Retrospective studies suggest the following risk factors for BED: emotional abuse, physical abuse, sexual abuse, emotional neglect and physical neglect (Grilo & Masheb, 2001; Striegel-Moore, Dohm, Pike, Wilfley, & Fairburn, 2002).
1.2.4.5 Comorbidity

Binge eating disorder has been reported to be associated with higher prevalences of comorbid mood disorders (Telch & Stice, 1998) and anxiety disorders compared to controls (Yanovski, 1993), but similar levels compared to other eating disorders (Johnson et al., 2001). High prevalences of Axis II disorders have been reported in BED. Whilst these appear to be at a similar level to AN, they may be lower levels than in BN groups (Sansone et al., 2005). Obsessive-compulsive personality disorder has been the most common personality disorder found in this group (Sansone & Levitt, 2005b).

1.2.4.6 Treatment

Four factors have been identified as targets for BED treatment: 1) binge eating, 2) eating disorder psychopathology, 3) general psychopathology and 4) excess body weight or obesity (Wilson, 2005). The current treatment-of-choice for BED is CBT. Although CBT is effective, it does not appear superior to other treatments such as IPT (Wilfley, Welch, Stein, Spurrell, Cohen, Saelens, Dounchis, Frank, Wiseman, & Matt, 2002), nor does it lead to weight loss (Brownley, Berkman, Sedway, Lohr, & Bulik, 2007). Behavioural weight loss treatment has been reported to be successful in treating binge eating, improving general psychiatric symptoms and depressive symptomatology (Wilson, 2005). Despite this, the effects of weight loss interventions are less sustainable than CBT at six and twelve month follow-up (Nauta, Hospers, & Jansen, 2001). Pharmacologic treatment for BED has been trialled with some promise shown for antidepressants and appetite suppressants. However, further efficacy studies need to be
conducted to clarify medication selection, dosing, duration and possible treatment combinations (Carter, Hudson, Lalonde, Pindyck, McElroy, & Pope, 2003b).

1.2.4.7 Outcome and predictors of outcome

Some studies have suggested that the course and outcome for BED is more favourable when compared to BN (Fairburn, Cooper, Doll, Norman, & O'Connor, 2000). However, remission rates for BED appear to be similar to those with BN with ranges between 50% to 57%, five and six years after treatment (Fairburn et al., 2000; Fichter, Quadflieg, & Gnutzmann, 1998). Many predictors of poor BED outcome have been suggested, including: early age-of-onset of binge eating and higher EDE scores at post-treatment (Safer, Lively, Telch, & Agras, 2002), psychiatric comorbidity, body dissatisfaction, sexual abuse and impulsivity (Fichter, Quadflieg, & Hedlund, 2008a), increased frequency of binge eating episodes at pre-treatment (Peterson, Crow, Nugent, Mitchell, Engbloom, & Mussell, 2000), higher levels of palliative reacting coping style (“trying to feel better by eating, smoking or drinking”; Dingemans, Spinhoven, & van Furth, 2007), and the presence of Cluster B personality disorders (Wilfley, Friedman, Dounchis, Stein, Welch, & Ball, 2000a).

1.3 CONTEXT OF THIS RESEARCH

The samples used in this thesis were participants recruited for two separate randomised controlled trials for eating disorders. The first study was the Bulimia Treatment Study (BTS; Bulik, Sullivan, Carter, McIntosh, & Joyce, 1998a). In this study women with BN were recruited for a treatment trial with long term follow-up. The trial evaluated cognitive therapy with randomisation to two exposure based treatments or relaxation as
a control. This thesis utilised personality data from that study to evaluate the impact of personality disorders and traits on the clinical characteristics of women with BN at pre-treatment and at one, three or five-year follow-up assessments.

The second sample was the Binge Eating Psychotherapy (BEP) study (Joyce, McIntosh, Jordan, McKenzie, Carter, & Frampton, 2003 - ongoing). In this study women were recruited for a randomised clinical trial of three different psychotherapies for BN and BED. The trial compared standard CBT with two variations of CBT - schema therapy or a nutrition and appetite focused cognitive behaviour therapy (Latner & Wilson, 2000; McIntosh, Jordan, Carter, Latner, & Wallace, 2007). This thesis utilised personality data from the BEP study to examine clinical differences between BN and BED in relation to personality comorbidity. The impact of personality disorders and traits on outcome is not examined for the BEP sample as this study is still actively recruiting participants.

1.4 THESIS AIMS

This thesis will employ several different models to assess personality and to examine the impact of personality on clinical characteristics at pre-treatment and outcome, assessed at one, three or five years in a sample of women with BN. Using the second sample, this thesis addresses personality functioning in relation to clinical characteristics in those with a BN or BED diagnoses and it makes comparisons between the groups.
The main aims of this thesis are:

1. To investigate the impact of personality traits and personality disorders on clinical characteristics at pre-treatment and long-term outcome in women with BN using categorical and dimensional methods

2. To investigate the impact of personality traits and personality disorders on clinical characteristics in women with BED compared to women with BN at pre-treatment

3. To compare personality profiles between women with BN and BED at pre-treatment and assess similarities and differences between the two eating disorder groups, in comparison to both psychiatric control and a healthy control groups
PART 2

PERSONALITY AND PERSONALITY DISORDERS

Chapter 2: Personality and personality disorders in general

2.1 OVERVIEW

This chapter provides an overview of the concepts of personality and personality disorders. A brief historical overview of personality with a focus on three major perspectives within personality theory (trait, biological and psychodynamic) will be presented. The concept of personality disorders is reviewed and the relationships between personality traits and personality disorders are explored. Finally, the literature on how personality and personality disorders impact in general on Axis I disorders is reviewed.

2.2 PERSONALITY

2.2.1 The history of personality

The origins of the word ‘personality’ are Latin, derived from the term ‘persona’ which represents a theatrical mask (Millon, Millon, Grossman, & Meagher, 2004). Although this suggests an attempt to disguise the appearance and identity of the character, persona was actually used to reveal the inner traits that typified that character (Millon et al., 2004).
The development of the concept of personality can be viewed in three historical phases: 1) the literary and philosophical phase, in which insight into personality was provided by the personal beliefs of playwrights and philosophers, 2) the proto-clinical phase which involved the organised observation and theorising of abnormal or “sick” behaviour, and 3) the quantitative and experimental phase, in which scientific study developed theories based on behavioural measurements (Cattell, 2009). Throughout these periods, numerous personality theories developed, such as biological, cognitive, humanistic, learning, psychodynamic and trait theories (Corr & Matthews, 2009). Corr and Matthews have presented a useful overview of key contributors in the table produced below (Corr & Matthews, 2009).

Table 2.1: Major perspectives in personality

<table>
<thead>
<tr>
<th>Perspective</th>
<th>Major concepts</th>
<th>Contributors</th>
</tr>
</thead>
<tbody>
<tr>
<td>Biological</td>
<td>Temperament, evolution, adaptation, altruism, sexual jealousy, heredity, neurotransmitter pathways, cerebral hemispheric function</td>
<td>Buss, Eysenck, Gray, Cloninger, Kagan</td>
</tr>
<tr>
<td>Cognitive</td>
<td>Expectancy, self-efficacy, outcome expectation, schema, cognitive person variable, personal construct, reciprocal determinism, modelling, constructive alternativism, life narrative</td>
<td>Mischel, Bandura, Kelly, Beck</td>
</tr>
<tr>
<td>Humanistic</td>
<td>Self-actualisation, creativity, flow, spirituality, personal responsibility, freedom, choice, openness to experience, unconditional positive regard, acceptance, empathy, real self, hierarchy of needs, peak experience, positive psychology</td>
<td>Maslow, Rogers, Seligman, Csikszentmihalyi</td>
</tr>
<tr>
<td>Perspective</td>
<td>Major concepts</td>
<td>Contributors</td>
</tr>
<tr>
<td>-------------------</td>
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</tr>
</tbody>
</table>
| Learning          | Reinforcement, punishment, stimulus, response, conditioning, extinction, shaping
                   | discrimination learning, generalisation, situation, act frequency, basic behaviour
                   | repertoire, labelling, gradients of approach and avoidance                   | Skinner            |
|                   |                                                                               | Staats             |
|                   |                                                                               | Dollard            |
|                   |                                                                               | Miller             |
| Psychodynamic     | Libido, conflict, id, ego, superego, defence mechanisms, oedipal conflict, fixation,
                   | repression, attachment, object-relations                                     | Freud              |
|                   |                                                                               | Jung               |
|                   |                                                                               | Adler              |
|                   |                                                                               | Erikson            |
|                   |                                                                               | Horney             |
|                   |                                                                               | Klein              |
|                   |                                                                               | Sullivan           |
|                   |                                                                               | Chodorow           |
|                   |                                                                               | Westen             |
|                   |                                                                               | Lohut              |
|                   |                                                                               | Kernberg           |
| Trait             | Trait, type facet, factors, neuroticism, emotional stability, extraversion    | Allport            |
|                   |                                                                               | Cattell            |
|                   |                                                                               | McCrae and Costa   |

This thesis will focus on aspects of trait, biological and psychodynamic perspectives.

2.2.2 Trait perspectives of personality

The concept of personality traits has existed since classical times although it has often been used interchangeably with the terms ‘temperament’ and ‘character,’ (Corr & Matthews, 2009). The concept of temperament was described in the second century by the physician Galen, who identified four categories, ‘melancholic’ (low mood), ‘choleric’ (anger), ‘phlegmatic’ (stolid calmness), and ‘sanguine’ (optimism and confidence), based on bodily humours (Kagan, 1998; Matthews, Deary, & Whiteman, 2009; Stelmack & Stalikas, 1991). In the last century, trait theories of personality have been largely influenced by the work of Gordon Allport and H.S. Odbert (1936),

Allport (1937) had a causal view of personality traits and believed that they were responsible for understanding motivation and adaptive behaviours of an individual (Allport, 1937 cited in Corr & Matthews, 2009). One of the earlier projects by Allport and Odbert identified 4500 words from the dictionary describing personality traits and categorised these into four levels of traits. These were: central traits - basic characteristics that form the foundation of personality; secondary traits - appearing only in certain situations and sometimes associated with an individual’s preference or attitude; common traits - pertaining to culture; cardinal traits - dominant in an individual’s life such that these traits might be strongly recognised in them (Allport, 1936 cited in Matthews et al., 2009).

In 1946, Cattell used factor analysis to reduce Allport and Odbert’s 4500 personality traits to 16 factors representing key personality traits (Cattell, 1946 cited in John, Robins, & Pervin, 2008). These were: warmth, reasoning, emotional stability, dominance, liveliness, rule consciousness, social boldness, sensitivity, vigilance, abstractedness, privateness, apprehension, openness to change, self-reliance, perfectionism and tension (Boyle, Matthews, & Saklofske, 2008). When Cattell further factor-analysed the 16 primary traits, he identified five broad “second-order” or global factors which he saw as providing meaning and structure for the primary traits (Boyle et al., 2008). Recently, a similar set of five personality traits have been identified by Costa and McCrae’s five-factor model (Costa, 1992 and McCrae, 1992 cited in Boyle et al., 2008).
The five-factor model was developed by Costa and McCrae in 1992 and was partly based on theorising as well as statistical concerns about other models (Matthews et al., 2009). The dimensions in the five-factor model of personality are labelled neuroticism, extraversion, openness, agreeableness and conscientiousness (McCrae & John, 1992). Neuroticism is the tendency for an individual to feel distress and its cognitive and behavioural consequences; extraversion is characterised by positive emotionality and sociability; openness is the need for variety and experience; agreeableness is the compassionate and nurturing aspect of humanity; conscientiousness is a combination of restraining impulsive behaviours and diligence (McCrae & John, 1992). These dimensions were not explicitly related to psychiatric concepts nor did they have prior bases in biological theory. However, the dimensions of the five-factor model have since been shown to be have moderate heritability and as such contribute to research on bridging the gap between behavioural phenotypes and expressions of genotypes (Matthews et al., 2009).

2.2.3 Biological perspectives of personality

Biological theories of personality began to dominate research in the latter part of the twentieth century. The origins of this research began with the observation that there was greater similarity in behavioural phenotypes among those who also shared a genetic similarity (Munafò, 2009). A major focus within the biological perspective is the investigation into heritable genes that influence psychological characteristics including personality (Munafò, 2009).

The work of Hans Eysenck (1967) and Jeffrey Gray (1970) influenced subsequent research emphasising the role of individual differences in the genes involved in brain
development (polymorphisms) that drive personality variation (Corr & Matthews, 2009). According to Eysenck’s “Big Three” model, these are expressed in traits such as extraversion, neuroticism and psychoticism. The extraversion and neuroticism constructs were the basis for Costa and McCrae’s higher-order dimensions, however, the psychoticism construct was controversial due to validity concerns (Bouchard & McGue, 2003). Psychoticism was added to Eysenck’s model in 1970.

Twin studies and family studies indicate a strong genetic influence on extraversion, neuroticism and psychoticism, accounting for up to half of the phenotypic variance and heritability (Corr & Matthews, 2009; Eysenck, 1990; Floderus-Myrhed, Pedersen, & Rasmuson, 1980; Ivkovic, Vitart, Rudan, Janicijevic, Smolej-Narancic, Skaric-Juric, Barbalic, Polasek, Kolcic, Biloglav, Visscher, Hayward, Hastie, Anderson, Campbell, Wright, Rudan, & Deary, 2007). There is still debate regarding specific genetic factors that may potentially influence personality. For example, Lesch, Bengel, Heils, Sabol, Greenberg, Petri, Benjamin, Muller, Hamer, & Murphy (1996) found that neuroticism was higher in individuals with at least one short copy of the serotonin transporter (5-HTTLPR) allele compared to individuals who carried two long copies of the 5-HTTLPR allele. This has also been found in impulsivity and harm avoidance (Steiger, Joober, Israel, Young, Ng Ying Kin, Gauvin, Bruce, Joncas & Torkaman-Zehi, 2005; Szekely, Ronai, Nemoda, Kolmann, Gervai & Sasvari-Szekely, 2004) Unfortunately, other studies have failed to replicate this finding and the influence of the serotonin transporter on personality remains unknown (Bouchard & McGue, 2003).

An alternative “normative” model has more recently been developed by Cloninger, with the aim of understanding the genetic structure of personality (Cloninger, Przybeck, &
Svrakic, 1993). The psychobiological model of personality initially began with considering temperament in 1986 (Cloninger, 1986) and then character was added in 1993 (Cloninger et al., 1993). Temperament encompasses four dimensions of personality (novelty-seeking, harm-avoidance, reward dependence and persistence) that involve automatic responses to perceptual stimuli. Robert Cloninger stipulates that these are “heritable, manifest early in life, and apparently involve preconceptual or unconscious biases in learning” (Cloninger et al., 1993, p. 977). Character encompasses three dimensions of personality (self-directedness, cooperativeness and self-transcendence) that influence our responses based on biases related to our self-concept. For example, how our automatic behaviour relates to who and what we are and our place in the world (Cloninger et al., 1993).

A large twin study found heritability of the four temperament dimensions ranged from 50% to 65% (Heath, Cloninger, & Martin, 1994). Although character was initially conceived of as less heritable, all character subscales have been found to have a substantial genetic contribution (Ando, Suzuki, Yamagata, Kijima, Maekawa, Ono, & Jang, 2004). Once again, there is some debate regarding the specific genetic factors that have been identified as potentially influencing personality. For example, a significant association has been found between the dopamine D4 receptor (DRD4) and novelty-seeking (Benjamin, Li, Patterson, Greenberg, Murphy, & Hamer, 1996; Ebstein, Novick, Umansky, Priel, Osher, Blaine, Bennett, Nemanov, Katz, & Belmaker, 1996). Novelty-seeking was higher in individuals with longer alleles (usually the 7-repeat allele) than those with shorter alleles (usually the 4-repeat allele). This finding has been replicated in some studies (Lee, Lee, Kim, Kim, Lee, Jung, Suh, & Kim, 2003; Munafo, Yalcin, Willis-Owen, & Flint, 2008; Schinka, Letsch, & Crawford, 2002) but not other
studies (Kluger, Siegfried, & Ebstein, 2002; Lynn, Lubke, Yang, McCracken, McGough, Ishii, Loo, Nelson, & Smalley, 2005; Sullivan, Fifield, Kennedy, Mulder, Sellman, & Joyce, 1998).

Cloninger’s psychobiological model of temperament and character is still evolving. More recent developments (beyond the scope of this thesis) explore relationships between temperament, character and wellbeing (Cloninger, 2006).

2.2.4 Psychodynamic perspectives of personality

Although the psychodynamic theory of personality is most identified as beginning with Sigmund Freud, German philosophers (Gottihilf Heinrich von Schubert, Carl Gustav Carus and Arthur Schoepenhauer) preceded the development of psychodynamic theories and are likely to have influenced many of Freud’s theories in the early nineteenth century (Ewen, 2003). Von Schubert proposed a tripartite theory similar to Freud’s id, ego and superego theory. Carus argued for a focus on the unconscious as a key to knowledge of the conscious life and Schoepenhaur formulated theories on ideas of will and intellect which related in part to Freud’s ideas of the id, ego and repression (Ewen, 2003). Furthermore, the term “id” was first developed in the latter part of the nineteenth century by Nietzsche to describe the self-destructive and self-deceptive nature of human beings, inhibition of threatening thoughts and unmasking of unconscious thoughts (Ewen, 2003).

Psychoanalysis was developed by Freud beginning in the 1890s and was defined by 1) theory of the mind or personality, 2) a method of investigation of unconscious processes and 3) a method of treatment (Westen, Gabbard, & Ortigo, 2008). Freud’s theory emphasised unconscious processes, conflicts, defenses, the Oedipus complex (the desire
to possess the parent of the opposite sex) and the centrality of sexual drive in the
development of personality and neurosis (Westen et al., 2008). Personality was seen to
result from conflicting wishes and goals and was explained through the concepts of the
id (unconscious, instinctual behaviours), ego (reality principle) and superego
(internalised experience of being punished or rewarded; Quintar, Lane, & Goeltz, 2004).
Whilst a number of psychologists accepted some of Freud’s theory, other aspects were
rejected and lead to the development of a broader perspective of psychodynamic theory
(Westen et al., 2008).

Carl Jung was originally a follower of Freud’s theory however he disagreed with several
of the important aspects and eventually detached to establish his own theory. Jung
identified introversion and extraversion as major features in personality and believed
that psychopathology occurred when personality became “one-sided” for example,
when important aspects of personality failed to develop or when there was an
overemphasis on aspects that were not true to our intrinsic nature (Ewen, 2003). At a
later stage, Erik Erikson revisited Freud’s theory and concluded that personality is
influenced to a greater extent by a child’s relationship with his/her parents than by
sexuality or instincts and personality constantly develops through a series of
psychosocial stages from infancy to old age (Ewen, 2003). Despite broader
psychodynamic principles evolving over time, psychodynamic theory continues to be
criticised for a lack of scientific progress.

In more recent times, psychodynamic theory has attempted to find common ground with
other theories such as cognitive, social and developmental paradigms, thereby
strengthening ties with mainstream psychology (Bornstein, 2009). There has been a
shift from the conflict model of illness previously seen in psychodynamic psychiatry to a deficit model of illness. This model implies that patients have a weakened representation of self and others (Gabbard, 2005). According to contemporary psychodynamic principles, unconscious conflicts, deficits and distortions are integrated with a biological understanding of illness (Gabbard, 2005).

2.3 PERSONALITY DISORDERS

2.3.1 The history of personality disorders

Personality disorders are diagnosed within a medical model of mental disorders using a categorical approach in which lists of symptoms are judged to be present or not.

The first diagnostic categories emerged in psychiatry as a result of work by Philippe Pinel (1809) and J. C. Prichard (1835) describing insanity without delusions (manie sans délire) and “moral insanity” (Pinel, 1809 and Prichard, 1835 cited in Livesley, 2001b). The term “moral insanity” referred to distorted feelings, habits, morals and impulses in the absence of intellectual or reasoning impairments and without the presence of hallucinations or delusions. This definition covered a wide range of disorders, including mood disorders, personality disorders and clinical syndromes such as mania (Hervé & Yuille, 2007; Livesley, 2001b). It has been suggested that through the development of descriptive psychopathology in relation to mood disorders, Pritchard was able to further differentiate between transient symptoms and more enduring characteristics (Berrios, 1993; Livesley, 2001b). This distinction contributed to the emergence of a separate category for mental disorders including personality disorders. Although moral insanity continued to be developed by many throughout the nineteenth
century, it was Kurt Schneider’s work (1923 and 1950) that had a significant impact on the overall model of personality disorders (Schneider, 1923 and Schneider, 1950 cited in Coolidge & Segal, 1998).

Schneider’s work entitled ‘Psychopathologic Personalities’ (1923) was one of first approaches to distinguish between abnormal and disordered personality (Schneider, 1923 cited in Coolidge & Segal, 1998). Abnormal personality was defined as “deviating from the average” and considered on the extreme end of the normal personality spectrum whereas psychopathologic personality was defined as ‘abnormal personality that is dysfunctional (Schneider, 1950 cited in Livesley, 2001b). Several other pivotal concepts emerged from Schneider’s taxonomy. Schneider’s model suggested that it was not necessary for what he termed ‘psychopathologic personalities’ to be antecedents for other mental disorders; psychopathologic personalities were likely developed in childhood and persist into adulthood; and there were ten types of psychopathologic personalities: hyperthymic, depressive, insecure, fanatical, attention-seeking, labile, explosive, weak-willed, asthenic, and affectionless personality (Coolidge & Segal, 1998; Livesley, 2001b; Schneider, 1950).

2.3.2 Personality disorder classification system

Over time, diverse concepts of personality psychopathology lead to confusion in attempting to classify personality dysfunction (Livesley, 2001b). The International Classification of Diseases, Injuries and Causes of Death (ICD) was originally developed in 1900. During the sixth revision (ICD-6) in 1948, it was modified to include a section for mental disorders for the first time. Several categories though, including many personality disorders, were excluded (First, Frances, & Pincus, 2004). This lead to
widespread dissatisfaction and limited international use. An alternative manual for mental disorders was produced in the United States in 1952 called the Diagnostic and Statistical Manual of Mental Disorder (DSM-I) with official diagnostic categories (including personality disorders) and a glossary of definitions of the categories (First et al., 2004).

Both the ICD and DSM were revised in the 1960s and 1970s. It was, however, the publication of the Diagnostic and Statistical Manual of Mental Disorder-Third Edition (DSM-III) that provided a fundamental shift in classifying mental disorders. In the DSM-III there was a shift in theory from a psychoanalytic perspective to an atheoretical perspective. For the first time, a multiaxial approach was introduced in which clinical syndromes (e.g., major depressive disorder, anxiety disorders, schizophrenia) were categorised as Axis I disorders and personality disorders and childhood problems were categorised as Axis II disorders (American Psychiatric Association, 1980). The new Axis II category included more thorough descriptions and specific criteria for the diagnoses of personality disorders. In addition to these changes, the DSM-III saw an emergence of structured and semi-structured clinical interviews that encompassed all Axis II disorders, for example, the Diagnostic Interview for Personality Disorders (DIPD, Zanarini, Frankenburg, Chauncey, & Gunderson, 1987), the Structured Clinical Interview for DSM-III-R Personality Disorders (SCID-II, Spitzer, Williams, & Gibbon, 1987) and the Personality Disorder Examination (PDE, Loranger, Susman, Oldham, & Russakoff, 1987). The use of standardised clinical interviews increased the reliability of diagnosis and enabled comparisons across studies (Zanarini et al., 1987).
The Diagnostic and Statistical Manual of Mental Disorder – Fourth Edition –Text Revision (DSM-IV-TR) and The International Classification of Diseases, Injuries and Causes of Death (ICD) – Tenth Edition (ICD-10) are the major categorical classification systems currently in use. The use of diagnostic criteria for personality disorders, although useful, does not address concepts of normal personality (Livesley, 2001a). In recent times, there have been efforts to integrate personality disorder and personality theory to normal and abnormal personality (Saulsman & Page, 2004).

2.4 HOW DO PERSONALITY TRAITS MAP ONTO PERSONALITY DISORDERS?

The rationale for mapping personality traits onto personality disorders is to reveal significant relationships between the two, and to establish that personality disorder categories are measuring what they are meant to, that is, pathological personality (Saulsman & Page, 2003). The bridging of personality traits and personality disorders has focused on the ability of models such as the five-factor model and the psychobiological model of temperament and character to accurately predict the presence of personality disorders.

In a meta-analytic review of the five-factor model and personality disorders, high neuroticism and low agreeableness findings were common across most personality disorders whereas low scores on extraversion and conscientiousness scales were unique to certain personality disorders (extraversion to schizoid, schizotypal and avoidant, conscientiousness to antisocial and borderline; Saulsman & Page, 2004). Findings in relation to the psychobiological model of temperament and character and personality disorders were that low self-directedness and cooperativeness had a strong inverse
association. The number of personality disorder symptoms and low reward dependence, high novelty-seeking and high harm avoidance differentiated Cluster A, B, and C respectively (Svrakic, Whitehead, Przybeck, & Cloninger, 1993). Despite these findings, the rationale for mapping personality traits onto personality disorders has been criticised for a lack of validity, reliability and utility of personality disorders in the first place (Saulsman & Page, 2003).

Future classification systems for personality disorders are likely to incorporate a dimensional approach to supplement rather than replace the categorical approach. Rounsaville et al. (2002, p. 13) states “There is a clear need for dimensional models to be developed and their utility compared with that of existing typologies in one or more limited fields, such as personality. If a dimensional system performs well and is acceptable to clinicians, it might be appropriate to explore dimensional approaches in other domains”. This implies that the Diagnostic and Statistical Manual of Mental Disorder-Fifth Edition (DSM-5) may use personality disorders as a test case for dimensional approaches in the diagnosis of mental disorders (Skodol & Bender, 2009).

Indeed, a hybrid model of personality and personality disorder assessment is under consideration for the DSM-5. This consists of: 1) an overall rating of personality functioning ranging from normal to severely impaired, 2) major personality (disorder) types described in a prototype, 3) personality traits on which the prototypes are based but that can be also be used in the absence of a personality disorder or in the presence of an atypical personality disorder that does not conform to the prototypes, 4) genetic criteria for personality disorders associated with severe impairments in self-differentiation and integration, and interpersonal relatedness, and 5) adaptive
functioning measures (Skodol & Bender, 2009). Mapping personality traits onto personality disorders remains an important step in evolving the current classification system towards a future diagnostic system combining categorical and dimensional approaches to obtain a clinically meaningful representation of an individual’s personality and/or personality disorder.

2.5 **Patterns and Clinical Significance of Personality Traits and Personality Disorders in Axis I Disorders**

Maladaptive personality traits and personality disorders are known to be elevated in those with Axis I psychiatric disorders. These elevations may be specific or non-specific to an Axis I disorder. Studies examining the clinical significance of personality traits and personality disorders (particularly borderline and antisocial personality disorders) in Axis I disorders have focused on personality profiles, functioning and impact on outcome of treatment for Axis I disorders. Reviewing the literature on personality comorbidity prevalence’s in other common psychiatric conditions provides a context for interpreting whether results found in these eating disorders samples are specific or non-specific.

2.5.1 **Major depressive disorder**

2.5.1.1 **Personality traits**

The five-factor model and the psychobiological model of temperament and character have been used to describe personality trait profiles in patients with major depressive disorder (MDD). On Cloninger’s Temperament and Character Inventory (TCI) there have been consistent findings of high harm avoidance and low self-directedness in
MDD (Celikel, Kose, Cumurcu, Erkorkmaz, Sayar, Borckardt, & Cloninger, 2009; Hansenne, Reggers, Pinto, Kjiri, Ajamier, & Anseaux, 1999; Marijnissen, Tuinier, Sijben, & Verhoeven, 2002; Smith, Duffy, Stewart, Muir, & Blackwood, 2005). Furthermore, in those with MDD, higher novelty-seeking and self-transcendence and lower reward dependence and cooperativeness have been identified compared to healthy controls (Nery, Hatch, Nicoletti, Monkul, Najt, Matsuo, Cloninger, & Soares, 2009). However, in that study, these differences in personality profiles were reported to be the result of acute mood states and comorbid anxiety disorders. In patients with remitted MDD, self-directedness was the only personality trait to remain significantly different from the healthy controls (Nery et al., 2009). Personality profiles of patients with MDD using the five-factor model have revealed higher neuroticism and lower extraversion in this group compared to normative means (Petersen, Bottonari, Alpert, Fava, & Nierenberg, 2001).

Personality traits have been examined in relation to their impact on Axis I disorders on both severity of symptoms and treatment outcome. A positive association has been found between harm avoidance and the intensity and number of episodes of MDD (Nery et al., 2009). Neuroticism has been reported to predict severity of depression (Petersen et al., 2001). Similarly, in a review of personality pathology and treatment outcome in MDD, higher neuroticism scores were highlighted as a consistent predictor of worse outcome (Mulder, 2002). In contrast, higher extraversion and openness to experience have been found to predict a better response to treatment for individuals with MDD (Quilty, De Fruyt, Rolland, Kennedy, Rouillon, & Bagby, 2008).
2.5.1.2 Personality disorders

Prevalences of personality disorders in inpatients and outpatients with MDD range widely from 20% to 85% (Corruble, Ginestet, & Guelfi, 1996; Zimmerman, Rothschild, & Chelminski, 2005). High prevalences of avoidant, borderline and dependent personality disorders have been associated with depressive disorders (Skodol, Stout, McGlashan, Grilo, Gunderson, Shea, Morey, Zanarini, Dyck, & Oldham, 1999). There is some debate on the impact of personality disorders on the functioning of patients with MDD. Some studies have found depressed patients with a personality disorder experience a poorer quality of social networks, worse employment status (Reich, 1990), emotional role limitations and poorer general health perceptions (Skodol, Grilo, Pagano, Bender, Gunderson, Shea, Yen, Zanarini, & McGlashan, 2005) compared to depressed patients with no personality disorder. These findings have been challenged in recent years by studies reporting no negative effect of a comorbid personality disorder on the functioning of patients with MDD. For instance, Brieger et al. (2002) found that patients with MDD and comorbid personality disorder were no more likely to have impaired psychosocial functioning, increased disability payments, a higher number of previous depressive episodes, longer inpatient treatment, lower general functioning or higher depression scores at the time of hospital discharge, than patients with MDD and no personality disorder (Brieger, Ehrt, Bloeink, & Marneros, 2002).

Regarding the impact of personality disorders on treatment outcomes, there have been longstanding assumptions in clinical psychiatry that comorbid personality disorders are associated with a poor outcome in MDD. Recent literature reviews on this topic remain inconclusive. A meta-analysis of studies using a categorical approach to personality
disorder diagnoses (personality disorder/no personality disorder) and outcome
(recovered/not recovered) found comorbid personality disorders with MDD doubled the
risk of a poor outcome compared with no personality disorder (Newton-Howes, Tyrer,
& Johnson, 2006). In contrast, reviews by Mulder (2002) and Kool et al., (2005) suggest
that the presence of personality disorder does not impact on the outcome in patients
receiving treatment for MDD (Kool, Schoevers, de Maat, Van, Molenaar, Vink, &
Dekker, 2007; Mulder, 2002). To further complicate our understanding of this
relationship, a study by Joyce, McKenzie, Carter, Rae, Luty, Frampton and Mulder
(2007) found personality disorders did not adversely affect treatment response to
depressed patients receiving CBT but did adversely affect treatment response for
patients receiving IPT. Further research examining mediating factors may be necessary
to better understand this issue but for now, conclusions on the effect of comorbid
personality disorders on MDD remain unclear.

2.5.2 Anxiety disorders

2.5.2.1 Personality traits

The literature on personality traits in anxiety disorders is broad and covers individual
anxiety disorders, anxiety disorders as a whole, or comorbid anxiety and depressive
disorders. A review by Bienvenu and Stein (2003) suggests that neuroticism appears
strongly associated with, though not specific to, anxiety disorders. Furthermore,
introversion appears more strongly associated with some anxiety disorders than others
(Bienvenu & Stein, 2003). Higher introversion has been identified as significantly
related to social phobia and agoraphobia but not panic disorder (Bienvenu, Nestadt,
Samuels, Costa, Howard, & Eaton, 2001). Using the TCI, patients with social phobia
were identified as having higher harm avoidance, lower persistence, self-directedness, cooperativeness and self-transcendence, than healthy controls (Marteinsdottir, Tillfors, Furmark, Anderberg, & Ekselius, 2003). Few differences in personality traits (using the NEO-Five-Factor Inventory) between subjects with anxiety disorders and MDDs have been found (Cuijpers, van Straten, & Donker, 2005).

Few studies have examined the impact of personality traits on functioning, symptoms or outcome in patients with anxiety disorders. Neuroticism and extraversion have been associated with greater comorbidity of mental disorders (Cuijpers et al., 2005). The likely implication of these findings is that an increased number of comorbid disorders may impair functioning and/or worsen symptoms of the anxiety disorder (Merikangas & Swanson, 2010). Of the few studies examining the impact of personality traits on outcome, low self-directedness and high neuroticism have been identified as potentially important variables influencing the treatment outcome of obsessive-compulsive disorders and anxiety disorders, more generally (Andrews, Creamer, Crino, Hunt, Lampe, & Page, 2003; Corchs, Corregiari, Ferrao, Takakura, Mathis, Lopes, Miguel, & Bernik, 2008).

### 2.5.2.2 Personality disorders

Prevalence rates of personality disorders among outpatients with anxiety disorders range from 35% to 47% (Kantojarvi, Veijola, Laksy, Jokelainen, Herva, Karvonen, Kokkonen, Jarvelin, & Joukamaa, 2006; Sanderson, Wetzler, Beck, & Betz, 1994). Those with anxiety disorders are most likely to have a Cluster C (anxious/fearful) personality disorder (Kantojarvi et al., 2006; Sanderson et al., 1994). There is a strong association between avoidant personality disorder and social phobia (Bienvenu & Stein,
Borderline personality disorder has been associated with post-traumatic stress disorder (Bienvenu & Stein, 2003; Gunderson & Sabo, 1993). Personality disorders may affect overall functioning and symptoms of those with anxiety disorders resulting in associations with more severe Axis I pathology (Dreessen, Arntz, Luttels, & Sallaerts, 1994), more initial severe anxiety symptoms, more symptom-related disability (Noyes, Reich, Christiansen, Suelzer, Pföhl, & Coryell, 1990), and higher treatment drop out rates (Reich, 2003). Findings of the effects of personality disorders on the outcome of anxiety disorders are mixed. A 6-year follow-up study of anxiety disorders in psychiatric outpatients suggests that the presence of borderline, obsessive-compulsive, paranoid, self-defeating or avoidant personality disorders predicted poor outcome (Alnaes & Torgersen, 1999). Furthermore, the presence of personality disorders increased the risk of chronicity and developing a new anxiety disorder (Alnaes & Torgersen, 1999). More recently, personality disorders have been found to lower the likelihood of remission in generalised anxiety disorder and social phobia but not panic disorder (Massion, Dyck, Shea, Phillips, Warshaw, & Keller, 2002; Yonkers, Dyck, Warshaw, & Keller, 2000). In contrast, a review of 15 best-evidence studies could not conclude that patients with a comorbid anxiety disorder and personality disorder responded less well to treatment (Dreessen & Arntz, 1998). As these findings suggest, conclusions about the impact of personality disorders on the outcome of anxiety disorders remain unresolved.
2.5.3 Substance use disorders

2.5.3.1 Personality traits

Substance use disorders, as a whole, appear to be associated with high neuroticism and novelty-seeking, and low agreeableness and conscientiousness (Ruiz, Pincus, & Schinka, 2008). Several personality traits specific to alcohol, drug and tobacco dependence symptoms have been identified by Ball et al. (2005; 2002). Alcohol dependence symptoms were predicted by extraversion and low openness to experience; drug dependence symptoms were predicted by low conscientiousness; and tobacco dependence symptoms were predicted by openness to experience and low conscientiousness (Grekin, Sher, & Wood, 2006). Disinhibitory personality traits (such as novelty-seeking, sensation-seeking, impulsivity and hostility/disagreeableness) were related to earlier age-of-onset, polydrug use, chronic/heavy use, substance dependence severity, violence, arrests, psychiatric symptoms and suicide attempts (Ball, 2005; Ball, 2002).

Little is published about the impact of personality traits on the outcome of substance use disorders. It has been reported that patients with disinhibitory personality traits drop-out from treatment early (Ball, 2005; Ball, 2002) and have a shorter time to relapse (Zikos, Gill, & Charney, 2010).

2.5.3.2 Personality disorders

Comorbidity between personality disorders and substance use disorders is approximately four times higher than the general population (Verheul, 2001). Prevalence rates range from 29% to 80% for inpatients and outpatients with substance
use disorders (Grant, Stinson, Dawson, Chou, Ruan, & Pickering, 2004; Skodol, Oldham, & Gallaher, 1999; Verheul, 2001). Antisocial, borderline and avoidant personality disorders are the most common disorders reported in individuals with substance use disorders (Ball, 2005; Ford, Gelernter, DeVoe, Zhang, Weiss, Brady, Farrer, & Kranzler, 2009).

The presence of a personality disorder comorbid with substance use disorders has been reported to have a significant impact upon symptoms and functioning of individuals with this comorbidity. Personality disorders and comorbid substance use disorders have been associated with greater involvement with illegal drugs, increased psychopathology, less life satisfaction, more impulsiveness, isolation and depression and more social disadvantage (incomplete schooling, having no qualifications, not working and receiving government benefits) compared to those without personality disorders (Moran, Coffey, Mann, Carlin, & Patton, 2006; Nace, Davis, & Gaspari, 1991). Antisocial personality disorder in an opiate abuse sample was associated with increased severity of alcohol, psychiatric and legal problems, and risky sexual behaviour (Ford et al., 2009). Borderline personality disorder in patients with substance use disorders has been associated with poorer overall mental health, committing more criminal acts and increased psychopathology (Darke, Ross, Williamson, & Teesson, 2005).

A review by Verheul (2001) summarising the impact of personality disorders on the outcome of substance use disorders, concluded that despite early opinions suggesting comorbid personality disorders predicted poor treatment response or outcome of substance abusers, many more recent studies have found that personality disorders were not a robust predictor of improvement, drop-out or motivation to change in patients with
substance use disorders (Verheul, 2001). However, other studies have also reported that individuals with substance use disorders and Axis II comorbidity have a short time to relapse following discharge (Thomas, Melchert, & Banken, 1999; Verheul, van den Brink, & Hartgers, 1998; Zikos et al., 2010). Antisocial and borderline personality disorders have been specifically linked to worse outcome at follow-up (Compton, Cottler, Jacobs, Ben-Abdallah, & Spitznagel, 2003; Haro, Mateu, Martinez-Raga, Valderrama, Castellano, & Cervera, 2004; Wolwer, Burtscheidt, Redner, Schwarz, & Gaebel, 2001).

2.6 CONCLUSIONS

The study of personality has evolved over centuries. Within the field of mental disorders, there has long been awareness of the relationship of more enduring personality traits with common Axis I conditions such as major depression, anxiety and substance use disorders. There is fairly consistent empirical evidence that the presence of a personality disorder is associated with more severe Axis I symptoms at pre-treatment across conditions. However, there is conflicting evidence regarding the impact of a personality disorder on outcome of treatment for Axis I conditions. Increasingly, normal and abnormal personality traits are being examined alongside personality disorders in relation to Axis I conditions and this may assist in clarifying some of the contradictory findings. A key focus of this thesis will be to examine personality traits and disorders in relation to clinical characteristics, global functioning and treatment outcome in women with eating disorders.
Chapter 3: Personality and personality disorders in eating disorders

3.1 Overview

This chapter extends the literature review of personality and personality disorders, addressing issues specific to eating disorders. The relationship between eating disorders and personality, as well as patterns of personality traits and disorders in eating disorders, will be reviewed to provide some context for comparisons between existing literature and findings from the BTS and BEP analyses presented in later chapters. Methodological issues associated with this comorbidity are examined.

3.2 The Relationship Between Eating Disorders and Personality

Understanding the relationship between eating disorders and personality is of high clinical relevance. Studying this relationship may identify risk factors, identify differences in eating disorder and other Axis I symptoms and identify whether there are differential treatment outcomes for those with or without a comorbid personality disorder.
3.2.1 Aetiology/vulnerability factors

3.2.1.1 Personality traits

Personality traits and eating disorders have been reported to be moderately heritable (Bulik, Sullivan, Wade, & Kendler, 2000; Cloninger et al., 1993; Farmer, Mahmood, Redman, Harris, Sadler, & McGuffin, 2003; Jang, McCrae, Angleitner, Riemann, & Livesley, 1998; Kendler, Maclean, Neale, Kessler, Heath, & Eaves, 1991). Specific personality traits associated with eating disorders include perfectionism, obsessive-compulsiveness, impulsivity, sensation-seeking and narcissism (Cassin & von Ranson, 2005). Perfectionism and obsessive-compulsive traits have been associated with increased risks of developing AN and BN (Anderluh, Tchanturia, Rabe-Hesketh, & Treasure, 2003b; Lilenfeld, Kaye, Greeno, Merikangas, Plotnicov, Pollice, Rao, Strober, Bulik, & Nagy, 1998; Tyrka, Waldron, Graber, & Brooks-Gunn, 2002). Impulsivity and sensation-seeking have been found to be associated with eating disorder behaviours such as binge eating and purging (Claes, Vandereyeken, & Vertommen, 2002; Diaz-Marsa, Carrasco, & Saiz, 2000; Steiger, Jabalpurwala, Champagne, & Stotland, 1997). Some studies have found a relationship between narcissism, pathological body image concerns and AN or BN (Lehoux & Howe, 2007; Steiger et al., 1997).

3.2.1.2 Personality disorders

Personality disorders in adolescence are considered a vulnerability factor for developing an eating disorder in the future. Rastam (1992) estimated that approximately two-thirds of a sample of adolescents with AN had a personality disorder prior to the onset of the eating disorder (Rastam, 1992). This risk may also continue into young adulthood. A further study showed that the presence of a personality disorder by the age of 22 years
has been associated with an increased risk of developing an eating disorder by the age of 33 (Johnson, 2006). Obsessive-compulsive personality disorder has been frequently singled out as a risk factor for developing AN (Sansone & Sansone, 2010b). The presence of Cluster B personality disorders (specifically borderline or histrionic) has been associated with the onset of binge eating and purging; depressive personality disorder has been associated with an elevated risk of dietary restriction; antisocial and schizotypal personality disorders have been associated with increased binge eating and obesity (Johnson, 2006). Cluster C personality disorders have been implicated as a significant contributor to the vulnerability of an eating disorder in a population-based twin study (Inceoglu, Franzen, Backmund, & Gerlinghoff, 2000).

3.2.2 Impact on symptoms and behaviours

3.2.2.1 Personality traits

Personality traits have been identified as a factor influencing the symptomatic expression of eating disorders. High neuroticism, low openness to experience and high agreeableness have been linked to more pathological eating disorder attitudes and behaviour factor scores on the Eating Disorder Examination-Questionnaire (EDE-Q; Tasca, Demidenko, Krysanski, Bissada, Illing, Gick, Weekes, & Balfour, 2009). Perfectionism has been associated with fasting and purging in undergraduate women (Forbush, Heatherton, & Keel, 2007). Low scores on the self-directedness trait has been identified as a predictor of increased severity of vomiting behaviours in women with BN (Abbate-Daga, Piero, Gramaglia, & Fassino, 2005). Finally, impulsive traits have been positively associated with bulimic symptoms (Diaz-Marsa et al., 2000) and treatment drop-out (Fassino, Abbate-Daga, Piero, Leombruni, & Rovera, 2003).
3.2.2.2 Personality disorders

Reports on the impact of personality disorders on eating disorders symptoms and attitudes have been mixed. Several studies have found Axis II psychopathology is related to greater body dissatisfaction (Bulik, Sullivan, Joyce, & Carter, 1995a), more severe binge eating (Picot & Lilienfeld, 2003; Wilfley et al., 2000a) and purging (Spindler, 2006). However, other studies found minimal or no difference in eating disorder behaviours (Steiger, Leung, Thibaudeau, Houle, & Ghadirian, 1993; Wonderlich & Mitchell, 1997).

Borderline personality disorder in BN is one of the most commonly studied combinations within this area. Findings on the impact of borderline personality disorder on eating disorder attitudes and behaviours have been mixed. Some studies have associated the presence of borderline personality disorder with increased duration of eating problems, increased vomiting frequency, increased laxative use and body dissatisfaction (Wonderlich, Swift, Slotnick, & Goodman, 1990). However, other studies challenge these findings and report that the impact of borderline personality disorder on eating behaviours and attitudes is minimal (Ben-Porath, Wisniewski, & Warren, 2009; Johnson et al., 1990; Steiger et al., 1993). The major effects of borderline personality disorder on those with eating disorders have been identified in relation to adverse effects on general psychiatric symptoms (Steiger & Stotland, 1996), psychosocial functioning, family environment, self-destructive behaviour (Johnson, Tobin, & Enright, 1989), increased feelings of ineffectiveness, increased disturbance in interoceptive awareness and greater levels of general psychopathology (Zeeck, Birindelli, Sandholz, Joos, Herzog, & Hartmann, 2007).
3.2.3 Treatment outcome

3.2.3.1 Personality traits

Higher self-directedness has been identified as a predictor of favourable outcome of BN at one-year follow-up (Bulik et al., 1998b). High harm avoidance and low self-directedness have been found in those with chronic AN (Karwautz, Troop, Rabe-Hesketh, Collier, & Treasure, 2003) in comparison to those in full recovery, partial recovery and controls (Bulik, Sullivan, Fear, & Pickering, 2000). Other personality traits that have been found to predict poor outcome in an eating disorder sample are high levels of impulsivity, maturity fears and perfectionism (Steiger & Bruce, 2004).

3.2.3.2 Personality disorders

The impact of personality disorders on eating disorder outcome is a highly debated topic within the literature. Some previous studies suggest that personality disorders negatively impact treatment outcome in an eating disorder sample (Johnson et al., 1990; Keel & Mitchell, 1997; Steiger & Stotland, 1996). However, other studies report that the course and outcome of eating disorders is not significantly influenced by the presence of a personality disorder (Bulik et al., 1998b; Grilo et al., 2003).

3.3 Patterns of Personality Traits and Personality Disorders in Eating Disorders

In a similar manner to other Axis I disorders, eating disorders are known to be associated with elevated maladaptive personality traits and disorders compared to healthy controls. This section reports the patterns of specific personality disorders
reported within AN, BN and BED. Eating disorder not otherwise specified will not be
reviewed here as there is very limited literature regarding personality patterns in this
heterogeneous group. Tables 3.1, 3.2, and 3.3 summarise prevalence rates in clinical
samples from studies published in the past decade.
Table 3.1: Personality disorder prevalence rates in women with AN 2000-2010

<table>
<thead>
<tr>
<th>First Author</th>
<th>Year</th>
<th>Eating disorder group</th>
<th>Measure</th>
<th>N</th>
<th>Any PD (%)</th>
<th>Av (%)</th>
<th>Bor (%)</th>
<th>Dep (%)</th>
<th>His (%)</th>
<th>OC (%)</th>
<th>Cluster A</th>
<th>Cluster B</th>
<th>Cluster C</th>
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<tbody>
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</table>

Notes: The most commonly occurring personality disorders within an eating disorder population are represented in this table. Percentages are rounded to the nearest whole number. Abbreviations: AN = Anorexia nervosa; AN-R = Anorexia nervosa restricting type; PD = Personality disorder; Av = Avoidant personality disorder; Bor = Borderline personality disorder; Dep = Dependent personality disorder; His = Histrionic personality disorder; OC = Obsessive-compulsive personality disorder; SCID-II = Structured Clinical Interview for DSM personality disorders; IPDE = International Personality Disorders Examination, PDQ-R = Personality Diagnostic Questionnaire Revised.
**Table 3.2: Personality disorder prevalence rates in women with BN 2000-2010**

<table>
<thead>
<tr>
<th>First Author</th>
<th>Year</th>
<th>Eating disorder group</th>
<th>Measure</th>
<th>N</th>
<th>Any PD (%)</th>
<th>Av (%)</th>
<th>Bor (%)</th>
<th>Dep (%)</th>
<th>His (%)</th>
<th>OC (%)</th>
<th>Cluster A</th>
<th>Cluster B</th>
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<tr>
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Notes: The most commonly occurring personality disorders within an eating disorder population are represented in this table. Percentages are rounded to the nearest whole number. Abbreviations: BN = Bulimia nervosa; PD = Personality disorder; Av = Avoidant personality disorder; Bor = Borderline personality disorder; Dep = Dependent personality disorder; His = Histrionic personality disorder; OC = Obsessive-compulsive personality disorder; SCID-II = Structured Clinical Interview for DSM personality disorders; IPDE = International Personality Disorders Examination, PDQ-R = Personality Diagnostic Questionnaire Revised.
Table 3.3: Personality disorder prevalence rates in women with BED 2000-2010

<table>
<thead>
<tr>
<th>First Author</th>
<th>Year</th>
<th>Eating disorder group</th>
<th>Measure</th>
<th>N</th>
<th>Any PD (%)</th>
<th>Av (%)</th>
<th>Bor (%)</th>
<th>Dep (%)</th>
<th>His (%)</th>
<th>OC (%)</th>
<th>Cluster A (%)</th>
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<tr>
<td>Van Hanswijck De Jonge</td>
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<td>IPDE</td>
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<td>1</td>
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</table>

Notes: The most commonly occurring personality disorders within an eating disorder population are represented in this table. Percentages are rounded to the nearest whole number. Abbreviations: BED = Binge Eating Disorder; PD = Personality disorder; Av = Avoidant personality disorder; Bor = Borderline personality disorder; Dep = Dependent personality disorder; His = Histrionic personality disorder; OC = Obsessive-compulsive personality disorder; SCID-II = Structured Clinical Interview for DSM personality disorders; IPDE = International Personality Disorders Examination, PDQ-R = Personality Diagnostic Questionnaire Revised.
3.3.1 Anorexia nervosa

3.3.1.1 Personality traits

Many studies have used the TCI to attempt to develop a profile of women with AN. Traits that have been found to characterise AN are high harm avoidance, low novelty-seeking, low self-directedness and low cooperativeness (Fassino, Abbate-Daga, Amianto, Leombruni, Boggio, & Rovera, 2002a; Klump, Bulik, Pollice, Halmi, Fichter, Berrettini, Devlin, Strober, Kaplan, Woodside, Treasure, Shabbout, Lilienfeld, Plotnicov, & Kaye, 2000). Furthermore, it has been hypothesised that temperament is a potential predictor of binge-purge status in AN (Klump et al., 2000). A differentiating characteristic between AN-R and AN-BP is the higher level of persistence demonstrated by those with AN-R (Cassin & von Ranson, 2005). High persistence has been associated with perfectionism, rigidity, obsessiveness and perseverance, which may help to maintain restrictive eating behaviours (Cassin & von Ranson, 2005). Personality traits for women with AN-BP are more similar to women with BN rather than AN-R (Klump et al., 2000; Rybakowski, Słojien, Zakrzewska, Hornowska, & Rajewski, 2004).

3.3.1.2 Personality disorders

Prevalence rates for personality disorders in AN-R generally range from 20% to 25% (Herzog et al., 1992; Maranon, Echeburua, & Grijalvo, 2004; Matsunaga, Kaye, McConaha, Plotnicov, Pollice, & Rao, 2000). A summary of the empirical literature reveals the most frequently occurring personality disorders in this subgroup are: obsessive-compulsive (22%), avoidant (19%), borderline (10%) and dependent (10%) personality disorders (Jordan et al., 2008; Sansone et al., 2005).
Prevalence of personality disorders are increased in those with AN-BP, ranging from 35% to 80% (Herzog et al., 1992; Maranon et al., 2004; Matsunaga et al., 2000). The most frequently occurring personality disorders in this subgroup are: borderline (25%), avoidant (15%), dependent (15%) and histrionic (10%) personality disorders (Sansone et al., 2005).

3.3.2 Bulimia nervosa

3.3.2.1 Personality traits

BN has been characterised as being associated with high novelty-seeking, high harm avoidance, low self-directedness and low cooperativeness (Diaz-Marsa et al., 2000; Fassino et al., 2002a). Although AN and BN appear to share many of the same personality traits, there are more specific differences with elevated novelty-seeking and low cooperativeness and low self-directedness in BN compared to AN (Fassino et al., 2002a).

Other personality traits linked to BN include higher stress reaction (Peterson, Thuras, Ackard, Mitchell, Berg, Sandager, Wonderlich, Pederson, & Crow, 2010), obsessionality, ineffectiveness, interpersonal distrust, impulsivity and affective instability (Grilo, 2002). These characteristics may contribute to the maintenance of binge eating and purging behaviours (Cassin & von Ranson, 2005).

3.3.2.2 Personality disorders

Research on comorbidity between BN and personality disorders is generally with much larger samples than found in the AN literature. Among these studies, prevalence rates of personality disorders in BN range from 21% to 67% (Herzog et al., 1992; Maranon et al., 2004).
al., 2004; Matsunaga et al., 2000). The most frequently occurring personality disorders in this subgroup are: borderline (28%), dependent, histrionic and avoidant (all with rates around 20%; Sansone, Levitt & Sansone cited in Sansone & Levitt, 2005b). Although Cluster B is reportedly the most common cluster of personality disorders in BN, many studies report similar or increased levels of Cluster C in this eating disorder subgroup (Godt, 2002; Lilenfeld et al., 1998; Matsunaga, Kiriike, Nagata, & Yamagami, 1998).

3.3.3 Binge eating disorder

3.3.3.1 Personality traits

Women with BED have been reported to have lower self-directedness (Fassino, Leombruni, Piero, Daga, Amianto, Rovera, & Rovera, 2002b), higher harm avoidance and lower positive emotionality compared to obese controls without BED and normal-weight controls (Peterson et al., 2010). However, in that study, reanalyses with depression as a covariate excluded positive emotionality as a significant factor (Peterson et al., 2010).

3.3.3.2 Personality disorders

The literature on comorbidity in BED has increased in recent years. The rates of personality disorders in BED range from 20% to 37% (Specker, de Zwaan, Raymond, & Mitchell, 1994; Telch & Stice, 1998; Wilfley et al., 2000a). The most frequently occurring personality disorders are: obsessive-compulsive (15%), avoidant and borderline (both around 12%; Sansone et al., 2005). Interestingly, several studies have reported a high prevalence of Cluster A personality disorders in BED samples (Raymond, Mussell, Mitchell, de Zwaan, & Crosby, 1995; Specker et al., 1994).
3.4 Methodological Issues in Assessing Personality and Personality Disorders in an Eating Disorder Population

Many methodological issues are present in the literature relating to personality and eating disorders. In most cases however, these issues relate to the personality and personality disorder literature, and to the broader comorbidity literature although there are a few specific concerns in relation to eating disorders. General issues include: problems with the construct of personality disorders, problems with personality disorder criteria, problems with diagnostic stability, problems with personality measures, problems with the timing of the assessment and problems with samples and comparison groups (Lilenfeld, 2006; Sansone & Levitt, 2005b). These general problems will be briefly reviewed followed by more specific difficulties relative to an eating disorder population such as crossover between eating disorder subtypes, age-of-onset and timing of the assessment, state effects resulting from the eating disorder and misrepresentation in self-report.

3.4.1 Problems with the construct of personality disorders

The assessment of personality within a clinical sample is frequently done using the DSM classification system. The use of a categorical system with some common symptoms often results in diagnostic overlap (Trull & Durrett, 2005). This suggests that the construct and classification of personality disorders is problematic. One way to minimise this problem is by replacing or supplementing the categorical model with a dimensional model of personality pathology (Livesley, 1998; Morey, Hopwood, Gunderson, Skodol, Shea, Yen, Stout, Zanarini, Grilo, Sanislow, & McGlashan, 2007;
Trull & Durrett, 2005). A dimensional approach attempts to measure personality features as continuous rather than discrete entities (Tomotake & Ohmori, 2002). A major advantage of dimensional models is the improved reliability and validity in comparison to the categorical model (Wonderlich & Mitchell, 2001).

3.4.2 Problems with personality disorder criteria

Livesley (1998) describes the lack of theoretical and empirical rationales for personality disorder categories as a flaw of the current classification system, which he describes as “arbitrary” (Livesley, 1998, p.138). The atheoretical approach in the DSM was formulated so it could be used by clinicians from different theoretical perspectives (American Psychiatric Association, 1987). Whilst it has been successful in appealing to a wide range of clinicians, this decision to remain atheoretical has been criticised for constraining scientific research (Follette & Houts, 1996).

The current Axis II personality disorder classification system is not exhaustive and there are many cases that fall into the ‘personality disorder not otherwise specified’ category (Livesley, 1998). Some research has shown this to be one of the most commonly occurring personality disorders with rates from 17% to 49% (Verheul & Widiger, 2004; Wilberg, Hummelen, Pedersen, & Karterud, 2008). It has been suggested that if the ‘personality disorder not otherwise specified’ category is to be retained in the DSM-IV-TR, then more specific criteria in the assessment of this disorder is warranted (Verheul, Bartak, & Widiger, 2007). An example of this is the subcategories that have been used with other ‘not otherwise specified’ diagnoses like EDNOS.

Minimal attention is also given to DSM subthreshold diagnoses. A study by Westen and Arkowitz-Westen (1998) revealed that the presence of clinically significant personality
pathology that did not reach Axis II diagnosis was as high as 61% (Westen & Arkowitz-Westen, 1998). This has contributed to a renewed interest in dimensional models of personality disorder classification which had been accorded less prominence with the development of the DSM categories (Huprich & Bornstein, 2007).

3.4.3 Problems with diagnostic stability

The literature shows that personality disorder diagnoses have low to moderate stability with many participants no longer meeting criteria for a personality disorder after treatment for an Axis I disorder (Ferro, Klein, Schwartz, Kasch, & Leader, 1998; Grilo & McGlashan, 1999). An example of this is the finding by Ames-Frankel that personality disorders decrease when eating disorders are treated (Ames-Frankel, Devlin, Walsh, Strasser, Sadik, Oldham & Roose, 1992). This ‘recovery’ from personality disorders may be the result of overdiagnosis at presentation, Axis I disorders affecting personality features and/or the effects of psychotherapy. In any scenario, the change in diagnosis over a short period of time is at odds with the supposed enduring nature of the categorical construct of personality disorders. Dimensional measures of personality demonstrate improved stability compared to categorical measures (Clark, 2007; Durbin & Klein, 2006). Methodological issues such as small sample sizes, high attrition rates, limited or no use of structured interviews diagnosing personality disorders in earlier studies and lack of inter-rater reliability (Shea, Stout, Gunderson, Morey, Grilo, McGlashan, Skodol, Dyck, Zanarini, & Keller, 2002), make it difficult to compare studies in order to provide a clearer understanding on the stability of personality disorders.
3.4.4  Problems with measures

The comparison of personality traits across studies is hindered by the wide range of measures used. Self-report measures of both normal and abnormal personality traits are used but some may exclude personality constructs important to those with an eating disorder such as perfectionism and impulsivity (Sansone & Levitt, 2005b).

Self-report questionnaires and semi-structured interviews are the usual measures for diagnosing personality disorders. Unfortunately, reliability and validity of the personality disorders classification system is only marginally satisfactory (Kirk, 1994; Tyrer, Coombs, Ibrahimi, Mathilakath, Bajaj, Ranger, Rao, & Din, 2007). Modest levels of agreement across different interviews have been established (Lobbestael, Leurgans, & Arntz, 2010; O'Boyle & Self, 1990) however, discriminant validity is weak for DSM-IV personality disorders. This suggests that high levels of comorbidity across Axis II diagnoses will continue (Blais & Norman, 1997).

3.4.5  Problems with the timing of assessment

The clinical assessment of personality traits and disorders is based on the assumption that personality is stable and enduring over time (American Psychiatric Association, 1994). Most studies report pre-treatment data of individuals presenting for treatment of an Axis I condition. Concern has been raised about state effects (occurring as a result of the Axis I disorder) influencing responses given during the personality assessment (Vitousek & Stumpf, 2005). For example, cognitive research has shown that patients with depression may experience distortions in their interpretation of experience due to a biased effect influencing information retrieval (Beck, 2008; Madigan & Bollenbach,
This may lead to an impression of more severe personality pathology and thus an overdiagnosis of personality disorders.

3.4.6 Problems with samples and comparison groups

The elevated prevalences of personality pathology seen in research trials may be attributable in part to Berkson’s bias, which suggests that individuals with two disorders are more likely to enter the health care system for treatment of either disorder (Berkson, 1946). This may account for some of the difference in rates of comorbidity between clinical patients and community samples (Galbaud du Fort, Newman, & Bland, 1993). Different rates of personality disorders are evident between outpatient and inpatient samples (Marinangeli, Butti, Scinto, Di Cicco, Petruazzi, Daneluzzo, & Rossi, 2000; Zimmerman et al., 2005). This sampling bias restricts comparisons among studies.

Appropriate comparison groups are lacking in many studies. Although many studies report results for patients, the absence of a comparison group limits the interpretation of these findings. Alternatively, some studies use a “healthy normal” comparison group that excludes any pathology. The differences found between such “ultra healthy normal” control groups and disordered groups fail to clarify the associations we are attempting to understand. It has been suggested that the more appropriate comparison is with another psychiatric control group (Vitousek & Stumpf cited in Sansone & Levitt, 2005b).

In addition to these limitations, there are specific difficulties in assessing personality and personality disorders within an eating disorder population. These specific issues (discussed below) are: crossover between eating disorder subtypes, age-of-onset at the time of assessment, state effects resulting from the eating disorder and possible
misrepresentations in self-report as a feature of the eating disorder (Vitousek & Stumpf, 2005).

3.4.7 Crossover between eating disorder subtypes

The stability of individual eating disorder diagnoses is relatively low, with approximately one-third to two-thirds of participants changing diagnoses (Eddy, Dorer, Franko, Tahlani, Thompson-Brenner, & Herzog, 2008; Milos et al., 2005; Tozzi, Thornton, Klump, Fichter, Halmi, Kaplan, Strober, Woodside, Crow, Mitchell, Rotondo, Mauri, Cassano, Keel, Plotnicov, Pollice, Lilienfeld, Berrettini, Bulik, & Kaye, 2005). This makes it difficult to ascertain if personality traits influence the way in which eating disorder symptoms are expressed. Recent research suggests that the use of personality trait variables is helpful in distinguishing meaningful eating disorder subgroups within diagnostic categories (Keel, Fichter, Quadflieg, Bulik, Baxter, Thornton, Halmi, Kaplan, Strober, Woodside, Crow, Mitchell, Rotondo, Mauri, Cassano, Treasure, Goldman, Berrettini, & Kaye, 2004).

3.4.8 Age-of-onset and assessment

The onset of eating disorder symptoms usually occurs in adolescence however, assessment of a personality disorder during this time is contentious. The DSM-IV does not rule out the possibility of personality disorders being diagnosed under the age of 18 in the “unusual instances in which the individual’s particular maladaptive personality traits appear to be pervasive, persistent, and unlikely to be limited to a particular developmental stage or an episode of an Axis I disorder” (American Psychiatric Association, 1994, p.631). The validity of a personality disorder diagnosis in adolescents has been questioned by some researchers due to developmental processes.
that result in frequent changes within adolescents, their relationships and their environment (Bleiberg, 1994). Bleiberg (1994) states that this makes it difficult or impossible to meet the criteria of an ‘enduring pattern which is inflexible’ (American Psychiatric Association, 1994; Bleiberg, 1994). In contrast, other clinical researchers and personality theorists suggest that personality pathology is evident in childhood or adolescence and identifying this earlier may help to limit dysfunctional patterns that develop over time (Kernberg & Shapiro, 1990; Miller, Muehlenkamp, & Jacobson, 2008). Due to the potentially changeable nature of personality symptoms during adolescence, it has been suggested that that a dimensional approach may be a more useful classification system as it better accounts for developmental variability and heterogeneity within this population (Miller et al., 2008).

3.4.9 State effects resulting from the eating disorder

The psychological effects of starvation are well documented within the literature (Fessler, 2002; Hagan, Tomaka, & Moss, 2000; Polivy, 1996) and are of special consideration within the eating disorder field. A landmark study ‘The Minnesota Semi-Starvation Experiment’ by Keys in 1950, examined the restriction of caloric intake in 36 physically and psychologically healthy men for six months (Keys, Brozek, Henschel, Mickelson, & Taylor, 1950). During this time they lost 25% of their body weight and found that this resulted in dramatic physical, psychological and social changes over time (Keys et al., 1950). Severe emotional distress that interfered with functioning, depression, occasional elation, irritability, impulsivity, outbursts of anger, anxiety, and apathy were increased in participants over nine weeks of semi-starvation (Keys et al., 1950). All subscales on the Minnesota Multiphasic Personality Inventory (MMPI;
Hathaway & McKinley, 1940) were elevated indicating severe personality disturbance (Keys et al., 1950).

Studies of the effects of dieting in obese subjects with no history of psychiatric disorders also report similar findings (Robinson & Winnik, 1973; Swanson & Dinello, 1970). Increased impulsivity, exhibitionism, anger and aggression were found in those subjects, illustrating the importance of these state effects on personality across all eating disorder subgroups (Fessler, 2002).

3.4.10 Misrepresentations in self-report

Denial and minimisation of eating disorder symptoms is relatively common in those with AN (Couturier & Lock, 2006; Viglione, Muratori, Maestro, Brunori, & Picchi, 2006). In addition, adolescents with eating disorders have been found to deny or under report lower levels of internalising disorders such as depression and anxiety (Salbach-Andrae, Klinkowski, Lenz, Pfeiffer, Lehmkuhl, & Ehrlich, 2008; Viglione et al., 2006). Some authors have proposed that severe restriction might block emotions so this apparent under-reporting may not be a misrepresentation (Corstorphine, 2006; Harrison, Sullivan, Tchanturia, & Treasure, 2009). With weight gain some patients experience more negative affect, possibly because of the experiencing of emotions but also because of the loss of sense of control or achievement that they desire from maintaining low weight and high levels of restriction. Although this has not been shown to extend to personality disorders, it suggests that this potential distortion may be another complication in assessing personality pathology in eating disordered patients (Vitousek & Stumpf, 2005). In contrast, Vitousek (2005) proposes that some women with AN may confess to personality disturbances that they do not have in order to provide non-eating
disorder related explanations for eating disorder inspired behaviours (Vitousek & Stumpf, 2005).

3.4 CONCLUSIONS

The concept of personality has evolved over time. Recently, there has been a focus on bridging personality traits and disorders in order to measure normal and abnormal personality. Comorbidity is common between Axis I disorders and personality pathology. This comorbidity has been found to impact the symptom severity, expression and functioning at pre-treatment of Axis I disorders such as depression, anxiety substance use disorders and eating disorders. The impact of a comorbid personality disorder on outcome of Axis I disorders is more contentious.

There has been a growth of interest in the relationship between eating disorders and personality over the past twenty years however, there are many areas that remain contentious or unexplored. It is important to understand the relationship between personality functioning and eating disorders as specific personality traits such as impulsivity or obsessive-compulsive traits have been implicated in the aetiology of eating disorders. Greater understanding of this comorbidity is especially important given that personality functioning has been implicated in some studies as a potential negative influence on the outcome of treatment. Few definite conclusions have been reached in research examining personality in eating disorders. Whilst general patterns of Axis II comorbidity in eating disorders are somewhat conceded, prevalence levels are highly variable. Methodological issues and limitations in the assessment of personality disorders are partly responsible for this. It is important to consider these methodological issues in the assessment of eating disorders, as invalid assessment information impacts
treatment planning and complicates efforts to understand the course and outcome of eating disorders. Incorporating personality traits and personality disorders into a broader assessment of normal and abnormal personality may offer a possible solution to some of the difficulties within this area.

This thesis attempts to address some of the unresolved areas in the relationship of eating disorders and personality. These include examining the impact of personality disorders on eating disorder symptoms and long-term outcome, a comparison of different ways of measuring personality pathology and clarification of similarities and differences in personality traits and disorders in BED compared to BN.
PART 3

METHODOLOGY

Chapter 4: Overview of the methodology in the Bulimia Treatment Study

4.1 OVERVIEW

This chapter describes the sample and methodology for the Bulimia Treatment Study from which empirical data are examined in later chapters (Part 3, Chapters 5-8).

4.2 INTRODUCTION – THE BULIMIA TREATMENT STUDY (BTS)

The BTS was a randomised controlled treatment trial for BN with long term follow-up. This dismantling trial evaluated the additive efficacy of exposure based versus non-exposure based behavioural treatments to a core of cognitive therapy. All participants received eight sessions of cognitive therapy before being randomised to a further eight sessions of one of three forms of behavioural therapy: a) exposure to pre-binge cues with binging being prevented (B-ERP), b) exposure to pre-purge cues with purging being prevented (P-ERP), or c) relaxation training (RELAX). The main assessment timepoints were: pre-treatment, mid-treatment, post-treatment, 6-month follow-up, one, two, three, and five-year follow-up. Figure 4.1 shows an overview of the BTS study design. Relevant aspects of the BTS study to be reported in this thesis are pre-treatment, one, three and five-year follow-up points.
Eligible
Consented to treatment
Pre-treatment assessment including:
- Blood test
- Self-report questionnaires
- Structured clinical interview for DSM-III-R with non-treating clinician
- Personality assessment
Cognitive Therapy
Mid-treatment assessment
Randomised and began exposure based or non-exposure based treatment
B-ERP
P-ERP
RELAX
Post-treatment assessment
Follow-up phase 6 month, 12 month, 2, 3, 5-year follow-up

Figure 4.1: Overview of the BTS design
4.3 METHODS

4.3.1 Ethical approval

The BTS received ethical approval from the Southern Regional Health Authority (Canterbury) and the University of Canterbury Ethics Committee. Participants provided written informed consent (see Appendix E).

4.3.2 Inclusion criteria

Inclusion criteria for the BTS study were:

- Female gender
- Ages 17-45
- Current primary diagnosis of BN

Below are the DSM-III-R diagnostic criteria for BN in the BTS study (American Psychiatric Association, 1987).

A Recurrent episodes of binge eating (rapid consumption of a large amount of food in a discrete period of time)

B A feeling of lack of control over eating behaviour during the eating binges

C The person regularly engages in either self-induced vomiting, use of laxatives or diuretics, strict dieting or fasting, or vigorous exercise in order to prevent weight gain

D A minimum average of two binge eating episodes a week for at least three months

E Persistent overconcern with body shape and weight
4.3.3 Exclusion criteria

Exclusion criteria for the BTS study were:

- Current AN
- Current obesity (Body Mass Index [BMI] >30)
- Current severe major depression (Hamilton Depression Rating Scale score ≥20) or active suicidal intent
- Current psychoactive substance dependence
- Current or past bipolar I disorder or schizophrenia
- Any developmental learning disorder or cognitive impairment contraindicating cognitive-behavioural treatment
- Major medical or neurological illnesses
- Current significant medical complications of BN
- Currently taking psychoactive medication and unwilling to undergo a supervised drug wash-out period (the longer of two weeks or five drug half-lives)

4.3.4 Recruitment

Participants were recruited using a broad range of strategies including direct mailings to general practitioners and mental health providers, advertisements at local universities and polytechnic institutes and in local media offering free treatment for BN.
4.4 PROCEDURE

Participants underwent telephone screening in which the nature of the study was described, the possible presence of BN was determined and the likely absence of exclusion criteria assessed. Three assessments were then scheduled.

4.4.1 Assessments

Assessment 1: Initial evaluation

The first assessment, conducted by the treating therapist, was a brief interview to ensure participants met criteria for BN and none of the exclusion criteria. Once eligibility was confirmed, informed consent was obtained. A history of BN, routine blood tests, height and weight were collected. In addition, a list of high-risk binge foods was generated along with intensity and frequency ratings for these foods. Participants were trained in self-monitoring which began that day and continued until the end of treatment. Self-report questionnaires were given to participants to take away and complete.

Assessment II: Diagnostic assessment and symptom review

The second assessment took place approximately one week later at which a non-treating clinician determined the presence of Axis I and Axis II disorders using SCID I and SCID-II interviews (American Psychiatric Association, 1987). The non-treating therapist assessed core bulimic symptomatology, mood and global functioning. Self-report questionnaires were collected and checked to ensure they had been fully completed.
Assessment III: Cue reactivity

The third assessment took place approximately one week later and tested information processing speed followed by a cue reactivity assessment.

Mid-treatment, post-treatment and follow-up assessments were clinician rated and examined mood, eating disorder symptomatology and global functioning. Self-report questionnaires were collected and checked for completeness.

4.4.2 Measures

Table 4.2 summarises when assessments were conducted and the measures used at each timepoint. Only the measures of the BTS relevant to this thesis will be described here.
Table 4.1 Relevant assessments and measures used in the BTS study

<table>
<thead>
<tr>
<th>Measure</th>
<th>Pre-treatment</th>
<th>Mid-treatment</th>
<th>Post-treatment</th>
<th>Follow-up</th>
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<td><strong>Diagnostic</strong></td>
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<td>SCID-I</td>
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<td>SCID-I partial¹</td>
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<td>SCID-II</td>
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<td><strong>Clinical Ratings</strong></td>
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<td>CBSI</td>
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<td><strong>Self-report questionnaires</strong></td>
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<td>EDI-2</td>
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<td>TCI</td>
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</table>

¹ Note: eating disorder and major depression were the only diagnostic sections examined at follow-up assessments

Abbreviations: SCID-I = Structured Clinical Interview for DSM-III-R Axis I, SCID-II = Structured Clinical Interview for DSM-III-R Axis II, HDRS = Hamilton Depression Rating Scale, GAF = Global Assessment of Functioning, CBSI = Comprehensive Bulimia Severity Index, EDI = Eating Disorder Inventory, EDI-2 = Eating Disorder Inventory-2, TCI = Temperament and Character Inventory
4.4.2.1 Diagnostic measures

The Structured Clinical Interview for DSM-III-R Patient Version (SCID-I)

This is a diagnostic interview that assesses the presence of current (past month) and lifetime DSM-III-R Axis I disorders (Spitzer, 1990)

This was used for the following diagnoses:

- **Mood disorders**: Major depression, Dysthymia, Bipolar II
- **Anxiety disorders**: Panic disorder with/without Agoraphobia, Social Phobia, Simple Phobia, Obsessive Compulsive Disorder, Generalised Anxiety Disorder
- **Eating disorders**: AN, BN, EDNOS
- **Psychoactive substance use disorders**: Alcohol, Sedatives/Hypnotics, Cannabis, Stimulant, Opioid, Hallucinogen, Polydrug, Other

The Structured Clinical Interview for DSM-III-R Personality Disorders Version (SCID-II)

This is a diagnostic interview that assesses the presence of 11 current Axis II disorders (Spitzer, 1990). The personality disorders can be further classified into three clusters:

- **Cluster A** – Paranoid, Schizotypal, Schizoid personality disorders
- **Cluster B** – Borderline, Histrionic, Narcissistic, Antisocial personality disorders
• **Cluster C** – Avoidant, Dependent, Obsessive-Compulsive, Passive Aggressive personality disorders

4.4.2.2 Clinical ratings

**The Hamilton Depression Rating Scale (HDRS)**

This is a 17-item clinician rating scale used to measure current depression severity (past week) on items such as low mood, feelings of guilt, agitation, anxiety, insomnia and somatic symptoms (Hamilton, 1960). Variables are measured either on a three point or five point scale. Internal consistency has been reported to range from 0.83 – 0.94 and inter-rater reliabilities 0.87 – 0.95 (Hamilton, 1986).

**The Global Assessment of Functioning (GAF)**

This is Axis V of the DSM-III-R (American Psychiatric Association, 1987). It is a clinician rating of current (past week) overall level of functioning (considering psychological, social and occupational functioning) on a scale ranging from 1-90 (1-10 = persistent danger of severely hurting self or others, 81-90 = absent or minimal symptoms). Intraclass correlation coefficients for (DSM-III) Axis V ratings range from 0.69 (test-retest evaluations) to 0.80 (joint interview method; Spitzer & Forman, 1979).

**Comprehensive Bulimia Severity Index (CBSI)**

The CBSI is a clinician rated instrument designed to measure the frequency and intensity of bulimic symptoms (binge frequency, vomiting frequency, purging frequency, dietary restriction and body dissatisfaction) and global functioning (mood, anxiety, substance use and social/occupational functioning; Sullivan, 1993). This
instrument was designed specifically for this study using questions that reflected concepts from the Eating Disorder Examination (EDE; Fairburn, 1993).

4.4.2.3 Self-report questionnaires

**Eating Disorder Inventory (EDI) and EDI-revised (EDI-2)**

The EDI (1983) is a 64-item self-report inventory used to assess the psychological characteristics relevant to eating disorders (Garner, 1983). This consists of eight subscales: drive for thinness, bulimia, body dissatisfaction, ineffectiveness, perfectionism, interpersonal distrust, interoceptive awareness and maturity fears (Garner, 1983) The EDI was revised (EDI-2) in 1991 as a 91-item self-report questionnaire and included 27 new items divided into three additional subscales: asceticism, impulse regulation and social insecurity (Garner, 1991). The original EDI subscales had internal consistency ranging from 0.83 to 0.93 for an eating disorder sample (Garner & Olmsted, 1984 in (Garner, 1991). Internal consistency for the three additional subscales (asceticism, impulse regulation and social insecurity) were 0.70, 0.77 and 0.80 respectively (Garner, 1991). Test-retest reliability for a non-patient sample was satisfactory after three weeks (Wear & Pratz, 1987) however, this was lower after a one-year interval (Crowther, Lilly, Crawford, Sheperd, & Oliver, 1990). At follow-up, a short version of the EDI was used. This had 41 items and five subscales: drive-for-thinness, bulimia, body dissatisfaction, perfectionism and interoceptive awareness.
The Temperament and Character Inventory (TCI)

The TCI (version 8) is a 238-item scale used to assess the personality dimensions of temperament and character (Cloninger et al., 1993). The four temperament dimensions consist of: novelty seeking, harm avoidance, reward dependence and persistence. The three character dimensions are: self-directedness, cooperativeness and self-transcendence. The authors report reliabilities of the TCI scales in the moderate to high range (0.76 to 0.92; Cloninger et al., 1993). The test-retest reliability of this scale has been reported as moderately high (0.85) over six months (Cloninger, Przybeck, Svrakic, & Wetzel, 1994).

4.5 Statistical Analyses

4.5.1 Computer packages

All data was entered into Paradox, a relational database (International, 1993). The Statistical Package for the Social Sciences (SPSS, Version 13) was used to analyse data.

4.5.2 Assessment of the normality of data

Tests for skewness were conducted to inspect the normal distribution of the data. Continuous variables were normally distributed except for the following: age-of-onset of eating disorder, duration of eating disorder, ethnicity, binge and purge frequencies, and number of personality disorder symptoms (Cluster A, B, C and total symptoms). Parametric and non-parametric tests were used however, means and standard deviations are presented for the ease of comparison with data from other studies.
4.5.3 Statistical techniques

Continuous variables which were normally distributed were examined using t-tests or analysis of variance (ANOVA). When significant differences were found, post-hoc analyses were undertaken using the least significant difference (LSD) test.

The chi-square test was used for dichotomous variables, with Fisher’s exact test being used where expected cell numbers were small.

Repeated measures ANOVAs were used to assess group differences across multiple timepoints. Any significant group differences identified were further explored using multiple regression analyses to determine independent responsibility for these differences. Paired samples t-tests were used to compare the mean scores on some continuous variables.

Binary logistic regression was used to examine the contribution of multiple variables to a dichotomous outcome variable, with variables entered simultaneously (enter) then using a stepwise method (forward conditional).

Pearson’s correlation coefficients were used to examine the association of normally distributed continuous variables.

4.5.4 Reliability of measures

No reliability checks were undertaken with the personality disorder (SCID-II) interviews however, there is some overlap with raters for this study and previous studies with published SCID-II reliability data. Kappa statistics for inter-rater reliability of Axis II disorders with the overlapping raters range from 0.78 to 0.81 (using a sample with
MDD and AN respectively; Carter, Joyce, Mulder, Sullivan, & Luty, 1999; Jordan et al., 2008).

4.5.5 Approach to analyses

Statistical significance was set at a 0.05 or 0.01 (two-tailed) level. It is acknowledged that multiple exploratory comparisons and the use of an alpha level of <0.05 increase the risk of Type I error. The choice of these statistics is considered justified in relation to questions that have not been addressed previously or where the literature is contradictory as correcting too stringently increases the risk of Type II error.
Chapter 5: Overview of the methodology in the Binge Eating Psychotherapy Study (BEP)

5.1 OVERVIEW

This chapter describes the sample and methodology for the Binge Eating Psychotherapy (BEP) study from which empirical data are examined in later chapters (Part 4, chapters 9-11).

5.2 INTRODUCTION

The BEP study is a randomised controlled treatment trial of three psychotherapies for BN and BED with long term follow-up. The trial compared the relative efficacy of standard CBT with two potentially enhanced forms: schema focused therapy (SFT) and nutrition and appetite enhanced CBT (CBT-A). All participants received approximately 20 weekly sessions (possible range 15-26 sessions) of psychotherapy followed by six months of monthly (possible range 3-8 sessions) maintenance sessions.

The main assessment timepoints were: pre-treatment, mid-treatment, end-treatment, two, three and five-year follow-up. Figure 4.1 shows an overview of the BEP study design.
Figure 5.1: Overview of the BEP study design

Phone screening interview

Pre-treatment clinical assessment to establish eligibility

Eligible

Consented to treatment

Pre-treatment assessment including:
- Biological assessment
- Self-report questionnaires
- Neuropsychological testing
- Structured Clinical Interview for DSM-IV with treating clinician

Randomised and began treatment

CBT

CBT-A

SFT

Personality assessment at week 4 point

Mid-treatment (week 26) assessment

Completed therapy. End assessment at week 52

Follow-up phase assessments at 2, 3, and 5 years

15-26 sessions, 6 months

3-8 sessions, 6 months
5.3 METHODS

5.3.1 Ethical approval

The BEP study received ethical approval from the Upper Regional South A Ethics Committee. Participants provided written informed consent (see Appendix F).

5.3.2 Inclusion criteria

Inclusion criteria for the BEP study were:

- Female gender
- Ages 16 and above
- Current primary diagnosis of BN or BED
- BMI > 17.5

Below are the DSM-IV diagnostic criteria for BN in the BEP study (American Psychiatric Association, 1994).

A Recurrent episodes of binge eating. An episode of binge eating is characterised by both the following:

1) eating, in a discrete period of time (e.g., within any 2-hour period), an amount of food that is definitely larger than most people would eating during a similar period of time and under similar circumstances
2) a sense of lack of control over eating during the episode (e.g., a feeling that one cannot stop eating or control what or how much one is eating)

B Recurrent inappropriate compensatory behaviour in order to prevent weight gain, such as: self-induced vomiting, misuse of laxatives, diuretics, enemas, or other medications, fasting, or excessive exercise
C The binge eating and inappropriate compensatory behaviours both occur, on average, at least twice a week for three months

D Self-evaluation is unduly influenced by body shape and weight

E The disturbance does not occur exclusively during episodes of AN

Specify type:

**Purging Type:** during the current episode of BN, the person has regularly engaged in self-induced vomiting or the misuse of laxatives, diuretics, or enemas.

**Nonpurging Type:** during the current episode of BN, the person has used other inappropriate compensatory behaviors, such as fasting or excessive exercise, but has not regularly engaged in self-induced vomiting or the misuse of laxative, diuretics, or enemas.

The diagnosis for BED is classified under EDNOS in the DSM-IV. Below are the criteria for BED in the BEP study (American Psychiatric Association, 1994)

**Table 5.2 DSM-IV research criteria for BED**

A Recurrent episodes of binge eating. Definition of binge eating is the same as that used in BN diagnostic criteria

B The binge eating episodes are associated with three (or more) of the following:

1) eating much more rapidly than normal
2) eating until feeling uncomfortably full
3) eating large amounts of food when not feeling physically hungry
4) eating alone because of being embarrassed by how much one is eating
5) feeling disgusted with oneself, depressed or very guilty after overeating

C Marked distress regarding binge eating is present

D The binge eating occurs, on average, at least two days a week for six months

E The binge eating is not associated with the regular use of inappropriate compensatory behaviours (e.g. purging, fasting, excessive exercise) and does not occur exclusively during the course of AN or BN

5.3.3 Exclusion criteria

Exclusion criteria for the BEP study were:

- Current severe major depression
- Current severe psychoactive substance dependence
- Current or past bipolar I disorder or schizophrenia
- Developmental learning disorder or cognitive impairment
- Major medical or neurological illness that could interfere with assessment or treatment
- Current severe medical complications of an eating disorder
- Currently taking psychoactive medication and unwilling to undergo a supervised drug wash-out period (the longer of two weeks or five drug half-lives)
- Failure to respond to a previous adequate trial of one of the psychotherapies in this study
5.3.4  **Recruitment**

Participants were recruited using a broad range of strategies including direct mailings to general practitioners and mental health providers, advertisements at local universities and polytechnic institutes, advertisements at local gyms and in local media offering free treatment for those with binge eating episodes.

5.4  **PROCEDURE**

Participants underwent telephone screening in which the nature of the study was described, the possible presence of BN or BED was determined and the likely absence of exclusion criteria assessed. Details were given to a clinical psychologist who contacted the participant and arranged an initial assessment.

5.4.1  **Assessments**

**Assessment 1: Initial evaluation**

The first assessment, conducted by the treating therapist, was a clinical assessment and interview to ensure participants met criteria for BN or BED and had none of the exclusion criteria. Once eligibility was confirmed, informed consent was obtained. Three further assessments were scheduled at this time (neuroendocrine, neuropsychological and diagnostic). Participants were trained in self-monitoring which began seven days prior to their neuroendocrine assessment.

**Assessment II: Neuroendocrine assessment**

The neuroendocrine assessment took place over the morning. This involved having physical measurements taken, fasting blood tests, an oral glucose load and completing
self-report questionnaires. Self-report questionnaires were collected and checked for completeness.

**Assessment III: Neuropsychological testing**

The third assessment was scheduled for an afternoon. This assessment involved a computerised assessment of neuropsychological functioning using the Cambridge Neuropsychological Test Automated Battery (CANTAB), a facial expression recognition task, several other pen and paper tasks and a verbal IQ test.

**Assessment IV: Diagnostic assessment**

The treating clinician determined the presence of Axis I disorders and current eating disorder status, assessed core bulimic and binge eating symptomatology, mood and global functioning.

All research assessments were completed within a three day period prior to randomisation.

**5.4.2 Measures**

Table 5.3 summarises when assessments were conducted and the measures used at each timepoint.

Only the measures of the BEP study relevant to this thesis will be described here.

The GAF and EDI-2 were discussed previously in Chapter 4 and will not be detailed in this section.
Table 5.3: Relevant assessments and measures used in the BEP study

<table>
<thead>
<tr>
<th>Measure</th>
<th>Pre-treatment</th>
<th>Week 4</th>
<th>Mid-treatment</th>
<th>End-treatment</th>
<th>Follow-up</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Diagnostic</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SCID-I</td>
<td></td>
<td></td>
<td></td>
<td>*</td>
<td>*</td>
</tr>
<tr>
<td>SCID-I partial¹</td>
<td></td>
<td></td>
<td>*</td>
<td>*</td>
<td>*</td>
</tr>
<tr>
<td>SCID-II</td>
<td></td>
<td></td>
<td>*</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Clinical ratings</strong></td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>MADRS</td>
<td></td>
<td></td>
<td></td>
<td>*</td>
<td></td>
</tr>
<tr>
<td>GAF</td>
<td></td>
<td></td>
<td></td>
<td>*</td>
<td>*</td>
</tr>
<tr>
<td>EDE</td>
<td></td>
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<tr>
<td><strong>Self-report questionnaires</strong></td>
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<td>EDI-2</td>
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<tr>
<td>TCI-R</td>
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<tr>
<td>SCL-90</td>
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</tr>
</tbody>
</table>

¹ Note: eating disorder, major depression, social phobia and obsessive-compulsive disorder were the only diagnostic sections examined at follow-up

Abbreviations: SCID-I = Structured Clinical Interview for DSM-IV Axis I, SCID-II = Structured Clinical Interview for DSM-IV Axis II, MADRS = Montgomery-Asberg Depression Rating Scale, GAF = Global Assessment of Functioning, EDE = Eating Disorder Examination, EDI-2 = Eating Disorder Inventory-2, TCI-R = Temperament and Character Inventory Revised, SCL-90 = Hopkins Symptom Checklist
5.4.2.1 Diagnostic measures

The Structured Clinical Interview for DSM-IV Patient Version (SCID-I)

This is a diagnostic interview that assesses the presence of current (past month) and lifetime DSM-IV Axis I disorders (First, Spitzer, Gibbon, & Williams, 1996).

This was used to assess the presence of the following diagnoses:

- **Mood disorders**: Major depression, Dysthymia, Bipolar II, Bipolar Not Otherwise Specified
- **Anxiety disorders**: Panic disorder with/without Agoraphobia, Social Phobia, Specific Phobia, Obsessive Compulsive Disorder, Posttraumatic Stress Disorder, Generalised Anxiety Disorder
- **Eating disorders**: AN, BN, BED
- **Substance-related disorders**: Alcohol, Sedatives/Hypnotics, Cannabis, Stimulant, Opioid, Hallucinogen, Polydrug, Other substance-related disorder

The Structured Clinical Interview for DSM-IV Personality Disorders Version (SCID-II)

This diagnostic interview assesses the presence of ten current Axis II disorders (First, Gibbon, Spitzer, Williams, & Benjamin, 1997). The disorders are grouped within three clusters:

- **Cluster A** – Paranoid, Schizotypal, Schizoid personality disorders
- **Cluster B** – Borderline, Histrionic, Narcissistic, Antisocial personality disorders
- **Cluster C** – Avoidant, Dependent, Obsessive-Compulsive personality disorders
5.4.2.2 Clinical rating scales

Montgomery Asberg Depression Rating Scale (MADRS)

This is a 10-item clinician rating scale used to measure behavioural, affective and vegetative symptoms of depression over the past week (Montgomery & Asberg, 1979). Variables are rated on six point scale of severity. Internal consistency and inter-rater reliability has been reported as satisfactory (Davidson, Turnbull, Strickland, Miller, & Graves, 1986; Montgomery & Asberg, 1979).

Eating Disorder Examination – 12th Edition (EDE)

The EDE is a clinician-rated semi-structured interview used to assess eating disorder psychopathology (Cooper & Fairburn, 1987). Questions from the EDE – 12th edition are largely concerned with the current state of patients over the past four weeks although the time frame is for three or six months for some items. The scores are used to form a profile of individuals on four subscales (Restraint, Eating Concern, Shape Concern, Weight Concern; Fairburn, 1993). The internal consistency of the original EDE ranges from 0.68 (Weight Concern subscale) to 0.90 (Bulimia subscale) and inter-rater reliability has been reported as uniformly high (Cooper & Fairburn, 1987; Fairburn, 1993). Concurrent and discriminant have been established (Rosen, Vara, Wendt, & Leitenberg, 1990).
5.4.2.3 Self-report questionnaires

The Temperament and Character Inventory Revised (TCI-R)

The TCI was revised (TCI-R) in 1999 to a 240-item scale with the most significant changes in the development of the ‘Persistence’ dimension. The mean reliability coefficient for the TCI-R subscales are: novelty-seeking = 0.72, harm avoidance = 0.81, reward dependence = 0.74, persistence = 0.78, self-directedness = 0.78, cooperativeness = 0.71, self-transcendence = 0.82. Reliability for the overall scale is 0.77 (Farmer & Goldberg, 2008).

The Hopkins Symptom Checklist (SCL-90)

This 90-item self-report measures psychiatric symptoms in outpatients in both clinical and research situations. A list of problems and complaints is rated for severity (0 = “not at all” and 5 = “extremely”) over the past week with higher scores indicating greater distress. The subscales are: somatization, obsessive-compulsive, interpersonal sensitivity, depression, anxiety, hostility, phobic anxiety, paranoid ideation and psychoticism. Internal consistency for the items range from 0.77 to 0.90 (Derogatis, Rickels, & Rock, 1976). Test-retest reliability range between 0.80 to 0.90 after one week (Derogatis, 1977).

5.5 Statistical Analyses

5.5.1 Computer packages

All data was entered into Paradox, a relational database (International, 1993). The Statistical Package for the Social Sciences (SPSS, Version 13) was used to analyse data.
5.5.2 Assessment of the normality of data

Tests for skewness were conducted to inspect the normal distribution of the data. Continuous variables were normally distributed except for the following: age-of-onset of eating disorder, duration of eating disorder, ethnicity, BMI, binge and purge frequencies and number of personality disorder symptoms (Cluster A, B, C and total symptoms). Parametric and non-parametric tests were used however, means and standard deviations are presented for the ease of comparison with data from other studies.

5.5.3 Statistical techniques

Continuous variables which were normally distributed were examined using t-tests or ANOVA. When significant differences were found, post-hoc analyses were undertaken using the LSD test. Where a normal distribution was not present, the Mann-Whitney U non-parametric test for two independent samples was used. For ease of comparison, means and standard deviations are presented. The chi-square test was used for dichotomous variables, with Fisher’s exact test being used where expected cell numbers were small. Paired samples t-test was used to compare the mean scores on some continuous variables. Stepwise discriminant analysis was used to explore the predictive ability of an independent variable with a categorical dependent variable. This showed which variables best predicted group membership.

5.5.4 Reliability of measures

Interrater reliability checks were performed on eight (10%) personality disorder (SCID II) interviews at two-year follow-up. The concordance between raters for Axis II
diagnoses (avoidant, borderline, obsessive-compulsive and paranoid personality disorders) was 100%. The raters agreed on the number of symptoms in five instances and disagreed in three cases, however the difference in all three cases was by a single symptom.

5.5.5 Approach to analyses

Statistical significance was set at a 0.05 (two-tailed) level. Multiple exploratory comparisons and use of a <0.05 alpha level increase the risk of Type I error (Bland & Altman, 1995). Given the multiple comparisons used in these exploratory analyses, a more stringent significance level of 0.01 might have been more appropriate. However, these are relatively small samples of women with BN and BED therefore corrections for multiple comparisons were not made as the analyses in chapters 9 to 11 were exploratory and used to generate further hypotheses. Both statistically significant and non-significant findings are reported in the table and text.
PART 4

PERSONALITY IN BN: IMPACT ON PRESENTATION AND OUTCOME

Chapter 6: The Impact of Borderline Personality Disorder on BN

6.1 OVERVIEW

This chapter compares the patterns of Axis I and Axis II comorbidity in groups of women with BN and borderline personality disorder (B-PD), other personality disorders (Other-PD) and no personality disorders (No-PD). The impact of borderline personality disorder on BN outcome is examined at one-year and three-years post-treatment and compared to the Other-PD and No-PD groups. Personality traits assessed by the TCI at pre-treatment, one-year and three-year follow-up, are compared among the B-PD, Other-PD and No-PD groups in women with BN.

6.2 INTRODUCTION

Comorbidity of BN and borderline personality disorder is common, with estimates that one quarter of women with BN also have borderline personality disorder (Sansone & Levitt, 2005a). A clinical bias, that the presence of comorbid borderline personality disorder in women with BN is associated with poorer eating disorders treatment outcome, is partially supported by outcome studies (Johnson et al., 1990; Keel & Mitchell, 1997; Steiger & Stotland, 1996). However, other studies report little adverse impact of borderline personality disorder on BN treatment outcomes over long-term
follow-up (Fallon et al., 1991; Norring, 1993; Wonderlich et al., 1994; Zanarini et al., 2003). A naturalistic prospective study found that the presence or severity of borderline personality disorder, avoidant personality disorder and obsessive-compulsive personality disorder did not significantly affect the natural course of BN (Grilo et al., 2007; Grilo et al., 2003).

In addition to differences in eating disorder outcome reported in some studies, broader clinical symptoms have been found to differ between women with BN with and without personality disorders. Those with a comorbid personality disorder have been characterised as having poorer general psychological functioning (depression, anxiety, self-mutilation, substance abuse), interpersonal problems and poorer social functioning (Cloninger et al., 1993; Fassino et al., 2002a; Johnson et al., 1989). Despite this, many studies show no differences in eating disorder symptoms such as binge eating, or attitudes such as drive for thinness and body dissatisfaction (Johnson et al., 1989).

Temperament and character dimensions of both BN and borderline personality disorder have been studied extensively using the TCI (Cloninger et al., 1993). Women with BN are characterised by high novelty seeking, high harm avoidance and low self-directedness (Bulik et al., 1995a; Fassino et al., 2002a; Mizushima, 1998). Cloninger links these personality features to pessimistic thoughts, rumination, immaturity and an inability to set and achieve goals (Fassino et al., 2002a). There is considerable overlap in TCI profiles between BN and borderline personality disorder. Individuals with borderline personality disorder also exhibit low self-directedness, high novelty seeking and high harm avoidance. In addition, borderline personality disorder patients have low cooperativeness scores, often making them socially intolerant and unhelpful (Joyce,
Mulder, Luty, McKenzie, Sullivan, & Cloninger, 2003). In both BN and borderline personality disorder these characteristics may influence treatment response and may predict outcome (Anderson, Joyce, Carter, McIntosh, & Bulik, 2002; Fassino, Amianto, Gramaglia, Facchini, & Abbate Daga, 2004; Joyce et al., 2003). Self-directedness has been found to be a strong predictor of outcome in BN (Bulik et al., 1998b). Higher harm avoidance, lower self-directedness and lower cooperativeness were found to persist in women recovered from eating disorders compared with control women (Klump, Strober, Bulik, Thornton, Johnson, Devlin, Fichter, Halmi, Kaplan, Woodside, Crow, Mitchell, Rotondo, Keel, Berrettini, Plotnicov, Pollice, Lilienfeld, & Kaye, 2004).

6.3 AIMS

Aim 1: To determine if the presence of borderline personality disorder (B-PD group) will have a negative impact on the pre-treatment clinical characteristics (eating disorder symptoms, depressive symptoms, global functioning) in women with BN compared to the Other-PD and No-PD groups

Aim 2: To determine if the presence of borderline personality disorder (B-PD group) will have a negative impact on BN outcome one-year and three-years after cognitive therapy for BN compared to the Other-PD and No-PDs groups

Aim 3: To determine if personality traits will differ at pre-treatment and follow-up in the B-PD group compared to the Other-PD or No-PD groups.
6.4 METHODS

6.4.1 Overview

The BTS, described earlier in Chapter 4, was used for these analyses. Full details of the study design and outcome, and three-year follow-up data have been presented elsewhere (Bulik et al., 1998a; Carter, McIntosh, Joyce, Sullivan, & Bulik, 2003a).

6.4.2 Participants

Participants were 134 women, aged 17 to 45 years, with a current DSM-III-R diagnosis of BN. Of the 135 participants entering the study, one was excluded from the analyses as Axis II data were missing. Exclusion criteria were outlined in Chapter 4.

6.4.3 Procedure

This study received ethical approval from the Southern Regional Health Authority (Canterbury) and the University of Canterbury Ethics Committee. Participants provided written informed consent.

6.4.3.1 Pre-treatment assessment

As described earlier, non-treating clinicians undertook assessments for Axis I and II disorders using the SCID-I and II for the DSM-III-R (Spitzer, 1990), eating disorder symptoms such as binging and purging frequency (CBSI), current depression severity (HDRS) and global functioning (GAF) over the past week. Participants completed self-report questionnaires including the EDI-2 and the TCI. These measures were described in detail in Chapter 4.
6.4.3.2 Follow-up assessment

Participants were reassessed at end treatment and one and three-year follow-up. Post-treatment assessment consisted of DSM-III-R diagnostic criteria for eating disorders, CBSI, HDRS, GAF, EDI (short version) and TCI assessments. Data were available for 134 participants at pre-treatment, 105 at end treatment, 107 at one-year follow-up and 112 at three-year follow-up.

6.4.3.3 Statistical analyses

The Statistical Package for the Social Sciences (SPSS, Version 12) was used to analyse data. Participants were divided into three groups: 1) B-PD, 2) Other-PD, and 3) No-PD. The division into these three groups follows previous studies that indicate differences in severity of psychopathology across these groups (Joyce et al., 2003; Steiger & Stotland, 1996; Steiger, Thibaudeau, Ghadirian, & Houle, 1992; Wonderlich & Swift, 1990). Chi-square tests were conducted on dichotomous variables. Analysis of variance among the groups was used to compare normally distributed continuous variables. T-tests were used to compare different time points for the groups. A repeated measures ANOVA was used to assess differences among the three groups over four time points. To reduce the risk of Type I error we have opted for a more stringent statistical significance level of \( p < 0.01 \) but by doing this the risk of Type II error is increased. In the few instances where follow-up data was missing, no attempt was made to estimate or impute these values.

6.5 RESULTS

The patient flow for the BTS study is presented in Figure 6.1.
Figure 6.1: Overview of the patient flow for the BTS

- Initial screening interview, n=152
- Pre-treatment clinical assessment, n=135
- Cognitive Therapy
- Mid-treatment assessment and point of randomisation, n=116
  - B-ERP, n=39
  - P-ERP, n=38
  - RELAX, n=39
- Post-treatment assessment, n=106
- Six-month follow-up assessment, n=107
- One-year follow-up assessment, n=102
- Two-year follow-up assessment, n=89
- Three-year follow-up assessment, n=113
- Five-year follow-up assessment, n=109

(Carter et al., 2003a)
6.5.1 Pre-treatment differences between B-PD, Other-PD and No-PD

The mean age of the sample was 26.1 years (SD=6.1) and 91% were New Zealand European. The remaining ethnicities were: Maori/European = 4.4%, Maori = 1.5%, Pacific Island/European = 1.5%, Asian = 0.7%, ‘Other’ = 0.7%. Sixty-two percent had never been married. Binge eating and purging frequency for the sample had a mean of 10.6 and 14.3 episodes respectively, at pre-treatment. A lifetime mood disorder was present in 64% of the sample. Diagnosis of an Axis II disorder was present in 56% of the sample. The most common personality disorders were borderline (28%), avoidant (28%) and paranoid (24%). Of the 75 individuals with a personality disorder diagnosis, 38 met criteria for borderline personality disorder (B-PD group) and 37 were diagnosed with having other personality disorders (Other-PD group). Borderline personality disorder often co-occurred with other personality disorders as seen in Table 6.1. In the cases where borderline personality disorder existed with another personality disorder, these individuals were categorised in the B-PD group.

There were no differences among the groups in age, age-of-onset of BN, duration of BN or history of AN. Table 6.1 shows that the B-PD group had worse general functioning and more severe clinician rated depressive symptomatology (HDRS), and greater Axis I comorbidity at presentation than those with No-PD. Participants in the B-PD group had significantly lower global functioning (GAF), and more frequent bipolar II disorder, simple phobia, and substance abuse/dependence (alcohol and cannabis) diagnoses than those with Other-PD and No-PD. No significant differences emerged in the prevalence of depression, social phobia, obsessive compulsive disorder, panic disorder and agoraphobia among B-PD, Other-PDs or No-PDs.
Table 6.1: Descriptive characteristics and Axis I comorbidity at pre-treatment of subjects with BN divided by the presence of B-PD, Other-PD and No-PD

<table>
<thead>
<tr>
<th></th>
<th>B-PD (N=38)</th>
<th>Other-PD (N=37)</th>
<th>No-PD (N=59)</th>
<th>Statistic</th>
<th>p</th>
<th>Post Hoc</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Demographics</strong></td>
<td></td>
<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>Age (years)</td>
<td>25.8 (5.5)</td>
<td>25.4 (5.7)</td>
<td>26.6 (6.7)</td>
<td><em>F</em> = 0.5</td>
<td>NS</td>
<td></td>
</tr>
<tr>
<td><strong>Bulimia history</strong></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Age of onset (year)</td>
<td>19.3 (4.9)</td>
<td>19.0 (3.4)</td>
<td>19.9 (5.1)</td>
<td><em>F</em> = 0.5</td>
<td>NS</td>
<td></td>
</tr>
<tr>
<td>Duration (years) ¹</td>
<td>6 (0-23)</td>
<td>4 (0-25)</td>
<td>5 (0-20)</td>
<td><em>x²</em> = 0.4</td>
<td>NS</td>
<td></td>
</tr>
<tr>
<td>% History of AN</td>
<td>1.5 (0.9)</td>
<td>1.7 (0.9)</td>
<td>1.5 (0.8)</td>
<td><em>F</em> = 0.8</td>
<td>NS</td>
<td></td>
</tr>
<tr>
<td><strong>Current functioning²</strong></td>
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<tr>
<td>GAF</td>
<td>52.9 (7.5)</td>
<td>56.3 (6.7)</td>
<td>57 (5.7)</td>
<td><em>F</em> = 4.9</td>
<td><strong>0.009</strong></td>
<td>B-PD&lt; Other-PD, No-PD</td>
</tr>
<tr>
<td>HDRS</td>
<td>11.1 (5.5)</td>
<td>10.0 (5.7)</td>
<td>6.1 (4.7)</td>
<td><em>F</em> = 12.3</td>
<td>&lt;<strong>0.001</strong></td>
<td>B-PD, Other-PD&gt; No-PD</td>
</tr>
<tr>
<td><strong>Lifetime Axis I Comorbidity³</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>Any Mood Disorder</td>
<td>34</td>
<td>90</td>
<td>28</td>
<td>76</td>
<td>28</td>
<td>48</td>
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<tr>
<td>Major Depressive Disorder</td>
<td>21</td>
<td>55</td>
<td>22</td>
<td>60</td>
<td>25</td>
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<td>Bipolar II</td>
<td>13</td>
<td>34</td>
<td>6</td>
<td>16</td>
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<td>Any Anxiety Disorder</td>
<td>27</td>
<td>71</td>
<td>21</td>
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<td>Obsessive Compulsive Disorder</td>
<td>1</td>
<td>3</td>
<td>2</td>
<td>5</td>
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<td>Panic Disorder</td>
<td>4</td>
<td>11</td>
<td>3</td>
<td>8</td>
<td>6</td>
<td>10</td>
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<tr>
<td>Agoraphobia without panic</td>
<td>1</td>
<td>3</td>
<td>1</td>
<td>3</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Social Phobia</td>
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<td>42</td>
<td>15</td>
<td>41</td>
<td>11</td>
<td>19</td>
</tr>
<tr>
<td>Axis II Comorbidity</td>
<td>B-PD (N=38)</td>
<td>Other-PD (N=37)</td>
<td>No-PD (N=59)</td>
<td>Statistic</td>
<td>p</td>
<td>Post Hoc</td>
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</tr>
<tr>
<td>Simple Phobia</td>
<td>17 (45)</td>
<td>11 (30)</td>
<td>8 (14)</td>
<td>$x^2 = 11.7$</td>
<td>0.003</td>
<td></td>
</tr>
<tr>
<td>Any Substance Abuse/Dependence</td>
<td>31 (82)</td>
<td>14 (38)</td>
<td>20 (34)</td>
<td>$x^2 = 23.4$</td>
<td>&lt;0.001</td>
<td></td>
</tr>
<tr>
<td>Alcohol Abuse / Dependence</td>
<td>31 (82)</td>
<td>13 (35)</td>
<td>18 (31)</td>
<td>$x^2 = 26.8$</td>
<td>&lt;0.001</td>
<td></td>
</tr>
<tr>
<td>Cannabis Abuse/Dependence</td>
<td>17 (45)</td>
<td>5 (14)</td>
<td>7 (12)</td>
<td>$x^2 = 16.7$</td>
<td>&lt;0.001</td>
<td></td>
</tr>
<tr>
<td>Stimulant Abuse/Dependence</td>
<td>9 (24)</td>
<td>2 (5)</td>
<td>4 (7)</td>
<td>$x^2 = 8.37$</td>
<td>NS</td>
<td></td>
</tr>
</tbody>
</table>

### Notes:
- Statistics are \(^1\) Kruskal-Wallis Tests; \(^2\)ANOVA; \(^3\)Chi-Square. Results for Lifetime Axis II comorbidity are shown without statistical significance as the specific personality patterns determined the composition of the groups. Abbreviations: AN = Anorexia Nervosa; GAF = Global Assessment of Functioning; HDRS = Hamilton Depression Rating Scale; PD = Personality Disorder.
Table 6.2 presents eating attitudes and behaviours, depression severity and global functioning at pre-treatment. Both the B-PD and Other-PD groups had significantly higher levels of disturbance than the No-PD group on EDI drive for thinness and body dissatisfaction subscales. As expected, the B-PD group scored higher on the EDI-2 impulse regulation subscale ($p<0.001$) than the Other-PD and No-PD groups. This was the only EDI-2 subscale with a specific elevation in the B-PD group compared with those with Other-PDs and No-PDs. Frequency of binge eating and purging behaviours did not differ significantly among the three groups at presentation.

### 6.5.2 Outcome at one and three-year follow-up for B-PD, Other-PD or No-PD

Women with BN and B-PD did not differ significantly from the Other-PD and No-PD groups in eating disorder symptoms and attitudes at one-year and three-year follow-up (Table 6.2).

At one-year follow-up none of the differences among the groups seen at pre-treatment on the EDI-2 drive for thinness and body dissatisfaction subscales were found. There were no differences among the three groups on eating disorder symptoms such as binge eating (Figure 6.2) and purging (Figure 6.3) at one-year follow-up. Overall no significant differences were found for the presence of any eating disorder diagnosis at one-year follow-up among the three groups (Table 6.3). However, at one-year follow-up there were several specific differences found in the B-PD group. They were more likely to still have a BN diagnosis but had lower prevalence of EDNOS than the other two groups. Anorexia nervosa at one-year follow-up was highest in the Other-PD group.
Levels of current depression (HDRS) and global functioning (GAF), showed improvements for all three groups at one-year follow-up. No significant differences among the groups were found at the one-year follow-up assessment.

Results at three-year follow-up were similar to those at one-year follow-up. At three-year follow-up, eating disorder symptoms were improved in all three groups and no differences were found among the three groups on EDI (short version) drive for thinness, bulimia and body dissatisfaction subscales, eating disorders symptoms (Figures 6.2 and 6.3) or eating disorder diagnoses. It is noteworthy that the B-PD group had the lowest rate of any eating disorder diagnoses at follow-up (35% and 24% at one and three years respectively; Table 6.3).
Table 6.2: EDI and outcome symptoms at pre-treatment, one-year and three-year follow-up divided by the presence of B-PD, Other-PD or No-PD

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<tr>
<th>Outcome measures</th>
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<th>No-PD</th>
<th>Repeated measures ANOVA Test of time &amp; group interaction</th>
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<td>7.6</td>
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<tr>
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<td>8.4</td>
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<td>6.7</td>
<td>6.2</td>
<td>4.6</td>
<td>6.1</td>
<td>0.9</td>
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| **Bulimia**       |      |          |       |    |   |        |        |
| pre-treatment     | 9.5  | 4.4      | 10.4  | 4.1 | 8.9  | 4.9  | 1.3   | NS      |
| one-year follow-up | 2.6  | 3.9      | 3.3   | 4.8 | 1.8  | 3.6  | 1.2   | NS      |
| three-year follow-up | 1.3  | 2.9      | 2.6   | 4.5 | 2.4  | 6.0  | 0.6   | NS      |

| **Body Dissatisfaction** |      |          |       |    |   |        |        |
| pre-treatment     | 21.4 | 6.0      | 20.7  | 7.0 | 15.5 | 7.9  | 9.7   | **<0.001** | B-PD, Other-PD>No-PD |
| one-year follow-up | 13.9 | 3.1      | 13.3  | 8.5 | 9.1  | 7.6  | 3.5   | NS      |
| three-year follow-up | 11.8 | 9.4      | 10.9  | 9.0 | 7.7  | 8.1  | 2.3   | NS      |

Note: the EDI-2 was used at pre-treatment and the EDI (short version) was used at follow-up.
Figure 6.2 Changes in binge frequency from pre-treatment to three-year follow-up

Figure 6.3 Changes in purge frequency from pre-treatment to three-year follow-up
Table 6.3: Eating disorder diagnoses at one-year and three-year follow-up assessments in BTS participants divided by B-PD, Other-PD or No-PD

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<td>N</td>
<td>%</td>
<td>N</td>
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<td>BN Current</td>
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<td></td>
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<tr>
<td>One-year follow-up</td>
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<td>22</td>
<td>4</td>
</tr>
<tr>
<td>Three-year follow-up</td>
<td>3</td>
<td>9</td>
<td>3</td>
</tr>
<tr>
<td>BN Past 6 Months</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>One-year follow-up</td>
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<td>22</td>
<td>4</td>
</tr>
<tr>
<td>BN Past Year</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Three-year follow-up</td>
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<td>15</td>
<td>6</td>
</tr>
<tr>
<td>AN Current</td>
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<td>One-year follow-up</td>
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<td>0</td>
<td>2</td>
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<td>Three-year follow-up</td>
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<td>0</td>
<td>0</td>
</tr>
<tr>
<td>AN Past 6 months</td>
<td></td>
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<td></td>
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<tr>
<td>One-year follow-up</td>
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<td>4</td>
<td>3</td>
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<tr>
<td>AN Past Year</td>
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<td>EDNOS Current</td>
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<td>One-year follow-up</td>
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<td>8</td>
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<td>Three-year follow-up</td>
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<td>15</td>
<td>6</td>
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<td>EDNOS Past 6 months</td>
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<td>One-year follow-up</td>
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<td>9</td>
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<td>EDNOS Past Year</td>
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<tr>
<td>Three-year follow-up</td>
<td>11</td>
<td>33</td>
<td>12</td>
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</table>

Current levels of depression and global functioning did not differ among the three groups.
6.5.3 Changes in the TCI over 3 years

Table 6.4 presents the TCI scales at pre-treatment, one-year and three-year follow-up in B-PD, Other-PD and No-PD groups. Pre-treatment TCI scores for participants with B-PD showed lower cooperativeness than those with Other-PDs or No-PDs. The B-PD and Other-PD groups had significantly higher harm avoidance and lower self-directedness than the No-PD group. Novelty seeking, reward dependence, and persistence scores did not differ among the groups.

At one-year follow-up, only the cooperativeness subscale scores had changed from pre-treatment, with the B-PD group no longer significantly different from the Other-PD and No-PD groups. The B-PD and Other-PD groups remained higher in harm avoidance and lower in self-directedness than the No-PD group at one-year follow-up.

At three-year follow-up, the Other-PD group had higher TCI harm avoidance and lower self-directedness compared to the No-PD group. The B-PD group was not significantly different from the Other-PD or No-PD groups on any TCI subscales.

Changes in the TCI were examined within groups over three years. A one-way repeated measures ANOVA compared harm avoidance and self-directedness scores at one-year and three-year follow-ups. There was a significant effect for harm avoidance in the B-PD [Wilks’ Lambda = 0.34, F(2, 14) = 13.88, p<0.001, multivariate partial eta squared = 0.67] and No-PD groups [Wilks’ Lambda =0.67, F(2, 34) = 8.5, p<0.001, multivariate partial eta squared = 0.33]. Self-directedness also showed significant within-group effects in the No-PD group across three years [Wilks’ Lambda = 0.51, F(2, 34) = 16.36, p<0.001, multivariate partial eta squared = 0.49]. Despite an increase of one standard deviation in self-directedness, the B-PD group had a smaller effect size than the no
personality disorder group [Wilks’ Lambda = 0.59, $F(2, 14) = 4.8$, $p<0.03$, multivariate partial eta squared = 0.41]. The Other-PD group showed no significant within-group changes in harm avoidance or self-directedness across three years.
Table 6.4: Personality characteristics at pre-treatment, one-year and three-year follow-up divided by the presence of BPD, Other-PD or No-PD

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<th>TCI subscales</th>
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</table>
This study examined the specific impact of borderline personality disorder in a sample of women with BN at pre-treatment and outcome at one and three-year follow-up. The sample was split into three groups: B-PD, Other-PD and No-PD. Overall, despite having a more severe clinical presentation at pre-treatment, women with BN and comorbid B-PD did not have a worse eating disorder or global functioning outcome at three-years post-treatment than those with Other-PDs or No-PDs.

There was some support for B-PD having a negative impact on the clinical characteristics in these women with BN compared to Other-PD and No-PD. Women with BN and B-PD had increased Axis I comorbidity, poorer global functioning and more severe eating disorder attitudes at pre-treatment. Despite this, the present study does not support previous research which suggests that women with bulimia and comorbid borderline personality disorder have “dismal prospects” and poorer outcome (Johnson et al., 1990; Rosenvinge et al., 2000; Steiger & Stotland, 1996; Wonderlich & Mitchell, 1997). On the contrary, the present study contributes to a growing literature that shows the presence of borderline personality disorder is not associated with poorer outcome in BN at long-term follow-up (Fallon et al., 1991; Norring, 1993; Wonderlich et al., 1994). A notable difference in the present study was the rate of improvement in the B-PD group. In contrast to findings from other studies (Herzog et al., 1991; Steiger et al., 1993; Steiger & Stotland, 1996), the B-PD group in the present study showed improvements at the same rate as the Other-PD and No-PD groups by one-year follow-up. The lack of adverse effects of borderline personality disorder comorbidity has also been seen in other clinical groups. For example, borderline personality disorder has
been associated with a more favourable outcome in major depression than those with other personality disorders (Joyce et al., 2003).

The low self-directedness and high harm avoidance personality traits seen at pre-treatment in both personality disorder groups are characteristic of personality profiles previously described in BN (Bulik et al., 1995a). However, findings of this present study did not support the hypothesis that personality traits differed at pre-treatment and follow-up in women with BN and B-PD compared to Other-PDs and No-PD. In the present study the only trait specifically associated with the B-PD group was lower cooperativeness. Self-directedness, reported to be a predictor of outcome in BN (Bulik et al., 1998a), improved the most in the No-PD and B-PD groups by three-year outcome. Interestingly, there was a trend for self-directedness to be lower in Other-PDs \( (p=0.01) \) at three-year follow-up, however this did not reach statistical significance. No significant differences in personality profiles at three-year follow-up were found between the B-PD and No-PD group.

A strength of the study was the independent assessment of personality disorder diagnosis by non-treating clinicians at pre-treatment. By keeping the treating clinician blind to any personality disorder diagnosis, potential bias that may have occurred during therapy was minimised. A strength of the treatment trial was that the B-PD group had the same amount of therapy as those with Other-PDs and No-PD, allowing direct comparisons of the extent of change in each group given the same amount of therapy. This is in contrast to much of the literature which recommends longer, more intensive therapy sessions for participants with BN and comorbid borderline personality disorder (Sansone & Sansone, 2010a; Steiger & Stotland, 1996). Despite the relatively short
course of therapy, those with B-PD had “caught up” in all measures to the No-PD group by one-year follow-up.

6.6.1 Limitations

There were several limitations in the present study. It has been reported that clinical samples are more likely to attract participants with comorbid disorders seeking treatment for one disorder or another, therefore results may not be generalisable to a community sample (Berkson, 1946; Vitousek & Stumpf, 2005). The personality disorder assessment was completed at pre-treatment during the acute phase of the eating disorder, increasing the risk of over-diagnosis of personality disorder as some state variables may be confused with trait variables (Vitousek & Stumpf, 2005). Despite this, the timing of the personality disorder assessment allows examination of change in the groups over time.

6.7 CONCLUSIONS

Although women with BN and borderline personality disorder have a worse clinical presentation at pre-treatment, they do not have a poorer outcome over three years of follow-up in comparison to women with BN and other comorbid personality disorders or women with BN without a personality disorder. The B-PD group improved to the same level after treatment as the Other-PD and No-PD groups. Few differences in personality profiles between the B-PD and No-PD group were evident at follow-up. The present study lends further support to a growing literature suggesting comorbid BN and borderline personality disorder do not predict poor outcome.
In addition, these results show a borderline personality disorder diagnosis to be only moderately stable and to decline over time. Further evidence of this can be seen in improvement in self-directedness which is consistent with the loss of the borderline personality disorder diagnosis at follow-up (Joyce et al., 2003; Mulder, Joyce, Sullivan, Bulik, & Carter, 1999). Future research should closely examine the factors that may influence remission of borderline personality disorder.
Chapter 7: The Impact of Avoidant Personality Disorder on BN

7.1 Overview

This chapter examines one of the most understudied comorbidities between BN and avoidant personality disorder. Because this is an unexplored area, the aims of the current chapter are to investigate the impact of avoidant personality disorder on the clinical characteristics and outcome three-years post-treatment for BN. This chapter will also control for the additional complexities of social phobia and any lifetime mood disorder that occur within this context.

7.2 Introduction

Avoidant personality disorder is one of the most prevalent personality disorders in the eating disorder literature (Sansone et al., 2005) but has received relatively little attention in contrast to borderline personality disorder (Grilo, 2004). Few studies have examined the impact of avoidant personality disorder on pre-treatment presentation and extent of change in psychopathology associated with BN following treatment. The DSM-IV reports prevalences of avoidant personality disorder between 0.5% and 1.0% in the general population however, across eating disorder diagnoses this increases to 16% to 27% making it one of the most common personality disorder diagnoses in the eating disorder population (Sansone & Levitt, 2005b).
Studies examining the impact of personality psychopathology co-occurring with an eating disorder have produced mixed findings on treatment outcome (Keel & Mitchell, 1997). Although, most of the research in this area has been conducted on Cluster B personality disorders, more specifically borderline personality disorder (Johnson et al., 1990; Rosenvinge et al., 2000; Steiger & Stotland, 1996; Wonderlich et al., 1994). In a two-year follow-up study of adults with chronic eating disorders, patients with avoidant personality disorder were found to have consistently higher levels of distress at pre-treatment, discharge, one-year follow-up and two-year follow-up compared to patients with borderline and obsessive-compulsive personality disorders. However, the extent of improvement in eating disorder symptoms was the same across the groups (Ro, Martinsen, Hoffart, Sexton, & Rosenvinge, 2005). In one of the few studies to focus specifically on BN and comorbid avoidant personality disorder, Bruce et al. (2004) compared three groups of women; women with BN and comorbid avoidant personality disorder (BNA+), women with BN but without avoidant personality disorder (BNA-) and control women, on eating symptoms, psychopathology, personality pathology, behavioral inhibition and disinhibition and serotonergic function (Bruce, Steiger, Koerner, Israel, & Young, 2004). Results from this study found the BNA+ group had higher levels of submissiveness, affective instability, self-harming behaviors and greater emotional restriction compared with the BNA- and control groups. Of note, however, no differences were found between the groups on eating symptomatology and psychiatric comorbidity. These results suggest poorer global functioning rather than elevated eating-related psychopathology in individuals with BN and avoidant personality disorder. This finding is largely supported by other studies suggesting personality disorders are more closely associated with deficits in general functioning than severity.
of eating disorder symptoms (Bruce & Steiger, 2005; Cassin & von Ranson, 2005; Grilo, 2002).

A comprehensive understanding of the role of avoidant personality disorder in BN must also consider carefully the presence of social phobia. Not only is social phobia commonly comorbid in individuals with BN (Swinbourne & Touyz, 2007) but, comorbid avoidant personality disorder has been reported in between 29% to 70% in studies with samples of patients with social phobia (Dyck, Phillips, Warshaw, Dolan, Shea, Stout, Massion, Zlotnick, & Keller, 2001; Hofmann, 1995; Perugi, Nassini, Socci, Lenzi, Toni, Simonini, & Akiskal, 1999; Sanderson et al., 1994). One perspective places social phobia and avoidant personality disorder on a spectrum rather than considering them as distinct disorders (Herbert, Hope, & Bellack, 1992; Ralevski, Sanislow, Grilo, Skodol, Gunderson, Shea, Yen, Bender, Zanarini, & McGlashan, 2005; Rettew, 2000; Widiger, 1992). Ralevski et al. (2005) examined whether patients with avoidant personality disorder experienced greater severity of symptoms if social phobia was co-occurring (avoidant personality disorder/social phobia; Ralevski et al., 2005). Comparisons between Axis I and Axis II comorbidity, personality traits, diagnostic stability, treatment utilization and functional impairment were made at pre-treatment and two-year follow-up. Results showed little difference in the severity of symptoms between the avoidant personality disorder and avoidant personality disorder/social phobia groups.

Mood disorders are also commonly comorbid with avoidant personality disorder. In a study of Axis I and Axis II comorbidity, Oldham et al., (1995) reported that prevalences of mood disorder were three times more likely to occur in those with
avoidant personality disorder than without (Alnaes & Torgersen, 1988; Jackson, Whiteside, Bates, Bell, & et al., 1991; Oldham, Skodol, Kellman, Hyler, Doidge, Rosnick, & Gallaher, 1995). Furthermore, Cluster C personality disorders have been found to predict mood disorder chronicity (Iacoviello, Alloy, Abramson, Whitehouse, & Hogan, 2007). Due to the frequent comorbidity of social phobia and mood disorders with avoidant personality disorder, studies of avoidant personality disorder must also consider possible confounding of these comorbidities on symptom presentation and outcome findings.

Despite the high prevalence of avoidant personality disorder, there is still limited understanding of the specific impact of avoidant personality disorder in women with BN at pre-treatment and more importantly on the medium to long-term outcome of CBT for BN. Better understanding of this impact has the potential to assist in improving treatment outcomes for the subset of women with this commonly presenting pattern of comorbidity.

### 7.3  AIMS

**Aim 1:** To compare pre-treatment characteristics of women with BN and avoidant personality disorder (AV-PD), other personality disorders (Other-PD) and no personality disorders (No-PD) groups

**Aim 2:** To examine the impact of AV-PD on BN outcome three-years post-treatment and compare this to Other-PD and No-PD groups
Aim 3: To attempt to examine the relative impact of social phobia and any lifetime mood disorders on the outcome treatment for BN

7.4 METHODS

7.4.1 Overview

The BTS, described earlier in Chapter 4, was used for these analyses. Details of the study design and outcome, and three-year follow-up data have been presented elsewhere (Bulik et al., 1998a; Carter et al., 2003a).

7.4.2 Participants

Participants were 134 women aged 17-45 years with a current DSM-III-R diagnosis of BN. Of the 135 participants entering the study, one was excluded from the analyses as Axis II data were missing. Exclusion criteria were outlined in Chapter 4.

7.4.3 Measures and procedure

This study received ethical approval from the Southern Regional Health Authority (Canterbury) and the University of Canterbury Ethics Committee. Participants provided written informed consent.

7.4.3.1 Pre-treatment assessment

As described earlier, non-treating clinicians undertook assessments for Axis I and II disorders using the SCID-I and II for the DSM-III-R (Spitzer, 1990), eating disorder symptoms such as binging and purging frequency (CBSI), current depression severity (HDRS) and global functioning (GAF) over the past week. Participants completed self-
report questionnaires including the EDI-2 and the TCI. These measures were described in detail in Chapter 4.

7.4.3.2 Follow-up assessment

Participants were reassessed at one-year and three-year follow-up. Follow-up assessments consisted of a structured interview for bulimic symptoms, HDRS, GAF, EDI (short version) and CBSI.

7.4.3.3 Statistical analysis

Participants were divided into groups based on the presence of avoidant personality disorder (AV-PD), the presence of other personality disorders (Other-PD) or the absence of any personality disorder (No-PD). Those with a diagnosis of avoidant personality disorder were included in the AV-PD group regardless of the presence of another personality disorder. There were seven cases in which avoidant personality disorder did not co-occur with any other personality disorders. Of the Other-PD groups, all diagnoses were included except for passive-aggressive and self-defeating personality disorders.

The Statistical Package for the Social Sciences (SPSS, Version 12) was used to analyse data. Chi-square tests were used to compare prevalences of Axis I disorders among AV-PD, Other-PD and No-PD groups. Analysis of variance with post-hoc pairwise comparisons and repeated measures ANOVA were conducted to compare pre-treatment scores and changes over three years in outcome measures among groups, and on absolute scores across the three groups at post-treatment (mentioned in text only). Any differences identified between AV-PD and the Other-PD or No-PD groups were further
explored using multiple regression to determine if AV-PD was independently responsible for these differences or whether they might be explained by the high comorbidity of AV-PD with specific Axis I disorders. Multiple regression was undertaken using the presence or absence of AV-PD, social phobia, any mood disorder and alcohol abuse/dependence as fixed factors and each outcome variable as the dependent variable. Paired t-tests were conducted to compare change on key variables from pre-treatment to one-year follow-up, and from one-year to three-year follow-up within the entire cohort. Statistical significance was considered at the two-tailed $p<0.05$ level.

7.5 RESULTS

7.5.1 Pre-treatment differences between AV-PD, Other-PD and No-PD

Pre-treatment characteristics of the BTS sample as a whole have previously been described in Chapter 6. Diagnosis of an Axis II disorder was present in 56% of the sample. The three most common personality disorders were borderline (28%), avoidant (28%) and paranoid (24%). Of the 75 individuals with a personality disorder diagnosis, 38 met criteria for AV-PD and 37 were diagnosed with having Other-PDs. The mean number of personality disorder diagnoses for the AV-PD and Other-PD groups were 2.9 and 2.2 respectively. The AV-PD group showed high Axis II comorbidity with paranoid (50%) and borderline (42%) personality disorders. The Other-PD group was largely made up of borderline (60%) and histrionic (46%) personality disorders.

Table 7.1 shows that the AV-PD and Other-PD groups had higher prevalence of any mood disorder and alcohol abuse/dependence at pre-treatment than those with No-PD.
In addition the AV-PD group showed a high comorbidity with any anxiety disorder, specifically social phobia and simple phobia.
Table 7.1: Axis I comorbidity of subjects with BN divided by the presence of AV-PD, Other-PD and No-PD

<table>
<thead>
<tr>
<th>Lifetime Axis I Comorbidity</th>
<th>AV-PD (N=37)</th>
<th>Other-PD (N=37)</th>
<th>No PD (N=60)</th>
<th>$x^2$</th>
<th>$p$</th>
<th>Post Hoc</th>
</tr>
</thead>
<tbody>
<tr>
<td>Any Mood Disorder</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Any Mood Disorder</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Major Depressive Disorder</td>
<td>23 62</td>
<td>19 51</td>
<td>25 42</td>
<td>3.9</td>
<td>NS</td>
<td></td>
</tr>
<tr>
<td>Bipolar II Disorder</td>
<td>10 27</td>
<td>9 24</td>
<td>3 5</td>
<td>10.4</td>
<td>0.006</td>
<td>AV-PD, Other-PD&gt;No-PD</td>
</tr>
<tr>
<td>Any Anxiety Disorder</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Any Anxiety Disorder</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Obsessive Compulsive Disorder</td>
<td>2 5</td>
<td>0 0</td>
<td>1 2</td>
<td>2.6</td>
<td>NS</td>
<td></td>
</tr>
<tr>
<td>Panic Disorder</td>
<td>3 8</td>
<td>3 8</td>
<td>6 10</td>
<td>0.15</td>
<td>NS</td>
<td></td>
</tr>
<tr>
<td>Agoraphobia without panic</td>
<td>2 5</td>
<td>0 0</td>
<td>0 0</td>
<td>5.3</td>
<td>NS</td>
<td></td>
</tr>
<tr>
<td>Social Phobia</td>
<td>23 62</td>
<td>8 22</td>
<td>11 18</td>
<td>22.7</td>
<td>0.001</td>
<td>AV-PD&gt;Other-PD, No-PD</td>
</tr>
<tr>
<td>Simple Phobia</td>
<td>17 46</td>
<td>10 27</td>
<td>8 13</td>
<td>12.6</td>
<td>0.001</td>
<td>AV-PD&gt;Other-PD, No-PD</td>
</tr>
<tr>
<td>Any Substance Abuse/Dependence</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Any Substance Abuse/Dependence</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Alcohol Abuse / Dependence</td>
<td>21 57</td>
<td>22 60</td>
<td>18 30</td>
<td>10.6</td>
<td>0.005</td>
<td>Other-PD, AV-PD&gt;No-PD</td>
</tr>
<tr>
<td>Cannabis Abuse/Dependence</td>
<td>13 35</td>
<td>9 24</td>
<td>7 12</td>
<td>7.7</td>
<td>0.03</td>
<td>AV-PD&gt;No-PD</td>
</tr>
<tr>
<td>Stimulant Abuse/Dependence</td>
<td>7 19</td>
<td>4 11</td>
<td>4 7</td>
<td>3.5</td>
<td>NS</td>
<td></td>
</tr>
</tbody>
</table>

Notes: Statistics are Chi-Square.
Figure 7.1 is a Venn diagram demonstrating the extent of comorbidity among AV-PD, social phobia and any lifetime mood disorder within this sample of women with BN. There were few ‘pure’ cases of avoidant personality disorder (n=1) or social phobia (n=5). The common pattern of comorbidities were any mood disorder and avoidant personality disorder (n=14), any mood disorder and social phobia (n=14). The combination of all three disorders (avoidant personality disorder, social phobia and any mood disorder; n=20) was even higher. There were 35 individuals in the sample who did not have any of these comorbid disorders.

Analysis of eating disorder symptoms (Table 7.2) yielded no significant differences in binge or purge frequency across the three groups at pre-treatment. Significantly higher levels of the drive for thinness and body dissatisfaction EDI-2 subscales were seen in both the AV-PD and Other-PD groups, relative to the No-PD group.

At pre-treatment, depressive symptoms from the HDRS and global functioning as rated by the GAF were significantly poorer in the AV-PD group than the Other-PD and No-PD groups.

7.5.2 Impact on outcome at follow-up

Of the 134 women, follow-up data were available for 101 (75%) women at one-year follow-up and 112 (84%) at three-year follow-up. There were no significant differences across the groups in the drop-out rate at one-year (AV-PD = 8, Other-PD = 11, No-PD = 14, $x_2^2 = 0.81, p = 0.67$) or three-year follow-up (AV-PD = 7, Other-PD = 6, No-PD = 9, $x_2^2 = 0.17, p = 0.92$).
Table 7.2 demonstrates the pre-treatment level and changes from pre-treatment to one-year follow-up and from one-year to three-year follow-up for each group. Paired t-tests confirm the entire cohort had statistically significant changes from pre-treatment to one-year follow-up on all outcome variables. Binge and purge frequency and all EDI (short version) subscales showed no significant differences in extent of change across the groups from pre-treatment to one-year follow-up. Diagnosis of current BN or any current eating disorder at one-year follow-up was not significantly different across the groups. Depressive symptoms in the AV-PD group showed a significant decrease from pre-treatment to one-year follow-up compared with the Other-PD and No-PD groups. Changes in global functioning did not differ significantly across the groups from pre-treatment to one-year follow-up.

Results from paired t-tests for the period from one-year to three-year follow-up showed no significant changes in any outcome variable in the cohort (Table 7.2). Binge or purge frequency and EDI (short version) subscales continued to show no differences in the extent of change across the groups over this time. The presence of any eating disorder diagnosis at three-year follow-up did not differ across the three groups (AV-PD = 29%, Other-PD = 26%, No-PD = 35%, \( x^2 = 0.89, p = 0.64 \)). There were no significant differences among the groups in the percentage of individuals still receiving a diagnosis of BN at three-year follow-up (AV-PD = 13%, Other-PD = 7%, No-PD = 22%, \( x^2 = 3.6, p = 0.17 \)).
Note: AVPD=Avoidant personality disorder. Any mood represents a lifetime diagnosis of MDD or bipolar II. All percentages are in reference to the full sample (n=134)

Figure 7.1: Combinations of Axis I comorbidity in the BN sample
Table 7.2: Symptom measures and change over three-year follow-up in women with BN divided by the presence of AV-PD, Other-PD and No-PD

<table>
<thead>
<tr>
<th>Symptom measures</th>
<th>Rate of change</th>
<th>AV-PD</th>
<th>Other-PD</th>
<th>No-PD</th>
<th>F</th>
<th>P</th>
<th>Post Hoc</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>M</td>
<td>SD</td>
<td>M</td>
<td>SD</td>
<td>M</td>
<td>SD</td>
</tr>
<tr>
<td>Binge Frequency</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>pre-treatment scores&lt;sup&gt;a&lt;/sup&gt;</td>
<td>10.1</td>
<td>12.0</td>
<td>9.3</td>
<td>9.1</td>
<td>11.8</td>
<td>11.4</td>
<td>0.67</td>
</tr>
<tr>
<td>pre-treatment to one-year follow-up&lt;sup&gt;b&lt;/sup&gt;</td>
<td>-8.1</td>
<td>13.1</td>
<td>-6.7</td>
<td>8.0</td>
<td>-10.0</td>
<td>11.6</td>
<td>0.73</td>
</tr>
<tr>
<td>one-year to three-year follow-up&lt;sup&gt;c&lt;/sup&gt;</td>
<td>-0.60</td>
<td>3.1</td>
<td>-0.52</td>
<td>4.4</td>
<td>.66</td>
<td>6.6</td>
<td>0.61</td>
</tr>
<tr>
<td>Purge Frequency</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>pre-treatment scores</td>
<td>18.2</td>
<td>30.0</td>
<td>11.6</td>
<td>9.7</td>
<td>13.4</td>
<td>12.9</td>
<td>1.2</td>
</tr>
<tr>
<td>pre-treatment to one-year follow-up&lt;sup&gt;b&lt;/sup&gt;</td>
<td>-14.9</td>
<td>33.6</td>
<td>-6.4</td>
<td>6.9</td>
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<td>1.2</td>
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<tr>
<td>one-year to three-year follow-up&lt;sup&gt;c&lt;/sup&gt;</td>
<td>-0.48</td>
<td>3.4</td>
<td>-1.6</td>
<td>5.3</td>
<td>-0.34</td>
<td>7.0</td>
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<tr>
<td>EDI subscales</td>
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<tr>
<td>Drive for thinness</td>
<td>pre-treatment scores&lt;sup&gt;a&lt;/sup&gt;</td>
<td>15.6</td>
<td>3.8</td>
<td>15.2</td>
<td>3.7</td>
<td>12.4</td>
<td>5.0</td>
</tr>
<tr>
<td>pre-treatment to one-year follow-up&lt;sup&gt;b&lt;/sup&gt;</td>
<td>-8.4</td>
<td>5.4</td>
<td>-7.8</td>
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<td>4.2</td>
<td>.5</td>
<td>5.1</td>
<td>1.2</td>
</tr>
<tr>
<td>Bulimia</td>
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<tr>
<td>pre-treatment scores&lt;sup&gt;a&lt;/sup&gt;</td>
<td>10.7</td>
<td>4.2</td>
<td>9.1</td>
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<td>-5.3</td>
<td>4.9</td>
<td>-7.3</td>
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<tr>
<td>one-year to three-year follow-up&lt;sup&gt;c&lt;/sup&gt;</td>
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<td>3.6</td>
<td>-1.2</td>
<td>3.0</td>
<td>.56</td>
<td>6.3</td>
<td>0.94</td>
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<sup>a</sup> pretreatment scores, <sup>b</sup> pre-treatment to one-year follow-up, <sup>c</sup> one-year to three-year follow-up.
Table 7.2: Continued

<table>
<thead>
<tr>
<th>Symptom measures</th>
<th>AV-PD</th>
<th>Other-PD</th>
<th>No-PD</th>
<th>F</th>
<th>P</th>
<th>Post Hoc</th>
</tr>
</thead>
<tbody>
<tr>
<td>Body dissatisfaction</td>
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<td></td>
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<tr>
<td>pre-treatment scores</td>
<td>21.7</td>
<td>6.4</td>
<td>20.3</td>
<td>6.6</td>
<td>15.6</td>
<td>7.8</td>
</tr>
<tr>
<td>pre-treatment to one-year follow-up</td>
<td>-7.4</td>
<td>8.7</td>
<td>-8.6</td>
<td>9.0</td>
<td>-6.9</td>
<td>7.1</td>
</tr>
<tr>
<td>one-year to three-year follow-up</td>
<td>-2.3</td>
<td>5.3</td>
<td>-1.7</td>
<td>5.7</td>
<td>.08</td>
<td>6.7</td>
</tr>
<tr>
<td>HDRS</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>pre-treatment scores</td>
<td>12.2</td>
<td>5.6</td>
<td>8.9</td>
<td>5.2</td>
<td>6.1</td>
<td>4.7</td>
</tr>
<tr>
<td>pre-treatment to one-year follow-up</td>
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<td>-2.4</td>
<td>7.2</td>
<td>-2.3</td>
<td>4.6</td>
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<td>7.5</td>
<td>-1.8</td>
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<td></td>
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<td>57.0</td>
<td>5.7</td>
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<tr>
<td>pre-treatment to one-year follow-up</td>
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<td>12.8</td>
<td>10.5</td>
<td>13.5</td>
<td>14.6</td>
<td>10.4</td>
</tr>
<tr>
<td>one-year to three-year follow-up</td>
<td>-3.7</td>
<td>13.7</td>
<td>2.3</td>
<td>17.4</td>
<td>-.14</td>
<td>12.9</td>
</tr>
</tbody>
</table>

Note: A negative result shows a decrease in score and a positive result shows an increase in score. The mean is significant at the .05 level. The EDI-2 was used at pre-treatment and the EDI (short version) was used at follow-up.

a = Participant numbers at pre-treatment for all variables were: AV-PD (n=37), Other-PD (n=37), No-PD (n=60); b= participant numbers from pre-treatment to 1-year follow-up were: AV-PD (n=29), other personality disorder (n=21), no personality disorder (n=44); c= participant numbers from 1-year to 3-year follow-up were: AV-PD (n=23), other personality disorder (n=14), no personality disorder (n=39).
Although there was no difference in the extent of change from one-year to three year follow-up across the groups (Table 7.2), notably at three-year follow-up there were significant differences between the AV-PD and No-PD groups in scores for the HDRS ($F_{2,109}=3.8$, $p=0.03$, AV-PD>No-PD) and GAF ($F_{2,109}=3.4$, $p=0.04$, AV-PD>No-PD). This finding highlights that despite the AV-PD group improving to the same extent as the Other-PD and No-PD groups, the AV-PD group remained significantly more impaired in depressive symptoms and global functioning three-years post treatment.

7.5.3 Controlling for Axis I disorders

A series of multiple regression analyses were conducted on all significant pre-treatment results and changes in the HDRS from pre-treatment to one-year follow-up, to control for the high comorbidity of Axis I disorders (Table 7.3). These analyses involved using the outcome variables as the dependent variables and avoidant personality disorder, social phobia, any mood disorder and alcohol abuse/dependence as independent variables. At pre-treatment, the EDI-2 subscales of drive for thinness and body dissatisfaction were predicted by the presence of any lifetime mood disorder. Current depressive symptomatology at pre-treatment was significantly associated with AV-PD and as expected, with any lifetime mood disorder. Global functioning was predicted by the presence of any lifetime mood disorder at pre-treatment.

The presence of AV-PD was the only comorbid diagnosis predicting improvement in depressive symptoms from pre-treatment to one-year follow-up. Social phobia, despite being highly comorbid with AV-PD, did not predict improvement in any of the variables.
Table 7.3: Multiple regression examining the impact of specific comorbid diagnoses in key symptom measures

<table>
<thead>
<tr>
<th>Symptom Measures</th>
<th>AV-PD (n=38)</th>
<th>Social Phobia (n=42)</th>
<th>Any Mood (n=90)</th>
<th>Alcohol Abuse/Dependence (n=61)</th>
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</thead>
<tbody>
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<td>F</td>
<td>p</td>
<td>F</td>
<td>p</td>
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<td>0.16</td>
<td>0.69</td>
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<td>Body dissatisfaction</td>
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<td><strong>0.05</strong></td>
<td>0.04</td>
<td>0.84</td>
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<td>HDRS</td>
<td>9.4</td>
<td><strong>0.003</strong></td>
<td>3.1</td>
<td>0.08</td>
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<td>GAF</td>
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<td><strong>Pre-treatment to 1-year follow-up</strong></td>
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<td></td>
</tr>
<tr>
<td>HDRS</td>
<td>4.8</td>
<td><strong>0.03</strong></td>
<td>0.10</td>
<td>0.76</td>
</tr>
</tbody>
</table>
7.6 DISCUSSION

This study has three main findings: First, AV-PD is associated with greater global and psychiatric impairment but not with greater eating disorder severity at pre-treatment. Second, recovery patterns for eating disorder related symptoms over one and three-years are not affected by the presence of AV-PD however, depressive symptoms continue to persist post-treatment in the AV-PD group. Third, additional analyses clarify that much of the impact on depressive symptoms and global functioning can be largely attributed to the presence of any lifetime mood disorder or the combination of mood disorder with AV-PD.

Findings from this study can be integrated into the growing literature that show personality disorders have little impact on eating disorder symptoms whilst negatively influencing depressive symptomatology and global functioning (Cassin & von Ranson, 2005; Grilo, 2002; Steiger & Stotland, 1996; Steiger, Thibaudeau, Leung, Houle, & Ghadirian, 1994). In combination, these findings of lack of impact of personality disorder on BN outcome challenge clinical assumptions that patients with comorbid BN and personality disorders have worse treatment outcomes and may provide reassurance to eating disorder clinicians who treat women with comorbid Axis II disorders.

In one of the few prospective outcome studies of BN and personality disorder, Grilo et al., (2007) found the presence of personality disorder overall had little influence on the natural course of BN regarding risk of relapse and remission at five-year follow-up. They did report however, that increasing numbers of avoidant personality disorder symptoms predicted a small reduction in the risk ratio for remission while borderline, obsessive-compulsive or schizotypal personality disorder symptoms did not.
Binge eating and purging were relatively unaffected by the presence of AV-PD and appeared to improve to the same extent in the AV-PD group as the Other-PD and No-PD groups at follow-up. Although AV-PD did not adversely affect eating disorder symptoms, it did have a specific adverse impact on mood over and above that seen in the Other-PD group. This finding highlights the importance of monitoring mood in this group. Most improvements in mood occurred from pre-treatment to one-year follow-up, particularly in the AV-PD group. Although the subsequent extent of change did not differ among groups, the long-term follow-up indicated that those with AV-PD were still doing worse in terms of depression level and global functioning than the other two groups. However, once Axis I comorbidity was controlled for, these analyses indicated that almost all significant findings were related to the presence of a lifetime mood disorder or in several cases, mood comorbidity with AV-PD. The high comorbidity of mood disorders in those with BN has been well recognised (Godart et al., 2007), as has frequent improvement in mood symptoms with successful treatment for BN symptoms (Bulik et al., 1998a; Mitchell, Pyle, Eckert, Hatsukami, Pomeroy, & Zimmerman, 1990; Openshaw, Waller, & Sperlinger, 2004). Indeed, improvement in depressive symptoms from pre-treatment to one-year follow-up did occur in this study, particularly in the AV-PD group; however at three-year follow-up the AV-PD group remained more depressed than those in this sample without AV-PD. It may be that in those with avoidant personality disorder, generalisation of therapeutic benefit from CBT aimed at eating symptoms to wider psychopathology such as mood and other life problems, is hindered due to the avoidant personality style, leading to persistence of depression. This could be related to failure to engage in potentially reinforcing life activities which would assist in improving depressive symptoms (Jacobson, 2001; Lewinsohn, 1973). The findings from
this study suggest that for those with BN and avoidant personality disorder, greater attention needs to be paid to mood treatment and longer treatments may be required to allow for multiple foci of treatment when such complex patterns of comorbidity occur.

Although this study design does not intentionally address the issue of avoidant personality disorder and social phobia as a spectrum relationship, the lack of any significant findings of social phobia on any symptoms measures, despite its high comorbidity within the AV-PD group, does not support the literature suggesting that avoidant personality disorder and social phobia have a spectrum relationship rather than being distinctly different disorders. It is important to note however, that the relative impact of these comorbidities was being examined within a sample of women with BN so this finding may not generalise to other populations.

A further caution is the high comorbidity of the AV-PD group with other Axis II personality disorders, with only seven participants with avoidant personality disorder as the only personality disorder diagnosis. The potential impact of this might be to reduce the chances of finding differences between the AV-PD group as defined here (with or without other comorbidity) and the Other-PD group.

Avoidant personality disorder is commonly found in the eating disorder population however there has been little research into its impact on presentation or outcome in those with BN. This study contributes to the small literature examining avoidant personality disorder in those with BN. The analyses of symptom change over time gives a more comprehensive picture of the outcome for those with comorbid BN and avoidant personality disorder and the symptoms that continue to persist post-treatment. In doing
so, possible gaps to address in treatment are suggested. These findings show the importance of closely examining and controlling for highly comorbid conditions.

### 7.6.1 Limitations

Despite raising the risk of Type I error through conducting multiple comparisons without correction, we did not find any evidence suggesting an adverse effect of avoidant personality disorder on the longer term outcome of those women with BN. It is possible that a larger sample might detect a small effect, however, for the most part, these results are consistent with existing literature.

The finding that the AV-PD group had a greater number of personality disorders than the Other-PD group suggests that severity of personality psychopathology, rather than the specific influence of avoidant personality disorder, may also be contributing to these results. Future studies could control for severity by using personality disorder symptom counts as a covariate.

Further research is needed to confirm the impact of avoidant personality disorder (with or without depression) in other samples of women with BN and other eating disorders. If confirmed, the need to specifically address mood symptoms in those with avoidant personality disorder is likely to have important clinical implications. Given that a sizeable proportion (28%) of those with eating disorders have avoidant personality disorder, further research on tailored treatments for this subgroup may be warranted.

### 7.7 Conclusions

In conclusion, the presence of comorbid avoidant personality disorder in women with BN had little or no impact on eating symptoms, or the trajectory of symptom change
over follow-up. Despite this, avoidant personality disorder appeared to have a specific impact on the level of depression and global functioning at pre-treatment, however multivariate analysis indicated that most of this effect was related to lifetime depression with or without avoidant personality disorder. The complex comorbidity of Axis I and Axis II disorders needs to be taken into account in research studies and clinical treatment of these with eating disorders.
Chapter 8: Complex Personality Disorder in BN

8.1 OVERVIEW

Research on overlapping Axis II diagnoses and severity is often overlooked in favour of individual diagnoses. The previous chapters examining the comorbidity of BN and borderline personality disorder or avoidant personality disorder have shown an impact on pre-treatment clinical characteristics but not outcome. The aim of this chapter is to broaden the area of eating disorder and personality disorder comorbidity by evaluating the use of Tyrer’s dimensional personality classification system to establish if personality disorder severity influences the course of BN.

8.2 INTRODUCTION

The categorical classification of personality disorders has been the subject of considerable debate for some years (Morey et al., 2007; Oldham, Skodol, Kellman, Hyler, Rosnick, & Davies, 1992; Widiger & Clark, 2000). Limitations of the categorical model have been widely noted and a number of authors have advocated dimensional models for the classification of personality disorders (Livesley & Jang, 2000; Westen & Shedler, 2000; Widiger & Simonsen, 2005). One dimensional approach proposed by Tyrer (2005) and Tyrer and Johnson (1996) uses severity as a means of categorising personality disorders. In an attempt to reconcile the frequently overlapping personality disorder diagnoses, this approach divides groups into a four-point severity scale: no personality disorder (does not meet criteria for actual or subthreshold personality disorder), personality difficulty (meets subthreshold criteria for one or several
personality disorders), simple personality disorder (meets criteria for one or more personality disorders within the same personality disorder cluster) and complex personality disorder (meets criteria for two or more personality disorders, across different clusters (Tyrer, 2005). This model of personality disorder severity has been used to examine the impact on mental disability and outcome in substance use, mood, anxiety and psychotic disorders (Pulay, Dawson, Ruan, Pickering, Huang, Chou, & Grant, 2008; Tyrer, Seivewright, & Johnson, 2004). However, to our knowledge Tyrer’s dimensional classification of personality disorder severity has not previously been used to assess the impact on outcome in an eating disordered group.

The prevalence of personality disorders in BN is reported to range from 21% to 67% (Diaz-Marsa et al., 2000; Godt, 2008; Sansone et al., 2005). The comorbidity of BN and personality disorders has been found to negatively impact clinical symptoms such as general functioning, interpersonal skills and social functioning (Bruce & Steiger, 2005; Johnson et al., 1990). In recent years, research has attempted to address the complexities of treating comorbid eating disorders and personality disorders by adapting psychotherapies such as DBT (Chen, Matthews, Allen, Kuo, & Linehan, 2008), CBT (Fairburn et al., 2009) and IPT (Fairburn, 1997), to attend to the combination of eating disorder symptoms and personality pathology (Bruce & Steiger, 2005). Mixed findings exist on the impact of personality pathology on eating disorder treatment outcome with some studies reporting greater binge eating severity (Picot & Lilenfeld, 2003) and more disturbed psychiatric symptoms post-treatment in those with personality pathology (Johnson et al., 1990; Wonderlich et al., 1994). In contrast, other studies report that the presence of a personality disorder did not predict outcome (Bulik et al., 1998b; Grilo et al., 2007).
The use of Tyrer’s dimensional personality classification attempts to address the issue of multiple personality diagnoses by considering severity in terms of both extent and breadth of personality dysfunction across personality clusters. The present study aims to evaluate the ability of Tyrer’s dimensional approach to predict pathology and outcome in a sample of women with BN participating in a randomised controlled trial of CBT (Bulik et al., 1998a). This will be achieved by: (1) comparing the pre-treatment characteristics of women with BN among the dimensional personality groups; (2) examining the impact of personality disorder severity on BN outcome at one and three-years post-treatment.

8.3 AIMS

**Aim 1:** To determine if complex personality disorder will negatively impact the pre-treatment clinical characteristics in women with BN compared to less severe personality disorders (no personality disorder, personality difficulty and simple personality disorder)

**Aim 2:** To determine if complex personality disorder will negatively impact BN outcome one-year and three-years after cognitive therapy for BN compared to less severe personality disorder groups
8.4 METHODS

8.4.1 Overview

The BTS, described earlier in Chapter 4, was used for these analyses. Details of the study design and outcome, and three-year follow-up data have been presented elsewhere (Bulik et al., 1998a; Carter et al., 2003a).

8.4.2 Participants

Participants were 134 women aged 17-45 years with a current DSM-III-R diagnosis of BN. Of the 135 participants entering the study, one was excluded from the analyses as Axis II data were missing. Exclusion criteria were outlined in Chapter 4.

8.4.3 Procedure

This study received ethical approval from the Southern Regional Health Authority (Canterbury) and the University of Canterbury Ethics Committee. Participants provided written informed consent.

8.4.3.1 Pre-treatment assessment

As described earlier, non-treating clinicians undertook assessments for Axis I and II disorders using the SCID-I and II for the DSM-III-R (Spitzer, 1990), eating disorder symptoms such as binging and purging frequency (CBSI), current depression severity (HDRS) and global functioning (GAF) over the past week. Participants completed self-report questionnaires including the EDI-2 and the TCI. These measures were described in detail in Chapter 4.
8.4.3.2 Follow-up assessment

Participants were reassessed at one and three-years after the end of treatment. Follow-up assessment consisted of re-evaluating eating disorder diagnosis and re-administering CBSI, HDRS, GAF and EDI (short version) measures.

8.4.3.3 Statistical analyses

The Statistical Package for the Social Sciences (SPSS, Version 12) was used to analyse data. Participants were divided into four groups: 1) No-PD (absent or minimal symptoms), 2) Personality Difficulty (subthreshold symptoms, that is, one symptom short of diagnosis), 3) Simple-PD (one or more personality disorders met within the same personality disorder cluster), 4) Complex-PD (two or more personality disorders from different clusters). Personality disorder clusters were: Cluster A (paranoid, schizoid, schizotypal personality disorders), Cluster B (antisocial, borderline, histrionic, narcissistic personality disorders) and Cluster C (avoidant, dependent, obsessive-compulsive personality disorders). Chi-square tests were conducted on dichotomous variables. Fisher’s exact and chi-square tests were used for post-hoc pairwise testing. One-way analyses of variance were conducted to examine the impact of personality pathology on binge and purge frequency, eating attitudes (EDI drive for thinness, bulimia, and body dissatisfaction), depressive symptoms (HDRS) and global functioning (GAF). Repeated measures ANOVA were used to assess differences among the four groups over three time points. To reduce the risk of Type I error we have opted for a more stringent statistical significance level of $p<0.01$ for all statistical testing.
8.5 RESULTS

8.5.1 Characteristics of personality groups at pre-treatment

Pre-treatment characteristics of the BTS sample as a whole have previously been described in Chapter 6. Diagnosis of an Axis II disorder was present in 56% of the sample (n=75). Figure 7.1 shows the overlap of Axis II diagnoses. Of the 75 participants with any personality disorder, 29 had a Simple-PD (mainly Cluster C), while 46 had a Complex-PD. Of those with a Complex-PD, 17 participants had personality disorders from all three clusters and 29 had combinations across two clusters.

Table 8.1 shows that those with Complex-PD had higher prevalence of any lifetime mood disorder than the No-PD and Personality Difficulty groups and higher prevalence of any lifetime anxiety disorder and any lifetime substance abuse/dependence than the other three groups. The Simple-PD group had significantly higher prevalence of lifetime mood disorder than the No-PD group. Prevalence of any lifetime anxiety disorder were significantly higher in the Personality Difficulty group than in the No-PD group.

At pre-treatment there were no significant differences in eating disorder symptoms such as binge eating and purging among the four groups (see Table 8.2). However, those with Simple-PD or Complex-PD had higher scores on drive for thinness and body dissatisfaction subscales of the EDI-2.
Figure 8.1: Comorbidity across personality disorder clusters in women with BN

Notes: Personality disorder clusters were: Cluster A (Paranoid, Schizoid, Schizotypal personality disorders), Cluster B (Antisocial, Borderline, Histrionic, Narcissistic personality disorders), and Cluster C (Avoidant, Dependent, Obsessive-compulsive personality disorders)
Table 8.1: Axis I comorbidity of participants with BN stratified by the presence of No-PD, Personality Difficulty, Simple-PD and Complex-PD

<table>
<thead>
<tr>
<th>Lifetime Axis I Comorbidity</th>
<th>No-PD&lt;sup&gt;a&lt;/sup&gt; (N=32)</th>
<th>Personality&lt;sup&gt;b&lt;/sup&gt; Difficulty (N=27)</th>
<th>Simple-PD&lt;sup&gt;c&lt;/sup&gt; (N=29)</th>
<th>Complex-PD&lt;sup&gt;d&lt;/sup&gt; (N=46)</th>
<th>$\chi^2$</th>
<th>$p$</th>
<th>Post Hoc</th>
</tr>
</thead>
<tbody>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<td>N</td>
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<td>15</td>
<td>22</td>
<td>40</td>
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<td>%</td>
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<td>76</td>
<td>87</td>
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<td>45</td>
<td>70</td>
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</table>

<sup>a</sup>=No-PD group, <sup>b</sup>=Personality Difficulty group, <sup>c</sup>=Simple-PD group, <sup>d</sup>=Complex-PD group

Notes: Post Hoc Statistics are Chi-Square or Fisher’s Exact Test

Abbreviations: PD=personality disorder
<table>
<thead>
<tr>
<th>Outcome measures</th>
<th>No-PD&lt;sup&gt;a&lt;/sup&gt;</th>
<th>Personality Difficulty&lt;sup&gt;b&lt;/sup&gt;</th>
<th>Simple-PD&lt;sup&gt;c&lt;/sup&gt;</th>
<th>Complex-PD&lt;sup&gt;d&lt;/sup&gt;</th>
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<td>M</td>
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<td></td>
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<td>1.9</td>
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</tr>
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</tr>
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<td>Drive for Thinness</td>
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</tr>
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<td>three-year follow-up</td>
<td>8.0</td>
<td>1.6</td>
<td>7.3</td>
<td>1.1</td>
<td>8.8</td>
</tr>
</tbody>
</table>

a=No-PD group, b=Personality Difficulty group, c=Simple-PD group, d=Complex-PD group

Note: Participant numbers for pre-treatment were: No-PD (n=32), Personality Difficulty (n=27), Simple-PD (n=29), Complex-PD (n=46); Participant numbers for 1-year follow-up were: No-PD (n=27), Personality-Difficulty (n=18), Simple-PD (n=22), Complex-PD (n=34); Participant numbers for 3-year follow-up were: No-PD (n=28), Personality Difficulty (n=22), Simple-PD (n=24), Complex-PD (n=38)
Table 8.3: Global functioning and depressive symptomatology at pre-treatment, one-year and three-year follow-up

<table>
<thead>
<tr>
<th>Outcome measures</th>
<th>No-PD a</th>
<th>Personality Difficulty b</th>
<th>Simple-PD c</th>
<th>Complex-PD d</th>
<th>Statistics</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>M</td>
<td>SE</td>
<td>M</td>
<td>SE</td>
<td>M</td>
</tr>
<tr>
<td>GAF</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>pre-treatment</td>
<td>56.1</td>
<td>0.8</td>
<td>58.0</td>
<td>1.3</td>
<td>56.9</td>
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<tr>
<td>one-year follow-up</td>
<td>73.8</td>
<td>2.2</td>
<td>67.6</td>
<td>2.6</td>
<td>65.9</td>
</tr>
<tr>
<td>three-year follow-up</td>
<td>74.4</td>
<td>2.6</td>
<td>66.8</td>
<td>3.1</td>
<td>64.5</td>
</tr>
<tr>
<td>HDRS</td>
<td></td>
<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>pre-treatment</td>
<td>6.0</td>
<td>0.9</td>
<td>6.1</td>
<td>0.8</td>
<td>9.7</td>
</tr>
<tr>
<td>one-year follow-up</td>
<td>3.0</td>
<td>0.6</td>
<td>4.8</td>
<td>1.0</td>
<td>7.0</td>
</tr>
<tr>
<td>three-year follow-up</td>
<td>4.8</td>
<td>0.9</td>
<td>4.5</td>
<td>0.9</td>
<td>8.7</td>
</tr>
</tbody>
</table>

a=No-PD group, b=Personality Difficulty group, c=Simple-PD group, d=Complex-PD group

Note: 1Participant numbers for pre-treatment were: No-PD (n=32), Personality Difficulty (n=27), Simple-PD (n=29), Complex-PD (n=46); 2Participant numbers for 1-year follow-up were: No-PD (n=27), Personality Difficulty (n=18), Simple-PD (n=22), Complex-PD (n=34); 3Participant numbers for 3-year follow-up were: No-PD (n=28), Personality Difficulty (n=22), Simple-PD (n=24), Complex-PD (n=38)
Significant differences were found in depressive symptoms between No-PD and Complex-PD and Personality Difficulty and Complex-PD groups. The Complex-PD group had considerably higher levels of current depressive severity (HDRS) compared with the No-PD and Personality Difficulty groups. At pre-treatment, there was a small but statistically significant difference in GAF scores between the Personality Difficulty group and the Complex-PD group.

### 8.5.2 Outcome at follow-up for the four groups

Of the 134 women, follow-up data were available for 101 (75%) women at one-year follow-up and 112 (84%) at three-year follow-up. There were no differences in the rate of missing follow-up assessments among the groups at one-year (No-PD = 16%, Personality Difficulty = 33%, Simple-PD = 24%, Complex-PD = 28%), ($\chi^2 = 2.7, p = 0.43$) or three-year follow-up (No-PD = 13%, Personality Difficulty = 19%, Simple-PD = 17%, Complex-PD = 8%), ($\chi^2 = 0.5, p = 0.9$). Ninety-two participants were assessed at all three time points and complete data were available for all participants for the clinical interview, HDRS, and GAF. Self-report questionnaire (EDI-2) data were available for 76 out of the 92 participants at all three time points.

At one-year follow-up there were no significant differences among the four groups in binge or purge frequency, current diagnoses of BN, AN or EDNOS. The overall remission rate from any eating disorder at this time point was 46% (n = 61). Differences among the groups at pre-treatment for eating attitudes (drive for thinness and body dissatisfaction, see Table 8.2), GAF and current depressive symptoms (see Table 8.3) were no longer present.
Similarly, at three-year follow-up, there were no significant differences among the groups in binge and purge frequency, eating disorder diagnosis or eating attitudes. At three-years, the remission rate from any eating disorder was 58% (n = 77). The relapse rate at three-years for the whole sample was 10% (n = 9) and there were no significant differences across the groups (No-PD = 12%, Personality Difficulty = 11%, Simple-PD = 16% and Complex-PD = 4%, $\chi^2 = 9.4, p = 0.40$). Similarly, no significant differences were found in global functioning or depressive symptoms across the groups.

Further analyses were conducted using repeated measures ANOVA to compare changes in outcome measures among the groups at pre-treatment, one-year and three-year follow-up. This revealed no significant difference in changes over time among groups in binge frequency [Group*time interaction, $F_{6, 176} = 0.4, p = 0.9$] or purge frequency [Group*time interaction $F_{6, 176} = 0.7, p = 0.7$]. Eating attitudes showed no significant differences in drive for thinness [Group*time interaction $F_{6, 144} = 0.6, p = 0.7$], bulimia [Group*time interaction $F_{6, 144} = 2.2, p = 0.05$] and body dissatisfaction subscales [Group*time interaction $F_{6, 144} = 0.6, p = 0.7$]. Similarly, there were no significant difference in changes over time among the groups in global functioning [Group*time interaction $F_{6, 174} = 1.5, p = 0.2$] and depressive symptomatology [Group*time interaction $F_{6, 176} = 1.5, p = 0.2$].

8.6 DISCUSSION

This study compared the pre-treatment clinical characteristics of women with BN among Tyrer’s dimensional personality groups and examined the impact of personality disorder severity on BN outcome at one and three-years post-treatment. There were three main findings. First, at pre-treatment, individuals with Complex-PD and BN
showed greater Axis I comorbidity, worse eating attitudes, poorer global functioning and more depressive symptoms than the No-PD, Personality Difficulty and Simple-PD groups. Despite this, the frequency of binge eating and purging were not affected by the presence of Complex-PDs or Simple-PDs. Second, BN outcome (that is, eating behaviours, eating attitudes and diagnosis) showed no significant differences among the groups at one-year and three-years post-treatment, indicating that improvement in eating disorder psychopathology after treatment is relatively unaffected by the number of personality disorders comorbid with BN. Third, although global functioning and depressive symptoms were significantly worse in the Complex-PD and Simple-PD groups at pre-treatment, this was mostly resolved by one-year and three-year follow-up with no significant differences found among the four groups.

Our findings are consistent with previous studies showing that comorbid BN and personality disorders have greater pathology and poorer global functioning at pre-treatment (Steiger & Stotland, 1996; Wonderlich et al., 1994). Unlike those studies that reported poorer treatment response, worse outcome and greater disability associated with Complex-PD (Moran, Walsh, Tyrer, Burns, Creed, & Fahy, 2003; Pulay et al., 2008; Tyrer et al., 2004), we found that significant pre-treatment differences in eating disorder attitudes (drive-for-thinness, body dissatisfaction), global functioning and depressive symptoms disappeared in the Complex-PD group by one-year follow-up and remained absent at three-year follow-up.

Although there was a pre-treatment difference in eating disorder attitudes in this sample, the absence of differences in eating disorder behaviours (binge eating or purging) in relation to personality disorder severity is consistent with earlier studies reporting that
personality disorders have little impact on the severity of eating behaviours (Bulik et al., 1998b; Johnson et al., 1990; Steiger et al., 1994). These results challenge the need for longer psychotherapy or psychotherapy specific to personality disorders within this population. However, this only pertains to the goal of remission of the eating disorder and not the improvement of global and psychiatric functioning or the treatment of the personality disorder. The presence and severity of personality pathology in participants in this psychotherapy trial did not affect their response to relatively brief CBT, with the Complex-PD group indistinguishable at follow-up from the No-PD group.

The present study highlights methodological issues in the assessment and interpretation of personality disorder diagnoses. As a result, some consideration should be given to the lack of impact of severe multiple Axis II diagnoses (Complex-PD) on outcome. Severity and pathology of Complex-PD may be partly illusory due the substantial overlap of personality disorder groups (Fossati, Maffei, Bagnato, Battaglia, Donati, Donini, Fiorilli, Novella, & Prolo, 2000; Oldham et al., 1992). This overlap may misrepresent the severity of personality disorders by increasing the symptoms and diagnoses in the Complex-PD group thus implying that they have greater pathology. This issue relates to some of the fundamental problems with the current classification system of personality disorders and may indirectly affect interpretation of findings. A further consideration is that the outcome of BN may not be affected by the presence of any personality disorder regardless of diagnosis or severity. This adds to the growing body of research that shows no long-term impact of personality disorders on the outcome of BN (Fallon et al., 1991; Grilo et al., 2003; Wonderlich et al., 1994).
8.6.1 Limitations

This study has several limitations. First, there is an absence of repeated personality assessments to establish personality status at follow-up given recent evidence of instability of personality disorder diagnoses over time and after treatment (Grilo, McGlashan, & Skodol, 2000; Lenzenweger, Johnson, & Willett, 2004; Tyrer, 2008). The fact that Complex-PD does not predict outcome in BN may be the result of confounding Axis II diagnosis by acute Axis I distress (Vitousek & Stumpf, 2005). Repeated personality assessments would have allowed a clearer understanding of changes in severity and course of the dimensional personality disorder groups over time. Second, reliability data were not available for our outcome measures however, these raters have conducted inter-rater reliability checks for Axis I and II diagnosis in an anorexia treatment study and a depression study. The overall kappa for these range from 0.78 to 0.85 which suggests good concordance for these raters (Carter et al., 1999; Jordan et al., 2008). Third, the foundation of Tyrer’s approach to personality disorders is based on the validity of current categorical diagnoses, and as such, addresses some but not all of the problem of reliability and stability of personality diagnoses. Finally, although we found that Tyrer’s model suggested that severity of personality pathology does not affect outcome in eating disorders, other methods of classifying personality disorder severity need to be examined for example, total personality disorder symptoms. Although personality disorder severity does not affect the course of an eating disorder, there is still conflicting evidence about whether the nature of the personality disorder may do so (Thompson-Brenner, Eddy, Franko, Dorer, Vashchenko, Kass, & Herzog, 2008). Further replication of this study is needed to establish the validity of Tyrer’s
dimensional personality classification for those with eating disorders and comorbid Axis II disorders.

8.7 CONCLUSIONS

To our knowledge, this is the first study to use Tyrer’s dimensional personality classification to assess the impact of Complex-PD on BN outcomes. We can conclude that despite having slightly poorer functioning at pre-treatment, participants with BN and comorbid Complex-PD did not have worse eating disorder or global functioning outcome one-year and three-years post-treatment compared to those with less severe personality symptoms (Simple-PD and Personality Difficulty) or no personality psychopathology (No-PD). Overall, this study suggests that the number of personality disorders comorbid with BN does not influence the course of an eating disorder. This challenges the idea that treatments such as CBT need to be adapted for those with comorbid eating disorders and personality disorders in order to achieve successful treatment outcomes for an eating disorder.
Chapter 9: Dimensional measures of personality as a predictor of outcome at five-year follow-up in women with BN

9.1 Overview

The dimensional classification system is remerging as a favoured option in examining personality pathology within Axis I disorders. This chapter will explore personality predictors of outcome in BN using dimensional measures. Because there are a broad range of personality measures available, this chapter will assess the predictive validity of three widely-used self-report and interview measures. These are: the TCI, the EDI-2 and personality disorder symptoms from the SCID-II.

9.2 Introduction

The assessment of personality remains an area of ongoing scientific debate with no consensus regarding an optimal approach to categorical and dimensional models. Personality has been implicated in predicting general functioning and symptomatic expression in BN however, there is some debate about its impact on outcome (Grilo et al., 2007; Rossiter et al., 1993; Wonderlich et al., 1994). Some of this discrepancy can be attributed to the different models (categorical and dimensional), different assessment methods (clinical interviews and self-report) and different measures of personality used with this population.

The current categorical model of personality disorders (DSM-IV-TR) has several limitations. Personality disorder symptoms in the categorical model are moderately
heterogeneous, which result in high rates of co-occurring personality disorder diagnoses (Livesley, 1998). Furthermore, the stability of Axis II diagnoses and the reliability of Axis II assessment instruments are relatively poor (Trull & Durrett, 2005). Historically, the dimensional approach has existed alongside the categorical approach however, these limitations have encouraged increased research investigating dimensional models of personality as an alternate approach (Goldner, Srikameswaran, Schroeder, Livesley, & Birmingham, 1999; Morey et al., 2007; Pukrop, 2002; Widiger & Simonsen, 2005). One personality traits model that has been frequently examined is Cloninger’s psychobiological model of temperament and character (Cloninger et al., 1993; Cloninger et al., 1994).

The TCI (Cloninger et al., 1993) examines four dimensions of temperament: novelty seeking, harm avoidance, reward dependence, and persistence; and three dimensions of character: self-directedness, cooperativeness, and self-transcendence (Cloninger et al., 1993). Bulimia is characterised by high harm avoidance, low self-directedness and high novelty seeking (Fassino et al., 2004; Fassino et al., 2002b). In a previous study by Bulik et al., (1998b), self-directedness was a predictor of BN outcome at one-year follow-up. However, this finding was not replicated in a study by Bloks et al (2004) at 2.5 year follow-up (Bloks, Hoek, Callewaert, & van Furth, 2004). Clarifying the role of self-directedness as a predictor of outcome may have important implications for the treatment of BN. For example, it has been suggested that increasing self-directedness as a pre-treatment intervention may be a useful approach (Anderson et al., 2002).

The EDI-2, is one of the most widely used self-report questionnaires in eating disorder research. In addition to the three eating disorder symptom scales (drive-for-thinness,
bulimia and body dissatisfaction), the EDI-2 has eight subscales assessing characteristics relevant to eating disorders, including personality (Garner, 1983). Elevated EDI-2 subscales have been noted in those eating disorder patients with other comorbid disorders such as affective, anxiety and personality disorders (Bizeul, Brun, & Rigaud, 2003; Hinrichsen, 2004; Milos, Spindler, & Schnyder, 2004; Sunday, Levey, & Halmi, 1993). Given the EDI-2 was specifically designed to examine personality features and psychological traits in an eating disorder population, it is surprising that few studies have used this as an outcome measure for these themes.

Future DSMs may consider the inclusion of a dimensional classification system as a new way of conceptualising personality pathology. In relation to this potential change, it would be useful to clarify the link between personality dimensions and their impact on outcome.

9.3 AIMS

Aim 1: To determine whether personality dimensions measured at pre-treatment by the TCI, EDI-2 and DSM-III-R personality symptoms among women with BN predict eating disorder outcome (past year) at five years

Aim 2: To determine whether personality dimensions measured at pre-treatment by the TCI, EDI-2 and DSM-III-R personality symptoms among women with BN predict mood disorder episode (past year) at five years

Aim 3: To determine whether personality dimensions measured at pre-treatment by the TCI, EDI-2 and DSM-III-R personality symptoms among women with BN predict global functioning at five years
9.4 **METHOD**

9.4.1 **Overview**

The BTS, described earlier in Chapter 4, was used for these analyses. Details of the study design and outcome, and three-year follow-up data have been presented elsewhere (Bulik et al., 1998a; Carter et al., 2003a)

9.4.2 **Participants**

Participants were 134 women aged 17-45 years with a current DSM-III-R diagnosis of BN. Of the 135 participants entering the study, one was excluded from the analyses as Axis II data were missing. Exclusion criteria were outlined in Chapter 4.

9.4.3 **Procedure**

This study received ethical approval from the Southern Regional Health Authority (Canterbury) and the University of Canterbury Ethics Committee. Participants provided written informed consent.

9.4.3.1 **Pre-treatment assessment**

As described earlier, non-treating clinicians undertook assessments for Axis I and II disorders using the SCID-I and II for the DSM-III-R (Spitzer, 1990), eating disorder symptoms such as binge eating and purging frequency (CBSI), current depression severity (HDRS) and global functioning (GAF) over the past week. Participants completed self-report questionnaires including the EDI-2 and the TCI. These measures were described in detail in Chapter 4.
9.4.3.2 Follow-up assessment

Participants were reassessed at five-year follow-up for current and past year variables. Past year diagnoses at the five-year assessment provided a more conservative and parsimonious evaluation of outcome, capturing both current (past month) and recent functioning. Our primary outcome was the presence of any eating disorder diagnosis at five years. For someone to be classified as ‘no diagnosis’ during the past year at follow-up, they must have been absent of regular (twice weekly for at least three months) binge eating and purging behaviours during the preceding 12 month period. Follow-up assessment consisted of a diagnostic re-evaluation of any eating disorder diagnosis (full-criteria AN, full-criteria BN, EDNOS), any mood disorder episode in the past year and GAF scores. Data were available for 109 (81%) participants at five-year follow-up.

9.4.3.3 Statistical analyses

The Statistical Package for the Social Sciences (SPSS, Version 12) was used for all statistical analyses. The five-year outcome variables were 1) any eating disorder diagnosis 2) any mood disorder diagnosis and 3) global functioning (GAF scores). Potential predictors of these outcomes were pre-treatment TCI subscales, EDI-2 personality subscales and SCID-II personality disorder symptoms. The presence of any eating disorder was chosen as the key eating disorder outcome at five years for parsimony in the number of analyses and tables, and to capture significant but subthreshold eating symptoms that would be missed if only BN diagnosis were reported. SCID-II personality disorder symptoms were examined in two ways: First, symptom sums for each of the four most common personality disorder diagnoses were used in this sample: avoidant, borderline, obsessive compulsive and paranoid
personality disorder. Second, the sum of Cluster A, B, and C symptoms were used to be consistent with previous literature. Logistic regressions were used to determine the univariate and multivariate independent associations of the predictor variables (TCI subscales, EDI-2 subscales, Axis II personality symptoms and a lifetime mood disorder) and the two binary outcome variables (diagnosis of any eating disorder and a mood disorder episode in the past year at five-year follow-up). An additional logistic regression was conducted to establish whether post-treatment levels of self-directedness were a better predictor of any eating disorder diagnosis at five-year follow-up. Univariate predictors (TCI subscales, EDI-2 subscales, Axis II personality symptoms) of the five-year measure of global functioning (GAF) were assessed using Pearson’s correlation coefficients. Significant results identified in these analyses were further analysed using a multiple linear regression. Statistical significance was determined at the two-tailed $p<0.05$ level.

9.5 RESULTS

9.5.1 Characteristics at pre-treatment

Pre-treatment characteristics of the BTS sample as a whole have been previously described in Chapter 6. Descriptive statistics for current mood disorder episodes and the GAF are presented in Table 9.1.
9.5.2 Descriptive outcome at follow-up

A diagnosis of any eating disorder (AN, BN, EDNOS) in the past year was present in 27% of the sample at five-year follow-up (AN = 2, BN = 19, EDNOS = 20). The EDNOS group was made up of three subgroups: those who were binge eating or purging throughout the past year but did not meet diagnostic frequency or duration for BN (n = 11), those who were binge eating only (n = 2), and those who were purging only (n = 7). The EDNOS group had an average of 2.5 binge eating episodes per month and 9.4 purging episodes per month over the past year at five-year follow-up. A mood disorder episode (past year) was present in 34% of the sample at five years post-treatment. Descriptive statistics for five-year follow-up outcome variables are shown in Table 9.1.

As expected, many of the TCI, EDI-2 and personality disorder symptom variables were inter-correlated. Of note, harm avoidance was correlated with almost all EDI-2 items.

### Table 9.1: Pre-treatment and five-year follow-up descriptive statistics of outcome variables

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<tr>
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<th>N</th>
<th>%</th>
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<tbody>
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<td></td>
</tr>
<tr>
<td>pre-treatment (current)</td>
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<td>100</td>
</tr>
<tr>
<td>five-year follow-up (past year)</td>
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<td>27</td>
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<tr>
<td><strong>Any mood disorder</strong></td>
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<td></td>
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<tr>
<td>pre-treatment (current)</td>
<td>30</td>
<td>21</td>
</tr>
<tr>
<td>five-year follow-up (past year)</td>
<td>48</td>
<td>34</td>
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<tr>
<td><strong>GAF</strong></td>
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<tr>
<td>pre-treatment (current)</td>
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</tr>
<tr>
<td>five-year follow-up (current)</td>
<td>70.7</td>
<td>13.7</td>
</tr>
</tbody>
</table>
and all personality disorder symptoms; self-directedness was negatively correlated with all EDI-2 items except for perfectionism and all personality disorders symptoms except for paranoid personality disorder symptoms. A correlation matrix showing the inter-correlations among the predictor variables at pre-treatment is presented in Table 9.2.
Table 9.2: Correlation coefficients across the personality measures (n = 134)

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Note: *=correlation is significant at the 0.05 level (two-tailed), **=correlation is significant at the 0.01 level (two-tailed)
Abbreviations: TCI = Temperament and Character Inventory, EDI-2 = Eating disorder Inventory-2, PD Sym = Personality disorder symptoms, NS=Novelty-seeking, HA=Harm Avoidance, RD=Reward Dependence, SD=Self-directedness, C=Cooperativeness, P=Persistence, ST=Self-transcendence, Ineffect=ineffectiveness, Perfect=Perfectionism, Distrust=Interpersonal distrust, Aware=Interceptive Awareness, Mat Fear=Maturity Fears, Ascert=Asceticism, Impul=Impulse Regulation, Insec=Social insecurity, Avod=Avoidant, Borderl=Borderline, Ob Com=Obsessive-Compulsive, Clust A=Cluster A, Clust B=Cluster B, Clust C=Cluster C
Table 9.3: Pre-treatment personality measures and their univariate associations with five-year outcomes

<table>
<thead>
<tr>
<th>TCI subscales</th>
<th>Pre-treatment mean (SD)</th>
<th>Any eating disorder diagnosis (past year) OR (CI)</th>
<th>Any mood diagnosis (past year) OR (CI)</th>
<th>GAF scores OR (CI)</th>
<th>r</th>
</tr>
</thead>
<tbody>
<tr>
<td>Novelty seeking</td>
<td>22.1 (6.6)</td>
<td>0.98 (0.93-1.05)</td>
<td>0.95 (0.90-1.01)</td>
<td>-0.01</td>
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</tr>
<tr>
<td>Harm avoidance</td>
<td>19.9 (7.1)</td>
<td>1.00 (0.94-1.05)</td>
<td>1.05 (0.99-1.11)</td>
<td>-0.00</td>
<td></td>
</tr>
<tr>
<td>Reward dependence</td>
<td>15.4 (4.6)</td>
<td>0.95 (0.87-1.04)</td>
<td>1.01 (0.93-1.12)</td>
<td>0.10</td>
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<tr>
<td>Self directedness</td>
<td>23.8 (8.5)</td>
<td>0.93† (0.89-0.98)</td>
<td>0.97 (0.93-1.02)</td>
<td>0.13</td>
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</tr>
<tr>
<td>Cooperativeness</td>
<td>33.3 (7.0)</td>
<td>1.00 (0.94-1.07)</td>
<td>1.03 (0.97-1.10)</td>
<td>0.03</td>
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<tr>
<td>Persistence</td>
<td>4.7 (2.0)</td>
<td>1.04 (0.85-1.23)</td>
<td>1.30† (1.06-1.60)</td>
<td>-0.07</td>
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<tr>
<td>Self-transcendence</td>
<td>11.1 (5.9)</td>
<td>1.00 (0.94-1.07)</td>
<td>1.03 (0.96-1.10)</td>
<td>0.07</td>
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</table>

<table>
<thead>
<tr>
<th>EDI-2 subscales</th>
<th>Pre-treatment mean (SD)</th>
<th>Any eating disorder diagnosis (past year) OR (CI)</th>
<th>Any mood diagnosis (past year) OR (CI)</th>
<th>GAF scores OR (CI)</th>
<th>r</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ineffectiveness</td>
<td>8.9 (6.4)</td>
<td>1.06 (1.00-1.12)</td>
<td>1.06† (1.00-1.13)</td>
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<tr>
<td>Perfectionism</td>
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<td>1.02 (0.94-1.10)</td>
<td>1.07 (0.99-1.16)</td>
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<td>Interpersonal distrust</td>
<td>5.3 (4.2)</td>
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<tr>
<td>Interceptive awareness</td>
<td>9.1 (6.2)</td>
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<td>1.08† (1.01-1.15)</td>
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<td>Maturity fears</td>
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<td>Ascetism</td>
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<td>1.19† (1.06-1.32)</td>
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<td>Impulse regulation</td>
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<td>1.06 (0.97-1.16)</td>
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<td>Social insecurity</td>
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<td>1.07 (0.98-1.16)</td>
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<table>
<thead>
<tr>
<th>PD symptoms</th>
<th>Any eating disorder diagnosis (past year) OR (CI)</th>
<th>Any mood diagnosis (past year) OR (CI)</th>
<th>GAF scores OR (CI)</th>
<th>r</th>
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<tr>
<td>Avoidant symptoms</td>
<td>2.3 (2.1)</td>
<td>1.04 (0.87-1.24)</td>
<td>1.17 (0.98-1.39)</td>
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<tr>
<td>Borderline symptoms</td>
<td>3.4 (2.2)</td>
<td>1.05 (0.88-1.27)</td>
<td>1.26† (1.04-1.52)</td>
<td>-0.21†</td>
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<td>OC symptoms</td>
<td>2.4 (2.0)</td>
<td>1.12 (0.92-1.37)</td>
<td>1.31† (1.07-1.61)</td>
<td>-0.09</td>
</tr>
<tr>
<td>Paranoid symptoms</td>
<td>2.1 (1.9)</td>
<td>0.88 (0.71-1.10)</td>
<td>1.17 (0.96-1.44)</td>
<td>-0.19†</td>
</tr>
<tr>
<td>Cluster A symptoms</td>
<td>4.2 (3.5)</td>
<td>1.00 (0.89-1.12)</td>
<td>1.13† (1.01-1.27)</td>
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<tr>
<td>Cluster B symptoms</td>
<td>7.7 (5.1)</td>
<td>1.03 (0.96-1.12)</td>
<td>1.05 (0.97-1.13)</td>
<td>-0.19†</td>
</tr>
<tr>
<td>Cluster C symptoms</td>
<td>6.4 (4.4)</td>
<td>1.03 (0.95-1.13)</td>
<td>1.15† (1.05-1.25)</td>
<td>-0.09</td>
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<tr>
<td>Total symptoms</td>
<td>18.4 (10.6)</td>
<td>1.01 (0.98-1.05)</td>
<td>1.05† (1.01-1.09)</td>
<td>-0.17†</td>
</tr>
</tbody>
</table>

† Indicates significance at the <0.05 level
Abbreviations: OC=Obsessive Compulsive, TCI=Temperament and Character Inventory, EDI=Eating Disorder Inventory, PD=Personality Disorder
9.5.3 Predictors of any eating disorder diagnosis at five-years

Table 9.3 presents univariate logistic regressions for the pre-treatment TCI and EDI-2 subscales and personality disorder symptoms in the prediction of five-year diagnostic outcome of any eating disorder.

The self-directedness subscale from the TCI was the only personality predictor of the presence of any eating disorder (past year) and for the presence of BN (past year) at five-year follow-up (OR = 0.92, \( p = 0.009 \)). None of the other TCI or EDI-2 subscales or SCID-II personality disorder symptoms predicted five-year diagnostic outcome of any eating disorder or BN specifically. For each unit increase on self-directedness, the odds of not having BN within the last year at five-year follow-up increases by 7%.

Additional analyses examined whether self-directedness at post-treatment was a better predictor of five-year outcome of any eating disorder than self-directedness at pre-treatment. It was found that only self-directedness at pre-treatment was a significant predictor of outcome (self-directedness pre-treatment OR = 0.92, \( p = 0.01 \), self-directedness post-treatment OR = 0.99, \( p = 0.32 \)). Closer examination using a logistic regression within the self-directedness subscale revealed three of the five subscales had significant associations: purposefulness (OR = 0.77, \( p = 0.05 \)), resourcefulness (OR = 0.61, \( p = 0.003 \)), and enlightened second nature (OR = 0.78, \( p = 0.01 \)). When all variables were included in a multivariate logistic regression only SD3 (resourcefulness) remained significant. A lifetime history of a mood disorder episode at pre-treatment did not predict any eating disorder diagnosis at five years (\( p = 0.34 \)).
9.5.4 Predictors of the presence of any mood disorder at five-years

The logistic regression analyses revealed several personality predictors of any episode of a mood disorder (past year) at five-years post-treatment. These variables were: TCI persistence, EDI-2 ineffectiveness, interoceptive awareness and asceticism subscales, and the personality disorder symptoms of borderline, obsessive compulsive, Cluster A, Cluster C and total personality disorder symptoms. A stepwise logistic regression analysis was used to determine whether the variables were independent predictors of a mood disorder episode at five-years. In this analysis, TCI persistence and EDI-2 asceticism subscales remained significant (OR = 1.24, \( p = 0.05 \) and OR = 1.16, \( p = 0.008 \) respectively) indicating an independent contribution from each variable. A lifetime history of mood disorder episodes predicted the presence of a mood disorder episode (past year) at five-year follow-up (OR = 2.0, \( p = 0.002 \)). Fifty-four percent of those with a lifetime diagnosis of a mood disorder at pre-treatment also had an episode of a mood disorder during the fifth year of follow-up. To control for the presence of a lifetime mood disorder, a further logistic regression was conducted using this variable in the model. This revealed the EDI-2 asceticism subscale remained an independent variable significantly associated with a mood disorder episode during follow-up in women with BN at five-year follow-up (OR = 1.1, \( p = 0.02 \)). To confirm internal consistency Cronbach’s alpha were conducted for the asceticism subscale at five different time points. The median Cronbach alpha was 0.60 with a range from 0.56 to 0.69.
9.5.5 Predictors of global functioning at 5-years

In the univariate analyses presented in Table 9.3, the EDI-2 asceticism subscale and the borderline, paranoid, Cluster B and total personality disorder subscales were significant predictors of global functioning at five-year follow-up. If these five correlations are recalculated as partial correlation coefficients allowing for the presence of any lifetime mood disorder, all associations with global functioning at five-years remain significant with the exception of total personality disorder symptoms ($r = -0.18$, $p = 0.06$).

To determine the independent influence of each of these significant variables (borderline, paranoid, Cluster B and total personality disorder subscales) on global functioning at five-years, a multiple linear regression was conducted. Borderline personality disorder symptoms were the only variable significantly associated with global functioning at five-years with this multivariate analysis.

9.6 DISCUSSION

This study examined dimensional measures of personality at pre-treatment (using the TCI, EDI-2 and SCID-II personality disorder symptoms) as a predictor of outcome at five-year follow-up among women with BN. The main findings from this study were: 1) self-directedness was the sole predictor of any eating disorder diagnosis in the past year at five-year follow-up; 2) The EDI-2 asceticism subscale and a lifetime mood disorder were predictors of a mood disorder episode in the year preceding the fifth year of follow-up; 3) borderline personality disorder symptoms were predictors of poorer global functioning at five-year follow-up.
The findings that self-directedness was a predictor of any eating disorder diagnosis and SCID-II personality disorder symptoms were not predictive at five-years, are consistent with previous reports from this sample at one-year follow-up (Bulik et al., 1998b). However, these findings differ from a study by Bloks et al (2004) that found self-directedness did not predict recovery in bulimic patients. This may be due to a difference in samples and instruments, for example, participants in this study were outpatients with BN that were subject to inclusion/exclusion criteria and diagnostic outcome was obtained by using the DSM-III-R. In contrast, the participants in the Bloks study had a range of diagnoses (with less than half the sample BN), a mixture of inpatient and outpatient groups and recovery was defined using scores on the Psychiatric Status Rating scale on the Longitudinal Interview Follow-up Evaluation (LIFE; Bloks et al., 2004).

None of the EDI-2 personality subscales predicted eating disorder outcome in the past year at five-years. Given that this study has shown self-directedness increases with treatment (Anderson et al., 2002), we explored whether post-treatment self-directedness was a superior predictor of five-year outcome than pre-treatment self-directedness. We found that self-directedness at pre-treatment was the only predictor of outcome for any eating disorder diagnosis and post-treatment self-directedness was not a significant predictor.

Cloninger et al., (1993) describes self-directedness as a developmental process with several stages including acceptance of responsibility for one’s own choices, identification of individually valued goals and purposes, development of skills and confidence in problem solving (resourcefulness), self-acceptance and congruent second
nature (automatic responses that match an individual’s goals and values). Results from our study showed that purposefulness (SD2), resourcefulness (SD3) and enlightened/congruent second nature (SD5) were the significant aspects of self-directedness although only resourcefulness remained in the logistic regression model. The key features of high purposefulness are an individual’s clear sense of direction and their ability to guide their activities based on their long-term goals and values. In contrast, individuals that score low on the purposefulness subscale lack long-term direction, they are described as being more reactive to their impulses and circumstances and have feelings of emptiness (Cloninger et al., 1994). The key features of high resourcefulness are described as productivity, being proactive, competence and initiative with problem solving. In contrast, individuals with low resourcefulness display a lack of confidence or skills with solving problems, helplessness, hopelessness and ineffectiveness (Cloninger et al., 1994). The key feature of high congruent second nature is good habits that become automatic and integrated with their long-term goals so that spontaneous acts, impulses or persuasions are safe and not conflicting with their priorities. In contrast, individuals that are low in this subscale are described as self-defeating and struggle with forming habits consistent with their goals (Cloninger et al., 1994).

Overall, our findings suggest that pre-treatment self-directedness offers potential prognostic information in that high self-directedness suggests a good outcome is more likely. Patients with low self-directedness may be less likely to play an active role, may have lower motivation to change and lower personal resourcefulness, reducing the potential effectiveness of therapy. Participation in therapy such as CBT for eating disorders, is likely to assist in increasing self-directness however, those with low self-
directness at pre-treatment are more likely to be depressed and are also more likely to dropout from therapy (Fassino, Piero, Tomba, & Abbate-Daga, 2009). Paying much greater attention to increasing self-directedness early in treatment may assist patients to stay in and benefit from therapy.

High self-directedness involves awareness of values and long-term goals, the person’s own personal processes and resources and displaying adaptive ways of acting to achieve long term goals. Some useful strategies to increase self-directedness are available in therapies such as CBT (Fairburn et al., 2009), DBT (Safer, Telch, & Agras, 2001), Acceptance and Commitment Therapy (Berman, Boutelle, & Crow, 2009) and mindfulness approaches (Baer, Fischer, & Huss, 2006; Proulx, 2008). Increasing self-directedness might be achieved by increasing the person’s self-awareness of their own processes (for example, mindfulness), developing affect tolerance and the ability to disengage from maladaptive emotional activation, developing long term goals consistent with core values (values work, goal setting), expanding adaptive coping behaviours (problem solving, assertiveness etc) in order to achieve these goals, and developing confidence that he/she has the resources required to achieve these goals (increasing self-efficacy beliefs through mastery experiences). It has been observed that self-directedness captures the optimal characteristics for the success of cognitive therapy; therefore, this may be an important quality to enhance (Bulik et al., 1998b).

In addition to our results, self-directedness has been found to predict good outcome in depression (Anderson et al., 2002; Sato, Hirano, Narita, Kusunoki, Kato, Goto, Sakado, & Uehara, 1999), in social phobia (Mortberg, Bejerot, & Wistedt, 2007), in alcohol dependence (Arnau, Mondon, & Santacreu, 2008), and in outcomes of pharmacotherapy
in bulimia (Rybakowski, Słopienn, Komorowska, Antkowiak, Ciesielski, & Rajewski, 2005) and panic disorder (Marchesi, Cantoni, Fonto, Giannelli, & Maggini, 2006, 2007 #451). Although it has been previously suggested that self-directedness encompasses the essence of psychological mindedness in CBT (Bulik et al., 1998b), research shows that it is a predictor of outcome in pharmacotherapy which suggests that it is not necessarily a specific predictor but a widespread predictor of outcome that draws on a basic sense of self as an effective individual (P. Joyce, Personal Communication, July 2010).

Our finding that borderline symptoms are a significant predictor of global functioning at five-year follow-up but not eating disorder outcomes, is consistent with previous findings from studies with shorter follow-up periods that show borderline features do not impact the eating disorder outcome but instead affect global functioning (Grilo et al., 2003; Steiger et al., 1994). Borderline personality symptoms and disorders have been shown to impair social functioning, decrease life satisfaction, predict lower academic and occupational attainment and increase reports of more negative life events associated with personal health, law and finances in clinical and nonclinical populations (Jovev & Jackson, 2006; Trull, Useda, Conforti, & Doan, 1997; Winograd, Cohen, & Chen, 2008). Adolescent borderline symptoms have been shown to persist into adulthood and in one study were found to be associated with the need for services at an average age of 33 years, despite the fact that borderline symptoms decline with age (Winograd et al., 2008). These findings have clinical and research implications. First, dimensional measures of borderline personality predicted five-year global functioning outcome whereas the categorical diagnostic measures did not. This further supports the use of a dimensional approach when evaluating the impact of personality characteristics.
Second, incorporating the persisting impact of personality symptomatology on global functioning suggests the need for therapies such as DBT, that address these personality characteristics (Bruce & Steiger, 2005).

Given that major depression is commonly a recurrent illness (Andrews, 2001; Lee & Murray, 1988; Mulder, Joyce, Frampton, Luty, & Sullivan, 2006), it was unsurprising that a lifetime depressive episode predicted a mood disorder episode in the fifth year. However, the significant finding that higher EDI-2 asceticism subscale predicted any mood disorder episode (past year) at five-year follow-up is novel. Garner (1991) describes asceticism as measuring control over one’s own behavior through self-denial, self-restraint and self-discipline in order to attain rigid spiritual ideals (Garner, 1991). Questions in the EDI-2 reflect the concepts of dieting as a form of purification, for example, “I would like to be in total control of my bodily urges”, “Self-denial makes me feel stronger spiritually”, and “Eating for pleasure is a sign of moral weakness” (Garner, 1991). Asceticism has been found to correlate with perfectionism and outcome of AN (Fassino, Daga, Amianto, Leombruni, Garzaro, & Rovera, 2001; Fassino, Piero, Gramaglia, Abbate Daga, Gandione, Rovera, & Bartocci, 2006). Its relationship with a mood disorder episode can be explained in the context of food restriction becoming reinforcing in order to obtain temporary respite from dysphoric mood (Kaye, 2008). Despite this, asceticism remains a neglected dimension. Although the original EDI has several studies establishing its reliability and validity, the three additional subscales in the revised version (asceticism, impulse regulation and social insecurity) have only moderate internal consistency (Cronbach’s alpha for asceticism range from 0.65-0.70; Eberenz & Gleaves, 1994; Garner, 1991). Given the uncertainty surrounding this subscale, its prognostic value in predicting a mood disorder episode at follow-up is
unclear. Further explication of the domains captured in the asceticism scale may assist with understanding its relation to other core features of eating disorders such as perfectionism and restraint and may shed light on its association with depression at follow-up.

9.6.1 Limitations

This was a post-hoc study in which the focus was on personality as a predictor of outcome and did not address other broader predictor variables identified in previous studies (Collings & King, 1994; Fairburn et al., 1993b; Herzog, Thomas, Kass, Eddy, Franko, & Lowe; Keel et al., 1999). The initial assessment of personality dimensions at pre-treatment was conducted during the acute phase of the eating disorder. This increases the likelihood of “state” and “trait” overlap in aspects of measurement. However, what is measured during the acute phase of the illness may be an exacerbation of core traits. State and trait effects are not stable constructs and exist despite the timing of pre-treatment assessment. This is shown by the modest changes in self-directedness and asceticism that were found over time. The internal consistency of the asceticism subscale limits our interpretation of the finding that asceticism predicted any mood disorder episode (past year) at five-year follow-up. It would preferable to examine asceticism with respect to correlations between the other subscale items in order to see if internal consistency could have been improved. Although this is a relatively large sample of women with BN, we acknowledge that multiple comparisons may increase the chance of Type I error particularly among the univariate associations. However, the broad consistency of the results across outcomes would perhaps suggest that these results represent genuine clinical associations rather than spurious statistical patterns.
Finally, the exclusion criteria results in a selective sample that is not representative for all eating disorder populations.

9.7 CONCLUSIONS

Using a dimensional model, this study found that self-directedness predicted the presence of any eating disorder at five-year follow-up. Future research is needed to evaluate ways to develop this important attribute in individuals with low scores at pre-treatment. That different pre-treatment measures predicted our three different outcome domains indicates that no single measure of personality dimensions captures all aspects of outcome. This underscores the importance of a broad pre-treatment assessment battery that includes both dimensional and categorical measures that effectively captures various dimensions of personality functioning.
PART 5

PERSONALITY IN BN AND BED

Chapter 10: A Comparison of the Impact of Personality Disorders on BN and BED

10.1 Overview

The goal of this chapter is to compare Axis I and Axis II comorbidity, eating disorder symptomatology, general psychiatric symptomatology and global functioning between BN and BED in an attempt to explore the similarities and differences between these groups.

10.2 Introduction

Bulimia nervosa and AN have been extensively studied however, information about BED is more limited. Although it has been suggested that BN and BED may be closely related disorders (Nunez-Navarro, Jimenez-Murcia, Alvarez-Moya, Villarejo, Sanchez Diaz, Masuet Augmantell, Granero, Penelo, Krug, Tinahones, Bulik& Fernandez-Aranda, 2010), findings are mixed on the similarities and differences that exist on between the groups (Fichter, Quadflieg, & Brandl, 1993; Masheb & Grilo, 2000; Raymond et al., 1995; Wilfley, Schwartz, Spurrell, & Fairburn, 2000b). It has been suggested that BN and BED differ in the following areas: eating and weight-related pathology (Fichter et al., 1993), levels of depression and anxiety, and rates of personality disorder (BN>BED; Raymond et al., 1995).
Personality disorders are common within an eating disorder population and have higher rates than some Axis I disorders (Braun et al., 1994). Prevalence rates for Axis II comorbidity in BN and BED range from 21% to 77% and 20% to 37% respectively (Herzog et al., 1992; Steiger et al., 1992; Telch & Stice, 1998; Wilfley et al., 2000a; Yanovski, 1993). Those rates found in the BED group are higher than non-BED obese samples (Specker et al., 1994). A summary of existing research shows borderline personality disorder is the most common personality disorder in BN, followed by dependent and histrionic personality disorders (Sansone et al., 2005). The most common Axis II comorbidity reported in those with BED is obsessive-compulsive personality disorder, followed by avoidant personality disorder and borderline personality disorder (Sansone et al., 2005).

Those with comorbid eating disorders and personality disorders have been reported to have increased general psychopathology, interpersonal problems and poorer general functioning (Cassin & von Ranson, 2005; Johnson et al., 1989; Telch & Stice, 1998). However, the effect of personality disorders on the severity of eating disorder symptoms in BED is unclear. The few studies to examine the impact of personality disorders on eating disorder symptoms in BED have found that the presence of Axis II psychopathology was significantly related to increased binge eating and frequency of binge episodes at pre-treatment (Picot & Lilienfeld, 2003; Wilfley et al., 2000a). In contrast, there are mixed findings within the BN literature with some studies reporting that the presence of a personality disorder has a negative effect on eating disorder symptoms (Matsunaga et al., 1998; Picot & Lilienfeld, 2003; Wonderlich et al., 1994) and other studies reporting few differences in eating disorder symptoms despite Axis II
comorbidity (Johnson et al., 1990; Steiger & Stotland, 1996; Steiger et al., 1994; Zeeck et al., 2007).

Comparisons between BN and BED and comorbid personality disorders are important to understand theoretical and clinical similarities and differences between the groups (Cassin & von Ranson, 2005; Raymond et al., 1995). If differences exist, this may have implications for treatment options for these groups.

10.3 AIMS

The aims of this exploratory study are:

Aim 1: To compare the prevalence of Axis I and Axis II diagnoses across BN and BED

Aim 2: To explore the effect of the presence of a personality disorder on eating disorder symptomatology, general psychiatric functioning and global functioning across both BN and BED at pre-treatment

10.4 METHOD

10.4.1 Overview

The BEP study, described earlier in Chapter 5, was used for these analyses.

10.4.2 Participants

BED and BN participants were consecutive referrals to the treatment trial. Inclusion criteria for this study were women aged 16 and over with a current primary DSM-IV
diagnosis of BN or BED and a BMI>17.5. Exclusion criteria were outlined in Chapter 5. The sample was comprised of 39 women with BN and 41 women with BED.

10.4.3 Procedure

This study received ethical approval from the Upper Regional South A Ethics Committee. Participants provided written informed consent.

10.4.3.1 Pre-treatment assessment

Participants were assessed for Axis I disorders using the Structured Clinical Interview for DSM-IV Patient version (SCID-I; American Psychiatric Association, 1994). Assessments for Axis I disorders were done by the treating clinician prior to randomisation. Eating disorder symptoms such as binge eating and purging frequency were assessed using the EDE (Fairburn, 1993). The clinician rated the GAF (American Psychiatric Association, 1987) and assessed depressive symptoms with the MADRS (Montgomery & Asberg, 1979). Participants completed a series of self-report questionnaires including the EDI-2 (Garner, 1983) and the SCL-90 (Derogatis, Lipman, & Covi, 1973) which are reported here.

10.4.3.2 Personality assessment

Participants were assessed for Axis II disorders using the Structured Clinical Interview for DSM-IV personality disorders version four weeks after beginning treatment. This assessment was done by non-treating psychologists, psychiatrists and a PhD student trained in administration of this assessment.
10.4.3.3 Statistical analyses

The Statistical Package for the Social Sciences (SPSS, Version 13) was used to analyse data. Standard descriptive statistics were used to summarise demographics and eating disorder history for the BN and BED groups. Continuous parameters were compared between these groups using one-way ANOVA. Axis I and II comorbid diagnoses were summarised for the two groups as frequencies and percentages. Key comorbidities (any mood disorder, any anxiety disorder and any substance abuse/dependence) were compared between the groups using chi-square tests.

The effect of presence/absence of personality disorder and eating disorder groups (BN/PD, BN/No-PD, BED/PD, BED/No-PD) on EDE, EDI-2, and SCL subscales and the MADRS total score were tested at pre-treatment using a two-way ANOVA using the presence/absence of personality disorder and eating disorder groups as between-subject factors. As this was an exploratory study, a two-tailed p-value (<0.05) was used to indicate statistical significance.

10.5 RESULTS

10.5.1 Descriptive statistics for the sample

The mean age of the eating disorder sample was 32.9 years (SD = 11.2), and the mean BMI was 29.1 (SD = 8.1). The sample was made up of New Zealand European (65%), Maori (11%), European (15%), Asian (8%) and Other (1%). The mean age-of-onset of the eating disorder was 20.9 years (SD=8.6) and duration of illness was 11.9 years (SD = 11.8). Prior eating disorder diagnoses (AN, BN, BED) occurred in 21% of the whole sample and there were no significant difference between the BN and BED groups (p =
0.88). Of those with a current BED diagnosis, 23% had a lifetime history of BN while of those with a current BN diagnosis, 8% had a history of BED. The current BN group had a greater proportion of those with a history of AN (14%) compared to 2% in the BED group (Fishers exact test, p = 0.02). The BED group was significantly older (M = 35.3, SD = 11.2, p = 0.04), had a higher BMI (M = 33.7, SD = 7.5, p<0.001) and longer duration of the eating disorder (M = 14.6, SD = 13.4, p = 0.04) compared than those with BN.

Frequencies of Axis I comorbidity are shown in Table 10.2. Due to low numbers in some groups, chi-square tests were only conducted on the any mood disorder, any anxiety disorder and any substance abuse/dependence variables. Results from the chi-square tests show no significant differences in the occurrence of any mood disorder ($\chi^2 = 0.40, p = 0.53$), any anxiety disorder ($\chi^2 = 0.76, p = 0.39$) and any substance abuse/dependence ($\chi^2 = 0.03, p = 0.86$) between the BN and BED groups.

Diagnosis of an Axis II disorder was present in 45% of the whole sample and the overall prevalence did not differ between BN and BED groups (Table 10.3). The most common personality disorders were avoidant (25%), obsessive-compulsive (19%) and borderline (14%). Avoidant personality disorder was the most common diagnosis for those with BN while obsessive-compulsive personality disorder was the most common in BED. Obsessive-compulsive personality disorder was the only Axis II disorder with a significantly different prevalence between the BN and BED groups ($p = 0.05$). There was no difference in ‘Any PD’ between the BN and BED groups ($\chi^2 = 0.04, p = 0.84$).
Table 10.1: Descriptive characteristics of subjects with BN and BED

<table>
<thead>
<tr>
<th>Demographics</th>
<th>Whole sample</th>
<th>BN group</th>
<th>BED group</th>
<th>( F )</th>
<th>( p )</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Mean (SD) or N (%)</strong></td>
<td><strong>Mean (SD) or N (%)</strong></td>
<td><strong>Mean (SD) or N (%)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Age (years)</strong></td>
<td>32.9 (11.2)</td>
<td>30.3 (10.9)</td>
<td>35.3 (11.2)</td>
<td>4.2</td>
<td>0.04</td>
</tr>
<tr>
<td><strong>BMI</strong></td>
<td>29.1 (8.1)</td>
<td>24.3 (5.3)</td>
<td>33.7 (7.5)</td>
<td>41.4</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td><strong>Ethnicity</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>NZ European</td>
<td>54 (68%)</td>
<td>27 (69%)</td>
<td>27 (66%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Maori</td>
<td>2 (3%)</td>
<td>0 (0%)</td>
<td>2 (5%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Other</td>
<td>24 (30%)</td>
<td>12 (31%)</td>
<td>12 (29%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Marital</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Never married</td>
<td>36 (45%)</td>
<td>17 (44%)</td>
<td>19 (46%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Married/de facto</td>
<td>33 (41%)</td>
<td>15 (39%)</td>
<td>18 (44%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Separated</td>
<td>6 (8%)</td>
<td>3 (8%)</td>
<td>3 (7%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Divorced</td>
<td>4 (5%)</td>
<td>3 (8%)</td>
<td>4 (2%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Widowed</td>
<td>1 (1%)</td>
<td>1 (3%)</td>
<td>0 (0%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Education (years)</strong></td>
<td>15.2 (2.5)</td>
<td>15.1 (2.4)</td>
<td>15.3 (2.6)</td>
<td>0.1</td>
<td>NS</td>
</tr>
<tr>
<td><strong>Employment</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Employed</td>
<td>38 (48%)</td>
<td>19 (49%)</td>
<td>19 (46%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Student</td>
<td>21 (26%)</td>
<td>13 (33%)</td>
<td>8 (20%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Housewife</td>
<td>12 (12%)</td>
<td>4 (10%)</td>
<td>8 (20%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Benefit recipient</td>
<td>9 (11%)</td>
<td>3 (8%)</td>
<td>6 (15%)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Table 10.2: Axis I comorbidity of subjects with BN and BED

<table>
<thead>
<tr>
<th>Lifetime Axis I Comorbidity</th>
<th>Whole sample (n=80)</th>
<th>BN group (n=39)</th>
<th>BED group (n=41)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>N (%)</td>
<td>N (%)</td>
<td>N (%)</td>
</tr>
<tr>
<td><strong>Any Mood Disorder</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Major Depressive Disorder</td>
<td>50 (63%)</td>
<td>23 (59%)</td>
<td>27 (66%)</td>
</tr>
<tr>
<td>Bipolar II</td>
<td>3 (4%)</td>
<td>1 (3%)</td>
<td>2 (5%)</td>
</tr>
<tr>
<td><strong>Any Anxiety Disorder</strong></td>
<td>47 (59%)</td>
<td>21 (54%)</td>
<td>26 (63%)</td>
</tr>
<tr>
<td>Obsessive Compulsive Disorder</td>
<td>8 (10%)</td>
<td>4 (10%)</td>
<td>4 (10%)</td>
</tr>
<tr>
<td>Panic Disorder</td>
<td>8 (10%)</td>
<td>4 (10%)</td>
<td>4 (10%)</td>
</tr>
<tr>
<td>Agoraphobia without panic</td>
<td>1 (1%)</td>
<td>0 (0%)</td>
<td>1 (2%)</td>
</tr>
<tr>
<td>Social Phobia</td>
<td>24 (30%)</td>
<td>11 (28%)</td>
<td>13 (32%)</td>
</tr>
<tr>
<td>Simple Phobia</td>
<td>18 (23%)</td>
<td>8 (21%)</td>
<td>10 (24%)</td>
</tr>
<tr>
<td>Post traumatic Stress Disorder</td>
<td>15 (19%)</td>
<td>6 (15%)</td>
<td>9 (22%)</td>
</tr>
<tr>
<td><strong>Any Substance Abuse/Dependence</strong></td>
<td>32 (40%)</td>
<td>16 (41%)</td>
<td>16 (39%)</td>
</tr>
<tr>
<td>Alcohol Abuse/Dependence</td>
<td>27 (34%)</td>
<td>13 (33%)</td>
<td>14 (34%)</td>
</tr>
<tr>
<td>Cannabis Abuse/Dependence</td>
<td>12 (15%)</td>
<td>5 (13%)</td>
<td>7 (17%)</td>
</tr>
<tr>
<td>Stimulant Abuse/Dependence</td>
<td>5 (6%)</td>
<td>1 (3%)</td>
<td>4 (10%)</td>
</tr>
</tbody>
</table>
### Table 10.3: Axis II comorbidity of subjects with BN and BED

<table>
<thead>
<tr>
<th>Lifetime Axis II Comorbidity</th>
<th>Whole sample (n=80)</th>
<th>BN group (n=39)</th>
<th>BED group (n=41)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>N (%)</td>
<td>N (%)</td>
<td>N (%)</td>
</tr>
<tr>
<td>Borderline PD</td>
<td>11 (14%)</td>
<td>5 (13%)</td>
<td>6 (15%)</td>
</tr>
<tr>
<td>Antisocial PD</td>
<td>3 (4%)</td>
<td>2 (5%)</td>
<td>1 (2%)</td>
</tr>
<tr>
<td>Histrionic PD</td>
<td>1 (1%)</td>
<td>0 (0%)</td>
<td>1 (2%)</td>
</tr>
<tr>
<td>Narcissistic PD</td>
<td>4 (5%)</td>
<td>0 (0%)</td>
<td>4 (10%)</td>
</tr>
<tr>
<td>Avoidant PD</td>
<td>20 (25%)</td>
<td>12 (31%)</td>
<td>8 (20%)</td>
</tr>
<tr>
<td>Dependent PD</td>
<td>2 (3%)</td>
<td>0 (0%)</td>
<td>2 (5%)</td>
</tr>
<tr>
<td>Obsessive-compulsive PD</td>
<td>15 (19%)</td>
<td>4 (10%)</td>
<td>11 (27%)</td>
</tr>
<tr>
<td>Paranoid PD</td>
<td>7 (9%)</td>
<td>4 (10%)</td>
<td>3 (7%)</td>
</tr>
<tr>
<td>Schizoid PD</td>
<td>2 (3%)</td>
<td>1 (3%)</td>
<td>1 (2%)</td>
</tr>
<tr>
<td>Schizotypal PD</td>
<td>1 (1%)</td>
<td>1 (3%)</td>
<td>0 (0%)</td>
</tr>
<tr>
<td>Any PD</td>
<td>36 (45%)</td>
<td>18 (46%)</td>
<td>18 (44%)</td>
</tr>
</tbody>
</table>

Note: PD=Personality Disorder
<table>
<thead>
<tr>
<th>Eating disorder symptomatology</th>
<th>BN (n=21)</th>
<th>PD (n=18)</th>
<th>No PD (n=23)</th>
<th>PD (n=18)</th>
<th>Main effect</th>
<th>Main effect</th>
<th>Interaction</th>
</tr>
</thead>
<tbody>
<tr>
<td>Objective binge days</td>
<td>17.0 (1.6)</td>
<td>14.2 (1.7)</td>
<td>12.0 (1.5)</td>
<td>16.0 (1.7)</td>
<td>0.33</td>
<td>0.73</td>
<td>0.04</td>
</tr>
<tr>
<td>Objective binge episodes</td>
<td>24.8 (2.8)</td>
<td>18.9 (3.0)</td>
<td>14.5 (2.7)</td>
<td>20.8 (3.0)</td>
<td>0.15</td>
<td>0.94</td>
<td>0.04</td>
</tr>
<tr>
<td>Subjective binge days¹</td>
<td>15.5 (2.7)</td>
<td>15.1 (2.6)</td>
<td>18.3 (2.8)</td>
<td>17.9 (3.2)</td>
<td>0.33</td>
<td>0.88</td>
<td>0.99</td>
</tr>
<tr>
<td>Subjective binge episodes¹</td>
<td>26.0 (7.4)</td>
<td>31.7 (7.1)</td>
<td>29.7 (7.7)</td>
<td>28.9 (8.9)</td>
<td>0.96</td>
<td>0.75</td>
<td>0.68</td>
</tr>
<tr>
<td><strong>EDE subscales</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Restraint over eating</td>
<td>4.5 (0.5)</td>
<td>5.7 (0.5)</td>
<td>3.4 (0.5)</td>
<td>3.7 (0.5)</td>
<td><strong>0.003</strong></td>
<td>0.14</td>
<td>0.46</td>
</tr>
<tr>
<td>Avoidance of eating</td>
<td>0.3 (0.2)</td>
<td>0.7 (0.3)</td>
<td>0.3 (0.2)</td>
<td>0.4 (0.3)</td>
<td>0.47</td>
<td>0.34</td>
<td>0.67</td>
</tr>
<tr>
<td>Empty stomach</td>
<td>3.0 (0.5)</td>
<td>3.0 (0.6)</td>
<td>1.3 (0.5)</td>
<td>1.6 (0.6)</td>
<td>0.005</td>
<td>0.71</td>
<td>0.78</td>
</tr>
<tr>
<td>Food avoidance</td>
<td>4.1 (0.5)</td>
<td>5.4 (0.5)</td>
<td>3.8 (0.5)</td>
<td>3.8 (0.5)</td>
<td><strong>0.05</strong></td>
<td>0.20</td>
<td>0.20</td>
</tr>
<tr>
<td>Dietary rules</td>
<td>4.2 (0.6)</td>
<td>5.2 (0.6)</td>
<td>2.4 (0.5)</td>
<td>4.2 (0.6)</td>
<td><strong>0.02</strong></td>
<td><strong>0.02</strong></td>
<td>0.51</td>
</tr>
<tr>
<td>Preoccupation with food, eating</td>
<td>2.9 (0.5)</td>
<td>4.4 (0.6)</td>
<td>2.4 (0.5)</td>
<td>3.6 (0.6)</td>
<td>0.24</td>
<td><strong>0.01</strong></td>
<td>0.77</td>
</tr>
<tr>
<td>Fear of losing control</td>
<td>4.8 (0.4)</td>
<td>4.9 (0.4)</td>
<td>3.4 (0.4)</td>
<td>5.5 (0.4)</td>
<td>0.37</td>
<td><strong>0.01</strong></td>
<td><strong>0.02</strong></td>
</tr>
<tr>
<td>Social eating</td>
<td>2.2 (0.4)</td>
<td>2.7 (0.4)</td>
<td>1.5 (0.4)</td>
<td>2.4 (0.4)</td>
<td>0.24</td>
<td>0.10</td>
<td>0.52</td>
</tr>
<tr>
<td>Secret eating</td>
<td>2.9 (0.5)</td>
<td>2.8 (0.5)</td>
<td>2.7 (0.4)</td>
<td>3.3 (0.5)</td>
<td>0.72</td>
<td>0.53</td>
<td>0.43</td>
</tr>
<tr>
<td>Guilt</td>
<td>4.1 (0.4)</td>
<td>3.7 (0.4)</td>
<td>3.0 (0.4)</td>
<td>3.6 (0.4)</td>
<td>0.13</td>
<td>0.86</td>
<td>0.17</td>
</tr>
<tr>
<td>Dissatisfaction with weight</td>
<td>4.4 (0.3)</td>
<td>4.6 (0.4)</td>
<td>4.5 (0.3)</td>
<td>5.1 (0.4)</td>
<td>0.42</td>
<td>0.24</td>
<td>0.51</td>
</tr>
<tr>
<td>Desire to lose weight</td>
<td>4.7 (0.4)</td>
<td>5.6 (0.4)</td>
<td>4.3 (0.4)</td>
<td>4.6 (0.4)</td>
<td>0.10</td>
<td>0.19</td>
<td>0.49</td>
</tr>
<tr>
<td>Reaction to prescribed weighing</td>
<td>1.6 (0.5)</td>
<td>3.0 (0.5)</td>
<td>1.5 (0.5)</td>
<td>1.9 (0.5)</td>
<td>0.24</td>
<td>0.07</td>
<td>0.32</td>
</tr>
<tr>
<td>Dissatisfaction with shape</td>
<td>4.1 (0.3)</td>
<td>4.9 (0.3)</td>
<td>4.3 (0.3)</td>
<td>4.7 (0.3)</td>
<td>0.98</td>
<td>0.07</td>
<td>0.50</td>
</tr>
<tr>
<td>Preoccupation with shape or weight</td>
<td>2.2 (0.6)</td>
<td>2.9 (0.6)</td>
<td>2.2 (0.5)</td>
<td>3.4 (0.6)</td>
<td>0.68</td>
<td>0.10</td>
<td>0.72</td>
</tr>
<tr>
<td>Importance of shape</td>
<td>4.2 (0.3)</td>
<td>4.9 (0.3)</td>
<td>4.0 (0.3)</td>
<td>4.6 (0.3)</td>
<td><strong>0.03</strong></td>
<td>0.94</td>
<td></td>
</tr>
<tr>
<td>Importance of weight</td>
<td>3.7 (0.4)</td>
<td>4.3 (0.4)</td>
<td>3.9 (0.4)</td>
<td>4.4 (0.4)</td>
<td>0.68</td>
<td>0.10</td>
<td>0.90</td>
</tr>
<tr>
<td>Fear of weight gain</td>
<td>5.0 (0.5)</td>
<td>5.0 (0.5)</td>
<td>3.1 (0.4)</td>
<td>4.7 (0.5)</td>
<td><strong>0.02</strong></td>
<td>0.11</td>
<td>0.11</td>
</tr>
<tr>
<td>Discomfort seeing body</td>
<td>3.3 (0.3)</td>
<td>4.6 (0.3)</td>
<td>3.5 (0.3)</td>
<td>4.1 (0.3)</td>
<td><strong>0.004</strong></td>
<td>0.25</td>
<td></td>
</tr>
<tr>
<td>Avoidance of exposure</td>
<td>3.6 (0.4)</td>
<td>4.4 (0.4)</td>
<td>4.0 (0.4)</td>
<td>4.8 (0.4)</td>
<td>0.28</td>
<td>0.06</td>
<td>0.98</td>
</tr>
<tr>
<td>Feelings of fatness</td>
<td>5.0 (0.3)</td>
<td>5.4 (0.4)</td>
<td>5.8 (0.3)</td>
<td>6.1 (0.4)</td>
<td><strong>0.03</strong></td>
<td>0.29</td>
<td>0.88</td>
</tr>
<tr>
<td>Flat stomach</td>
<td>5.1 (0.5)</td>
<td>5.3 (0.5)</td>
<td>5.4 (0.5)</td>
<td>5.4 (0.5)</td>
<td>0.66</td>
<td>0.89</td>
<td>0.82</td>
</tr>
</tbody>
</table>

Abbreviations: BN = bulimia nervosa, BED = binge eating disorder, PD = personality disorder, vs = versus, EDE = Eating Disorder Examination
Note: The significance level is set at p<0.05
Analyses controlled for age and BMI however, there were no significant differences across the results therefore the raw data with no covariates are presented in Table 10.4. Table 10.4 presents exploratory analyses using a two-way ANOVA to test the main effect differences between eating disorder diagnoses (BN versus BED), personality disorder diagnosis (personality disorder versus no personality disorder) and the interaction between the personality disorder and eating disorder diagnoses. The mean number of objective binge days and episodes is significantly higher in the BED/PD (M = 16.0) compared to the BED/No-PD group (M = 12.0) (also see Figure 10.1). In contrast, among the BN group the presence of a personality disorder decreased the
number of objective binge days (M = 14.2) compared to the BN/No-PD group (M = 17.0). Consistent with this finding, the number of objective binge episodes in the BED/PD group (M = 20.8) was higher than in the BED/No-PD group (M = 14.5) whereas the BN/PD group (M = 18.9) was again lower when compared to the BN/No-PD group (M = 24.8). There were no significant differences in the mean number of subjective binge days and episodes across the groups.

Scores on a number of EDE subscales were significantly higher in the BN group compared to the BED group, including restraint over eating, empty stomach, food avoidance, dietary rules and fear of weight subscales. The BED group was higher than the BN group on the EDE ‘feelings of fatness’ subscale. The following EDE subscales were significantly higher in the personality disorder groups (BN/PD and BED/PD) than the no personality disorder groups (BN/No-PD and BED/No-PD): dietary rules, preoccupation with food and eating, fear of losing control over eating, importance of shape and discomfort over seeing body subscales. The only EDE subscale to significantly differ in the comorbid eating disorder and personality disorder groups was the fear of losing control over eating subscale. In the BED group, the presence of a personality disorder was associated with higher scores on the fear of losing control (M = 5.5) subscale compared to the no personality disorder group (M = 3.4). There was no difference in the BN group on the fear of losing control over eating subscale (M = 4.9 and M = 4.8 respectively) with and without a personality disorder.

Eating disorder attitudes from the EDI-2 are presented in Table 10.5. Significant differences were found on the EDI-2 subscales ‘drive for thinness’ and ‘body dissatisfaction’ between the BN and BED groups. No differences were found between
the no personality disorder/personality disorder groups or in the comorbid eating disorder and personality disorder groups.
Table 10.5: The effect personality disorders on BN and BED eating disorder attitudes, psychiatric symptoms and global functioning

<table>
<thead>
<tr>
<th></th>
<th>BN (n=21)</th>
<th>PD (n=18)</th>
<th>BED (n=18)</th>
<th>Main effect BN vs BED</th>
<th>Main effect No PD vs PD</th>
<th>Interaction BN/BED x No PD/PD</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>EDI-2 subscales</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Drive for thinness</td>
<td>13.3 (1.2)</td>
<td>14.3 (1.3)</td>
<td>9.7 (1.1)</td>
<td>13.2 (1.3)</td>
<td><strong>0.05</strong></td>
<td>0.07</td>
</tr>
<tr>
<td>Bulimia</td>
<td>9.9 (0.9)</td>
<td>10.4 (0.9)</td>
<td>8.5 (0.8)</td>
<td>10.2 (0.9)</td>
<td>0.37</td>
<td>0.22</td>
</tr>
<tr>
<td>Body dissatisfaction</td>
<td>15.3 (1.6)</td>
<td>19.2 (1.7)</td>
<td>21.3 (1.5)</td>
<td>22.9 (1.7)</td>
<td><strong>0.004</strong></td>
<td>0.10</td>
</tr>
<tr>
<td><strong>SCL-90 subscales</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Somatization</td>
<td>0.7 (0.1)</td>
<td>1.1 (0.1)</td>
<td>0.7 (0.1)</td>
<td>1.2 (0.1)</td>
<td>0.50</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Obsessive-compulsive</td>
<td>1.1 (0.2)</td>
<td>1.4 (0.2)</td>
<td>1.0 (0.2)</td>
<td>1.7 (0.2)</td>
<td>0.49</td>
<td><strong>0.001</strong></td>
</tr>
<tr>
<td>Interpersonal sensitivity</td>
<td>0.9 (0.1)</td>
<td>1.6 (0.1)</td>
<td>1.1 (0.1)</td>
<td>1.6 (0.1)</td>
<td>0.43</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Depression</td>
<td>1.2 (0.2)</td>
<td>1.7 (0.2)</td>
<td>1.2 (0.2)</td>
<td>1.9 (0.2)</td>
<td>0.56</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Anxiety</td>
<td>0.7 (0.1)</td>
<td>1.1 (0.1)</td>
<td>0.7 (0.1)</td>
<td>1.3 (0.1)</td>
<td>0.62</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Anger-Hostility</td>
<td>0.5 (0.1)</td>
<td>0.9 (0.1)</td>
<td>0.6 (0.1)</td>
<td>0.7 (0.1)</td>
<td>0.74</td>
<td>0.08</td>
</tr>
<tr>
<td>Phobic anxiety</td>
<td>0.3 (0.1)</td>
<td>0.5 (0.1)</td>
<td>0.2 (0.1)</td>
<td>0.5 (0.1)</td>
<td>0.60</td>
<td><strong>0.005</strong></td>
</tr>
<tr>
<td>Paranoid ideation</td>
<td>0.5 (0.1)</td>
<td>0.9 (0.1)</td>
<td>0.7 (0.1)</td>
<td>1.1 (0.1)</td>
<td>0.11</td>
<td><strong>0.003</strong></td>
</tr>
<tr>
<td>Psychoticism</td>
<td>0.5 (0.1)</td>
<td>1.0 (0.1)</td>
<td>0.5 (0.1)</td>
<td>0.9 (0.1)</td>
<td>0.78</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td><strong>MADRS total</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>MADRS total</td>
<td>11.5 (1.9)</td>
<td>17.2 (2.0)</td>
<td>10.1 (1.7)</td>
<td>14.4 (2.0)</td>
<td><strong>0.01</strong></td>
<td>0.72</td>
</tr>
<tr>
<td><strong>GAF total</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>GAF total</td>
<td>53.3 (1.0)</td>
<td>54.2 (1.1)</td>
<td>56.9 (1.0)</td>
<td>53.8 (1.1)</td>
<td>0.14</td>
<td>0.31</td>
</tr>
</tbody>
</table>

Abbreviations: BN = BN, BED = binge eating disorder, PD = personality disorder, vs = versus, EDI = Eating Disorder Inventory, SCL-90 = Hopkins Symptom Checklist Revised, MADRS = Montgomery and Asberg Depression Rating Scale, GAF = Global Assessment of Functioning scale
Note: The significance level is set at p<0.05
10.5.3 Psychiatric symptoms and global functioning

Psychiatric symptoms showed no significant differences on the SCL-90 obsessive-compulsive, interpersonal sensitivity and anxiety subscales between the BN and BED groups (Table 10.5). In contrast, all SCL-90 subscales except for the ‘anger-hostility’ subscale and the MADRS total score were significantly higher in the personality disorder group compared to the no personality disorder group.

Global functioning, measured by the GAF, was not significantly different between any of the groups.

Analyses controlled for age and BMI however, there were no significant differences in the results therefore the raw data with no covariates are presented in Table 10.5.

10.6 DISCUSSION

Women with BN and BED are remarkably similar on Axis I and II comorbidity, eating disorder symptoms, general psychiatric functioning and global functioning at pre-treatment. Across BN and BED, those with a comorbid personality disorder have more general psychopathology however, the impact of Axis II comorbidity has the opposite effect on BN and BED eating behaviours, in which it is associated with increased binge eating in the BED group and decreased binge eating in the BN group.

Our finding that the BED/PD group had more frequent binge episodes is consistent with other studies showing that the presence of a personality disorder significantly affects eating disorder symptoms in the BED group (Picot & Lilenfeld, 2003; Wilfley et al., 2000a). However, our results do not support other findings suggesting that eating
disorder symptoms in BN are exacerbated by the presence of a personality disorder as binge frequency was lower in this sample in those with BN and comorbid Axis II disorders (Matsunaga et al., 1998; Picot & Lilenfeld, 2003; Wonderlich et al., 1994). Although statistically significant, the effect sizes were modest and may have limited clinical significance.

The presence of a personality disorder on eating disorder psychological symptoms does not differ between the BN and BED groups with the exception of the EDE fear of losing control over eating subscale which was higher in the BED/personality disorder group. This may be a direct result of the increased binge frequency in this group.

In both the BN and BED samples, the presence of a personality disorder was associated with an adverse impact on general psychiatric functioning (almost all SCL-90 and MADRS subscales). It has been shown that a comorbid Axis II disorder impacts on general psychiatric functioning more severely than eating disorder symptoms (Bruce et al., 2004; Johnson et al., 1989). Our study confirms this finding and further clarifies that this result is not dependent on BN or BED diagnosis.

Overall, the clinical profiles of BN and BED are comparable in eating disorder psychopathology, general psychiatric functioning and global functioning. The few significant differences in eating disorder symptoms between the BN and BED groups were in the direction of BN being slightly more pathological. However, when a comorbid personality disorder was present this finding reversed and the BED group had increased eating psychopathology in comparison to those with BED and no personality disorder.
The similarities of clinical profiles between the BN and BED groups may have important implications. Within New Zealand where this study has taken place, treatment for BED is limited or unavailable. Specialist treatment is generally reserved for those with AN, BN or morbid obesity. For those individuals that do not meet diagnostic criteria for AN or BN, generalist services providing counselling and support may be the only treatment option available. The similar pathology between BN and BED shows a need for specialist evidence-based treatment of BED to be incorporated into healthcare services, particularly in the cases where an Axis II disorder is present and possibly worsening the eating disorder behaviours. Treatment for BED may be complex because there are both psychological and weight issues to treat.

10.6.1 Limitations

A limitation of this study was the small size of this treatment seeking sample which limits the generalisability of these findings. Corrections for multiple comparisons were not made as this was an exploratory study used to generate further hypotheses; therefore there is a risk of type I error with regards to these analyses. A standard Bonferroni test would be too strict as we were looking at correlated outcomes.

10.7 Conclusions

The present study compared the prevalence of Axis I and II comorbidity within BN and BED groups, as well as the effects of comorbid personality disorders on BN and BED. Our study suggests that overall BN and BED have similar clinical profiles. However, having a personality disorder did negatively affect eating disorder symptoms in those with BED compared to the comorbid BN and personality disorder group. Replication of
this study is needed in a larger sample to verify the finding. If replicated, there may be implications for treatments for BED such as, increasing specialist evidence-based treatment facilities and greater attention to addressing personality dysfunction. Future research should examine individual personality disorders (for example, obsessive-compulsive personality disorder) in the BED group in order to clarify the extent to which specific personality disorders contribute to potential increases in eating disorder pathology. Furthermore, investigations of the long-term impact of Axis II comorbidity on BED outcome would be of value.
Chapter 11: Temperament and Character Profile of BN and BED

11.1 Overview

This chapter examines differences in personality traits in women with BN, BED, depressive controls and a normal control group. Comparisons between the groups are made and the extent to which these differences are independent of age and mood is examined.

11.2 Introduction

Temperament and character constructs have been widely studied within the eating disorder literature (Bulik, Sullivan, Weltzin, & Kaye, 1995b; Diaz-Marsa et al., 2000; Fassino et al., 2004; Klump et al., 2004). Personality profiles in eating disorder subgroups have mainly focused on AN and BN, or comparisons between these groups (Bulik et al., 1995a; Rybakowski et al., 2004; Vervaeet, van Heeringen, & Audenaert, 2004b). Few studies have examined temperament and character in relation to BED, limiting our understanding in this area.

The TCI is an instrument designed to assess personality using four temperament dimensions and three character dimensions (Cloninger et al., 1993). Temperament is defined as “automatic emotional responses to experience that are moderately heritable and stable throughout life” (Cloninger et al., 1994, p.1), and is assessed by novelty seeking, harm avoidance, reward dependence and persistence dimensions. Character
refers to “self-concepts and individual differences in goals and values which influence voluntary choices, intentions, and the meaning of what is experienced in life” (Cloninger et al., 1994, p.1), and is measured by self-directedness, cooperativeness and self-transcendence dimensions.

Eating disorder studies using the TCI have characterised women with AN as having high harm avoidance, low cooperativeness (Klump et al., 2000) and high persistence (Diaz-Marsa et al., 2000). Women with BN have been reported to have high harm avoidance, high novelty seeking (Mizushima, 1998) and low self-directedness (Bulik et al., 1995a). One of the few studies to examine temperament and character in BED (compared to obese women) showed lower self-directedness in this group and suggested that this character dimension may be the strongest predictor for the development of BED (Fassino et al., 2002b).

The TCI has been used for many purposes within eating disorders research. Studies examining the TCI have used this instrument for defining personality profiles (Fassino et al., 2002a; Rybakowski et al., 2004), highlighting differences between eating disorder subgroups (Klump et al., 2000; Vervaet et al., 2004b), as a measure of severity of eating disorder behaviour (Abbate-Daga et al., 2005), examining personality characteristics before and after recovery of an eating disorder (Klump et al., 2004), creating a profile of dropouts from brief psychotherapy (Fassino et al., 2003) and as a predictor of outcome (Bulik et al., 1998b).

Studies of personality dimensions using a range of measures in BED have found personality characteristics and emotional disturbance were significantly related to binge-eating severity (greater emotional disturbance led to increased binge eating;
Kolotkin, Revis, Kirkley, & Janick, 1987). One of the only studies to compare BED with other eating disorder subgroups and control groups found that patients with BED scored lower on well-being (on the Multidimensional Personality Questionnaire) and higher on harm avoidance than a normal-weight comparison group (Peterson et al., 2010). Levels of well-being were comparable between the BN and BED group although the BN group showed greater stress reaction than the BED or control groups (Peterson et al., 2010).

Further research into temperament and character has the potential to increase understanding regarding the development, maintenance and prognosis of BED. If fundamental differences exist in personality between BN and BED this may help to explain differences seen in other areas such as course and outcome of the eating disorder (Agras, Crow, Mitchell, Halmi, & Bryson, 2009; Fairburn et al., 2000; Fichter, Quadflieg, & Hedlund, 2008b).

11.3 AIMS

The aims of this exploratory study are:

Aim 1: To examine specific personality traits using TCI scales in women with BN and BED and compare with a depressive control group and a healthy control group of women

Aim 2: To evaluate facets of the TCI to provide greater sensitivity in further distinguishing between the eating disorder subtypes, depressive control groups and healthy control group
11.4 **Method**

11.4.1 **Overview**

The BEP study, described earlier in Chapter 5, was used for these analyses.

11.4.2 **Participants**

BED and BN participants were consecutive referrals to the treatment trial. Inclusion criteria for this study were women aged 16 and over with a current primary DSM-IV diagnosis of BN or BED and a BMI>17.5. Exclusion criteria were outlined in Chapter 5. Of the 80 participants entering the study, one was excluded from analyses as TCI data were missing.

The depressive control group was made up of participants recruited for ‘The Molecular Genetics of Depression and Personality’ family study (Joyce, McHugh, Light, Rowe, Miller, & Kennedy, 2009). This study aimed to address the genes which contribute to the vulnerability to suffer from MDD and which influence symptom patterns, comorbid diagnoses, risk taking behaviours or personality traits. Inclusion criteria were: participants that have suffered from MDD, aged 18 years or above and had two first degree relatives (parents, sibling or children) who were willing to participate in the assessment. From this study, a group of women aged 15 to 70 with current MDD were selected to represent the depressive control group for these analyses.

The healthy control group was recruited as an age-and-BMI-matched comparison group for the eating disorder participants in BEP study. Inclusion criteria were women aged 16 and over. Exclusion criteria were: current or past eating disorders, current or past binge
eating or purging behaviours, current or past bipolar I, schizophrenia, developmental learning disorder or cognitive impairment and current psychoactive medication.

11.4.3 Procedure

This study received ethical approval from the Upper Regional South A Ethics Committee. Participants provided written informed consent.

11.4.3.1 Pre-treatment assessment

Participants completed a comprehensive assessment protocol at pre-treatment including clinician rated diagnostic assessment for Axis I diagnoses and rating of mood and eating disorder symptoms and status; biological measures; a neuropsychological assessment; and a range of self-report measures assessing eating disorder related attitudes and symptoms, psychopathology, personality and other psychological functioning. Measures relevant to this are the TCI-R (Cloninger, 1999) and the MADRS (Montgomery & Asberg, 1979).

*The TCI-R*

The TCI-R is a 240-item self-report questionnaire providing a comprehensive assessment of personality using the seven dimensions of temperament and character (Cloninger, 1999). The dimensions can be subdivided into facets which use high and low score descriptors (opposite pairs) to summarise the temperament and character traits (resulting in an overall total score for the scale; Cloninger et al., 1994) (Cloninger, 1999).
Temperament dimensions include novelty-seeking, harm avoidance, reward dependence and persistence scales. Novelty seeking consists of four facets: exploratory excitability versus stoic rigidity (NS1), impulsiveness versus reflection (NS2), extravagance versus reserve (NS3) and disorderliness versus regimentation (NS4). Harm avoidance consists of four facets: anticipatory worry and pessimism versus uninhibited optimism (HA1), fear of uncertainty versus confidence (HA2), shyness with strangers versus outgoing (HA3) and fatigability versus vigour (HA4). Reward dependence consists of three facets: sentimentality versus tough mindedness (RD1), openness to warm communication versus reserved (RD2), attachment versus detachment (RD3) and dependence versus independence (RD4). Persistence consists for four facets: eagerness of effort versus laziness (P1), work hardened versus spoiled (P2), ambitious overachieving versus underachieving (P3) and perfectionistic perseveration versus pragmatic quitting (P4; Cloninger et al., 1994).

Character is evaluated by self-directedness, cooperation and self-transcendence dimensions. Self-directedness consists of six facets: responsibility versus blaming (SD1), purposefulness versus lack of goal direction (SD2), resourcefulness versus inertia (SD3), self-acceptance versus self-striving (SD4) and congruent second nature versus bad habits (SD5). Cooperativeness consists of five facets: social acceptance versus social intolerance (C1), empathy versus social disinterest (C2), helpfulness versus unhelpfulness (C3), compassion versus revengefulness (C4) and integrated conscience versus self-serving advantage (C5). Self-transcendence consists of three facets: creative self-forgetfulness versus self-consciousness (ST1), transpersonal identification versus self-isolation (ST2) and spiritual acceptance versus rational materialism (ST3; Cloninger et al., 1994).
Age effects on the TCI dimensions have been consistently identified in which self-directedness and cooperativeness increases with age and novelty-seeking decreases with age (Brandstrom, Sigvardsson, Nylander, & Richter, 2008; Cloninger et al., 1993; Parker, Cheah, & Parker, 2003). The effects of age will be controlled for within the analyses. Furthermore, harm avoidance and self-directedness are widely associated with MDD therefore the effects of depression will be controlled for within the analyses (Abrams, Yune, Kim, Jeon, Han, Hwang, Sung, Lee, & Lyoo, 2004; Farmer et al., 2003; Hansenne et al., 1999; Richter, Polak, & Eisemann, 2003; Smith et al., 2005).

11.4.3.2 Statistical analyses

The Statistical Package for the Social Sciences (SPSS, Version 13) was used to analyse data. Analysis of variance with post hoc comparisons was used to compare BN, BED, the depressive control group and the healthy control group with regard to the TCI scales and facets. Analyses of covariance were conducted with age as a covariate to correct for any age effects on TCI dimensions and with MADRS scores as a covariate to correct for depressive effects on harm avoidance and self-directedness subscales. As this was an exploratory study, a two-tailed p-value (<0.05) was used to indicate statistical significance.
11.5 RESULTS

11.5.1 Demographic and clinical features for the groups

Table 11.1 shows the pre-treatment descriptive characteristics of the group. The ages of the eating disorder sample ranged from 16 to 63 years and the BMI ranged from 17.9 to 56. The majority of the sample were New Zealand European or ‘Other’ and 45% had never been married. Almost half the sample was employed. The BED group had a significantly higher age, BMI and longer duration of the disorder than the BN group.

The mean age of the depressive control group was 40.0 years (SD=13.0) and the mean age-of-onset of MDD was 18.7 years (SD=8.3). The sample was made up of New Zealand European (97%) and Other (3%). There was no BMI measurement for this group.

The mean age of the healthy control group was 30.5 years (SD=10.5) and the mean BMI was 26.6 (SD=5.4). The sample was made up of New Zealand European (72%), Maori (6%) and Other (22%). There was no significant difference between the eating disorders group and the no eating disorders comparison groups on age ($p=0.23$) however, differences in BMI were significant among the groups ($p=0.05$).

11.5.2 TCI scores

Analyses were conducted with age as a covariate however, few differences with the raw data were found. Age was significantly associated with ambitious overachieving (P3) which became a non-significant result ($p=0.25$) and self-acceptance (S4) which became a significant result ($p=0.03$). Although age did not significantly affect helpfulness (C3, $p=0.19$), this result became significant when age was corrected ($p=0.05$).
The effects of mood on harm avoidance and self-directedness subscales were controlled for by using MADRS scores as a covariate. Controlling for depression did not alter the statistical significance of any differences found between BN and BED in terms of harm avoidance or self-directedness.

11.5.3 Temperament and character in BN and BED in comparison to a depressive control group and a healthy control group

The pattern of TCI profiles across the four groups is displayed in Figure 11.1. Women with BN and BED were significantly lower in harm avoidance and self-transcendence compared to the depressive control group (Table 11.1). In comparison to healthy controls, the BN and BED groups had higher harm avoidance, lower self-directedness and lower self-transcendence. Furthermore, the BED group was lower in cooperativeness compared to the healthy control group. There were no significant differences for novelty seeking, persistence or reward dependence subscales across the four groups.

11.5.4 Comparisons in temperament and character between BN and BED

Self-directedness and cooperativeness were significantly lower in the BED group than the BN group but there were no significant differences for other TCI scales.
11.5.5 Specific differences in TCI facets across the four groups

Harm avoidance (and all its facets) was elevated in all three clinical groups relative to the healthy control group. Several harm avoidance facets (anticipatory worry (HA1) and fatigability (HA4)) were higher in the depressive group relative to both eating disorder groups and the healthy controls. The difference in fatigability was still significant when pre-treatment depression scores were taken into account however, this was not the case for anticipatory worry. Within the eating disorder groups, the only significant difference on the harm avoidance subscale was for the BED group who had higher fatigability compared to the BN group.
Table 11.1: Comparison of TCI personality traits across BN, BED, depressive controls and healthy controls

<table>
<thead>
<tr>
<th>TCI subscales</th>
<th>BN group a (n=38)</th>
<th>BED group b (n=41)</th>
<th>Depressive Controls c (n=39)</th>
<th>Healthy Controls d (n=44)</th>
<th>F</th>
<th>p</th>
<th>Post Hoc</th>
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</thead>
<tbody>
<tr>
<td><strong>Novelty seeking</strong></td>
<td></td>
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<tr>
<td>NS1</td>
<td>32.1 (0.9)</td>
<td>29.4 (0.8)</td>
<td>29.5 (0.9)</td>
<td>33.4 (0.6)</td>
<td>6.3</td>
<td>&lt;0.001</td>
<td>a, d&gt;b, c</td>
</tr>
<tr>
<td>NS2</td>
<td>25.0 (0.9)</td>
<td>26.1 (0.9)</td>
<td>23.0 (0.9)</td>
<td>24.84 (0.6)</td>
<td>2.6</td>
<td>NS</td>
<td></td>
</tr>
<tr>
<td>NS3</td>
<td>27.3 (1.4)</td>
<td>31.6 (1.2)</td>
<td>28.4 (1.02)</td>
<td>29.8 (0.9)</td>
<td>2.7</td>
<td>0.05</td>
<td>b&gt;a, c</td>
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<tr>
<td>NS4</td>
<td>20.7 (0.6)</td>
<td>21.1 (0.6)</td>
<td>18.6 (0.7)</td>
<td>18.3 (0.5)</td>
<td>5.7</td>
<td>0.001</td>
<td>a, b&gt;c, d</td>
</tr>
<tr>
<td>NSTOT</td>
<td>105.0 (2.6)</td>
<td>108.2 (2.4)</td>
<td>99.5 (2.4)</td>
<td>105.9 (1.7)</td>
<td>5.8</td>
<td>NS</td>
<td></td>
</tr>
<tr>
<td><strong>Harm Avoidance</strong></td>
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<td></td>
</tr>
<tr>
<td>HA1</td>
<td>33.4 (1.1)</td>
<td>34.3 (1.3)</td>
<td>38.6 (0.6)</td>
<td>27.4 (0.9)</td>
<td>20.0</td>
<td>&lt;0.001</td>
<td>c&gt;a, b&gt;d</td>
</tr>
<tr>
<td>HA2</td>
<td>24.1 (0.8)</td>
<td>25.6 (0.9)</td>
<td>27.3 (0.6)</td>
<td>21.7 (0.6)</td>
<td>11.0</td>
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</tr>
<tr>
<td>HA3</td>
<td>23.3 (1.1)</td>
<td>23.1 (1.0)</td>
<td>25.7 (0.9)</td>
<td>19.9 (0.8)</td>
<td>6.7</td>
<td>&lt;0.001</td>
<td>a, b, c&gt;d</td>
</tr>
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<td>HA4</td>
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<td>30.6 (0.8)</td>
<td>21.4 (0.9)</td>
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<td>HATOT</td>
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<td>111.0 (3.2)</td>
<td>122.2 (2.3)</td>
<td>90.4 (2.3)</td>
<td>24.8</td>
<td>&lt;0.001</td>
<td>c&gt;a, b&gt;d</td>
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<tr>
<td><strong>Reward Dependence</strong></td>
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<td>RD1</td>
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<td>0.008</td>
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<td>36.4 (1.0)</td>
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<td>0.02</td>
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<td>0.02</td>
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<td>103.4 (2.3)</td>
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<tr>
<td><strong>Persistence</strong></td>
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</tr>
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<td>29.7 (1.0)</td>
<td>26.9 (1.0)</td>
<td>26.8 (1.0)</td>
<td>30.1 (0.8)</td>
<td>3.3</td>
<td>0.02</td>
<td>a, d&gt;b, c</td>
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<td>26.2 (0.9)</td>
<td>24.3 (0.9)</td>
<td>25.5 (0.8)</td>
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<tr>
<td>P3</td>
<td>34.4 (1.2)</td>
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<td>29.4 (1.0)</td>
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<td>PTOT</td>
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<td>107.6 (3.0)</td>
<td>113.6 (2.8)</td>
<td>2.0</td>
<td>NS</td>
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Table 11.1: Continued

<table>
<thead>
<tr>
<th>TCI subscales</th>
<th>BN group&lt;sup&gt;a&lt;/sup&gt; (n=38)</th>
<th>BED group&lt;sup&gt;b&lt;/sup&gt; (n=41)</th>
<th>Depressive Controls&lt;sup&gt;c&lt;/sup&gt; (n=39)</th>
<th>Healthy Controls&lt;sup&gt;d&lt;/sup&gt; (n=50)</th>
<th>F</th>
<th>p</th>
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<tr>
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<tr>
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<td>26.3 (0.9)</td>
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<td>14.9 (0.6)</td>
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<td>35.7 (0.9)</td>
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<tr>
<td>SD5</td>
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<td>27.4 (0.9)</td>
<td>33.8 (1.2)</td>
<td>40.8 (0.9)</td>
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<td>d&gt;c&gt;a&gt;b</td>
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<td>120.4 (3.1)</td>
<td>126.7 (3.1)</td>
<td>150.7 (2.8)</td>
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<td><strong>Cooperativeness</strong></td>
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<tr>
<td>C1</td>
<td>31.3 (0.6)</td>
<td>29.0 (0.7)</td>
<td>30.3 (0.7)</td>
<td>32.4 (0.6)</td>
<td>5.4</td>
<td>0.001</td>
<td>d&gt;b, c</td>
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<tr>
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<td>18.0 (0.6)</td>
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<td>26.9 (0.8)</td>
<td>29.6 (0.6)</td>
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<td>0.001</td>
<td>d&gt;b, c</td>
</tr>
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<td>C5</td>
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<td>29.8 (0.6)</td>
<td>30.7 (0.6)</td>
<td>31.5 (0.6)</td>
<td>1.7</td>
<td>NS</td>
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</tr>
<tr>
<td>CTOT</td>
<td>139.0 (2.1)</td>
<td>132.2 (2.5)</td>
<td>136.7 (2.0)</td>
<td>144.1 (2.1)</td>
<td>5.3</td>
<td>0.002</td>
<td>d&gt;b, c</td>
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<td><strong>Self-transcendence</strong></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>ST1</td>
<td>22.9 (1.2)</td>
<td>24.4 (1.1)</td>
<td>27.2 (1.1)</td>
<td>26.0 (1.2)</td>
<td>2.5</td>
<td>NS</td>
<td></td>
</tr>
<tr>
<td>ST2</td>
<td>16.3 (0.7)</td>
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<td>STOT</td>
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<td>61.2 (2.5)</td>
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<td>4.6</td>
<td>0.004</td>
<td>c, d&gt;a, b</td>
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</tbody>
</table>

Abbreviations: BN= BN, BED=Binge Eating Disorder
Notes: Analyses are presented are raw data and without age as a covariate
The healthy control group had higher levels of self-directedness (and all its facets) than the three clinical groups. The BN group had higher scores on three of the five self-directedness facets in relation to the BED group (responsibility (SD1), resourcefulness (SD3) and congruent second nature (SD5)). The differences in these in responsibility, resourcefulness and congruent second nature remained significant when pre-treatment depression scores were taken into account. On the congruent second nature facet (SD5) significant differences were found between each of the groups, ranked from highest to lowest as follows: healthy controls, depressive group, BN and BED.

Low levels of cooperativeness (and its facets) were found in all three clinical groups in comparison to the healthy controls. The BED group resembled the depressive group in having significantly lower scores on the cooperativeness facets of social acceptance (C1) and compassion (C4) compared to the BN and healthy control group.

11.6 DISCUSSION

Women with BN and BED have largely similar personality traits that distinguish them from a healthy control group but not necessarily from a depressive control group. Despite these similarities, there are some interesting differences that exist between BN and BED on self-directedness and cooperativeness. Interestingly, the TCI profile presents the BED group as less responsible, less resourceful, less self-accepting, less socially accepting and less compassionate than the BN group. This may challenge assumptions that the BN group has greater problems of personality than the BED group.

This study suggests that specific personality traits common to psychiatric disorders are greater harm avoidance (HATOT) and lower self-directedness (SDTOT). Lower self-
transcendence (STTOT) is specific to BN and BED and lower cooperativeness (CTOT) appears in women with BED and depressive controls, but not necessarily at a significant level in women with BN. Within these dimensions, only lower congruent second nature (SD5) and creative self-forgetfulness (ST1) are specifically associated with an eating disorder. The following facets are significantly associated with any psychiatric disorder (BN, BED and depressive controls): greater anticipatory worry and pessimism (HA1), greater fear of uncertainty (HA2), greater shyness with strangers (HA3), lower responsibility (SD1), lower purposefulness (SD2) and lower resourcefulness (SD3).

These findings are consistent with the literature that describe women with BN as high in harm avoidance and low in self-directedness (Bulik et al., 1995a; Diaz-Marsa et al., 2000), but are not consistent with studies that show high novelty seeking in women with BN (Kleifield, Sunday, Hurt, & Halmi, 1994; Mizushima, 1998; Waller, Gullion, Petty, Hardy, Murdock, & Rush, 1993). In addition, our results demonstrating low self-directedness and cooperativeness as character traits seen in those with BED were consistent with previous research (Fassino et al., 2002b; Grucza, 2006).

The role of self-directedness may also be of importance. Self-directedness has been implicated as a predictor of outcome for women with eating disorders (Bulik et al., 1998b). Improvements in self-directedness can be achieved with psychotherapy or pharmacotherapy (Anderson et al., 2002; Rybakowski et al., 2005). Given the significant difference in self-directedness between the BN and BED groups in our study, a particular focus on increasing self-directedness in women with BED may be necessary for the improvement in eating disorder symptoms and/or global functioning. Using the TCI data above, it may be useful to focus on improving the level of acceptance of one’s
actions, developing problem-solving skills and instilling good habits that become automatic responses and override impulses (Cloninger et al., 1994).

In addition to self-directedness, the cooperativeness scale was the only other difference in temperament and character scales between BN and BED. Cooperativeness has been correlated with the presence of a personality disorder (Mulder et al., 1999) and frequently occurs across all eating disorder types (Fassino et al., 2002a; Klump et al., 2000). It has been suggested that the combination of low cooperativeness, high harm avoidance and low self-directedness may impair an individual’s response to stressful situations through “behavioral inhibition, fear, anxiety, and depression” (Cassin & von Ranson, 2005, p. 902). The significant difference between BN and BED suggests an increased tendency for the BED group to experience these emotions and provides additional information to inform treatment goals.

There are contradictory studies about the extent of differences between BN and BED in the course and outcome of the eating disorder. Studies by Fairburn et al., (2000) and Agras et al., (2009) suggest that BN and BED have a different course and outcome, with BN having a poorer prognosis. In contrast, Fichter et al., (2008b) found a similar course and outcome between BN purging type and BED. The few differences found in personality profiles in our current study were in the direction for BED to appear worse than the BN group. Therefore, if self-directedness is a predictor of outcome, we would expect a poorer prognosis in the BED group rather than the BN group. It is possible however, that the differences found between BN and BED on self-directedness and cooperativeness may not be significant enough to have negative prognostic implications.
on the BED group. Future research investigating the impact of temperament and character on the outcome of BED is necessary to answer these questions.

11.6.1 Limitations

A limitation of this study is that it did not distinguish between purging and non-purging BN. Research has shown a personality difference in the TCI persistence subscale between the non-purging BN and BED groups (Vervaet, van Heeringen, & Audenaert, 2004a) however, this study’s small sample size precluded examination of this issue. Analyses of all facets, regardless of the significance of the overall scale, may increase the chance of Type I error. A correction of these statistics was not done as this study was exploratory in an attempt to uncover individual contributions from the facets that are potentially masked by only examining the overall scale.

11.7 Conclusions

In conclusion, our findings suggest that temperament and character profiles are fundamentally similar between BN, BED and depressive controls. However, the significant differences in self-directedness and cooperativeness detected between the BN and BED groups warrants further exploration in relation to the possible contribution of these factors to the course and outcome of these disorders.
PART 6

Chapter 12: Discussion

12.1 SUMMARY OF KEY FINDINGS

The relationship between Axis I disorders such as eating disorders, and Axis II personality disorders is not independent, with personality disorders impacting the clinical presentation but not the outcome of eating disorders. The four key findings from this thesis were: 1) personality disorders had a negative impact on current depressive severity and global functioning of women with BN at pre-treatment but did not affect the long-term outcome three-years post-treatment, 2) self-directedness was the only personality predictor of any eating disorder diagnosis at five-year follow-up, 3) there was a differential impact of the presence of personality disorders on binge eating severity in BN (fewer binges) and BED (more binges), 4) TCI personality profiles were largely similar in BN and BED with the exception of self-directedness and cooperativeness which were slightly worse in BED compared to BN.

In Chapters 6, 7 and 8, borderline personality disorder, avoidant personality disorder and complex personality disorders were found to have a negative impact on the clinical characteristics in women with BN at pre-treatment but not long-term outcome. Women in the BN and B-PD group had increased Axis I comorbidity, poorer global functioning and elevated eating disorder attitude scores at pre-treatment compared to Other-PDs and No-PD groups. Similarly, AV-PD had a negative impact on current depressive severity and global functioning at pre-treatment compared to women with Other-PDs and No-
PD. The Complex-PD group showed greater Axis I comorbidity, poorer global functioning, greater current depressive severity and poorer eating disorder attitudes than the groups with less severe personality disorder dysfunction (No-PD, Personality Difficulty and Simple-PD). Despite this adverse effect at pre-treatment, by three-year follow-up there were no significant differences on eating disorder symptomatology, current depressive severity or global functioning between the personality disorder groups and no personality disorder groups at three-year follow-up for the B-PD, AV-PD or Complex-PD analyses. Overall, these findings indicate that at pre-treatment the presence of a personality disorder does have a negative impact on depression severity and global functioning. On all these analyses however, the presence of a personality disorder did not impact on long-term outcome at one-year and three-year follow-up.

In Chapter 9, self-directedness alone predicted the presence of any eating disorder diagnosis at five-year follow-up whereas other dimensional measures of personality characteristics from three different instruments (TCI, EDI-2 and SCID-II personality disorder symptoms) did not. Other predictors of five-year outcome using those three instruments were asceticism (EDI-2) which predicted any mood disorder episode (past year), and borderline symptoms (SCID-II) which predicted poor global functioning in the past year.

In Chapter 10, both BN and BED groups had worse general psychopathology when any personality disorder was present however, a differential impact of personality disorders on BN and BED was found regarding eating disorder symptomatology, with a decrease in binges in those with BN and an increase in binges in those with BED. Overall, there were remarkably few other differences on Axis I and II comorbidity, eating disorder
symptomatology, general psychiatric functioning and global functioning variables between BN and BED groups.

In Chapter 11, women with BED were significantly lower on self-directedness and cooperativeness subscales compared to women with BN. Overall, women with BN and BED had similar personality traits compared to depressive controls but differed from the normal control group in having more personality dysfunction.

12.1.1 Impact of personality disorders at pre-treatment

The findings from this study are consistent with previous reports in the eating disorder literature that personality disorders adversely affect general psychiatric functioning and global functioning but not necessarily eating disorder symptomatology (Ben-Porath et al., 2009; Cassin & von Ranson, 2005; Johnson et al., 1989; Steiger & Stotland, 1996; Zeeck et al., 2007). In this study, those with borderline, avoidant or complex personality disorders had increased depressive severity and poorer global functioning but similar levels of binge eating and purging behaviours at pre-treatment.

The impact of Axis II disorders in these eating disorders is in general psychopathology and life functioning as it is across a range of Axis I disorders (Dreessen et al., 1994; Nace et al., 1991; Noyes et al., 1990; Reich, 1990; Skodol et al., 2005).

The finding in this thesis, that in those with BED the presence of a personality disorder is associated with increased binge eating frequency, supports previous studies that have reported this association (Picot & Lilienfeld, 2003; Wilfley et al., 2000a).
12.1.2 Differential impact of personality disorders on BN and BED

Women with BED and comorbid personality disorders had increased binge eating episodes and fear of losing control over eating compared to women with BN and comorbid personality disorders in this study. This challenges clinical assumptions that those with BED have less psychopathology than those with BN. It also suggests the importance of considering Axis II comorbidity in the assessment and treatment of those with BED.

In one of the few studies measuring differences in personality pathology between women with BN and BED, the only significant difference reported has been in the prevalence rates of borderline personality disorder (BED<BN; van Hansijck De Jonge, Van Furth, Lacey, & Waller, 2003). In the BED group, the presence of Axis II disorders has been associated with more severe binge eating and other eating disorder psychopathology (Wilfley et al., 2000a). Furthermore, increased binge eating one-year post-treatment was predicted by the presence of Cluster B personality disorders in women with BED (Wilfley et al., 2000a). Outside the findings from this thesis, few direct comparisons have been made between BN and BED on the impact of Axis II comorbidity on eating disorder symptomatology, general psychiatric functioning and global functioning.
12.1.3 Impact of personality disorders on outcome in BN

Findings from this study suggest that the presence of a personality disorder does not adversely affect the outcome of BN despite clinical assumptions that comorbid personality disorders result in poor outcome for eating disorders. The results presented in this thesis are inconsistent with studies reporting a negative effect on BN outcome (Johnson et al., 1990; Keel & Mitchell, 1997; Steiger & Stotland, 1996).

The wider literature is mixed regarding the impact of comorbid personality disorders on the outcome of Axis I disorders. Whilst some studies have found that the presence of a personality disorder predicts poorer outcome in MDD (Newton-Howes et al., 2006), anxiety disorders (Alnaes & Torgersen, 1999) and substance use disorders (Thomas et al., 1999; Zikos et al., 2010), other studies did not find any adverse effect on the outcome of these Axis I disorders (Dreessen & Arntz, 1998; Kool et al., 2007; Mulder, 2002; Verheul, 2001). Similarly to findings for other Axis I disorders, the literature is mixed regarding the impact of comorbid Axis II disorders with eating disorders (Bruce & Steiger, 2006). Bruce et al., (2006) suggested that contradictory findings may be the result of the use of trait dimensions (such as self-directedness or impulsivity) rather than categories representing more general personality organisation (such as borderline personality disorder). Findings from this study concur with this view and suggest that the personality trait self-directedness may offer more reliable prognostic information than categorical measures such as DSM-IV diagnoses.

12.1.4 Self-directedness as a predictor of any eating disorder outcome

The only positive predictor of any eating disorder outcome found in this study was pre-treatment self-directedness. The important facets of self-directedness in relation to
predicting outcome were identified as purposefulness, resourcefulness and congruent second nature.

The findings from this thesis are generally consistent with the literature that describe women with BN as having low self-directedness and high harm avoidance (Diaz-Marsa et al., 2000; Fassino et al., 2002a). Previous studies using the same data from this BN (BTS) sample reported that self-directedness predicted BN outcome at one-year follow-up (Anderson et al., 2002; Bulik et al., 1998b). In this thesis, those analyses were replicated and extended to show that self-directedness predicts eating disorder outcome at five-year follow-up. Low self-directedness has been related to higher rates of drop-out in women with BN which may be a contributing factor to poorer outcome (Fassino et al., 2003). The character facets identified as significant in this study (purposefulness, resourcefulness and congruent second nature) have also been previously identified as significantly lower in a BN group compared to healthy controls and may be important in highlighting areas to develop in women with BN (Fassino et al., 2002a).

Self-directedness appears to be capturing a critical aspect of personality functioning that has predictive validity across Axis I disorders. Self-directedness has been reported to be a general predictor of outcome for other Axis I disorders such as depression (Corruble, Duret, Pelissolo, Falissard, & Guelfi, 2002), and for other treatment outcomes such as pharmacotherapy (Black & Sheline, 1997; Sato et al., 1999). Self-directedness also appears to encapsulate many of the important characteristics required for successful treatment. Increasing this characteristic may be of benefit for the treatment of women with BN. Existing therapy strategies that relate to self-directedness include increasing the patient’s self-efficacy beliefs through mastery experiences, mindfulness, improving
problem solving skills, increasing assertiveness and goal setting, all of which target the three important facets identified in this thesis. It may be particularly important to emphasise these strategies whenever low self-directedness is identified at pre-treatment.

12.1.5 Differences between BN and BED

Women with BN and BED were found to have few differences on Axis I and II comorbidity, eating disorder symptoms, general psychiatric functioning and global functioning at pre-treatment. One of the few differences found was the higher rates of obsessive-compulsive personality disorder in the BED group compared to the BN group. Prevalence rates of obsessive-compulsive disorder have been reported to be around 18% in AN and BN samples (Jordan et al., 2008) however, this reached 27% in the BED (BEP) sample. Higher rates of obsessive-compulsive personality disorder have been consistently found in BED samples and those with this comorbidity (BED and obsessive-compulsive personality disorder) may represent a distinct subgroup (Sansone et al., 2005). In other eating disorders such as AN, obsessive-compulsive personality disorders and traits have been identified as a potential marker of vulnerability to an eating disorder (Anderluh et al., 2003b; Halmi, 2005) and have been associated with persistence of eating disorder symptoms and poorer general functioning at follow-up (Pollice, Kaye, Greeno, & Weltzin, 1997).

Personality traits measured by the TCI were similar between BN and BED groups with the exception of self-directedness and cooperativeness. The few differences between BN and BED suggest a similar psychopathology in these groups, contrary to clinical assumptions that the diagnosis of BN is associated with greater pathology. With respect to the differences though, TCI scores indicated that the BED group was less responsible,
less resourceful, less self-accepting, less socially accepting and less compassionate than the BN group. These differences should be examined for their prognostic value in predicting BED outcome.

The presence of inflexible, high expectations of self and others (very high levels of obsessive-compulsive personality disorder, low self acceptance, low social acceptance and compassion) combined with low resourcefulness suggests that women with BED may be experiencing a significant discrepancy between expectations of themselves, the world and the reality they experience. This discrepancy is likely to be associated with negative affect, especially when combined with fewer adaptive coping resources (low resourcefulness). Negative affect is well recognised as a maintaining factor for binge eating (Agras & Telch, 1998; Deaver, Miltenberger, Smyth, Meidinger, & Crosby, 2003; Stice, Akutagawa, Gaggar, & Agras, 2000). Rigid high expectations are characteristics of other eating disorders as well, however the combination of these with low resourcefulness (which appears to be more problematic in the BED group) may be contributing to the higher level of general and eating psychopathology seen here.

Although these findings may support existing etiological models and maintaining factors in eating disorders (Fairburn, Marcus, & Wilson, 1993a; Peterson, Wonderlich, Mitchell, & Crow, 2004; Wonderlich, Mitchell, Peterson, & Crow, 2001), the findings of this thesis suggest that for most, the presence of personality pathology does not impact on long-term outcome and so specific treatment targeting personality disorders is not required. Elements of existing therapies such as CBT and Integrative Cognitive-Affective Therapy may already be addressing expectations and working to increase
resourcefulness for those with BN as a whole (Wonderlich, Engel, Peterson, Robinson, Crosby, Mitchell, Smith, Klein, Lysne, Crow, Strauman, & Simonich, 2008).

12.2 **METHODOLOGICAL STRENGTHS AND LIMITATIONS**

12.2.1 **Methodological strengths**

12.2.1.1 **Sample and comparison groups**

The sample size of the BTS study (n = 135) is moderate in relation to other studies examining comorbidity between eating disorders and personality disorders (Maranon et al., 2004; Sansone & Levitt, 2005b; Thompson-Brenner & Westen, 2005).

The use here of psychiatric and healthy control groups (see Chapter 11) addresses the criticism regarding a lack of appropriate comparison groups in this field of study. Using these control groups allowed for examination of disorder-specific personality profiles and assisted in clarifying the associations found in the study. For example, much of the personality pathology was present across all three clinical groups and was not necessarily specific to eating disorders.

12.2.1.2 **Assessments**

The use of structured clinical interviews and self-report questionnaires provided a comprehensive assessment of personality functioning. Selecting the DSM-IV and the TCI allowed for direct comparisons with other studies that used the same instruments. Independent assessments of personality disorder diagnoses by non-treating clinicians minimised potential bias that may have occurred during treatment.
12.2.1.3 Analyses

Controlling for other conditions reduced the risk that significant associations were a result of other confounding variables. In this study, an attempt was made to control for the presence of lifetime MDD, social phobia, age and current depressive symptoms.

12.2.2 Methodological limitations

12.2.2.1 Study design

This study did not address other broader predictor variables of outcome previously identified in the literature however, broader predictor variables of treatment using demographics, lifetime history (of Axis I disorders) and pre-treatment eating disorder status have been reported previously for the BTS sample (Bulik et al., 1998b).

12.2.2.2 Sample

As noted earlier in this thesis, this was a clinical sample which increases the likelihood of referral bias and Berkson’s bias resulting in inflated prevalence rates of personality disorders, and so these results may not be generalisable to community samples (Berkson, 1946; Vitousek & Stumpf, 2005).

The samples in this thesis were limited to women; therefore, findings cannot be generalised beyond this demographic. However, this is consistent with the majority of other clinical samples of those with BN and BED, and so allows for comparisons with other studies. Although the BN (BTS) had a moderately large sample size, the sample size of BN and BED groups in the BEP study were relatively small, limiting the power to detect differences. However, these were mainly exploratory analyses and were meant to generate further hypotheses that could be conducted with a larger sample.
Finally, the influence of the exclusion criteria for these trials might have resulted in lower prevalence rates of other Axis I disorders such as current severe major depression or current psychoactive substance use disorder. Despite this possibility, elevated rates of mood and alcohol use disorders were still found in the BTS sample and these were consistent with prevalence ranges in the literature (Duncan, Neuman, Kramer, Kuperman, Hesselbrock, Reich, & Bucholz, 2005; Hudson et al., 2007; Wonderlich & Mitchell, 1997). This suggests little or no effect of the exclusion criteria on sample selection.

12.2.2.3 Assessments

The personality disorder assessments for the BTS and BEP trials were conducted at pretreatment during the acute phase of the eating disorder, consistent with the methodology in much of the literature. As noted earlier, this practice has been criticised as it may increase recall and other biases increasing the risk of over-diagnosing personality disorders if some state variables are confused with trait variables.

12.2.2.4 Analysis

Use of multiple comparisons without correction to explore the dataset raises the risk of finding statistically significant results by chance (Type I error), but was considered justified for exploratory purposes when there was little or no information available from previous studies to generate hypotheses.

Reliability data were not available for the BTS or BEP study. However, there was some overlap in investigators and raters between these studies and a previously conducted anorexia treatment study and a depression study. The inter-rater reliability for those
raters on SCID-I and II assessments suggests good concordance with ranges from 0.78 to 0.85 (Carter et al., 1999; Jordan et al., 2008).

The relative failure of categorical personality disorders to predict outcome might be accounted for to some extent by the lesser power available in using dichotomous various relative to continuous variables. Indeed borderline personality traits did predict global functioning at five-year follow-up whereas the dichotomous B-PD diagnosis did not. Importantly though, even within dimensional measures, only a few variables were useful predictors. Of these, self-directedness was the strongest example.

Finally, where cross-sectional analyses have been conducted, the use of the term ‘impact’ in this context refers to associations and is not meant to imply causality.

12.3 IMPLICATIONS OF THESE FINDINGS FOR EATING DISORDER TREATMENT AND SERVICES

The American Psychiatric Association (American Psychiatric Association, 2000b) and NICE, (2004) support special clinical attention being given to patients with eating disorders and comorbid personality disorders (Bruce & Steiger, 2006). The guidelines suggest “the presence of comorbid personality disorders, particularly borderline personality disorder, dictates the need for longer-term therapy that focuses on the underlying personality structure and dealing with interpersonal relationships in addition to the symptoms of the eating disorder (American Psychiatric Association, 2000b, p. 27)”. However, findings from this thesis challenge the necessity for longer psychotherapy or more complex psychotherapy that is aimed at addressing personality
disorders within an eating disorder population, for example DBT for people with an eating disorder and borderline personality disorder (Palmer, Birchall, Damani, Gatward, McGrain, & Parker, 2003). The finding that the presence of a comorbid personality disorder in women with BN does not predict poor outcome at long-term follow-up should also work towards destigmatising clinical attitudes for this eating disorder group. The clear message for clinicians should be that those who are assessed with borderline personality disorder at pre-treatment should be given the same focussed eating disorder treatment as individuals without that diagnosis.

Women with BED had similar clinical characteristics to women with BN at pre-treatment however, the presence of a personality disorder had a negative impact on eating disorder behaviours in a way that was not seen in the BN group. This provides support for the small amount of literature that shows Axis II comorbidity negatively affects eating disorder symptomatology in women with BED. The implications of these findings may be significant. In New Zealand, specialist eating disorder services primarily focus on AN and BN while BED is often excluded from entry criteria for these services (Ministry of Health, 2006). Planning for eating disorder services in New Zealand appears to make no mention of BED, signalling little intention of incorporating treatment for this disorder into publically funded healthcare services in the years to come (Ministry of Health, 2007). Furthermore, the New Zealand Ministry of Health has declared BED to be a public health problem rather than a mental health problem which results in no funding for the treatment of BED (Bulik, 2010). Although BED does not have the same acute medical risks as AN or BN, this may be made up for by long-term conditions such as obesity. Findings from this thesis suggest that treatment should be available to women with BED in New Zealand.
The finding in this thesis that self-directedness predicts long-term outcome of eating disorders adds to the existing literature showing self-directedness as a predictor of outcome for multiple Axis I disorders (Anderson et al., 2002; Arnaud et al., 2008; Mortberg et al., 2007; Rybakowski et al., 2005; Sato et al., 1999). This suggest that self-directedness may be representing a core aspect of personality dysfunction that is related to impairment in broader life functioning. If this is so, then it would be important to measure the trait of self-directedness in future outcome studies. A further implication of this finding is that although eating disorder patients may not need treatment targeting personality disorders, they may benefit from more focussed therapeutic interventions to increase self-directedness prior to commencing therapy or within therapies such as CBT in order to increase the benefits from therapy.

12.4 SUGGESTIONS FOR FUTURE RESEARCH EXAMINING PERSONALITY AND EATING DISORDERS

Further examination of personality functioning in eating disorders is important to improve understanding of the relationships within this comorbidity. That they are related is well established however, going beyond the presence of the comorbidity relationship to understand the mechanisms of action and how the mechanisms function needs further exploration. Although there is a general consensus within the literature that personality disorders are associated with increased general psychopathology in women with eating disorders at pre-treatment, there is less agreement on the impact of personality disorders on eating disorder symptoms. As a result of these mixed findings, further research is required, including exploration of mediating factors or the possibility
of subgroups related to variables beyond the eating disorder and personality disorder symptoms.

The finding here of a differential impact of personality disorders on binge eating frequency in BED compared to BN requires replication and if confirmed, further exploration of possible mechanisms of action that could explain this effect may be required.

Research is also required to explore the relationship between BED and obsessive-compulsive personality disorder and related traits as this may establish whether those with this comorbid pattern form a potential subgroup within BED with prognostic significance. Given previous research suggesting that obsessive-compulsive personality disorder is one of the more stable personality disorders and may be associated with poorer outcome of AN, examining the specific effects of obsessive-compulsive personality disorder and related traits on the outcome of BED may be important. To do this, further research is required into identifying the “best” methods of assessing personality. Other measures aside from the SCID-II may be more applicable to understanding the personality traits involved in the construct of obsessive-compulsive personality disorder. In addition to categorical obsessive-compulsive personality disorder variables, future research should include alternative measures of persistence and perfectionism to comprehensively assess obsessive-compulsive personality features. More recently it has been suggested the following scales should be given greater future consideration for measuring obsessive-compulsive personality features: the Schedule for Nonadaptive and Adaptive Personality (SNAP), Wisconsin Personality Inventory-IV (WISPI-IV) and the OMNI Personality Inventory (Samuel & Widiger, 2010).
Further research is required to establish the replicability in other samples of the findings that categorical DSM-IV personality disorders did not predict outcome but the dimensional TCI character measure of self-directedness did predict any eating disorder outcome in women with BN at long-term follow-up.

Future research should also consider the construct of self-directedness. Further exploration is required into what the TCI self-directedness subscale is measuring and related to this, to compare the TCI self-directedness subscale with other measures of this construct such as the ‘self-aware consciousness’ scale in the Toronto Alexithymia Scale (Bishop Jr, 2008).

Finally, if low self-directedness predicts poor eating disorder outcome, would a pre-treatment focus on increasing self-directedness get a better outcome? Future research could examine whether increased sessions of cognitive therapy that have an initial focus on increasing self-directedness (with a particular emphasis on resourcefulness) might improve eating disorder outcome over long-term follow-up.

12.5 CONCLUSIONS

In this thesis, the personality trait of self-directedness was the only predictor of any eating disorder outcome at five-years in women with BN. Furthermore, despite adversely affecting pre-treatment clinical characteristics, the presence of a personality disorder did not impact on BN outcome three-years post-treatment whether examined in relation to the impact of specific personality disorders or a complex personality typology. These findings contribute to the literature by clarifying aspects of the relationship between eating disorders and personality.
Findings from this thesis suggest that when similar recruitment strategies are used in a clinical research setting, those with BED have significant difficulties that resemble those with BN regarding severity of general psychopathology and personality profiles. Indeed, these findings suggest that those with BED who have a personality disorder have worse binge frequency. The implications of these findings are that those with BED arguably have as great a need for treatment as those with BN and that service providers need to take this into account.

Personality functioning is an important factor to consider in those with eating disorders although the impact and role of personality may differ from long-held clinical assumptions. Current personality disorder categories have significant limitations and a change in focus from personality disorders to personality traits may provide more useful information regarding potential targets for interventions and in predicting the long-term success of treatment. It must also be acknowledged that in addition to personality, other factors such as genetic, biological and cultural factors impact upon the clinical characteristics and outcome of BN and BED. Further understanding is required of the relationship between these broader factors, personality and eating disorder factors in relation to outcome in order to improve treatments for those with eating disorders.
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APPENDICES

Appendix A  Borderline personality disorder paper
Appendix B  Avoidant personality disorder paper
Appendix C  Complex personality disorder paper
Appendix D  Dimensional personality paper
Appendix E  Information sheet and consent form for BTS
Appendix F  Information sheet and consent form for BEP study
APPENDIX A

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APPENDIX B

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APPENDIX D

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APPENDIX E
INFORMATION SHEET

BULIMIA NERVOSA TREATMENT STUDY

This study is being conducted by Dr. C. Bulik (Psychology Department, University of Canterbury), Dr. P. Sullivan, Professor P. Joyce and Ms F. Carter from the Clinical Research Unit, Adult Specialty Services, The Princess Margaret Hospital. The purpose of the study is to examine the effectiveness of psychological treatments for bulimia nervosa.

We are seeking women aged between 18-40 years who suffer primarily from bulimia nervosa (recurrent episodes of binge eating, a feeling of lack of control over eating during binges, purging to prevent weight gain and persistent preoccupation with body shape and weight). While subjects with mild or moderate depression, alcohol abuse, or a history of anorexia nervosa will be included in the study, we are not able to serve people with more serious medical or mental health problems.

Participation is voluntary and free of charge. Subjects are free to withdraw from the study at any time with appropriate referrals being provided. We do ask for a $50 (negotiable) refundable deposit for treatment materials. This will be refunded at follow-up appointments ($10 at the end of therapy; $20 at the 6-month follow-up; and $20 at the 13-month follow-up). Otherwise this money will be donated to WEDRC (Women with Eating Disorders Resource Centre).

The study involves participation in assessment, treatment and follow-up as follows:

ASSSESSMENT
1. We will examine your eating behaviour and attitudes, body image, alcohol and drug usage, mood, personality and mental health. This will be done via an interview, completing questionnaires and self-monitoring (keeping a diary) of problem behaviours.
2. A neuropsychological assessment will be conducted examining the following areas: concentration/attention, memory, perceptual abilities and intelligence.
3. Physiological Assessment - this involves measuring blood pressure and pulse following looking, smelling and eating a binge food.

These assessments will take place in three sessions over a span of two weeks.

TREATMENT
Cognitive Therapy
Treatment will occur in two phases. The first phase is eight sessions over a six week period. This treatment is called cognitive therapy. You and your therapist will explore information about bulimia nervosa, she will teach you the language used in cognitive therapy, and then you will work with her to identify and change problematic thoughts that are often associated with
bulimia nervosa. For example, after eating a binge food, women with bulimia sometimes say “I’ve blown my diet already, I may as well binge”. You will examine this thought and learn how to replace it with a healthier one that helps you stop binging. You will also learn better ways to solve problems and how to prevent relapses in the future.

**Behavioural therapies.**
After the cognitive therapy, you will have another assessment and you will be assigned randomly to one of three alternative therapies. Each of these therapies has been used in the treatment of eating disorders. You will not be able to choose which therapy you will receive. This second phase will involve eight sessions over a six week period. Descriptions of the three treatments follow:

**Exposure treatments.** Two of the treatments are called exposure with response prevention. These sessions teach you how to cope better with facing high risk situations up front. Exposure means putting yourself (with the help of your therapist) in a situation that would normally lead to a binge or a purge, but then restraining from doing so. For example, if you commonly binge in a fast food restaurant, you and your therapist might go to that restaurant. Initially you might feel the urge to binge and you might even feel anxious. However, over time, and by doing it more often without binging, you will find that the urge decreases and it will build your confidence so you do not have to binge every time you are there. Your therapist will monitor your pulse and blood pressure during exposure sessions and you will tell her how you are feeling as the session goes on.

**Relaxation training.** The third therapy is relaxation training. People with eating disorders often find that they have difficulty managing stress. Relaxation training involves learning breathing techniques, imagery, and deep muscle relaxation to help you deal with these stressful times.

All of the therapies involve “self monitoring” or keeping a diary of what you eat and your mood and thoughts. You will also be assigned homework so that you will be working on treatment even when you are not with your therapist. All treatments will be conducted on an individual basis by a clinical psychologist.

**FOLLOW-UP**
After treatment, we will ask you to come back to The Princess Margaret Hospital after 6 and 13 months during which time you will have an assessment to see how you are doing. At those times we will also ask you to keep a food and mood diary for another two weeks.

Throughout the study all reasonable measures will be taken to protect your confidentiality. Numbers, not names, will be used on all forms and participants’ identities will not be revealed on any publications that will result from the project.

If you are interested in participating in the study or would like to have more information, please contact: Leslie Livingston ph. 337-7696 372-9401
BULIMIA TREATMENT STUDY
CONSENT FORM

Please take your time and read through this consent form carefully. If you have any questions, please feel free to ask them now. It is important that you understand the nature of the study and the types of treatment we are offering you.

Brief description of the project: You are being offered the opportunity to participate in a treatment study for bulimia nervosa because you currently have bulimia and are between the ages of 18 and 40. The study will compare three different psychotherapies. All three of the therapies include eight sessions of cognitive therapy. This means that we will help you understand irrational and distorted thoughts that you may have about food, eating, yourself and the world in general. We will work with you to learn how to replace them with more healthy ways of viewing and thinking about these things. Everyone in the study will have this type of cognitive therapy.

In the beginning of the study, you will be randomly assigned (this means that you will not have a choice over which behavioural treatment you receive) to one of three behavioural treatments. One of the treatments involves you being exposed to the situations that commonly lead you to binge-eat with your therapist's help. For example, if you commonly have the urge to binge when you are in a fast food restaurant, you and your therapist will spend some time there until the urge to binge decreases. The second treatment involves you being exposed to foods that commonly give you the urge to purge. For example, if you commonly feel like purging after you eat pizza, you will be asked by your therapist to eat some pizza until you have the urge to purge. You will then remain with your therapist until the urge decreases. The third treatment involves you learning new techniques for relaxation that you will be able to use in stressful situations. All three of these treatments have been used extensively in the treatment of bulimia nervosa. You will only receive one of these treatments.

Risks associated with participation: There are very few risks associated with participation in this treatment study. You will be offered free treatment, so there will be no cost to you. We will ask for a $50 deposit (sliding scale if necessary) which will be refunded to you upon completion of your follow-up appointments at 6 and 13 months. Sometimes, people find that their bulimia may not improve with treatment. If this appears to be the case, we will discuss it with you and recommend other options for treating your eating disorder. You may also find the behavioural treatment to be uncomfortable at first. For example, sometimes people with an eating disorder do not like to eat in front of others or do not like to eat "forbidden foods." These behavioural treatments will not begin until you have been in the study for at least six weeks. By then you should be feeling more comfortable with these types of situations. We will try to make you as comfortable as possible throughout the treatment.

Time required: Before the actual treatment begins, you will be asked to keep a daily diary of what you eat and drink for two weeks. During that two week period, you will have two meetings with us in addition to our meeting today. You will be asked to fill out some questionnaires, undergo some cognitive evaluation, estimate your body size, and
have your blood pressure and pulse monitored first as you touch and smell and later as you eat a small amount of a food that you normally associate with binging. After the assessments, you will then start therapy. For the first four weeks you will meet with your therapist twice a week. For the remaining two weeks, you will meet once per week. The same pattern will hold for the behavioural therapy. The length of the behavioural sessions may vary. After your last treatment session, you will have another assessment (questionnaires, estimation of body size, blood pressure monitoring associated with eating). We will then contact you by phone after three months to see how you are coming along. Six months after treatment, we will ask you to self-monitor your eating again for two weeks and we will make another appointment for an assessment. We will contact you again by phone at 9 months. Finally, we will ask you to self-monitor for two weeks again after thirteen months and make one final appointment for your last assessment. If you have any questions at any other time, you can feel free to phone either one of the numbers listed below or your therapist.

Finally, if you are interested, we would be happy to send you general results of the study after everyone has completed treatment. Remember that this is a three year treatment trial, so it may be a while.

Name of researcher(s): Cynthia M Bulik, Ph.D. 364 2169 or 364 2992
Patrick F Sullivan, MD 337 7692
Peter R Joyce, MB ChB., PhD, FRANZCP
Frances Carter, MA, Dip Clin Psyc

I agree to participate in the project described above, on the understanding that if at any time I wish to withdraw from the study I may, without prejudice, do so. All information collected will be confidential as will the identity of participants.

Name: ____________________________
Signature: _________________________ Date: ___________
Witness: __________________________ Date: ___________
Communication with other health professionals may enhance the healthcare you receive. Any contact with these professionals would be brief and limited to summary information.

If your GP or a mental health professional referred you to the Bulimia Treatment Study, we are probably obliged to communicate with them.

Do you give us permission to communicate with the person who referred you to this study?

☐ NO

☐ YES . . . Name __________________________________________
Address ________________________________________________
Telephone ______________________________________________
Occupation ______________________________________________

Do you give us permission to communicate with your GP (If not the referral source)?

☐ NO

☐ YES . . . Name __________________________________________
Address ________________________________________________
Telephone ______________________________________________

Signature ______________________________________________
Date _______________
A comparison of three psychotherapies for binge eating

Information Sheet

Introduction

You are invited to take part in a clinical treatment trial of psychotherapies for women who have problems with binge eating, being conducted by Virginia McIntosh, Jenny Jordan, Janet Carter, Jan McKenzie, Janet Latner, Chris Frampton, Andrea Bartram and Sarah Rowe and Peter Joyce.

The major focus of this study is to compare the effectiveness of three psychotherapies (talking therapies) for binge eating problems. The three types of psychotherapy to be investigated in this study are: cognitive-behavioural therapy (CBT), Schema Therapy (ST) and nutrition and appetite focused CBT (NAFCBT). Cognitive-behavioural therapy helps people to understand and change cues that lead to binge eating, including unhelpful patterns of thinking, feeling and behaving, particularly those that relate to nutrition, food and eating. Schema therapy focuses on long-standing beliefs and patterns of behaving that contribute to binge eating. Nutrition and appetite focused CBT uses many of the same approaches as standard CBT, but has a particular emphasis on revised nutritional guidelines and emphasises the role of appetite, hunger, and fullness in problems with binge eating.

All three therapies will be conducted approximately weekly for six months (with the exact number of sessions in the range of 15-30), followed by approximately monthly sessions for 6 months. We would like to follow participants after the end of treatment to obtain a clear picture of the extent of the improvement and whether or not this is maintained. This will involve being interviewed and completing questionnaires at regular follow-up intervals.

Your participation in this study is completely voluntary (your choice). If you agree to take part you may withdraw at any time for any reason and this will in no way affect your future health care.

More about this study

What are the aims of this study? We hope to determine whether there are differences between the three types of therapy: cognitive-behavioural therapy, schema therapy, and nutrition and appetite focused CBT in the treatment of binge eating problems.

Who can participate in this study? If you are suffering from significant problems with binge eating, are medically well and above a body mass index (BMI, weight (kgs)/height (m)²) of 17.5 (ie, not severely underweight), 16 years or over and not taking psychotropic medications or receiving other treatments for binge eating, you may participate in this study.

What is binge eating? This is when you eat a large amount of food in a short time and feel out of control while doing so. This may or may not be followed by attempts to counteract the effects of the binge eating, including fasting, self-induced vomiting, taking laxatives or other substances to try to get rid of the food or to prevent weight gain, or over-exercising. The binge eating may have been present for months or even years.

How will participants be selected for this study and who will select them? If you meet the above inclusion criteria, you may be referred to the study by your general practitioner, psychiatric emergency
service or other mental health services, or you may contact the Clinical Research Unit yourself if you think you are suffering this kind of problem with binge eating.

**How many participants will be involved?** We hope to study at least 200 women.

**Where will the study be held?** This study will be held in Terrace House, near Christchurch Hospital, Cnr. Antigua Street & Oxford Terrace.

**What is the time span for the study?** Your psychotherapy will occur over one year, approximately weekly for the first 6 months then approximately monthly for 6 months. We would also like to follow your progress after completion of treatment so you will be contacted one, two and three and four years after treatment ends.

**What will happen during the study?** Your participation in this study first involves an assessment by a clinical psychologist, psychiatrist, or psychiatric registrar. At this time the researcher will explain the study in more detail and answer any questions you have. If you feel that you may benefit from our treatment and there is no reason why our treatment would not be appropriate, the researcher will obtain your written consent to participate in the study.

After you have given consent to participate we will organize with you:

a. A time to complete a more detailed structured assessment with your therapist.

b. A neuroendocrine assessment to examine certain of your body’s hormones (including insulin, glucagon, cholecystokinin, leptin and ghrelin) that are potentially related the regulation of appetite and satiety. This assessment will involve coming to the clinical research unit for a morning (from 8.30am), without having eaten breakfast. During this assessment we will take a series of blood samples while you will be resting comfortably with a needle inserted in a vein. After the first blood samples you will be given a glucose drink, to be drunk within a 5 minute period. We are also interested in your report of hunger, fullness, depression and anxiety over this time.

c. A series of tasks to be completed on a computer, which assess abilities such as planning, thinking and memory. This will take approximately two hours and will be done at the clinical research unit.

d. To complete a booklet containing questions about your binge eating symptoms, current relationships, and aspects of your childhood and personality. This takes approximately an hour to complete.

Following the assessment you will be randomly allocated to receive one of the three psychotherapies and begin treatment with your therapist. Randomly allocated means that neither you nor the therapist will choose which psychotherapy you will receive, but that it is chosen “by the flip of a coin”. All therapists in this study are clinical psychologists, senior psychiatric registrars or consultant psychiatrists. You will have six months of weekly psychotherapy sessions, followed by monthly sessions for 6 months. All treatment is free of charge. If at any stage your binge eating is not improving or is getting worse, there is the opportunity to consider other treatments.

You will be asked if you consent to having therapy sessions audiotaped. This is to ensure a high quality of treatment. Some audiotapes may be heard by other members of the research team. You have the option of stopping the taping or having the tapes destroyed at any time. Clinical notes, however, are the property of the CDHB and are subject to their regulations.

You will also be asked to complete a questionnaire before each therapy session so we can record any changes in your symptoms and functioning. These questionnaires take only about 5 to 10 minutes of your time.

There is no obligation for you to take part in this study. If you choose not to participate we will refer you back to your general practitioner or other appropriate health professional.
Will my GP know I am in the study? We prefer to advise your GP that you are involved with this treatment programme, however, this is your decision.

Why the detailed research assessment? The detailed research assessment is for a number of reasons. The questionnaires you complete will help us to assess how clinical factors (e.g. severity or subtype of binge eating, and other problems, such as mood or anxiety) and personality influence how people progress during and after treatment. Similarly, the computer assessment will enable us to examine the association between how people with binge eating perform on tasks measuring such things as memory, planning and attention and the relationship of that performance to progress in treatment.

Some of the blood tests are to check that you are currently physically healthy (e.g. blood count, thyroid tests). We also wish to extract and store DNA (genetic material). Each person has a DNA make-up (their genes) that is different from that of everybody else – except in the case of identical twins. This genetic make-up is a mixture of the genes of the individual’s mother and father. The precise way they are mixed varies from child to child within the same family, so having the same parents does not mean that two children will have exactly the same genes. The research you are invited to participate in will investigate how genes are related to binge eating, to specific eating disorder symptoms and behaviours such as self-induced vomiting, and to personality traits, such as impulsiveness, tendency to worry and perfectionism, and to related factors such as depression and anxiety.

DNA samples will be identified only with a code and as with all other material gathered in this research will be confidential and will not be disclosed or used in any way without your informed consent. In particular the researchers will not claim any right, ownership or property in your individual genetic information or that of your kinship group, hapu or iwi, without you having first sought or obtained informed consent to the transfer of any such right, ownership or property. Your consenting to participate in DNA sampling for the proposed study will not be construed as creating any right or claim on the part of the researchers to your genetic information. Occasionally, when genetic analyses are completed it is necessary to introduce your DNA into a laboratory bacterium (E. coli.)

DNA samples will be retained for 5 years after completion of the study. If you decide to withdraw your consent to the storage of your DNA samples during this storage period, you may do so by contacting the Clinical Research Unit, Ph 372 0400, or by writing to the address at the end of this Information Sheet.

Risks and Benefits

The major benefit for participants is that they will receive free high quality psychotherapy for binge eating over a period of one year. This is a therapeutic study using treatments specifically designed to help individuals with binge eating problems. Without treatment it is possible that binge eating may persist for months or even longer.

There may be some discomfort associated with talking about personal issues in psychotherapy. With the taking of blood, there may occasionally be, as with any blood tests, slight bruising. Participants in this study will not receive any payment or reimbursement of expenses.

If you choose to participate in this study, there may be repercussions from your whanau/family because you have given away genetic information. Whilst the sample given is from you, it will contain information shared by other whanau/family members and they may consider you do not have the right to give that information to others. Te Runanga O Ngai Tahu does not support genetic research.

Participation

• Your participation in this study is entirely voluntary (your choice).
If you agree to take part, you are free to withdraw from this study at any time, for any reason. If you choose not to take part or to withdraw, this will not affect any of your future care or treatment. We will refer you back to your general practitioner or other health professionals as appropriate. You may have a friend, family or whanau support to hear about the study, including the risk and/or benefits and any other explanations you require. While we anticipate that most people participating in this study will be treated as outpatients, if your eating disorder or other problems indicated the need for inpatient care or alternative treatments then these would be organised.

If you have any queries or concerns about your rights as a participant in this study you are free to contact a Health and Disability Services Consumer Advocate, Ph. (03) 377 7501.

Confidentiality

We will take all precautions to maintain confidentiality. All forms and computer files will be marked with numbers only, not names. No material that could personally identify you will be used in any reports based on this study. The data from this study will be available only to the study investigators. All data will be stored in secure areas.

You do not have to answer all questions and you may stop an interview at any time.

Results

How can I obtain results of this research? When this study is over you may have a summary of the key results. Detailed results will be published in international scientific journals.

Compensation

In the unlikely event of a physical injury as a result of your participation in this study, you may be covered by ACC under the Injury Prevention, Rehabilitation and Compensation Act. ACC cover is not automatic and your case will need to be assessed by ACC according to the provisions of the 2002 Injury Prevention Rehabilitation and Compensation Act. If your claim is accepted by ACC, you still might not receive any compensation. This depends on a number of factors such as whether you are an earner or non-earner. ACC usually provides only partial reimbursement of costs and expenses and there may be no lump sum compensation payable. There is no cover for mental injury unless it is a result of physical injury. If you have ACC cover, generally this will affect your right to sue the investigators.

If you have any questions about ACC, contact your nearest ACC office or the investigator.

This study has received ethical approval from the Canterbury Ethics Committee.

Where can I get more information about the study? Andrea Bartram or Sarah Rowe may be contacted by telephone or by letter: ‘Psychotherapy for Binge Eating’, Clinical Research Unit, University Department of Psychological Medicine, Terrace House, 4 Oxford Terrace, Christchurch, Ph. 372 0400.
A comparison of three psychotherapies for binge eating

You are invited to take part in a clinical treatment trial of psychotherapies for people who have major depression, being conducted by Gini McIntosh, Janet Carter, Jenny Jordan, Jan McKenzie, Janet Latner, Martin Kennedy, Chris Frampton, Andrea Bartram, Sarah Rowe, and Peter Joyce.

I have read and I understand the information sheet dated June 2004 for those taking part in this study.

I have been given the opportunity to discuss this study. I am satisfied with the answers I have been given.

I understand that taking part in this study is voluntary (my choice) and that I may withdraw from the study at any time and this will in no way affect my future health care.

I understand that my participation in this study is confidential and that no material that could identify me will be used in any report on this study.

I understand the compensation provisions for this study.

I have had time to consider whether to take part.

I know whom to contact if I have any questions about this study.

I consent to the researchers storing a specimen of my blood for its later use as part of this study. YES / NO

I consent to blood samples being destroyed at the end of the study. ........................................YES / NO

I consent to my therapy sessions being audio-taped.................................................................YES / NO

I wish to receive a copy of the results of this study. ...............................................................YES / NO

I understand there will be a significant delay between the information I provide and receiving the results.

I agree to my GP being informed of my participation in this study........................................YES / NO

GP’s name________________________________ Address___________________________________________

I would be willing to be contacted to discuss participation in future research in this area.........YES / NO

I am aware that the proposed study will involve analysis of my genetic makeup. I consent to such an analysis being performed.................................................................YES / NO

I understand that if I consent to such analysis, no rights will be created for the researcher to my genetic information.................................................................YES / NO

I am aware that the proposed study may involve storage of my genetic makeup and give my consent.................................................................YES / NO

I ________________________________ (print full name) hereby consent to take part in this study.

Date: ____________ Phone number: ________________

Signature: _______________________ Signature of witness: ______________________

Project explained by: ___________________________ Role: ______________________________

Contact phone numbers: Andrea Bartram or Gini McIntosh (03) 372 0400

A comparison of three psychotherapies for binge eating