Set Shifting Impairments in an Outpatient

Eating Disorder Sample

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Dedication

This thesis is dedicated to my Granny, Lily Howarth.

Acknowledgements

Declaration

I confirm that all this work is my own except where indicated otherwise.

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Helen M. Swanson

Abstract

Background: Patients with anorexia nervosa have been consistently reported to show impairments in set shifting ability. Such deficits may be associated with characteristics commonly observed in this patient group, such as obsessive thoughts and behaviours around eating, maladaptive problem solving and a rigid thinking style. **Objective:** Much of the preceding literature on set shifting ability has involved inpatient samples meeting strict diagnostic criteria for anorexia nervosa. However most eating disorder patients are outpatients and commonly do not meet full criteria for anorexia nervosa. This study thus aimed to investigate the relationship between set shifting ability and psychological characteristics in a community sample of outpatients with symptoms of anorexia nervosa. **Methods:** Performance on selected measures of set-shifting ability (Wisconsin Card Sort Test, WCST; Delis-Kaplan Executive Function System, Hayling & Brixton) were compared between an eating disorders group comprising 17 female outpatients with symptoms of anorexia nervosa and a control group comprising 27 students. Set shifting performance was then correlated with eating disorder severity (Eating Disorders Examination), obsessive-compulsive symptoms (Yale-Brown Obsessive Compulsive Scale), and the Social Problem Solving Inventory. **Results:** The eating disorder group demonstrated significantly worse set shifting ability than the healthy control group on the primary outcome measure (WCST), with 47% of eating disorder participants showing impairment on this measure. Severity of obsessive-compulsive symptoms and an impulsive and careless approach to problem solving were associated with poorer scores on the WCST in the eating disorder group. Although the eating disorder group were significantly more impaired in set shifting than

controls, set shifting ability was not associated with eating disorder severity.

Conclusions: The results indicate that set shifting impairments are present in outpatients with eating disorders with anorexic symptoms, and may be trait characteristics. Impaired set shifting was associated with obsessive-compulsive symptoms and maladaptive problem solving. These findings highlight a need for neuropsychological assessment of eating disorder outpatients in order to identify individuals who may benefit from psychological interventions to reduce the impact of these impairments.

Chapter 1: Introduction

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1.1 Introduction

Patients with anorexia nervosa have been consistently reported to show impairments in the executive function of set shifting. Set shifting involves the ability to switch attention, allowing the individual to change their thinking or behaviour to adapt to the demands of the environment or situation they are in. It is thought that deficits in set shifting ability may be associated with characteristics commonly observed in this patient group such as obsessive thoughts and behaviours around eating, maladaptive problem solving and a rigid thinking style. Much of the preceding literature has reported on inpatient samples meeting strict diagnostic criteria for anorexia nervosa. Set shifting ability has not been widely investigated in outpatients. This study is the first to investigate the relationship between neuropsychological test performance on measures of set shifting ability and psychological characteristics in a community sample of outpatients presenting to specialist eating disorder services with symptoms of anorexia nervosa. The findings of this study have the potential to enhance our understanding of risk and maintenance factors in eating disorders, help inform new interventions and future service development.

As an introduction to the research, Section 1.2 provides background information on anorexia nervosa, outlining the definition of the disorder, prevalence and incidence rates, aetiology including evidence to suggest neuropsychological dysfunction, current treatments and outcome. Section 1.3 provides a brief overview of neuropsychological research in patients with anorexia nervosa demonstrating that the disorder is not thought to be associated with a widespread pattern of cognitive

impairment. Section 1.4 reviews the literature on set shifting ability, the most consistently reported neuropsychological impairment in patients with anorexia nervosa. Section 1.5 reviews the literature on social problem solving in anorexia nervosa, outlining how problem solving and neuropsychological status may be related. Section 1.6 reviews the literature on neuropsychological functioning in obsessive-compulsive disorder and highlights parallels with anorexia nervosa. Section 1.7 then details the aims and hypotheses of the current study.

1.1.1 Search Strategy

The literature reviewed was obtained by searching the Medline, PsychInfo and EMBASE databases for articles on eating disorders published since 1987. The keywords used were variants of anorexia nervosa, eating disorders, eating disorders not otherwise specified, neuropsychology, executive function, set shifting, flexibility, obsessive-compulsive disorder, and social problem solving. The truncation function was used to increase the number of articles returned. Only articles written in English and translated abstracts were reviewed. Additional searches were undertaken using Google Scholar. Key references which did not come up using the above search criteria were identified from the literature. Professional books about eating disorders and neuropsychology written in English were also reviewed.

1.2 Anorexia Nervosa

1.2.1 Definitions and Subtypes of Anorexia Nervosa

The classification of eating disorders is a controversial issue. Current diagnostic systems subdivide eating disorders into three distinct categories: anorexia nervosa

(AN), bulimia nervosa (BN) and eating disorders not otherwise specified (EDNOS). Anorexia nervosa is a condition characterised by the maintenance of excessive low weight, bulimia nervosa is characterised by binge eating and compensatory purging behaviours and EDNOS has no distinct clinical characteristics and is largely a diagnosis of exclusion.

The Diagnostic and Statistical Manual of Mental Disorders (DSM-IV, American Psychiatric Association, 1994) and the International Classification of Diseases (ICD-10, World Health Organisation, 1993) define anorexia nervosa as a disorder where the individual maintains a body weight of at least 15% below their expected weight. This is usually indicated by a body mass index (BMI) of ≤ 17.5. Other criteria for a diagnosis within DSM-IV include an intense fear of weight gain or becoming fat; disturbance in the way one's body shape and weight are perceived, placing undue emphasis on body shape and weight as aspects of self evaluation; and amenorrhea. ICD-10 criteria specify that patients' weight loss must be self-induced by the avoidance of fattening foods and the use of one or more of the following strategies: self induced vomiting, excessive exercise, misuse of appetite suppressants and/or diuretics. ICD-10 explicitly specifies presence of widespread endocrine disorder involving the hypothalamic-pituitary-gonadal axis, which in women is expressed in amenorrhoea and in men results in reduced libido. Endocrine disorder may also include elevated levels of cortisol, growth hormone and abnormalities in insulin regulation.

DSM-IV indicates two specific subtypes of anorexia: restricting and binge-eating/purging. Individuals with restricting type anorexia do not regularly engage in binge eating or purging behaviours and maintain a low weight by restricting their calorie intake and exercising excessively (Grave, Calugi & Marchesini, 2008). The binge-eating/purging subtype regularly engage in binge-eating or purging behaviours such as self induced vomiting, and misuse of laxatives or diuretics. A number of studies have investigated the clinical and psychological characteristics of patients on the basis of diagnostic subtype, and have consistently provided support for the sub-classification of anorexia nervosa based on the presence or absence of purging behaviours (Bollen & Wojciechowski, 2004; Garner, Garner & Rosen, 1993; Gleaves & Eberenz, 1993; Herzog et al., 1996).

The "not otherwise specified" category of the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV, American Psychiatric Association, 1994) is designed as a category for disorders of clinical significance that do not meet specific criteria and are thus atypical in their presentation. In the field of eating disorders the category is not atypical with up to 60% of patients presenting at eating disorder speciality clinics meeting diagnostic criteria for EDNOS rather than criteria for other defined eating disorders (Fairburn et al., 2007). In non-speciality settings the prevalence of EDNOS is even higher. One community based outpatient psychiatric clinic reported that 90% of eating disorder patients had a diagnosis of EDNOS (Zimmerman, Francione-Witt, Chelminski, Young & Tortolani, 2008); while another community study reported 75% prevalence (Machando, Machando, Gonclaves & Hoek, 2007).

Recently a core of leading researchers within the field have proposed a "transdiagnostic" theory of categorisation, based on the principle that all eating disorders share distinctive clinical features and are maintained by similar psychopathological processes (Fairburn, Cooper & Shafran, 2003). Support for this model was provided by a recent study that followed up 192 females with eating disorders at three time points over a 30-month period (Milos, Spinalder, Schnyder & Fairburn, 2005). Results revealed that while eating disorder status remained stable, there was low stability between the diagnoses with only one third retaining their original diagnosis. However a recent meta-analysis of 125 studies has demonstrated that significant differences exist between the diagnostic categories of bulimia and EDNOS, suggesting the application of a transdiagnostic approach could potentially eliminate genuine diagnostic distinctions (Thomas, Vartanian & Brownell, 2009).

Traditionally EDNOS was thought to represent 'sub-clinical' or mild eating disorders, however, severity of psychopathology and degree of functional impairment in EDNOS have been shown to be comparable to anorexia and bulimia nervosa (Ricca et al., 2001; Turner & Bryant-Waugh, 2004). It has been proposed that two subgroups exist within EDNOS (Fairburn & Walsh, 2002; Mitchell, Pyle, Hatsukami & Eckert, 1986); the first is where patients closely resemble either AN or BN but do not fully meet diagnostic criteria and the other is where the presentation is 'mixed' and may include features of AN, BN, binge eating disorder or other eating pathology. The 'mixed' presentation is significantly more prevalent within the EDNOS category (Fairburn & Bohn, 2005; Fairburn et al., 2007).

On the basis that a subgroup of patients with EDNOS may closely resemble patients with AN, a number of suggestions have been made within the literature for relaxing the diagnostic criteria for AN. These include removing amenorrhea (Bulik, Sullivan & Kendler, 2000; Watson & Andersen, 2003) and increasing body weight to over 85% of healthy weight (McInosh et al., 2004; Watson & Andersen, 2003). Neither the presence of amenorrhea nor body weight within a 'low weight' range have been shown to reliably distinguish clinical samples on the basis of demographics, illness history, psychopathology, or response to treatment (McIntosh et al., 2005; Watson & Andersen, 2003). Other authors have suggested that weight phobia may not be a necessary inclusion criteria, based on the premise that this only emerged as a motive for food refusal in the 1930's (Hsu & Lee, 1993).

Thomas et al., (2009) conducted a meta analysis investigating the relationship between EDNOS and officially recognised eating disorders. In order to conduct their analysis the authors classified AN type EDNOS into 4 subgroups, as detailed in Table 1.1

Table 1.1: Anorexia Nervosa type EDNOS subgroups (adapted from Thomas et al., 2009).

EDNOS subgroup	Description of clinical features
AN with menses	Meets all diagnostic criteria for AN except has menses.
High weight AN	Meets all diagnostic criteria for AN except weighs >85% of expected body weight.
Non-fat phobic AN	Meets all diagnostic criteria for AN except does not endorse intense fear of gaining weight or becoming fat.
Partial AN	Presents with features of AN but missing at least 2 of the 4 diagnostic criteria.

In relation to anorexia nervosa, the meta-analysis concluded that patients meeting strict diagnostic criteria for anorexia nervosa did not differ in terms of eating pathology from AN groups with menses, high weight AN or partial AN. The only difference observed was that patients with non-fat phobic AN displayed less severe eating pathology than patients meeting strict diagnostic criteria for AN. The authors concluded that the largely non-significant differences between AN and AN type EDNOS suggests that AN represents the severe end of a spectrum of abnormal eating. The authors further suggest it may be possible to drop the amenorrhea criteria and increase the weight criteria without influencing the homogeneity of the AN diagnosis.

Given the similarity between AN and AN type EDNOS, and the fact that there is very little literature on AN type EDNOS, the review of the literature that follows focuses primarily on anorexia nervosa.

1.2.2 Prevalence and Incidence

Point prevalence of anorexia nervosa is estimated to be 0.3% in young women (Hoek, 2006), lifetime prevalence amongst women is estimated to be 0.9% (Hudson, Hiripi, Pope & Kessler, 2007). In the UK in 2000, the primary care incidence rate of anorexia nervosa adjusted for age and gender was 4.7 per 100,000 population. The incidence rate showed huge variation on the grounds of gender and age, with the disorder being most common in females aged 10-19 years. The overall incidence rate for females was 8.6 per 100,000, and for males was 0.7 per 100,000. This translates to a relative risk for females to males of 12:1 (Currin, Schmidt, Treasure, & Jick, 2005). In Scotland, approximately 1200 women aged 15-24 suffer from the disorder (Carter & Miller, 2004).

It is estimated that males account for 5-10% of reported cases of anorexia nervosa (Alexander-Mott, 1994), however the disorder is likely to be underreported and misdiagnosed in this population. The literature on male anorexia nervosa is extremely limited, but suggests that the disorder is very similar to female anorexia nervosa in terms of clinical features and presentation (Carta, Zappa, Carugati, De Coppi & Garghentini, 2006); however there are unique differences in predisposition, course and onset (Andersen, 1992; Crosscope-Happel, Hutchins, Getz & Hayes, 2000).

1.2.3 Aetiology

A number of theories have been proposed regarding the development and maintenance of anorexia nervosa, briefly these include psychodynamic (Dare &

Crowther, 1995), cognitive-behavioural (Fairburn et al., 2003), physiological (Robinson & McHugh, 1995), neurological (Braun & Chouinard, 1992), neuropsychological (e.g. Roberts, Tchanturia, Stahl, Southgate & Treasure, 2007a; Southgate, Tchanturia & Treasure, 2008; Tchanturia, Cambell, Morris & Treasure, 2005), neurodevelopmental (Connan, Campbell, Katzman, Lightman & Treasure, 2003), feminist (Orbach, 1985; Palmer, 1982) and family systems theory (Colahan & Senior, 1995). Despite a multitude of theories the cause of anorexia nervosa is unknown. Discussion of all of these theoretical models is outwith the scope of this study, therefore the focus in this section will be on neurological and neurodevelopmental theories, which are relevant to the study hypotheses. Neuropsychological literature will be discussed in Sections 1.3 and 1.4.

1.2.3.1 Evidence for a Neurological Component

Biopsychosocial models have provided evidence for central nervous system dysfunction in eating disorders (Braun & Chouinard, 1992); and heritability factors for anorexia nervosa are thought to be related to genes involved in brain development, energy homeostasis and serotonin pathways (Klump, Bulik, Kaye, & Strober, 2002). The largest population based twin study conducted to date indicated a substantial contribution of genetic factors to liability for developing anorexia as defined using both broad and narrow diagnostic criteria (Bulik, et al., 2006), with current heritability estimates ranging from 56-70% (Bulik et al., 2006; Gorwood, Kipman & Foulon, 2003; Lilenfeld, et al., 1998).

Neurological imaging studies in the field of anorexia report conflicting findings due to methodological differences across studies, for example in state of starvation, co-morbidity, psychotropic medication, age of onset, heterogeneity of eating disorder samples and lack of suitable control groups (Key, O'Brien, Gordon, Christie & Lask, 2006). However consistent structural neural abnormalities have been found in patients with anorexia in the form of decreased brain volume (Katzman et al., 1996, Katzman, Christensen, Young & Zipursky, 2001; Swayze et al., 2003), lateral ventricular enlargement, dilated sulci (Dolan, Mitchell, & Wakeling, 1988; Kingston, Szmukler, Andrews, Tress, & Desmond, 1996; Palazidou, Robinson & Lishman, 1990; Swayze et al., 2003) subcortical changes in the thalamus and basal ganglia (Uher et al., 2002), reduced hippocampus (Connan et al., 2006) and pituitary gland size (Kingston et al., 1996).

Structural abnormalities are largely resolved with re-feeding, although persistent grey matter volume decrease has been reported in recovered patients with anorexia nervosa from 1-23 years post recovery (Lambe, Katzman, Mikulis, Kennedy & Zipursky, 1997).

Physiological abnormalities observed in anorexia nervosa include reduced cerebral blood flow and metabolism (Delvenne et al., 1995), altered neurotransmitter levels (Kaye, Gendall & Kye, 1998) and disturbance in event related potentials at the synaptic level (Bradley et al., 1997). Functional imaging studies of adolescents with early onset anorexia (onset before age 15) have demonstrated unilateral decreased blood flow in the temporal lobe and associated areas in 75% of early onset patients

(Chowdhury et al., 2003; Lask et al., 2005). These abnormalities have been demonstrated to remain following weight gain (Råstam et al., 2001).

Persistent abnormalities in the serotonin system have been reported in patients with anorexia and those who are fully recovered (Frank et al., 2001; Katzman, Zipursky, Lambe, & Mikulis, 1997; Kaye, Klump, Frank & Strober, 2000; Råstam et al., 2001). The serotonin system has been implicated in behavioural regulation, impulsivity and cognitive flexibility (Clarke, Dalley, Crofts, Robbins & Roberts, 2004; Winstanley, Theobald, Dalley, Glennon & Robbins, 2004). These studies appear to provide supporting evidence for the hypothesis that serotonin dysfunction contributes to anorexic psychopathology. However, some authors have suggested that the evidence to support a dysfunctional serotonin system as a factor in anorexia is weak (Mondelli, 2006), and treatment with Selective Serotonin Reuptake Inhibitors (SSRI's) has not been shown to be particularly effective (Treasure & Schmidt, 2008).

1.2.3.2 Evidence for a Neurodevelopmental Component

Peri-natal factors associated with increased risk for anorexia include premature delivery, cephalhematoma and low birth weight for gestational age (Cnattingius, Hultman, Dahl & Sparén, 1999; Lindberg & Hjern, 2003). Obstetric complications such as maternal anaemia, diabetes, pre-eclampsia, placental infarction, neonatal cardiac problems and hyporeactivity have been associated with the development of anorexia nervosa; and the risk for developing anorexia has been found to increase with the total number of obstetric complications (Favaro, Tenconi & Santonastaso, 2006).

Family factors play a key role in healthy development. Preterm and low birth weight children are at increased biological risk of interrupted development, including difficulties coping and adapting to their environment, which can negatively influence relationships with caregivers (Bennet, 1987). Studies of attachment have reported that patients with eating disorders develop anxious-ambivalent and avoidant attachments in adolescence and adulthood (O'Kearney, 1996; Rhodes & Kroger, 1992; Ward, Ramsay, Turnbull, Benedettini & Treasure, 2000). Poor attachment is demonstrated by low levels of parental care, and high levels of control (Parker, Tupling, & Brown, 1989). Lack of perceived control is seen as a major factor in some eating disorder formulations (e.g. Bruch, 1973; Slade, 1982); and high maternal over control has been associated with anorexia nervosa (Swanson et al., In Press; Walters & Kendler, 1995).

Connan et al. (2003) propose a neurodevelopmental model for anorexia nervosa which brings together genetic, perinatal and developmental factors. They suggest genetic and early attachment experiences have a critical role in the development of neurobiological systems including the hypothalamic-pituitary-adrenal (HPA) axis. The HPA axis modulates stress response. Dysregulation of the HPA axis is associated with failure to adapt to chronic stressors. Psychosocial and pubertal changes during the transition through adolescence increase vulnerability such that when stress is encountered the HPA axis response and behavioural coping response is maladaptive. Disruption of the HPA axis is thought to cause loss of appetite and weight leading to anorexia. Once initiated, they propose that anorexia is maintained by underlying biological and psychological systems.

1.2.3.3 Summary

The studies presented above provide evidence for neurological dysfunction in anorexia nervosa. Consistent structural neural abnormalities have been reported in patients with anorexia. Physiological abnormalities observed in anorexia nervosa include reduced cerebral blood flow and metabolism, altered neurotransmitter levels and disturbance in event related potentials at the synaptic level. Neurodevelopmental theories bring together genetic, peri-natal and developmental factors, and suggest genetic and early attachment experiences have a critical role in the development of neurobiological systems.

1.2.4 Co-Morbidity

Anorexia nervosa is associated with high levels of medical and psychiatric co-morbidity (Su & Birmingham, 2003). Data from a national co-morbidity survey replication in the USA (Hudson et al., 2007) indicated that 56% of patients with anorexia nervosa met diagnostic criteria for at least one psychiatric disorder as assessed by DSM IV. Anxiety (47.9%), mood (42.1%), impulse control (30.8%) and substance use (27.0%) were the most prevalent. Depression and Obsessive-Compulsive Disorder (OCD) are commonly noted to present alongside anorexia (Wentz, Gillberg, Gillberg & Råstam, 2001). Due to its high prevalence, some authors have suggested depression should be considered part of the psychopathology of anorexia rather than as a discrete co-morbid condition (Herzog, 1984; Hudson, Pope, Jonas & Yurgelen-Todd, 1983; Piran, Kennedy, Garfinkel & Owens, 1985).

It is unclear whether depression and other affective disorders precede the diagnosis of anorexia or are a consequence of starvation (Råstam, Gillberg & Gillberg, 1996), however OCD appears to precede anorexia nervosa in the majority of cases where co-morbidity is observed (Wentz et al., 2001). Indeed obsessive-compulsive behaviours in anorexia have been shown to remain relatively stable over time and to be unrelated to weight problems (Råstam et al., 1996). Many patients with anorexia may not meet full criteria for OCD, however, as discussed in Section 1.6, there is evidence that the two disorders share psychological and neuropsychological features.

1.2.5 Current Treatments

The Scottish Executive in its "Delivering for Mental Health" report (2006) recommends effective use of evidence to produce better mental health outcomes. The evidence base for treatment of eating disorders varies by condition, and for anorexia nervosa there is very little research. Le Grange & Lock (2005) state there is a "dearth" of psychological treatment outcome studies, largely due to difficulties with recruitment, ethical implications of randomising patients to treatment, and high drop out rates. This is highlighted in the NICE Guidelines for Eating Disorders (2004), in which all recommendations are made at the lowest evidence based grading.

Anorexia nervosa is difficult and expensive to treat, patients are commonly in denial of their symptoms, chronically symptomatic, frequently relapse and are ambivalent about engaging in treatment (Guarda, 2008, Pike 1998). Drop out from inpatient treatment ranges from 30-50% (Kahn & Pike, 2001; Vandereycken & Pierloot, 1983; Woodside & Stabb, 2006; Zeeck, Hartman, Buchholz & Herzog, 2005). Current

treatments include inpatient admission, individual psychotherapy, family therapy and pharmacotherapy.

A range of psychotherapies have been shown to have some effectiveness in the treatment of anorexia nervosa, including Cognitive Behavioural Therapy (Gowers & Bryant-Waugh, 2004; Pike, Walsh, Vitousek, Wilson & Bauer, 2003), Cognitive Analytical Therapy (Dare, Eisler, Russell, Treasure, Dodge, 2001), Psychodynamic Psychotherapy (Fairburn & Harrison, 2003), Cognitive Remediation Therapy (Davies & Tchanturia, 2005; Tchanturia, Davies & Campbell, 2007), Focal Analytic Therapy (Dare et al., 2001), and Family Therapy (Dare et al., 2001). Most research comparing psychotherapies has found no difference in treatment effectiveness, although family therapy has been shown to have moderate efficacy in the adolescent population (for a review see Treasure & Schmidt, 2008).

The role of pharmacological treatments for anorexia nervosa is unclear as anti-anxiety drugs and tricyclic antidepressants are likely to be ineffective or harmful, and Cyproheptadine, Oestrogen and SSRI's have unknown effectiveness (Treasure & Schmidt, 2008). The lack of evidence based treatments for anorexia nervosa highlights the need for research that can enhance our understanding of this disorder and inform the development of more efficacious interventions.

1.2.6 Outcome

Reported recovery rates vary and are generally low over the short term. A prospective, naturalistic, longitudinal study carried out over 7.5 years, mapped the

course of anorexia and bulimia nervosa in 246 women. At median 90 month follow up, 33% of patients with anorexia nervosa had fully recovered, while 83% achieved at least partial recovery. Approximately a third of patients were found to relapse following full recovery, and a diagnosis of anorexia nervosa was the single best predictor of poor outcome (Herzog et al., 1999). A large outcome study reviewing 119 studies of anorexia nervosa suggested that following intervention, approximately half of surviving patients were considered to be "recovered", a third "improved" and 20% remained "chronically ill" (Steinhausen, 2002). Of all psychiatric conditions, anorexia has the highest rate of premature mortality; with up to 22% mortality in some long-term studies (Steinhausen, 2002).

The course of recovery is long. A discrete time survival analysis of 69 inpatients with anorexia nervosa over a 12-year period, revealed that patients did not show significant improvements until six years after the first inpatient treatment (Herzog, Schellberg & Deter, 1997).

1.2.7 Summary

Anorexia nervosa is a persistent disorder in which morbidity and mortality are high. It is significantly more prevalent in women than men (Alexander-Mott, 1994; Currin et al., 2005) and commonly develops in adolescence. It is associated with high levels of medical and psychiatric co-morbidity and significant functional impairment (Su & Birmingham, 2003). The cause of anorexia nervosa is unknown, however research suggests that it is likely to be multi-componental. There is increasing evidence of a neurological component including genetic vulnerability, central nervous system

changes including structural and physiological brain changes, influence of peri-natal factors, and developmental considerations.

The disorder is difficult and expensive to treat, patients commonly lack insight and are ambivalent about engaging in treatment, consequently drop out and relapse rates are high (Guarda, 2008). Current treatments include medical management, psychotherapy and pharmacotherapy, however there is limited evidence for efficacious treatments and outcome is generally poor (Le Grange & Lock, 2005; Steinhausen, 2002). Research with the potential to enhance our understanding of the disorder and contribute to efficacious interventions is therefore crucial.

1.3 Neuropsychological Functioning in Anorexia Nervosa

As described in Section 1.2.3 above, there is increasing evidence for a neurological and neurodevelopmental component in anorexia nervosa. The following section will discuss evidence for neuropsychological dysfunction in this disorder.

Neuropsychology is defined as the study of the relationship between brain function and behaviour (Kolb & Wishaw, 1989). Neuropsychological assessment was originally developed to examine the effects of brain lesions or trauma, however over recent decades has been increasingly applied in the study of psychiatric disorders. Whilst investigation of neuropsychological functioning in anorexia is a relatively new field; it is widely recognised as having the potential to advance our understanding of eating disorders at a clinical and theoretical level (Keefe, 1995). A recent literature review identified 52 published studies investigating

neuropsychological function in patients with eating disorders (Southgate, Tchanturia & Treasure, 2006). Many aspects of cognitive functioning have been investigated, including intelligence, memory, learning, visuo-spatial abilities, speed of information processing, motor speed, and a range of executive functions.

The literature suggests that eating disorders are not associated with a widespread pattern of cognitive deficits. The most consistently reported deficit associated with anorexia is in the executive function of set shifting (Roberts et al., 2007a). The following sections briefly outline the literature on intelligence, memory, learning, visuo-spatial abilities and speed of information processing/motor speed in anorexia nervosa. Section 1.4 will then discuss set shifting ability in more detail.

1.3.1. Intelligence

Early neuropsychological research investigated Intelligence Quotient (IQ) in patients with anorexia nervosa. Such individuals are characterised as being high achievers academically and initial hypotheses predicted that people with anorexia would have higher IQ's than the general population. Eating disorder samples have been shown to have higher IQ scores than other psychiatric groups (Blanz, Detzner, Lay, Rose & Schmidt, 1997) however there is no evidence that they are of higher intelligence than the general population (Gillberg, Gillberg, Råstam & Johanasson, 1996). It is thought high academic achievement is likely to be a reflection of increased effort associated with clinical perfectionism, a trait commonly observed in patients with anorexia (Shafran, Cooper & Fairburn, 2002). Indeed, there is evidence that people with anorexia perform better than controls on tasks requiring substantial cognitive effort

but often do badly on tasks that require automatic processing (Kingston et al., 1996; Fassino et al., 2002; Struup, Weingartner, Kaye & Gwirtsmann, 1986).

1.3.2 Memory and Learning

Overall the literature suggests that anorexia nervosa is not associated with significant impairments in memory and learning. To date, deficits have been reported in both verbal and visual memory and in learning ability in patients with anorexia nervosa (Southgate et al., 2006). The literature is somewhat inconsistent, with a small number of studies reporting significant differences between patients with anorexia and controls in tests of verbal memory (Mathias & Kent, 1998; Kingston et al., 1996); paired associate learning (Witt, Ryan & Hsu, 1985), and conditional associative learning (Murphy, Nutzinger, Paul & Leplow, 2002, 2004; Witt et al., 1985). Other studies have however reported no significant differences between patients with anorexia and non-clinical control groups on tasks of immediate and delayed list learning (Gillberg, Råstam, Wentz & Gillberg, 2007; Szmukler et al., 1992; Kingston et al., 1996); and conditional associative learning tasks (Seed, Dixon, McCluskey, & Young, 2000). There is some evidence that these discrepancies in the literature may be explained by inconsistencies in study methodology, for example, the diagnostic criteria used across studies is variable with some using DSM-III and others using DSM-III-R or DSM-IV, which includes the separation of patients into diagnostic subtypes. Of the above studies Mathias & Kent, (1998) and Kingston et al. (1996) employed sound methodology including a relatively large sample size, and detailed reporting of participant characteristics; whereas Witt et al. (1985), employed a small

sample, used older diagnostic criteria for anorexia and did not report participant characteristics in detail.

1.3.3 Visuo-spatial Functioning

There is no significant evidence to suggest that impairments in visuo-spatial functioning exist in anorexia nervosa, though some studies have reported poor performance on tasks of copying a complex figure, constructional ability, haptic tests and other visuo-spatial tasks (Grunwald et al., 2001; Kingston et al., 1996; Mathias & Kent, 1998; Palazidou et al., 1990; Pendleton-Jones, Duncan, Brouwers & Mirsky, 1991; Thompson, 1993).

Southgate et al. (2006) suggest that a cognitive style associated with weak central coherence may account for both impaired and optimal performance on tests of visuo-spatial ability depending on the demands of the task. For example, the ability to observe the individual details of a pattern would be advantageous on a task such as Block Design (WAIS-III, Wechsler, 1997), but would impair performance on Object Assembly (WAIS-III, Wechsler, 1997), or a copy of the Rey-Osterrieth Complex Figure (Osterrieth, 1944), in which the whole design needs to be observed simultaneously.

1.3.4 Speed of Processing

Speed of information processing has been found to be slowed in anorexia nervosa, particularly amongst individuals with lower weight (Hamsher, Halmi & Benton, 1981). Slowed speed of information processing is characteristic of low mood and

Depression (Christensen, Griffiths, Mackinnon, & Jacomb, 1997) which is commonly co-morbid with anorexia, and may therefore have influenced patients' performance on these tests. There is some evidence that patients with anorexia nervosa show psychomotor slowing (Green, Elliman, Wakeling, & Rogers, 1996; Kingston et al., 1996; Szmukler et al., 1992) which if present could influence performance on neuropsychological tests that include a timed component.

1.3.5. Summary

Neuropsychology is defined as the study of the relationship between brain function and behaviour and has the potential to advance our understanding of psychiatric illness. Many aspects of cognitive functioning have been investigated in patients with anorexia nervosa. The literature has been confounded by inconsistencies in study methodology, but there is a general consensus that anorexia nervosa is not associated with a widespread pattern of cognitive impairment. However patients with anorexia nervosa have been consistently reported to have deficits in the executive function of set shifting ability (Roberts et al., 2007a), which will be discussed in Section 1.4.

1.4 Set Shifting in Anorexia Nervosa

1.4.1. Definition of Set Shifting

Set shifting is an integral component of cognitive and behavioural flexibility. It involves the ability to adapt behaviour or thoughts in line with changing demands of the situation or environment, by switching attention between different topics. Set shifting is one of the executive functions. Executive functions include a range of skills which help organise and execute brain activity, including attention, problem

solving, sequencing, decision making, planning, goal setting, organisation, initiating and inhibiting responses, regulation and monitoring of behaviour, and set shifting (Lezak, Howieson & Loring, 2004).

The term "executive function" defines complex cognitive processing requiring the coordination of several sub-processes to achieve a specific goal (Elliott, 2003). Baddeley & Hitch (1974) proposed a model of working memory to describe the interface between short and long term memory and the coordination of cognitive processes. Part of this model was named the central executive, which was conceptualised as being responsible for directing attention, and selecting and manipulating information from long-term memory. Norman & Shallice's (1986) supervisory attention system (SAS) model, explains well the role of the central executive. It is proposed that the executive controls attentional resources to bring unconscious automatic behaviours and cognitions into conscious awareness. This direction of attentional resources is referred to as "executive" because it is thought to operate in a supervisory or executive capacity to unify a range of cognitive functions and to action sequences of behaviour, cognition and emotion (Burgess, 2003).

Successful set shifting involves the coordination of several cognitive functions including attention, working memory, initiation and inhibition of responses, and regulation and monitoring of behaviour. Failure to effectively shift set results in perseverative responses that are inappropriate to the changing demands of the situation.

1.4.2 Neurology of Set Shifting and Evidence for Impairment

The frontal lobes are known to play a key role in executive functioning and therefore the ability to shift set (Shallice, 1982; 1988). In evolutionary terms they are the most recently developed part of the brain (Lezak et al., 2004), and their size in relation to the rest of the cortex is unique in humans, who have much larger frontal lobes than other animals (Malloy, Cohen, Jenkins & Paul, 2005). The frontal lobes are thus thought to be involved in many of the processes that make us human, and have been identified as the prime location of general intelligence or Spearman's g because of their role in so many diverse cognitive functions (Duncan & Owen, 2000). Neurodevelopment of the frontal lobes occurs during adolescence and they are typically fully functional by adulthood (Malloy et al., 2005). Anorexia nervosa commonly develops in adolescence, raising the possibility of frontal lobe involvement in this disorder.

The frontal lobes have profuse connections with most other areas of the brain, and neurotransmitter systems converge in the prefrontal cortex, including dopamine. Dopamine plays a vital role in mediating interactions at a synaptic level, in many areas of the cortex and in sub-cortical structures (Fuster, 2003). The frontal cortex is closely connected to posterior structures specifically the thalamic nuclei and basal ganglia with which they form complex interconnected systems. These frontal lobe systems are thought to be important for regulating a stream of conscious thought and behaviour (Malloy et al., 2005).

Collectively, medial and orbital areas are referred to as the ventral prefrontal cortex and are thought to have a key role in regulation and maintenance of set and ongoing behaviour (Malloy, Bihrle, Duffy & Cimino, 1993; Stuss et al., 1983). The dorsolateral prefrontal cortex has been identified as being associated with other cognitive functions (Stuss & Levine, 2002). Some cases have documented the development of an anorexic type syndrome, including characteristic psychopathology following discrete acquired brain lesions in the right prefrontal cortex (Uher & Treasure, 2005). Other brain regions thought to be involved in anorexia nervosa include the prefrontal cortex (Delvenne et al., 1996, Delvenne, Goldman, de Maertalaer, & Lotstra, 1999), dorsolateral prefrontal cortex, anterior cingulate (Ohrmann et al., 2004) and caudate nucleus (Delvenne et al., 1996, 1999). The caudate nucleus has been implicated in explaining obsessionality observed in both anorexia nervosa and OCD (Thompson, 1993).

1.4.3 Impairments in Set Shifting Ability

A meta-analysis of set shifting impairment in patients with anorexia nervosa identified 15 published studies, with a minimum sample size of 30 and including both control and anorexia nervosa samples (Roberts et al., 2007a). A consistent deficit in set shifting ability was found across most measures, with analyses indicating wide variation in effect size depending on the neuropsychological measure employed. Estimated effect size ranged from 0.36 (for the Trail Making Test; Reitan, 1958), to 0.62 (for the Wisconsin Card Sort Test, WCST; Heaton, Chelune, Talley, Kay & Curtis, 1993) to 1.05 (for Haptic illusion; Uznadze, 1966; Tchanturia, Serpell, Troop & Treasure, 2001).

There is a growing evidence base to suggest that set shifting difficulties identified in anorexia nervosa may be trait rather than state characteristics (Holliday, Tchanturia, Landau, Collier & Treasure, 2005; Tchanturia et al., 2004a). A number of studies have investigated the effects of refeeding/ weight gain on executive impairments, in which set shifting has been consistently shown not to improve with weight gain (Green et al., 1996; Tchanturia et al., 2004c). Recent research has suggested that set shifting difficulties may persist after recovery from anorexia nervosa and are therefore not simply a function of acute illness state (Tchanturia, Morris, Surguladze & Treasure, 2002; Holliday et al., 2005). However this has mainly been investigated in inpatients with severe anorexia, and has not been studied in an outpatient population.

Theoretical papers suggest set shifting may predate the onset of eating disorders (Lena, Fiocco & Leyenaar, 2004) and be a predisposing factor for anorexia (Southgate, Tchanturia, & Treasure, 2005; Steinglass & Walsh, 2006; Tchanturia et al., 2005). Poor performance on set shifting tasks has been associated with childhood rigidity and inflexibility (Tchanturia et al., 2004c), and a number of authors have argued that premorbid cognitive deficits affect the development of self esteem, identity formation, social functioning, problem solving and development of autonomy in adolescence which may increase the risk of developing an eating disorder (Fox & Mahoney, 1998; Lena et al., 2004; Peck, 1981; Roman, 1998).

Neuropsychological testing of patients with anorexia and their unaffected sisters has demonstrated the presence of set shifting impairment and reduced mental flexibility

in unaffected sisters at a higher proportion than in the general population, suggesting set shifting difficulty may be a biological marker or endophenotype of anorexia nervosa (Holliday et al., 2005). However given that the scores on tasks of set shifting were very similar between affected and unaffected sisters, the presence of set shifting impairment alone clearly does not explain why some sisters developed anorexia nervosa and others did not. Bulik et al. (2007) have suggested endophenotypes are useful in deconstructing complex disorders. Wilksch & Wade (2009) conducted a well designed study investigating seven potential temperament risk factors for disordered eating. Six hundred and ninety nine female twins aged 12-15 years participated in the study, which identified 4 temperament endophenotypes for importance of shape and weight. These included an internalised thin-ideal, sense of ineffectiveness, body dissatisfaction and sensitivity to punishment/anxiety.

1.4.4 Impairments in Executive Functions Related to Set Shifting

As mentioned previously, successful set shifting involves the coordination of attention, working memory, initiation and inhibition of responses, as well as regulation and monitoring of behaviour. Deficits have been reported in patients with anorexia in many of these executive functions, including attention (Bosanac et al., 2007; Fassino et al., 2002; Kingston et al., 1996; Pendleton-Jones et al., 1991), working memory (Kemps, Tiggemann, Wade, Ben-Tovim & Breyer, 2006), and response inhibition (Lauer, 2002; Southgate, 2005).

The literature suggests immediate memory and basic attention abilities measured by tasks such as Digit Span (WMS-III, Wechsler, 2002) are intact in patients with

anorexia nervosa (Southgate et al., 2006). However there is consistent evidence for impairment in sustained attention (Laessle, Krieg, Fichter & Pirke, 1989; Seed, Dixon, McCluskey, & Young, 2000; Seed, Mccue, Wesnes, Dahabra & Young, 2002) and complex/divided attention tasks (Kingston et al., 1996; Lauer, Gorzewski, Gerlingloff, Backmund & Zhil, 1999). Studies of neuropsychological functioning in patients recovering from anorexia have shown attention deficits to resolve following re-feeding (Kingston et al., 1996) and in patients with anorexia who have achieved a normal weight (Pendleton-Jones et al., 1991).

The term inhibition is used to describe a wide variety of cognitive functions at differing levels of complexity (Kok, 1999). In the context of the current study, inhibition is defined as one's ability to deliberately inhibit dominant or automatic responses (Miyake et al., 2000). It is thus a conscious effortful process. Response inhibition is measured using tasks such as the Matching Familiar Figures Test (Kagan, Rosman, Day, Albert & Philips, 1964) and the Stroop Test (Trenerry, Crosson, DeBoe & Leber, 1989). Inhibition has not been widely study in anorexia. Two studies have been identified using the Matching Familiar Figures Test, both compared subtypes of anorexia nervosa and reported those with binge purge symptoms demonstrated a more impulsive cognitive style (Kaye, Bastiani, & Moss, 1995; Toner, Garfinkel, & Garner 1987). Four studies have utilised the Stroop Test in eating disorder samples. Kingston et al. (1996) reported the difference in performance between 46 inpatients and 41 healthy controls was approaching significance on the colour word task, with a trend towards poor performance in the eating disorder group. Two other studies have reported no significant differences on

Stroop Test performance between patients with anorexia and healthy controls (Steinglass, Walsh & Stern, 2006; Key et al., 2006). However both these studies employed small samples and the analyses are likely to have been underpowered.

1.4.5 Manifestation of Deficits

It is thought that deficits in set shifting ability may be associated with clinical characteristics commonly observed in the population of individuals with anorexia, such as a rigid obsession with food, perfectionism, an inflexible personality, obsessive thoughts and behaviours around eating, concrete and rigid approaches to problem solving, and perseverative and stereotyped behaviour (Bulik et al., 2003; Fassino et al., 2001; Tchanturia et al., 2005). Indeed, set shifting impairment in anorexia has been associated with obsessive-compulsive personality traits (Tchanturia et al., 2004c). Interestingly set shifting impairments have been reported in a wide range of psychiatric conditions including patients with Bipolar Disorder (Robinson et al., 2006), Schizophrenia (Ceaser et al., 2008) Autism, and Attention Deficit Hyperactivity Disorder (ADHD) (Happé & Frith, 1996).   First-degree relatives of people with Bipolar Disorder (Clark, Sarna & Goodwin, 2005) and Schizophrenia (Snitz, Macdonald & Carter, 2006), have also shown impaired performance on tests of set shifting. In addition extremely obese individuals have been found to show subtle impairments in the executive functions of planning, problem solving, mental flexibility and set shifting (Boeka & Lokken, 2008; Roberts, Demetriou, Treasure & Tchanturia, 2007b). Impaired set shifting ability may therefore represent a general risk factor for psychopathology and disordered eating.

1.4.6 Summary

Set shifting is an integral component of cognitive and behavioural flexibility. It involves the ability to adapt behaviour or thoughts in line with changing demands of the situation or environment, by switching attention between different topics. Successful set shifting involves the coordination of several sub-processes, and is considered to be an aspect of executive functioning. Psychological models of executive functioning highlight the role of attention and direction of neurological resources in successful neural coordination and processing (Baddeley & Hitch, 1974; Norman & Shallice, 1986). Neuropsychological studies of patients with anorexia have shown consistent deficits in set shifting ability, (Roberts et al., 2007a). Such deficits have been shown to persist following refeeding (Tchanturia et al., 2004c), exist in first degree relatives of patients with anorexia (Holliday et al., 2005); and may be a precursor to the development of the disorder. The right hemisphere, caudate nucleus and prefrontal cortex have been implicated as areas of dysfunction. It is likely dysfunction is related to impaired neuronal networks located in these areas (Cavedini et al., 2004; Fassino et al., 2002; Naruo et al., 2000; Steinglass & Walsh, 2006). Set shifting impairments are thought to result in inappropriate or dysfunctional patterns of behaviour and thought, including obsessive-compulsive and perseverative responses.

1.5 Social Problem Solving in Anorexia Nervosa

Deficits in set shifting ability may manifest clinically in a number of ways, for example applying the same solutions to different problems, stereotyped behaviour, or

concrete and rigid approaches to problem solving, all elements that are commonly observed in individuals with anorexia nervosa.

Social problem solving has been defined as a set of instrumental, cognitive behavioural skills necessary for adaptation in everyday life (D'Zurilla & Nezu, 1982). It has been suggested that effective problem solving is an important coping strategy, which can minimise psychological stress and negative affect. Problem solving outcomes are thought to be related largely to two processes, orientation to problems and application of actual problem solving strategies (D'Zurilla & Nezu, 1990). Problem orientation schemas are assumed to reflect automatic thoughts and feelings based on past experience (D'Zurilla, 1986; D'Zurilla & Chang, 1995), whereas application of actual problem solving strategies are thought to reflect conscious, effortful processes.

Generally, practical approaches to problem solving are associated with good psychological functioning, whereas emotional and avoidance orientated strategies are related to psychological dysfunction, health problems and somatic complaints (Endler & Parker, 1990a, 1990b; Ptacek, Smith & Zanas, 1992). In a non-clinical sample, worry has been shown to be associated with a more negative problem orientation (Gosselin, Dugas & Ladouceur, 2002); in addition there is some evidence that a negative orientation to problems can affect actual problem solving strategies over successive trials (Schewchuk, Johnson & Elliott, 2000).

Deficits in problem solving have been identified in a range of clinical populations, including patients with Depression (Marx, Williams & Claridge, 1992), Generalised Anxiety Disorder (Dugas, Gagnon, Ladouceur & Freeston, 1998) and Post Traumatic Stress Disorder (Nezu & Carnevale, 1987). In addition maladaptive problem solving has been connected to eating pathology in individuals with anorexia (Bloks, Spinhoven, Callewaert, Willemse & Turksma, 2001; Garcia-Grau, Fuste, Miro, Saldana & Bados, 2002; Ghaderi & Scott, 2000; Troop, Holbery & Treasure, 1998). Evidence from the neuropsychological literature demonstrates patients with anorexia nervosa make more perseverative errors than healthy controls, are unable to effectively utilise feedback and have difficulty coming up with novel and dynamic solutions to problems.

Interestingly, deficits in social problem solving are more often in relation to a negative orientation to problems rather than a deficit in actual problem solving skills (D'Zurilla & Nezu, 1999). It has been suggested that disordered eating may be a manifestation of maladaptive coping (Troop et al., 1994), and may be used to avoid current stressors (Ball & Lee, 2000). Indeed, women with eating disorders are more likely than women without an eating disorder to use cognitive avoidance and rumination as coping strategies, and thus are less effective in their coping (Troop, Holbery & Treasure, 1998). Avoidance style coping has been linked with predisposition to eating disorder development in a number of non-clinical (Garcia-Grau et al. 2002; Ghaderi & Scott, 2000; Koff & Sangani, 1996) and clinical samples (Neckowitz & Morrison, 1991; Troop et al., 1994).

1.5.1 Summary

Maladaptive social problem solving has been associated with anorexia nervosa, specifically women with anorexia have been shown to use cognitive avoidance and rumination as coping strategies. This may be linked to neuropsychological impairments in set shifting ability observed in individuals with anorexia.

1.6 Neuropsychological Functioning in Obsessive-Compulsive Disorder: Comparison with Anorexia Nervosa

Obsessive-compulsive Disorder is a highly disabling condition characterised by dysfunctional cognitive and behavioural symptoms. Recurrent aversive ideas, thoughts or images (obsessions) intrude, and ritualised behaviours such as cleaning, checking, counting and touching (compulsions) function to reduce anxiety and distress associated with the obsessions. Anorexia nervosa has been noted to have an obsessive-compulsive element, with sufferers becoming obsessed with thoughts of food, weight and shape and exhibiting a range of compulsions including body checking and ritualised eating behaviour. Obsessions regarding food and ritualised eating behaviours have been argued by some authors to be an adaptive response to food shortage (Södersten, Bergh & Zandian, 2006). In non-anorexic individuals exposed to conditions of starvation, weight gain significantly reduces obsessions and compulsions around food (Keys, Brozek, Henschel, Michelsen, & Longstreet Taylor, 1950). However, in individuals with anorexia nervosa these features persist under conditions of adequate nutrition and weight restoration suggesting that they are not merely an acute consequence of starvation (Sysko, Walsh, Schebendach, & Wilson, 2005). There is a growing evidence base to suggest an underlying neurological

component in obsessive-compulsive disorder similar to that seen in anorexia nervosa (Kuelz, Hohagen & Voderholzer, 2004).

1.6.1 Evidence for a Neurological Component in OCD

A neurological component in OCD is suggested by the high prevalence of OCD in several neurological conditions including Tourette's Syndrome, Sydenham's chorea, temporal lobe epilepsy, and postencephalitic Parkinsons Disease (Bihari, Pato, Hill, & Murphy, 1991; Hollander et al., 1990; Jenike, 1984; Malloy, Rasmussen, Braden & Haier, 1989; Rapoport, 1990). Further evidence is provided in that OCD is responsive to biological treatments including neurosurgery for mental disorder and SSRI's (Baer et al., 1995; Humble, Bejerot, Bergqvist & Bengtsson, 2001).

Functional neuroimaging has provided robust evidence for the involvement of the right cortex (Baxter et al., 1992; Lucey et al., 1997; Saxena et al., 1999), frontal areas including orbitiofrontal cortex (Saxena & Rauch, 2000), anterior cingulate (Saxena et al., 1999) and thalamus (Saxena & Rauch, 2000) in patients with OCD. Abnormal activity in limbic and paralimbic systems has been associated with depression and OCD (Breiter et al., 1996).

1.6.2 Cognitive Impairments in OCD

The most consistently reported neuropsychological deficits in OCD are in visuo-spatial functioning (Aronowitz et al., 1994; Cohen et al., 1996; Hollander et al., 1993), memory and executive functions (Alcaron, Libb & Boll, 1994). Unlike anorexia nervosa, there is no evidence for attention impairment in OCD (Kuelz et al.,

2004). Neuropsychological studies have provided evidence for deficits in immediate non verbal and verbal memory, as well as reduced confidence in memory in non-depressed patients with OCD (Zitterl et al., 2001). It has been hypothesised that memory deficits seen in OCD may be a result of poor encoding due to weak executive processes (Savage et al., 1995; 1999), and there is some evidence to support this from neuro-imaging and neuropsychological studies (Penadés, Catalán, Andrés, Salamero & Gastó, 2005; Savage et al., 2000). Similarly there is evidence that visuospatial deficits may also be executive in nature (Kulez et al., 2004). Thus OCD can be conceptualised as a neuropsychiatric disorder with executive dysfunction as the main component.

1.6.3 Set Shifting in OCD

Set shifting ability has been extensively studied in OCD. Mixed findings have been reported in relation to the different neuropsychological tests employed to measure set shifting performance. In relation to the Wisconsin Card Sort Test (WCST), some studies have reported significant impairments on the subscale of number of categories completed (Boone, Ananth, Philpott, Kaur, & Djenderedjian, 1991; Okasha et al., 2000). Other studies have reported no differences between patients with OCD and normal controls on this particular subscale (Abbruzzese, Bellodi, Ferrri & Scarone, 1995a; Abbruzzese, Ferri, & Scarone, 1995b; Zielinski, Taylor, & Juzwin, 1991). Generally the studies reporting no difference were of a better quality, and included larger sample sizes. Boone et al. (1991) recruited over half of their control subjects from siblings of patients with OCD, which may have affected their

findings given that unaffected first degree relatives of patients with OCD have been shown to have executive functioning impairments (Chamberlain et al., 2007).

On the WCST, patients with OCD have been shown to make more perseverative errors than normal controls, indicating impairment in set shifting ability (Roh et al., 2005). Studies of set shifting using the Object Alternation Task (OAT, Freedman, 1990), Delayed Attention Test (DAT, Freedman & Oscar-Berman, 1986) and the Intra Dimensional/Extra Dimensional test from the CANTAB battery (Downes et al., 1989) have consistently reported impairments in patients with OCD (Abbruzzese, Ferri & Scarone, 1997; Aycicegi, Dinn, Harris & Erkmen, 2003; Olley, Malhi & Sachdev, 2007).

One study investigated neuropsychological performance as measured by the WCST and regional cerebral blood flow in 14 patients with OCD and 14 matched controls (Lacerda et al., 2003). Results indicated negative correlations between perseverative errors on the WCST and regional cerebral blood flow in the right thalamus. Positive correlations were noted between non-perseverative errors and regional cerebral blood flow in frontal areas and the anterior cingulate. In addition, performance on neuropsychological testing was correlated with severity of OCD symptoms. These findings support prior studies that have suggested involvement of dysfunction of frontal subcortical networks in the right hemisphere (Lucey et al., 1997; Martinot et al., 1990). Tests of set shifting which are suggested to be sensitive to orbitofrontal damage have indicated marked impairments in patients with OCD (Abbruzzese et al., 1995a, 1997; Cavendini, Ferri, Scarone, & Bellodi, 1998; Gross-Isseroff et al., 1996;

Moritz, Fricke & Hand, 2001; Spitznagel & Suhr, 2002), providing support for the involvement of orbitofrontal areas in obsessive and compulsive responses.

Mixed findings have been reported in relation to set shifting in OCD as measured by tests of Verbal Fluency. Of 17 studies identified investigating verbal letter fluency in a recent review (Kulez et al., 2004), nine found no significant differences compared with normal controls, five reported significant differences, with OCD sufferers significantly worse than controls, one reported OCD sufferers scored significantly better than controls, and two studies had no control group. Neuropsychological studies reported in the literature have been compounded by methodological limitations such as extensive comorbidity, medication effects and poorly selected control groups, which makes drawing conclusions from the present literature difficult.

There is some evidence to suggest that executive impairments in OCD in the form of poor set shifting, alternation, response inhibition and non verbal memory are trait deficits (Bannon, Gonsalvez, Croft & Boyce, 2006) and could be endophenotype markers, due to their continued presence in patients recovered from OCD (Rao, Reddy, Kumar, Kandavel & Chandrashekar, 2008) and in unaffected first degree relatives of patients with OCD (Chamberlain et al., 2007).

Steinglass & Walsh (2006) present a neurocognitive model of anorexia nervosa based on comparisons with OCD. They outline parallel clinical features between OCD and anorexia, including cognitive preoccupation, repetitive and stereotyped

behaviours, and rigid, perfectionistic personality style. Their model of anorexia theorises that an inability to alter perseverative eating behaviours is a major maintaining factor, linked to difficulty learning and developing alternative patterns of behaviour, a commonality seen in both OCD (Deckersbach et al., 2002; Joel et al., 2005) and anorexia nervosa (Tchanturia et al., 2004b).

1.6.4 Summary

There is a growing evidence base to suggest an underlying neurological component in obsessive-compulsive disorder similar to that seen in anorexia nervosa. Consistent deficits in set shifting ability similar to those seen in anorexia nervosa have been implicated in OCD. The literature has implicated the involvement of dysfunction in orbitiofrontal areas, including the limbic network and fronto-striatal circuits, in set shifting impairments in OCD and anorexia.

1.7 Present Study

Anorexia nervosa is an eating disorder characterised by persistent failure to maintain a healthy weight, preoccupation with weight and shape, fear of fatness and expression of ritualistic and perseverative behaviours. Onset usually occurs during adolescence, a time when the frontal lobes of the brain develop to maturity. Neurological and Neuropsychological research indicates central nervous system dysfunction in eating disorders, involving the right hemisphere, caudate nucleus and prefrontal cortex. Cognitive impairments in set shifting ability have been consistently reported in patients with anorexia nervosa, and may be a precursor to the development of the disorder. Much of the research conducted to date has included

inpatients meeting strict diagnostic criteria for anorexia nervosa. Given that "most" patients with anorexia nervosa are managed on an outpatient basis (NHS QIS 2006, NICE 2004), much of the preceding literature may not generalise to outpatient populations where up to 60% of patients meet diagnostic criteria for EDNOS rather than anorexia nervosa (Fairburn et al., 2007).

It is not clear how set shifting impairments manifest clinically, however inability to effectively utilise feedback and poor social problem solving have been associated with anorexia. In addition, deficits in set shifting similar to those seen in anorexia nervosa have been implicated in OCD. Anorexia is known to have an obsessive-compulsive element and it is possible that such features are a result of underlying organic pathology. Much of the previous literature investigating neuropsychological functioning in anorexia nervosa has been fraught with methodological difficulties, including lack of consistency in patient sampling, small sample sizes, failure to define case status or recovery, failure to separate patients by diagnostic subtype, lack of healthy control groups and poor follow up.

Tchanturia et al. (2005) state that neuropsychological findings are somewhat inconsistent due to the wide range of test batteries employed. They suggest the use of a hypothesis driven approach to encourage in depth assessment of identified deficits. The current study has thus adopted a hypothesis driven approach drawing together aspects of the literature which suggest set shifting and problem solving deficits in anorexia nervosa that seem to have some parallels with obsessive-compulsive disorder.

1.7.1 Aims

This study is the first to investigate the relationship between neuropsychological test performance on measures of set shifting ability and psychological characteristics in a community sample of outpatients presenting to specialist eating disorder services with symptoms of anorexia nervosa. This study seeks to address some of the methodological concerns of the previous literature and to advance our understanding of how deficits in set shifting manifest clinically. The findings of this study have the potential to enhance our understanding of risk and maintenance factors in eating disorders, help inform new interventions and future service development.

1.7.2 Hypotheses

1. The eating disorder group will have worse set shifting ability than a healthy control group.

2. Poor set shifting ability will be associated with greater eating disorder severity in the eating disorder group.

3. Greater severity of obsessive-compulsive symptoms will be associated with poor set shifting ability in the eating disorder group.

4. Maladaptive social problem solving will be associated with poor set shifting ability in the eating disorder group.

The primary outcome measure of set shifting ability is the number of perseverative errors on the Wisconsin Card Sort Test, (WCST, Heaton et al., 1993). Additional key measures of set shifting include the Trail Making Test, Verbal Fluency and Colour Word Inhibition subtests of Delis-Kaplan Executive Function System (DKEFS;

Delis, Kaplan & Kramer, 2001); and the Hayling and Brixton tests (Burgess & Shallice, 1997). Eating disorder severity will be measured using the Eating Disorder Examination (EDE; Cooper & Fairburn, 1987; Fairburn & Cooper, 1993). Obsessive-compulsive traits will be measured using the Yale Brown Obsessive-Compulsive Scale (YBOCS; Goodman et al., 1989a; 1989b). Maladaptive social problem solving will be measured using the Social Problem Solving Inventory-Revised (SPSI-R; D'Zurilla & Maydeu-Oliveres, 1995). These measures are outlined in detail in the Methodology chapter.

Chapter 2: Methodology

Chapter 2: Methodology

The methodology outlined below is based on clinical guidelines and best research practice for eating disorder populations (NHS QIS, 2006; NICE, 2004).

2.1 Design

The study is quantitative and employs a between groups, cross sectional design involving formal neuropsychological testing, questionnaire and interview data. The study was designed to address some of the methodological limitations highlighted in a recent review of the neuropsychological literature in eating disorder populations (Southgate et al., 2006). Methods undertaken to address such limitations include stringent inclusion/exclusion criteria to minimise the effect of confounding variables; detailed reporting of participant characteristics such as clinical status, co-morbidity, current medication and drug/alcohol use; use of a prospective power calculation, and reporting of effect sizes within the results.

2.2 Ethical Issues

2.2.1 Approval

Ethical approval was granted from the University of Edinburgh, the University of Stirling and NHS Tayside Research Ethics Committees. Management approval was granted from NHS Tayside R&D Department and NHS Grampian R&D Department (see Appendix A for letters of approval).

2.2.2 Confidentiality

Personally identifiable data was only recorded on consent forms and on a key designed to anonymise data. Interview, questionnaire and neuropsychological assessment data was encoded with a unique identifier. Only the Chief Investigator and her Clinical Supervisor had access to personally identifiable data.

2.2.3 Data Storage

Consent forms were stored in a locked filing cabinet in a locked department on NHS Tayside property. A key to anonymised data was stored in another locked filing cabinet on NHS Tayside property. Only the Chief Investigator and her Supervisor had access to these cabinets. Transfer of any documents (e.g. consent forms) with patient identifiable data occurred through use of personal transport. Documents were transferred in a locked briefcase. No personally identifiable information was stored on computer.

Non identifiable data was stored in a locked filing cabinet, in a locked department on NHS Tayside property. All data stored on computer was anonymised and all computers used were password protected. Non identifiable data was also stored on a home laptop which was password protected and had anti virus software to prevent loss of data and corruption of files. Data was stored in line with the Data Protection Act (Department of Health, 1998), NHS Code of Practice on Protecting Patient Confidentiality, (Scottish Executive, 2003) NHS Tayside Information Governance Security Policy (2007) and NHS Tayside Information Security and Confidentiality Policy (2007). All memory sticks used to transfer non-identifiable data between sites

were encrypted and password protected in line with NHS Tayside Information Security and Confidentiality Policy (2007).

As part of the research methodology other researchers (e.g. Academic Supervisor at the University of Edinburgh) had temporary access to anonymised data to give advice on statistical analysis and interpretation of the data.

Following the date of study completion, as recommended by NHS Tayside Research and Development Office, personally identifiable data will be stored for 6-12 months and then destroyed. Non-identifiable data will be stored for 5 years from the date of publication.

2.2.4 Risks/Burdens and Benefits

The eating disorder group are potentially vulnerable, and there was a risk that they may find participation in the research upsetting. The Chief Investigator liaised closely with clinical staff and accepted their judgement in identifying patients who may have been too emotionally or physically frail to participate. The study did not ask anything about eating difficulties that had not already been assessed within the respective clinical teams. Systems were devised in collaboration with the Priory Hospital Glasgow, Tayside Eating Disorders Service and Grampian Eating Disorders Service for dealing with any distress caused by participation in the research. This included participants having access to clinical staff following their involvement in the study.

Participation required approximately 1.5 – 2 hours of the participants time, including allocated time for a break in testing. Participants were offered a break in between neuropsychological testing and questionnaire completion, to reduce potential for fatigue.

Potential benefits for the eating disorder group included having their experience taken seriously and being given the opportunity to play a role in helping others understand their condition. Participants may have felt good about helping to develop new treatments, which could benefit future patients. Benefits for the healthy control group include increasing their knowledge and experience of psychological research. Clinical experience and neuropsychological research indicate that individuals generally find the experience of participating in neuropsychological testing enjoyable.

2.3 Power Calculation

The number of participants was decided upon following power calculation and statistical advice. G*Power 3.0.10 (Faul, Erdfelder, Lang, & Buchner, 2007) was used for the power calculation. A recent meta analysis (Roberts et al., 2007a) suggested the effect size of a set shifting impairment in subjects with eating disorders, on a range on neuropsychological measures was medium.

In relation to Hypothesis 1 (see Section 1.7.2), analysis consisting of t-tests between two independent means calculated for a medium effect size (0.5) at power 0.8 with alpha level of 0.05, suggests 51 participants are required per group. Using an effect

size of 0.62 as reported by Roberts et al. (2007a) in relation to the WCST the primary outcome measure in the current study, with the above parameters suggests 33 participants are required per group.

In relation to Hypotheses 2, 3 and 4 (see Section 1.7.2), analysis consisting of correlation for a medium effect size (0.5) at power 0.8 with alpha level of 0.05, suggests 64 participants are required. As far as I am aware no studies have been published in this area with a sample as large as 64. Of the 15 studies included in the Roberts et al. (2007a) meta-analysis, sample sizes ranged from 10-47 with a mean of 24. Due to time constraints the present study intended to collect 24 participants per group. It is recognized that this may leave the study underpowered but still represents a reasonable sample size in the context of previous literature within this population group.

2.4 Participants

2.4.1 Inclusion/Exclusion Criteria

The selection criteria for each group are listed below.

Group 1: Eating Disorder Group

Inclusion Criteria:
- Female.
- English as first language.
- Age 18−65.
- Receiving treatment for an eating disorder with anorexic symptoms on an outpatient basis as part of Tayside Eating Disorders Service, Grampian Eating Disorders Service or Priory Hospital Glasgow, Outpatient Eating Disorders Service.

Exclusion Criteria:
- Diagnosis of Bulimia Nervosa.
- Deemed by Clinical staff to be too emotionally or physically frail to participate.
- Current Psychosis.
- History of Learning Disability/Developmental Disorder.
- History of head injury involving loss of consciousness.
- History/Current Neurological Disorder.
- Uncorrected significant visual or motor impairment.
- Past substance abuse/related disorder (i.e. has previously received treatment from an addictions service).
- Knowledge of Neuropsychological tests (i.e. has been employed to administer tests to patients in a clinical or research setting; or has undergone neuropsychological testing previously as a patient).

Group 2: Healthy Control Group

Inclusion Criteria:
- Female.
- English as first language.
- Age 18−65.
- Student studying Psychology at the University of Stirling.

Exclusion Criteria:
- Previous history of eating disorder requiring consultation with a health care professional.
- EDE Global score above 4.
- Significant current Psychiatric Disorder (significant psychiatric disorder is identified if the individual is currently receiving treatment from specialist mental health services).
- History of Learning Disability/Developmental Disorder.
- History of head injury involving loss of consciousness.
- History/Current Neurological Disorder.
- Uncorrected significant visual or motor impairment.
- Past substance abuse/related disorder.
- Knowledge of Neuropsychological tests.

2.4.2 Sample

Group 1: Eating Disorder Group

Participants in Group 1 comprised a sample of 17 female outpatients recruited from

multi-site specialist eating disorder services, who were receiving treatment for an

eating disorder with anorexic symptoms. Research suggests that the majority of patients being treated for anorexia nervosa are female, with relatively few male patients (Fairburn & Harrison, 2003). There is very little published literature on male anorexia. One study has identified differences between male and female patients with anorexia on the personality construct of extraversion (Fichter, Daser & Postpischil, 1985). However, a number of studies have reported similar clinical presentation in males and females (Burns & Crisp, 1985; Carta, et al., 2006; Crisp, Burns & Bhat, 1986; Hall, Delahunt & Ellis, 1985). Much of the research consists of small sample sizes or single case studies (Carta et al., 2006). As there may be differences between male and females with anorexia and the study was unlikely to be able to recruit many males, the present study included only female participants.

All participants met diagnostic criteria for anorexia nervosa or an eating disorder not otherwise specified (EDNOS) as outlined in DSM-IV (American Psychiatric Association, 1994). All participants with an EDNOS diagnosis met at least 2 of the 4 key diagnostic criteria for anorexia nervosa in DSM-IV. NICE Guidelines on eating disorders (2004) and NHS QIS (2006) recommendations for the management of eating disorders in Scotland state that most people with anorexia nervosa can be managed on an outpatient basis. Much of the previous literature investigating neuropsychological functioning in anorexia nervosa has included largely inpatient samples. Recruitment of an outpatient sample allowed for a broader examination of the population, and is more representative of the majority of patients with eating disorders.

Participation was voluntary and patients were advised that participation or non-participation would not affect their treatment. Clinicians approached patients who they considered were well enough medically and psychiatrically to participate in the study. Twenty eating disorder patients were approached; one patient was excluded on the basis she had ongoing memory problems; one patient agreed to take part in the study, but did not subsequently attend her appointment; and one patient declined to participate in the study.

Group 2: Healthy Control Group

Participants in Group 2 comprised a sample of 27 female students studying Psychology at the University of Stirling. This group was chosen as they are reasonably similar to the eating disorder group in terms of age, occupation, education and intellectual functioning. Previous research conducted at the Priory Hospital Glasgow, reported the mean age of patients with anorexia nervosa to be 25.4, and 38% reported their occupation as students (Personal communication, Priory Hospital). There are no existing normative data for females of this age group and educational status for the range of measures included in the current study. Participation was voluntary and students did not receive course credit or payment for taking part. They were advised that participation or non-participation would not affect their academic standing or position at the university in any way. Of 50 students who initially indicated that they would like to participate, 18 did not respond to an email inviting them to arrange a time to take part, three were excluded as English was not their first language, one was excluded due to previous head injury involving loss of consciousness, and one was excluded due to dyslexia and dyspraxia.

2.5 Measures

All measures used in the study can be seen in Table 2.1.

Table 2.1: Psychological and Neuropsychological Measures

Measure	Domain
Psychological Measures	
Eating Disorders Examination (EDE)	Eating psychopathology
Symptom Check List−90 Revised (SCL−90R)	Psychiatric pathology
Yale−Brown Obsessive-compulsive Scale (Y−BOCS)	Obsessive-compulsive symptoms and severity
Social Problem Solving Inventory Revised (SPSI−R)	Social problem solving
Neuropsychological Measures	
National Adult Reading Test (NART)	Pre-morbid intellectual functioning
Wisconsin Card Sort Test (WCST)	Ability to shift and maintain set, form abstract concepts and to utilise feedback
Delis−Kaplan Executive Function System (DKEFS)	Set shifting, divided attention and response inhibition
Hayling & Brixton Test	Set shifting and response inhibition

2.5.1 Psychological Measures

Eating Disorders Examination (EDE) (Cooper & Fairburn, 1987; Fairburn & Cooper, 1993).

Regarded as the 'gold standard' assessment of anorexia and bulimia nervosa, the EDE is a 23-item semi structured, investigator based clinical interview. Questions are rated on a seven-point scale (0-6) with higher scores representing greater psychopathology. The EDE contains four subscales (Dietary Restraint, Eating Concern, Shape Concern and Weight Concern), as well as frequency measures of

binge eating and compensatory behaviours over the preceding 28 days. A global score can be calculated by adding the subscale mean scores and dividing by 4. Total scores on Dietary Restraint, Eating Concern and Weight Concern range from 0-30; while total scores on Shape Concern range from 0-48. All subscale mean scores and the Global score range from 0-6. There are no defined cut-offs for 'caseness' of eating disorders. The EDE can be used to generate operationally defined eating disorder diagnoses for DSM-IV. However it does not generate sufficient information to accurately determine subtypes of anorexia nervosa (Fairburn & Cooper, 1993). The good internal consistency, (Beumont, Kopec-Schrader, Talbot & Touyz, 1993; Cooper, Cooper & Fariburn, 1989); concurrent (Rosen, Vara, Wendt & Leitenberg, 1990) and discriminant (Cooper et al., 1989; Rosen et al., 1990; Wilson & Smith, 1989) validity, and inter-rater reliability (Beglin, 1990; Cooper & Fairburn, 1987; Rosen et al., 1990; Wilson & Smith, 1989) of the EDE have been well documented in adults.

Symptom Check List−90 Revised (SCL−90R) (Derogatis, 1994).

The SCL-90R is a 90-item self-report symptom inventory designed to reflect the psychological symptom patterns of community, medical and psychiatric respondents. Each item is rated on a five-point scale of distress (0-4) ranging from 'Not at all' to 'Extremely.' Distress is measured over the last 7 days. The SCL-90R is scored and interpreted in terms of nine primary symptom dimensions: Somatisation, Obsessive-Compulsive, Interpersonal Sensitivity, Depression, Anxiety, Hostility, Phobic Anxiety, Paranoid Ideation and Psychoticism. Mean scores (range 0-4) are calculated for each primary symptom dimension. Three global indices of distress can be

calculated: Global Severity Index (GSI), Positive Symptom Total (PST) and Positive Symptom Distress Index (PSDI). The GSI is calculated by adding all items and dividing by the total number of items completed (usually 90). GSI thus ranges from 0-4. The PST is calculated by counting all items which have been responded to, i.e. all responses which have not been scored zero. PST thus ranges from 0-90. The PSDI is calculated by dividing the sum of all items by the PST, and ranges from 0-4.

Good internal consistency of the primary symptom dimensions has been demonstrated, with alpha coefficients ranging from 0.77 for the Psychoticism sub-scale to 0.90 for the Depression sub-scale in one study of symptomatic volunteers (Derogatis, Rickels & Rock, 1976). Another study with a sample of psychiatric outpatients reported alpha coefficients ranging from 0.79 for Paranoid Ideation to 0.90 for Depression (Horowitz, Rosenberg, Baer, Ureno & Villasensor, 1988). These studies demonstrated good test-retest reliabilities. Good convergent-discriminant validity for the SCL-90R has also been documented in symptomatic and healthy volunteers (Boleloucky & Horvath, 1974; Derogatis et al., 1976).

Yale−Brown Obsessive-Compulsive Scale (Y−BOCS) (Goodman et al., 1989a; 1989b).

Regarded as the 'gold standard' for the assessment of obsessive-compulsive symptoms (Frost, Steketee, Krause & Trepaner, 1995; Moritz et al., 2002), the Y-BOCS offers a symptom checklist as well as a discrete measure of symptom severity. The symptom checklist is composed of 58 items, which cover a range of obsessions and compulsions, clustered by behavioural expression (e.g. checking compulsions)

and thematic content (e.g. contamination obsessions). The present study used a version of the Y-BOCS symptom check list published by Baer (1991); this version assesses the same 15 categories as the interview version and is used widely.

Following completion of the symptom checklist respondents were asked to identify their main symptoms and to complete the Y-BOCS scale with these in mind. The Y-BOCS scale is a 10-item measure of severity, with five items relating to obsessions and five to compulsions. The items assess time spent, degree of interference, distress, resistance and degree of control. Items are rated on a 5-point scale from 0 (no symptoms) to 4 (extreme symptoms.) Scores on the Y-BOCS thus range from 0-40. Scores of <10 indicate very mild OCD symptoms; 10-15 mild OCD symptoms; 16-25 moderate OCD symptoms; and >25 severe OCD symptoms. The scale also includes a number of supplementary items for clinical assessment purposes, which are not included in subscale or total scores. The Y-BOCS can be administered in the form of interview or self-report. A number of studies have demonstrated the validity and reliability of the interview version (Goodman et al. 1989a; 1989b; Kim, Dysken & Kuskowski, 1990; 1992; Woody, Steketee & Chambless, 1995). Comparison of interview and self-report administration showed good agreement between both the symptom checklist and severity ratings (Steketee, Frost & Bogart, 1995). The authors suggest a self-report version would save time and costs involved in administration. The current study used a self-report version (Baer, 1991) of the Y-BOCS on this basis.

Social Problem Solving Inventory Revised (SPSI−R) (D'Zurilla & Maydeu-Oliveres, 1995).

The SPSI-R is a 52-item, multidimensional, self-report measure of social problem solving ability derived from a factor analysis of the original 70-item theory based measure. Each item is responded to on a five-point scale from 0 'not at all true of me' to 4 'extremely true of me.' The SPSI-R consists of two problem orientation subscales, and three actual problem-solving subscales, detailed in Table 2.2. Positive problem orientation and rational problem solving subscales measure adaptive problem solving, whereas negative problem orientation, impulsivity/careless style and avoidance style subscales measure maladaptive problem solving.

There is evidence that the actual problem solving subscales of the SPSI-R are ecologically valid in that they have been shown to accurately predict academic success after controlling for aptitude (D'Zurilla & Sheedy, 1992). Acceptable convergent, construct and discriminant validity have been reported (Chang & D'Zurilla, 1996; D'Zurilla & Maydeu-Olivares, 1995; D'Zurilla, Nezu & Maydeu-Olivares, 1998). Test-retest reliabilities range from of 0.72 for positive problem orientation to 0.88 for negative problem orientation (D'Zurilla et al., 1998).

Table 2.2: Subscales of the SPSI-R

Subscale	Domain Measured
Problem Orientation	
Positive Problem Orientation (PPO)	Positive appraisal, realistic beliefs and positive outcome expectancies.
Negative Problem Orientation (NPO)	Inhibited cognitive processes, perceived threat, low frustration tolerance and self-inefficacy.
Actual Problem Solving	
Rational Problem Solving (RPS)	Ability to define problems and formulate solutions, make decisions, implement solutions and generate alternatives.
Impulsivity/Carelessness Style (ICS)	Impulsive, incomplete or careless attempts to solve problems.
Avoidance Style (AS)	Avoidance of problem through procrastination, passivity and inability to accept responsibility.

2.5.2 Neuropsychological Measures

National Adult Reading Test (NART) (Nelson, 1982).

The NART is widely used in clinical and research practice as a measure of pre-morbid intellectual ability. It is a reading test of graded difficulty composed of 50 short, irregularly spelt words (e.g. cough), which could not be correctly pronounced without prior knowledge of the word. The examiner presents the participant with a word card and asks them to pronounce each word aloud. The examiner records the number of errors (range 0-50) and from this can estimate Wechsler Adult Intelligence Scale (WAIS-R, Wechsler, 1981) verbal, performance and full scale IQ

scores. The development of the NART was based on the observation that reading ability is highly correlated with IQ in the general population, and is usually preserved despite widespread cognitive decline associated with dementia (Nelson & McKenna, 1975). More recently research has demonstrated that NART performance remains preserved despite cognitive impairment of neurological or psychiatric origin, with the exception of moderate to severe Alzheimer's Disease and some specific conditions (Crawford, 1992; Franzen, Burgess & Smith-Seemiller, 1997; O'Carroll, 1995).

The internal consistency (Crawford, Stewart, Garthwaite, Parker & Besson, 1988; Nelson & Willison, 1991), test-retest reliability (Crawford, Stewart, Besson, Parker, & De Lacey, 1989), and inter-rater reliability (O'Carroll, 1987; Crawford et al., 1989) of the NART have been well documented. A recent study investigating the retrospective validity of the NART over a 66 year interval reported that IQ at age 11 was highly correlated with NART performance at age 77, and was able to account for over 50% of the variance in psychometric intelligence (Crawford, Deary, Starr & Whalley, 2001).

Wisconsin Card Sort Test (WCST) (Heaton et al., 1993).

The WCST is a measure of an individual's ability to form abstract concepts, shift and maintain set, and to utilise feedback (Strauss, Sherman & Spreen, 2006). The test consists of 4 stimulus cards, placed in front of the participant, the first with a red triangle, the second with 2 green stars, the third with 3 yellow crosses and the fourth with 4 blue circles on them. The participant is then given two packs each containing 64 response cards, which have designs similar to those on the stimulus cards, varying

in colour, shape and number. The participant is asked to match each of the cards to one of the 4 key cards and is given feedback each time whether they are right or wrong. No warning is provided that the sorting rule changes. Performance is scored on seven dimensions, detailed in Table 2.3. In the current study number of perseverative errors is identified as the primary outcome measure of set shifting ability.

Table 2.3: Subscales of the Wisconsin Card Sort Test

Subscale	Definition
Number of categories completed	Number of sequences of 10 correct consecutive card sorts.
Trials to complete first category	Number of cards required to achieve first sequence of 10 correct consecutive sorts.
Perseverative errors	Number of persisting responses to a stimulus characteristic that is incorrect.
Percent perseverative errors	Percentage of perseverative errors in relation to total number of cards sorted.
Failure to maintain set	Number of instances when a participant has made 5 or more correct responses and then makes an incorrect response before the sorting rule has changed.
Percent conceptual level responses	Percentage of correct responses in runs of 3 or more.
Learning to learn	Mean change score in conceptual efficiency across categories completed.

Test-retest reliabilities for the WCST are confounded by practice effects and so reported correlations are generally low (Paolo, Axelrod & Troester, 1996). Inter-rater reliability is variable with some studies reporting excellent correlations, up to 0.83

(Axelrod, Goldman, & Woodard, 1992) and others reporting quite low correlations (Flashman, Horner & Freides, 1991). A recent exploratory factor analysis identified 3 factors, the ability to shift set, problem solving/hypothesis testing and response maintenance (Greve, Stickle, Love, Bianchini & Stanford, 2005).

Delis−Kaplan Executive Function System (Delis et al., 2001).

- **Trail Making Subtest**

This test assesses set shifting ability and divided attention, and is made up of 3 subtasks: the number task, the letter task and the number/letter switch task. The main subtask of interest in this study is the number/letter switch task, as this involves set shifting. In the number task, participants are asked to connect encircled numbers randomly arranged on a page in consecutive order. In the letter task, participants are asked to connect encircled letters randomly arranged on a page in consecutive order. In the number/letter switch task, participants are asked to switch between connecting encircled numbers and letters by alternating between the two sequences. Performance time is the primary measure on all tasks. Scaled scores based on completion time can be calculated, as can error measures. Good internal consistency and moderate test retest reliability are described in the technical manual (Delis et al., 2001).

- **Verbal Fluency Subtest**

This test assesses spontaneous production of words under restricted search conditions. Outcome measures on this test include letter fluency, category fluency, and category switching accuracy. It is also possible to calculate error scores. The main variable of interest in the current study is letter fluency, which requires the

participant to say as many words as possible starting with a specified letter during a 60 second interval. Three letter fluency trials are conducted, requiring the participant to generate words beginning with the letters F, A, and S respectively. The outcome measure is the number of correct words generated over the 3 trials. Correct words are those that are in keeping with the rules of the trial and are not repetitions. Secondary variables of interest are category fluency and category switching accuracy. During the category fluency task participants are asked to name as many things as they can within the categories of animals and boys names, during two 60 second intervals. The outcome measure is the number of correct words generated over the 2 trials. During the switching task participants are asked to alternate betweens saying words from two different categories, fruits and furniture, during a 60 second interval. The outcome measure is number of correct alternations. High internal consistency has been shown for the Verbal Fluency component and moderate consistency was shown for the category switching component (Delis et al., 2001).

- **Colour Word Inference Subtest (Stroop)**

This is also known as the Stroop Test, and assesses selective attention and set shifting, on 2 subtasks. The main variable of interest in this study is Colour Word inhibition task. During the Colour Word inhibition task, the participant is asked to name aloud the colour of the ink in which colour words are printed, as quickly and accurately as they can. In the Colour Word inhibition/switch task, participants are asked to switch back and forth between naming dissonant ink colours and reading conflicting words. The primary measure for both tasks is seconds taken to complete the task. In addition the number of errors is recorded. The good internal consistency

and test retest reliability has been shown (Delis et al., 2001). The validity of other versions of the Trail Making Test, Verbal Fluency and Stroop Tests have been widely demonstrated (Strauss et al., 2006).

Hayling & Brixton (Burgess & Shallice, 1997).

The Hayling and Brixton tests are measures of response inhibition and set shifting ability (Strauss et al., 2006). The Hayling test consists of two sets of 15 sentences, each with the last word missing. In the first part the examiner reads the sentence aloud and asks the participant to complete the sentence as quickly as possible. In the second part the participant is asked to complete the sentence with a word that is totally unconnected to the sentence. Participants must therefore inhibit the primed response and come up with a new unconnected response. Outcome measures include total time taken to complete the second part (section 2 total time); and number of errors. The measure of interest in the current study is number of errors, as this is thought to provide the best reflection of failure to inhibit inappropriate responses.

The Brixton test consists of a 56-page booklet, with each page showing the same array of 10 circles set in two rows of five, with each circle numbered from 1 to 10. On each page one of the circles is filled with blue ink, the position of which changes from one page to the next. The changes in position are governed by a series of rules which vary without warning. The participant is shown one page at a time and asked to predict where the next filled circle will be, by trying to find a pattern or rule based on previous pages. The Brixton test is scored by recording the total number of errors.

The reliability and validity of these tests are documented in the technical manual (Burgess & Shallice, 1997). The test retest reliabilities for the Hayling and Brixton tests are reported as adequate (Burgess & Shallice, 1997). One study investigated inter-rater reliability on the Hayling test and reported 76.5% agreement (Andres & Van der Linden, 2000).

2.6 Procedure

A summary diagram detailing the participant journey can be seen below in Figure 2.1.

Figure 2.1: Participant Journey

Group 1: Eating Disorder Patients identified as suitable for approach by Clinical Staff.	**Group 2: Healthy Controls** Presentation of study outline by Chief Investigator during lectures.
Written information sheets given out by Clinical Staff. Interested participants fill in slips giving contact details.	Written information sheets given out by Chief Investigator. Interested participants fill in slips giving contact details.

Minimum 24 hours consideration time

Groups 1 and 2
Potential participants contacted by Chief Investigator to give opportunity to discuss research and ask questions.

Groups 1 and 2
Interested participants sign up for meeting.

Group 1: Eating Disorder Meet with Chief Investigator within local NHS setting, screened for inclusion/exclusion criteria and consent to participate.	**Group 2: Healthy Controls** Meet with Chief Investigator at the University of Stirling, screened for inclusion/exclusion criteria and consent to participate.

Groups 1 and 2
Complete Neuropsychological tests and questionnaires.

Groups 1 and 2
General discussion and feedback following participation.

Group 1: Eating Disorder Group

Study Sites: Tayside Eating Disorders Service, Grampian Eating Disorders Service, Priory Hospital Glasgow, Outpatient Eating Disorders Service.

Prior to study recruitment, the Chief Investigator attended Eating Disorder Service team meetings to present information about the background, rationale and procedure for the study to staff working within the respective services. Meetings were attended periodically during the course of study in order to liaise with staff.

Any outpatients attending Tayside Eating Disorders Service, Grampian Eating Disorders Service or Priory Hospital Outpatient Eating Disorders Service for treatment of an eating disorder with anorexic symptoms and meeting inclusion criteria were considered for participation in the study. Clinical staff within the respective services approached patients who in their clinical judgement were deemed as medically fit and without acute psychological distress or co-morbidity. Patients who expressed an interest in finding out more about the research were given verbal and written information about the study by their clinician (see Appendix B). With their agreement their details were passed onto the Chief Investigator who contacted them by telephone or email to further discuss the study. Previous research has used similar methodology to contact potential participants and has demonstrated a high response rate (Crombie et al., 2008; Kiezebrink, et al., 2009).

There was a period of at least 24 hours between receiving the written study information and being contacted by the Chief Investigator, to ensure potential

participants had sufficient time to consider whether they would like to take part in the study. If patients decided to participate, a meeting was arranged. Meetings took place at one of the following locations: Neuropsychology Department, Ninewells Hospital, Dundee; Clinical Psychology Adult Mental Health Department, Dundee, Perth; Clinical Psychology Eating Disorder Service, Dundee; Clinical Psychology Eating Disorder Service, Royal Cornhill Hospital, Aberdeen; Outpatient Eating Disorder Service, The Priory Hospital, Glasgow.

At the meeting patients were given the opportunity to ask any further questions, and if appropriate completed consent forms (see Appendix C). They were informed that their participation was confidential and that they did not have to write their names on any of the questionnaires or neuropsychological tests. Confidentiality was limited if there was an issue of risk to the patient or others, in which instance the clinical staff were informed. The limits to confidentiality were made clear to participants in the information sheet and when they completed the consent form. They were informed participation was voluntary and that they could withdraw from the study at any time without giving a reason, and that participation or non-participation would in no way impact on their treatment.

The assessment procedure involved screening for inclusion/ exclusion criteria, a brief interview to collect demographic information, formal neuropsychological testing, time for a break and then administration of a semi-structured interview regarding eating behaviour and thoughts around shape and weight, followed by completion of the questionnaires. In total, this took approximately 1.5−2 hours.

Measures were administered in the following order with all participants: Demographic information, NART, WCST, Delis-Kaplan: Trail Making Test, Verbal Fluency, Colour Word Inhibition Test; Hayling and Brixton Tests, EDE, Y-BOCS, SPSI, and SCL-90. Demographic information regarding Body Mass Index (BMI) was calculated by asking participants their current height and weight. To reduce potential influence of mood/distress on neuropsychological test scores, the EDE interview and psychological questionnaires were administered after the neuropsychological tests. Having the Chief Investigator present at all times allowed for discussion of any concerns about the study, or any other issues raised.

Following study completion participants had a general discussion with the Chief Investigator and were offered feedback on their neuropsychological test performance. More detailed written feedback was available on request. Participants were given the opportunity to request a summary of the findings, which will be provided on completion of the study. In the event of the procedure causing distress, participants were advised to speak to their treating clinician or were directed to external sources of support.

Group 2: Healthy Controls.
Study site: University of Stirling.

Students enrolled in Psychology courses at the University of Stirling were recruited through short presentations at the beginning of lectures. The Chief Investigator presented some brief information at the beginning of 3 lectures and handed out

participant information sheets (see Appendix B). At the end of lectures interested students provided their contact details and were subsequently contacted by telephone or email by the Chief Investigator to further discuss the study, and if appropriate to arrange a meeting for participation in the research. There was a period of at least 24 hours between receiving the written study information and being contacted by the Chief Investigator. This ensured that potential participants were given sufficient time to consider whether they would like to take part in the study.

The meetings took place in a quiet office at the University of Stirling. At the meeting students were given the opportunity to ask any further questions and if appropriate completed consent forms (see Appendix C). The assessment procedure for the Healthy Controls was as described above for the Eating Disorder participants, except the Healthy Controls were weighed with a set of portable scales, and completed the EDE interview at the beginning of the test battery, following collection of the demographic information. After study completion participants had a general discussion with the Chief Investigator and were offered feedback on their neuropsychological test performance. More detailed written feedback was available on request. Participants were given the opportunity to request a summary of the findings, which will be provided on completion of the study. In the event of the procedure causing distress, participants were advised to speak to their Director of Studies and were directed to external sources of support.

Additional Data Collection

Study site: Priory Hospital, Glasgow, Inpatient Eating Disorders Unit.

As part of a larger study, inpatient data was collected by an Assistant Psychologist from patients admitted to The Priory Hospital, Glasgow, Eating Disorders Unit. It was intended that data from 50 inpatient participants would be collected for comparison with the outpatient data collected in the present study. This would have improved the statistical power of the study and enabled a wider range of analyses. Unfortunately due to difficulties with recruitment the number of participants collected was not sufficient to be included as a separate group in the analyses on this occasion.

2.7 Data Analysis

In relation to Hypothesis 1 (see Section 1.7.2), independent samples t-tests were conducted to determine any statistically significant differences between the eating disorder and healthy control group on neuropsychological measures of set shifting. To reduce the number of comparisons within the data, a number of set shifting measures were identified a priori as key variables. The WCST perseverative errors subscale was identified as the primary outcome measure of set shifting ability. The following set shifting measures were identified as key secondary variables: Delis-Kaplan Number/Letter Switch, Delis-Kaplan Letter Fluency, Delis-Kaplan Colour Word Inhibition, Hayling and Brixton, errors.

Clinically significant impairments in set shifting ability, were determined on a case by case basis by converting neuropsychological test scaled scores to z-scores, using means and standard deviations from published normative data. This allowed identification of impairments on a case by case basis by comparing an individual's z-score with an "expected" z-score calculated on the basis of their estimated premorbid intellectual ability. Scores that fell at least one standard deviation (i.e. one z-score) below the expected level were defined as clinically significant impairments.

In relation to Hypotheses 2, 3 and 4 (see Section 1.7.2), analysis consisted of calculating Pearson's *r* correlations between the measures of set shifting that were identified a priori as key variables, and the corresponding psychological measures. Thus, to investigate Hypothesis 2, the key set shifting measures were correlated with eating pathology, as measured by BMI and EDE. To investigate Hypothesis 3, these key set shifting measures were correlated with severity of obsessive-compulsive symptoms as measured by the YBOCS total score. To investigate Hypothesis 4, the same key set shifting measures were correlated with social problem solving, as measured by the SPSI-R. Data analysis is reported in the Results chapter.

Chapter 3: Results

Chapter 3: Results

3.1 Exploring Assumptions

3.1.1 Examination of the Normal Distribution of the Data

The use of parametric statistical tests assumes that the sampling distribution is normally distributed. If this assumption is correct, the data being tested should also be of a normal distribution. The assumption of normality was tested in the current study firstly by visual examination using histograms with normal curve overlay and Probability-Probability (P-P) plots. Distributions for all variables looked uni-modal, however some variables appeared skewed. Skewness and kurtosis were investigated by examining the ratio of the skewness or kurtosis index to its standard error by converting it to a z score. If z >1.96 or < -1.96, the distribution is considered significantly positively or negatively skewed (Field, 2009). Using these criteria, in the eating disorder group the following variables were positively skewed: WCST perseverative errors, percent perseverative errors, set failure; Delis-Kaplan (DK) number letter switch, Colour Word inhibition switch; Hayling errors and SCL-90 phobic anxiety. Conversely the following variables were negatively skewed: FSIQ, EDE shape concern, WCST conceptual level responses, learning to learn; and YBOCS total score. All other variables were normally distributed.

In the healthy control group the following variables were positively skewed: age, all EDE subscales, all WCST subscales except conceptual level responses and learning to learn; DK number letter switch, Colour Word inhibition/switch, Verbal Fluency-category; Hayling section 2 total time, errors; YBOCS total and all subscales of the

SCL-90. Conversely WCST conceptual level responses was negatively skewed. All other variables were normally distributed.

Use of non-parametric statistical tests, which are not based on the assumption of an underlying normal distribution have considerable benefits as opposed to transforming the data (Field, 2009), however they do not allow reporting of effect sizes and are considered less powerful than parametric methods. Parametric tests like t-tests and MANCOVA are considered robust even under departures from the underlying assumptions (Field, 2009). In the present study the data were analysed using both non-parametric and parametric statistics. In addition skewed variables were log transformed to base 10. The results were largely analogous. Thus parametric statistics are reported in the main analysis, while non-parametric results are reported in Appendix D, and log transformed analyses are reported in Appendix E.

3.1.2 Homogeneity of Variance

For the assumption of homogeneity of variance to be sustained, the variance should be reasonably consistent on individual measures throughout the data (Field, 2009). In order to test this, Levene's test was applied to identify any significant differences between groups on a test by test basis. If group variance was significantly different test statistics were read from the "*equal variances not assumed*" row.

3.1.3 Statistical Analyses

The data were entered into a spreadsheet and analysed using the Statistical Package for the Social Sciences (SPSS) Version 16.0. For neuropsychological test data

statistical analysis was carried out using raw scores, unless otherwise stated. Significant outliers ($p<0.05$) were assessed using Grubbs method (Barnett & Lewis, 1998). Two data points (of 510) were identified as outliers in the eating disorder group (1 on WCST learning to learn and 1 on DK Colour Word inhibition). Seventeen data points (of 810) were identified as outliers in the healthy control group (9 on set shifting variables and 8 on psychological variables). Removal of outliers acts to reduce skewness in the distribution and reduce the effect of outlying data points on statistical modelling (Field, 2009). However outliers should only be removed if they are believed to be unrepresentative of the population being sampled. All outliers were believed to give an accurate reflection of participants' performance on the neuropsychological tests administered, with the exception of one of the healthy controls who became frustrated with her performance on the WCST. As the test progressed she began to sort cards at random rather than attempting to decipher the pattern of correct and incorrect responses. Thus her 4 outlying data points relating to the WCST subscales of total errors, perseverative errors, percent perseverative errors and percent conceptual level responses were replaced with the highest value for that variable within that group plus one unit, in accordance with standard procedure (Tabachnik & Fidell, 2001). All other outliers were retained in the analyses, unless otherwise stated.

To reduce the number of comparisons within the data, certain set shifting measures were identified a priori as key variables. The WCST perseverative errors subscale was identified as the primary outcome measure of set shifting ability. The following set shifting measures were identified as key secondary variables: Delis-Kaplan

Number/Letterletter Switch, Delis-Kaplan Letter Fluency, Delis-Kaplan Colour Word Inhibition, Hayling and Brixton, errors.

3.2 Descriptive Statistics

3.2.1 Eating Disorder Group

Seventeen outpatients were recruited to the eating disorder group. All participants met inclusion/exclusion criteria as outlined in Section 2.4.1. No participants reported a head injury involving loss of consciousness, however 2 participants had previously suffered a mild concussion as defined by Gennarelli (1986).

Age ranged from 20 to 44 years (M=28.0, SD=8.28). At the time of assessment, Body Mass Index (BMI) ranged from 13.67-21.03 (M=16.83, SD=2.32). Diagnostic status at the time of participation in the study was determined using information collected from administration of the EDE, and was based on DSM-IV (1994) criteria. Five participants (29%) met criteria for anorexia nervosa; four (23%) with restricting symptoms and one (6%) with binge/purge symptoms. Twelve (71%) participants met criteria for EDNOS. Using Thomas et al's., (2009) criteria as described in the Introduction (Section 1.2.1; Table 1.1) the EDNOS group can be further categorised into 4 subgroups. Five (29%) participants met criteria for partial anorexia nervosa (AN), four (24%) for non fat phobic AN, two (12%) for high weight AN and one (6%) AN with menses.

Most patients had previously received treatment for their eating disorder. Eight (47%) had received outpatient treatment alone, three (18%) had one or more previous

inpatient admissions to a specialist eating disorders unit, and six (35%) reported having no previous treatment for their eating disorder. On average participants reported having their eating disorder for 8.13 years (range 1-20 years). Length of current treatment ranged from 1 to 104 months (M=16.8 months, Median=12 months, SD=24.9).

Two participants reported alcohol use in the preceding 24 hours. Alcohol consumption in this period did not exceed 4.7 units. Two participants reported they were current recreational drug users, both reported using cannabis between 2-4 weeks prior to assessment. Twelve participants were on medication at the time of assessment. Medications included anti-depressants, anti-anxiety, gastro-intestinal, sleep tablets and prescribed vitamins. For more detailed reporting of medication status, please see Table F.1 Appendix F.

Complications reported by three participants during their mother's pregnancy or during their delivery, included distress during delivery e.g. breech birth, use of forceps; and specific health problems such as growth retardation.

3.2.2 Healthy Controls

Twenty-seven psychology students were recruited to the healthy control group. All participants met inclusion/exclusion criteria as outlined in section 2.4.1. No participants reported a head injury involving loss of consciousness, however 2 participants had previously suffered a mild concussion (Gennarelli, 1986).

In the healthy control group age ranged from 19 to 52 years (M=27.19, SD=10.55). At the time of assessment, Body Mass Index (BMI) ranged from 16.68-33.31 (M=23.68, SD=4.37). Two participants had a BMI <18.5, which is classified as underweight, 18 had a BMI 18.5-25, which is classified as healthy, four had a BMI 25-30, which is classified as overweight and three had a BMI>30, which is classified as obese (WHO, 1995, 2000).

Of the participants who reported that they were current alcohol drinkers, six (22%) participants reported alcohol use in the preceding 24 hours. Alcohol consumption in this period did not exceed 3.15 units for five of these participants. One participant had consumed 9 units approximately 15 hours before participating in the study. One unit of alcohol takes approximately one hour to metabolise (Evans, O'Brien & Chafetz, 1991), therefore within 15 hours she should have processed all units of alcohol consumed. Her neuropsychological test performance was not significantly lower than the healthy control group mean, thus her data was included in the final analysis. Seven participants were on medication at the time of assessment. For more detailed reporting of medication status, please see Table F.2 Appendix F.

Complications reported by six participants during their mother's pregnancy or during their delivery, included distress during delivery e.g. breech birth, use of forceps; premature birth, and specific health problems such as tachycardia.

To aid comparisons between groups demographic information for categorical data is presented in Table 3.1.

Table 3.1: Demographic Information for Categorical Data in Eating Disorder and Healthy Control Group.

	Eating Disorder Group n=17		Healthy Control Group n=27	
	n	*%*	*n*	*%*
Handedness				
Right	14	82	22	81
Left	2	12	4	15
Ambidextrous	1	6	1	4
Educational History				
High School	1	6	0	0
College	2	12	0	0
Undergraduate Degree	10	59	23	85
Postgraduate Qualifications	4	23	4	15
Occupational Status				
Professional	8	47	0	0
Student	6	35	27	100
Unemployed	3	18	0	0
Time Last Eaten				
Within 3 hours	10	59	24	89
3-6 hours ago	3	18	1	4
6-24 hours ago	4	23	2	7
Current Alcohol User	12	71	23	85
Current Drug User	2	12	0	0
Currently Taking Medication	12	71	7	26
Pre/Post Natal Complications	3	18	6	22

3.3. Comparison of Groups on Background and Psychological Variables

Fishers Exact tests were used to compare the eating disorder and control group on categorical variables which might have affected individuals' neurological status (See Table 3.1). The two groups did not differ in terms of handedness (p=0.587), incidence of mild concussion (p=0.504), birth complications (p=0.635), alcohol use (p=0.215), and drug use (p=0.144). The groups were significantly different in terms of medication use (p=0.004) with the eating disorder group reporting higher medication use than the healthy control group. From these tests it was concluded that the two groups were comparable in terms of handedness and possible pre-existing neurological damage as a result of drug or alcohol use and possible pre or post natal injury.

Independent samples t-tests were performed to determine whether the eating disorder and healthy control groups were comparable on continuous demographic variables. Table 3.2 details means, standard deviations, t-values, p-values and effect sizes. Results revealed that the eating disorder group had a significantly lower BMI than the healthy control group, however they did not differ in terms of age or premorbid intellectual ability as measured by the NART.

Table 3.2: Independent Samples *t*-tests Between Eating Disorder and Healthy Control Group, on Demographic Variables.

	Eating Disorder Group n= 17		Healthy Control Group n= 27		*t*	*p*	*d*
	Mean	*SD*	*Mean*	*SD*			
Age	28.00	8.28	27.19	10.55	0.27	0.789	0.08
BMI	16.83	2.32	23.68	4.37	5.79	<0.001**	1.81
NART errors	15.41	4.93	18.81	7.28	1.85	0.072	0.52
NART FSIQ	114.88	4.06	112.26	5.96	1.59	0.119	0.48

*Note: BMI=Body Mass Index, NART=National Adult Reading Test, FSIQ=Full-Scale intelligence Quotient. ** Indicates significant difference p<0.01.*

Further independent samples *t*-tests were performed to determine any group differences on psychological characteristics. Table 3.3 details means, standard deviations, *t*-values, *p*-values and effect sizes. Due to the number of comparisons conducted the use of Bonferroni's correction was applied. Bonferroni suggested when conducting multiple comparisons alpha should be divided by the number of tests conducted. Twenty-two comparisons were conducted. Dividing an alpha of 0.05 by 22 results in a significance level of 0.002. As expected, there were significant differences between the eating disorder and healthy control group on psychological variables. The eating disorder group displayed significantly higher eating pathology on all subscales of the EDE, significantly higher general psychiatric pathology on all subscales of the SCL-90, with the exceptions of hostility and phobic anxiety, and significantly more severe obsessive-compulsive symptoms as measured by the Y-BOCS. In addition, significant differences were noted in terms of social problem solving, with the eating disorder group reporting higher negative problem orientation than healthy controls.

Table 3.3: Independent Samples *t*-tests Between Eating Disorder and Healthy Control Group, on Psychological Variables.

	Eating Disorder Group n= 17		Healthy Control Group n= 27		*t*	*p*	*d*
	Mean	*SD*	*Mean*	*SD*			
Psychological Measures							
EDE							
Restraint	3.53	1.37	0.58	0.70	8.21	< 0.001**	2.92
Eating concern	2.96	1.27	0.19	0.27	8.91	< 0.001**	3.14
Shape concern	4.24	1.50	0.90	0.70	8.62	< 0.001**	3.10
Weight concern	3.22	1.81	0.65	0.64	5.64	< 0.001**	2.29
Global score	3.60	1.29	0.62	0.52	9.09	< 0.001**	3.33
SCL-90							
Somatisation	1.33	0.76	0.34	0.44	4.93	< 0.001**	1.70
Obsessive-compulsive	1.81	0.89	0.49	0.53	5.51	< 0.001**	1.91
Interpersonal sensitivity	1.84	0.86	0.41	0.39	6.47	< 0.001**	2.33
Depression	1.94	0.90	0.29	0.36	7.23	< 0.001**	2.65
Anxiety	1.32	0.74	0.16	0.21	6.30	< 0.001**	2.39
Hostility	0.61	0.46	0.32	0.45	2.03	0.049	0.64
Phobic anxiety	0.83	0.94	0.03	0.08	3.54	0.003	1.37
Paranoid ideation	0.91	0.67	0.24	0.31	3.85	0.001**	1.40
Psychoticism	0.82	0.43	0.08	0.20	6.57	< 0.001**	2.40
Global severity index	1.39	0.58	0.28	0.25	7.46	< 0.001**	2.72
YBOCS							
Total score	18.18	4.92	1.20	1.53	13.80	< 0.001**	5.20
SPSI-R							
Positive problem orientation	8.29	5.29	12.23	3.50	2.71	0.012	0.92
Negative problem orientation	22.12	8.76	10.81	5.94	4.67	< 0.001**	1.58
Rational problem style	38.47	18.34	41.65	13.42	0.66	0.515	0.21
Impulsivity/carelessness style	9.71	6.27	10.38	6.95	0.33	0.747	0.10
Avoidance style	10.82	6.35	7.42	4.78	2.00	0.052	0.63
Global score	10.84	3.47	13.25	2.35	2.50	0.019	0.85

** Indicates significant difference with application of Bonferroni's correction p<0.002.

3.4 Set Shifting Impairment

3.4.1 Hypothesis 1: The eating disorder group will have worse set shifting ability than a healthy control group.

Independent samples *t*-tests were performed to determine any statistically significant group differences on key neuropsychological measures of set shifting. Table 3.4 details means, standard deviations, *t*-values, *p*-values and effect sizes for key neuropsychological measures. Secondary analysis detailed in Table 3.5 reports means, standard deviations, *t*-values, *p*-values and effect sizes for additional neuropsychological measures. Due to the number of comparisons conducted in Table 3.5, the use of Bonferroni's correction was applied, resulting in a threshold significance level of 0.005.

Table 3.4: Independent Samples *t*-tests Between Eating Disorder and Healthy Control Group, on Key Neuropsychological Measures of Set Shifting Ability.

	Eating Disorder Group n= 17		Healthy Control Group n= 27		*t*	*p*	*d*
	Mean	*SD*	*Mean*	*SD*			
WCST Perseverative errors	16.59	11.57	7.59	5.39	3.01	0.007**	1.08
DK Trail Making test Number/letter switch	54.11	13.67	63.58	23.33	1.51	0.137	0.47
DK Verbal Fluency Letter	43.35	10.73	41.70	12.10	1.30	0.202	0.14
DK Colour Word Inhibition	45.55	8.85	44.08	8.93	0.55	0.582	0.17
Hayling Errors	1.59	1.84	1.56	1.53	0.06	0.949	0.02
Brixton Errors	12.06	4.10	11.85	3.38	0.18	0.856	0.06

Note: Higher scores on DK verbal fluency indicates better performance.
*** Indicates significant difference p<0.01*

Table 3.5: Independent Samples *t*-tests Between Eating Disorder and Healthy Control Group, on Additional Neuropsychological Measures of Set Shifting Ability.

	Eating Disorder Group n= 17		Healthy Control Group n= 27		*t*	*p*	*d*
	Mean	*SD*	*Mean*	*SD*			
WCST							
Trials administered	104.65	21.99	81.96	17.78	3.76	0.001**	1.16
Total error	30.18	18.27	14.96	11.49	3.07	0.005	1.05
Percent perseverative error	14.71	8.11	8.67	3.73	2.89	0.009	1.04
Set failure	1.00	1.17	0.15	0.46	2.86	0.010	1.05
Percent conceptual level responses	63.94	16.18	78.19	10.98	3.20	0.004**	1.08
Learning to learn	-2.80	6.18	-0.40	1.60	1.52	0.148	0.60
Delis-Kaplan							
Verbal Fluency							
Category	45.41	6.87	46.15	9.16	0.28	0.778	0.09
Switch total accuracy	14.53	3.02	13.11	3.61	1.35	0.185	0.42
Colour Word							
Inhibition/switch	49.65	7.44	51.65	7.10	0.89	0.381	0.28
Hayling							
Section 2 total time	15.53	15.49	17.70	16.56	0.43	0.666	0.13

*Note: ** Indicates significant difference with application of Bonferroni's correction p<0.005. Higher scores on the following tests indicate better performance: WCST percent conceptual level responses, learning to learn; DK verbal fluency all subscales. For learning to learn n=16 in eating disorder group, n=26 in healthy control group.*

Results revealed that the eating disorder group made significantly more perseverative errors on the WCST than the healthy control group. No significant differences were found between the eating disorder and healthy control groups on the other five key measures of set shifting ability (Table 3.4 and Figure 3.1). Secondary analysis revealed that the eating disorder group took more trials to complete the WCST and made fewer conceptual level responses than healthy controls (Table 3.5 and Figure 3.1). Reporting of effect sizes allows group differences to be quantified and emphasises the size of the difference regardless of whether analysis reveals statistically significant differences (Coe, 2002). Using Cohen's criteria (1969), effect

sizes on all WCST subscales with the exception of learning to learn demonstrated large effects. Learning to learn and Verbal Fluency switching accuracy demonstrated a medium effect size between groups, whereas Colour Word inhibition switch demonstrated a small effect size. Effect sizes between groups on other measures of set shifting ability were small to medium (Table 3.5).

Figure 3.1: WCST Performance in Eating Disorder and Healthy Control Group.
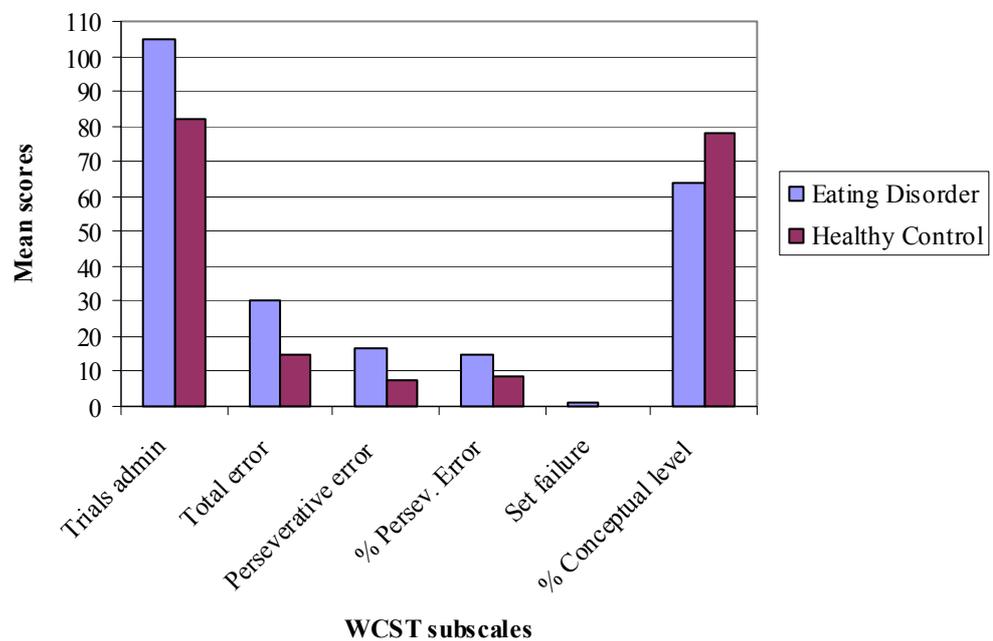
Note: Higher scores indicate poorer performance except on percent conceptual level responses.

Clinically significant impairments in set shifting ability were investigated by converting neuropsychological age normed scaled scores to *z*-scores, using means and standard deviations from published normative data. This allowed identification of impairments on a case by case basis by comparing an individual's z-score with an "expected" z-score, calculated on the basis of their estimated premorbid level of

intellectual ability (For a detailed description of how this was done see Appendix G).

Individual premorbid intellectual ability was estimated on the basis of NART scores.

Scores that fell at least one standard deviation below the expected level were defined

as clinically significant impairments. Results are demonstrated for key

neuropsychological measures of set shifting in Table 3.6.

Table 3.6: Number of Participants Falling Between 1–2 or > 2 Standard Deviations Below the Expected Range on Key Neuropsychological Measures of Set Shifting Ability.

	1-2 SD below expected n, (%)		>2 SD below expected n, (%)		Total n, (%)	
	ED	*Control*	*ED*	*Control*	*ED*	*Control*
WCST Perseverative error	5 (29)	3 (11)	3 (18)	0	8 (47)	3 (11)
Trail Making Test Number letter switch	0	3 (11)	0	1(4)	0	4 (15)
Verbal Fluency Letter	1 (6)	1 (4)	0	0	1 (6)	1 (4)
Colour Word Inhibition	1 (6)	3 (11)	0	0	1 (6)	3 (11)
Hayling Total	0	1 (4)	0	0	0	1 (4)
Brixton Total	2 (12)	1 (4)	0	0	2 (12)	1 (4)

Note: ED=Eating Disorder Group

Forty seven percent of the eating disorder group were found to have impairments on

the perseverative errors subscale of the WCST, the primary outcome measure for set

shifting. On other tests of set shifting the percentage of eating disorder participants

with impairments was considerably less. Further investigation of those with

impairments revealed that 9 (53%) patients had neuropsychological test scores that

fell at least 1 standard deviation below the level expected for them. Of those with deficits, three had a diagnosis of anorexia nervosa and six a diagnosis of EDNOS.

To summarise, the eating disorder group were found to have significantly worse set shifting ability than the healthy control group on the primary outcome measure, WCST number of perseverative errors. Forty seven percent of the eating disorder sample showed impaired performance on this measure. Overall 53% of the sample demonstrated performance at least one standard deviation below the level expected for them on tests of set shifting ability. Hypothesis 1 is therefore supported.

Further exploratory analyses relevant to Hypothesis 1 are reported in Section 3.6.

3.5 Relationship Between Set Shifting and Psychological Variables

3.5.1 Hypothesis 2: Poor set shifting ability will be associated with greater eating disorder severity in the eating disorder group.

Pearson's *r* correlations were calculated between key measures of set shifting and eating pathology as measured by BMI and EDE. Correlations were run for all EDE subscales and set shifting measures that were identified a priori as key variables. Results are displayed in Table 3.7. Scatterplots of the data can be seen in Appendix H (Figures H.1-H.12).

Table 3.7: Pearson's Correlations (*r*) Between Eating Pathology and Key Neuropsychological Measures of Set Shifting Ability, in the Eating Disorder Group, n=17.

		EDE Subscales				
	BMI	**Global**	**Restraint**	**Eating Concern**	**Shape Concern**	**Weight Concern**
WCST Perseverative error	0.118	0.243	0.475	0.301	0.108	0.092
DK Trail Making test Number/letter switch	-0.022	-0.559*	-0.466	-0.598*	-0.438	-0.437
DK Verbal Fluency Letter	0.272	0.102	0.206	-0.033	0.167	0.014
DK Colour Word Inhibition	0.165	0.560*	0.413	0.360	0.476	0.637**
Hayling Errors	-0.123	-0.186	-0.230	-0.274	-0.211	0.063
Brixton Errors	0.221	-0.131	-0.164	-0.432	-0.033	0.084

*Correlation is significant at the 0.05 level (2-tailed) ** Correlation is significant at the 0.01 level (2-tailed). †Indicates significant difference with application of Bonferroni's correction p<0.004

In the eating disorder group there were no significant correlations between any of the key measures of set shifting and BMI. There were significant negative correlations between performance on the DK Trail Making Test, number/letter switch and both the EDE eating concern subscale (*r* =-0.598, *p*=0.011) and the global EDE score (*r* =-0.559, *p*=0.020). As higher scores on the Trail Making Test indicate poorer performance, this inverse correlation indicates that higher levels of pathology were associated with better performance on the Trail Making Test. However this relationship was not significant with application of Bonferroni's correction.

Significant positive correlations were noted between performance on the DK Colour Word inhibition, weight concern (*r* =0.637, *p*=0.006) and global EDE score (*r* =0.560, *p*=0.019). This indicates an association between high eating pathology and

poor test performance on the Colour Word inhibition test. The same correlations were run in the healthy control group, and none were significant.

Overall there was not a consistent pattern of highly significant correlations between set shifting ability and measures of eating pathology. Hypothesis 2 is therefore not supported, as poorer set shifting ability was not associated with greater eating disorder severity in the eating disorder group.

3.5.2 Hypothesis 3: Greater severity of obsessive-compulsive symptoms will be associated with poor set shifting ability in the eating disorder group.

Pearson's *r* correlations were calculated between key measures of set shifting and severity of obsessive-compulsive symptoms as measured by the YBOCS total score. Results presented in Table 3.8 demonstrate a highly significant positive correlation between YBOCS total score and WCST perseverative errors in the eating disorder group (For scatterplot see Appendix H, Figure H.13).There were no significant correlations between any other key measures of set shifting and YBOCS total score. These correlations indicate that poorer performance on the WCST is associated with greater severity of obsessive-compulsive symptoms.

In the healthy control group there were no significant correlations between YBOCS total score and key measures of set shifting.

Table 3.8: Pearson's Correlations Between Severity of Obsessive-Compulsive Symptoms and Key Neuropsychological Measures of Set Shifting Ability, in the Eating Disorder Group, n=17.

	YBOCS Total score (r)	p
WCST Perseverative error	0.707	0.002**
DK Trail Making test Number/letter switch	0.135	0.604
DK Verbal Fluency Letter	0.416	0.097
DK Colour Word Inhibition	0.027	0.917
Hayling Errors	-0.399	0.112
Brixton Errors	-0.018	0.945

*** Correlation is significant at the 0.01 level (2-tailed)*

Hypothesis 3 is therefore supported, as greater severity of obsessive-compulsive symptoms in the eating disorder group was associated with poor scores on the primary outcome measure. However no other key measures of set shifting were correlated with obsessive-compulsive symptoms.

3.5.3 Hypothesis 4: Maladaptive social problem solving will be associated with poor set shifting ability in the eating disorder group.

Pearson's r correlations were calculated between key measures of set shifting and social problem solving. Adaptive social problem solving is measured by the subscales of positive problem orientation (PPO) and rational problem solving (RPS). Conversely, maladaptive problem solving is measured by negative problem orientation (NPO), impulsivity/carelessness style (ICS), and avoidance style (AS). Results presented in Table 3.9 demonstrate a highly significant positive correlation between Impulsivity/carelessness style (ICS) problem solving and number of perseverative errors on the WCST. In addition a significant negative correlation was

noted between Negative problem orientation (NPO) and number of errors on the Brixton test. There were no significant correlations between any other measures of set shifting and adaptive or maladaptive social problem solving scores (For scatterplots see Appendix H, Figures H.14-H.19).

In the healthy control group there was a significant correlation between avoidance style and DK Colour Word inhibition (r =0.402, p=0.021, n=26). There were no other significant correlations.

Table 3.9: Pearson's Correlations (r) Between Social Problem Solving and Key Neuropsychological Measures of Set Shifting Ability, in the Eating Disorder Group, n=17.

	PPO	NPO	RPS	ICS	AS
WCST Perseverative error	0.119	0.070	-0.056	0.706**	0.236
DK Trail Making test Number/letter switch	-0.087	-0.205	-0.092	-0.348	-0.046
DK Verbal Fluency Letter	0.283	-0.254	0.124	0.281	-0.194
DK Colour Word Inhibition	0.127	0.193	0.316	-0.075	0.020
Hayling Errors	-0.212	-0.276	-0.240	-0.369	0.133
Brixton Errors	0.054	-0.524*	-0.206	-0.096	-0.173

*Correlation is significant at the 0.05 level (1-tailed) ** Correlation is significant at the 0.01 level (1-tailed). †Indicates significant difference with application of Bonferroni's correction p<0.004.*

Overall there was not a consistent pattern of highly significant correlations between set shifting ability and maladaptive problem solving, though impulsivity/carelessness style problem solving was associated with poor performance on the WCST. Hypothesis 4 is therefore only partially supported.

3.6 Exploratory Analyses

3.6.1 Correlations Between Set Shifting Performance on Neuropsychological Tests

Pearson's *r* correlations were run in order to assess the relationship between performance on different neuropsychological measures of set shifting in the eating disorder group. Results (Table 3.10) revealed a significant correlation between number of perseverative errors on the WCST and letter fluency. In addition, number of errors on the Hayling and Brixton tests were significantly correlated. However with the application of Bonferroni's correction to address the issue of multiple comparisons, differences were no longer significant.

Table 3.10: Pearson's (*r*) Correlation Matrix for Neuropsychological Test Performance in Eating Disorder Group, n=17.

	WCST	TMT	VF Letter	CW Inhibition	Hayling	Brixton
WCST	1					
TMT	-0.177	1				
VF Letter	0.526*	0.198	1			
CW Inhibition	0.135	-0.206	-0.051	1		
Hayling	-0.276	0.209	-0.394	0.197	1	
Brixton	0.045	0.297	0.250	0.070	0.575*	1

Note: TMT=trail making test, VF=verbal fluency, CW=colour word
*Correlation is significant at the 0.05 level (2-tailed) ** Correlation is significant at the 0.01 level (2-tailed) †Indicates significant difference with application of Bonferroni's correction p<0.004.

3.6.2 Associations Between Set Shifting Performance and Mood

Additional Pearson's *r* correlations were run to investigate the relationship between anxiety and depression (as measured by subscales of the SCL-90) and key measures of set shifting ability. Results are presented in Table 3.11.

Table 3.11: Pearson's Correlations Between SCL-90 Anxiety and Depression and Key Neuropsychological Measures of Set Shifting Ability, in the Eating Disorder Group, n=17.

	SCL-90 Anxiety (*r*)	*p*	*SCL-90 Depression (r)*	*p*
WCST Perseverative error	0.608	0.010**	0.274	0.278
DK Trail Making test Number/letter switch	-0.453	0.068	-0.266	0.301
DK Verbal Fluency Letter	0.336	0.188	0.088	0.738
DK Colour Word Inhibition	0.475	0.054	0.355	0.163
Hayling Errors	-0.262	0.310	-0.049	0.851
Brixton Errors	-0.242	0.350	-0.172	0.509

*** Correlation is significant at the 0.01 level (2-tailed)*

In the eating disorder group, anxiety was significantly correlated with performance on the WCST, and was approaching significance with DK Colour Word inhibition. There were no significant correlations between any other measures of set shifting and SCL-90 Anxiety score. There were no significant correlations between any measures of set shifting and SCL-90 Depression score. In the healthy control group, there were no significant correlations between mood measures and key set shifting variables.

3.6.3 Differences in Set Shifting Ability after Controlling for Obsessive-Compulsive Symptoms and Anxiety

An analysis of covariance (ANCOVA) was performed, in order to determine whether there were any overall differences in set shifting ability between eating disorder participants and healthy controls when the effect of severity of obsessive-compulsive symptoms and anxiety were taken into account. Significant group differences were reported on WCST subscales of perseverative errors, trials administered, set failure and percent conceptual level responses (see Tables 3.5 & 3.6), thus these subscales

were entered into the analysis as dependent variables. YBOCS total and SCL-90 Anxiety were used as covariates because the eating disorder and healthy control group were found to differ on these variables and they were found to correlate with performance on the WCST (see Tables 3.8 & 3.10).

One participant in the healthy control group had scores that did not quite reach significance to qualify as outliers, however she demonstrated high scores on measures of eating pathology (EDE), severity of obsessive-compulsive symptoms (YBOCS) and anxiety (SCL-90). The issue of high scores on the YBOCS was raised with the individual following participation in the study and she indicated having previously received treatment for OCD. Given that the study's exclusion criteria only excluded participants currently undergoing treatment for psychological disorders her data was retained in the analysis. However her particular pattern of scores had significant influence on the ANCOVA analysis. When her data was included in the analysis, results revealed that when the effects of obsessive-compulsive symptoms and anxiety were partialled out, group differences lost significance on the following WCST subscales: perseverative errors (F (1,44)= 0.980, p=0.328); trials administered (F (1,44)= 0.182, p=0.672), set failure (F (1,44)=2.33, p=0.135) and percent conceptual level responses (F (1,44)=0.366, p=0.549).

When her data were removed from the analysis results revealed WCST perseverative errors ($F_{(1,43)}$= 4.27, p=0.046) continued to differ between the eating disorder and healthy control groups. However, group differences on WCST trials administered ($F_{(1,43)}$= 0.067, p=0.798), set failure ($F_{(1,43)}$=1.606, p=0.213) and percent conceptual level responses ($F_{(1,43)}$=2.075, p=0.158) lost significance.

Chapter 4: Discussion

Chapter 4: Discussion

4.1 Summary of Previous Research

The investigation of neuropsychological functioning in anorexia nervosa is a relatively new field that it is widely recognised as having the potential to advance our understanding of eating disorders at a clinical and theoretical level. Anorexia nervosa is not thought to be associated with widespread cognitive impairment (Southgate et al., 2006). However, patients with anorexia have consistently been reported to show impairments in the executive function of set shifting (Roberts et al., 2007a). Set shifting involves the ability to switch attention, allowing the individual to change their thinking or behaviour to adapt to the demands of the environment or situation they are in. It is thought that deficits in set shifting ability may be associated with characteristics commonly observed in this patient group such as obsessive thoughts and behaviours around eating, maladaptive problem solving and a rigid thinking style. Much of the preceding literature has been fraught with methodological difficulties and has largely reported on inpatient samples meeting strict diagnostic criteria for anorexia nervosa. Set shifting ability has not been widely investigated in outpatients. Given that "most" patients with anorexia nervosa are managed on an outpatient basis (NHS QIS 2006, NICE 2004), much of the preceding literature may not generalise to outpatient populations where up to 60% of patients meet diagnostic criteria for EDNOS rather than other eating disorders (Fairburn et al., 2007).

The current study aimed to investigate relationships between neuropsychological measures of set shifting ability and defined clinical and psychological variables in a community sample of outpatients presenting to specialist eating disorder services

with symptoms of anorexia nervosa. It also aimed to determine whether set shifting ability was poorer in the outpatient eating disorder group than in a healthy control sample. The main findings are discussed below.

4.2 Set Shifting Impairment

4.2.1 Hypothesis 1 – Summary of Results

Hypothesis 1: The eating disorder group will have worse set shifting ability than a healthy control group.

Results revealed that the eating disorder group had significantly worse set shifting ability than the healthy control group on the primary outcome measure, WCST number of perseverative errors. Forty seven percent of the eating disorder sample showed impaired performance on this measure. Overall 53% of the sample demonstrated performance at least one standard deviation below the level expected for them on tests of set shifting ability. Hypothesis 1 was therefore supported.

4.2.2 Hypothesis 1 – Comparison with Previous Research

As highlighted in the introduction, set shifting impairments have been consistently reported in individuals with anorexia across a range of neuropsychological measures (Roberts et al., 2007a). Wide variations in effect size have been observed depending on the neuropsychological measure employed. Roberts et al.'s (2007a) meta-analysis of set shifting ability reported estimated effect sizes ranging from 0.36 (for the Trail Making Test), to 0.62 (for the Wisconsin Card Sort Test, WCST).

4.2.2.1 Wisconsin Card Sort Test

The present study revealed significant differences between the eating disorder group and healthy controls in set shifting ability as measured by the WCST. Differences in the number of perseverative errors remained when the effects of anxiety and severity of obsessive-compulsive symptoms were controlled for. The WCST is considered primarily a test of set shifting ability, however, successful performance also involves the cognitive functions of inhibition, problem solving, attention, working memory, and categorisation (Miyake et al., 2000).

In the present study, the effect size for number of perseverative errors on the WCST was 1.08. This represents a very large effect and is somewhat greater than the pooled effect size reported by Roberts et al. (2007a). Other subscales of the WCST also demonstrated medium to large effect sizes. The findings of the current study are in agreement with a number of previous studies which have demonstrated impairments in set shifting as measured by performance on the WCST in patients with anorexia nervosa (Fassino et al., 2002; Koba, Shrie, & Nabeta, 2002; Ohrmann et al., 2004; Steinglass et al., 2006; Thompson, 1993).

Thompson's (1993) study is perhaps the most comparable to the present research in that it is one of the only other studies that included a purely outpatient sample. The study reported on 10 outpatients meeting DSM-III criteria for anorexia nervosa, and 10 matched healthy controls. Unfortunately the authors only report summary statistics and do not report test statistics for all comparisons, hence it is unclear on which subscales of the WCST the clinical group demonstrated impaired

performance. Roberts et al. (2007a) included the Thompson paper in their meta-analysis and report an effect size of 0.50 on the WCST perseverative errors subscale, which is somewhat smaller than that noted in the present study.

Steinglass et al. (2006) investigated performance on the WCST in 10 inpatients and 5 outpatients, meeting DSM-IV criteria for anorexia nervosa with the exception of low weight status (BMI's in their sample ranged from 16.7 to 20.2). They reported significantly worse performance in the group of patients with anorexia relative to healthy controls on subscales of total errors and perseverative errors. The inclusion of higher weight patients suggests set shifting impairments cannot be attributed to low weight status; and that findings may be generalised to patients not meeting strict diagnostic criteria for anorexia. Fassino et al. (2002) reported on 20 subjects with restricting anorexia, and 20 healthy controls. The admission status of these patients is unclear as the authors state the patients were attending a psychiatric clinic, but not if this was on an inpatient or outpatient basis.

Ohrmann et al's. (2004) study included 11 inpatients meeting DSM-IV criteria for anorexia nervosa, and 11 age and gender matched controls. Results indicated significant correlations between WCST performance on subscales of total trials and perseverative errors with glutamate/glutamine metabolism in the anterior cingulate cortex, implicating this region in set shifting. Other neurological studies have linked set shifting in the WCST to activation in the prefrontal cortex and ventral striatum (Monchi, Petrides, Petre, Worsley & Dagher, 2001; Shafritz, Kartheiser, & Belger, 2005); and to hypoperfusion in patients with anorexia (Lask, et al., 2005).

Two studies have reported no significant differences between patients with anorexia and healthy controls in set shifting ability, as measured by the WCST. Cavedini et al. (2004) reported no significant differences between a large sample of 59 inpatients meeting DSM-IV criteria for anorexia nervosa and 82 healthy controls on WCST, number of categories completed, total errors and perseverative errors. In the eating disorder group patients were excluded on the basis of multiple psychiatric diagnoses. The exclusion of patients with co-morbid anxiety, depression and OCD is likely to have impacted on test performance, and may explain why no significant differences in set shifting ability were demonstrated in this study.

In a longitudinal study Gillberg et al. (2007) reported on a group of 51 patients 10 years after adolescent onset of anorexia nervosa. Forty one patients participated in neuropsychological testing using a computerised version of the WCST, 10 years after onset of AN. At this time it is reported that of the overall group of 51 patients, three met diagnostic criteria for AN, and 11 met criteria for other disorders, including bulimia nervosa and EDNOS. The diagnostic status of the 41 patients who underwent testing with the WCST is not reported. Comparison of the 41 eating disorder patients with healthy controls did not demonstrate a significant difference in WCST performance, suggesting that patients are relatively unimpaired on the WCST in the longer term of anorexia after re-feeding and rehabilitation.

Overall, the findings of this study and much of the previous literature provide robust evidence for increased perseveration on the WCST, suggesting impairment in set shifting ability. Studies of anorexia that have combined neurological and

neuropsychological assessment, using the WCST, have implicated the anterior cingulate cortex, prefrontal cortex and ventral striatum as key brain areas involved in set shifting. This study is the first to confirm deficits in set shifting ability in a community sample of outpatients who presented with symptoms of anorexia, but largely met DSM-IV criteria for EDNOS (71%) rather than anorexia nervosa.

4.2.2.2 Trail Making Test

The present study did not reveal significant differences between the eating disorder group and healthy controls in set shifting ability as measured by the Delis-Kaplan Trail Making Test, Number/Letter Switch task. It is possible that this is because successful performance on the Trail Making Test relies on multiple cognitive domains, particularly the ability to divide attention, and set shifting is not necessarily the primary determinant of performance (Lezak et al., 2004).

A number of previous studies have investigated performance on the Trail Making Test in eating disorder populations with conflicting findings. Some studies have reported no significant differences between individuals with anorexia and healthy controls (Mathias & Kent, 1998; Murphy et al., 2002; Steinglass et al., 2006; Witt et al., 1985).

Touyz, Beumont & Johnstone (1986) investigated the presence of impairments on the Trail Making Test in patients with anorexia, which they defined as performance below the 10th percentile compared with age norms. They did not report significant impairments; however it may be that they applied too stringent a criterion, given that

impairments identified in other studies have been of a subtle nature. Bayless et al. (2002) reported mild impairments or failure to complete the test in approximately 22% of patients with anorexia.

Other studies have reported significant impairments on the Trail Making Test in individuals with anorexia (Ohrmann et al., 2004; Pendleton-Jones et al., 1991; Tchanturia et al., 2004a; 2004c; Thompson, 1993) and bulimia nervosa (Brand, Franke-Sievert, Jacoby, Markowitsch & Tuschen-Caffier, 2007). Impairments have been found to persist after controlling for the effect of depression (Thompson, 1993) and have been associated with childhood perfectionism (Tchanturia et al., 2004c). Roberts et al.'s (2007a) meta analysis included the Trail Making Test and reported a small pooled standardised mean difference of 0.36, relative to healthy controls, suggesting the presence of subtle impairments.

Patients' attentional abilities are known to improve with weight gain (Kingston et al., 1996). As only 9 of the 17 patients in the current study were underweight (BMI<17.5), this may explain why no significant differences were observed between the eating disorder and healthy control groups on the Trail Making Test, which is known to have a strong attention component.

In the present study, despite there being no statistically significant group differences, there was a medium effect size (0.47) reported for performance on the Trail Making Test, with the eating disorder group displaying better performance than healthy controls. No other studies have been identified which suggest improved attention in

anorexia nervosa. Good performance on tests of attention may indicate increased effort, as a result of underlying anxiety regarding test performance (Eysenck, Derakshan, Santos & Calvo, 2007). Patients in the current study had significantly higher levels of anxiety than healthy controls, and other studies have shown patients with anorexia to demonstrate enhanced effort on cognitive tests (Fassino et al., 2002; Kingston et al., 1996; Struup et al., 1986), which may explain the current findings.

4.2.2.3 Verbal Fluency

The present study did not reveal significant differences between the eating disorder and healthy control group in set shifting ability, as measured by the Delis-Kaplan Verbal Fluency task. Successful performance on Verbal Fluency requires set shifting ability, flexibility of thought and use of strategy to deal with the demands of the task (Lezak et al., 2004).

Previous research investigating performance on tests of Verbal Fluency in anorexia has demonstrated largely non-significant findings (Mathias & Kent, 1998; Murphy et al., 2002; Steinglass et al., 2006; Tchanturia et al., 2002; Tchanturia et al., 2004a). The results of this study thus add support to the previous literature suggesting patients with anorexia do not have impairments in Verbal Fluency.

4.2.2.4 Colour Word Inhibition Task (Stroop)

The present study did not reveal significant differences between the eating disorder group and healthy controls in set shifting ability, as measured by the Delis-Kaplan Colour Word inhibition task. Successful performance on the Colour Word inhibition

task requires coordination of set shifting, selective attention and response inhibition (Lezak et al., 2004).

Whilst the "Emotional Stroop" test has been investigated extensively in patients with eating disorders (Lee & Shafran, 2004), the standard format has not been widely used in patients with anorexia nervosa. In line with the findings of this study, two prior studies have reported no significant differences between patients with anorexia and controls (Key et al., 2006; Steinglass et al., 2006). Kingston et al. (1996) reported non-significant differences between performance on the Stroop Test in patients with anorexia and healthy controls, however there was a trend towards poor performance in the group of patients with anorexia with significantly more individuals demonstrating impairments on this task.

4.2.2.5 Hayling & Brixton

The present study did not reveal significant differences between the eating disorder group and healthy controls in set shifting ability as measured by the Hayling and Brixton tests. The Hayling test is considered a test of response inhibition and set shifting ability (Shallice & Burgess, 1998). The function of inhibition is related to set shifting as one may be required to deactivate or inhibit previous correct responses in order to accurately shift set (Miyake et al., 2000). However, people who are successful on the Hayling test have been shown to come up with a strategy for dealing with the demands of the task, such as looking around the room and naming objects. The use of such a strategy may overcome the need to inhibit primed responses (Shallice & Burgess, 1998). The Brixton test is considered primarily a

measure of set shifting, with the potential for participants to perseverate following rule changes. Successful performance requires the coordination of attention, working memory, planning, and inhibition (Shallice & Burgess, 1998).

No previous studies have been identified in the literature using the Hayling test in patients with eating disorders. Roberts et al. (2007a) reported that three studies have previously employed the Brixton test in patients with eating disorders (Tchanturia et al., 2004a; 2004c; Holliday et al., 2005). An average standardized effect size of 0.21, was reported with wide variation in effect size noted across studies. Studies which reported on patients with anorexia nervosa (Holliday et al., 2005; Tchanturia et al., 2004c) generally reported larger effect sizes than those reporting on participants with bulimia nervosa (Tchanturia et al., 2004a). However of the three published studies using the Brixton test, one did not report statistically significant differences (Holliday et al., 2005) and in two studies the 95% confidence interval for effect size overlapped with zero (Holliday et al., 2005; Tchanturia et al., 2004a). There is therefore some evidence to suggest a mild impairment in set shifting ability as measured by the Brixton test, however this is far from robust. The findings of the present study do not provide support for a mild impairment in set shifting ability as measured by the Brixton test in outpatients with eating disorders.

4.2.2.6 General Discussion in Relation to Hypothesis 1

The results of this study did not demonstrate impairments in set shifting ability in the eating disorder group on all neuropsychological measures employed. The WCST was the only one in which impaired performance was observed. Exploratory analysis

revealed a lack of correlation between performances on the separate measures of set shifting in the eating disorder group. There is some evidence in the literature of dissociation in performance among executive tasks in neuropsychiatric, amnesic and Korsakoff's patients (Crockett, Bilsker, Hurwitz & Kozak, 1986; Kopelman, 1991; Shoqeirat, Mayes, MacDonald, Meudell & Pickering, 1990). Similar to the results of the current study, individuals may perform poorly on one executive task (for example set shifting) but perform normally on a related task (such as inhibition). Individual difference studies using exploratory factor analysis have demonstrated low intercorrelations among different executive tasks, which are often non-significant (Miyake et al., 2000). The differences in test performance may be related to the different demands of each task. While all tests selected are considered measures of set shifting ability, they all incorporate additional cognitive functions (Lezak et al., 2004).

By definition, executive functions involve the coordination and employment of multiple cognitive processes to deal with novel stimuli. Thus tests of executive function often tap multiple cognitive resources and are considered less "pure" measures than non executive ones (Burgess, 1997). Failure or difficulty on any one task of set shifting does not therefore imply impairment in that one function. This is referred to as the "task impurity" problem (Burgess, 1997). To overcome the task impurity problem clinically a wide test battery is used to formulate the pattern of impairment, and inform interpretation of an individual's test results. In a research setting this is not practical due to time constraints and the use of a hypothesis driven approach. It is therefore possible that participants may have had non-executive

cognitive impairments, for example of working memory, which could have influenced their performance on the executive tasks used. However the literature reviewed suggests that anorexia nervosa is not generally associated with widespread cognitive impairment, and every effort was taken to ensure the sample selected were free from neurological insult through the use of use of rigorous inclusion/exclusion criteria.

Many of the tests used in the study of neuropsychological functioning were developed to assess the effects of neurological trauma or illness but have been used increasingly for psychiatric populations where impairments are likely to be less severe (Tchanturia et al, 2005). It is possible therefore that the tests available may not be sensitive enough to discriminate or pick up on subtle deficits present in psychiatric disorders (Kuelz et al., 2004). For this reason, Keefe (1995) states that you may expect to see significant results on only the most difficult tests employed. Indeed, in the present study significant differences were seen only on the WCST, which could be considered to be the most difficult test due to its length and lack of clear instruction.

One of the major obstacles in examining executive functioning is the need to structure a situation in which patients can show whether and how they can make a structure for themselves (Lezak et al., 2004). The WCST does this well, providing enough instruction for participants to be able to begin the test, but not enough that they know what to expect or how to adapt to the changing demands of the test. The Brixton test is thought to be equivalent to the WCST, however in the current study

no impairments were noted in the eating disorder group and performance between the two measures was not correlated. This may in part be due to the different instructions given prior to the test. The Brixton test is more structured, including information that the pattern of blue circles will change without warning, and that the participant must try and work out the new pattern. This limits the difficulty of the task and may reduce the demand on executive functioning required to complete the task.

This study reported that 47% of the eating disorder group had impairments in set shifting ability on the WCST. This is in keeping with previous literature, where impairments in selective attention and inhibition have been identified in approximately 40% of patients (Kingston et al., 1996; Lauer, 2002). The method of identification of impairments used in the current study is an improvement on previous research (e.g. Mathias & Kent, 1998) as impairments were based on expected level of functioning predicted from premorbid intellectual ability rather than against mean normative scores. The level of impairment seen in the current study is thus likely to be an accurate reflection of that present in the outpatient population of eating disorder patients with anorexic symptoms.

4.3 Relationship Between Set Shifting and Psychological Variables

4.3.1 Hypothesis 2 – Summary of Results

Hypothesis 2: Poor set shifting ability will be associated with greater eating disorder severity in the eating disorder group.

Overall there was not a consistent pattern of highly significant correlations between set shifting ability and measures of eating pathology (BMI and EDE scores). Hypothesis 2 was therefore not supported, as poor set shifting ability was not associated with greater eating disorder severity in the eating disorder group.

4.3.2 Hypothesis 2 – Comparison with Previous Research

The suggestion that there may be a relationship between severity of impairment on set shifting tasks and severity of illness implies that impairments are not fixed and vary as a function of disease, i.e. that they are state rather that trait characteristics. In relation to BMI, there is some evidence in the literature for a relationship between severity of anorexia nervosa and set shifting ability, suggesting impairments may be state dependent. In a large sample of 41 inpatients meeting DSM-IV criteria for anorexia nervosa, BMI on admission was shown to be correlated with attention and flexibility (Kingston et al., 1996). In addition number of previous hospital admissions was significantly related to poorer performance on a test of cognitive flexibility. Further evidence that set shifting ability may be state dependent is that neuropsychological functioning has been shown to improve over the course of inpatient treatment (Moser et al., 2003); and set shifting impairments have not been

reported in patients 10 years after adolescent onset of anorexia nervosa, following refeeding and rehabilitation (Gillberg et al., 2007).

However there is also evidence from studies of patients who have recovered from anorexia to suggest that impaired set shifting may be a trait characteristic. Tchanturia et al. (2002) compared 14 patients with a diagnosis of anorexia to 16 patients recovered from anorexia. Results revealed similar performance on tests of set shifting between these two groups. Holliday et al. (2005) compared 23 women who had fully recovered from anorexia nervosa with 24 acutely ill women. Neuropsychological results were largely similar on tests of set shifting; the only significant difference reported was that the recovered group made fewer errors on the Trail Making Test. This was not unexpected as successful performance on the Trail Making Test requires the ability to divide attention, which has been shown to improve with weight gain (Kingston et al., 1996). Unaffected sisters of patients with anorexia nervosa have also been shown to have impairments in set shifting ability. These studies provide evidence to suggest impairments may be trait characteristics, which increase vulnerability to the development of AN (Holliday et al., 2005).

BMI as an indicator of disease severity has not been found to correlate with cognitive impairment related to set shifting on a range of neuropsychological tests, in inpatients with anorexia nervosa (Bayless, et al., 2002; Fassino et al., 2002; Gillberg et al., 2007; Holliday et al., 2005; Mathias & Kent, 1998; Tchanturia et al., 2004a). However, the relationship between eating pathology and set shifting ability has not been previously investigated in an outpatient sample. In keeping with a number of

other studies, the present research did not report significant correlations between BMI and set shifting ability.

Body mass index is a rather crude measure of severity, and does not take psychological status into account. Psychological measures of disease severity (such as the EDE and Eating Disorder Inventory, EDI) have also been investigated in relation to their ability to predict cognitive impairment. In keeping with the results of the current study no significant correlations have been reported between WCST performance (Fassino et al., 2002; Steinglass et al., 2006) and eating pathology as measured by the EDI. The results of the current study thus add to a growing evidence base to suggest that impairments in set shifting ability may represent a risk factor for anorexia nervosa and are likely to be trait rather than state dependent.

4.3.3 Hypothesis 3 – Summary of Results

Hypothesis 3: Greater severity of obsessive-compulsive symptoms will be associated with poor set shifting ability in the eating disorder group.

Results demonstrated an association between greater severity of obsessive-compulsive symptoms and poorer scores on the primary outcome measure (perseverative errors, WCST) in the eating disorder group. Hypothesis 3 was therefore supported. However no other key measures of set shifting were found to be correlated with obsessive-compulsive symptoms.

Additional exploratory analyses revealed that when the effects of obsessive-compulsive symptoms and anxiety were taken into account, WCST perseverative errors continued to differ between the eating disorder and healthy control groups.

4.3.4 Hypothesis 3 – Comparison with Previous Research

The findings of the current study suggest an association between greater severity of obsessive-compulsive symptoms and set shifting impairment in outpatients with symptoms of anorexia nervosa. This finding is not in keeping with studies that have reported no significant correlations between set shifting performance and OCD symptoms as measured by the Maudsley Obsessive-Compulsive Inventory (MOCI), despite the eating disorder groups in these studies demonstrating increased obsessionality compared with healthy controls (Holliday et al., 2005; Tchanturia et al., 2002; 2004a).

The current study found that group differences on number of perseverative errors on the WCST remained significant after partialling out the effects of anxiety and severity of obsessive-compulsive symptoms. However, this finding should be interpreted with caution given that the inclusion of an outlying participant and log transformation of the data had a significant impact on the analysis. Previous literature has demonstrated that effects of obsessive-compulsive symptoms cannot account for impairments in set shifting ability in inpatients with eating disorders (Tchanturia et al., 2002; Tchanturia et al., 2004a). However, this is the first time that this relationship has been tentatively demonstrated in a community sample of outpatients with symptoms of anorexia.

No studies have been identified which directly compare performance between patients with anorexia and obsessive-compulsive disorder, in neuropsychological tests of set shifting. However, associations between neuropsychological functioning, and childhood and adult obsessive-compulsive personality traits was investigated in patients with acute anorexia, patients currently in long term recovery and healthy controls (Tchanturia et al., 2004c). The authors utilised the EATATE semi structured interview which assesses obsessive-compulsive personality traits including perfectionism, inflexibility, rule driven behaviour, drive for order and symmetry, excessive doubt and cautiousness. Childhood perfectionism, rigidity and inflexibility were found to be associated with performance on the Trail Making and Brixton tests, which the authors suggest provides support for set shifting impairments being trait characteristics which may represent a vulnerability factor for the development of anorexia nervosa. This is further evidenced by the finding that unaffected sisters of patients with anorexia nervosa have higher levels of obsessive-compulsive symptoms than healthy unrelated women (Holliday et al., 2005).

Pendleton-Jones et al. (1991) state that the idea that changes in neuropsychological functioning may be "due to depression," and that partialling out the effects of depression, will leave the changes that are "due to the eating disorder," reflects a fundamental misconception about eating disorders. They assert that depressive symptoms are part of the eating disorder, rather than representing some separate unrelated pathology. The same could be said for obsessive-compulsive symptoms, and thus the analysis applied may be somewhat simplistic in providing an

explanation for the relationship between severity of obsessive-compulsive symptoms and set shifting impairments in this population.

4.3.5 Hypothesis 4 – Summary of Results

Hypothesis 4: Maladaptive social problem solving will be associated with poor set shifting ability in the eating disorder group.

Overall there was not a consistent pattern of highly significant correlations between set shifting ability and maladaptive problem solving, though impulsivity/carelessness style problem solving was associated with poor performance on the WCST. Hypothesis 4 was therefore only partially supported.

4.3.6 Hypothesis 4 – Comparison with Previous Research

The findings of the current study demonstrated a significant correlation between impulsivity/carelessness style problem solving and impaired set shifting ability on the WCST in the eating disorder group. No other significant correlations were reported between maladaptive problem solving and set shifting. No previous studies have been identified in the literature on eating disorders investigating social problem solving in combination with neuropsychological testing.

Research in schizophrenia has demonstrated a link between performance on tests of executive functioning including Verbal Fluency, Design Fluency and sustained attention (but not set shifting on the WCST) and social problem solving skills as measured by a video based test (Zanello, Perrig & Huguelet, 2006). The authors

suggest that the deficits observed in social problem solving may relate to social anxiety and theory of mind impairment. Another review study in schizophrenia demonstrated a relationship between sustained attention and social problem solving (Green, 1996). Set shifting ability as measured by the WCST and other card sorting tasks predicted community functioning but not social problem solving (Green, 1996).

Effective problem solving is associated with enhancement of a person's ability to cope with stress, whereas ineffective problem solving is associated with poor coping (D'Zurilla & Chang, 1995). Disordered eating may be a manifestation of maladaptive coping (e.g. Troop et al., 1994); indeed in the current study comparisons between the eating disorder and healthy control groups on self reported social problem solving revealed the eating disorder group displayed a significantly more negative orientation to problems than healthy controls. A negative orientation to problems is associated with inhibited cognitive processes, a tendency to perceive problems as threatening rather than challenging, low frustration tolerance and self-inefficacy (D'Zurilla & Chang, 1995). This finding is in keeping with previous literature reporting on inpatients with anorexia nervosa (Paterson, Power, Yellowlees, Park & Taylor, 2007; Paterson et al., In Submission; Swanson et al., In Press).

High impulsivity/carelessness style problem solving is characterised by the use of quick fix solutions, which tend to be narrowed, impulsive, hurried, careless and incomplete (D'Zurilla & Chang, 1995). In keeping with the findings of Swanson et al. (In Press) this style of problem solving was not predominant in the eating disorder sample in the current study. It has been suggested that individuals with anorexia have

an obsessive and perfectionist cognitive set (Bornstein, 2001; Kaye, Bastriani & Moss, 1995) and this finding adds support to this view.

In opposition to the findings of the current study, previous literature has demonstrated inpatients with anorexia to have higher impulsivity/carelessness style problem solving that normal controls (Paterson et al., 2007; In Submission). Despite the fact that in the current study there were no significant differences in impulsivity/carelessness style between the eating disorder and healthy control groups, a significant correlation was observed between set shifting performance on the WCST and impulsivity/carelessness style problem solving in the eating disorder group. This suggests that set shifting impairments may facilitate the use of impulsive approaches to problem solving.

Low self-esteem and a pervasive sense of ineffectiveness have been cited as contributory factors in the development and maintenance of anorexia (McLaughlin, Karp & Herzog, 1985; Silverstone, 1990). Tafarodi & Swann, (1995) propose a two dimensional theory of self-esteem, made up of self-liking and self-competence. Self-liking includes the concepts of self worth and conformity whereas self-competence is related to self-appraisal, efficacy and confidence in ones ability. The self-competence component of self-esteem has been shown to correlate with eating pathology (Paterson et al., 2007); and to mediate the relationship between social problem solving (specifically negative problem orientation and avoidance style), and eating pathology (Paterson et al., 2007; In Submission). This indicates that a sense of

inefficacy in patients with eating disorders is likely to impact on their ability to problem solve, and therefore inhibit successful coping.

Clinical perfectionism has also been associated with anorexia nervosa (Shafran et al., 2002). It is suggested that people who are clinical perfectionists react to failure to meet their standards with self-criticism. If standards are met they are re-evaluated as not being sufficiently demanding. Thus, perfectionism contributes to low self-competence. Perfectionism has been shown to be inversely related to social problem solving, with high levels of perfectionism associated with the use of maladaptive problem solving (Chang, 2002). This provides further support for the role of self esteem in effective problem solving.

There is some evidence that maladaptive problem solving in eating disorder samples continues to exist beyond recovery (Ghaderi & Scott, 2000) and beyond discharge from inpatient treatment despite improvement in eating pathology (Bloks et al., 2001). This suggests that similar to set shifting impairments, maladaptive problem solving may be a trait rather than a state characteristic.

In the current study self-esteem was not measured and it is therefore not possible to examine the relationship between self-esteem, set shifting ability and social problem solving. It may however be tentatively hypothesised than impairments in set shifting ability could contribute to the use of maladaptive problem solving strategies, which could impact on the maintenance of a sense of inefficacy and poor self competence. Given that the analysis in the present study was correlational, and therefore does not

implicate a causal relationship, confirmation of this hypothesis requires further exploration.

4.4 Strengths and Limitations

This study is the first to investigate the relationship between neuropsychological test performance on measures of set shifting ability and psychological characteristics in a community sample of outpatients presenting to specialist eating disorder services with symptoms of anorexia nervosa. The use of an outpatient sample increases the generalisability of the findings, given that most patients with eating disorders are managed on an outpatient basis. The study employed rigorous methodology, including detailed recording of drug and alcohol use, previous head injury, peri-natal complications, current medication, and educational history. A broad range of reliable and valid measures of executive functioning were used to assess cognition. Further methodological strengths include the way the data was collected and analysed. For example the effect size for the prospective power calculation was derived from meta analysis rather than using Cohen's (1988) standardised effect sizes, and statistical analysis using ANCOVA allowed for control of confounding variables (Allison, Allison, Faith, Paultre, & Pi-Sunyer, 1997).

Several methodological limitations must be taken into account in the overall consideration of the research. Firstly it was impossible for the investigator to be blind to group due to the different geographical locations used for recruitment of patients and healthy controls, and because of the physical effects of anorexia nervosa. However every effort was taken to standardise the research procedure for all

participants. Secondly all the neuropsychological tests used were pen and paper versions administered by the investigator, which may leave more room for experimental bias and error. Computerised versions of the WCST, Stroop Test, Verbal Fluency and Trail Making Test are available and their use may have reduced measurement error. All tests selected for use in the study are reliable and valid measures and it is unlikely that use of non-computerised tests would have a significant impact on the findings of the study. Tests were all administered in the same order. An improvement in the methodology would be to counterbalance test order to reduce effects of confounding variables such as anxiety at the beginning of testing or fatigue at the end.

Whilst the study achieved a good sample size in the context of previous literature, the analysis was underpowered according to the a priori power calculation, especially the analyses in relation to Hypotheses 2, 3 and 4. Achieving sufficient statistical power is recognized as being difficult for research in the field of anorexia nervosa (Brocklehurst, Elbourne, Garcia & McCandlish, 1996). Multi-centre methodology was used in the current study to increase the sample size and reduce the influence of site idiosyncrasies (Armstrong & Droter, 1997).

In terms of sampling method, one limitation in the selection of the healthy control group was that participants were not asked about current dieting behaviour. In non eating disordered participants dieting has been associated with impairments in attention, word recall, reaction time and working memory (Bosanac et al., 2007). It has also been associated with impaired performance on a vigilance task, with poorer

immediate memory and slower reaction times (Green, Rogers, Elliman, & Gatenby, 1994; Green & Rogers, 1995; 1998). Given that up to 80% of British women report their current dieting status as "watching their weight" or "trying to lose weight" (Wardle & Johnstone, 2002), it is likely the healthy control group may have included some current dieters.

4.5 Clinical Implications

The findings of the current study have significant clinical implications, as they demonstrate for the first time the presence of set shifting impairments in a sample of outpatients with eating disorders, who do not meet strict diagnostic criteria for anorexia nervosa. This adds support to the assertion that set shifting impairments may represent an endophenotype for the development of eating disorders (Holliday et al., 2005), with anorexia nervosa representing the severe end of a particular spectrum of disturbed eating. The findings of this study have implications for the way eating disorders are conceptualised and defined, and provide support for the reclassification of anorexia nervosa along the lines suggested by Thomas et al. (2009), (see Section 1.2.1; Table 1.1). Dropping the amenorrhea criterion and increasing the weight criterion, would place more of an emphasis on the core psychopathology of eating disorders. With the upcoming publication of DSM-V this is a highly relevant issue and will have ramifications on the future research and treatment of eating disorders with anorexic symptoms.

The literature suggests approximately a third of individuals with anorexia are impaired on cognitive tests (Kingston et al., 1996; Lauer 2002). The current study

identified clinically significant set shifting impairments in 53% of the eating disorder group. Given that cognitive impairments are noted in a large number of patients presenting with eating disorders, the question of whether neuropsychological testing should be included in the routine assessment of eating disorders is pertinent. Centres of clinical excellence such as the Institute of Psychiatry, Kings College London, routinely conduct neuropsychological assessments prior to inpatient admission for anorexia nervosa. The findings of the current study suggest that it may be beneficial to include such assessment more widely in outpatients who present to specialist eating disorder services. Neuropsychological assessment has been shown to contribute to the understanding of psychiatric illness in relation to predictors of course of illness, diagnostic classification, and aids to treatment strategies (Keefe, 1995). The ability to tailor treatment to an individuals' particular profile of cognitive function and dysfunction is likely to improve the efficacy of psychological intervention (Keefe, 1995). Indeed, many therapeutic modifications have been suggested to overcome impairments in attention and concentration, memory, executive functioning, and language difficulties in the context of brain injury (Tyerman & King, 2004).

Patients with anorexia nervosa are notoriously ambivalent about treatment and existing inpatient treatments show large rates of dropout, with figures ranging from 20-50% (Kahn & Pike, 2004; Surgenor, Magiure & Beaumont, 2004; Vandereycken & Pierloot, 1983; Woodside, Carter & Blackmore, 2004). Many patients are chronically symptomatic and relapse is common (Pike, 1998). The presence of cognitive impairments, including a lack of flexibility in thinking style may make it

difficult for patients to reflect on the nature and seriousness of their problems, and hinder ability to participate in cognitive therapies. It has been suggested that Cognitive Remediation Therapy (CRT) could be a useful precursor to engagement in more complex cognitive therapies (Davies & Tchanturia, 2005). Cognitive remediation therapy aims to stimulate mental activities, improve set shifting ability and flexibility of thinking, and has the potential to improve patients' management and daily functioning. Contrary to other cognitive therapies, CRT aims to change the process of thinking rather than content. Davies & Tchanturia, (2005) suggest that CRT treatment may be beneficial in the treatment of acute anorexia nervosa, when other therapies may be too emotionally distressing and cognitively demanding. Participation in CRT at this time would allow the individual to begin working towards recovery without having to address any of the cognitive or behavioural aspects of their eating disorder, which may be advantageous given the common resistance to engagement in treatment and denial of the seriousness of low weight status (Davies & Tchanturia, 2005).

Given the recency of the discovery of set shifting impairments in patients with anorexia nervosa, there are no published randomised controlled studies of CRT in this population. A model of CRT was developed by Delahunty & Morice (1993) for the treatment of Schizophrenia. This model involves individual executive functioning training and has been shown to have some efficacy in schizophrenic populations (Wykes, Reeder, Corner, Williams, & Everitt, 1999; Wykes, Reeder, Williams, & Corner, 1999; Wykes & van der Gaag, 2001). Davies & Tchanturia, (2005) adapted Delahunty & Morice's CRT model for use with anorexia nervosa, specifically

focusing on improving the executive functions of flexibility, working memory and planning. They describe a single N case study in which results indicated a marked improvement in set shifting ability following ten sessions of CRT, with treatment gains still observable at 6 month follow up. In a follow up study Tchanturia, Davies & Campbell (2007) conducted CRT with four patients with anorexia nervosa, and demonstrated medium to large improvements in set shifting ability, following ten 45-minute sessions of CRT.

Given that set shifting impairments are thought to predate the onset of anorexia nervosa, the early identification and treatment of such deficits could potentially reduce the severity of the disorder (Lena et al., 2004). This observation taken in combination with the findings of the current study suggests the use of CRT may be beneficial for use in outpatient as well as inpatient eating disorder populations.

The current study indicated maladaptive problem solving was more prevalent in the eating disorder group. The role of low self-competence and clinical perfectionism was highlighted. Treatment programmes that aim to improve self-efficacy, modify approaches to problems, enhance actual problem solving skills, and reduce the use of avoidance are therefore worthy of consideration. Bloks et al. (2001) found that patients with eating disorders reported significant improvements in coping style after inpatient treatment, however at discharge they were still using more avoidance and passive style coping strategies than normal controls. A coping style that included reassuring thoughts predicted better outcome at six month follow up, however Bloks et al. note that patients attending follow up were those who were higher weight at

discharge and showed better psychological adjustment. The results of the current study suggest that focused treatment on the use of positive problem solving strategies, may be beneficial during outpatient treatment.

4.6 Theoretical Implications

The findings of the current study have significant theoretical implications. Impaired set shifting ability may represent a general risk factor for psychopathology, as set shifting impairments have been reported in a wide range of psychiatric conditions including patients with OCD (Kulez et al., 2004), Bipolar Disorder (Robinson et al., 2006), Schizophrenia (Ceaser et al., 2008) Autism and ADHD (Happé & Frith, 1996). The literature reviewed has demonstrated set shifting impairments in patients with anorexia nervosa at different stages of illness and following recovery. The present study demonstrates the presence of impairments in outpatients who do not meet strict diagnostic criteria for anorexia, and highlights parallels between cognitive functioning in eating disorders and obsessive-compulsive disorder.

Within eating disorder populations set shifting has been shown to predate and underlie the development of the disorder (Lena et al., 2004). It is has been suggested by Tchanturia et al. (2004c) that impairments in set shifting in anorexia nervosa may occur as a result of malnutrition in adolescence, which could interfere with frontal lobe development. Lena et al. (2004) state that the fact that identified impairments in set shifting are subtle should not be downplayed, given their potential developmental influence. A number of authors have argued that premorbid cognitive deficits affect the development of self-esteem, identity formation, social functioning, problem

solving and development of autonomy in adolescence, which may increase the risk of developing an eating disorder (Fox & Mahoney, 1998; Lena et al., 2004; Peck, 1981; Roman, 1998).

Connan et al. (2003) propose a neurodevelopmental model for anorexia nervosa which brings together genetic, perinatal and developmental factors. They suggest genetic and early attachment experiences have a critical role in the development of neurobiological systems regarding stress response. The serotenergic system has been found to be abnormal in patients with anorexia (Frank et al., 2001) and has been strongly implicated in the regulation of impulsivity and cognitive inflexibility (Clarke, Dalley, Crofts, Robbins & Roberts, 2004; Winstanley, Theobold, Dalley, Gennon & Robbins, 2004). Persistent neural abnormalities have been reported following recovery from anorexia nervosa including structural abnormalities (Katzman, Zipursky, Lambe, & Mikulis, 1997) and abnormal activation of the orbitofrontal cortex and anterior cingulate in response to food cues (Uher et al., 2003). This provides evidence for a neurological component in anorexia nervosa. The findings of the study indicate impaired set shifting ability, which has been linked to activity in the prefrontal cortex and ventral striatum (Monchi et al., 2001; Shafritz et al., 2005) and thus add support to this view.

In relation to the findings regarding severity of obsessive-compulsive symptoms, the present study demonstrated significant associations between obsessionality and set shifting, providing further support for a Neurocognitive model of anorexia based on comparisons with OCD. Steinglass & Walsh, (2006) proposed such a model, in

which they suggest that patients with anorexia may have a premorbid disturbance in implicit learning, which makes them vulnerable to over learning dieting behaviours, which are then difficult to extinguish. This in combination with biological and neurological changes contributes to the persistence of the disorder.

4.7 Future Research

The current study highlights many avenues for future research. Given the difficulties in achieving sufficient statistical power in eating disorder projects, collaborative studies are indicated. Increasing the sample size would increase the chance of detecting small or medium cognitive impairments.

The current study initially planned to include an additional group of inpatients with anorexia nervosa to allow direct comparisons between inpatient and outpatient samples. This would be likely to improve the current study. It would also be interesting to include an additional clinical group of patients with OCD. A further modification of the research would be conducting analyses on the basis of diagnostic status. For example, comparing EDNOS patients with symptoms of anorexia to patients who meet strict diagnostic criteria for anorexia. As EDNOS patients with symptoms of anorexia nervosa have demonstrated subtle differences in eating pathology (Thomas et al., 2009), if a large enough sample were collected it would be interesting to see if there were any differences in cognitive impairment across subtypes.

As set shifting impairments have been identified in approximately a third of patients, and cannot be predicted on the basis of clinical characteristics, further investigation of the cause of set shifting difficulties is warranted. One way of doing this would be to compare two groups of patients with anorexia nervosa who present with and without set shifting impairments.

In the current study self-esteem was not measured and it is therefore not possible to examine the relationship between self-esteem, set shifting ability and social problem solving. However in light of the tentative hypothesis described earlier it would be interesting to repeat this study including a mutli-component measure of self-esteem

4.8 Conclusion

The current study is the first to investigate the relationship between neuropsychological test performance on measures of set shifting ability and psychological characteristics in a community sample of outpatients presenting to specialist eating disorder services with symptoms of anorexia nervosa.

Seventeen female outpatients recruited from multi-site specialist eating disorder services and 27 university students participated in the study, which involved clinical interview, neuropsychological testing, and completion of psychological questionnaires. Results revealed significant impairments in set shifting ability in 53% of the eating disorder sample. Severity of obsessive-compulsive symptoms and impulsivity/carelessness style problem solving were associated with impaired set shifting ability in the eating disorder group. The eating disorder group performed

significantly worse on some measures of set shifting than the healthy control group, with these differences not explained by the effects of anxiety or obsessive-compulsive symptoms alone.

The set-shifting impairments observed in around half of the outpatient eating disorder group indicates that routine neuropsychological screening should be considered at any early stage for such patients. Promising recent interventions, such as Cognitive Remediation Therapy, are available which could reduce the impact of difficulties associated with such impairments and potentially reduce eating disorder symptomatology.

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Appendix A: Letters of Ethical Approval

Appendix B: Participant Information Sheets

Appendix C: Consent Form

Appendix D: Non-Parametric Analyses

Appendix D: Non-Parametric Analyses

Table 3.2.D: Mann-Whitney tests for skewed variables between eating disorder and healthy control group, on demographic variables.

	Eating Disorder Group n= 17		Control Group n= 27		*U*	*z*	*p*
	Mean	*SD*	*Mean*	*SD*			
Age	28.00	8.28	27.19	10.55	186.0	-1.06	0.290
NART FSIQ	114.88	4.06	112.26	5.96	161.5	-1.65	0.100

Note: NART=National adult reading test, FSIQ=full-scale intelligence quotient.

Table 3.3.D: Mann-Whitney tests between eating disorder and healthy control group, on psychological variables.

	Eating Disorder Group n= 17		Healthy Control Group n= 27		*U*	*z*	*p*
	Mean	*SD*	*Mean*	*SD*			
Psychological Measures							
EDE							
Restraint	3.53	1.37	0.58	0.70	12	-5.27	< 0.001**
Eating concern	2.96	1.27	0.19	0.27	2.5	5.59	< 0.001**
Shape concern	4.24	1.50	0.90	0.70	15	5.18	< 0.001**
Weight concern	3.22	1.81	0.65	0.64	28.5	4.87	< 0.001**
Global score	3.60	1.29	0.62	0.52	7	5.37	< 0.001**
SCL-90							
Somatisation	1.33	0.76	0.34	0.44	68.5	3.89	< 0.001**
Obsessive compulsive	1.81	0.89	0.49	0.53	46.5	4.43	< 0.001**
Interpersonal sensitivity	1.84	0.86	0.41	0.39	27	4.91	< 0.001**
Depression	1.94	0.90	0.29	0.36	28	4.87	< 0.001**
Anxiety	1.32	0.74	0.16	0.21	20.5	5.09	< 0.001**
Hostility	0.61	0.46	0.32	0.45	121.5	2.66	0.008
Phobic anxiety	0.83	0.94	0.03	0.08	39	5.16	< 0.001**
Paranoid ideation	0.91	0.67	0.24	0.31	79	3.71	< 0.001**
Psychoticism	0.82	0.43	0.08	0.20	30	5.0	< 0.001**
Global severity index	1.39	0.58	0.28	0.25	27	5.47	< 0.001**
YBOCS							
Total score	18.18	4.92	1.20	1.53	9.5	5.36	< 0.001**
SPSI-R							
Positive problem orientation	8.29	5.29	12.23	3.50	117.5	2.58	0.010
Negative problem orientation	22.12	8.76	10.81	5.94	61.5	3.97	< 0.001**
Rational problem style	38.47	18.34	41.65	13.42	208	0.32	0.747
Impulsivity/carelessness style	9.71	6.27	10.38	6.95	211	0.25	0.803
Avoidance style	10.82	6.35	7.42	4.78	146	-1.87	0.062
Global score	10.84	3.47	13.25	2.35	122.5	2.48	0.014

** Indicates significant difference with application of Bonferroni's correction p<0.002.

Table 3.4.D: Mann-Whitney tests between eating disorder and healthy control group,

on key neuropsychological measures of set shifting ability.

	Eating Disorder Group n= 17		Healthy Control Group n= 27		*U*	*z*	*p*
	Mean	*SD*	*Mean*	*SD*			
WCST Perseverative error	16.59	11.57	7.59	5.39	92.5	3.32	0.001**
DK Trail Making test Number/letter switch	54.11	13.67	63.58	23.33	187.5	1.01	0.311
DK Verbal Fluency Letter	43.35	10.73	41.70	12.10	193	0.881	0.378
DK Colour Word Inhibition	45.55	8.85	44.08	8.93	193	0.880	0.379
Hayling Errors	1.59	1.84	1.56	1.53	220.5	0.224	0.823
Brixton Errors	12.06	4.10	11.85	3.38	207.5	0.533	0.594

Note: High scores on DK verbal fluency indicates good performance.
*** Indicates significant difference p<0.01*

Table 3.5.D: Mann-Whitney tests between eating disorder and healthy control group, on additional neuropsychological measures of set shifting ability.

	Eating Disorder Group n= 17		Healthy Control Group n= 27		U	z	p
	Mean	*SD*	*Mean*	*SD*			
WCST							
Trials administered	104.65	21.99	81.96	17.78	87.5	3.44	0.001*
Total error	30.18	18.27	14.96	11.49	87	3.44	0.001*
Percent perseverative error	14.71	8.11	8.67	3.73	99	3.16	0.002*
Set failure	1.00	1.17	0.15	0.46	118	3.35	0.001*
Percent conceptual level responses	63.94	16.18	78.19	10.98	87	3.44	0.001*
Learning to learn	-2.80	6.18	-0.40	1.60	125.5	2.15	0.032
Delis-Kaplan							
Verbal Fluency							
Category	45.41	6.87	46.15	9.16	221	-0.205	0.837
Switch total accuracy	14.53	3.02	13.11	3.61	171.5	1.41	0.160
Colour Word							
Inhibition/switch	49.65	7.44	51.65	7.10	182	1.15	0.252
Hayling							
Section 2 total time	15.53	15.49	17.70	16.56	189	0.978	0.328

*Note: ** Indicates significant difference with application of Bonferroni's correction $p<0.005$. Higher scores on the following tests indicate better performance: WCST percent conceptual level responses, learning to learn; DK verbal fluency all subscales. For learning to learn n=16 in eating disorder group, n=26 in healthy control group.*

Table 3.7.D: Spearman's rho correlations (r_s) between eating pathology and key neuropsychological measures of set shifting ability, in the eating disorder group, n=17.

		EDE Subscales				
	BMI	**Global**	**Restraint**	**Eating Concern**	**Shape Concern**	**Weight Concern**
WCST Perseverative error	0.069	0.066	0.260	0.217	-0.010	0.020
DK Trail Making test Number/letter switch	0.041	-0.584*	-0.401	-0.539*	-0.413	-0.409
DK Verbal Fluency Letter	0.194	0.007	0.050	-0.098	0.047	0.050
DK Colour Word Inhibition	0.326	0.587*	0.341	0.310	0.570*	0.661**
Hayling Errors	-0.093	-0.130	-0.196	-0.237	-0.064	-0.035
Brixton Errors	0.333	-0.023	-0.163	-0.364	0.008	0.087

Correlation is significant at the 0.05 level (2-tailed)
** Correlation is significant at the 0.01 level (2-tailed)

Table 3.8.D: Spearman's rho correlations between severity of obsessive compulsive symptoms and key neuropsychological measures of set shifting ability, in the eating disorder group, n=17.

	YBOCS Total score (r_s)	***p***
WCST Perseverative error	0.754	< 0.001**
DK Trail Making test Number/letter switch	0.021	0.937
DK Verbal Fluency Letter	0.370	0.144
DK Colour Word Inhibition	0.021	0.937
Hayling Errors	-0.332	0.193
Brixton Errors	0.002	0.992

** Correlation is significant at the 0.01 level (2-tailed)

Table 3.9.D: Spearman's rho (r_s) between social problem solving and key neuropsychological measures of set shifting ability, in the eating disorder group, n=17.

	PPO	NPO	RPS	ICS	AS
WCST Perseverative error	0.057	0.157	0.021	0.608**	0.155
DK Trail Making test Number/letter switch	-0.165	-0.266	-0.033	-0.387	-0.022
DK Verbal Fluency Letter	0.514*	-0.327	0.172	0.088	-0.036
DK Colour Word Inhibition	0.102	0.106	0.226	0.034	0.016
Hayling Errors	-0.170	-0.362	-0.090	-0.466	0.066
Brixton Errors	-0.004	-0.539*	-0.176	-0.212	-0.122

Correlation is significant at the 0.05 level (1-tailed)
*** Correlation is significant at the 0.01 level (1-tailed)*

Table 3.10.D: Spearman's rho (r_s) Correlation Matrix for Neuropsychological Test Performance in Eating Disorder Group, n=17.

	WCST	TMT	VF Letter	CW Inhibition	Hayling	Brixton
WCST	1					
TMT	0.058	1				
VF Letter	0.338	0.064	1			
CW Inhibition	0.081	-0.186	-0.097	1		
Hayling	-0.223	0.464	-0.331	0.152	1	
Brixton	-0.072	0.330	0.159	0.123	0.675**	1

Note: TMT=trail making test, VF=verbal fluency, CW=colour word
Correlation is significant at the 0.05 level (2-tailed)
*** Correlation is significant at the 0.01 level (2-tailed)*

Table 3.11.D: Spearman's rho (r_s) correlations between SCL-90 anxiety and depression and key neuropsychological measures of set shifting ability, in the eating disorder group, n=17.

	SCL-90 Anxiety (*r*)	***p***	**SCL-90 Depression (r)**	***p***
WCST Perseverative error	0.471	0.056	0.173	0.507
DK Trail Making Test Number/letter switch	-0.612	0.009**	-0.264	0.306
DK Verbal Fluency Letter	0.261	0.312	0.078	0.765
DK Colour Word Inhibition	0.300	0.242	0.449	0.071
Hayling Errors	-0.395	0.116	-0.050	0.849
Brixton Errors	-0.280	0.276	-0.022	0.932

*** Correlation is significant at the 0.01 level (2-tailed)*

Appendix E: Log Transformation (to the Base 10) Analyses

Appendix E: Log Transformation to the base 10 Analyses

Table 3.2.E: Independent samples *t*-tests between eating disorder and healthy control group, on log transformed demographic variables.

	Eating Disorder Group n= 17		Control Group n= 27		*t*	*p*
	Mean	*SD*	*Mean*	*SD*		
Age	1.43	0.12	1.41	0.15	0.52	0.605
NART FSIQ	2.06	0.01	2.05	0.02	1.75	0.087

Note: BMI=body mass index, NART=National adult reading test, FSIQ=full-scale intelligence quotient.

Table 3.3.E: Independent samples *t*-tests between eating disorder and healthy control group, on log transformed psychological variables.

	Eating Disorder Group n= 17		Healthy Control Group n= 27		*t*	*p*
	Mean	*SD*	*Mean*	*SD*		
Psychological Measure						
EDE						
Restraint	0.636	0.139	0.165	0.165	9.77	< 0.001**
Eating concern	0.573	0.160	0.065	0.087	12.05	< 0.001**
Shape concern	0.698	0.153	0.254	0.143	9.74	< 0.001**
Weight concern	0.583	0.204	0.190	0.147	6.91	< 0.001**
Global score	0.524	0.184	0.332	0.333	10.97	< 0.001**
SCL-90						
Somatisation	0.342	0.165	0.109	0.144	5.52	< 0.001**
Obsessive compulsive	0.424	0.155	0.151	0.142	6.02	< 0.001**
Interpersonal sensitivity	0.433	0.138	0.135	0.105	8.12	< 0.001**
Depression	0.466	0.156	0.099	0.099	9.03	< 0.001**
Anxiety	0.343	0.146	0.057	0.069	7.55	< 0.001**
Hostility	0.190	0.120	0.105	0.108	2.43	0.019
Phobic anxiety	0.221	0.189	0.010	0.031	4.54	< 0.001**
Paranoid ideation	0.256	0.155	0.082	0.099	4.13	< 0.001**
Psychoticism	0.248	0.109	0.029	0.062	7.51	< 0.001**
Global severity index	0.365	0.121	0.100	0.078	8.86	< 0.001**
YBOCS						
Total score	1.267	0.123	0.323	0.368	12.29	< 0.001**

*** Indicates significant difference with application of Bonferroni's correction p<0.002.*

Table 3.4.E: Independent samples *t*-tests between eating disorder and healthy control group, on log transformed key neuropsychological measures of set shifting ability.

	Eating Disorder Group n= 17		Healthy Control Group n= 27		*t*	*p*
	Mean	*SD*	*Mean*	*SD*		
WCST Perseverative error	1.124	0.302	0.814	0.220	3.94	<0.001**
DK Trail Making Test Number/letter switch	1.720	0.107	1.780	0.140	1.47	0.147
Hayling Errors	0.312	0.305	0.335	0.258	0.27	0.778

** Indicates significant difference p<0.01

Table 3.5.E: Independent samples t-tests between eating disorder and healthy control group, on additional log transformed neuropsychological measures of set shifting ability.

	Eating Disorder Group n= 17		Healthy Control Group n= 27		*t*	*p*
	Mean	*SD*	*Mean*	*SD*		
WCST						
Trials administered	2.010	0.095	1.911	0.081	3.89	<0.001**
Total error	1.397	0.286	1.099	0.233	3.78	<0.001**
Percent perseverative error	1.110	0.230	0.908	0.157	3.48	0.001**
Set failure	0.239	0.234	0.040	0.118	3.25	0.004**
Percent conceptual level responses	1.790	0.127	1.890	0.073	2.90	0.008
Learning to learn	1.170	0.339	1.290	0.036	1.45	0.166
Delis-Kaplan						
Verbal Fluency						
Category	1.650	0.067	1.675	0.078	0.200	0.842
Colour Word						
Inhibition/switch	1.69	0.063	1.718	0.074	1.26	0.215
Hayling						
Section 2 total time	0.908	0.565	1.14	0.323	1.45	0.136

Note: ** Indicates significant difference with application of Bonferroni's correction p<0.005. Higher scores on the following tests indicate better performance: WCST percent

conceptual level responses, learning to learn; DK verbal fluency. For learning to learn n=16 in eating disorder group, n=26 in healthy control group.

Table 3.7.E: Pearsons' correlations (*r*) between log transformed eating pathology and log transformed key neuropsychological measures of set shifting ability, in the eating disorder group, n=17.

	EDE Subscales				
	Global	**Restraint**	**Eating Concern**	**Shape Concern**	**Weight Concern**
WCST † Perseverative error	0.183	0.261	0.288	0.084	0.050
DK Trail Making test † Number/letter switch	-0.545*	-0.446	-0.607**	-0.408	-0.434
DK Verbal Fluency Letter	0.120	0.164	-0.048	0.215	-0.012
DK Colour Word Inhibition	0.453	0.385	0.330	0.380	0.589*
Hayling † Errors	-0.269	-0.187	-0.270	-0.288	-0.041
Brixton Errors	-0.202	-0.199	-0.411	-0.101	0.040

*Correlation is significant at the 0.05 level (2-tailed)
** Correlation is significant at the 0.01 level (2-tailed)
† Indicates neuropsychological variables that have been log transformed.

Table 3.8.E: Pearsons' correlations between log transformed severity of obsessive compulsive symptoms and log transformed key neuropsychological measures of set shifting ability, in the eating disorder group, n=17.

	YBOCS Total score (*r*)	***p***
WCST Perseverative error †	0.648	0.005**
DK Trail Making test Number/letter switch †	0.102	0.698
DK Verbal Fluency Letter	0.345	0.175
DK Colour Word Inhibition	0.022	0.932
Hayling Errors †	-0.361	0.155
Brixton Errors	-0.054	0.836

** Correlation is significant at the 0.01 level (2-tailed)
† Indicates neuropsychological variables that have been log transformed.

Table 3.9.E: Pearsons' correlations (*r*) between social problem solving and log transformed key neuropsychological measures of set shifting ability, in the eating disorder group, n=17.

	PPO	**NPO**	**RPS**	**ICS**	**AS**
WCST Perseverative error	0.054	0.101	-0.116	0.660**	0.248
DK Trail Making test Number/letter switch	-0.064	-0.213	-0.040	-0.419	-0.041
Hayling Errors	-0.132	-0.321	-0.131	-0.471	-0.020

*** Correlation is significant at the 0.01 level (1-tailed)*

Table 3.10.E: Pearsons' (*r*) Correlation Matrix for log transformed Neuropsychological Test Performance in Eating Disorder Group, n=17.

	WCST	**TMT**	**Hayling**
WCST	1		
TMT	-0.052	1	
Hayling	-0.197	0.373	1

Note: TMT=trail making test

Table 3.11.E: Pearsons' (*r*) correlations between log transformed SCL-90 anxiety and depression and log transformed key neuropsychological measures of set shifting ability, in the eating disorder group, n=17.

	SCL-90 Anxiety (*r*)	***p***	***SCL-90 Depression (r)***	***p***
WCST Perseverative error †	0.561	0.019*	0.320	0.211
DK Trail Making test Number/letter switch †	-0.472	0.056	-0.231	0.373
DK Verbal Fluency Letter	0.321	0.209	0.082	0.755
DK Colour Word Inhibition	0.372	0.142	0.287	0.263
Hayling Errors †	-0.345	0.175	-0.103	0.694
Brixton Errors	-0.301	0.240	-0.232	0.370

**Correlation is significant at the 0.05 level (2-tailed)*
† Indicates neuropsychological variables that have been log transformed.

3.6.3.E Differences in Set Shifting Ability after Controlling for Obsessive-Compulsive Symptoms and Anxiety

ANCOVA analysis with log transformed WCST subscales, log transformed Anxiety and Depression.

When the outlying data was included in the analysis, results revealed that when the effects of obsessive-compulsive symptoms and anxiety were partialled out, group differences lost significance on the following WCST subscales: perseverative errors ($F_{(1,44)}= 1.422$, $p=0.240$); trials administered ($F_{(1,44)}= 1.919$, $p=0.174$), set failure ($F_{(1,44)}=1.898$, $p=0.176$) and percent conceptual level responses ($F_{(1,44)}=0.049$, $p=0.825$).

When the outlying data were removed from the analysis results revealed that when the effects of obsessive-compulsive symptoms and anxiety were partialled out, group differences lost significance on the following WCST subscales: perseverative errors ($F_{(1,43)}= 0.137$, $p=0.713$); trials administered ($F_{(1,43)}= 0.704$, $p=0.406$), set failure ($F_{(1,43)}=0.989$, $p=0.326$) and percent conceptual level responses ($F_{(1,43)}=0.106$, $p=0.746$).

Appendix F: Medication Status

Appendix F: Medication Status

Table F.1: Medication in Eating Disorder Group n=12

Medication	n
Anti-depressants	
Fluoexitine	5
Citalopram	2
Sertraline	1
Venlafaxine	1
Anti-anxiety	
Diazepam	2
Beta-blockers	2
Sleep	
Temazepam	1
Valium	1
Zolpidem	1
Gastro-intestinal	
Ranitidine	2
Domperidone	1
Vitamins	
Multivitamin	3
Calcium	1
Sando-K (potassium)	1
Other	
Lamotrogene	1
Cocodamol	1
Omeprazole	1

Table F.2: Medication in Healthy Control Group n=7

Medication	n
Antihistamine	2
Amytriptaline	1
Cuprafen	1
Insulin	1
Prochlorperazine	1
Thyroxine	1

Appendix G: Calculation of Neuropsychological Impairments

Appendix G: Calculation of Neuropsychological Impairments

NART estimated full scale IQ scores are based on mean score of 100 and a standard deviation of 15. Given that one standard deviation equals 15 IQ points and 1 standard deviation of a z score is equal to 1 it is possible to work out the z score which corresponds to each IQ point (15/1=0.066). This is demonstrated in Table F.1 for IQ scores from 99 to 130. For each individual person their expected z score was taken from their estimated IQ. For example an individual with an IQ of 110 would be expected to achieve a test score, equivalent to a z of 0.67, for that person a score of 1.67 would be one standard deviation above their expected range and a score of - 1.67 would be one standard deviation below their expected range.

Table F.1: NART Estimated Full Scale IQ and Corresponding z Scores.

IQ	99	100	101	102	103	104	105	106	107	108	109
Z	-0.07	0	0.07	0.14	0.20	0.27	0.34	0.4	0.47	0.54	0.6
IQ	110	111	112	113	114	115	116	117	118	119	120
Z	0.67	0.74	0.8	0.87	0.94	1	10.7	1.13	1.20	1.27	1.33
IQ	121	122	123	124	125	126	127	128	129	130	
Z	1.40	1.47	1.54	1.6	1.67	1.74	1.8	1.87	1.94	2	

Appendix H: Scatterplots

Appendix H: Scatterplots

In Relation to Hypothesis 2

Figure H.1: BMI and WCST Perseverative Errors in Eating Disorder Group n=17
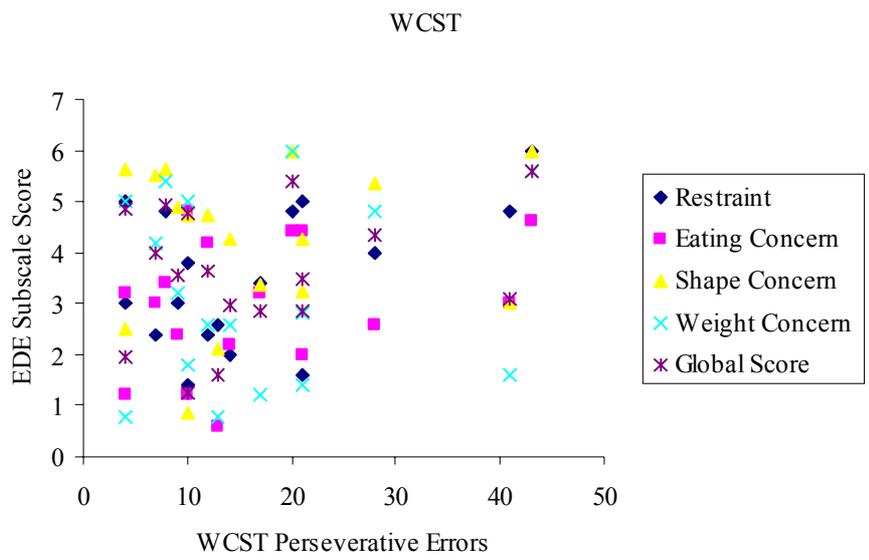
WCST
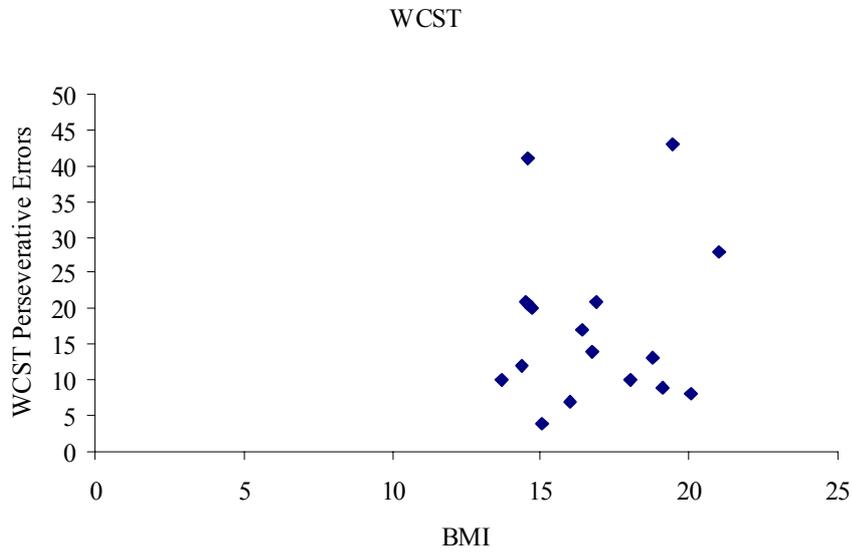
Figure H.2: BMI and Delis-Kaplan Trail Making Test Number Letter Switch in Eating Disorder Group n=17
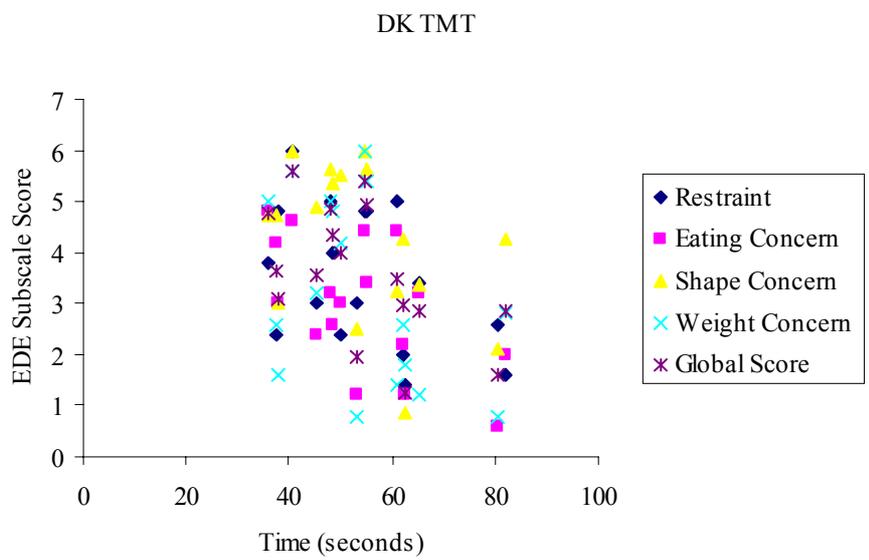
DK TMT
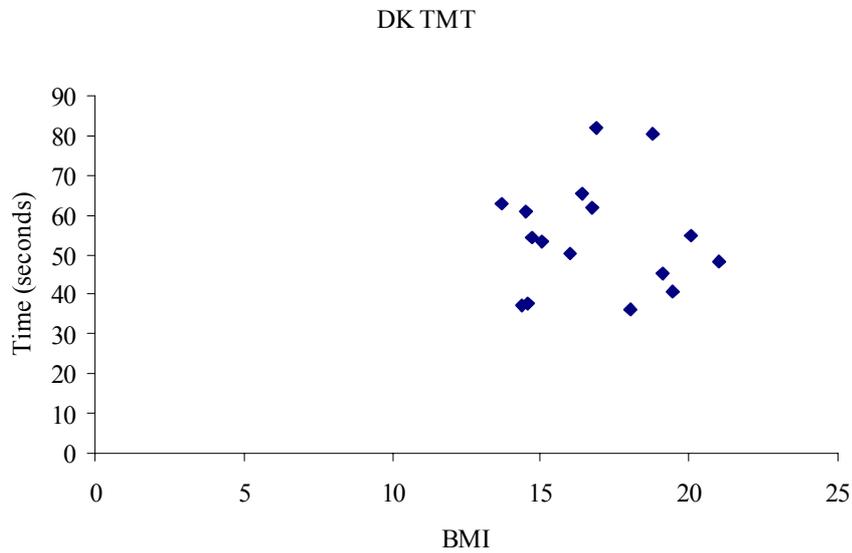
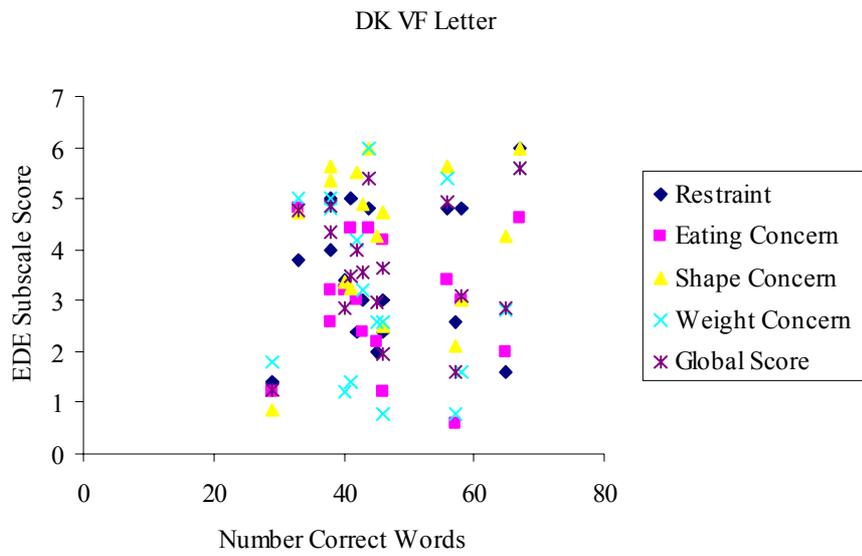**Figure H.3:** BMI and Delis-Kaplan Verbal Fluency Letter in Eating Disorder Group n=17

DK VF Letter
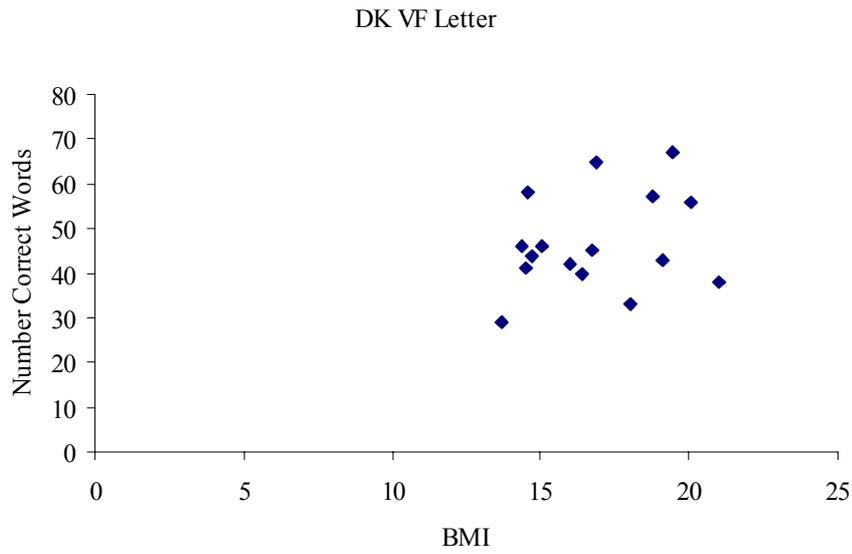
Figure H.4: BMI and Delis-Kaplan Colour Word Inhibition (Stroop) in Eating Disorder Group n=17

DK CW Inhibition (Stroop)
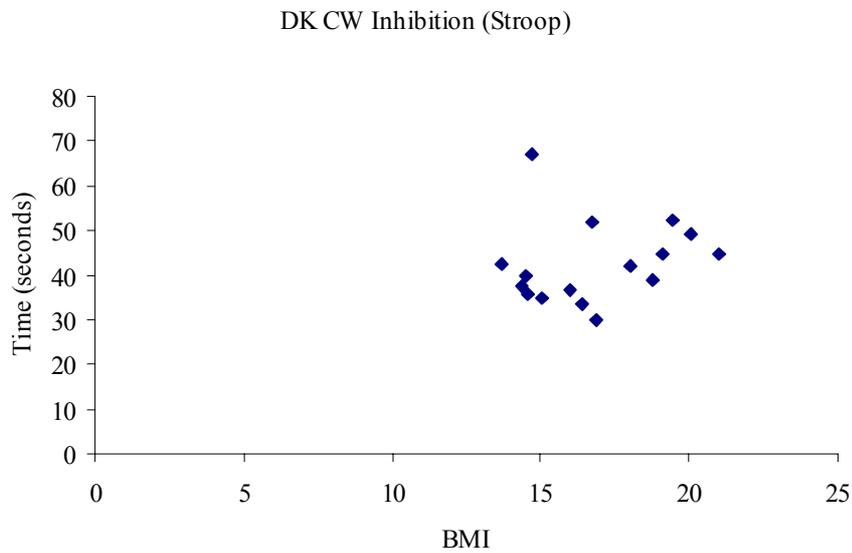
Figure H.5: BMI and Hayling Test in Eating Disorder Group n=16

Hayling
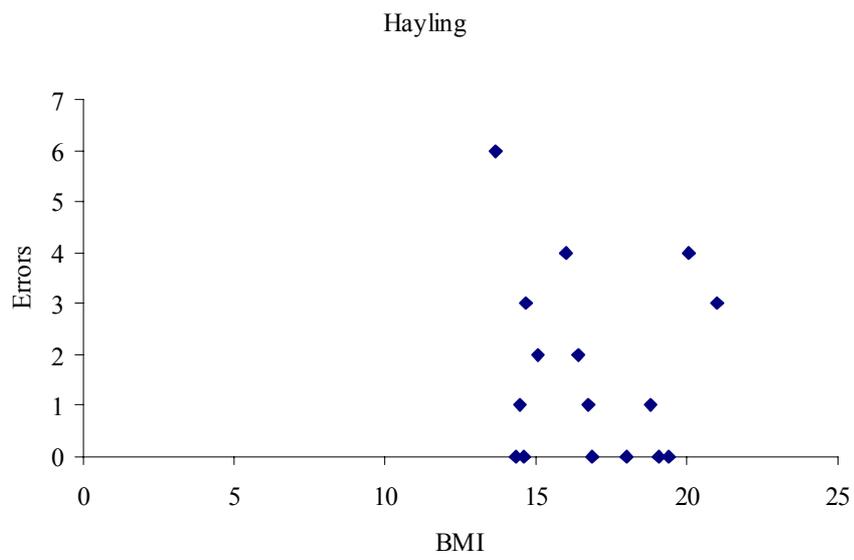
Figure H.6: BMI and Brixton Test in Eating Disorder Group n=16

Brixton
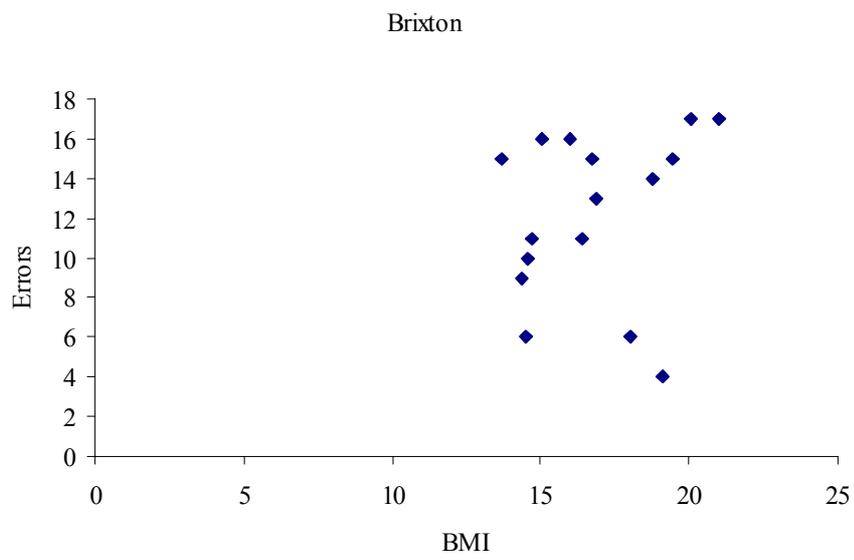
Figure H.7: EDE and WCST Perseverative Errors in Eating Disorder Group n=17

WCST

Figure H.8: EDE and Delis-Kaplan Trail Making Test Number Letter Switch in Eating Disorder Group n=17

DK TMT

213

Figure H.9: EDE and Delis-Kaplan Verbal Fluency Letter in Eating Disorder Group
n=17

DK VF Letter

Figure H.10: EDE and Delis-Kaplan Colour Word Inhibition (Stroop) in Eating
Disorder Group n=17

DK CW Inhibition (Stroop)
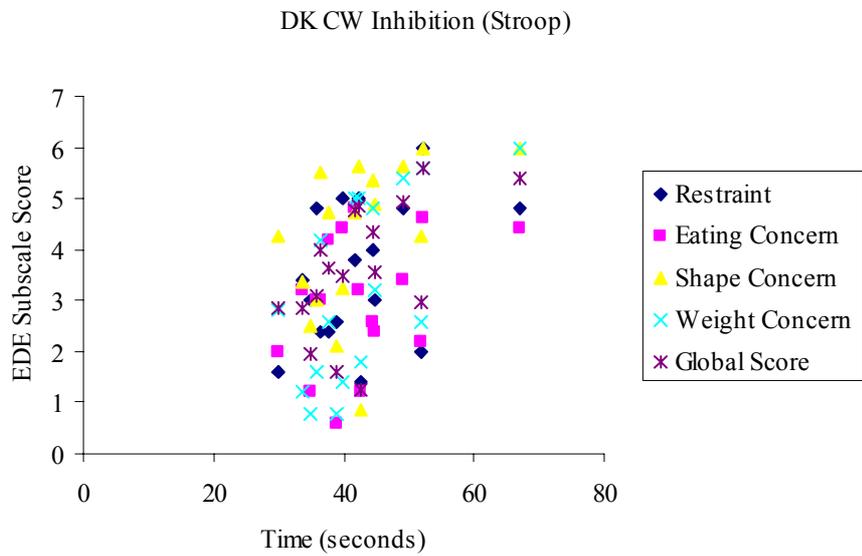
Figure H.11: EDE and Hayling Test in Eating Disorder Group n=17

Hayling
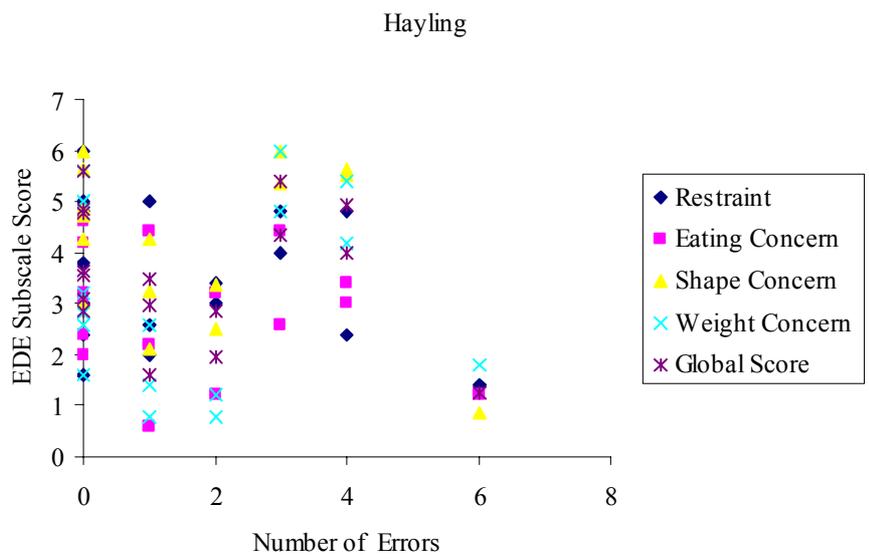
Figure H.12: EDE and Brixton Test in Eating Disorder Group n=17

Brixton
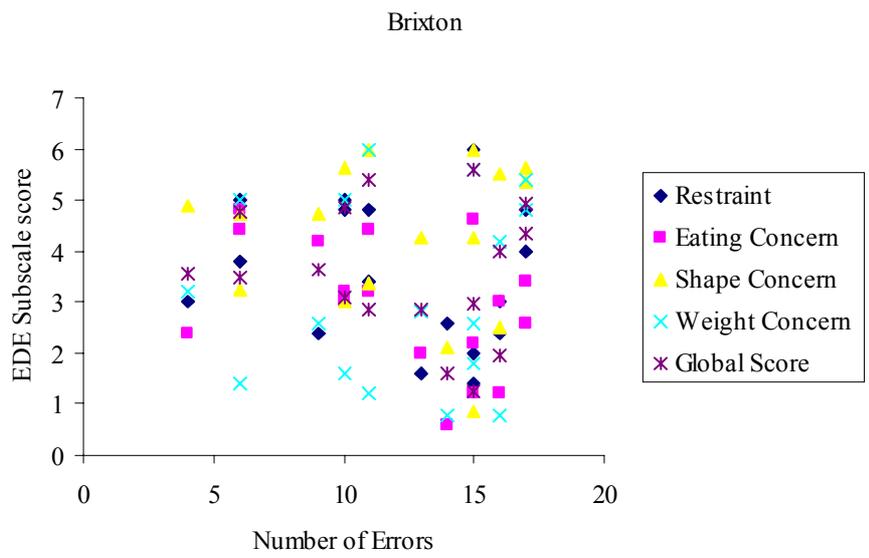
In Relation to Hypothesis 3

Figure H.13: WCST Perseverative Errors and YBOCS Total Score in Eating Disorder Group n=17
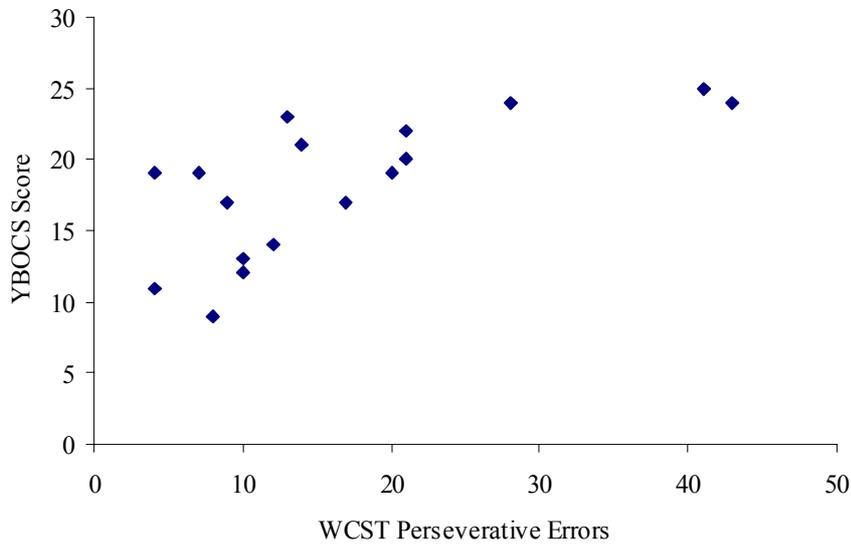
In Relation to Hypothesis 4

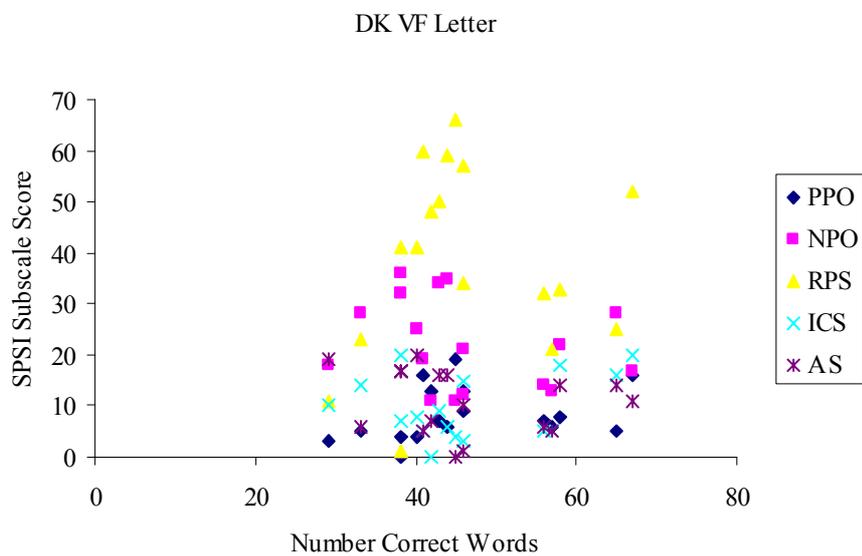**Figure H.14:** SPSI and WCST Perseverative Errors in Eating Disorder Group n=17
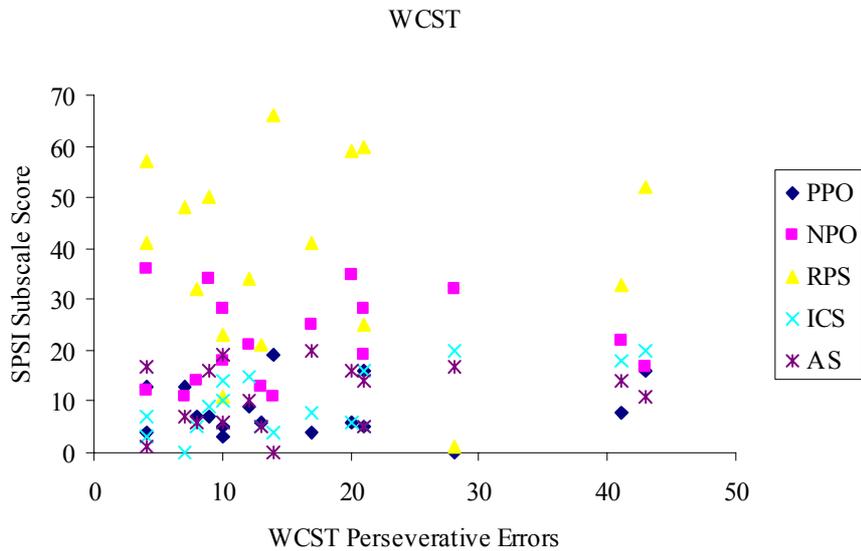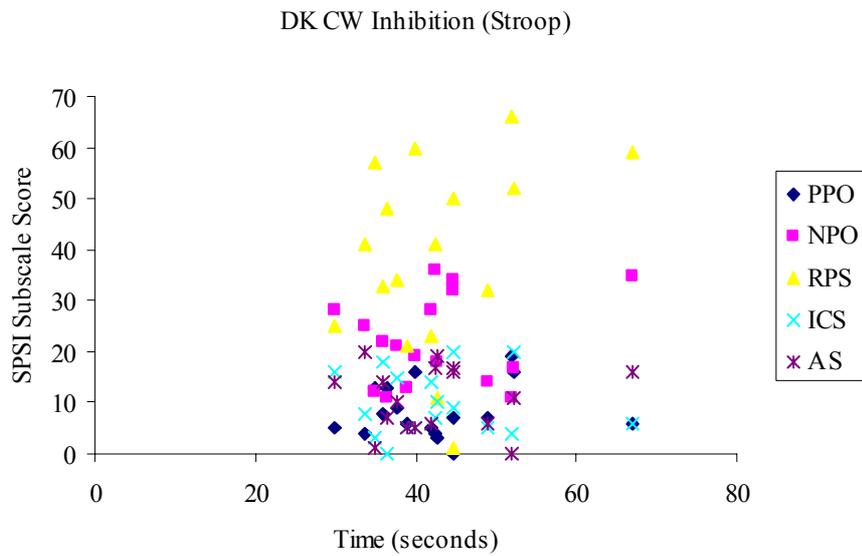

NB: PPO=positive problem orientation, NPO=negative problem orientation, RPS=rational problem solving style, ICS=impulsivity/carelessness style, AS=avoidance style.

Figure H.15: SPSI and Delis-Kaplan Trail Making Test Number Letter Switch in Eating Disorder Group n=17
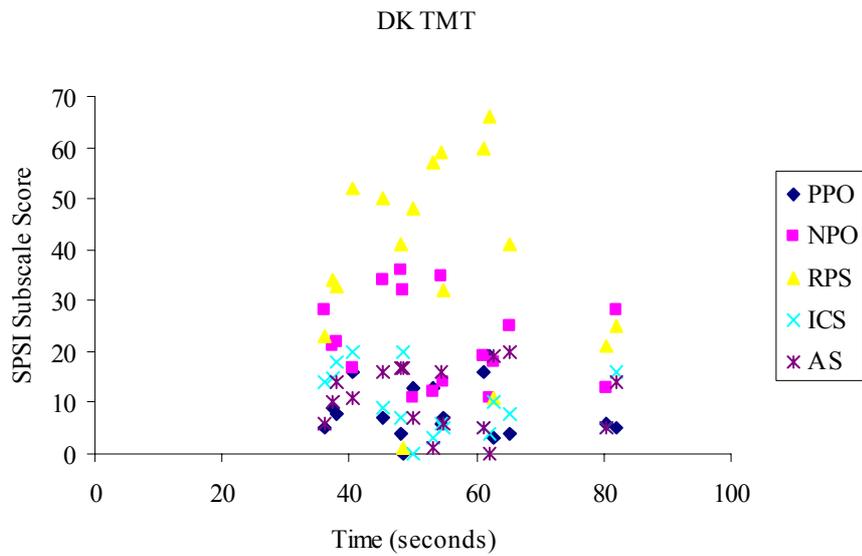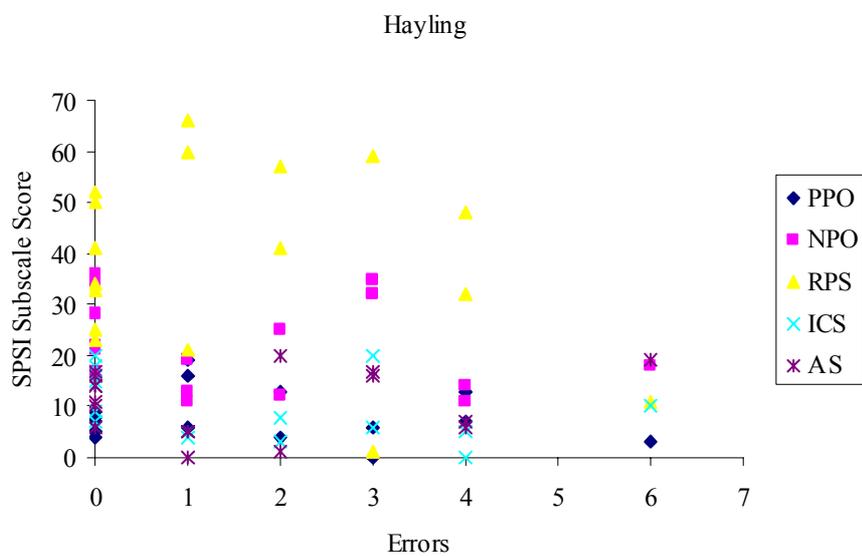

NB: PPO=positive problem orientation, NPO=negative problem orientation, RPS=rational problem solving style, ICS=impulsivity/carelessness style, AS=avoidance style.

Figure H.16: SPSI and Delis-Kaplan Verbal Fluency Letter in Eating Disorder Group n=17

NB: PPO=positive problem orientation, NPO=negative problem orientation, RPS=rational problem solving style, ICS=impulsivity/carelessness style, AS=avoidance style.

Figure H.17: SPSI and Delis-Kaplan Colour Word Inhibition (Stroop) in Eating
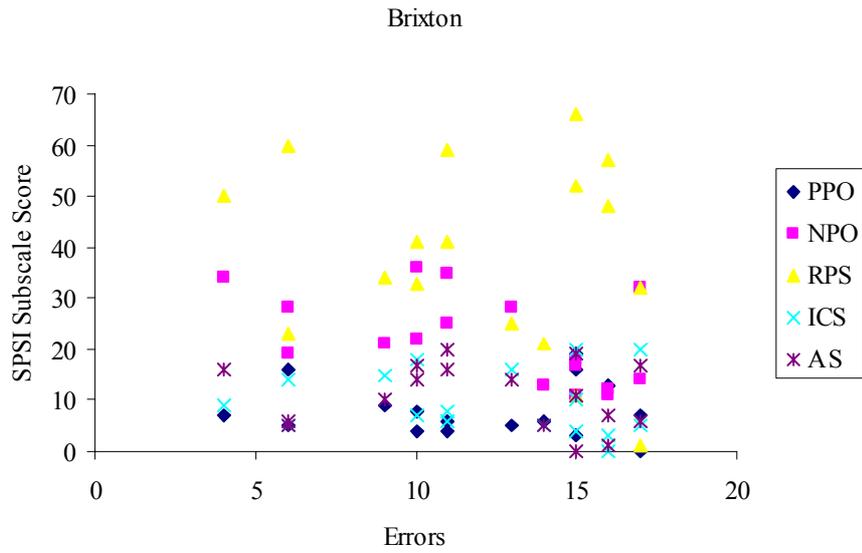Disorder Group n=17

DK CW Inhibition (Stroop)

*NB: PPO=positive problem orientation, NPO=negative problem orientation, RPS=rational problem
solving style, ICS=impulsivity/carelessness style, AS=avoidance style.*

Figure H.18: SPSI and Hayling Test in Eating Disorder Group n=17

Hayling

*NB: PPO=positive problem orientation, NPO=negative problem orientation, RPS=rational problem
solving style, ICS=impulsivity/carelessness style, AS=avoidance style.*

Figure H.19: SPSI and Brixton Test in Eating Disorder Group n=17

NB: PPO=positive problem orientation, NPO=negative problem orientation, RPS=rational problem solving style, ICS=impulsivity/carelessness style, AS=avoidance style.