NEUROPSYCHOLOGICAL CONTRIBUTIONS TO SYMPTOMATOLOGY IN
EATING DISORDERED PATIENTS

By

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Abstract
The present study examined the claim that neuropsychological deficits in set-shifting and emotional decision making are present in eating disordered patients, and to what extent these deficits relate to specific aspects of disordered eating. Sixteen eating disordered patients and 38 controls were given a battery of neuropsychological measures, as well as questionnaires measuring disordered eating. Compared to controls, patients demonstrated poorer performance on tasks of set-shifting, but not decision making, psychomotor speed, working memory, or IQ. Across groups, poor set-shifting was correlated with food-, shape-, and weight concerns, and restricting, whereas poor decision making was correlated to restricting. The study demonstrates that set-shifting deficits are present in eating disordered patients, and that specific relations exist between cognitive performance in different domains and disordered eating.
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Neuropsychological Contributions to Symptomatology in Eating Disordered Patients

Eating disorders (ED) are characterized by atypical eating behaviour, preoccupations with food, and disproportionate self-worth attributed to weight and body size (American Psychiatric Association, 1994; Grilo, 2006). Although both anorexia nervosa (AN) and bulimia nervosa (BN) are characterized by restriction and binge eating symptoms, these symptoms may have separate causal factors in the two disorders. Thus, understanding the putative factors that contribute to specific symptoms in ED would provide a clearer explanation of eating disorders in general.

Impulsivity and Eating Disorders

A growing body of literature described below suggests that the concept of impulsivity is a contributing factor to ED symptomatology, particularly in those diagnoses characterized primarily by bingeing behaviour (i.e., BN and binge eating disorder [BED]), as opposed to anorexia nervosa (AN), which is characterized primarily by restrictive eating behaviour. Impulsivity in this context refers to an inability to exert self-control in the presence of strong emotional and/or motivational factors, such as positive or negative affect, or tangible reinforcers such as money or food. This definition reflects Whiteside & Lynam’s (2001) definition of the “urgency” factor of impulsivity, which they hypothesize is “related to an inability to resist cravings, binging, and acting rashly while upset” (Whiteside & Lynam, 2001, p. 677). This is distinct from other aspects of impulsivity, such as lack of premeditation, sensation-seeking, and lack of perseverance, all of which were derived from the authors’ factor analyses of a myriad of different impulsivity self-reports.
This conception of impulsivity for psychopathologies in general and in ED in particular has face validity, if one considers the various references to impulsivity in the DSM-IV diagnostic criteria (e.g., antisocial personality disorder, borderline personality disorder, substance abuse). In the case of BN, recurrent binge eating must occur while experiencing a lack of control over the behaviour (i.e., an inability to inhibit eating). Although both AN and BN are commonly co-morbid with axis I disorders, BN also shares increased co-morbidities with disorders characterized by poor behavioural control (i.e., panic disorder, borderline personality disorders, & substance abuse; Grilo, 2006; Steiger & Bruce, 2007).

More importantly, individuals suffering from BN and to a lesser extent AN, exhibit marked impulsivity. Kaye, Bastiani, and Moss (1995) conducted the first comparison of anorexic and bulimic patients on cognitive measures and found that bulimics displayed more impulsive cognitive styles compared to anorexics, who showed more reflective cognitive styles. Claes, Vandereycken, and Vertommen (2002) tested both bulimics and anorexics on a number of self-report measures of impulsivity and personality disorder traits. Bulimics reported higher levels of impulsivity compared to both anorexics and healthy controls, a difference which remained significant even after controlling for the impulsive Axis II cluster B personality disorder traits (i.e., antisocial, borderline, histrionic, & narcissistic). Furthermore, bulimics reported more concerns over loss of control over mental activities and motivated motor behaviour (i.e., aggressive, sexual acts), compared to anorexics.

Similarly, studies utilizing behavioural paradigms to measure impulsivity have provided converging evidence for the presence of impulsivity in ED patients (Kaye et al.,
1995; Rosval, Steiger, Bruce, Israel, Richardson, & Aubut, 2006; Bruce, Koerner, Steiger, & Young, 2003). In the only study to date to examine both self-report and behavioural measures of impulsivity, Rosval and colleagues measured 87 bulimic and 37 anorexic patients, using a self-report measure of impulsivity and the go/no-go task. In this task, participants must learn to respond to one stimulus and not the other, in order to gain symbolic monetary rewards. Periodically, the reinforcing stimulus is changed, and participants must learn to respond to the new reinforcer. Incorrect responses in the form of commissions (i.e., inappropriately responding) or omissions (failing to respond when appropriate) both result in a loss of money. Thus, the task requires one to learn the contingencies, and more importantly, to exercise the proper response inhibition for trials where inappropriate responding results in monetary loss (i.e., punishment). Rosval et al. found that bulimics, but not anorexics, showed behavioural impulsivity as evidenced through both the go/no-go task and self-report. In accordance with these results, Bruce et al. (2003) found that bulimics who reported laxative use were more impulsive on a go/no-go task, compared to bulimics who did not use laxatives. Moreover, these results were unrelated to other reported psychopathology (i.e. depression, general anxiety, and posttraumatic stress).

Similar results have been found in normal females, who show a positive correlation between self-reported bulimic traits and impulsivity traits (Fischer, Smith, & Anderson, 2003). Moreover, evidence suggests that overeating tendencies are mediated indirectly by reward sensitivity (Davis, Strachan, & Berkson, 2004; Davis, Patte, Tweed, & Curtis, 2007); this suggests that an individual’s increased sensitivity to positively reinforcing stimuli (i.e., food) may contribute to his/her tendency to overeat in response
to negative affect, which is then likely to influence BMI (Davis et al., 2007). Thus, both self-report and behavioural measures have demonstrated a greater association of EDs that involve binge eating with motivational impulsivity, highlighting it as a potential contributor to disordered eating in response to negative affect.

Compulsivity and Eating Disorders

In addition, there is growing evidence to suggest that the rigid beliefs and restrictive behavioural aspects of ED may share commonalities with obsessive-compulsive disorder (OCD). The original conception of ED as a variant of an OCD dates back to Palmer & Jones (1939). Conceptually, there are many similarities in clinical symptomatology between OCD and ED. Patients with AN are rigidly preoccupied with thoughts pertaining to food, weight, and body image, and frequently engage in ritualistic-like behaviours such as excessive exercise, dieting, and other routines to promote weight loss (Davis & Kaptein, 2006). As the disorder progresses and weight loss continues, these rituals may become more ingrained and resistant to change (Klein & Walsh, 2004). This is analogous to the classical pattern of obsessions (i.e., intrusive, distressing thoughts), which drive a patient to compulsions (specific, regimented acts) that result in temporary amelioration of the anxiety underlying the obsession (American Psychiatric Association, 1994).

Indeed there are a number of links between OCD and ED (Swinbourne & Touyz, 2007). Increased incidence rates of OCD within eating disordered populations are clearly evident (Jacobi, Haywood, de Zwaan, Kraemer, & Agras, 2004; Rothernburg, 1986), and are estimated to range from 31 to 69% (Bastiani, Altemus, Rubenstein, Weltzin, & Kaye, 1996; Swinbourne & Touyz, 2007). AN generally shows higher co-morbidity rates with
OCD, compared to co-morbidity rates between BN and OCD. Matsunaga et al. (1999) found a 33% prevalence rate of comorbidity between OCD and BN, whereas Von Ranson, Kaye, Weltzin, Rao, and Matsunaga (1999) confirmed the presence of OC-symptoms in BN, which persisted even after recovery from BN. Thus, the general co-occurrence of obsessive compulsive symptoms with ED suggests that certain features of ED may share common etiology with OCD.

Determining Causes of Eating Disorders

The periodic loss of control predominant in bingeing diagnoses such as BN and BED, as well as the characteristic highly restrictive eating behaviour and inflexible views about the self in AN, raise the question of what underlying cognitive, emotional, and motivational processes are at work in ED. The vast majority of work on characterizing the beliefs, personality traits, and symptomatology of ED has created clear descriptions of these disorders. However, causal factors underlying various core factors of disordered eating remain unknown, particularly those relating to bingeing, restricting, and rigid beliefs. Thus far, attempts to establish causal factors of ED have focused on genetics.

Substantial work on genetic predispositions to EDs suggest that genetic factors may account for between 58 – 76% of the variance for susceptibility to developing AN, with interactions between genetic predisposition and proper environmental influences being a likely process for expression of AN (Klein & Walsh, 2004). However, genes in and of themselves cannot exhaustively explain behaviour, since they merely code for protein expression, and there are numerous steps between protein expression, stimulus perception, information processing, and overt behaviour. The majority of genetic research in ED focuses on aberrant neurotransmitters or neurotransmitter receptors within the
brain, with primary focus on serotonin and neurohormones (Klump & Gobrogge, 2005). Because the role of neurotransmitters in the brain is for cellular signalling, their influence and thus any genetic influences ultimately reside in the ability to properly regulate information processing in the brain. Therefore, studying the known psychological functions of the brain could afford a better understanding of the rudimentary factors that underlie ED (Treasure, 2006).

Neuropsychology is concerned with the links between different brain regions and their respective information processing functions, and thus is a useful tool in ascertaining how humans process information, and the various brain regions that subserve such processes. In general, the major neuropsychological domains include language, memory, visuospatial processing, attention, and executive function (Lezak, Howieson, & Loring, 2004). Each domain is further subdivided into specific aspects: for example, memory is broken down into short-term memory, long-term memory, semantic memory, etc. Numerous neuropsychological tests have been developed to obtain a measurement of any of the cognitive domains, using either qualitative or quantitative data. Typically, the data obtained from an individual’s performance is compared to a larger set of normative data, to determine the individual’s level of performance relative to the general population.

Neuropsychological Impairment in Eating Disorders

The concept of utilizing neuropsychological paradigms to examine psychological disorders is not novel. Numerous other psychopathologies have been found to contain neuropsychological impairments, which have aided in explaining their puzzling manifestations. For example, substance abuse has also been characterized as an inability to use internal emotional information to guide decision-making to maximize benefit and
minimize loss (Bechara, 2005). Such individuals appear to have a ‘myopia for the future’, failing to use negative emotions to inhibit drug-taking behaviour, which is immediately rewarding. This impairment in integrating emotion into decision-making appears to rely on the ventromedial prefrontal cortex (VMPFC). Other such theories linking clinical symptomatology to cognitive impairments exist for borderline personality disorder, OCD, and Parkinson’s and Huntington’s diseases (Brendel, Stern, & Silbersweig, 2005; van Veen & Carter, 2007; Yechiam, Busemeyer, Stout, & Bechara, 2006).

With respect to ED, several investigators have also suggested that neuropsychological impairments may play a role in the etiology or maintenance of the disorder. Jones, Duncan, Brouwers, and Minsky (1991) were the first to suggest that ED patients may have cognitive impairments that contribute to their clinical presentation, showing that both AN and BN patients showed slightly lower scores in numerous cognitive domains. Ferraro, Wonderlich, & Jocic (1997) furthered this notion, suggesting that examining cognitive impairments within this population may help explain group and individual differences at the symptom level. Treasure, Tchanturia, and Schmidt (2005) suggested a developmental model incorporating interactions between personality styles, genetic factors, social influences, and brain function in the development of an ED. It is reasonable to consider that if eating disordered patients are fundamentally different from the normal population in how they process information, this impairment may play some role in the evolution, maintenance, or treatment of the eating disorder.

However, there is a paucity of research to date on the neuropsychological impairments found in impulsive ED, with the majority of existing research focusing on AN. With respect to AN, impairments have been noted in attention, visuospatial
perception, and executive functioning (Bosanac et al., 2007; Duchesne, Mattos, Fontenelle, Veiga, Rizo, & Appolinario, 2004; Lauer, Gorzewski, Gerlinghoff, Backmund, & Zihl, 1999), although no clear pattern of impairment seems to emerge. Aside from the handful of studies showing a motivational impulsivity deficit in BN, there is some evidence to suggest that executive functioning (EF) impairments, including set-shifting, may also be present in BN (Duchesne et al., 2004; Lena, Fiocco, & Leyenaa, 2004; Tchanturia et al., 2004). No studies have examined the putative role of neuropsychological impairment in BED patients. In sum, there is some evidence for EF impairment in bingers compared to normal controls, although further evidence is mixed.

It is theoretically plausible to suggest that the periodic loss of control in bingers may be indicative of impaired in executive functioning (EF). It is generally agreed that EF is composed of higher-level processes that operate upon various forms of information, usually in service of a specific goal. Examples of EF processes include problem solving, set-shifting, planning, and response inhibition (Turner & Levine, 2004). Specifically, it may be that ED patients are impaired on set-shifting tasks. These tasks require the participant to consciously alternate attention between multiple sets of stimuli that differ in terms of perceptual or conceptual properties. For example, the Trail Making Test (TMT; Reitan, 1958) simply requires an individual to connect a series of dots with a pencil, in alphanumeric order (i.e. 1-A-2-B, etc), requiring one to shift between the conceptual sets of letters and numbers.

For the ED patient, poor set-shifting (i.e., difficulties in volitional alternating to other forms of information) may preclude the development of maladaptive cognitions, including the distorted emphasis on food, weight, and body shape to determine self-
worth, and the inflexible rules surrounding eating behaviour. Supporting this notion, Roberts, Tchanturia, Stahl, Southgate, and Treasure (2007) conducted a meta-analysis of set-shifting ability in eating disordered patients based on 15 studies in the literature, and found evidence of set-shifting impairments, which varied in effect size based on the task used.

Neuropsychology of Decision-Making in ED

A recent development in the neuropsychological underpinnings of psychopathology has focused on real-world decision making, that is, those decisions that govern the course of our actions in everyday life. Humans are constantly presented with situations affording numerous possibilities and factors to consider, and must choose based on the most favourable outcome. For example, we make decisions on investing in the stock market, whether or not to buy a car, or which job offer to accept. Indeed, our functional capacity permits us to consider a myriad of objective and subjective factors, and then act appropriately.

Traditional theories of decision-making developed from economics, which presupposes humans to be primarily rational in nature. Early models of consumer decision behaviour relied heavily on probabilistic formulae that weighed the consumer’s choices based on the most favourable outcome (Kahneman, Slovic, & Tversky, 1982). However, these theories were not reflective of the true nature of consumer decisions, which often are based on heuristic principles and are vulnerable to framing effects (De Martino, Kumaran, Seymour, & Dolan, 2006; Naqvi, Shiv, & Bechara, 2006; Sanfey, 2007). For example, in one of these studies, participants who were told they had won $50 were more reluctant to gamble with that money if they were told they could keep $30,
compared to if they were told they would lose $20 (De Martino et al., 2006). In this scenario, both results had the same outcome, but the notion of being able to maintain some acquired amount of money seemed to dissuade willingness to gamble.

In contrast, seminal work by Bechara, Damasio and colleagues provided an alternate paradigm to model real-world decision-making. Their work focused on patients who suffered lesions to an area of the frontal lobes called the ventromedial prefrontal cortex (VMPFC). The majority of these patients show normal intellectual and cognitive functioning, and yet have a marked impairment in real-life decision-making: They often showed difficulty gaining and maintaining employment, choosing friends, deciding in their best interests, and learning from their mistakes (Bechara, Damasio, Damasio, & Anderson, 1994). It is argued that these patients show decision-making that is geared towards immediate reward, with a lack of consideration of long-term punishments that, for them, are not salient in the precise moment of decision-making (Bechara, 2005; Bechara, Dolan, & Hindes, 2002). For example, in alcoholism, the presence of alcohol or related contextual cues triggers intense craving, concurrent emotional reactions, and subsequent alcohol abuse, despite the negative long-term consequences of deterioration of one’s family, loss of employment, or social isolation (Bechara, 2005).

Bechara and colleagues (1994) designed the Iowa Gambling Task as a paradigm to measure this type of decision-making function, which requires the integration of emotional information into the cognitive operation in order to be successful. The IGT consists of 4 decks of cards, labelled ‘A’, ‘B’, ‘C’, and ‘D’. The subject is told to pick cards from any of the decks, with the goal of gaining as much money as possible. Decks ‘A’ and ‘B’ are referred to as the disadvantageous decks, since they give large amounts of
reward (i.e. $100, $200), but occasionally result in substantially large losses of money ($1250, $2500). Although initially appealing, continued selection from these decks will result in a net loss of money. Decks ‘C’ and ‘D’ are the advantageous decks, since they give moderate rewards (i.e. $50, $75), and periodically give proportionately smaller punishments (i.e. lose $45, $30). During the task, subjects learn that the two decks that initially are more rewarding also give rise to larger losses in the long-term, and that it is more beneficial to switch to the other two decks.

The IGT requires one to switch between choices that are immediately rewarding to those that are not as appealing at the moment, but will ultimately be more advantageous (Bechara et al., 1994, Bechara, Tranel, & Damasio, 2000). Moreover, due to the inconsistent nature of reward and punishment, the exact contingencies of each deck cannot be ascertained within a few trials. As a result, it has been argued that success on the IGT requires emotional information with respect to the positive affect experienced by reward, and negative affect experienced with punishment, in order to guide decisions trial by trial. In agreement with this notion, evidence from neuroimaging studies confirms that the VMPFC is consistently involved in decision-making tasks where emotional valence is involved, and where the outcomes are unexpected or not easily computed (De Martino et al., 2006; Northoff et al., 2006; O’Doherty, Kringelbach, Rolls, Hornack, & Andrews, 2001). Furthermore, activation of the VMPFC in normal participants correlates with performance on the IGT (Northoff et al., 2006).

However, successful performance on the IGT also relies on a number of other cognitive operations. Impaired memory (Gutbrod et al., 2006), executive function (Brand et al., 2007), and random responding due to poor sustained attention (Yechiam et al.,
can all result in impaired IGT performance, although not necessarily. Therefore, IGT performance in and of itself should not be interpreted as poor decision-making function, without discounting other potential contributing factors (Levine et al., 2005).

Consequently, a limitation of the IGT is a lack of specificity, due to its multifaceted nature. Nevertheless, one can conceptualize impairment on the IGT as being either primarily emotionally-based (i.e. poor integration of emotional information) or primarily cognitively-based (e.g. impaired EF, poor attention). If a patient population shows poor decision-making, but no impairments in any of the contributing cognitive constructs, their impairment can be assumed to be due to poor utilization of emotional information to guide decision-making. Conversely, if such patients are impaired in one or more of the contributing cognitive constructs, these may be able to account for their poor decision-making. For example, an individual who has learned that responding to certain stimuli is disadvantageous, but cannot change their actions due to poor set-shifting, does show poor decision-making, but not because of poor emotional processing. It follows, then, that if these contributing factors are tested in conjunction with the IGT, the specific factor underlying poor decision-making can be parsed out.

The merit of utilizing the IGT is due to its ecological validity, i.e., as a measure of decision-making function in real-world situations (Dunn, Dalgleish, & Lawrence, 2006). As mentioned earlier, VMPFC lesion patients show poor IGT performance, despite having no observable impairments in general intellectual functioning or specific cognitive function. The IGT has also been shown to be a successful indicator of treatment prognosis in OCD and AN (Cavedini et al., 2002; Cavedini et al., 2006), and is a moderately sensitive measure of VMPFC damage and real-life executive and emotional
problems in patients suffering from traumatic brain injury (Levine et al., 2005). In terms of ED, it may be that patients also show this poor affective decision-making, and this impairment may relate to specific symptoms.

Specifically, it may be that the poor emotional regulation in bingeing ED patients is associated with the poor emotional decision-making as measured through the IGT. There is ample evidence to suggest that emotional eating occurs in response to negative situations such as ego-threat and stress, and this tendency towards eating serves to divert attention towards reinforcing stimuli in the environment (i.e., food), and away from negative self-awareness (Heatherton & Baumeister, 1991). Moreover, there is evidence to suggest that impulsivity interacts with poor emotional regulation to influence emotional eating (Bekker, Meerendonk, & Mollerus, 2004; Elfhag & Morey, 2008; Stice, 2002). At a glance, this parallels the reward-seeking behaviour typified by other patient groups who perform poorly on the IGT, who impulsively seek immediate rewards despite long-term negative consequences (Bechara, 2005).

Preliminary results regarding IGT performance in bingeing ED patients are mixed. Boeka & Lokken (2006) found poorer IGT performance in bulimics compared to controls. However, Bosanac and colleagues (2007) found no difference between bulimics and controls in their study. No studies to date have looked at IGT performance in BED; however, IGT performance seems to be poorer in obese individuals, compared to normal controls (Davis, Levitan, Muglia, Bewell, & Kennedy, 2004). Conversely, anorexic patients seem to consistently show impaired IGT performance (Cavedini et al., 2004; 2006; Tchanturia et al., 2007). Cavedini and colleagues (2004) were the first to report that anorexic patients showed poorer IGT performance compared to normal controls (but
see Bosanac et al., 2007). In a follow up study, Cavedini et al. (2006) confirmed their previous findings, and found that better performance on the IGT predicted better treatment outcome (i.e. higher BMI) following cognitive behaviour therapy and drug treatment.

It is interesting to note that the neuroimaging literature of ED largely coincides with the literature on decision-making and the VMPFC. Activation of the orbitofrontal cortex (OFC), a section within the medial prefrontal cortex, occurs during passive viewing of food vs. non-food stimuli (Killgore et al., 2003; Porubska, Veit, Preissl, Fritsche, & Birbaumer, 2006) and with making decisions about food choice based on palatability (Hinton et al., 2004). Also, activation in the OFC is mediated by satiety state, although the nature of this relationship is not clear (Beaver, Lawrence, van Ditzhuijzen, Davis, Woods, & Calder, 2006; Santel, Baving, Krauel, Munte, & Rotte, 2006). Moreover, aberrant medial prefrontal cortex activity is observed in both anorexics and bulimics (Uher et al., 2003), with increased activation during symptom provocation (Uher et al., 2004). Taken together, these studies suggest that indeed this region is involved with evaluation of choices (i.e., food choices) based partially on subjective motivational value, consistent with the claim of the IGT as a task measuring emotional decision-making.

**Current Limitations**

A key limitation in the eating disorders literature to date has been the lack of attempts to connect cognitive impairments with psychopathological behaviour. There is some evidence to suggest that neuropsychological impairment may play a role in some aspects of ED, but not others. Specifically, preliminary evidence suggests that self-reported EF deficits correlate with the number of bingeing episodes in normal controls.
(Spinella and Lyke, 2004). However, it should be noted that the EF measure was a self-report questionnaire, and is not a pure test of EF. Conversely, Mikos and colleagues (2008) showed no correlation between cognitive performance on a myriad of neuropsychological tasks, and overall eating disorder symptomatology in anorexic patients at intake, discharge, and at a two-year follow-up. Thus, it appears that certain neuropsychological impairments may play a role more in bingeing eating disordered patients, compared to more restrictive patients. However, the question remains as to whether or not neuropsychological impairments contribute to impulsive behaviour and inflexible views, in an impulsive ED sample. The purpose of this study is to determine a) whether impulsive ED patients show set-shifting and decision making impairments, and if so, b) to which specific psychological symptomatologies are these impairments related. Moreover, measures of psychomotor speed, working memory, and IQ were included to rule out their potential influence upon the variables of interest.

**Hypotheses**

In line with the notions that poor set-shifting would relate to inflexible beliefs and impulsive decision-making with emotional eating, it was hypothesized that ED patients would show impaired performance on set-shifting tasks, as well as on the IGT. Moreover, set-shifting performance would be associated with inflexible cognitive beliefs surrounding eating, food, and weight, whereas decision making performance on the IGT would correlate with emotional eating. Finally, no group differences would be found on measures of working memory, psychomotor speed, and general intellectual function.
Method

Participants:

Sixteen women between the ages of 18-47 were recruited from the outpatient eating disorders clinic at the Centre for Addiction and Mental Health (CAMH). The diagnoses were established through use of the Structured Clinical Interview for DSM-IV (SCID) axis I and II (American Psychiatric Association, 1994), as well as the Eating Disorder Examination Questionnaire (EDE-Q; Fairburn & Beglin, 1994). All diagnoses were made by a licensed clinical psychologist, with 10 years of experience in the eating disorders field. Of the 16 patients, nine had a diagnosis of BN, two had a diagnosis of BED, three had a diagnosis of AN-bingeing subtype, and two had a diagnosis of eating disorder not otherwise specified. Exclusion criteria for the outpatient group included: the presence of a psychiatric/psychological disturbance aside from depression/anxiety, prior history of neurological insult (e.g., traumatic brain injury, infection, cerebrovascular accident), prior history of exposure to organic solvents or toxins (i.e., lead, industrial solvents), or any other disease of the central nervous system. No patients in this study were excluded based on these criteria. Participants received $10 remuneration for the duration of the experiment. Thirty-eight healthy controls were recruited from the University of Toronto through an online recruitment system, and received two course points towards their final mark in their an introductory psychology class. Exclusion criteria for the controls included: no history of neurological insult, exposure to organic solvents, or any current psychiatric diagnosis. No participants were excluded based on these criteria. All studies were approved by Review Ethics Boards at both the University of Toronto and the Centre for Addiction and Mental Health.
Materials:

All clinical measures were collected through an intake assessment by a certified clinical psychologist, or supervised PhD practicum students with adequate training in the psychological assessment of eating disorders. These sessions were separate from the primary experimental testing session. Eating disorder symptom severity was measured through the Eating Disorders Examination Questionnaire (Fairburn & Beglin, 1994), and the Emotional Eating Scale (EES; Arnow et al., 1995).

*The Eating Disorders Examination Questionnaire.*

The EDE-Q is a 33-item self-report questionnaire with four subscales measuring restraint, and concerns with weight, eating, and shape. It is modeled after the Eating Disorders Examination (EDE; Fairburn & Cooper, 1993), one of the most popular clinical interviews for assessment of eating disorder symptomatology. The EDE-Q has high rates of agreement with the EDE (Fairburn & Beglin, 1994).

*The Emotional Eating Scale.*

The EES consists of 25 questions scored on a 5-point Likert scale, and was designed to quantify eating in response to negative affect. The three subscales include eating in response to depression, anxiety, and anger/frustration, and all significantly correlate with binge eating behaviour in eating disordered patients (Arnow et al., 1995).

*Test of Nonverbal Intelligence (TONI).*

The TONI (Brown et al., 1997) is a nonverbal measure of abstract reasoning, problem solving, and general intellectual function. It consists of 46 incomplete figures, which the participant must complete by pointing to the appropriate choice of missing pieces at the bottom of the stimulus page. Testing is discontinued after a participant
makes three errors out of the last five administered items. A total score gives an estimation of IQ.

**Trail Making Test.**

The TMT (Reitan, 1958) is a measure of cognitive set-shifting. Part A tests psychomotor speed by having the participant connect lettered dots alphabetically as quickly as possible. Part B consists of connecting dots in alternating alpha-numeric order, and is considered a measure of set-shifting, as participants must constantly shift between numbers and letters. Time to completion and number of errors are used as measures of set-shifting performance.

**Card Sorting Task (CST).**

The CST subtest of the Delis-Kaplan Executive Functioning Scales measures cognitive set-shifting, as well as perseveration (Delis et al., 2001). The participant is shown 6 cards that can be sorted into two equal groups, based on eight different attributes (e.g., shape, colour, size, perceptual design, meaning of words printed on the cards, etc). The task is to sort the cards as many ways as the participant can think of. Scores are determined by the total number of confirmed correct sorts, and the time required to complete the task. Indexes of descriptive capability and perseveration can also be ascertained. For this study, the number of confirmed correct sorts was used as the primary outcome measure of set-shifting.

**Digit Symbol Coding.**

The digit symbol coding subtest of the Wechsler Adult Intelligence Scale III (WAIS-III) is a measure of attention and psychomotor speed (Lezak et al., 2004; Wechsler, 1997). Participants are shown a number of symbols that correspond to specific
numeric digits, and are given two minutes to fill in two rows of blanks with the symbol that corresponds to the digit listed above each blank. The score is determined by the total number of squares filled in correctly.

**Digit Span.**

The digit span subtest of the WAIS-III is a measure of working memory (Lezak et al., 2004; Wechsler, 1997). In the forward condition, participants are read strings of numbers, and are asked to repeat the string out loud. The length of strings increases until the participant fails to reproduce a string length twice. In the backward condition, the task is the same except the participant is to repeat the numbers in the reverse order that they were told. The score is determined by the total number of trials completed correctly.

**Balloon Analogue Risk Task (BART).**

The BART (Lejuez et al., 2002) is a computerized measure of pure impulsive risk-taking. On the screen, participants have to pump up a balloon via clicking on a button. Each pump results in a gain of 5 cents and the subjects can choose to stop any time and cash their earnings by pressing a separate button. However, the balloon may pop at any time, and if it explodes before the subjects’ cash their earnings, they gain no money. The task lasts for 30 trials, and measures of impulsivity are ascertained by the average number of pumps, and the total number of explosions. The BART has reliably measured impulsive behaviour in psychopathy, adolescents, smokers, and drug abusers (Hopko et al., 2006; Lejuez et al., 2002; 2003; Vigil-Colet, 2007).

**Iowa Gambling Task (IGT).**

The IGT is a measure of emotional decision-making (Bechara et al., 1994). The task consists of 100 trials, whereby participants are required to choose from four decks of
cards to earn money, and to determine which decks are the most advantageous overall. Performance on this task is measured by breaking the trials into 5 blocks of 20 trials, and subtracting the number of choices from disadvantageous decks (‘A’ and ‘B’) from the advantageous decks (‘C’ and ‘D’) within each block. Good performance is characterized by proportionately more selections from the good decks in the last two blocks of the task (Boeka & Lokken, 2006; Brand et al., 2007), as the first three blocks are associated with learning the task (Bechara, Damasio, Tranel, & Damasio, 1997; Maia & McClelland, 2004).

Procedure:

Testing sessions lasted between 45 – 60 minutes. After obtaining consent, participants were told that they would be given a number of tests to see how well they can take in information, and use it for a task. As an example, they were told that they may have to remember some numbers, or make decisions based on a card game. Participants were encouraged to do their best, and not be discouraged if they found any of the tasks difficult. To avoid any order effects, the tests were administered in random order. After the testing session, participants were remunerated, and any questions regarding the study were answered.

Statistical analyses

All statistical measures were conducted using SPSS version 15.0 (SPSS Inc., 2005). All measures were checked for normality of the distribution using the Kolmogorov-Smirnov statistic. The eating concern subscale of the EDE-Q was the only measure not normally distributed. Thus, Spearman’s rho was used to examine correlations with this subscale. All other correlations were calculated using Pearson’s
product-moment correlations. Between group analyses were conducted using mixed ANOVA for the IGT, and independent samples t-tests for all other dependent measures.

Participants in the control group were excluded if their EDE-Q total score was four or greater, which is two SD above the mean score in a normal population (see Mond, Hay, Rodgers, & Owen, 2006). Based on these criteria, three participants from the control group were excluded. Moreover, three participants in the control group had outlier scores on the TMT-B, and thus their data was not included on those analyses. No other outliers were found on any measures of interest (i.e. IGT, CST, BART, EDE-Q, & EES).

One participant in the ED group chose not to complete the TONI, and another participant’s EDE-Q and EES scores were not available. Moreover, four patients and four controls were missing IGT scores, due to limitations of the task that will be addressed in the discussion.

Results

Group demographics are listed in Table 1. There was a significant age difference between the ED group \( (M = 26.19, SD = 8.03) \) and the control group \( (M = 18.83, SD = 1.50) \), \( t(49) = 5.28, p < .001 \). As expected, there were highly significant differences between groups on all eating disorder measures, and no significant differences in overall IQ or education.

With respect to neuropsychological measures, we found a significant difference in set-shifting performance on the TMT-B, as measured by time to complete the task, \( t(46) = 2.36, p < .05, d = .72 \). In general, ED patients took longer to complete the task compared to normal controls. However, ED patients did not differ from controls on the
CST, the other measure of set-shifting, \( t(46) = 1.03 \), ns. No other group differences were significant (Table 1).

**Table 1. Clinical demographics and neuropsychological performance**

<table>
<thead>
<tr>
<th></th>
<th>ED Patients</th>
<th>Controls</th>
<th>( t )</th>
<th>df</th>
<th>( p )</th>
<th>Cohen's ( d )</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Demographics</strong></td>
<td></td>
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<tr>
<td>Age</td>
<td>26.19 (8.03)</td>
<td>18.83 (1.50)</td>
<td>5.28</td>
<td>49</td>
<td>&lt; .001</td>
<td>1.59</td>
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<tr>
<td>Education (years)</td>
<td>14.13 (2.36)</td>
<td>13.34 (.91)</td>
<td>1.72</td>
<td>49</td>
<td>0.09</td>
<td>0.52</td>
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<tr>
<td>TONI (raw)</td>
<td>30.13 (6.12)</td>
<td>29.09 (6.40)</td>
<td>&lt; 1</td>
<td>48</td>
<td>0.59</td>
<td>0.17</td>
</tr>
<tr>
<td><strong>Neuropsychological Tasks</strong></td>
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<tr>
<td>TMT-A (time)</td>
<td>(16.14)</td>
<td>(17.63)</td>
<td>2.36</td>
<td>46</td>
<td>&lt; .05</td>
<td>0.72</td>
</tr>
<tr>
<td>Digit Symbol</td>
<td>(11.88)</td>
<td>(12.31)</td>
<td>-1.8</td>
<td>49</td>
<td>0.08</td>
<td>-0.54</td>
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<tr>
<td>BART (average pumps)</td>
<td>(11.49)</td>
<td>(11.42)</td>
<td>&lt; 1</td>
<td>46</td>
<td>0.72</td>
<td>0.11</td>
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<tr>
<td>BART(exploding)</td>
<td>8.25 (3.70)</td>
<td>7.77 (2.94)</td>
<td>&lt; 1</td>
<td>45</td>
<td>0.63</td>
<td>0.15</td>
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<td><strong>Eating Disorder Symptomatology</strong></td>
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<td>EDE-Q Restraint</td>
<td>3.46 (1.26)</td>
<td>1.15 (1.10)</td>
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<td>&lt; .001</td>
<td>2.48</td>
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<tr>
<td>EDE-Q Eating Concern</td>
<td>3.87 (1.33)</td>
<td>.76 (1.04)</td>
<td>9.87</td>
<td>48</td>
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<td>3.04</td>
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<td>EDE-Q Shape Concern</td>
<td>4.02 (1.47)</td>
<td>2.13 (1.27)</td>
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<td>&lt; .001</td>
<td>1.83</td>
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<tr>
<td>EDE-Q Weight Concern</td>
<td>4.13 (1.48)</td>
<td>1.76 (1.28)</td>
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<td>48</td>
<td>&lt; .001</td>
<td>2.11</td>
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<tr>
<td>EES</td>
<td>65.5 (30.26)</td>
<td>(13.30)</td>
<td>8.15</td>
<td>47</td>
<td>&lt; .001</td>
<td>2.53</td>
</tr>
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</table>

To examine IGT performance, we used the conventional method of dividing the total number of trials into blocks of 20, and conducting a 2 X 5 mixed-ANOVA with group as the between-subjects variable, and blocks as the within-subjects variable (Bechara et al., 2000). There was a significant main effect of block, \( F_{\text{Block}} (4, 164) = 6.52, \quad p < .001 \), indicating that performance across blocks differed in general. However, neither the main effect of group, nor the interaction, was significant, \( F_{\text{Group}} (1, 41) < 1, \ F_{\text{GxB}} (4, \ 164) < 1 \) (Figure 1).
Figure 1. Iowa Gambling Task performance as a function of blocks of 20 trials

Correlations between neuropsychological measures of set-shifting and decision-making with ED symptomatology are listed in Figure 2. Associations between variables of interest and the IGT were done by correlating the appropriate outcome scores with the net score of the last 40 trials of the IGT. This was done based on recent evidence suggesting that the last sections of the IGT are when decision-making differences manifest (Brand, Recknor, et al., 2007). With respect to set-shifting performance, TMT-B performance was positively associated with all four subscales of the EDE-Q; however, performance on the CST was not associated with any eating disorder scales. IGT performance was associated only with the restricting subscale of the EDE-Q; however, the relation was significant only at the $p < .05$ level. Neither the BART nor the EES correlated with any measures in a meaningful way. Although total number of pumps on
the BART was correlated with number of confirmed sorts on the CST, it is unclear what
this relation indicates.

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<td>.77**</td>
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<td>.34*</td>
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<td>11</td>
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<td>.65**</td>
<td>.46**</td>
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* p < .05
** p < .01
Note: TONI = Test of Nonverbal Intelligence, TMT-B = Trail Making Test B, CST = Card Sorting Task, IGT = Iowa Gambling Task, RES = EDE-Q restraint subscale, EC = EDE-Q Eating Concern Subscale, SC = EDE-Q Shape Concern Subscale, WC = EDE-Q Weight Concern Subscale, EES = Emotional Eating Scale

Figure 2. Correlations among set-shifting, decision making, and symptomatology measures

It is possible that other variables may have contributed to these associations.

Psychomotor speed and working memory can be affected by restrictive dieting (Green, Elliman, Wakeling & Rogers, 1996; Green & Rogers, 1998), and the TMT-B was significantly correlated with the IGT. To account for these potential effects, I calculated partial correlations between the TMT-B and the EDE-Q subscales, controlling for psychomotor speed (TMT-A), working memory (digit span) and the IGT. All prior correlations between the TMT-B and the EDE-Q remained significant at the $p < .05$ level, even after adjusting for potential confounding variables. With respect to decision making, the IGT has previously been associated with set-shifting in a normal sample (Brand et al., 2007), and with level of education (Evans, Kemish, & Turnbull, 2004). However, even
after controlling for TMT-B scores and years of education, the partial correlations between the IGT and the restricting subscale of the EDE-Q remained significant (Figure 3).

<table>
<thead>
<tr>
<th>Measure</th>
<th>TMT-B†</th>
<th>IGT‡‡</th>
</tr>
</thead>
<tbody>
<tr>
<td>RES</td>
<td>.35*</td>
<td>- .36*</td>
</tr>
<tr>
<td>EC‡</td>
<td>.46**</td>
<td>- .03</td>
</tr>
<tr>
<td>SC</td>
<td>.40*</td>
<td>- .16</td>
</tr>
<tr>
<td>WC</td>
<td>.41*</td>
<td>- .16</td>
</tr>
<tr>
<td>EES</td>
<td>.17</td>
<td>- .04</td>
</tr>
</tbody>
</table>

* p < .05  
** p < .01  
† Controlling for psychomotor speed (TMT-A), working memory (digit span), and IGT  
‡‡ Controlling for TMT-B and years of education  
‡ nonparametric correlations

Figure 3: Partial correlations between select eating disorder symptomatology and neuropsychological measures

Discussion

The purpose of this study was to determine whether set-shifting and decision-making impairments exist in a sample of bingeing ED patients, and to see whether these differences are associated with distinct aspects of ED. It may be that if neuropsychological impairments exist in ED patients, such impairments would contribute to the psychopathology of the ED in a meaningful way.

It was hypothesized that set-shifting impairments, which have been demonstrated in AN patient samples, would also occur in an impulsive ED patient sample with various diagnoses. Consistent with the hypothesis, ED patients showed a significant deficit in set-shifting performance on the TMT-B. However, there were no differences in the CST, the second measure of set-shifting. Presumably, an undifferentiated deficit in set-shifting performance should manifest consistently across a variety of tasks designed to tap the
same construct, which was not the case in this study. It may be that although both tasks challenged set-shifting ability; the timed nature of the TMT may have exerted greater cognitive demand on participants, whereas the CST may not have elicited the same degree of speeded information processing. Although the CST has a maximum 4-minute time restriction, no participants in this study exceeded the time restriction before completing the task to the best of their ability. Indeed, Roberts et al. (2007) found that effect sizes for set-shifting impairment in ED patients varied according to the task used, perhaps due to varying levels of sensitivity.

Moreover, in general agreement with the hypothesis, poorer set-shifting was positively correlated with higher scores on all subscales of the EDE-Q. These results suggest that individuals who place greater emphasis and though on food, shape, and weight have a more difficult time cognitively shifting between different sets of information within a given task. For the ED patient, a deficit in the ability to switch attention may contribute to the rigidity of her beliefs surrounding the importance of body shape and weight. Having been exposed to factors that encourage the development of self-worth through body shape (i.e., cultural norms encouraging thinness, genetic predisposition), an additional deficit in set-shifting may hinder her likelihood of considering alternate means of determining self-worth, thus facilitating the rigidity of the current beliefs. Given the observed relations between set-shifting performance and all subscales of the EDE-Q, it appears that this influence of set-shifting extends to concerns regarding food, weight, shape, and to beliefs about restricting.

It was posited that ED patients would show poor performance on a task of emotional decision-making, and that this would relate to their tendency to eat in response
to negative affect: That is, in the face of negative affect, ED patients impulsively engage in bingeing and purging cycles that are immediately rewarding (removal of negative affect), despite the looming negative consequences (e.g., negative feelings toward purging, loss of self-control, negative effects on health). However, there was no significant between group difference in IGT performance, and IGT performance was not correlated with emotional eating across groups. Thus, it seems that although bingeing ED patients are more impulsive with respect to eating (i.e., high levels of emotional eating), this impulsivity bears little relation to the impulsivity measured by the IGT. It may be that emotional eating relates to other neuropsychological processes such as behavioural and cognitive inhibition (Bruce et al., 2003; Rosval et al., 2006).

It is worth considering that the lack of a group difference may have been due partially to a flaw in the paradigm. In this study, four ED patients and four normal controls did not complete the IGT, and thus their data was not included in the analysis. This was because there are 100 trials in total, but only 60 cards per deck. Thus, it is possible for a participant to run out of cards before completing the task. In effect, this reduces the sensitivity of the task to between group differences because extreme responders’ data cannot be included in any analyses (Dunn et al., 2006). Such responders will include those who solve the task quicker than average and choose preferentially from the good decks, and those who show the poorest performance and are highly biased towards the bad decks. This is because it is only in the latter trials (i.e., blocks 4 & 5) when decision-making performance is parsed out (Bechara et al., 1997; Brand et al., 2007).
Surprisingly, IGT scores were negatively correlated with restraint scores on the EDE-Q, even after controlling for set-shifting performance and years of education. Prima facie, the fact that impulsive individuals who show a preference for immediate rewards (i.e., who have poor IGT scores), also tend to show higher restraint scores on the EDE-Q, seems counter-intuitive. However, consideration of the typical system of beliefs held by ED patients may resolve logical inconsistencies between theory and the present data.

It is widely accepted that self-mastery and control over eating behaviour are seen as the ultimate goal in ED patients, whether achieved by exercise, adherence to a strict diet, and/or more severe caloric restriction (Fairburn & Harrison, 2003; Grilo, 2006). Numerous cognitive-behavioural theories suggest that for patients, the need to exhibit self-control is the focus for subsequent restrictive behaviours, and is probably reinforced by a temporary increased sense of self-control and self-worth, a sense of achievement, and positive feedback from friends and family from the initial weight loss (Shafran & de Silva, 2003). However, in parallel, repeated instances of restrictive behaviour also result eventually in serious negative consequences, such as the deterioration of physical health and psychosocial disturbance. The correlation between IGT performance and restricting may reflect the ED patient’s relative inability to consider these negative consequences to guide the decision as to whether or not to continue restricting, instead focusing primarily on the immediate rewards.

Indeed, current research suggesting that the VMPFC is involved for the flexible assignment of stimulus-reward contingencies (Fellows & Farah, 2003; Rolls, Hornak, Wade, & McGrath, 1994) supports the notion of impaired reversal learning as a contributory factor in restrictive eating behaviour. In these studies, patients with damage
to the VMPFC must initially learn that responding to a certain set of stimuli results in reward. After a period of time, the contingency is switched and subjects must eventually learn the new contingencies (reversal learning). However, these patients cannot learn these new associations, regardless of the number of trials. It is conceivable that in ED patients, a similar mechanism may explain partially the initial positive association with restrictive eating, and the relative ineffectiveness of objectively negative consequences in dissuading such behaviour.

It is of interest that although patients and controls differed on the EES, there were no differences on the BART, a reported measure of impulsivity, nor was there any association between the BART and any symptomatology scales. Although no hypothesis was posited for this task, it would be expected that differences would exist on a measure of impulsive risk taking, given the known association of personality measures of impulsivity with bulimic symptomatology (Bekker et al., 2004; Elfhag & Morey, 2008; Stice, 2002). However, the concept of impulsivity is multi-faceted, and it may be that the BART is a more pure measure of sensation-seeking and venturesomeness, rather than impulsivity related to present versus future bias in decision making.

Overall, the results indicate that two separate neuropsychological factors have distinct roles in disordered eating. Set-shifting appears to be poorer in individuals who have high ratings of concern over their eating and shape in general, whereas decision-making biased towards immediate goals seems to relate selectively to an individual’s tendency to restrict her eating in order to achieve the goal of weight control. Collectively, these results suggest that specific relations occur between psychopathological symptoms and neuropsychological constructs. This may account for some of the mixed findings in
terms of correlating symptomatology with cognitive performance. For example, Mikos
and colleagues (2008) failed to find correlations between any neuropsychological
measures and EDE-Q total scores, based on a 2-year longitudinal study of AN patients.
Similarly, Steinglass, Walsh, and Stern (2006) failed to correlate poor set-shifting with
scores on the Eating Disorder Inventory. In contrast, Spinella and Lyke (2004) found
correlations between number of bingeing episodes and self-report executive function
impairment. It seems that utilizing total scores on questionnaires may mask underlying
correlations that are present between specific facets and neuropsychological tests.

The fact that the TMT and IGT both correlated with different subscales on the
same questionnaire raises the possibility that these correlations are spurious, rather than
veridical. This is plausible given the high intercorrelations between all subscales on the
EDE-Q, and the fact that both the TMT-B and IGT correlated with different subscales of
the same questionnaire. However, all set-shifting correlations were significant at the \( p < .01 \) level, and both TMT-B and IGT measures shared consistent partial correlations with
the same EDE-Q scales, albeit only at the \( p < .05 \) level. Moreover, specific correlations
between different neuropsychological tests and subscales of the same questionnaire have
been reported. Lawrence and colleagues (2006) found that within OCD patients, set-
shifting scores correlated with symmetry/ordering symptoms, whereas IGT scores were
correlated with hoarding symptoms within the same clinical sample measured with the
Obsessive Compulsive Inventory—Revised. Although this lends support to the
plausibility of the present findings, replication is required to confirm their veracity.

Assuming that the neuropsychological tests in use are reliable and valid metrics of
cognitive performance in ED patients, one should find evidence of neural function
abnormality in ED patients. Thus far, neuroimaging evidence is consistent with the notion of aberrant VMPFC function in ED patients (Hinton et al., 2004; Uher et al., 2004; 2006). However, there is mixed evidence regarding the relations between neuropsychological performance and structural brain changes in ED patients (Connan et al., 2006; Kingston, Szmuckler, Andrewes, Tress, & Desmond, 1996). Future research should elucidate the relations between psychopathology, neuropsychological test performance, and measures of brain structure and activity.

There were several limitations to the current study. As with the majority of studies on neuropsychological performance in ED, the small sample size of the patient group may have limited the statistical power to detect significant effects. Although the findings from this study suggest specific associations between symptoms and cognitive impairments, the small number of patients prevented any regression analyses within group to determine if similar relations occur across both normal and patient populations. In addition, the patient sample was drawn from an outpatient clinic, with only a small portion of clients consenting to participate in the present study. Also, patients were not excluded if they met the criteria for depression or substance abuse, given their high incidence of co-morbidity with ED. It is unclear whether or not these factors may have limited the generalizability of the present findings: however, there are practical and logistical limits to the extent of experimental control available when conducting research with clinical populations.

Moreover, this study did not account for the potential effects of nutrient depletion and starvation on cognitive performance. Although only a few patients were diagnosed as anorexic, this does not preclude the possibility that nutritional deficiencies due to
disordered eating in general affected cognition, although few studies have found such an association in this population (Bosanac et al., 2007; Gillberg, Rastam, Wentz, & Gillberg, 2007; Kingston et al, 1996).

Conclusions

In summary, this study found evidence that ED patients show some deficit in set-shifting performance, which relates generally to their eating and weight concerns and restrictive tendencies. The magnitude of this set-shifting deficit was relatively small by clinical convention, but may have profound effects in concert with dysfunctional beliefs and attitudes. Conversely, patients did not show a decision-making impairment, and decision-making performance did not show any relation with impulsive emotional eating. However, poor IGT performance was related to restrictive behaviour, paralleling previous work suggesting that self-control over eating behaviour is a powerful reinforcer that exerts a large influence on eating behaviour.
References


