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Quantitative evidence for distinct cognitive impairment in anorexia nervosa and bulimia nervosa

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It is generally agreed that at least some aspects of abnormal eating behaviour is indeed due in part to disordered cognition. The accumulated literature illustrates cognitive impairment in patients with anorexia nervosa (AN) and bulimia nervosa (BN). Yet beyond being inconsistent, these independent studies also do not reveal the magnitude of impairment within and across studies and fail to give due consideration to the magnitude of impairment so as to understand the severity and breadth of impairment and/or differences in cognitive profiles between patients with AN and BN. Hence, the present review on the subject sought to articulate the magnitude of cognitive impairment in patients with AN and BN by quantitatively synthesizing the existing literature using meta-analytic methodology. The results demonstrate modest evidence of cognitive impairment specific to AN and BN that is related to body mass index in AN in terms of its severity, and is differentially impaired between disorders. Together, these results suggest that disturbed cognition is figural in the presentation of eating disorders and may serve to play an integral role in its cause and maintenance. Implications of these findings with respects to future research are discussed.

The prevalence of eating disorders (ED) in North America is on the rise and it is estimated that 8% of females currently suffer from either anorexia nervosa (AN) or bulimia nervosa (BN) (American Psychiatric Association Work Group on Eating Disorders, 2000). Whilst putative social and personality theories of causation and maintenance have served to shed light on these disorders, interest has recently also developed in the idea that the central nervous system plays an integral role. To this end, it is generally agreed that at least some aspects of abnormal eating behaviour is indeed due in part to cognitive impairment (Lezak, Howieson, & Loring, 2004).

In regard to the existing literature on cognitive impairment, researchers have posited that ED may share the same cognitive profile as that seen in persons with problem gambling, addictions, and psychopathy (Treasure, 2006). For example, individuals who

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are problem gamblers have been shown to be inappropriate decision makers (Brand *et al.*, 2005) whilst the same has been found in patients diagnosed with AN and BN (Boeka & Lokken, 2006; Brand, Frank-Sievert, Jacoby, Markowitsch, & Tuschen-Caffier, 2007; Cavedini *et al.*, 2004, 2006; Tchanturia *et al.*, 2007). These findings suggest that impaired decision making is common between groups. Furthermore, the finding that patients with BN are often found to have co-morbid problems related to addiction is also supportive of this theory (Favaro *et al.*, 2008).

Beyond impaired decision making, other component processes of executive functioning have been examined in the ED perhaps given that impaired executive function in various other psychopathologies (e.g. problem gamblers, addiction, psychopathy) has been found to be most theoretically fundamental to their causes (Cavedini, Riboldi, Keller, D'Annucci, & Bellodi, 2002). To this end, the literature illustrates impaired mental flexibility and set shifting in patients with AN consistent with the behavioural patterns that characterizes the disorder. Indeed, rigid and obsessional focus as it relates to avoidance of weight gain has been thought to be the result of impaired mental flexibility and the inability to shift cognitive set. Predictably, researchers have found that patients with AN perform more poorly on clinical test measures of mental flexibility and set shifting (Cavedini *et al.*, 2004; Roberts, Tchanturia, Stahl, Southgate, & Treasure, 2007; Steinglass, Walsh, & Stern, 2006; Tchanturia *et al.*, 2004; Thompson, 1993). In contrast, the research literature regarding cognitive function in patients with BN illustrates impulsivity and risk taking (Fisher, Smith, & Anderson, 2003; Peñas-Lledó, Vaz, Ramos, & Waller, 2002; Rosval *et al.*, 2006; Steiger, Lehoux, & Gauvin, 1999). To this end, impulsivity has been measured in two ways using traditional neuropsychological testing; disinhibtion of unwanted or automatic responses and response latency. A number of researchers have found that patients with BN are quicker on measures of response speed (Cooper, Anastasiades, & Fairburn, 1992; Ferraro, Wunderlich, & Jocic, 1997; Murphy, Nutzinger, Paul, & Leplow, 2002, 2004) and are less able to inhibit unwanted responses (Ben-Tovim & Walker, 1991; Rosval *et al.*, 2006).

Whilst component cognitive processes of executive functioning have been well articulated, the cognitive profile of ED is seemingly variable and particularly inconsistent. Indeed, in a recent review of cognitive functioning in AN, there was no consensus on the existence of neuropsychological impairment despite research which has identified the presence of neurobiological abnormalities (Tchanturia, Campbell, Morris, & Treasure, 2005). The authors attributed the incongruity to small sample size and heterogeneous clinical groups (i.e. level of clinical severity). At the primary study level, examples are plentiful. For instance, Thompson (1993) found that patients with AN achieved significantly more categories on a measure of cognitive flexibility (i.e. the Wisconsin Card Sorting Test, WCST) compared to normal controls whilst Fassino *et al.* (2002) found that patients with AN achieved significantly less. Similar inconsistencies are noted in terms of test measure performance as it relates to attention and vigilance. For example, Green, Elliman, Wakeling, and Rogers (1996) examined attention and vigilance across controls and patients with AN but did not identify any significant group differences. Similarly, Jones, Duncan, Brouwers, and Mirsky (1991) examined sustained attention across controls, and patients with BN and AN, and also failed to identify any significant group differences. Notwithstanding however, Kingston, Szmukler, Andrews, Tress, and Desmond (1996) found that patients with AN performed significantly worse than controls prior to weight gain but were noted to perform comparably in the post-

weight gain period. Further, Szmukler *et al.* (1992) reported that patients with AN in the pre-weight gain period performed worse on the Trail Making Test (parts A and B) but performed similarly on the Wechsler Adult Intelligence Test – Revised (WAIS-R) digit symbol task (also see Bosanac *et al.*, 2007; Ohrmann *et al.*, 2004). Within the cognitive domain of learning, further inconsistencies are apparent. For example, Kingston *et al.* (1996) did not find significant differences in learning performance across controls and patients with AN before or after weight gain whilst Szmukler *et al.* (1992) did not find any group differences on the Rey Auditory Verbal Learning Test or Serial Digit Learning Task. Yet, Witt, Ryan, and Hsu (1985) found that performance on the Symbol-Digit Learning Test was impaired in patients with AN relative to a control group. Inconsistencies are also apparent in the domain of memory. For example, Witt *et al.* (1985) did not find any significant differences on the Wechsler Memory Scales (WMS) when patients with AN were compared to controls. In contrast, however, Bayless *et al.* (2002) did not utilize a control group but did note that patients with AN performed poorly relative to normative scores on the recall and recognition components of the word list recall subtest of the WMS-III. Moreover, however, in a study that compared underweight patients with AN, weight-restored patients with AN, BN, and controls, it was found that only the underweight AN group performed significantly worse on memory measures (Jones *et al.*, 1991). As well, on a measure of free recall, Ferraro *et al.* (1997) reported that, relative to controls, patients with BN performed significantly poorer on the first trial of the test but not on subsequent trials (also see Bosanac *et al.*, 2007; Seed, Dixon, McCluskey, & Young, 2000). Lastly, according to the review by Lena, Fiocco, and Leyenaar (2004), visual-spatial deficits in ED have been consistently noted across studies (e.g. Jones *et al.*, 1991; Palazidou, Robinson, & Lishman, 1990; Sherman *et al.*, 2006; Szmukler *et al.* 1992; Thompson, 1993) suggesting that this aspect of cognition may be particularly important as it relates to the understanding of ED.

Together, the accumulated literature does suggest the presence of cognitive impairment in patients with AN and BN. Yet beyond being inconsistent, these independent studies also do not reveal the magnitude of impairment within and across studies whilst traditional narrative reviews typically only champion statistically significant group differences as evidence of cognitive impairment, and fail to give due consideration to the magnitude of impairment so to understand the severity and breadth of impairment and/or differences in cognitive profiles between patients with AN and BN. To this end, differential patterns of cognitive impairment between patients with AN and BN would seemingly be important to articulate so to further our understanding of their putative causes, maintenance behaviour, and treatment. Hence, the present review on the subject seeks to articulate the magnitude of cognitive impairment in both patients with AN and BN by quantitatively synthesizing the existing literature using meta-analytic methodology so to address the following questions:

(1) What is the strength and consistency of cognitive impairment in patients with AN and BN? In other words, do cognitive tests provide reliable evidence of impairment in AN and BN and what is the average magnitude of difference between patients and healthy controls (HCs)?
(2) Do tests of specific cognitive function (e.g. mental flexibility, memory) reveal similar magnitudes of difference between patients with AN and BN and controls or are some aspects of cognitive performance spared in these illnesses?

(3) Are there relationships between cognitive impairment and clinical and demographic attributes of patients and controls?
(4) Do patients with AN and BN differ in terms of their respective cognitive profiles?

Method

Meta-analysis

We employed standard meta-analytic techniques to our review of the literature (Cooper & Hedges, 1994; Hedges & Olken, 1985; Rosenthal, 1991, 1995). In addition to solving problems with traditional narrative reviews (Wolf, 1986), meta-analysis provides tools for the analysis of magnitude. Magnitude can be indexed with the effect size estimate *d* that is meant to reflect the degree to which the dependent variable is present in the sample group or the degree to which the null hypothesis is false (Cohen, 1988). In mathematical terms, *d* is the difference between two group means calibrated in pooled standard deviation units. Eligible research studies comprising a common dependent variable as well as statistics that can be transformed into effect sizes are viewed as a population to be systematically sampled and surveyed. Individual study results (typically means and standard deviations from each group) and relevant moderator variables can be abstracted, quantified, and coded, and assembled into a database that is statistically analysed (Lipsey & Wilson, 1993). The main statistic presented in a meta-analysis is the mean effect size, which is meant to reflect the average individual effect across the sample of studies included in the synthesis. Moderator variables are then correlated to the effect size in order to tease out relationships of subject characteristics that may influence the magnitude of the size of effect between the groups being compared. To ascertain how robust our findings were, Orwin's (1983) fail safe *N* formula was also utilized so to provide an index of how many studies would be theoretically needed to overturn the obtained effect size and yield an insignificant effect (i.e. $d = 0.2$). The effect sizes were also transformed into a non-overlap percentage using Cohen's (1988) idealized distributions, which can be further transformed into an overlap percentage (OL%) to articulate the meaningfulness of an effect size (Zakzanis, 1998, 2001). The OL% statistic represents the degree of overlap by subtracting the non-overlap from 100. In the present context, this hypothetical overlap statistic used represents cognitive test sensitivity, or the percentage of patients who perform unlike any normal control participant in terms of cognitive impairment on a given cognitive test measure. Lastly, any moderator variables of interest were regressed to the effect size in order to tease out relationships of subject characteristics that may have impacted upon the cognitive test findings.

Finally, it should be noted that statistical analysis of meta-analytic studies is not entirely uncontroversial (see Hunter & Schmidt, 1990). A problem with any meta-analytical review of the literature is that primary studies vary in sample size, and that independent variables are not uncorrelated. As Van Horn and McManus (1992) did, we have used a correlational analysis to assess the independent effects of moderator variables. However, the crucial characteristic is the number of subjects, which paradoxically can sometimes mean that their statistical power is surprisingly low, despite apparently large subject numbers (Van Horn & McManus, 1992). In using univariate and multivariate analysis of studies we have followed Van Horn and McManus (1992) in not attempting to take any account of the differing sample sizes in

studies, since despite the concerns of Hedges and Olkin (1985), we have also accepted the argument of Hunter and Schmidt (1990; p. 408) that such problems pale into insignificance in comparison with the problems posed by low power in such studies. In assessing the potential effects of moderator variables we have therefore used unweighted population estimates from individual studies (see Van Horn & McManus, 1992).

Search strategy, selection criteria, and effect size analysis

Articles were identified by way of an extensive literature search of on-line databases (The Cochrane Library, PsychINFO, and MEDLINE). The search was limited to published English-language articles with human participants. The key terms *eating disorder*, anorexia, and bulimia* were used in combination with a number of neuropsychology-related terms including; *neuropsych**, *neurocog**, *cognitive impairment*, *attention*, *vigilance*, *memory*, *decision making*, *impuls**, *visuospatial*, *executive function*, *executive dysfunction*, *motor*, *psychomotor*, *processing speed*, *set shifting*, *rigidity*, *flexibility*, *perseveration*, *working memory*, *intelligence*, *Wisconsin Card Sort*, *Trails*, *Brixton*, *Stroop*, and *Gambling Task*. A secondary search involved checking the reference sections of relevant review and meta-analytic papers for articles that may have been missed in the computerized search.

The search identified 71 potential studies. These studies were chosen based on the following criteria; (a) comparison of a clinical group to a HC group, (b) inclusion of a clinical population sample composed of either BN or AN, (c) inclusion of at least one neuropsychological test measure, (d) published between 1980 (to coincide with the publication of the *Diagnostic and Statistical Manual of Mental Disorders*, DSM-III) and 2008, and (e) studies had to include sufficient statistical information to allow for effect sizes to be calculated. To this end specifically, effect sizes were derived whenever means and standard deviations were reported. Effect sizes were calculated from inferential statistics based on formulas provided by Wolf (1986) when primary studies did not report central tendency and dispersion data. Effect sizes were not derived from *p* values due to the unreliability of making inferences about magnitude on the basis of this statistic. The statistical software Meta-Analysis 5.3 was used to calculate effect sizes (Schwarzer, 1989).

Of the 71 articles, 16 were subsequently excluded due to (a) lack of an HC group ($N = 3$), (b) the authors combining the AN and BN groups for analysis ($N = 3$), (c) absence of statistics needed to calculate an effect size ($N = 5$), or (d) lack of appropriate neuropsychological test measures [i.e. experimental tasks and neuropsychological tests that did not fall under the predetermined cognitive domains ($N = 5$)]. Additionally, 5 of the articles were foreign language articles which were unable to be translated and 13 of the articles were unobtainable (i.e. no response from authors when contacted). In addition, the same data was reported twice across two articles, accordingly, only the first article was used to acquire the statistical data (i.e. Murphy *et al.*, 2002, 2004) Overall, a total of 36 studies were included in the present analysis.

Recorded variables

Recorded variables from each study used in our meta-analysis included the full study reference and any moderator variables reported [e.g. age, duration of illness, age onset, body mass index (BMI)]. Effect sizes were calculated for each neuropsychological test

that measured some aspect of cognitive functioning. To this end, organizing the myriad of cognitive test variables reported in the literature into a coherent classification was a major challenge. Several strategies exist in the literature for organizing diverse tests into categories of cognitive function, and each of these strategies has advantages and disadvantages. First, there exist *a priori* approaches such as the Lezak *et al.* (2004) classification, which are influenced by theoretical and practice related considerations about the test measures and their putative underlying processes. For example, Lezak *et al.* (2004) include motor and executive ability tasks in the chapter, presumably on the basis of a common substrate in the frontal brain or some other assumed link. Such classifications have no quantitative statistical underpinning, and even advocates of this approach admit to an element of arbitrariness in test organization (see Lezak *et al.*, 2004). A second approach is based on factor analytic studies of neuropsychological test batteries (see Goldstein, 1984). Factor analysis provides a quantitative description that relates different tests to a smaller number of underlying abilities. The validity of this approach however as a general strategy for organizing tests in a meta-analysis depends in part on the availability of factor analyses that include all of the test in the literature on cognitive function in ED. In the present case, we were unable to find factor analytic studies of eating disorder patient samples that included all, or even most, of the cognitive test variables reported in the literature. Finally, it is possible to avoid constructs altogether and simply compile effects for individual tests. This approach incorporates the fewest assumptions about the date, although it is unwieldy in view of the dozens of tests in common use and the inconsistency with which different scores from the same test are reported in the literature (e.g. categories vs. perseverative errors on the WCST; see Heaton, Chelune, Talley, Kay, & Curtiss, 1993).

A further consideration is that regardless of classification method several component processes probably influence many cognitive tests. Thus, scores on the Vocabulary subtest of the WAIS may reflect both language abilities and general intelligence whilst scores on the Trail Making Test may reflect visual scanning and perception but also motor speed, hand–eye coordination, and attention (Lezak *et al.*, 2004). Hence it may be misleading to categorize tests on the basis of a faulty assumption that test performance is determined by only one process. Accordingly, we tried to avoid aggregating different tests and their effect sizes into our own hypothetical categories and adopted those defined by Lezak *et al.* (2004). These included, intelligence (full, performance, and verbal), memory [verbal and visual (immediate, short delay, long delay, and recognition)], working memory (including spatial working memory), processing speed, psychomotor speed, motor function, attention, and executive function (set shifting, inhibition, decision making, and problem solving). Table 1 illustrates the specific neuropsychological tests that were aggregated into each generated category.

To meet the assumption of independence, when multiple test variables in a study contributed to any one neuropsychological domain, the effect sizes were pooled together into a mean effect size. For each neuropsychological domain, these mean effect sizes were aggregated and analysed further. As per Hedges and Olkin (1985, p. 256), homogeneity statistics were used in parallel to confirm the identity of outliers. If the homogeneity statistic (i.e. Q_T) was significant at a alpha level of .05, effect size outliers were first identified using the interquartile range (i.e. between the 25th and 75th percentile) as a reference point. As a general criterion, effect sizes that were greater than two times the interquartile range from the nearest quartile were considered outliers and were subsequently removed. This procedure identified four outliers for the AN dataset and two outliers for the BN dataset. If there were no outliers according to the above

Table 1. Neurocognitive categories and tests used in the meta-analysis

Construct	Recorded test variables
Intelligence	CFT-20
	MWT-P
	NART
	WAIS
	WIP-Q
	WTAR
	VIQ
	PIQ
Visuospatial skills	Block design
	BMAPS-visuospatial and visual analysis
	BORB 1 and 2
	CANTAB-match-to-sample
	Eckman faces
	Facial recognition
	Rey-CFT copy
	WAIS-object assembly, picture completion
Verbal skills	Vocabulary
	Confrontation naming
	Verbal fluency
Verbal memory	BMAPS
	CDR-Ver
	CVLT
	RAVLT
Visual memory	BMAPS
	CDR-visual-recognition
	RCFT
Working memory	Block span
	CANTAB-spatial-span
	CDR-quality-of-working-memory
	CFT-20-number-sequencing
	Digit span
	Supraspan
Processing speed	BMAPS
	CDR-RT-think
	Stop-signal (signal task)
	Stroop W
	Stroop C
	Symbol digit modalities
	TMT A
	WAIS-digit symbol
Psychomotor speed	CDR-RT-Sim
	Simple RT
	TMT-motor
Motor	CDR tapping
	Finger tapping test
Attention	CPT
	Focused attention
	PASAT 2 and 4
	Selective attention

Table 1. (*Continued*)

Construct	Recorded test variables
Executive function	Vigilance WMS-attention/concentration Decision making: IGT, GDT Inhibition: Go/No Go, stop-signal, Stroop I Set shift: BMCST, Brixton, COWAT, TMT B, WCST, Weigl's sorting Problem solving: Tower of London, Tower of Hanoi, matrix reasoning

criterion or if the homogeneity statistic remained significant despite the removal of outliers, a random effects model was employed (i.e. vs. a fixed effects model).

Results

Twenty-seven and fourteen studies of cognitive functioning in patients with AN and BN, respectively, met criteria for inclusion into the present analysis. In total, cognitive test results from 608 patients with AN, and 347 patients with BN were recorded across studies published between 1980 and mid-2008. Descriptive statistics for the study sets are shown in Table 2. Descriptive data available for demographic and clinical variables, by patient group, is presented in Table 3. The published literature reflects patient samples (both for AN and BN) aged approximately 23–24 years of age with a duration of illness of approximately 5–6 years. It is worth noting that a great deal of variability was noted between studies with respect to the patients samples used, neuropsychological measures administered, and descriptive statistics reported. To this end, symptomatic variability between patient groups was found in AN patient samples only as BN patients did not differ with respect to BMI or illness subtype (i.e. BN-purging and BN-non-purging). For AN samples, however, patients ranged from acute stage AN to weight-recovered AN and long-term recovered AN and also differed with respect to subtype (i.e. AN-binge/purge and AN-restrictive). For the purposes of this analysis, however, all AN patient samples, irrespective of stage of illness or subtype, were included into the meta-analysis so as to ensure greater generalizability of the findings.

A second aspect of the study set worth noting relates to symptomatic variability between patient groups and the rate and type of diagnosed comorbid disorder. Nine of the studies reported that their BN or AN patient samples were also diagnosed with a second disorder, namely an affective disorder [anxiety, depression, dysthymia, panic disorder ($N = 9$)], conduct disorder ($N = 1$), or personality disorder [borderline, histrionic ($N = 2$)] according to DSM-III or DSM-IV (American Psychiatric Association, 1994) criteria (*Note*. neither comorbidity or psychiatric exclusion criteria was mentioned in 21 of the papers). In addition, although these demographic variables, including history of substance abuse, were controlled for in many of the studies, scores on depression and anxiety scales were almost universally higher in patient populations (compared to normed scores as well as the HC group used for comparison in the study).

The study set also revealed inconsistencies in the types of cognitive test measures employed across studies. For example, although some researchers, interested in

Table 2. Descriptive statistics for study sets

	AN			BN		
	N.St	N.HC	N.AN	N.St	N.HC	N.BN
Full IQ	6	115	124	3	52	49
Performance IQ	3	58	65	0	0	0
Verbal IQ	3	58	65	0	0	0
Visuospatial skills	9	226	237	2	44	39
Verbal skills	7	159	158	2	30	30
Verbal memory						
Immediate	9	173	189	4	88	75
Short delay	4	70	77	0	0	0
Long delay	8	163	184	3	74	61
Recognition	4	76	87	2	58	48
Visual memory						
Immediate	2	35	34	0	0	0
Long delay	5	123	130	2	30	30
Recognition	4	72	80	0	0	0
Working memory	9	190	196	3	72	59
Processing speed	12	344	349	5	172	103
Psychomotor speed	6	161	189	1	17	19
Motor	3	58	62	1	16	13
Attention	7	132	134	4	81	80
Executive function						
Set shifting	15	419	453	6	137	142
Inhibition	6	242	157	5	244	158
Decision making	4	169	168	3	50	47
Problem solving	3	62	61	1	14	14

Notes. N.St, number of studies; N.HC, total number of HCs; N.AN, number of patients with AN; N.BN, number of patients with BN. If a cognitive domain contained only one effect size, meta-analytic calculations were not performed.

Table 3. Descriptive statistics for patients samples across study sets

	Mdn	*M*	*SD*	Min	Max	*N*
AN						
Age	24.50	23.60	3.68	14.50	29.12	27
BMI	15.30	16.13	2.09	13.20	22.20	35
Age of onset	17.34	17.03	1.60	14.30	20	10
Duration (years)	5.67	5.90	3.64	.74	12.91	13
BN						
Age	23.50	23.74	2.70	19.10	28.31	12
BMI	21.69	21.32	1.58	18.28	23.52	10
Age of onset	NR	NR	NR	NR	NR	0
Duration (years)	6.40	7.52	2.65	5.61	10.54	3

Notes. Mdn, median; *M*, mean; *SD*, standard deviation; *N*, number of studies; BMI, body mass index; NR, not reported. Descriptive statistics for AN only include those in acute stages (i.e. not fully recovered).

Table 4. Weighted mean effect sizes for neuropsychological variables in BN and AN

Neuropsychological variables	ED group	Nd	Md	*SE*	Lower CI	Upper CI	*Q*	*p* for *Q*	Nfs	*U*
Full IQ	AN	8	.29	.16	0.05	0.53	11.33	.12	4	20.76
	BN*	3	.11	.69	−1.16	1.39	2.26	.32	1	8.70
Performance IQ	AN*	3	.18	.41	−0.58	0.94	2.13	.35	0	13.46
Verbal IQ	AN	3	.21	.24	−0.15	0.57	3.39	.18	0	15.27
Visuospatial skills	AN	9	.37	.12	0.18	0.55	9.13	.33	8	25.41
	BN	2	.43	.21	0.00	0.87	.85	.36	2	29.27
Verbal skills	AN	7	.03	.06	−0.19	0.25	2.13	.91	6	2.24
	BN	2	.23	.19	−0.28	0.73	.51	.48	0	16.49
Verbal memory										
Immediate	AN*	9	.55	.20	0.23	0.88	9.39	.31	16	35.72
	BN	4	.68	.19	0.36	0.99	3.00	.39	10	41.87
Short delay	AN	4	.29	.21	−0.04	0.62	3.94	.27	2	20.54
Long delay	AN	8	.16	.17	−0.05	0.37	12.78	.08	2	12.02
	BN*	3	.70	.41	−0.01	1.40	2.43	.30	7	42.75
Recognition	AN	4	.36	.13	0.05	0.68	1.43	.70	3	25.14
	BN	2	.14	.10	−0.25	0.52	.22	.64	1	10.47
Visual memory										
Immediate	AN	2	.73	.36	0.24	1.23	2.06	.15	5	44.53
Long delay	AN	5	.59	.11	0.34	0.85	3.94	.41	10	37.77
	BN*	2	.26	.88	−1.45	1.98	1.00	.32	1	19.06
Recognition	AN	4	.43	.10	0.10	0.75	1.07	.78	5	28.96
Working memory	AN	9	.35	.08	0.15	0.55	3.94	.86	7	24.52
	BN	3	.12	.11	−0.22	0.47	.67	.72	1	9.30
Processing speed	AN	12	.52	.08	0.36	0.68	15.54	.16	19	34.04
	BN	5	−.03	.08	−0.28	0.22	1.45	.84	4	2.41
Psychomotor speed	AN	6	.62	.14	0.40	0.84	5.33	.38	13	39.27
Motor function	AN*	3	.93	.58	−0.30	2.15	1.74	.42	11	52.56
Attention	AN	7	.23	.09	−0.02	0.47	3.62	.73	1	16.62
	BN*	4	.26	.42	−0.58	1.11	2.84	.42	1	19.03

Table 4. (*Continued*)

Neuropsychological variables	ED group	Nd	Md	*SE*	Lower CI	Upper CI	*Q*	*p* for *Q*	Nfs	*U*
Executive function										
Set shifting	AN	15	.35	.06	0.21	0.48	10.87	.70	11	24.29
	BN	7	.16	.16	−0.07	0.39	9.59	.14	1	11.97
Inhibition	AN	6	.34	.13	0.14	0.55	4.37	.50	4	24.01
	BN	5	.43	.13	0.21	0.64	5.18	.27	6	28.85
Decision making	AN	4	.57	.12	0.35	0.79	3.27	.35	7	36.66
	BN	3	.85	.11	0.43	1.26	.55	.76	10	49.39
Problem solving	AN	3	.43	.08	0.07	0.79	.40	.82	3	29.13

Notes. An asterisk (*) denotes where random effects were employed in the calculations. Negative scores imply that the ED group performed better than the HC group. Nd, number of independent effect sizes; Md, mean effect size; *SE*, standard error of estimate of Cohen's *d*; CI, confidence interval (95% limits); *Q*, *Q* statistic (a test of homogeneity of variance); *p* for *Q*, probability that *Q* statistic is significantly different than zero; Nfs, fale-safe *N* statistic (Orwin, 1983); *U*, percent non-overlap of effect sizes based on Cohen's (1988) idealized population distribution.

executive functioning in the ED have used measures such as the WCST, trail making test (TMT), and Stroop, others have used newer, less established measures such as the Computer Automated Neuropsychological Test Battery (CANTAB) and Computerized Drug Research Battery (CDR). Additionally, the way in which data was analysed by each researcher was highly variable. For example, on the Iowa Gambling Task (IGT), a decision-making task in which participants are required to choose cards from one of four decks, two of which are advantageous and two disadvantageous, there are many differences in the methods used to analyse performance. Cavedini *et al.* (2006) examined the number of advantageous picks minus disadvantageous whereas Bosanac *et al.* (2007) looked only at the total number of advantageous picks. Still different was Boeka and Lokken's (2006) analysis of only the last 50 trials (out of 100). Although data reports on standard cognitive test measures did not reach the same degree of variability, there are still moderate discrepancies in what individual studies chose to report. For example, on the TMT some researchers reported the latency to completion as well as the errors made while some researchers chose only to report the latency. Lastly, a degree of variability was also found amongst the studies with respect to the data that was actually reported. As already noted, many of the studies chose to omit important information regarding patient characteristics (e.g. psychiatric history or co-morbidities, duration of illness).

Finally, effect size summaries are presented for each cognitive domain by eating disorder in Table 4. Mean effect sizes weighted by their inverse variance are reported. In the correction procedure, effect sizes are combined by averaging d values, with each d weighted by the reciprocal of its variance (see Wolf, 1986). This procedure gives greatest weight to the most reliably estimated study effect sizes – those with the largest sample sizes (Hedges & Olkin, 1985). In addition, standard error scores for each mean effect size, 95% confidence intervals, homogeneity statistics, Orwin's fail-safe N statistic (using a conservative criterion of $d = 0.2$; Orwin, 1983), and per cent of non-overlap of effect size based on Cohen's (1988) idealized population distribution are also presented. Overall, for patients with AN, the largest effect sizes were noted for verbal memory (immediate), visual memory (immediate and long delay), psychomotor speed, motor function, and decision making. For patients with BN, immediate verbal memory and decision-making ability were relatively large effect sizes, but in contrast to patients with AN, so was long delay verbal memory.

Discussion

The present review of cognitive test findings in patients with AN and BN sought to articulate the magnitude of cognitive impairment in both patient groups by quantitatively synthesizing the existing literature using meta-analytic methodology. Overall, the results illustrate that cognitive tests provide modest evidence of impairment in AN and BN. In addition, the results also suggest that the breadth and magnitude of cognitive impairment is more severe in patients with AN than patients with BN. As well, there seems to be no relationship between cognitive impairment and clinical and demographic attributes of patients and controls beyond BMI and severity of cognitive impairment in patients with AN. That is, as body mass decreases, the severity of cognitive impairment increases. The results of the meta-analysis also suggests that patients with AN and BN differ in terms of their respective cognitive profiles. It is important to note however that due to the relatively small number of published studies

investigating cognitive impairment of BN (a total of 14 studies reported here), this limited the number of valid comparisons that could be made across cognitive domains.

With regards to cognitive impairment in AN specifically, it was surprising to find that only a small number of studies have been conducted investigating higher order functioning, such as decision making and problem solving, in patients with AN. Our results indicate that patients with AN are more likely than HCs to make disadvantageous choices on measures of motivated decision making and take significantly more time to solve complex problems. Traditional theory with respect to motivated decision making is that decision-making deficits are the result of either set-shifting deficits (Lawrence *et al.*, 2006) or impulsivity/disinhibition (Bechara, Damasio, Damasio, & Anderson, 1994). To this end, set-shifting ability was commonly investigated. Interestingly, however, it seems that researchers may have overemphasized the reliability of set-shifting impairment in patients with AN given that the current review of the literature did not find much in terms of support for impairment by way of a negligible effect size. An important distinction however is noted between performance on visual or perceptual set-shifting tasks (e.g. Trail Making Task B; WCST) and verbal set-shifting tasks (e.g. FAS, Controlled Oral Word Association Task) in that performance on the former tasks suggest more pronounced impairment on the later. In keeping with the obtained effect sizes for visuospatial skills, visual memory (immediate, delayed, and recognition), and to some extent also, spatial working memory, it is plausible to conclude that patients with AN are primarily impaired in terms of spatial perception or representation. To this end, impairment in spatial perception and representation might explain the distorted assessment of body image in patients with AN (e.g. Raudenbush & Zellner, 1997; Zellner, Harner, & Adler, 1989) who typically rate their ideal figures (what they would like to look like) and opposite figures (what they believe the opposite sex finds attractive) thinner than their current figures (how they believe they currently appear).

At the same time, although spatial perceptual and representation impairment may be most pervasive in the neuropsychology of AN, this does not suggest that verbal impairment is nonexistent. That is, the results of our meta-analysis illustrate that verbal skills are also impaired in AN. Additionally, patients with AN illustrate moderately impaired verbal recall. To this end specifically, a more qualitative analysis of the findings suggest that impairment is more evident in the first two or three trials of a learning task suggesting that learning for these patients is more effortful and hence, repetition is required so to ensure consolidation and consequent retrieval. This interpretation is consistent with primary experimental findings that have revealed that patients with AN display deficits in working memory (Thompson, 1993).

Processing speed has also been well researched in patients with AN. To this end, the present meta-analysis found evidence of moderate impairment in terms of processing speed in that patients were found to perform slower on these measures when compared to matched HCs. This impairment may however be peripheral, and secondary to slowed motor functioning, rather than slowed mental processing speed given that a large percentage of patients with AN were found to be impaired on specific test measures of psychomotor speed. This interpretation seems at the very least plausible in keeping with the observation that as task demand became more complex (i.e. from motor speed, to manual dexterity speed, to processing speed) smaller effect sizes were garnered between patients diagnosed with AN and HCs.

With regards to cognitive impairment in BN, patients illustrate little to absolute no impairment in terms of performance on aggregate intelligence tests (e.g. on measures

of IQ), visuospatial skills, visual memory, working memory, spatial working memory, processing speed, or set shifting compared to HCs. Patients with BN do however illustrate some evidence of impairment in terms of impulsivity or disinhibition, in that approximately 21.3% of patients scored worse on measures of impulsivity or disinhibition compared to controls. It is important to note however that traditional cognitive test measures of impulsivity or disinhibition have been questioned in terms of their ecological validity and as such, may not be both sensitive and predictive of real world behaviour (see Zakzanis, Graham, Campbell, & Mraz, 2004). Similar to patients with AN, patients with BN were impaired on measures of immediate verbal memory which is inconsistent with the hypotheses regarding the cognitive mechanisms of the observed behavioural patterns in BN. Furthermore, patients with BN performed significantly worse than HCs on measures of delayed verbal memory. It is important to note, however, that with a limited number of studies in hand, additional research is necessary so to articulate this finding further.

Also consistent with AN, patients with BN made more disadvantageous choices on measures of motivated decision making compared to controls, but at a greater severity than patients with AN. The underlying mechanism(s) for this finding is unclear and warrants investigation. Indeed, it has been posited that impulsivity may account for disadvantageous decision making although this is inconsistent in keeping with the findings of the present meta-analysis as there exists little difference on measures of inhibition between patients with BN and HCs. Again however, it has been noted that neuropsychological test measures may not be sensitive enough to detect impulsive behaviour, and as such, future research with various experimental measures of impulsivity should be employed in hand with decision-making paradigms to help elucidate the contributory role of this cognitive function.

Summary and conclusions

In keeping with the results of this meta-analysis of cognitive impairment in AN and BN, it is apparent that further investigations are needed so to address a number of matters inherent in the existing literature and brought forth by way of this review. That is, future research might wish to examine the differences between verbal and visual stimuli so as to determine whether the type of stimuli contributes to the observed performance deficits on cognitive test measures with similar constructs (e.g. set shifting). Additionally, future investigations might wish to examine the contributory role of co-morbid disorder(s) on cognitive test performance in patients with AN and BN. The contributory role of depression, anxiety, personality disorder, and learning disability on cognitive in eating disordered patients must be examined if a clearer picture of cognitive impairment is to emerge. Furthermore, the effects of malnutrition and consequent BMI should be examined directly so to determine more directly its impact on cognitive functioning. As well, the observation that there were sometimes large differences in effect size magnitude between variable measures on the same cognitive test (e.g. latency vs. errors) suggests that experimental measures may serve more purposeful when delineating multifactorial cognitive constructs. For example, Seed, Mccue, Wesnes, Dahabra, and Young (2002) found that patients with AN were slower to process information than normal controls but were less likely to make an error on an attentional task. This demonstrates that averaging scores on a single cognitive test measure may mask important findings that may help further articulate the neuropsychology of ED.

Although further studies are needed to help elucidate the nature and pattern of cognitive functioning in ED, the present meta-analysis demonstrates reliable evidence of cognitive impairment specific to AN and BN that is related to BMI in AN in terms of its severity, and is differentially impaired between disorders. Together, these results suggest that disturbed cognition is figural in the presentation of ED and may serve to play an integral role in its cause and maintenance.

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