Central Coherence in Adolescents with Bulimia Nervosa Spectrum Eating Disorders

Article *in* International Journal of Eating Disorders · August 2014

Impact Factor: 3.13 · DOI: 10.1002/eat.22340

11 authors, including:

Alison M Darcy
Stanford University
27 PUBLICATIONS **168** CITATIONS
SEE PROFILE

Kathleen Kara Fitzpatrick
Stanford Medicine
39 PUBLICATIONS **732** CITATIONS
SEE PROFILE

Vandana Aspen
Stanford University
20 PUBLICATIONS **237** CITATIONS
SEE PROFILE

James Lock
Stanford Medicine
162 PUBLICATIONS **3,802** CITATIONS
SEE PROFILE

Central Coherence in Adolescents with Bulimia Nervosa Spectrum Eating Disorders

Alison M. Darcy, PhD[1]*
Kathleen Kara Fitzpatrick, PhD[1]
Stephanie M. Manasse, BA[1]
Nandini Datta, BS[1]
Megan Klabunde, PhD[2]
Danielle Colborn, PhD[1]
Vandana Aspen, PhD[1]
Colleen Stiles-Shields, MA, MS[3]
Zandre Labuschagne, MA[3]
Daniel Le Grange, PhD[3]
James Lock, MD, PhD[1]

ABSTRACT:
Background: Weak central coherence—a tendency to process details at the expense of the gestalt—has been observed among adults with bulimia nervosa (BN) and is a potential candidate endophenotype for eating disorders (EDs). However, as BN behaviors typically onset during adolescence it is important to assess central coherence in this younger age group to determine whether the findings in adults are likely a result of BN or present earlier in the evolution of the disorder. This study examines whether the detail-oriented and fragmented cognitive inefficiency observed among adults with BN is observable among adolescents with shorter illness duration, relative to healthy controls.

Method: The Rey-Osterrieth Complex Figure Test (RCFT) was administered to a total of 47 adolescents with DSM5 BN, 42 with purging disorder (PD), and 25 healthy controls (HC). Performance on this measure was compared across the three groups.

Results: Those with BN and PD demonstrated significantly worse accuracy scores compared to controls in the copy and delayed recall condition with a moderate effect size. These findings were exacerbated when symptoms of BN increased.

Discussion: Poorer accuracy scores reflect a fragmented and piecemeal strategy that interferes with visual-spatial integration in BN spectrum disorders. This cognitive inefficiency likely contributes to broad difficulties in executive functioning in this population especially in the context of worsening bulimic symptoms. The findings of this study support the hypothesis that poor global integration may constitute a cognitive endophenotype for BN.

Keywords: bulimia nervosa; neurocognition; central-coherence; endophenotype; eating disorders; adolescents

Resumen
Antecedentes: La coherencia central débil– que es la tendencia a procesar detalles a expensas de la gestalt – ha sido observada en adultos con bulimia nervosa (BN) y es un candidato potencial de endofenotipo para trastornos de la conducta alimentaria. Sin embargo, dado que los comportamientos de la BN se inician típicamente durante la adolescencia, es importante evaluar la coherencia central en este grupo de edad joven para determinar si los hallazgos en adultos son resultado de la BN o están presentes antes de la evolución de este trastorno. Este estudio analiza si la ineficacia cognitiva fragmentada y orientada a los detalles que se observa entre los adultos con BN es observable entre adolescentes con una duración más corta de la enfermedad, relativo a un control saludable.

Métodos: El test de la figura compleja de Rey-Osterrieth (RCFT) fue aplicado a 47 adolescentes con BN DSM5, a 42 con trastorno purgativo (PD) y a 25 controles saludables (HC). El desempeño en este estudio se comparó a través de los 3 grupos.

Resultados: Aquellos individuos con BN y PD mostraron puntajes de precisión significativamente peores en la copia, en comparación con los controles (HC), y condición de recuerdo diferido con tamaño de efecto moderado. Estos hallazgos se encontraban exacerbados cuando los síntomas de la BN se incrementaban.

Accepted 15 July 2014
Supported by NIH SPO#37129 and R01 MH079979 (Clinical Trial Registration Identifier: NCT00879151) from National Institutes of Health and by Hilda and Preston Davis Foundation.
*Correspondence to: Alison M. Darcy, PhD, Department of Psychiatry and Behavioral Sciences, Stanford University School of Medicine, 401 Quarry Road, Stanford, CA 94305. E-mail: adarcy@stanford.edu
[1] Department of Psychiatry and Behavioral Sciences, Child and Adolescent Psychiatry, Stanford University School of Medicine, Stanford, California
[2] Department of Psychiatry and Behavioral Sciences, Center for Interdisciplinary Brain Sciences Research, Stanford University School of Medicine, Stanford, California
[3] Department of Psychiatry and Behavioral Neuroscience, The University of Chicago Medical Center, Chicago, Illinois.
Published online 00 Month 2014 in Wiley Online Library (wileyonlinelibrary.com). DOI: 10.1002/eat.22340

Conclusiones: Las puntuaciones que exhibieron precisión más pobre reflejaron una estrategia secuencial y fragmentada, la cual interfiere con la integración visuo espacial en trastornos del espectro de la BN. Esta ineficiencia cognitiva posiblemente contribuye a aumentar las dificultades en el funcionamiento ejecutivo de esta población, especialmente en el contexto del empeoramiento de los síntomas de la BN. Los hallazgos de este estudio sostienen la hipótesis que la integración global pobre puede constituir un endofenotipo cognitivo para la BN. © 2014 Wiley Periodicals, Inc.

Palabras clave: Bulimia Nervosa; Neurocognición; Coherencia-central; endofenotipo; trastornos de la conducta alimentaria; adolescentes

(Int J Eat Disord 2014; 00:000000)

Bulimia nervosa (BN) is a serious psychiatric disorder that usually arises in adolescence and is characterized by recurrent episodes of binge eating and compensatory behaviors.[1] Recent work on neurocognition in eating disorders (EDs) suggests that weak central coherence—a cognitive-processing bias toward superior local processing and a reduced tendency toward global, or gestalt, processing—may be a cognitive endophenotype for EDs.[2] Among individuals with autism disorder, data to support the validity of the central coherence construct are somewhat mixed[3–5] leading some researchers to explore alternative underlying mechanisms.[6] In the ED field, data suggests that central coherence is a somewhat complex, non-unitary construct. For example, while global processing difficulty has been observed in adults across the ED spectrum,[7] superior attention to detail appears to be a core feature in anorexia nervosa (AN) and poor global integration appears to be more relevant to BN.[2] For example, several studies using the Rey-Osterrieth Complex Figure Test (RCFT) have found that adults with BN demonstrate inefficiencies in central coherence relative to healthy controls (HCs)[7–10] by scoring lower on central coherence and accuracy scores on immediate and delayed recall. Accuracy scores in the delayed recall condition on the RCFT provide information on attention, concentration, visual-spatial ability and non-verbal memory. These lower accuracy scores from the delayed recall condition suggest an inefficiency of detail-focus for non-verbal memory. In addition, Harrison et al.[11] found that recovered ED patients demonstrated superior detail processing compared to HCs. However, global integration difficulties were more pronounced among those with AN than BN relative to HCs. In addition, body mass index (BMI) was negatively associated with performance on a global integration task leading the authors to conclude that global integration difficulties may be state-related while the detail processing dimension of central coherence may represent a trait characteristic for EDs more generally. Similarly, more recent work incorporating healthy sisters of those with BN, AN, and a healthy control comparison group revealed that those with BN and their unaffected sisters demonstrate a profile more in line with global integration difficulties, whereas superior detail focus was more strongly associated with AN.[2]

Overall, the literature demonstrates that adults with AN and BN exhibit relative inefficiencies in central coherence. However to our knowledge no study has explored whether these problems exist in adolescent samples who present with shorter illness duration compared to adult samples. This is important for two reasons. First, the underlying neurophysiological mechanism hypothesized to underline central coherence is a disturbance in the natural transition from localized to distributed or collaborative brain function that usually occurs as a result of significant myelination and synaptic pruning during the critical period of adolescence.[12] Second, because illness onset often occurs during adolescence, failing to observe difficulties earlier on in the course of illness might suggest that difficulties observed in adults are due to the effects of illness over time. Additionally, in refining this concept, it is unclear whether one facet of central coherence (i.e., difficulties with global integration or superior detail focus) is of most relevance to BN. The aim of this study was to assess whether and what types of central coherence difficulties are evident in adolescents with BN-spectrum disorders relative to HCs. A secondary aim was to explore the relationship between central coherence and illness variables that might affect cognitive process (e.g., weight, binge/purge frequency) to better understand this construct. Given the heterogeneity in symptom presentation, we explored constructs among individuals with BN separately from those with purging disorder.

Method

One hundred and fourteen adolescents aged 12–18 years with BN-spectrum ED were recruited as part of a 2-site randomized clinical treatment trial for adolescents with BN (trial registration NCT00879151). The current study of neurocognitive process is a secondary study associated with this primary treatment study. For the purposes of the current study, 25 adolescent HCs were recruited from the community by advertisements placed on local and University notice boards and email listservs. The HC samples were recruited for two different studies. Prior to participation in the study, subjects signed informed consent (signed by parents for participants <18 years old and participants >18 years old signed assent forms). Institutional Review Boards at Stanford University and the University of Chicago approved the primary study from which the clinical data were drawn, and at Stanford for approval of the unrelated studies from which the HCs were recruited.

All clinical patients were female and recruited through a treatment trial for BN which for the purposes of the trial was defined as an average of one binge-eating episode (objective or subjective) and one purging episode per week within the past 3 months, with at least one binge-eating or purging episode occurring in the past month. Inclusion criteria for the treatment trial also included medical stability for outpatient treatment, English language proficiency with at least one parent speaking fluent English, and if on psychiatric medication, on a stable dose for at least two months. Exclusion criteria were diagnosis of a psychotic disorder or taking anti-psychotic medication, and any medical condition (e.g., diabetes, seizure, head injury) that could interfere with cognitive ability or weight maintenance. Participants with BN either met Diagnostic and Statistical Manual of Mental Disorders-Fifth Edition (DSM5) criteria for BN, or purging disorder (PD), diagnoses were determined at baseline by the Eating Disorder Examination (EDE, see below) administered by trained interviewers.

HCs were all female; had no psychiatric history as determined by the KSADS; no immediate family member with a history of eating disorder as measured by self-report; and were of normal weight, defined as >85% of median body weight. All participants were screened for eligibility and assessed by trained research assistants.

Measures

At baseline, all study participants were administered a full neuropsychological test battery including the following assessments in addition to the RCFT: four subtests from The Delis-Kaplan Executive Functioning System (Trail Making Task, Towers, Color-Word Interference, and Verbal Fluency) and the Brixton Spatial Anticipation Task. Data on tasks other than the RCFT have been reported elsewhere (Ref. Darcy et al., 2012). In addition, participants were given a measure of intelligence and a clinical measure of eating disorder psychopathology (EDE or EDE-Q, see below).

General Intelligence

Weschler Abbreviated Scale of Intelligence; Weschler Intelligence Scale for Children; and the Weschler Adult Intelligence Scale. For the purposes of this study, we used four subtests as a proxy for the full Weschler Abbreviated Scale of Intelligence test (Vocabulary, Similarities, Block Design, Matrix Reasoning) for the clinical samples, thus estimated, rather than full-scale IQ values, are used. HC children up to age 16 were administered the Weschler Intelligence Scale for Children and HC adolescents aged 16–18 were administered the Weschler Adult Intelligence Scale.

Psychological Assessments

Eating Disorders Examination and EDE-Questionnaire. The Eating Disorders Examination[13] (EDE) is a standardized, semi-structured interview that is considered the "gold standard" measure of ED psychopathology measuring severity of ED symptomology across four subscales: Dietary Restraint, Eating Concern, Shape Concern, and Weight Concern. The frequency of key ED behaviors, such as binging and purging, and severity of psychopathology such as dissatisfaction with weight and shape are rated over the past 28 days, or for diagnostic items, the previous 3 months. The psychometric properties of the EDE are sound and have been used in many treatment studies.[14,15] The EDE was administered to BN participants at baseline.

The EDE-Questionnaire (EDE-Q)[16] was administered to HC subjects and is a validated, short-form, self-report version of the EDE, that has demonstrated reliability for estimation of binge eating and purging among community samples.[17]

Schedule for Affective Disorders and Schizophrenia for School-Aged Children, Present and Lifetime Version. Psychiatric co-morbidity, duration of BN, and psychotropic medication use among BN subjects was determined by the Schedule for Affective Disorders and Schizophrenia for School-Aged Children, Present and Lifetime Version (KSADS-PL).[18] A widely used semi-structured interview, the KSADS-PL is a diagnostic tool for psychiatric disorders in children and adolescents.[18]

Central Coherence

Rey-Osterrieth Complex Figure Test. The RCFT[19,20] is a measure of visual-spatial constructional ability, nonverbal memory, and visual conceptual integration, used to assess central coherence. The RCFT consists of copy

TABLE 1. Demographic and descriptive variables (means [standard deviations]) for the groups

	BN ($n = 47$)	PD ($n = 42$)	HC ($n = 25$)	Comparison
Age (years)	16.19 (1.14)	15.59 (1.88)	15.52 (1.66)	$F(2, 111) = 2.24$, $p = .11$
Age (range)	14–18	12–18	12–18	
Estimated IQ	105.91 (9.87)	109.85 (12.57)	116.24 (10.90)	$F(2, 109) = 6.90$, $p = .002$
%MBW	109.90 (28.10)	107.21 (16.07)	103.72 (11.31)	$F(2, 110) = 0.681$, $p = .51$
Motor Speed	25.88 (10.70)	28.30(13.31)	23.36 (6.15)	$F(2, 96) = 1.503$, $p = .23$
Verbal fluency	38.95 (11.59)	40.64 (8.41)	41.27 (10.91)	$F(2, 91) = .345$, $p = .71$
One comorbidity (n)	9	17	NA	$\chi^2(2) = .474$, $p = .79$
Multiple comorbidity (n)	5	8	NA	
Depression diagnosis (n)	13	18	NA	$\chi^2(1) = 0.021$, $p = .89$
Ill duration (months)	18.78 (15.10)	18.89 (16.16)	NA	$t(86) = 0.034$, $p = .97$
Medication use (n)	5	3	NA	[a]
EDE-RES	3.36 (1.38)	3.36 (1.61)	NA	$t(87) = 0.014$, $p = .99$
EDE-EC	3.19 (1.47)	2.81 (1.39)	NA	$t(87) = 1.22$, $p = .22$
EDE-WC	3.92 (1.59)	3.70 (1.60)	NA	$t(87) = 0.629$, $p = .53$
EDE-SC	4.20 (1.58)	4.11 (1.25)	NA	$t(87) = 0.293$, $p = .77$
OBE episodes	18.68 (15.22)	4.00 (10.48)	NA	$t(81.88) = 5.344$, $p < .001$
SBE episodes	11.85 (13.09)	17.95 (19.80)	NA	$t(87) = 1.731$, $p = .087$
Vomiting episodes	31.57 (56.96)	21.21 (20.04)	NA	$t(87) = 1.118$, $p = .267$
Laxatives episodes	1.57 (5.08)	2.03 (9.50)	NA	$t(87) = 0.291$, $p = .772$
Driven exercise	13.62 (16.07)	15.51 (13.85)	NA	$t(87) = 0.596$, $p = .553$

Note: BN = bulimia nervosa group; PD = bulimia-type eating disorder not otherwise specified group; HC = healthy control group; MBW = median body weight; EDE = eating disorder examination; OBE = objective binge eating; SBE = subjective binge eating.
[a]Too few participants in each cell to conduct analysis.

and delayed recall conditions along with Style, Order, and Central Coherence Indexes and is videotaped to assist with inter-rater reliability. Subjects are first presented with the figure and instructed to copy the figure onto a blank piece of paper with colored pencils (Copy condition). The assessor changes the color of the pencil upon completion of an element of the figure, using a previously determined sequence of colors. In the first recall condition, the figure is removed from view and the participant is asked to re-draw the figure from memory immediately after completing the copy (immediate recall). Thirty minutes after the immediate recall, the participant is again asked to re-draw the figure from memory (delayed recall). Both recall conditions were presented without warning to the participants and no spatial tasks were administered to participants between recall conditions.

The scoring procedure was originally developed by Booth[21] to assess the drawing accuracy of the RCFT figures (Accuracy) in copy and recall conditions, as well as Indexes. The Performance Index score is derived from accuracy and placement of each of the 18 elements of the figure. Each element is given an accuracy score of 0, 0.5, 1, or 2 for a maximum score of 36. The Central Coherence Index ranges from a minimum score of 0 to a maximum score of 2. It is calculated from the Order Index, which scores the first six global and local elements drawn by the participant, and the Style Index, the degree of continuity in the drawing process.[8] Each drawing was independently scored by two assessors for both accuracy and central coherence, and was then scored together to obtain a consensus score.

Procedure

Assessors at both sites were research assistants trained, certified, and regularly supervised by a licensed clinical psychologist. Participants with BN-spectrum disorder were assessed prior to the first treatment session and HCs during a single testing session. All participants received $50 for completing the neuropsychological battery.

Statistical Analysis

Between-site differences were conducted using independent *t*-tests on the combined data from the clinical groups since all of the HC came from one site only. One-way analysis of variance (ANOVA) tests were conducted to ascertain whether significant differences existed between the three groups on demographic variables. Baseline characteristics that differed between the groups were entered as covariates, and five analyses of covariance (ANOCVA) tests were conducted to identify differences between the groups on Accuracy Copy, Accuracy Delay, Style, Order, and Central Coherence Index. For the main outcome variables, alpha was adjusted using Bonferonni correction to 0.01 to guard against Type 1 error (0.05/5). Pairwise Cohen's *d* effect sizes were calculated. Cohen suggests that *d* values of 0.2, 0.6, and 0.8 represent small, moderate, and large effect sizes, respectively. Pearson's *r* correlations were conducted between RCFT variables and clinical variables among the clinical groups. Because of the difference in size between the HC and clinical samples, we employed bootstrapping procedures to guard against bias. All analyses were conducted using SPSS version 22.

TABLE 2. Analysis of variance between the groups on the primary outcome variables (means [standard deviations]

	BN (n =47)	PD (n = 42)	HC (n = 25)	Comparison	BN vs PD d	BN vs. HC d	PD vs HC d
Accuracy Delay	13.10 (5.90)	10.85 (6.36)	17.80 (5.95)	$F(2, 105) = 7.537$, $p < .001$[a] HC > BN; HC > PD	0.37	0.81	1.14
Accuracy Copy	23.84 (5.58)	20.97 (5.15)	24.96 (4.51)	$F(2, 105) = 7.122$, $p < .001$[a] HC > BN; HC > PD	0.54	0.22	0.83
Order Index	1.82 (.65)	1.70 (.67)	2.03 (.66)	$F(2, 105) = 0.1.415$, $p = .247$	0.18	0.33	0.50
Style Index	1.06 (.47)	1.03 (.51)	1.39 (.46)	$F(2, 105) = 2.917$, $p = .058$ HC > BN; HC > PD[b]	0.06	0.72	0.74
Central Coherence Index	1.08 (.41)	1.03 (.43)	1.26 (.41)	$F(2, 105) = 2.954$, $p = .249$	0.12	0.45	0.55

[a]Remains statistically significant after Bonferonni correction.
[b]Significant relationships are based on bootstrapped pairwise comparisons.
Notes: All variables were raw scores; BN = bulimia nervosa group; PD = bulimia-type eating disorder not otherwise specified group; HC= healthy control group.

Results

We failed to find evidence of differences between the sites on any demographic, baseline, or outcome variable. Demographic characteristics of the three groups are presented in **Table 1**. There were no significant differences between groups on demographic variables with the exception of estimated IQ. ANCOVAS with estimated IQ were conducted on the outcome variables (Accuracy Copy, Accuracy Delay, Style, Order, and Central Coherence Index) and revealed a statistically significant difference between the groups on Accuracy Copy and Delay, and in Style Index. Both clinical groups scored significantly worse than HCs on Accuracy scores with larger effect sizes when recalling the figure after 30 min (see **Table 2**). There was also a suggested difference between both clinical groups and HCs on Style Index with moderate–large effect sizes though this was no longer significant after Bonferonni correction.

Exploratory analysis of the relationship between clinical variables and RCFT variables among the BN-spectrum participants revealed a significant negative relationship between %Median Body Weight (MBW) and Accuracy Copy (r (87) = −.415; $p < .001$); Accuracy Delay (r (86) = −.356; $p = .001$); and Style Index (r (87) = −.226; $p = .038$) such that higher %MBW was related to poorer scores. These relationships were not observed when the analysis was conducted in the HC group.

Discussion

The study aimed to ascertain whether adolescents with BN-spectrum disorders demonstrate central coherence and related neurocognitive features relative to HCs as measured by the RCFT, thereby elucidating some understanding of the construct as well as its role in BN spectrum disorders. Our results suggest that adolescents with BN and PD demonstrate worse visual spatial integration in the context of the copy condition and especially in the delayed recall condition. In addition, both clinical groups performed worse in relation to style relative to a healthy comparison group though this difference was not statistically significant in the ANCOVA overall. These findings are largely in concert with previous research among adults with BN showing significantly poorer accuracy relative to HCs without superior attention to detail as per AN adults, emphasizing the specificity of global integration difficulties to BN.[2]

We found significantly lower accuracy scores on the copy condition of the RCFT in BN as compared to controls. Lower accuracy scores suggest that those with BN employed a disorganized strategy in the construction of the figure. The BN participants appeared to employ a piecemeal approach that lead to poor integration of the figure. Accuracy deficits became more pronounced—as evidenced by very large effect sizes—during the recall tests, thus suggesting that performance was decreased when adding a memory component to the task. Poor initial accuracy scores may impact one's ability to accurately recall and correctly draw elements of the RCFT figure over time.

Higher weight was associated with poorer accuracy scores in both copy and delay conditions, as well poorer performance in style indexes. These findings in an adolescent sample support the findings of Roberts et al., who describe a disorganized approach to completion of the figure in overweight/obese adults and its relationship to binge eating.[2] This is particularly noteworthy in the context of findings from the adult literature that lower BMI among adults with AN is also associated with greater difficulties with central coherence, suggesting that

extreme weight disturbance in either direction may impair coherence. Although these associations were not large and need to be replicated, taken together, it is reasonable to tentatively conclude that higher weights may be associated with global integration difficulties and further research should explore this relationship.

The findings in this study are supportive of the notion that central coherence is not a one-dimensional construct such as its conceptualization as being composed of two distinct elements—attention to detail and global integration.[22] That the effects observed among older adult samples were also observed in this study of younger adolescents with BN may support the hypothesis that theneurocognitive inefficiency associated with a disorganized strategy when drawing the figure may not be directly related to length of illness and may instead be a predisposing factor that could contribute to the development of BN in adolescence. Results also suggest that memory may play an important role in neurocognitive functioning in adolescent BN. Memory deficits are a novel finding in BN. Future studies should further assess the role that memory deficits may play on various aspects of neurocognitive processing in BN, specifically whether memory deficits may lead to poor accuracy when copying the RCFT or whether poor accuracy (as evidenced by disturbances in planning or impulsivity) may influence accuracy sores in the recall condition. The implied role of memory in this earlier stage of illness also argues for a more complex strategy when assessing these neurocognitive features. While the RCFT is a sophisticated measure of visual spatial integration and memory, it may be important to investigate how performance may change as a function of the introduction of illness-related stimuli. The need for this type of investigation is supported by findings from a systematic review of the adult literature that found that those with BN tend to perform worse than HCs on certain tasks only when illness-specific constructs are introduced in the measure (e.g., body-related words in a Stroop task).[23]

Unfortunately this study was limited by only one measure of central coherence. Further examination of executive functioning, including memory, would have enriched our interpretation of findings and are needed in future studies. In addition, assessment of the control group's ED symptoms was by self-report, as was the presence of an ED among family members thus they may have been subject to bias. Aside from employing just one measure of central coherence, the study was also limited by a relatively small number of controls. However, we maintained the same scoring system as previously published to allow for direct comparison to the adult data, and to our knowledge this is the first report of central coherence difficulties among adolescents with BN. These findings should be replicated and broader assessments should be administered (e.g., employing illness-related stimuli).

How might difficulty in global integration be related to the specific clinical features of BN-spectrum disorders? One possibility is that global integration difficulties prevent parsing of information in an appropriate context, giving rise to a dichotomous cognitive style, extreme mood states, and behavioral impulsivity, all features that characterize bulimic syndromes and their associated comorbidity.[24,25] Over time a failure to incorporate information into a broader context or overall framework may exacerbate challenges in executive function such that the default organizing principal becomes disorganization. Thus, without a coherent framework into which to integrate and prioritize relevant information, adolescents with BN may become overwhelmed both cognitively and emotionally. This is consistent with current biophysiological theory that these difficulties may be related to inefficient collaborative brain function and thereby undermine top-down emotional regulation.

Understanding and improving the neurocognitive features that comprise central coherence may be an important strategy to improve outcomes in BN-spectrum disorders. Studies suggest that remediation of this cognitive style can be achieved in AN.[26,27] Further, addressing the bias toward fragmented strategies and increasing coherence in remediation therapy among adolescent populations may increase response to treatment.[28] For example, current choice therapies such as cognitive behavioral therapy rely upon the ability to coherently integrate and efficiently recall information over time, and improvements in central coherence may improve utilization of therapeutic skills.[29]

Declaration of Interest: Drs Lock and Le Grange receive royalties from Guildford Press.

References

1. APA. Diagnostic and Statistical Manual of Mental Disorders, 4th text rev. ed. Washington DC: Author, 2000.
2. Roberts ME, Tchanturia K, Treasure JL. Is attention to detail a similarly strong candidate endophenotype for anorexia nervosa and bulimia nervosa? World J Biol Psychiatry 2013;14:452–463.

3. Bernadinao I, Mouga S, Almeida J, van Asselen M, Oliveira G, Castelo-Branco A. Direct comparison of local-global integration in autism and other developmental disorders: Implications for the central coherence hypothesis. PLoS One 2012;7:339351.
4. Mottron L, Burack J, Iarocci G, Belleville S, Enns J. Locally oriented perception with intact global processing among adolescents with highfunctioning autism: Evidence from multiple paradigms. J Child Psychol Psychiatry 2003; 44:904–913.
5. Plaisted K, Saksida L, Alcantara J, Weisblatt E. Towards an understanding of the mechanisms of weak central coherence effects: Experiments in visual configural learning and auditory perception. Phil Trans R Soc B: Biol Sci 2003;358:375–386.
6. Plaisted K. Reduced generalization in autism: An alternative to weak central coherence. In: Burack JA, Charman T, Yirmiya N, Zelazo P, editors. The Development of Autism: Perspectives from Theory and Research. xvii. Mahwah, NJ: Lawrence Erlbaum Associates Publishers, 2001, pp. 149–169.
7. Lopez C, Tchanturia K, Stahl D, Treasure J. Weak central coherence in eating disorders: A step towards looking for an endophenotype of eating disorders. J Clin Exp Neuropsychol 2009;31:117–125.
8. Lopez C, Tchanturia K, Stahl D, Treasure J. Central coherence in women with bulimia nervosa. Int J Eat Disord 2008;41:340–347.
9. Brand M, Franke-Sievert C, Jacoby GE, Markowitsch HJ, Tuschen-Caffier B. Neuropsychological correlates of decision making in patients with bulimia nervosa. Neuropsychology 2007;21:742–750.
10. Lang K, Lopez C, Stahl D, Tchanturia K, Treasure J. Central coherence in eating disorders: An updated systematic review and meta-analysis. World J Biol Psychiatry 2014;1–13. (doi: 10.3109/15622975.2014.909606)
11. Harrison A, Tchanturia K, Treasure J. Measuring state trait properties of detail processing and global integration ability in eating disorders. World J Biol Psychiatry 2011;12:462–472.
12. Southgate L, Tchanturia K, Treasure J. Neuropsychology in eating disorders. In: Wood SJ, Allen NB, Pantelis. C, editors. The Neuropsychology of Mental Illness. Cambridge, UK: Cambridge University Press, 2009, pp. 316–325.
13. Fairburn CG, Cooper PJ. The Eating Disorder Examination. In: Fairburn CG, Wilson GT, editors. Binge Eating: Nature, Assessment and Treatment, 12th ed. New York: Guildford Press, 1993, pp. 317–360.
14. Carter JC, Aime AA, Mills JS. Assessment of bulimia nervosa: A comparison of interview and self-report questionnaire methods. Int J Eat Disord 2001;2: 187–192.
15. Le Grange D, Crosby R, Rathouz P, Leventhal BL. A randomized controlled comparison of family-based treatment and supportive psychotherapy for adolescent bulimia nervosa. Arch Gen Psychiatry 2007;64:1049–1056.
16. Fairburn CG, Beglin SJ. Eating Disorder Examination Questionnaire (EDE-Q 6.0). In: Fairburn CG, editor. Cognitive Behavior Therapy and Eating Dsiorders. New York: Guilford Press, 2008;265–308.
17. Mond JM, Hay PJ, Rodgers B, Owen C. Validity of the Eating Disorder Examination Questionnaire (EDE-Q) in screening for eating disorders in community samples. Behav Res Ther 2004;42:551–567.
18. Kaufman J, Birmaher B, Brent D, Rao U, Flynn C, Moreci P. Schedule for affective disorders and schizophrenia for school-age children-present and lifetime version (K-SADS-PL): Initial reliability and validity data. J Am Acad Child Adolesc Psychiatry 1997;36:980–987.
19. Osterrieth P. Test of copying a complex figure: Contribution to the study of perception and memory. Arch Psychol 1944;20:206–356.
20. Strauss E, Sherman E, Spreen O. A Compendium of Neuropsychological Tests: Administration, Norms and Commentary, 3rd ed. New York: Oxford University Press, 2006.
21. Booth R. Local–Global Processing and Cognitive Style in Autism Spectrum Disorders and Typical Development. London: King's College Institute of Psychiatry, 2006.
22. Happe F, Frith U. The weak coherence account: Detail-focused cognitive style in autism spectrum disorders. J Autism Dev Disord 2006;36: 5–25.
23. Van den Eynde F, Guillaume S, Broadbent H, Stahl D, Campbell IC, Schmidt U, et al. Neurocognition in bulimic eating disorders: A systematic review. Acta Psychiatr Scand 2011;124:120–140.
24. Treasure J, Claudino AM, Zucker N. Eating disorders. Lancet 2010;375: 583–593.
25. Engelberg MJ, Steiger H, Guavin L, Wonderlich SA. Binge antecedents in bulimic syndromes: An examination of dissociation and negative affect. Int J Eat Disord 2007;40:531.
26. Davies H, Fox J, Naumann U, Treasure J, Schmidt U, Tchanturia K. Cognitive remediation and emotion skills training for anorexia nervosa: an observational study using neuropsychological outcomes. Eur Eat Disord Rev 2012;20: 211–217.
27. Pretorius N, Dimmer M, Power E, Eisler I, Simic M, Tchanturia K. Evaluation of a cognitive remediation therapy group for adolescents with anorexia nervosa: Pilot study. Eur Eat Disord Rev 2012;20:321–325.
28. Lock J1 AW, Fitzpatrick KK, Bryson SW, Jo B, Tchanturia K. Is outpatient cognitive remediation therapy feasible to use in randomized clinical trials for anorexia nervosa? Int J Eat Disord 2013;46:567–575.
29. Tchanturia K, Lock J. Cognitive remediation therapy for eating disorders: development, refinement and future directions. Curr Top Behav Neurosci 2011;6:269–287.