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BRIEF REPORT

Executive functioning and central coherence in anorexia nervosa: Pilot investigation of a neurocognitive endophenotype

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Abstract

A neurocognitive profile characterized by problems in set shifting, executive functioning, and central coherence may pre-date and maintain anorexia nervosa (AN). To test this pattern as a possible endophenotype for AN, 10 youth with current AN, 14 healthy youth, and their biological parents, participated in a neuropsychological battery. Youth with AN demonstrated significantly weaker central coherence, related to enhanced detail-focused processing. Youth with AN and their parents demonstrated significantly greater psychopathology relative to controls, and youth–parent scores were significantly correlated. The study, limited by a small sample size, found little evidence supporting a neuropsychological endophenotype for AN. Identifying a neurocognitive profile for children and adolescents with AN has important implications for the treatment of young patients.

KEYWORDS

adolescents, anorexia nervosa, central coherence, endophenotype, neurocognitive profile

1 | INTRODUCTION

Recent attempts to understand the development, course, and treatment of anorexia nervosa (AN) have shifted from focusing on overt behaviours to studying underlying cognitive differences that may represent vulnerabilities to the disorder (Treasure, Kanakam, & Macare, 2011). Several such potential cognitive markers have been identified. These include problems with set-shifting and other elements of executive functioning and central coherence (Lang, Lopez, Stahl, Tchanturia, & Treasure, 2014; Westwood, Stahl, Mandy, & Tchanturia, 2016).

Among adults, inefficiencies in set-shifting are thought to be present in the acute, weight restored, and recovered states of AN relative to controls (Tchanturia et al., 2011). In youth, more subtle, largely nonsignificant differences in set-shifting are found in acute AN when compared with controls (Lang et al., 2015; Lang, Stahl, Espie, Treasure, & Tchanturia, 2014; Westwood et al., 2016). Although the same levels of set-shifting difficulties have not been shown in youth as in adults, evidence suggests that AN youth present with a similar, attenuated cognitive profile in terms of set-shifting and central coherence relative to adults (Lang et al., 2015; Lang, Stahl, et al., 2014; Westwood et al., 2016). Lower levels of set-shifting ability in AN may be related to weight status, other disorder features, associated psychopathology, or stable, trait markers (e.g., Giel et al., 2012; Roberts, Tchanturia, & Treasure, 2010; Tchanturia et al., 2004). Most studies have investigated set-shifting, but other areas of executive functioning may be affected in AN, including planning, decision-making, and verbal inhibition (e.g., Galimberti et al., 2013).

Weak central coherence (WCC) refers to the tendency to prioritize a focus on details as well as difficulty processing information in a global context (Happé, 2005; Happé & Booth, 2008). The concept has been extensively studied in relation to autism (Happé, 2005) and more recently in AN (Lang, Lopez, et al., 2014). Results from a meta-analysis found evidence that two aspects of WCC, detail-focused processing and poor global integration, are associated with acute AN and to a lesser extent recovered AN (Lang, Lopez, et al., 2014). Among youth with AN, studies indicate that children and adolescents with AN exhibit a cognitive profile characterized by WCC (Lang et al., 2015; Stedal, Rose, Frampton, Landrø, & Lask, 2012). Poor set-shifting ability and WCC may both contribute to the rigid and detail-oriented cognitive style that underlies the obsessive–compulsive personality traits observed in AN (Zucker et al., 2007). This cognitive style, associated with abnormality in neurobiological function (Zucker et al., 2007), could explain in part commonalities in cognitive and behavioural rigidity and social cognitive problems found in AN, autism spectrum disorders, and obsessive–compulsive personality disorder.

Whether possible neurocognitive weaknesses in AN are associated with acute state of malnutrition, trait risk factors, "scarring" that persists after recovery, or some combination of these remains unclear (Treasure et al., 2011). Preliminary research suggests difficulty in set-shifting and WCC may be inherited traits, part of an endophenotype for AN (Galimberti et al., 2013; Roberts, Tchanturia, & Treasure, 2013; Tenconi et al., 2010; Treasure et al., 2011; Zucker et al., 2007). In youth with AN, there is some evidence that inefficient set-shifting and WCC represent familial traits (Lang, Treasure, & Tchanturia, 2016). Such an endophenotype could represent a risk factor for the development of AN implicated in its maintenance and chronicity.

1.1 | Aims

This study sought to replicate neuropsychological findings in AN and incorporate a novel parent–child dyad comparison that could potentially signal an endophentype for AN. We investigated executive functioning and central coherence in youth with AN and in their biological parents using tests from a global standardized neuropsychological battery, the Ravello Profile (Rose, Davis, Frampton, & Lask, 2011) and several additional measures of constructs of interest. It adds to the relatively small body of literature on the cognitive profile of youth with AN and tests the hypothesis that specific cognitive differences characterize an endophenotype for AN, a hypothesis that would be supported if biological parents of individuals with AN exhibit similar neurocognitive features as those reported in AN. We predicted that youth with AN would demonstrate significant problems with executive functioning and central coherence compared with healthy controls (HCs). Further, it was expected that parents of youth with AN would demonstrate similar poorer performance compared with control parents.

2 | METHOD

2.1 | Participants

Ten youth under the age of 18 and diagnosed with current AN, along with a biological parent, were recruited from a multilevel, adolescent medicine-based eating disorders program, through physician referral or flyers. Youth with AN were first diagnosed by their treating physician, with diagnosis then confirmed via parent interview using diagnostic items from the Eating Disorder Examination, Edition 17.0D (EDE; Fairburn, Cooper, & O'Connor, 2014) and the Parent Version of the Eating Disorder Examination (P-EDE; Loeb, 2008). Fourteen HCs under the age of 18, along with a biological parent, were recruited through pediatric referrals and flyers at the medical center. Exclusionary criteria for all participants, both youth and parents, were a history of head injury or neurological disorder and current psychotic, bipolar, and substance use disorders. Current eating disorder or history of an eating disorder were exclusionary criteria for HC youth. Current eating disorder was also an exclusionary criterion for parents in both the AN and HC groups. All participants were screened by parent report for any current and/or past psychiatric diagnoses.

Of 38 youth–parent dyads ($N = 76$) that initially expressed interest in the study, 10 patient dyads and 14 healthy control dyads (24 dyads, $N = 48$) were deemed eligible and completed the study.

Demographic information is found in Table 1. All youth with AN were currently in treatment for AN, with half taking psychoactive medication for anxiety and/or depression. None of the HCs, both youth and parent, had a current or history of any psychiatric diagnoses by parent report, including eating disorders; none were taking psychoactive medications. No participants taking part in this study were diagnosed with an autism spectrum disorder or other neurodevelopmental disorder.

2.2 | Measures

2.2.1 | Executive functioning

Tests from the Delis–Kaplan Executive Function System (Delis, Kaplan, & Kramer, 2001) were utilized to measure

TABLE 1 Demographic characteristics

Characteristic	AN child	AN parent	HC child	HC parent
Gender				
Female	9 (90%)	9 (90%)	12 (86%)	12 (86%)
Male	1 (10%)	1 (10%)	2 (14%)	2 (14%)
Age (years)	14.85 (2.09)	44.67 (4.77)	13.66 (2.98)	46.92 (5.31)
BMI	17.91 (2.66)	26.75 (5.63)	27.16 (7.55)	34.26 (9.37)
BMI-for-age percentile	30.20 (26.99)		87.00 (14.40)	
Ethnicity				
Hispanic/Latino(a)	1 (10%)	1 (10%)	1(7%)	1(7%)
Native American	0	0	0	0
Asian	1 (10%)	1 (10%)	0	0
Black	0	0	2 (14%)	2 (14%)
Native Hawaiian/Pacific Islander	0	1 (10%)	1(7%)	3 (21%)
White	7 (70%)	7 (70%)	9 (64%)	8 (57%)
Other	1 (10%)	0	1 (7%)	0
Child education (years completed)	8.30 (1.95)		7.14 (3.09)	
Parent education level				
Less than high school		0		0
High school		2 (20%)		1 (7%)
Some college/associate degree		2 (20%)		6 (43%)
Bachelor degree		4 (40%)		6 (43%)
Graduate degree		2 (20%)		1(7%)
Total household income (parent–child dyad)				
Less than $40,000	0		3 (21%)	
$40,000 to $59,999	0		2 (14%)	
$60,000 to $79,999	2 (20%)		3 (21%)	
$80,000 to $99,999	1 (10%)		0	
$100,000 or more	7 (70%)		6 (43%)	

Note. AN = anorexia nervosa; HC = healthy control; BMI = body mass index. Values are either frequencies (percents) or means (standard deviations). BMI percentile calculated using Centers for Disease Control and Prevention calculators, which provides BMI and BMI-for-age percentile.

cognitive inhibition and flexibility (Color-Word Interference Test Conditions 3 and 4), fluency (Verbal Fluency Test Conditions 1 and 2), set-shifting (Verbal Fluency Test Condition 3 and Trail Making Test Condition 4), and planning (Tower Test).

2.2.2 | Central coherence

Central coherence was assessed using the Central Coherence Index (CCI) of the Rey–Osterrieth complex figure test (RCFT; Osterrieth, 1944), which measures global integration of processing visual information, one aspect of central coherence. The CCI scoring system developed by Booth (2006; see also Lopez et al., 2008) was utilized for the RCFT copy and delayed recall trials. Two raters independently scored six participants (12.5% of the total sample) on the CCI. Intraclass correlation coefficients (two-way random, absolute agreement, and single measures) were .99 for copy trial and .97 for the delayed recall trial. Additionally, central coherence was assessed with the Group Embedded Figures Test (GEFT; Oltman, Raskin, & Witkin, 1971; Witkin, Oltman, Raskin, & Karp, 2002), a measure of detail-focused processing of visual information.

2.2.3 | Intellectual ability

Verbal intelligence was assessed with the vocabulary subtest from the Wechsler Adult Intelligence Scale—Fourth Edition (WAIS-IV; Wechsler, 2008) and Wechsler

Intelligence Scale for Children–Fourth Edition (WISC-IV; Wechsler, 2003). Performance intelligence was assessed with the Matrix Reasoning subtest from the WAIS-IV and the WISC-IV.

2.2.4 | Psychopathology

Anxiety was measured with the State Trait Anxiety Inventory for Adults (Spielberger, Gorsuch, Lushene, Vagg, & Jacobs, 1983) and the children's version of the State Trait Anxiety Inventory (Spielberger, Gorsuch, Lushene, Vagg, & Jacobs, 1973). Depressive symptoms were assessed using the Beck Depression Inventory-II (Beck, Steer, & Brown, 1996) and the Children's Depression Inventory 2 (Kovacs, 1992). Eating disorder symptoms were measured with the Eating Disorder Examination-Questionnaire (EDE-Q; Fairburn & Beglin, 2008) and the parent version of the Eating Disorder Examination-Questionnaire (P-EDE-Q; Loeb, 2007). Obsessive–compulsive symptoms were assessed with a self-report version of the Yale-Brown Obsessive Compulsive Scale (Baer, 1991) and the Children's Obsessive Compulsive Inventory—Revised (Shafran et al., 2003).

2.3 | Procedure

Dual ethics approvals were received from the first author's academic institution and the hospital system at the performance site. Participants were recruited through treatment providers and posted flyers. Following phone screening, eligibility was determined in a formal face-to-face screening interview based on Diagnostic and Statistical Manual of Mental Disorders (5th ed.; DSM–5; American Psychiatric Association, 2013) criteria with the parent. Participants were then administered the battery detailed above by a doctoral level clinician trained in neuropsychological testing. Height and weight were measured at the start of the testing session or obtained from the patient chart. Financial compensation of $100 was awarded to each participant.

3 | RESULTS

We first examined correlations for all dependent variables of interest. Correlations for all dependent variables can be found in Table S1.

Table 2 shows the means, standard deviations, mean differences, confidence intervals for mean differences, and effects sizes (Cohen's *d*s) for youth with AN compared with HCs. Table 3 provides these for the two groups of parents. Confidence intervals are the recommended statistic for drawing interpretations from replication studies such as this one (Cumming & Maillardet, 2006). In addition, to adjust for multiple comparisons in the neuropsychological battery and control for Type I error, we set a conservative alpha level of .01, consistent with other similar studies (Lang et al., 2015). Using this conservative cutoff, youth with AN demonstrated WCC relative to HCs with large effect size (GEFT $d = 1.21$), but no differences in set-shifting or executive functioning were found. Likewise, no differences in parent scores on tests of executive functioning or central coherence were demonstrated using this alpha criterion. Greater levels of psychopathology were found for both AN youth and their parents compared with controls with very large effect sizes ($p < .01$). Differences in verbal fluency were notable for both youth and parent samples, with large effect sizes for differences, but these results were significant at the $p < .05$ level only. Parent performance on verbal fluency was also negatively correlated with parent depression ($r = -.45$).

As an exploratory analysis of youth–parent concordance on the neuropsychological measures, we examined the correlations of youth and parent scores for each variable using the total sample, the AN youth–parent group, and the HC youth–parent group (see Table S1). Youth–parent scores were correlated on measures of anxiety and depression.

4 | DISCUSSION

This study investigated set-shifting, executive functioning, and central coherence in children and adolescents with AN and in their biological parents. Findings demonstrated partial support for the hypotheses comparing youth with AN with controls but little evidence supporting a neuropsychological profile as an endophenotype for AN.

Our findings did not support our hypotheses regarding inefficiencies in set-shifting and executive function in youth with AN. Consistent with the results of this study, some investigations in children and adolescents with AN have not found the significant difficulties with set-shifting and executive function demonstrated in adults with AN (Andrés-Perpiña et al., 2011; Fitzpatrick, Darcy, Colborn, Gudorf, & Lock, 2012; Shott et al., 2012; Westwood et al., 2016). However, subtle differences in set-shifting, as well as central coherence, have been found in AN youth in a methodologically robust study (Lang et al., 2015) that employed the Wisconsin Card Sorting Test (WCST; Heaton, 1993). The WCST may be a more sensitive measure of set-shifting in youth with AN, who are hypothesized to exhibit similar but less pronounced cognitive inefficiencies relative to affected adults

TABLE 2 Descriptive statistics for anorexia nervosa versus healthy children

Variable	Group	*n*	*M*	*SD*	*MD*	CI	*d*
Executive functioning							
D-KEFS Color Word Interference-Condition 3	AN	10	11.90	1.97	1.28	−.34 to 2.90	.69
	HC	13	10.62	1.76			
D-KEFS Color Word Interference-Condition 4	AN	10	11.80	2.20	1.30	−1.05 to 3.65	.48
	HC	14	10.50	3.06			
D-KEFS Verbal Fluency-Condition 1	AN	10	13.20	3.74	3.20	.69 to 5.71	1.09*
	HC	14	10.00	2.18			
D-KEFS Verbal Fluency-Condition 2	AN	10	13.50	3.60	1.86	−1.05 to 4.76	.55
	HC	14	11.64	3.23			
D-KEFS Verbal Fluency-Condition 3	AN	10	11.60	2.76	−.54	−2.68 to 1.59	−.22
	HC	14	12.14	2.28			
D-KEFS Trail Making-Condition 4	AN	10	10.70	2.54	.78	−1.43 to 2.98	.31
	HC	13	9.92	2.50			
D-KEFS Tower	AN	10	10.40	2.17	.83	−.87 to 2.53	.42
	HC	14	9.57	1.83			
Central coherence							
RCFT copy-central coherence index	AN	10	1.05	0.37	.09	−.26 to .46	.23
	HC	14	.96	0.45			
RCFT delayed recall-central coherence index	AN	10	1.10	0.35	−.05	−.40 to .30	−.12
	HC	14	1.15	0.45			
GEFT	AN	10	10.60	3.69	5.24	1.53 to 8.96	1.21**
	HC	14	5.36	4.72			
Intellectual ability							
Matrix reasoning	AN	10	12.40	2.41	.04	−1.96 to 2.05	.02
	HC	14	12.36	2.27			
Vocabulary	AN	10	11.90	.99	.54	−.56 to 1.64	.42
	HC	14	11.36	1.45			
Psychopathology							
STAI state	AN	10	55.40	16.13	15.54	5.53 to 25.56	1.32**
	HC	14	39.86	7.08			
STAI trait	AN	10	64.80	14.47	25.59	15.56 to 35.61	2.18***
	HC	14	39.21	9.25			
CDI 2	AN	10	8.65	4.56	6.36	3.67 to 9.05	2.02***
	HC	14	2.29	1.49			
EDE-Q	AN	10	3.68	1.66	2.87	1.73 to 4.01	2.20***
	HC	13	0.81	0.94			
CHOC-R	AN	10	19.10	6.61	12.48	6.24 to 18.73	1.75***
	HC	13	6.62	7.52			

*$p < .05$.

**$p < .01$.

***$p < .001$.

Note. AN = anorexia nervosa; HC = healthy control; CDI 2 = Children's Depression Inventory 2; CHOCI-R = Children's Obsessive Compulsive Inventory—Revised; D-KEFS = Delis–Kaplan Executive Function System; GEFT = Group Embedded Figures Test; EDE-Q = Eating Disorder Examination-Questionnaire; RCFT = Rey Complex Figure Test; STAI = State Trait Anxiety Inventory; *M* = mean; *SD* = standard deviation; *MD* = mean differences; CI = confidence intervals for mean differences; *d* = effects sizes (Cohen's *d*s). Raw scores for all D-KEFS, WAIS, and WISC subtests were standardized using normal procedures for those instruments ($M = 10$ and $SD = 3$). Raw scores for the STAI were converted to *t* scores. Raw scores were used for all other measures.

TABLE 3 Descriptive statistics for parents of anorexia nervosa versus healthy children

Variable	Group	*n*	*M*	*SD*	*MD*	CI	*d*
Executive functioning							
D-KEFS Color Word Interference-Condition 3	AN	10	11.10	2.89	.31	−2.27 to 2.89	.10
	HC	14	10.79	3.09			
D-KEFS Color Word Interference-Condition 4	AN	10	10.90	2.64	−.10	−2.36 to 2.16	−.04
	HC	14	11.00	2.63			
D-KEFS Verbal Fluency-Condition 1	AN	10	10.30	4.24	−1.06	−4.76 to 2.65	−.25
	HC	14	11.36	4.36			
D-KEFS Verbal Fluency-Condition 2	AN	10	9.20	2.90	−2.66	−5.29 to −.02	−.87*
	HC	14	11.86	3.18			
D-KEFS Verbal Fluency-Condition 3	AN	10	11.30	3.34	−.84	−3.82 to 2.13	−.24
	HC	14	12.14	3.55			
D-KEFS Trail Making-Condition 4	AN	10	11.10	1.66	.24	−1.68 to 2.17	.11
	HC	14	10.86	2.57			
D-KEFS Tower	AN	10	10.40	1.43	.69	−.72 to 2.10	.42
	HC	14	9.71	1.77			
Central coherence							
RCFT copy-central coherence index	AN	10	1.25	.27	−.09	−.33 to .15	−.32
	HC	14	1.34	.29			
RCFT delayed recall-central coherence index	AN	10	1.09	.30	−.20	−.46 to .07	−.64
	HC	14	1.29	.31			
GEFT	AN	10	8.80	4.37	.59	−3.03 to 4.21	.14
	HC	14	8.21	4.12			
Intellectual ability							
Matrix reasoning	AN	10	12.00	1.63	.93	−1.15 to 3.01	0.39
	HC	14	11.07	2.84			
Vocabulary	AN	10	10.60	2.55	−.11	−2.48 to 2.25	−0.04
	HC	14	10.71	2.89			
Psychopathology							
STAI state	AN	10	53.10	10.92	13.64	6.25 to 21.02	1.58***
	HC	14	39.46	6.52			
STAI trait	AN	10	55.30	10.39	13.23	5.04 to 21.42	1.39**
	HC	14	42.07	8.90			
BDI-II	AN	10	12.40	6.72	8.26	3.28 to 13.24	1.42**
	HC	14	4.14	5.07			
EDE-Q	AN	10	1.18	1.14	.25	−.62 to 1.13	0.25
	HC	14	0.93	0.93			
Y-BOCS	AN	10	4.30	6.29	2.94	−1.25 to 7.13	0.60
	HC	14	1.36	3.59			
P-EDE-Q	AN	10	3.68	1.27	3.28	2.50 to 4.06	3.59***
	HC	14	0.40	0.52			

*$p < .05$.

**$p < .01$.

***$p < .001$.

Note. AN = anorexia nervosa; HC = healthy control; BDI-II = Beck Depression Inventory-II; D-KEFS = Delis–Kaplan Executive Function System; GEFT = Group Embedded Figures Test; EDE-Q = Eating Disorder Examination-Questionnaire; P-EDE-Q = Parent Version Eating Disorder Examination-Questionnaire; RCFT = Rey Complex Figure Test; STAI = State Trait Anxiety Inventory; Y-BOCS = Yale-Brown Obsessive Compulsive Scale; *M* = mean; *SD* = standard deviation; *MD* = mean differences; CI = confidence intervals for mean differences; *d* = effects sizes (Cohen's *d*s). Raw scores for all D-KEFS, WAIS, and WISC subtests were standardized using normal procedures for those instruments ($M = 10$ and $SD = 3$). Raw scores for the STAI were converted to *t* scores. Raw scores were used for all other measures.

(Lang et al., 2015; Lang, Stahl, et al., 2014; Westwood et al., 2016). Higher order cognitive functions may be more susceptible to the prolonged effects of starvation, as adults, by virtue of their age relative to the onset of AN, have had AN for a longer duration.

The WCC hypothesis was partially supported in this study. Youth with AN scored significantly higher on the GEFT than on the HCs, indicating enhanced detail processing, which is one aspect of WCC, and the effect size for this difference was large. In terms of weak global processing, a second aspect of WCC, we did not find significant group differences on the CCI because AN and HC youth groups scored well below norms for the CCI (AN $Mdn = 1.38$ and HC $Mdn = 1.50$; Lang, Roberts et al., 2016). These adult norms are not strictly comparable but do suggest that both our AN and HC youth samples demonstrated WCC related to global processing. Similarity between the affected and control groups on WCC could have affected our ability to detect significant group differences. Our results do indeed replicate Lang et al.'s (2015) findings regarding the absolute values of CCI scores for the AN youth group (RCFT Copy CCI $M = 1.05$). Overall, these results with respect to central coherence are consistent with a growing body of literature supporting a cognitive profile characterized by WCC in youth with AN (Lang et al., 2015; Stedal et al., 2012).

Additionally, our findings regarding the intellectual ability of the AN group replicate studies that have shown higher than average intelligence quotient scores in AN compared with the normative population (Lopez, Stahl, & Tchanturia, 2010). Although higher normative intelligence was not a specific hypothesis tested in our study, we found that AN youth scored approximately .67 standard deviations above the mean of the normative population on performance and verbal measures of intelligence. Our AN youth group was well matched with controls in terms of intelligence scores, with no significant differences between the youth groups. This suggests that the differential cognitive profile demonstrated by AN youth compared with controls was not solely an artifact of intellectual ability.

Few findings for parents were consistent with our hypotheses. Parents of youth with AN displayed lower scores on verbal fluency ($p < .05$), but differences did not meet statistical significance using a stringent p value criterion ($p < .01$). Parent differences in verbal fluency may be explained by higher levels of parent depression in AN parents. Regarding central coherence, although no significant differences were found comparing parent groups, AN parents scores were lower on the CCI than expected compared with norms (HC $Mdn = 1.50$; Lang, Roberts, et al., 2016), indicating that the AN parent group did indeed demonstrate WCC.

It is notable that the mean body mass index-for-age percentile for HC youth was in the overweight range (87th percentile; Barlow, 2007), with three of the 14 HC youth participants in the obese range. Overweight and obesity have been associated with poorer cognitive functioning in youth (Li, Dai, Jackson, & Zhang, 2008; Rienert, Po'e, & Barkin, 2013). In the present study, weight status of our youth HC could have affected our ability to detect relative differences in executive function and other aspects of cognition in AN youth. However, body mass index-for-age percentile was not correlated with cognitive performance for the HC youth group, suggesting that weight status of this group did not confound results. Further supporting this, intelligence quotients on both verbal and performance measures were well matched for the youth affected and control groups as mentioned previously.

A major limitation of this pilot study was small sample size; however, corrections were made for multiple comparisons, differences were significant at a conservative alpha level of .01, and effect sizes were large. Another potential limitation is heterogeneity of the youth sample in terms of age, developmental level, and gender. Other measures, such as the WCST to detect set-shifting differences, may also better target neurocognitive constructs, allowing for more robust interpretations. Higher levels of anxiety and depression in the AN groups could have affected cognitive function; however, AN youth actually performed "better" on the neuropsychological measures for which significant differences were found. Finally, half of AN youth were taking psychoactive medication for anxiety and/or depression, which may affect cognition (Snyder, 2013).

This study replicates and extends limited literature investigating the neurocognitive profile of youth with AN and parent–child dyads (Lang et al., 2015; Lang, Stahl, et al., 2014; Lang, Treasure, & Tchanturia, 2016; Stedal et al., 2012; Westwood et al., 2016). Though we found scant evidence of an endophenotype for AN, WCC appears to be a neurocognitive feature for youth as well as adults with AN. More research is needed to determine whether WCC is trait- or state-based or aggregates in families. Reasons for differences between AN cognitive profiles of youth and adults are unclear. Factors may include the greater severity and duration of illness and developmental impact of brain maturation and endocrine function during puberty. Set-shifting difficulties are associated with frontal lobe dysfunction, and childhood and adolescence are periods of substantial maturation in the prefrontal cortex and development of executive function (Gur et al., 2012).

An important clinical implication of this study is that the AN cognitive profile and possible underlying mechanisms for youth may be different than that for adults. Given greater brain plasticity, younger individuals may be more resilient to the neurocognitive impact of AN, making early intervention more effective before a rigid, obsessive, detail-focused cognitive style becomes entrenched. As such, rapid renourishment is important in youth to divert a chronic course of cognitive deterioration. In addition to renourishment and weight gain, adjunctive cognitive remediation therapy (Tchanturia, Giombini, Leppanen, & Kinnaird, 2017; Tchanturia, Lounes, & Holttum, 2014) for AN may be effective in improving features of cognitive functioning, such as over focus on detail, along with psychological factors addressed in standard treatment for AN.

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SUPPORTING INFORMATION

Additional Supporting Information may be found online in the supporting information tab for this article.

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