BRIEF REPORT

The Early Cognitive Development of Children at High Risk of Developing an Eating Disorder

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Abstract

Diagnosis of an eating disorder (ED) has been associated with differences in cognition. Recent evidence suggests that differences may be present prior to onset. Children at familial high risk for ED show cognitive differences at ages 8–10 years. Research is required to investigate differences in cognitive development at various time points. This is the first study to investigate cognitive development in children at high risk at 18 months (Griffiths Mental Development Scale; n = 982) and 4 years old (Wechsler Preschool and Primary Scale of Intelligence—Revised; n = 582), in comparison with children not at risk, using a general population sample, the Avon Longitudinal Study of Parents and Children. Children of women with lifetime anorexia nervosa revealed difficulties in social understanding, visual-motor function, planning and abstract reasoning. Cognitive differences observed here have also been observed in clinical groups. This suggests difficulties may be present prior to onset, potentially affecting risk status for development of ED. Findings contribute to an understanding of aetiology, and design of prevention/intervention strategies. Copyright © 2013 The Authors. *European Eating Disorders Review* published by John Wiley & Sons Ltd.

Keywords
ALSPAC; eating disorder; high-risk; cognitive development; aetiology

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Diagnosis of an eating disorder (ED) has been associated with differential cognitive function (Shott et al., 2012; Treasure & Schmidt, 2013). Whether differences are present prior to onset, possibly contributing to the development of an ED, or whether differences are a consequence of secondary features of the disorder (i.e. low/irregular nutritional intake) is not conclusive. Retrospective studies are subject to recall bias, and differences observed in recovered groups may be scars of the disorder (Lindner, Fichter, & Quadflieg, 2013). One method of investigating cognitive function prior to onset of a disorder is to investigate those that are at high risk of developing that disorder. The first-degree relatives of probands have been shown to be at higher risk of developing an ED than the general population (Strober, Freeman, Lampert, Diamond, & Kaye, 2000). We carried out the first study on cognitive function in children at familial high risk for ED (aged 8–10 years; Kothari, Solmi, Treasure, & Micali, 2012) and found that children of women with anorexia nervosa (AN) showed superior performance on tests of intelligence/global cognition and working memory, but poorer attentional control compared with children not at risk. The children of women with bulimia nervosa (BN) were found to have poor visuo-spatial function. To understand whether children at risk for ED follow a differential developmental trajectory to those not at risk, it is vital to investigate cognitive function at various time points. This type of research could provide valuable information regarding premorbid differences that may affect risk for ED, as well as informing prevention/intervention strategies. In order to extend our previous findings we sought to investigate early cognitive development in a sub-sample of the Avon Longitudinal Study of Parents and Children. We hypothesised that the children of women with AN would show higher IQ but poorer social function, and the children of women with BN would show poorer performance on tests of visuo-spatial function, when compared with children not at risk.

Methods

The Avon Longitudinal Study of Parents and Children (ALSPAC) is a population-based cohort of women recruited during pregnancy (n = 14 541), with the children they were pregnant with at the time (Boyd et al., 2012). Women were eligible for the study if they lived in a predefined study area of Bristol and if their expected date of delivery was between 1 April 1991 and 31 December 1992. A subset of children from the ALSPAC cohort (approximately 10%) were randomly selected, and mothers were invited to bring their children to clinics for behavioural and biological assessment at several ages between 4 and 61 months. The number of children seen ranged from 994 to 1314. Children's cognitive function was assessed during these clinics using the Griffiths Mental Development Scale (Griffiths, 1984) (18 months), and the Wechsler

Preschool and Primary Scale of Intelligence–Revised (Wechsler, 1990; WPPSI-R; 4 years). Data from a questionnaire sent to mothers at 12 weeks gestation, which asked about history of ED, were used to define high risk status of children. Mothers were able to report history of AN, BN or both. Previous studies have found these groups to be distinct with regard to ED cognitions (Micali, Northstone, et al., 2012; Micali, Simonoff, & Treasure, 2011), body mass index and frequency of compensatory behaviours, with women reporting AN and BN resembling an AN-binge/purge subtype (AN-BP; Micali, Simonoff, & Treasure, 2007). This kind of self-report indicator of diagnosis of AN and BN has also shown high sensitivity and specificity in a Netherlands-based population sample (Micali, De Stavola, et al., 2012). Mother–child pairs were excluded if mothers reported history of any psychiatric disorders other than ED only, because of lack of information of specific disorder making this a heterogeneous group. Mothers who had multiple births were also excluded as twins are known to experience a different developmental trajectory. For inclusion in the current study, data had to be available on both maternal exposure and at least one of the two cognitive assessments (outcome variable). At 18 months n = 982 (at risk = 45, 4.5%) and at 4 years n = 852 (at risk = 33, 3.9%). It is noted that high risk groups are small, especially in comparison with the samples of children not at risk; however, the prevalence of ED in this sample is consistent with that in the general population (Micali, Northstone, et al., 2012).

Statistical analysis

To analyse differences in cognitive function, a series of linear regression analyses were run using maternal history of ED to predict children's performance on each task. Children at high risk for ED, were compared with children of women with no history of psychiatric disorder. Socio-demographic predictors of attrition were investigated. Initially, a minimally adjusted model (model 1) was run, including a priori confounders (child age, child gender and tester). In a second model (model 2), potential confounders and predictors of attrition were included (birth weight, ethnicity, maternal age at delivery, marital status and parity). Maternal education was determined to be a potential mediator and was therefore additionally included individually in a fully adjusted model (model 3). Missing data on all potential confounders/mediators were dealt with using multiple random imputations. All predictor and outcome variables were used as predictors in the imputation model, which was set for 10 imputations. Analyses were run on complete case and imputed datasets, and a comparison of results showed that differences were negligible. Only results based on imputed datasets are presented here as complete case analysis is thought to suffer from more chance variation, and multiple random imputation is assumed to correct any bias. All analyses were carried out on SPSS 21 and a significance level of $p \leq 0.05$ was used. Because of the small size of the at-risk samples, it was decided that the significance level would not be adjusted for multiple comparisons to avoid missing clinically significant differences.

Procedure

The study was approved by the ALSPAC Law and Ethics Committee and the Local Research Ethics Committees. Questionnaires and assessments were conducted after giving full information to participants and acquiring consent in accordance with the ALSPAC study design.

Results

Griffiths developmental scales

In the fully adjusted model, children of women with lifetime AN showed lower scores on the locomotor development [B: −9.10, 95% confidence interval (CI): −14.24, −3.95, $p < 0.001$] and the personal-social development (B: −5.88, 95% CI: −11.25, −0.51, $p = 0.03$) subtests, as well as the general intelligence quotient (B: −5.25, 95% CI: −9.59, −0.92, $p = 0.02$), in comparison with unexposed children (Table 1).

Wechsler Preschool and Primary Scale of Intelligence–Revised

In the fully adjusted model, children of women with lifetime AN showed lower scores on two performance subtests: geometric design (B: −1.53, 95% CI: −2.91, −0.14, $p = 0.03$) and block design (B: −1.79, 95% CI: −3.50, 0.08, $p = 0.04$). This group also showed lower scores in two verbal subtests: comprehension (B: −1.81, 95% CI: −3.32, −0.30, $p = 0.02$) and similarities (B: −1.48, 95% CI: −2.86, −0.11, $p = 0.03$). Finally, children of women with AN showed lower verbal IQ scores (B: −8.20, 95% CI: −15.55, 0.85, $p = 0.03$) and lower full-scale IQ scores (B: −8.24, 95% CI: −15.91, 0.57, $p = 0.04$), in comparison with unexposed children (Table 2).

Discussion

This study investigated early cognitive development in children at high risk for an ED, due to being born to a mother with history of an ED, in comparison with children not at high risk. At 18 months (Griffiths development scales), children of women with lifetime AN showed comparatively poorer motor skills such as balance, coordination and movement control (Locomotor Development Score) and appeared to have less proficiency in daily activities such as feeding/dressing one's self, lower independence and less interaction with other children (Personal–Social Development score). At age 4 years (WPPSI), the children of women with AN showed poor perceptual skills, visual-motor coordination and possible figure ground deficits (Geometric and Block Design scores); poor social intelligence/understanding and social isolation (Comprehension score); and poor planning, logical thinking and abstract reasoning (Similarities score).

Lower full-scale IQ scores were observed in children of women with AN, with the main contribution to this appearing to be lower verbal IQ. This finding differs from the finding in our previous study investigating cognition at age 8 years (Kothari et al., 2012), where children of women with AN were found to have higher full-scale IQ with the main contribution coming from performance IQ. This could suggest a developmental change in ability, perhaps driven by a third factor such as perfectionism or persistence, traits that have previously been associated with AN (Halmi et al., 2000) and observed in first-degree relatives of probands (Woodside et al., 2002), or it may relate to biases within the samples studied at the various time points. At age 8 years, all children still part of the ALSPAC cohort were invited to participate; therefore, cognitive

Table 1 Linear regression analysis of children's Griffiths Scores: exposed versus unexposed (B coefficients, 95% confidence intervals and *p*-values)

		Model 1 B (95% CI)	*p*-value	Model 2 B (95% CI)	*p*-value	Model 3 B (95% CI)	*p*-value
Locomotor	Unexposed	Ref.		Ref.		Ref.	
	AN	−9.63 (−12.25, −7.02)	<0.001	−9.12 (−14.26, −3.98)	<0.001	−9.10 (−14.24, −3.95)	0.001
	BN	−0.32 (−2.63, 1.99)	0.89	−0.16 (−4.66, 4.34)	0.94	−0.17 (−4.70, 4.36)	0.94
	AN and BN	−5.13 (−7.84, −2.42)	0.06	−5.06 (−10.34, 0.23)	0.06	−5.16 (−10.42, 0.10)	0.06
Personal/social	Unexposed	Ref.		Ref.		Ref.	
	AN	−6.38 (−9.13, −3.62)	0.02	−5.97 (−11.29, -0.66)	0.03	−5.88 (−11.25, −051)	0.03
	BN	−0.76 (−3.19, 1.68)	0.76	−0.55 (−5.26, 4.16)	0.82	−0.57 (−5.28, 4.15)	0.82
	AN and BN	2.35 (−0.51, 5.21)	0.41	2.52 (−3.03, 8.07)	0.38	2.13 (−3.43, 7.69)	0.45
Hearing/speech	Unexposed	Ref.		Ref.		Ref.	
	AN	−4.87 (−9.06, −0.69)	0.24	−4.61 (−12.65, 3.42)	0.27	−4.30 (−12.35, 3.74)	0.30
	BN	1.20 (−2.51, 4.91)	0.75	0.97 (−5.82, 7.76)	0.79	0.93 (−6.00, 7.85)	0.80
	AN and BN	2.88 (−1.46, 7.23)	0.51	3.03 (−4.77, 10.82)	0.48	1.75 (−6.66, 10.16)	0.68
Hand/eye	Unexposed	Ref.		Ref.		Ref.	
	AN	−3.30 (−5.97, −0.64)	0.22	−3.51 (−8.68, 1.66)	0.19	−3.40 (−8.61, 1.80)	0.20
	BN	1.74 (−0.62, 4.10)	0.46	1.75 (−0.92, 4.42)	0.46	1.74 (−1.70, 5.18)	0.46
	AN and BN	2.74 (−0.02, 5.51)	0.32	2.65 (−2.75, 8.06)	0.34	2.21 (−3.19, 7.61)	0.43
Performance	Unexposed	Ref.		Ref.		Ref.	
	AN	−3.95 (−7.37, −0.53)	0.68	−3.80 (−10.35, 2.75)	0.26	−3.59 (−10.07, 2.90)	0.28
	BN	1.95 (−1.01, 4.90)	0.51	2.02 (−3.62, 7.65)	0.50	1.99 (−3.70, 7.67)	0.50
	AN and BN	3.81 (0.34, 7.28)	0.27	3.74 (−2.92, 10.40)	0.28	2.83 (−0.61, 6.27)	0.41
Average	Unexposed	Ref.		Ref.		Ref.	
Development	AN	−5.63 (−7.86, −3.39)	0.01	−5.40 (−9.77, −1.04)	0.02	−5.25 (−9.59, −0.92)	0.02
	BN	0.76 (−1.22, 2.74)	0.70	0.80 (−3.04, 4.65)	0.68	0.78 (−3.01, 4.58)	0.69
	AN and BN	1.33 (−0.99, 3.65)	0.57	1.38 (−3.13, 5.88)	0.55	0.75 (−3.70, 5.20)	0.74

Note:
AN, anorexia nervosa; BN, bulimia nervosa.
Model 1: Adjusted for child age and gender, and tester.
Model 2: Adjusted for child age, gender, birth weight and ethnicity; maternal age at delivery and marital status; parity and tester.
Model 3: Adjusted for child age, gender, birth weighte and ethnicity; maternal age at delivery, marital status and educational standard; parity and tester.
Sample sizes: AN = 14 (1.4%), BN = 18 (1.8%), AN and BN = 13 (1.3%) and unexposed = 937 (95.5%).

testing was undertaken on a larger sample but one that was subject to attrition from the study over time. Unfortunately, a direct comparison of children in this study and children investigated in Kothari et al. (Kothari et al., 2012) is not possible because of the attrition of a large proportion of children assessed at early time points, by age 8 years. Though the samples are too small for analysis, a visual inspection of the data showed high attrition of the children of women with AN who had the lowest full-scale IQ scores at age 4 years. Post hoc contrast analysis of the whole sample in this study showed that children with lower full-scale IQ score at age 4 years were less likely to attend at age 8 years. This may indicate a potential differential bias, but it is difficult to tell because of the small number of children at risk in this study. However, findings from both studies indicate the presence of difficulties in social understanding, visual-motor ability and planning/reasoning, in children at high risk for ED. These are all difficulties that have previously been observed in ED groups, those recovered from ED and first-degree relatives (Danner et al., 2012; Tchanturia et al., 2012; Tenconi, Santonastaso, et al., 2010; Treasure & Schmidt, 2013). It is also worth noting that while previous studies have reported comparatively high IQ in AN groups (Lopez, Stahl, & Tchanturia, 2010), particularly high verbal ability (Ranseen & Humphries, 1992), a recent study investigating intellectual function in ED patients found that individuals with AN showed poorer performance than the healthy control group on eight out of 13 subtests of the Wechsler Adult Intelligence Scale – III (Weider, Indredavik, Lydersen, & Hestad, 2013).

Strengths of this study are prospective data collection and longitudinal design, use of a large, population-based sample that improves generalizability of findings and availability of socio-demographic information that allows for the analysis of confounding features. The findings of the study were however limited by the small size of at-risk groups and self-report of ED by mothers at only one time point. Of particular interest is evidence of a potential bias in the previous study investigating children at high risk (Kothari et al., 2012), which highlights the need for more research. However, social, visuo-spatial and decision-making difficulties have been observed in the ED groups, (Danner et al., 2012; Tchanturia et al., 2012; Tenconi, Santonastaso, et al., 2010; Treasure & Schmidt, 2013) and in children at risk at both time points; therefore, differences in these constructs may be present prior to onset of ED. Further research should use longitudinal studies to investigate how the development of cognition and executive function in children at risk for ED varies from normal development. Finding from such research could have far reaching implications for discovering the aetiology of ED and for the design of prevention and early intervention strategies.

Table 2 Linear regression analysis of children's Wechsler Preschool and Primary Scale of Intelligence scores: exposed versus unexposed (B coefficients, 95% confidence intervals and *p*−values)

		Model 1 B (95% CI)	*p*-value	Model 2 B (95% CI)	*p*-value	Model 3 B (95% CI)	*p*-value
Object	Unexposed	Ref.		Ref.		Ref.	
Assembly	AN	−1.51 (−3.34, 0.33)	0.11	−1.53 (−3.37, 0.32)	0.11	−1.56 (−3.40, 0.27)	0.10
	BN	1.24 (−0.52, 2.99)	0.17	1.28 (−0.32, 2.88)	0.15	1.17 (−0.53, 2.86)	0.19
	AN and BN	−0.22 (−2.14, 1.70)	0.82	−0.28 (−2.14, 1.57)	0.77	−0.57 (−2.49, 1.34)	0.56
Geometric	Unexposed	Ref.		Ref.		Ref.	
Design	AN	−1.43 (−2.84,−0.01)	0.05	−1.50 (−2.89, −0.10)	0.04	−1.71 (−3.09, −0.33)	0.02
	BN	0.47 (−0.89,1.82)	0.50	0.58 (−0.62, 1.78)	0.39	0.43 (−0.88, 1.74)	0.52
	AN and BN	0.22 (−1.26,1.71)	0.77	0.14 (−1.04, 1.33)	0.85	−0.15 (−1.58, 1.28)	0.84
Block	Unexposed	Ref.		Ref.		Ref.	
Design	AN	−1.52 (−3.26, 0.23)	0.09	−1.75 (−3.48, −0.02)	0.05	−1.79 (−3.50, −0.08)	0.04
	BN	1.09 (−0.58,2.76)	0.20	1.07 (0.05, 2.09)	0.20	0.94 (0.11, 1.77)	0.26
	AN and BN	−0.44 (−2.26,1.39)	0.64	−0.62 (−2.37, 1.14)	0.50	−0.95 (−2.60, 0.69)	0.30
Mazes	Unexposed	Ref.		Ref.		Ref.	
	AN	0.75 (−0.96,2.46)	0.39	0.65 (−1.07, 2.37)	0.46	0.62 (−1.09, 2.33)	0.48
	BN	−0.75 (−2.39,0.89)	0.37	−0.79 (−1.62, 0.05)	0.34	−0.90 (−2.45, 0.66)	0.28
	AN and BN	0.32 (−1.48,2.11)	0.73	0.27 (−1.45, 1.98)	0.77	−0.01 (−1.79, 1.78)	1.00
Picture	Unexposed	Ref.		Ref		Ref.	
Completion	AN	−0.25 (−1.73,1.24)	0.74	−0.32 (−1.79, 1.16)	0.67	−0.35 (−1.81, 1.12)	0.64
	BN	1.36 (−0.06,2.79)	0.06	1.33 (−0.06, 2.72)	0.06	1.24 (−0.14, 2.61)	0.08
	AN and BN	0.11 (−1.45,1.66)	0.89	−0.02 (−1.55, 1.51)	0.98	−0.27 (−1.79, 1.26)	0.73
Information	Unexposed	Ref.		Ref.		Ref.	
	AN	−1.23 (−2.99,0.52)	0.17	−1.28 (−2.98, 0.42)	0.14	−1.33 (−3.00,0.33)	0.12
	BN	1.08 (−0.60,2.76)	0.21	0.96 (0.13, 1.79)	0.25	0.78 (−0.03, 1.58)	0.34
	AN and BN	0.81 (−1.02,2.65)	0.39	0.72 (−0.98, 2.42)	0.43	0.25 (−1.51, 2.02)	0.78
Comprehension	Unexposed	Ref.		Ref.		Ref.	
	AN	−1.72 (−3.30,−0.14)	0.03	−1.75 (−3.30, −0.20)	0.03	−1.81 (−3.32, −0.30)	0.02
	BN	0.56 (−0.95,2.08)	0.47	0.51 (−0.82, 1.84)	0.50	0.31 (−0.43, 1.04)	0.68
	AN and BN	0.86 (−0.80,2.51)	0.31	0.82 (−0.79, 2.42)	0.33	0.31 (−1.26, 1.88)	0.70
Arithmetic	Unexposed	Ref.		Ref.		Ref.	
	AN	−0.78 (−2.24,0.69)	0.30	−0.81 (−2.27, 0.64)	0.28	−0.85 (−2.28, 0.57)	0.24
	BN	0.26 (−1.15,1.66)	0.72	0.25 (−0.46, 0.95)	0.73	0.11 (−1.19, 1.41)	0.88
	AN and BN	−0.16 (1.69,1.38)	0.84	−0.18 (−0.95, 0.60)	0.82	−0.53 (−2.02, 0.97)	0.49
Vocabulary	Unexposed	Ref.		Ref.		Ref	
	AN	−0.76 (−2.44, 0.93)	0.38	−0.82 (−2.51, 0.86)	0.34	−0.87 (−2.53, 0.80)	0.31
	BN	0.34 (−1.27, 1.95)	0.68	0.27 (−0.55, 1.09)	0.74	0.13 (−0.68, 0.93)	0.87
	AN and BN	0.54 (−1.23,2.30)	0.55	0.42 (−1.36, 2.19)	0.64	0.06 (−1.67, 1.80)	0.94
Similarities	Unexposed	Ref.		Ref.		Ref.	
	AN	−1.35 (−2.75,0.05)	0.06	−1.44 (−2.84, −0.05)	0.04	−1.48 (−2.86, −0.11)	0.03
	BN	0.30 (−1.05,1.64)	0.67	0.23 (−1.05, 1.50)	0.74	0.10 (−0.21, 0.40)	0.89
	AN and BN	0.34 (−1.13,1.81)	0.65	0.26 (−1.19, 1.71)	0.73	−0.08 (−1.50, 1.35)	0.92
Performance	Unexposed	Ref.		Ref.		Ref.	
IQ	AN	−4.94 (−13.42,3.53)	0.25	−5.73 (−14.11, 2.65)	0.18	−5.97 (−14.19, 2.25)	0.15
	BN	4.74 (−3.37,12.86)	0.25	4.87 (−2.96, 12.70)	0.23	4.06 (−2.43, 10.54)	0.31
	AN and BN	0.15 (−8.73,9.02)	0.97	−0.61 (−9.28, 8.07)	0.89	−2.66 (−11.18, 5.86)	0.54
Verbal IQ	Unexposed	Ref.		Ref.		Ref.	
	AN	−7.51 (−15.30,0.28)	0.06	−7.89 (−15.53, −0.25)	0.04	−8.20 (−15.55,− 0.85)	0.03
	BN	3.42 (−4.04,10.89)	0.37	3.05 (−3.70, 9.81)	0.41	2.02 (−1.55, 5.58)	0.57
	AN and BN	2.94 (−5.23,11.10)	0.48	2.46 (−5.27, 10.18)	0.54	−0.16 (−7.77, 7.45)	0.97
Full-scale IQ	Unexposed	Ref.		Ref.		Ref.	
	AN	−7.24 (−15.38,0.90)	0.08	−7.92 (−15.88, 0.04)	0.05	−8.24 (−15.91, −0.57)	0.04
	BN	4.66 (−3.14,12.45)	0.24	4.52 (−2.81, 11.84)	0.24	3.44 (−0.28, 7.16)	0.36
	AN and BN	1.52 (−7.01,10.05)	0.73	0.80 (−7.40, 9.00)	0.85	−1.92 (−9.87, 6.02)	0.64

Note:
AN, anorexia nervosa; BN, bulimia nervosa.
Model 1: Adjusted for child age and gender, and tester.
Model 2: Adjusted for child age, gender, birth weight and ethnicity; maternal age at delivery and marital status; parity and tester.
Model 3: Adjusted for child age, gender, birth weight and ethnicity; maternal age at delivery, marital status and educational standard; parity and tester.
Sample sizes: AN = 11 (1.3%), BN = 12 (1.4%), AN and BN = 10 (1.2%) and unexposed = 819 (96.1%).

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REFERENCES

Boyd, A., Golding, J., Macleod, J., Lawlor, D. A., Fraser, A., Henderson, J., & Davey Smith, G. (2012). Cohort profile: The 'children of the 90s'—the index offspring of the avon longitudinal study of parents and children. *International journal of epidemiology*, *42*(1), 111–127. DOI: 10.1093/ije/dys064

Danner, U. N., Sanders, N., Smeets, P. A. M., van Meer, F., Adan, R. A. H., & Hoek, H. W., et al. (2012). Neuropsychological weaknesses in anorexia nervosa: Set-shifting, central coherence, and decision making in currently ill and recovered women. *International Journal of Eating Disorders*, *45*(5), 685–694. DOI: 10.1002/eat.22007

Griffiths, R. (1984). The abilities of young children: A comprehensive system of mental measurement for the first eight years of life. Bucks, England: The Test Agency.

Halmi, K. A., Sunday, S. R., Strober, M., Kaplan, A., Woodside, D. B., & Fichter, M., et al. (2000). Perfectionism in anorexia nervosa: Variation by clinical subtype, obsessionality, and pathological eating behavior. *American Journal of Psychiatry*, *157*(11), 1799–1805.

Kothari, R., Solmi, F., Treasure, J., & Micali, N. (2012). The neuropsychological profile of children at high risk of developing an eating disorder. *Psychological Medicine*, *43*(7), 1543–1554.

Lindner, S. E., Fichter, M. M., & Quadflieg, N. (2013). Central coherence in full recovery of anorexia nervosa. *European Eating Disorders Review*, *21*(2), 115–120. DOI: 10.1002/erv.2213

Lopez, Stahl, D., & Tchanturia, K. (2010). Estimated intelligence quotient in anorexia nervosa: A systematic review and meta-analysis of the literature. *Annals of General Psychiatry*, *9*(1), 40.

Micali, N., De Stavola, B., dos-Santos-Silva, I., Steenweg-de Graaff, J., Jansen, P. W., Jaddoe, V. W., et al. (2012). Perinatal outcomes and gestational weight gain in women with eating disorders: A population-based cohort study. *BJOG: an international journal of obstetrics and gynaecology*, *119*(12), 1493–1502. DOI: 10.1111/j.1471-0528.2012.03467.x

Micali, N., Northstone, K., Emmett, P., Naumann, U., & Treasure, J. L. (2012). Nutritional intake and dietary patterns in pregnancy: A longitudinal study of women with lifetime eating disorders. *The British journal of nutrition*, *108*(11), 2093–2099. DOI: 10.1017/S0007114512000256

Micali, N., Simonoff, E., & Treasure, J. (2007). Risk of major adverse perinatal outcomes in women with eating disorders. *The British Journal of Psychiatry*, *190*(3), 255–259. DOI: 10.1192/bjp.bp.106.020768

Micali, N., Simonoff, E., & Treasure, J. (2011). Pregnancy and postpartum depression and anxiety in a longitudinal general population cohort: The effect of eating disorders and past depression. *Journal of Affective Disorders*, *131*(1–3), 150–157.

Ranseen, J. D., & Humphries, L. L. (1992). The intellectual functioning of eating disorder patients. *Journal of the American Academy of Child and Adolescent Psychiatry*, *31*(5), 844–846.

Shott, M. E., Filoteo, J. V., Bhatnagar, K. A. C., Peak, N. J., Hagman, J. O., Rockwell, R., & Frank, G. K. W. (2012). Cognitive set-shifting in anorexia nervosa. *European Eating Disorders Review*, *20*(5), 343–349. DOI: 10.1002/erv.2172

Strober, M., Freeman, R., Lampert, C., Diamond, J., & Kaye, W. (2000). Controlled family study of anorexia nervosa and bulimia nervosa: Evidence of shared liability and transmission of partial syndromes. *American Journal of Psychiatry*, *157*(3), 393–401. DOI: 10.1176/appi.ajp.157.3.393

Tchanturia, K., Liao, P.–C., Forcano, L., Fernández-Aranda, F., Uher, R., Treasure, J., & Campbell, I. C. (2012). Poor decision making in male patients with anorexia nervosa. *European Eating Disorders Review*, *20*(2), 169–173. doi: 10.1002/erv.1154

Tenconi, Santonastaso, P., Degortes, D., Bosello, R., Titton, F., Mapelli, D., & Favaro, A. (2010). Set-shifting abilities, central coherence, and handedness in anorexia nervosa patients, their unaffected siblings and healthy controls: Exploring putativeendophenotypes. *The World Journal of Biological Psychiatry*, *11*(6), 813–823.

Treasure, J., & Schmidt, U. (2013). The cognitive-interpersonal maintenance model of anorexia nervosa revisited: A summary of the evidence for cognitive, socio-emotional and interpersonal predisposing and perpetuating factors. *Journal of Eating Disorders*, *1*, 1–13.

Wechsler, D. (1990). Wechsler preschool and primary scale of intelligence - Revised Edition. San Antonio, TX: The Psychological Corp., Harcourt Brace.

Weider, S., Indredavik, M. S., Lydersen, S., & Hestad, K. (2013). Intellectual function in patients with anorexia nervosa and bulimia nervosa. *European Eating Disorders Review*, *22*(1), 15–24.

Woodside, D. B., Bulik, C. M., Halmi, K. A., Fichter, M. M., Kaplan, A., Berrettini, W. H., & Klump, K. (2002). Personality, perfectionism, and attitudes toward eating in parents of individuals with eating disorders. *International Journal of Eating Disorders*, *31*(3), 290–299.