

TITLE PAGE

Title: Childhood Risk Factors for Lifetime Bulimic or Compulsive Eating by Age 30 Years in a British National Birth Cohort

D Nicholls MD ^{1,2}

R Statham BSc ¹,

S Costa PhD ²,

N Micali PhD ²,

RM Viner PhD ²

¹ Department of Child and Adolescent Mental Health, Great Ormond Street Hospital, London, WC1N 3JH

² UCL Institute of Child Health, 30 Guilford Street, London, WC1N 1EH

Corresponding author:

Dr Dasha Nicholls, Department of Child and Adolescent Mental Health, Great Ormond Street Hospital, London, WC1N 3JH

Telephone: + 44 (0)20 7829 8679

Email: d.nicholls@ucl.ac.uk

Abstract: 164

Word count: 4517 text (excluding references) plus 1458 tables

Key words: Eating disorders, risk factors, childhood, bulimia nervosa, binge eating, compulsive eating

ACKNOWLEDGEMENTS/DISCLOSURE OF CONFLICTS:

The study was unfunded. The authors have no conflicts of interest to declare.

Accepted Manuscript

ABSTRACT

Objective: To examine whether previously identified childhood risk factors for bulimia or compulsive eating (BCE) predict self-reported lifetime BCE by age 30 years in a prospective birth cohort.

Method: Using data from the 1970 British Cohort Study at birth, 5, and 10 years, associations between 22 putative childhood risk factors and self-reported lifetime BCE at 30 years were examined, adjusting for sex and socioeconomic status. **Results:** Only female sex (odds ratio (OR): 9.2; 95% confidence interval (CI): 1.9-43.7; $p=0.005$), low self-esteem (OR:2.9; 95%CI: 1.1-7.5; $p=0.03$) and high maternal education (OR:5.4; 95%CI: 2.0-14.8; $p=0.001$) were significantly associated with higher risk of BCE, whereas high SES at 10 years was significantly protective (OR:0.2; 95%CI: 0.1-0.8; $p=0.022$) of BCE in fully adjusted multivariable logistic regression analysis. **Discussion:** Our findings do not support a strong role for childhood weight status and eating behaviours in the development of bulimia and compulsive eating pathology, rather suggesting a focus on self esteem may have greater relative importance. Findings in relation to maternal education and SES need further exploration.

BACKGROUND

Identification of childhood risk factors that might predict later problem eating behaviours is important for understanding etiology and developing prevention and early intervention strategies to combat rising rates of obesity and eating disorders (Jacobi, Fittig et al. 2011). Research has suggested that loss of control over eating is associated with increased general as well as eating related psychopathology, and for overweight and obesity (Kalarchian and Marcus 2012). Bulimic, compulsive or binge eating, abbreviated hereafter as BCE (bulimic or compulsive eating) is defined as regularly consuming large quantities of food over a short period of time in the absence of hunger associated and with loss of control. It is a common feature of adolescent and adult onset eating disorders and is a risk factor for other mental health difficulties such as depression, as well as for obesity. The development of BCE is multifactorial, involving a combination of genetic, metabolic, environmental, and behavioural factors. Whilst research continues in both animal models (Di Segni, Patrono et al. 2014) and humans (Balodis, Grilo et al. 2015) to understand the neuroscientific basis of the reward and reinforcing processes that underlie BCE (Kessler, Hutson et al. 2016), there is a pressing need to identify potentially modifiable social and psychological aspects of risk in light of the physical and psychological morbidity associated with BCE. For example, several studies suggest a link between stress, access to highly palatable food, and BCE, as a pathway to the development of obesity (Sominsky and Spencer 2014).

Risk factor research is fraught with methodological challenges however. Most studies of risk for eating disorders do not have sufficient power to distinguish risk factors for restrictive eating, as seen in anorexia nervosa (AN), from BCE, seen in patients with bulimia nervosa (BN) and binge eating disorder (BED) (Stice, Ng et al. 2010). Risk factor research is also often limited by cross-sectional or retrospective design and small sample sizes. Kraemer et al. (Kraemer, Kazdin et al. 1997) emphasise precedence as an essential requirement for a risk factor (with the exception of fixed markers such as female status), thus requiring validation by longitudinal research. Data from national birth cohorts addresses many methodological weaknesses of childhood risk factor research. Samples are large, representative of the population and assess

risk factors across the lifetime of the sample. Furthermore, cohort data lack systematic bias with regard to hypotheses, since data were not collected to investigate eating-related behaviours.

The current study tested hypotheses posed in the existing literature regarding possible psychological and social risk factors for BCE, using data from the British 1970 birth cohort (BCS70) cohort. Risk factors for BCE were identified from recently published authoritative reviews (Jacobi 2005, Stice, Ng et al. 2010, Keel and Forney 2013). We tested factors that had previously been confirmed from longitudinal research (replication); where longitudinal findings were inconclusive due to inadequate power (confirmation); or where risk factor status was based on retrospective or cross sectional data and had not been confirmed by longitudinal design (validation). We focussed on potentially modifiable risk factors typically found in cohort data such as perinatal factors, childhood behaviours (both eating and non eating related), parental factors including eating disorders and substance misuse, psychological factors such as low self esteem, and childhood or parental obesity. Previous findings regarding the role of prenatal and perinatal factors in risk for EDs have been mixed (Krug, Taborelli et al. 2013) and therefore positive findings require replication. Findings for BMI and childhood eating behaviour as risk factors for bulimic and binge eating disorders are inconsistent in the literature, most studies being retrospective in nature, meaning validation as risk factors for, rather than consequences of, disordered eating behaviour is required. Other childhood factors such as inattention may also play a role (Sonneville, Calzo et al. 2015). The role of parental factors is also extremely important to clarify, since many early intervention treatment strategies involve parents. Our aim was to seek the most robust markers predicting BCE by age 30.

METHODS

The BCS70 longitudinal study enrolled 16,567 babies from England, Scotland, Wales and Northern Ireland born in one week in April 1970, with follow-up at five years (n=13,135: 21% attrition), 10 years (n=14,875: 10% attrition) and 30 years (n=11,261: 32% attrition). Datasets were obtained electronically from the UK Data Service (<http://ukdataservice.ac.uk>), who gave approval for analysis. As efforts were made to recruit 'difficult to reach' participants at 30 years, there was

only 3.9% additional attrition from lower socioeconomic status participants (those who had fathers in manual employment) between birth and the 30 year follow-up. Ethics consent for participation and for future secondary analyses of anonymized data was obtained at each survey from parent and participant (when adult). Specific ethics approval was not necessary for these analyses.

Case Definition

At age 30, participants were interviewed and completed a questionnaire asking if they had ever had or been told that they had: "*Anorexia Nervosa*", "*Bulimia or compulsive eating*", "*problems with swallowing*" or "*some other kind of eating problem?*". Those answering "no" to all eating problems formed the control group (n=10,805). Participants who reported "*Bulimia or compulsive eating*" formed the BCE group. No definition of BCE was provided. Participants answering yes to an eating problem other than BCE (n=299), and those answering yes to BCE plus AN (n=16) were excluded from analyses. Participants were also asked "Have you had an eating disorder in the last 12 months?", "Have you seen a doctor in the past 12 months about your eating disorder?" and "How old were you when you first had an eating disorder?". Individuals reporting onset of BCE before age 10 were excluded to ensure risk factors preceded onset. The BCE group comprised 91 people, approximately 1% of the whole sample.

Risk Factor Variables

The most appropriate variables in the BCS70 dataset to represent risk factors identified from the literature were identified (table 1). There were no variables from the five or 10-year data representative of the following hypothesised risk factors: thin body preoccupation, body dissatisfaction, interoceptive awareness, perfectionism, social support from family, sexual victimization, parental eating attitudes, parental perception of child's weight, negative comments about weight, and family history of depression and drug use.

Obstetric risk factors. Prematurity was defined as gestational age <37 weeks. Very low birth weight was defined as <1500g. Data on anaemia during pregnancy, maternal diabetes, antenatal admissions into hospital, administration of oxygen to baby, abnormal foetal heart rate (<120 or >160 beats/minute) and eclampsia during labour were combined to give a six-factor obstetric

risk score to indicate perinatal problems. Risk-scores were dichotomised into “none” and “ ≥ 1 risk factor” for analyses.

Child risk factors Health problems before age five were assessed from maternal report (at five years) of ‘biliousness’, head and stomach aches more than monthly for the previous 12 months, dichotomised into “none” and “ ≥ 1 health problem”. Sleep problems were by maternal report at five years to the question “Does your child have any sleeping difficulty?”, dichotomised into “yes”/“no”.

At 10 years, children were weighed and measured by a Medical Officer, school nurse or health visitor, and BMI z-scores (zBMI) for gender calculated using UK 1990 revised growth reference (Cole, Freeman et al. 1995) (contemporaneous BMI reference data were not available). Obesity at 10 years was defined as BMI $> 95^{\text{th}}$ centile. Mean zBMI for the cohort at 10 years was -0.10 (standard deviation (SD) 1.00), reflecting the rightwards shift in mean BMI between 1980 and derivation of the growth reference. zBMI at age 10 was investigated as a continuous variable. Evidence of puberty at 10 years (yes/no) was identified by the school physician after physical assessment.

The Social Development Scale contained items from Conner’s Teacher Rating Scale and Rutter Teaching Scale. A 14-item conduct problems/impulsivity/hyperactivity subscale and an eight-item attention deficit subscale has been previously identified by factor analysis (Irving 2002). We defined high scorers as > 1 SD from the sample mean in the direction of greater problems for all child behaviour scales. Self-esteem was assessed with the Lawrence Self-Esteem Questionnaire (Lawrence 1981), and categorised into: “medium” for scores between -1 SD and +1 SD of the population mean, “low” for scores > 1 SD below and “high” for scores > 1 SD above the population mean (Hart 1985). Childhood anxiety and negative affect/emotionality was investigated with reports of absence from school due to emotional reasons over the last 12 months (yes/no) and scores > 1 SD above the sample mean on the Rutter scale of emotional problems at 10 years. Escape-avoidance coping or personal ineffectiveness were identified by an external locus of control score > 1 SD below the sample mean on the Caraloc scale of ability

to control destiny. Impulsivity was defined by teacher and mother reports of the child being 'impulsive/excitable' at 10 years (yes/no) in the Rutter scale. Adverse family experience was defined by ever having been in social care (maternal report at ages five and 10).

Childhood eating problems were explored using maternal report of the following variables at age five and 10: eating problems (yes/no), under-eating (yes/no), overeating (yes/no), and other eating problems (yes/no). Child report of not eating anything for lunch at school at 10 years was used as a proxy for dietary restriction. Self-report of smoking regularly (>1 cigarette/week) and having tried >1 cigarette at 10 years were used as indicators for substance misuse in childhood.

Parental risk factors. Parental obesity was defined by having one or both parents with BMI $\geq 30 \text{ kg/m}^2$.

Maternal and paternal BMI were also investigated as continuous variables, based on mother-reported height and weight. BMI was converted to a z-score (zBMI) using the mean and SD from within the cohort for each parent.

In the absence of EDs measures, parental BMI $< 18.5 \text{ kg/m}^2$ was used as a proxy for family history of AN. Parental substance misuse was investigated by maternal report (at 10 years) of drinking alcohol >1 times per week during early and/or late pregnancy (yes/no), smoking during pregnancy (non-smoker, stopped periconception, or smoked during pregnancy), and parental smoking patterns between 1970-1980 (never smoked, smoked ≤ 5 years, or persistent smoker).

The Rutter Malaise Inventory is derived from the Cornell medical index and designed to assess adult psychiatric morbidity (Rutter 1970). At five years a total score between 0-24 was obtained by summing all positive responses and score ≥ 7 used to define high scorers for maternal Malaise, which has been reported to indicate high risk for psychiatric disorder (Rodgers, Pickles et al. 1999) and AN (Nicholls and Viner 2009). At 10 years, mothers were asked to rate themselves on a novel Likert scale between zero ("Seldom or never") to 100 ("Most of the time") to derive a score comparable to the standard scoring system. High maternal Malaise at 10 years was defined as a score >1 SD above the mean.

Analyses controlled for maternal education and family socioeconomic status (SES) at 10 years, and participant's SES at 30 years. Maternal education at 10 years was dichotomised into "lower than A-levels" (University entry requirement) and "A-levels or higher" for analyses. Childhood SES was assessed from father and mother's social class (mother's report at 10 years), dichotomised into 'Professional/Managerial' and "other". SES at 30 years was assessed from participant's social class, dichotomised as above.

Data Analysis

Chi-square and Fisher's exact tests (where >20% cells have expected counts <5) were used to assess differences between groups. Mann-Whitney U test was used to investigate differences where data were non-normally distributed. Logistic regression with robust standard errors (Huber-White sandwich) was used to investigate associations between risk factors and lifetime BCE. Univariable logistic regression used BCE as the outcome and each of the risk factors separately as independent variables, adjusting for sex, maternal education and SES at 10 years, and participant's SES at 30 years. Risk factors where $p \leq 0.1$ were introduced into a multivariable logistic regression model. Post-hoc correlation matrices were used to check for collinearity, and model fit was checked with the Hosmer-Lemeshow goodness of fit test and Bayesian information criteria. Participant's SES at 30 years was dropped from the final multivariable model due to collinearity with SES at 10 years ($r = -0.51$) and lack of association with BCE in both partly adjusted univariable and multivariable regression models. This improved the standard errors and 95% confidence interval (CI) for SES at 10 years, the correlation matrix and final multivariable regression model fit according to the Bayesian information criterion. Significance was set at $p < 0.05$, and all data were analysed in Stata (v.12.1).

We chose to investigate our power prior to carrying out the analyses, as this is a less biased approach compared to testing for power after the analyses have been carried out. Power calculations were conducted for potential risk factors to ensure that power was adequate despite relatively low prevalence of some variables. Our sample was sufficient to provide 80%

power to detect an odds ratio (OR) of 2 for common exposures (i.e. prevalence over 10%) where the ratio of exposure in cases to controls was > 1:1. For uncommon exposures (e.g. <2% prevalence) we had sufficient power to detect OR \geq 4.

RESULTS

At 30 year follow-up, 11,211 participants (99.6% of the cohort) answered questions about lifetime eating disorders (EDs), of which 406 reported an eating difficulty and 91 reported having BCE alone.

The BCE group consisted of nine males (9.9%) and 82 females (90.1%). Median age of onset of the EDs was 18 years (Inter-quartile range (IQR): 16-23) and median BMI at 30 years was 22.8 Kg/m² (IQR: 21.0-26.5), of whom 61% were normal weight, 2% underweight, and 37% overweight or obese according to the World Health Organisation definitions. Twenty-eight participants (30.8%) in the BCE group reported an eating problem in the last 12 months. Of these, 17 (60.7%) had seen a physician about their eating in the last 12 months. The control group consisted of 5359 males (49.6%) and 5446 females (50.4%), with a median BMI at 30 years of 24.2 Kg/m² (IQR: 21.9-27.1). Amongst controls, 56% had normal weight, 2% were underweight, and 42% were overweight or obese (difference between BCE and control groups' BMI status: $p < 0.05$). Descriptive statistics for the BCE and control groups on all variables are seen in table 2. Sample sizes vary by response rate.

Female sex, eating problems and overeating at five years, zBMI and low-self-esteem at 10 years, absence from school for emotional reasons, teacher-reported impulsivity, paternal smoking between 1970-1980, maternal smoking in pregnancy, alcohol consumption during early and late pregnancy, and high maternal malaise score all reached $p \leq 0.1$ in partly adjusted univariable analyses, and were entered into the multivariable model.

In the multivariable model (table 3), only female sex (OR:9.2; 95%CI: 1.9-43.7; $p=0.005$), low-self-esteem (OR:2.9; 95%CI: 1.1-7.5; $p=0.03$) and high maternal education (OR:5.4; 95%CI: 2.0-

14.8; $p=0.001$) were significantly associated with higher risk of lifetime BCE, whereas high family SES at 10 years was protective (OR:0.2; 95%CI: 0.1-0.8; $p=0.022$) of lifetime BCE.

Accepted Manuscript

DISCUSSION

This large prospective study explored possible childhood risk factors for bulimic or compulsive eating by age 30. After adjustment for SES, lifetime BCE was associated with female sex, eating problems and overeating at five years, obesity at 10 years, absence from school for emotional reasons, low-self-esteem, teacher-reported impulsivity, parental smoking, and maternal smoking and alcohol consumption during pregnancy, and high maternal Malaise score at five years at the univariable level. However, only female sex, low-self-esteem and high maternal education remained significant predictors of lifetime BCE in multivariable analysis, while high childhood SES was protective.

We found no significant association between lifetime BCE and many of the risk factors proposed by previous prospective, retrospective or cross-sectional research. For example, we found no association between perinatal (i.e. obstetric risk) factors and lifetime BCE. Among the previous findings regarding prenatal and perinatal risk, very low birth weight, preeclampsia and prenatal complications have been implicated in later bulimic and binge eating presentations (Foley, Thacker et al. 2001, Wehkalampi, Hovi et al. 2010). In this large cohort, measures of obstetric and perinatal risk were highly reliable, albeit measured over 35 years ago.

Our study found that childhood obesity was not associated with BCE risk and zBMI at 10 years was significant in univariable analysis but not in the multivariable model. Using the more inclusive International Obesity Task Force overweight definition produced similar non-significant findings (data not shown) (Cole, Bellizzi et al. 2000). Higher childhood BMI is associated with eating problems, in particular BED, in some studies (Vollrath, Koch et al. 1992, Killen, Hayward et al. 1994, Fairburn, Welch et al. 1997, Micali, Holliday et al. 2007) and unrelated to later eating disturbance, caseness or partial syndromes in others (Killen, Taylor et al. 1996, Patton, Selzer et al. 1999, The McKnight 2003) although the retrospective design of many studies is a limitation. This mixed evidence mirrors our finding that childhood BMI was not sufficiently specific as a risk factor to carry significance in a multivariable model, and supports the hypothesis that obesity is heterogeneous with respect to etiology and risk, with only a proportion of obese subjects showing compulsive eating behaviour and obesity being a risk

factor for only a proportion of those with later problem eating. Allen et al. (Allen, Byrne et al. 2009) found that parental perception of child weight was more important than actual weight in predicting later disordered eating, a hypothesis we were unable to investigate.

Prospective research reports a relationship between childhood eating problems and later BN (Micali, Holliday et al. 2007) (Marchi and Cohen 1990, Kotler, Cohen et al. 2001) although the nature of the eating problem is not consistent between these studies. Again, we found overeating at age five predicting later BCE at a univariable but not multivariable level. No other eating problems showed any significant associations.

Elevated stress levels may precede BED, although causal direction is difficult to establish (Striegel-Moore, Dohm et al. 2007). In our study, associations at a univariable level for factors relating to both child and parental emotional factors did not persist after adjustment for covariates in multivariable analyses. This contrasts with the findings of Striegel-Moore et al. (Striegel-Moore, Fairburn et al. 2005), who identified higher rates of familial eating problems, parental mood and substance misuse, perfectionism, separation from parents, and maternal problems with parenting in subjects with BED compared to healthy controls. However the findings were not based on prospective data. Longitudinal studies do identify psychiatric morbidity and negative emotionality as risk factors for general eating pathology (Jacobi 2005). For example, Moorhead et al. (Moorhead, Stashwick et al. 2003) found that maternal report of pregnancy complications, health problems in their daughters before age five and anxiety/depression in their daughters at age nine predicted later EDs. We did not replicate this finding. Childhood sleep problems have also been associated with BED, even after accounting for obesity (Trace, Thornton et al. 2012), another finding not replicated in the BC70 cohort based on reported sleep problems at age five. Together these findings suggest that whilst some emotional, parental and health factors may be important at an individual level, the association is not sufficiently robust to imply causation or to generalise across patient cohorts.

There are reported associations between BN and substance misuse or addiction (Krug, Pinheiro et al. 2009). Montgomery et al. found that adjusted OR for BN in offspring were lower for mothers

who gave up before pregnancy than for those who gave up during pregnancy or smoked throughout pregnancy, and suggested a causal relationship operating through compromised central nervous system development (Montgomery, Ehlin et al. 2005). In our sample, maternal smoking throughout pregnancy and paternal smoking during childhood was significant at the univariable but not the multivariable level. This suggests that a direct mediational pathway is unlikely to be correct. Smoking is significantly associated with SES, which was controlled for. Maternal alcohol consumption in pregnancy was also significant only at the univariable level in our cohort. It may be that these risk factors operate through other mechanisms, and hence lose significance as indicators of risk in their own right.

Media, family and peer influence on development and maintenance of EDs has been highlighted previously (Field, Javaras et al. 2008, Eisenberg and Neumark-Sztainer 2010, Haines, Kleinman et al. 2010, Skinner, Haines et al. 2012), but high maternal education status as a risk factor and high family SES as protective are new findings from our study. Poor correlation between maternal education and family SES ($r=-0.074$) in post-hoc analyses excludes collinearity as an explanation. It is possible that maternal education and SES mediate help seeking behaviour or self-identification (and therefore reporting) of problem behaviours, rather than BCE directly. Almost two thirds of the sample had seen a physician for their eating difficulties, which seems higher than would normally be expected.

Our study confirms the importance of self-esteem for development of problem eating. Low-self-esteem, low social support from family, and escape avoidance coping style have previously been implicated in the development of EDs (Ghaderi 2003).

Critical comments about shape, weight, or eating and negative life events are known to increase short term risk for BCE (Pike, Wilfley et al. 2006), but could not be investigated using BCS70 data. We were also unable to examine the role of perfectionism, which could mediate the association between high maternal education and disordered eating in the presence of low-self-esteem. Future research should focus on possible mediational pathways.

Strengths and limitations

National birth cohort data provide large nationally representative samples and prospective data collection, reducing the possibility of memory or information bias. Data collection was unrelated to EDs and therefore unbiased by the hypotheses. Nonetheless, there were a number of limitations. Self-report of BCE was highly specific but not defined and could have resulted in under-reporting of symptoms. As noted above, awareness of loss of control eating in the general population was also limited at the time of data collection. The prevalence of BCE in the entire cohort was close to 1%, which is lower than expected (Trace, Thornton et al. 2012). EDs in general are under-reported and under-detected, although self-report is more reliable than interview (Fursland and Watson 2014). Possibly those with severe symptoms reported BCE, reflecting differences between population and help seeking samples; a hypothesis supported by the high number (61%) who had seen a physician in the last 12 months for eating difficulties. We are relatively confident that BCE is not simply a correlate of morbid obesity. Median BMI was 22.81 kg/m² (IQR=20.98-26.60) in the BCE group, compared to 24.21 kg/m² (IQR=21.93-27.11) in controls. However this finding also suggests that the sample may be biased towards those who also had other ED pathology including compensatory behaviours seen in BN, rather than BCE alone, as occurs in BED.

Whilst we cannot assume that in general self report of BCE is equivalent to a diagnosis BN or BED, this particular sample, with a normal weight distribution and high levels of contact with health services for eating difficulties, may more closely resemble a clinical population with eating disorders than those with BCE in the general population. Age of onset of EDs reported by the BCE group was comparable to that reported by Hudson and colleagues for BN (Hudson, Hiripi et al. 2007). This suggests a reasonable predictive validity of the findings of our study with respect to clinical significant outcomes.

Some risk factors could have been defined in a number of ways, and this may in part explain the lack of association seen for some risk factors that had been identified in previous research.

However, findings for sleep problems at five years were the same whether categorized as

present “yes”/“no” or as “no”/“mild”/“severe”. Similarly, childhood obesity defined as >95th centile had a low prevalence, reducing power; but increasing prevalence using the International Obesity Task Force threshold for overweight did not change the results. zBMI as a continuous variable was significant at the univariable but not multivariable level.

Analyses were limited by the data available. Some risk factors could not be explored; others were not optimally measured (e.g. parental underweight as a proxy for AN). Dichotomising risk factors may be too simplistic to detect associations. Our power calculations suggest that the data was powered to detect strong but not weaker associations. False negatives due to small numbers and low prevalence of the outcome may explain the lack of association with some previously identified risk factors. We were unable to explore the role of childhood maltreatment, including sexual abuse, which has been implicated in development of disordered eating in some patients (Wonderlich, Brewerton et al. 1997, Chou 2012, Backholm, Isomaa et al. 2013). Dietary restraint, body dissatisfaction and ‘weight concerns’ or thin body preoccupation have also been investigated across the ED spectrum with variable definitions, measurements and inconsistent findings (Killen, Taylor et al. 1996, Johnson and Wardle 2005, Stice, Martinez et al. 2006) but could not be explored in this study as data were not available.

The sample for multivariable analyses was considerably smaller than the sample meeting inclusion criteria (25.5% of the 10,896); however, there was no significant difference in the proportion of participants reporting BCE ($p>0.59$) or for the majority of risk factors between those included and not included in the multivariable regression analyses. For those where there were differences, these were small (1-4%). We therefore have some confidence in the generalizability of the final results to the wider sample, although this may have played a part in the lower number of significant findings in the adjusted multivariable regression compared to the univariable analyses.

The entire cohort was born in April. Some research has suggested a season of birth variation for EDs (Javaras, Austin et al. 2011, Brewerton, Dansky et al. 2012), while others find no association (Winje, Torgalsboen et al. 2013). Where an association has been found, spring has the lowest

relative number of births for all categories, suggesting the BCS70 sample might have a lower chance of reporting BCE than if the births were evenly distributed throughout the year.

Implications

In conclusion, self-reported BCE was independently predicted by female sex, low-self-esteem and high maternal education in multivariable analyses by age 30 in this sample. All other previously identified risk factors for which analysis was possible were not significant in multivariable analysis. Conclusions are tentative and may be more applicable to those with BN than BED, given the likely bias towards help-seeking individuals with compensatory behaviours. A previous study using the BCS70 cohort explored risk factors for AN, and found female sex, infant feeding problems, maternal depressive symptoms and reported history of undereating at ages five and 10 years predicted lifetime self-reported AN at age 30 (Nicholls and Viner 2009), while high self-esteem and higher maternal BMI were protective. Together these studies emphasise that whilst some risk factors are transdiagnostic, there is also differential risk for restrictive versus bulimic and compulsive eating within the ED spectrum.

Despite possible false negatives, which might be a result of low power, this study adds to the growing body of longitudinal research supporting a general psychological, familial and biological risk profile for lifetime BCE. We did not identify any single risk factor (apart from being female) as sufficiently specific to target prevention on the basis of childhood risk factors alone. Even the strong association with female gender clearly does not preclude males being at risk. Future research focussing on the mechanisms precipitating those at high risk into developing EDs is needed.

References

- Allen, K. L., S. M. Byrne, D. Forbes and W. H. Oddy (2009). "Risk factors for full- and partial-syndrome early adolescent eating disorders: a population-based pregnancy cohort study." J Am.Acad.Child Adolesc.Psychiatry **48**(8): 800-809.
- Backholm, K., R. Isomaa and A. Birgegard (2013). "The prevalence and impact of trauma history in eating disorder patients." Eur.J Psychotraumatol. **4**.
- Balodis, I. M., C. M. Grilo and M. N. Potenza (2015). "Neurobiological features of binge eating disorder." CNS Spectr **20**(6): 557-565.
- Brewerton, T. D., B. S. Dansky, P. M. O'Neil and D. G. Kilpatrick (2012). "Seasonal patterns of birth for subjects with bulimia nervosa, binge eating, and purging: results from the National Women's Study." Int.J Eat.Disord. **45**(1): 131-134.
- Chou, K. L. (2012). "Childhood sexual abuse and psychiatric disorders in middle-aged and older adults: evidence from the 2007 Adult Psychiatric Morbidity Survey." J Clin.Psychiatry **73**(11): e1365-e1371.
- Cole, T. J., M. C. Bellizzi, K. M. Flegal and W. H. Dietz (2000). "Establishing a standard definition for child overweight and obesity worldwide: international survey." BMJ **320**(7244): 1240-1243.
- Cole, T. J., J. V. Freeman and M. A. Preece (1995). "Body mass index reference curves for the UK, 1990." Archives of Disease in Childhood **73**(1): 25-29.
- Di Segni, M., E. Patrono, L. Patella, S. Puglisi-Allegra and R. Ventura (2014). "Animal models of compulsive eating behavior." Nutrients **6**(10): 4591-4609.
- Eisenberg, M. E. and D. Neumark-Sztainer (2010). "Friends' dieting and disordered eating behaviors among adolescents five years later: findings from Project EAT." J Adolesc.Health **47**(1): 67-73.
- Fairburn, C. G., S. L. Welch, H. A. Doll, B. A. Davies and M. E. O'Connor (1997). "Risk factors for bulimia nervosa. A community-based case-control study." Arch Gen.Psychiatry **54**(6): 509-517.
- Field, A. E., K. M. Javaras, P. Aneja, N. Kitos, C. A. Camargo, Jr., C. B. Taylor and N. M. Laird (2008). "Family, peer, and media predictors of becoming eating disordered." Arch.Pediatr.Adolesc.Med. **162**(6): 574-579.
- Foley, D. L., L. R. Thacker, S. H. Aggen, M. C. Neale and K. S. Kendler (2001). "Pregnancy and perinatal complications associated with risks for common psychiatric disorders in a population-based sample of female twins." Am.J Med.Genet. **105**(5): 426-431.
- Fursland, A. and H. J. Watson (2014). "Eating disorders: a hidden phenomenon in outpatient mental health?" Int.J Eat.Disord. **47**(4): 422-425.
- Ghaderi, A. (2003). "Structural modeling analysis of prospective risk factors for eating disorder." Eat.Behav. **3**(4): 387-396.
- Haines, J., K. P. Kleinman, S. L. Rifas-Shiman, A. E. Field and S. B. Austin (2010). "Examination of shared risk and protective factors for overweight and disordered eating among adolescents." Arch.Pediatr.Adolesc.Med. **164**(4): 336-343.
- Hart, J. G. (1985). "LAWSEQ: its relation to other measures of self-esteem and academic ability." British Journal of Educational Psychology **55**(2): 167-169.
- Hudson, J. I., E. Hiripi, H. G. Pope, Jr. and R. C. Kessler (2007). "The prevalence and correlates of eating disorders in the national comorbidity survey replication." Biol.Psychiatry **61**(3): 348-358.
- Irving, B. B., C (2002). Predicting adolescent delinquent behaviour and criminal conviction by age 30: Evidence from the 1970 British Birth Cohort London, Police Foundation.

- Jacobi, C. (2005). Psychosocial Risk Factors for Eating Disorders. Eating Disorders Review: Part 1. S. Wonderlich, J. Mitchell, M. de Zwann and H. Steiger. Abingdon, Oxon, Radcliffe Publishing: 59-86.
- Jacobi, C., E. Fittig, S. W. Bryson, D. Wilfley, H. C. Kraemer and C. B. Taylor (2011). "Who is really at risk? Identifying risk factors for subthreshold and full syndrome eating disorders in a high-risk sample." Psychol.Med. **41**(9): 1939-1949.
- Javaras, K. N., S. B. Austin and A. E. Field (2011). "Season of birth and disordered eating in a population-based sample of young U.S. females." Int.J Eat.Disord. **44**(7): 630-638.
- Johnson, F. and J. Wardle (2005). "Dietary restraint, body dissatisfaction, and psychological distress: a prospective analysis." J Abnorm.Psychol. **114**(1): 119-125.
- Kalarchian, M. A. and M. D. Marcus (2012). "Psychiatric comorbidity of childhood obesity." Int.Rev.Psychiatry **24**(3): 241-246.
- Keel, P. K. and K. J. Forney (2013). "Psychosocial risk factors for eating disorders." Int.J Eat.Disord. **46**(5): 433-439.
- Kessler, R. M., P. H. Hutson, B. K. Herman and M. N. Potenza (2016). "The Neurobiological Basis of Binge-Eating Disorder." Neurosci Biobehav Rev.
- Killen, J. D., C. Hayward, D. M. Wilson, C. B. Taylor, L. D. Hammer, I. Litt, B. Simmonds and F. Haydel (1994). "Factors associated with eating disorder symptoms in a community sample of 6th and 7th grade girls." Int.J Eat.Disord. **15**(4): 357-367.
- Killen, J. D., C. B. Taylor, C. Hayward, K. F. Haydel, D. M. Wilson, L. Hammer, H. Kraemer, A. Blair-Greiner and D. Strachowski (1996). "Weight concerns influence the development of eating disorders: a 4-year prospective study." J Consult Clin.Psychol. **64**(5): 936-940.
- Kotler, L. A., P. Cohen, M. Davies, D. S. Pine and B. T. Walsh (2001). "Longitudinal relationships between childhood, adolescent, and adult eating disorders." J.Am.Acad.Child Adolesc.Psychiatry **40**(12): 1434-1440.
- Kraemer, H. C., A. E. Kazdin, D. R. Offord, R. C. Kessler, P. S. Jensen and D. J. Kupfer (1997). "Coming to terms with the terms of risk." Arch Gen.Psychiatry **54**(4): 337-343.
- Krug, I., A. P. Pinheiro, C. Bulik, S. Jimenez-Murcia, R. Granero, E. Penelo, C. Masuet, Z. Aguera and F. Fernandez-Aranda (2009). "Lifetime substance abuse, family history of alcohol abuse/dependence and novelty seeking in eating disorders: comparison study of eating disorder subgroups." Psychiatry Clin.Neurosci. **63**(1): 82-87.
- Krug, I., E. Taborelli, H. Sallis, J. Treasure and N. Micali (2013). "A systematic review of obstetric complications as risk factors for eating disorder and a meta-analysis of delivery method and prematurity." Physiol Behav. **109**: 51-62.
- Lawrence, D. (1981). "The Development of a Self Esteem Questionnaire." British Journal of Educational Psychology **51**(2): 245-251.
- Marchi, M. and P. Cohen (1990). "Early childhood eating behaviors and adolescent eating disorders." J Am Acad Child Adolesc Psychiatry **29**(1): 112-117.
- Micali, N., J. Holliday, A. Karwautz, M. Haidvogel, G. Wagner, F. Fernandez-Aranda, A. Badia, L. Gimenez, R. Solano, M. Brecelj-Anderluh, R. Mohan, D. Collier and J. L. Treasure (2007). "Childhood eating and weight in eating disorders: a multi-centre European study of affected women and their unaffected sisters." Psychother.Psychosom. **76**(4): 234-241.
- Montgomery, S. M., A. Ehlin and A. Ekblom (2005). "Smoking during pregnancy and bulimia nervosa in offspring." J Perinat.Med. **33**(3): 206-211.
- Moorhead, D. J., C. K. Stashwick, H. Z. Reinherz, R. M. Giaconia, R. M. Streigel-Moore and A. D. Paradis (2003). "Child and adolescent predictors for eating disorders in a community population of young adult women." Int.J Eat.Disord. **33**(1): 1-9.
- Nicholls, D. E. and R. M. Viner (2009). "Childhood risk factors for lifetime anorexia nervosa by age 30 years in a national birth cohort." J.Am.Acad.Child Adolesc.Psychiatry **48**(8): 791-799.

- Patton, G. C., R. Selzer, C. Coffey, J. B. Carlin and R. Wolfe (1999). "Onset of adolescent eating disorders: population based cohort study over 3 years." BMJ **318**(7186): 765-768.
- Pike, K. M., D. Wilfley, A. Hilbert, C. G. Fairburn, F. A. Dohm and R. H. Striegel-Moore (2006). "Antecedent life events of binge-eating disorder." Psychiatry Res. **142**(1): 19-29.
- Rodgers, B., A. Pickles, C. Power, S. Collishaw and B. Maughan (1999). "Validity of the Malaise Inventory in general population samples." Soc.Psychiatry Psychiatr.Epidemiol. **34**(6): 333-341.
- Rutter, M. T., J; Whitmore, K (1970). Education, health and behaviour. . London, Longmans.
- Skinner, H. H., J. Haines, S. B. Austin and A. E. Field (2012). "A prospective study of overeating, binge eating, and depressive symptoms among adolescent and young adult women." J Adolesc.Health **50**(5): 478-483.
- Sominsky, L. and S. J. Spencer (2014). "Eating behavior and stress: a pathway to obesity." Front Psychol **5**: 434.
- Sonnevile, K. R., J. P. Calzo, N. J. Horton, A. E. Field, R. D. Crosby, F. Solmi and N. Micali (2015). "Childhood hyperactivity/inattention and eating disturbances predict binge eating in adolescence." Psychol Med **45**(12): 2511-2520.
- Stice, E., E. E. Martinez, K. Presnell and L. M. Groesz (2006). "Relation of successful dietary restriction to change in bulimic symptoms: a prospective study of adolescent girls." Health Psychol. **25**(3): 274-281.
- Stice, E., J. Ng and H. Shaw (2010). "Risk factors and prodromal eating pathology." J Child Psychol.Psychiatry **51**(4): 518-525.
- Striegel-Moore, R. H., F. A. Dohm, H. C. Kraemer, G. B. Schreiber, C. B. Taylor and S. R. Daniels (2007). "Risk factors for binge-eating disorders: an exploratory study." Int.J Eat.Disord. **40**(6): 481-487.
- Striegel-Moore, R. H., C. G. Fairburn, D. E. Wilfley, K. M. Pike, F. A. Dohm and H. C. Kraemer (2005). "Toward an understanding of risk factors for binge-eating disorder in black and white women: a community-based case-control study." Psychol.Med. **35**(6): 907-917.
- The McKnight, I. (2003). "Risk factors for the onset of eating disorders in adolescent girls: results of the McKnight longitudinal risk factor study." Am.J Psychiatry **160**(2): 248-254.
- Trace, S. E., L. M. Thornton, T. L. Root, S. E. Mazzeo, P. Lichtenstein, N. L. Pedersen and C. M. Bulik (2012). "Effects of reducing the frequency and duration criteria for binge eating on lifetime prevalence of bulimia nervosa and binge eating disorder: implications for DSM-5." Int.J Eat.Disord. **45**(4): 531-536.
- Trace, S. E., L. M. Thornton, C. D. Runfola, P. Lichtenstein, N. L. Pedersen and C. M. Bulik (2012). "Sleep problems are associated with binge eating in women." Int.J Eat.Disord. **45**(5): 695-703.
- Vollrath, M., R. Koch and J. Angst (1992). "Binge eating and weight concerns among young adults. Results from the Zurich cohort study." Br.J Psychiatry **160**: 498-503.
- Wehkalampi, K., P. Hovi, S. Strang-Karlsson, K. Raikonen, A. K. Pesonen, K. Heinonen, O. Makitie, A. L. Jarvenpaa, J. G. Eriksson, S. Andersson and E. Kajantie (2010). "Reduced body size and shape-related symptoms in young adults born preterm with very low birth weight: Helsinki study of very low birth weight adults." J Pediatr. **157**(3): 421-427, 427.
- Winje, E., A. K. Torgalsboen, C. Brunborg and B. Lask (2013). "Season of birth bias and bulimia nervosa--results from a multi-centre collaboration." Eur.Eat.Disord.Rev. **21**(2): 170-174.
- Wonderlich, S. A., T. D. Brewerton, Z. Jovic, B. S. Dansky and D. W. Abbott (1997). "Relationship of childhood sexual abuse and eating disorders." J Am.Acad.Child Adolesc Psychiatry **36**(8): 1107-1115.