

Title: Weight trajectories and disordered eating behaviour in 11-12-year-olds: a longitudinal study within the Danish National Birth Cohort

ABSTRACT

Objective: To examine how childhood weight trajectories are associated with disordered eating behaviours (DEBs) in early adolescence.

Methods: Self-reports on DEBs (fasting, purging, and binge eating) were obtained from 18,337 children in the 11-year follow-up of the Danish National Birth Cohort. For this population birth register information on gestational age and birth weight was categorized into: small-, appropriate-, and large-for-gestational age. Prospective parent-reported height and weight data at child ages 1 and 7 years were dichotomized using standardized cut-offs into non-overweight and overweight. A 12-category weight trajectory variable was created, and the associations between weight trajectory and DEBs were estimated using logistic regression.

Results: In total, 7.0% 11-12 year olds reported DEBs. Compared to children born appropriate for gestational age, and being non-overweight at age 1 and 7, children born small-for-gestational age, and who were overweight at age 1 and 7 had a very high risk of disordered eating (OR 7.00; CI: 2.57-19.40). The statistical analyses revealed, however, that overweight at age 7 was the main contributor and independently of trajectory increased the risk of disordered eating at age 11-12 significantly (OR 3.16 (CI: 2.73-3.65)).

Conclusion: Overweight not in the first year of life, but at age 7 was more predictive for DEBs.

Highlights

- There is increasing evidence that DEBs co-exists with overweight, but the temporal relation remains unclear.
- This longitudinal study examined the association of childhood weight trajectories with DEBs within a contemporary cohort of 11-12-year-olds.
- Mid-childhood overweight was significantly associated with DEBs at age 11-12, independently of prior weight trajectories.

Keywords: childhood overweight, eating disorder behaviours, cohort study

INTRODUCTION

Although eating disorders are rare before the age of 12 years (Campbell & Peebles, 2014; Zerwas et al., 2015), symptoms of eating disorders, hereafter referred to as DEBs, often debut during childhood (Evans et al. 2017). Indeed, studies of non-clinical samples of children aged 6-11 years indicate that approximately 9-14% of girls and 5-8% of boys report DEBs (Ricciardelli & McCabe, 2001). In a recent study of 11-12-year-olds, we observed that overweight co-occurred with DEBs (Larsen, Strandberg-Larsen, Olsen, Micali, & Nybo Andersen, 2018). This is in line with increasing evidence showing that childhood overweight predicts later onset of DEBs (Forrester-Knauss, Perren, & Alsaker, 2012; Micali et al., 2015; Munkholm et al., 2016; Neumark-Sztainer, Paxton, Hannan, Haines, & Story, 2006; Neumark-Sztainer et al., 2007; Reed, Micali, Bulik, Smith, & Wade, 2017; Stice, Presnell, & Spangler, 2002; Wiklund et al., 2018). However, not all research concurs; other studies report no association between childhood BMI and disordered eating behaviors in early adolescence (Evans et al. 2017; Gardner, Stark, Friedman, & Jackson, 2000). The majority of research within the field of early-onset DEBs relies on small cross-sectional studies (Larsen, Strandberg-Larsen, Micali, & Nybo Andersen, 2015), and we therefore have little (if any) insight in how trajectories of weight across childhood relate to DEBs in adolescence. As elevated BMI seems to increase the risk of DEBs, it is plausible that timing of onset of obesity in childhood matters. Birth weight is associated with development of obesity (Hong, & Chung, 2018). Both small for gestational age (SGA), typically defined as the smallest 10% of birth weights at any given gestational age, and large for gestational age (LGA) defined as the 10% largest birth weights given gestational age, are correlated with obesity and obesity related morbidities over the life course (Hong, & Chung, 2018). Children who are born SGA tend to gain weight more rapidly during the early postnatal period. Catch-up growth and accelerated postnatal weight gain is also associated with an increased risk of obesity, with central fat deposition, and metabolic diseases later in life

(Barker, 2007; Hong, & Chung, 2018). Our aim was to examine the associations of childhood weight ‘trajectories’, covering weight status at birth, and at age 1 and 7, with DEBs at 11-12 years, as we hypothesized that children born SGA who catch up growth and become overweight in early childhood have increased risks of DEBs. Similarly, we hypothesized that children born LGA and overweight throughout childhood to have increased risk of DEBs.

METHODS

The Danish National Birth Cohort (DNBC) was approved by the Danish National Committee on Health Research Ethics, and from 1996 to 2002, around 100,000 pregnancies were enrolled in the cohort. By signing an informed consent form at the first antenatal care visit in general practice, the women gave consent to participation and to linkage of data to the Danish population registers. Further details about the cohort are available in the cohort description (Olsen et al., 2001) and at www.dnbc.dk.

This study was based on 90,986 children invited for the 11-year follow-up. The population was restricted to children aged 11-12 with complete information on DEBs ($n=46,051$), and on weight status at birth ($n=45,716$), at age 1 ($n=28,154$), and 7 ($n=21,495$). Because the age-span for the 1- and the 7-year weight status measures was wide, we only included children providing information in the age-spans: 10-15 months ($m=12.4$; $SD=0.6$), and 5-8 years ($m=7.0$; $SD=0.3$), respectively. Finally, data were restricted to complete information on the covariates adjusted for ($n=18,377$).

In the 11-year follow-up of the DNBC, information on unhealthy weight control behaviours during the past 12 months was collected by means of a web-based questionnaire to the adolescents using items adapted from the Youth Risk Behaviors Surveillance System survey (Kann et al., 1996).

Furthermore, information on binge eating with loss of control over eating was collected using a two-part question (Field, Taylor, Celio, & Colditz, 2004). Detailed information about the questions and response categories on DEBs used in the 11-year follow-up can be found elsewhere (Larsen et al., 2018). The outcome measure for the present study was the presence or absence of DEBs. DEBs were defined as present if the child reported engaging in either fasting, purging, or binge eating at a monthly basis during the last year. Fasting was defined as not eating anything for a full day in order to control weight, purging was defined by vomiting and/or use of laxative to control weight, and binge eating was defined by loss of control over eating. (Larsen et al., 2018).

From the Danish Medical Birth Registry (MBR, Knudsen & Olsen, 1998), we obtained information on gestational age and birth weight, where the latter was based on a combination of ultrasounds measurements, last menstrual period, and clinical assessments. According to age- and sex-specific intrauterine growth curves (Marsál et al., 1996), we created a categorical variable of weight-for-gestational age categorized into: SGA ($\leq 10^{\text{th}}$ percentile), appropriate-for-gestational age (AGA, $> 10^{\text{th}}$ and $< 90^{\text{th}}$ percentiles), and LGA ($\geq 90^{\text{th}}$ percentile). From the 18-month interview, we obtained parent-reported information on height and weight, as measured by the general practitioner at the 1-year routine health examination. Likewise, in the 7-year follow-up, the parent reported on the child's latest height and weight measures, which have been validated in a sub-sample of the cohort (Andersen, 2012). Based on the weight and height measures, we calculated the child's body mass index (BMI, in kg/m^2), and created two binary variables of the child's weight status (non-overweight, overweight) at age 1 and 7, respectively. The 1-year weight status variable was categorized according to the World Health Organization (WHO) age- and sex-specific growth references, where the cut-off for overweight was defined as ≥ 2 standard deviations (SD) above the mean (WHO, 2006). Weight status at age 7 was categorized according to the International Obesity

Task Force (IOTF) age- and sex-specific curves (Cole, 2000), where the cut-off for overweight was defined as the BMI percentiles corresponding to $BMI \geq 25$ at age 18. The three weight status variables were combined into a 12-category weight trajectory variable (Hallqvist, Lynch, Bartley, Lang, & Blane, 2004), where children being AGA, and non-overweight at both age 1 and 7 were used as reference (Figure 1).

Parental age at birth was obtained from the MBR and categorized into two categorical variables of respectively maternal and paternal age (<25, 25-29, ≥ 30 years). As an indicator of socioeconomic status, we extracted information on parental educational level (highest achieved/ongoing) from the Danish Education Registers (Jensen & Rasmussen, 2011) up to the date of child's BMI measurement at the 7-year follow-up. Using the International Standard Classification of Education categories (primary and lower secondary, upper secondary, lower tertiary, upper tertiary), we created two variables of respectively maternal and parental education level. We used both maternal self-reported eating disorder and maternal hospital-diagnosed eating disorder up to the date of the child's BMI measurement at the 7-year follow-up, as defined elsewhere (Larsen, Nybo Andersen, Olsen, Micali, & Strandberg-Larsen, 2016; Larsen et al., 2018), to create a binary variable of maternal eating disorder (yes, no). We extracted information on parental psychopathology from the Danish National Patient Register (Lyngge, Sandegaard, & Rebolj, 2011) and Psychiatric Central Research Register (Mors, Perto, & Mortensen, 2011) from 1978 up to the date of child's BMI measurement at the 7-year follow-up. Using this information, we created two binary variables of respectively maternal and paternal psychopathology (yes, no, Larsen et al., 2018; Pedersen et al., 2014). Finally, in the 7-year follow-up the parent reported on his/her own height and weight and their partner's height and weight. From this we created two variables for maternal and paternal weight status categorized into: underweight ($BMI < 18.5 \text{ kg/m}^2$), normal weight ($BMI: 18.5-24.9 \text{ kg/m}^2$), and overweight ($BMI > 25 \text{ kg/m}^2$, WHO, 1995).

To examine the association of childhood weight trajectories with DEBs, we used logistic regression and adjusted *a priori* for parental age at birth, educational level, psychopathology, as well as for parental weight status, and maternal eating disorder (Larsen et al., 2018). As the 12-category weight trajectories measure included all interactions between the three weight status measures, we tested whether the effects of the three separate weight status measures could be described in an additive model by using a likelihood ratio test (LRT). We further tested if the additive model could be reduced to include only weight status at age 7, and we tested for interaction with child's sex. Finally, we examined the association between weight status at age 7 and DEBs adjusted for all parental characteristics.

RESULTS

Of the 18,377 children (48.6% boys, 51.4% girls) aged 11-12 years ($M=11.3$ years, $SD=0.5$) included in this study, 1,292 (7.0%) reported any DEBs, and no statistically significant sex difference was observed ($p=0.1973$).

In total, 8.5% ($n=1,553$) and 13.0% ($n=2,387$) were born SGA and LGA respectively, and 13.4% ($n=2,458$) were defined as overweight at age 1, and 8.7% ($n=1,594$) at age 7. When we compared the parental characteristics according to the three weight status measures, we observed that young age of the mother (<25 years) was associated with being SGA at birth and overweight at age 1. Furthermore, lower educational level was associated with being SGA at birth and overweight at age 7. Finally, parental overweight was associated with being LGA at birth, and overweight at age 1 and 7 years (Table 1).

Compared to children born AGA, and who were non-overweight at age 1 and 7, the odds of DEBs was 7.00 (CI: 2.52-19.40) in those born SGA, who were overweight at age 1 and 7 (Table 2). All trajectories that included overweight at age 7 was strongly associated with DEBs, and this finding was supported by results from the additive model, which indicated that overweight at age 7 was associated with DEBs (OR: 3.61, CI: 3.13-4.17; LRT for model reduction gave $p=0.32$), but not weight for gestational age or weight at 1 year. A likelihood-ratio test showed that the model with the 12-category weight trajectory measure did not fit the data significantly better than the additive model ($p=0.31$).

In a final model where we, as consequence of the abovementioned results, only included weight status at age 7 as the independent variable and adjusted for the a priori selected parental characteristics, we found a threefold risk of DEBs in overweight compared to non-overweight 7-year-olds (OR: 3.16, CI: 2.73-3.65). We did not observe any difference in the associations between boys and girls (test for interaction: $p=0.69$).

DISCUSSION

In this large cohort study, including nearly 18,500 11-12-year-olds, DEBs were common. We found that overweight at age 7 was significantly associated with DEBs at age 11-12 independently of prior weight status.

In accordance with previous findings (Ricciardelli & McCabe, 2001), we observed a prevalence of any DEBs of 7.0%, and we did not observe significant sex differences. Also in line with the existing evidence, we observed that childhood overweight was associated with DEBs (Forrester-Knauss et al., 2012; Munkholm et al., 2016; Neumark-Sztainer et al., 2007; Stice et al., 2002). According to

our hypothesis, we expected the risk of DEBs to be higher in children born born SGA, and who were overweight at age 1 and 7 years as compared to the reference. The seven-fold increased risk for DEBs observed for that specific trajectory was, however, based on only six cases and the statistical tests indicated that overweight at age 7 was responsible for the excess risk. We had no information on weight status between age 1 to 7, and it is likely that overweight occurring in this period, as well as after age 7 may increase the risk of DEBs in late childhood/early adolescence. Unfortunately, no prospectively collected weight measures from age 7 to 11 were available, and we have previously shown that overweight at age 11 is associated with higher risk of DEBs (Larsen et al., 2018).

The definition of childhood weight status and cut-offs for overweight vary considerably. Rather than using a uniform measure, we decided to use three different references (Cole, 2000; Marsál et al., 1996; WHO, 2006), that we regarded as the best available standards at each specific age.

Weight status at birth was defined from sex-specific intrauterine growth curves based on ultrasound measures of Danish and Swedish foetuses conceived in the mid-90's (Marsál et al., 1996). We used this reference as: allowance is made for gestational age; and the growth curves are representative of our study population, conceived between 1996 and 2002. Weight status at age 1 was defined using the WHO Child Growth Standards from 2006 available for children less than 2 years of age (WHO, 2006). Finally, weight status at age 7 was defined using the IOTF references that are internationally based and established for 2-18-year-olds (Cole, 2000). As these references are constructed to pass through adult cut-offs, the cut-offs are suggested to be less arbitrary and more intuitive to interpret compared to other cut-offs (Rolland-Cachera, 2011).

Although this study was restricted to approximately 18,500 children with data on weight status and DEBs, the study size and the fact that both childhood weight status and parental characteristics were prospectively collected are major strengths of the study. However, it is well-established that

selection bias is of concern in longitudinal studies with implication for distortion of the findings. Previously, it has been shown that child psychopathology are among the factors associated with loss to follow-up (Dreier et al., 2017). Moreover, as compared to the source population, the prevalence of mid-childhood overweight is found to be lower among children enrolled in the DNBC (Pearson, Hansen, Sørensen, & Baker, 2010). Thus, in the present study representing the healthiest and least vulnerable children, the association between overweight and DEBs is likely attenuated due to these selection mechanisms (Pizzi et al., 2011).

The outcome of interest in this study, DEBs, was based on self-report. We were specifically concerned that binge eating was over-reported, as children may have difficulties in understanding the concept of binge eating (Shapiro et al., 2007). To address this concern, we conducted a number of sensitivity analyses, where we excluded binge eating from the outcome definition. The results of these analyses were essentially the same as those reported here. The height and weight measures at birth and 1 year were reported by health professionals, however, the heights and weights at age 7 were parent-reported. We have no reason to believe that the parental misclassification of growth measures at age 7 should be strongly related to the children-reported DEBs, but such misclassification cannot be ruled out.

Due to the low number of cases, we were unable to study single eating behaviours, but findings from a recent study within the DNBC showed co-occurrence of fasting, purging, and binge eating, and therefore we found it comprehensive to combine the outcomes in an overall measure of any DEBs (Larsen et al., 2018). However, it could be argued that our measure reflects dieting behaviours as a response to being overweight, but due to the wording of the eating behaviour questions, we believe that our measure captures something other than just dieting, as fasting for a whole day to control weight, and vomiting and use of laxatives are considered to be severe outcomes.

In conclusion, DEBs are common among 11-12-year-olds, and strongly associated with mid-childhood overweight, while intrauterine growth measures and weight status in infancy seemed to be unrelated to DEBs at age 11-12. If the findings are causal, prevention of childhood overweight may also prevent DEBs in adolescence.

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Table 1. Distributions of parental and child characteristics according to weight status of the child at birth, age 1 and 7, respectively. The Danish National Birth Cohort (n=18,377).

	<i>Total</i>		<i>Weight status at birth</i>				<i>Weight status at age 1</i>			<i>Weight status at age 7</i>		
			<i>SGA</i> <i>(n=1,553)</i>	<i>AGA</i> <i>(n=14,437)</i>	<i>LGA</i> <i>(n=2,387)</i>	<i>X²</i>	<i>Non-overweight</i> <i>(n=15,919)</i>	<i>Overweight</i> <i>(n=2,458)</i>	<i>X²</i>	<i>Non-overweight</i> <i>(n=16,783)</i>	<i>Overweight</i> <i>(n=1,594)</i>	<i>X²</i>
	<i>N</i>	<i>%</i>	<i>%</i>	<i>%</i>	<i>%</i>	<i>p-value</i>	<i>%</i>	<i>%</i>	<i>p-value</i>	<i>%</i>	<i>%</i>	<i>p-value</i>
<i>Maternal age at birth (years)</i>						<0.0001			0.0213			0.2556
<25	992	5.4	6.8	5.5	3.9		5.2	6.5		5.4	5.9	
25-29	6900	37.4	41.2	38.2	31.3		37.4	38.2		37.6	36.6	
≥30	10485	57.1	52.0	56.3	64.9		57.3	55.3		57.0	57.5	
<i>Paternal age at birth (years)</i>						<0.0001			0.2599			0.2280
<25	460	2.5	3.5	2.5	1.9		2.4	3.0		2.4	3.1	
25-29	4652	25.3	28.8	25.8	20.3		25.3	25.5		25.4	24.5	
≥30	13265	72.2	67.7	71.7	77.8		72.3	71.5		72.2	72.4	
<i>Maternal eating disorder</i>						0.4183			0.9958			0.6844
No	17433	94.9	94.2	95.0	94.7		94.9	94.8		94.9	94.6	
Yes	944	5.1	5.8	5.0	5.3		5.1	5.2		5.1	5.4	
<i>Maternal psychopathology</i>						0.0219			0.0219			0.2013
No	17624	95.9	95.6	96.1	94.9		96.0	95.1		96.0	95.4	
Yes	753	4.1	4.4	3.9	5.1		4.0	4.9		4.09	4.6	
<i>Paternal psychopathology</i>						0.0878			0.3739			0.0068
No	17955	97.7	97.0	97.7	98.1		97.7	98.0		97.8	97.2	
Yes	422	2.3	3.0	2.3	1.9		2.3	2.0		2.2	2.8	

Table 1. Distributions of parental and child characteristics according to weight status of the child at birth, age 1 and 7. The Danish National Birth Cohort (n=18,377). Cont'n.

		<i>Weight status at birth</i>				<i>Weight status at age 1</i>			<i>Weight status at age 7</i>		
		<i>SGA (n=1,553)</i>	<i>AGA (n=14,437)</i>	<i>LGA (n=2,387)</i>	<i>χ²</i>	<i>Non- overweight (n=15,919)</i>	<i>Overweight (n=2,458)</i>	<i>χ²</i>	<i>Non- overweight (n=16,783)</i>	<i>Overweight (n=1,594)</i>	<i>χ²</i>
<i>Total</i>											
<i>N</i>	<i>%</i>	<i>%</i>	<i>%</i>	<i>%</i>	<i>p-value</i>	<i>%</i>	<i>%</i>	<i>p-value</i>	<i>%</i>	<i>%</i>	<i>p-value</i>
Maternal educational level					0.0001			0.3022			<0.0001
<i>Primary and lower secondary</i>	896 4.9	6.6	4.8	4.1		5.0	4.3		4.6	7.5	
<i>Upper secondary</i>	6738 36.7	39.8	36.4	36.2		36.4	38.2		36.1	42.1	
<i>Lower tertiary</i>	7948 43.2	40.1	43.2	45.4		43.3	42.8		43.5	40.3	
<i>Upper tertiary</i>	2795 15.2	13.6	15.5	14.3		15.3	14.7		15.7	10.0	
Paternal educational level					<0.0001			0.7346			<0.0001
<i>Primary and lower secondary</i>	1865 10.1	12.4	9.9	9.9		10.0	10.9		9.9	13.1	
<i>Upper secondary</i>	8293 45.1	46.9	44.9	45.2		45.2	44.4		44.5	51.6	
<i>Lower tertiary</i>	5126 27.9	27.0	27.9	28.7		27.9	28.2		28.3	23.7	
<i>Upper tertiary</i>	3093 16.8	13.8	17.3	16.2		16.9	16.6		17.3	11.6	
Maternal weight status (BMI)					<0.0001			<0.0001			<0.0001
<i><18.5</i>	373 2.0	3.7	2.1	0.5		2.1	1.3		2.2	0.1	
<i>18.5-24.9</i>	12041 65.5	67.5	66.6	57.9		66.3	60.2		67.3	47.2	
<i>>25.0</i>	5963 32.4	28.8	31.3	41.6		31.5	38.5		30.5	52.7	
Paternal weight status (BMI)					0.0004			<0.0001			<0.0001
<i><18.5</i>	43 0.2	0.4	0.2	0.4		0.3	0.1		0.3	0.1	

18.5-24.9	8337	45.4	46.2	45.9	41.6	45.9	41.6	47.1	27.6
>25.0	9997	54.4	53.4	53.9	58.0	53.8	58.3	52.7	72.3

Table 2. Odds ratios (OR) for disordered eating according to childhood weight trajectories. The Danish National Birth Cohort (n=18,377).

	<i>Total</i>		<i>Any disordered eating (n=1,292)</i>			
	<i>N</i>	<i>n</i>	<i>Crude OR</i>	<i>(95% CI)</i>	<i>Adjusted OR*</i>	<i>(95% CI)</i>
<i>Weight trajectories*</i>						
<i>SGA, non-overweight 1y, non-overweight 7y</i>	1351	85	1.09	(0.87-1.38)	1.07	(0.85-1.35)
<i>SGA, non-overweight 1y, overweight 7y</i>	63	11	3.44	(1.79-6.63)	2.97	(1.53-5.67)
<i>SGA, overweight 1y, non-overweight 7y</i>	123	11	1.60	(0.86-2.98)	1.57	(0.84-2.94)
<i>SGA, overweight 1y, overweight 7y</i>	16	6	9.76	(3.54-26.90)	7.00	(2.52-19.40)
<i>AGA, non-overweight 1y, non-overweight 7y</i>	11728	679	1.00	(ref.)	1.00	(ref.)
<i>AGA, non-overweight 1y, overweight 7y</i>	869	155	3.53	(2.92-4.27)	3.05	(2.51-3.70)
<i>AGA, overweight 1y, non-overweight 7y</i>	1509	98	1.13	(0.91-1.41)	1.10	(0.88-1.37)
<i>AGA, overweight 1y, overweight 7y</i>	331	59	3.53	(2.64-4.73)	3.00	(2.23-4.03)
<i>LGA, non-overweight 1y, non-overweight 7y</i>	1710	94	0.95	(0.76-1.18)	0.91	(0.73-1.14)
<i>LGA, non-overweight 1y, overweight 7y</i>	198	47	5.06	(3.62-7.09)	4.25	(3.02-5.97)
<i>LGA, overweight 1y, non-overweight 7y</i>	362	26	1.26	(0.84-1.89)	1.24	(0.83-1.87)
<i>LGA, overweight 1y, overweight 7y</i>	117	21	3.56	(2.21-5.74)	2.98	(1.84-4.82)

Adjusted for parental age, psychopathology, educational level, weight status, and maternal eating disorder, obese children were included in the overweight category,

**SGA: small-for-gestational age*

AGA: appropriate-for-gestational age

LGA: large-for-gestational age

y: year

Figure 1. Illustration of the 12-category childhood weight trajectory measure, including all possible combinations