Pregnancy loss of control over eating: a longitudinal study of maternal and child outcomes

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ABSTRACT

Background: To our knowledge, no previous studies have investigated longitudinal outcomes of maternal loss of control over eating (LOC) in pregnancy in a general population sample.

Objective: We aimed to determine whether pregnancy LOC is associated with dietary, gestational weight gain, and offspring birthweight outcomes in a large population-based prospective study of pregnant women and their children. We also explored the association with offspring weight at age 15.5 y.

Design: Women ($n = 11{,}132$) from the Avon Longitudinal Study of Parents and Children (ALSPAC) were included. Crude and adjusted logistic and multinomial regression models were used. LOC in pregnancy and diet at 32 wk of gestation were assessed by self-report. Pregnancy weight gain and birth weight were obtained from obstetric records. Child weight and height were objectively measured at age 15.5 y.

Results: LOC in pregnancy was common (36.3%). Women with pregnancy LOC reported higher total energy intake, consumed more snacks, and had lower vitamin B-6, A, and C intake compared with women without LOC. Women with frequent LOC had lower vitamin B-1 and folate intake [respectively: b = −0.05 (95% CI: −0.07, −0.02) and b = −7.1 (95% CI: −11.8, −2.3) in adjusted analyses], and gained on average 3.74 kg (95% CI: 3.33, 4.13 kg) more than women without LOC. Frequent and occasional LOC were associated with higher birth weight [respectively: b = 0.07 (95% CI: 0.03, 0.1), b = 0.04 (95% CI: 0.02, 0.06)]. Offspring of mothers with frequent pregnancy LOC had 2-fold increased odds of being overweight/obese at 15.5 y [OR = 2.02 (95% CI: 1.37, 3.01)].

Conclusions: Pregnancy LOC eating is common and has an adverse short- and long-term impact on mother and offspring, but has received very limited attention. Our findings further the understanding of risk factors for obesity and highlight a need for improved identification of maternal pregnancy loss of control eating. This trial was registered at clinicaltrials.gov as NCT03269253. *Am J Clin Nutr* 2018;108:101–107.

Keywords: pregnancy, eating, loss of control, ALSPAC, adverse outcomes

INTRODUCTION

Maternal eating and lifestyle patterns in pregnancy have an important influence on both mother and child obesity and metabolic outcomes (1–3). Maternal diet has been shown to affect child adiposity, growth, and body composition, and might program child intake and appetite (4–6).

There is good evidence that excess weight and gestational weight gain (GWG) during pregnancy have short-term and long-lasting effects on child and maternal physical and psychological health, including gestational diabetes, heart disease, offspring obesity, and metabolic abnormalities (7–9). Although maternal overweight and obesity have received much attention, disordered eating has been less studied. Loss of control over eating (LOC) is the subjective experience of feeling out of control while eating, irrespective of the amount consumed, and it is a

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Supplemental Table 1 is available from the "Supplementary data" link in the online posting of the article and from the same link in the online table of contents at https://academic.oup.com/ajcn.

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Abbreviations used: ALSPAC, Avon Longitudinal Study of Parents and Children; BED, binge eating disorder; FFQ, Food-Frequency Questionnaire; GWG, gestational weight gain; IOM, Institute of Medicine; LOC, loss of control over eating.

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characteristic feature of binge eating. LOC affects 9–30% of individuals in the community (10, 11) and is associated with higher BMI, disordered eating, and psychiatric symptoms (12, 13). Amongst adults from community and clinical samples, the experience of LOC has been shown to be a better predictor of distress and disability, psychopathology, and impairment in psychosocial functioning than the amount of food consumed during a LOC or a binge eating episode (14, 15). Two studies (1 in Brazil and 1 in Canada) have investigated binge eating (LOC that results in abnormally large intake) in pregnancy and observed that it is positively associated with GWG (16) and macrosomia (17). A recent study of 200 overweight and obese women found a high prevalence of LOC during pregnancy, and an association between engaging in LOC, stress, and depression (18). Despite LOC being common, to our knowledge no previous studies have investigated LOC in pregnancy in a population sample across the weight range, its dietary correlates, and its effects on GWG and offspring weight. The aim of our study was to investigate the relations between LOC during pregnancy, dietary intake (particularly macronutrient and vitamin intake) and patterns, GWG, weight at 8 wk postpartum, offspring birth weight, and adolescent weight in a large population-based birth cohort study.

METHODS

Participants and procedures

The Avon Longitudinal Study of Parents and Children (ALSPAC) is a longitudinal, prospective study designed to examine the effects of environment, genetics, and other factors on health and development (7). All pregnant women living in the geographic area of Avon, UK, who were expected to deliver their infant between 1 April 1991 and 31 December 1992, were recruited. 14,541 women were enrolled. Amongst these pregnancies, there were a total of 14,676 fetuses, resulting in 14,062 live births and 13,798 children who were alive at 1 y of age and were singletons. The ALSPAC study website contains details of all the data that are available through a fully searchable data dictionary (http://www.bris.ac.uk/alspac/researchers/data-access/data-dictionary/).

Women were eligible to be included in the current study if they had completed the questionnaire at 32 wk of gestation including questions on the exposure ($n = 11{,}132$). This trial was registered at clinicaltrials.gov as NCT03269253.

Measures

Exposure

At 32 wk of gestation women were asked to report whether they had experienced any LOC during the current pregnancy ("Have you experienced a loss of control over eating during this pregnancy?"). Answers were coded on a 3-point Likert scale (Not at all, Yes occasionally, Yes most of the time). Hence we categorized this variable as frequent LOC, occasional LOC, and no LOC. Data on LOC in pregnancy were available on 11,132 women (91.4%).

Women were also asked whether they had dieted during the current pregnancy and whether they were dissatisfied with their shape.

Diet during pregnancy. A food frequency questionnaire (FFQ) was sent to women at 32 wk of gestation, enquiring about the frequency of consumption of a wide variety of foods and drinks [for details about the questionnaire, and its validation, see Micali et al. (19) and Northstone et al. (20)].

Dietary patterns during pregnancy were identified with the use of principal components analysis. This is described in detail in Northstone et al. (20). Each score had a mean of 0 and a higher score indicated closer adherence to that dietary pattern. Five components were identified: "health conscious," "traditional," "processed," "snacking," and "vegetarian."

Daily nutrient intakes were estimated from the FFQ with the use of the 5th edition of McCance and Widdowson's *The Composition of Food* and its supplements, based on standard portion sizes; detailed information on the methodology is published elsewhere (21, 22). Previous analysis of these data showed this questionnaire to produce mean nutrient intakes similar to those obtained for women in the British National Diet and Nutritional Survey for adults (23).

Outcomes

Pregnancy weight gain and postpartum weight. Net weight gain in pregnancy was derived from obstetric medical records by subtracting the first from the last weight measurement in pregnancy to derive total weight gain during the pregnancy (24).

The Institute of Medicine (IOM) 2009 recommendations were used to ascertain the adequacy of GWG obtained from obstetric records, given pre-pregnancy BMI, to derive 3 categories: weight gain as recommended, lower weight gain than recommended, and higher weight gain than recommended [for a detailed description of these measures see Fraser et al. (24)].

Postpartum weight was obtained via questionnaire at 8 wk postpartum.

Offspring birth weight. Birth weight was abstracted from obstetric records. Fifty-nine ($n = 59$) women with gestational diabetes were excluded from these analyses; the prevalence of gestational diabetes did not vary across those exposed and unexposed (those with and without LOC).

Offspring adiposity at 15.5 y of age. Children still enrolled in the study were measured at the ALSPAC base at mean age 15.5 y ($n = 5515$). Height was measured in standing position with the use of a Harpenden stadiometer and weight was measured in light clothing on a mechanical scale. BMI was obtained as objective kg/m^2. Age- and gender-adjusted BMI z scores (with the use of UK references) (25, 26) were obtained from the Stata user-defined program "Z-anthro". Age- and gender-adjusted cutoffs for adolescents (from the International Obesity Task Force) (25) were used to define the overweight and obese categories.

Covariates

Sociodemographic (maternal education, parity, and maternal age), weight, and height data were obtained by self-completion questionnaires at 12 and 18 wk of gestation. BMI was calculated as pre-pregnant weight/height squared (kg/m^2).

Attrition

Complete data on pregnancy nutrient intake and pregnancy LOC were available on 9903 women (89%); data on dietary patterns and pregnancy LOC on 9911 women (89%) (see

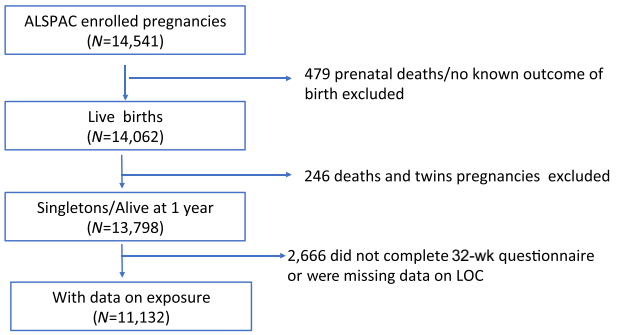

FIGURE 1 Flowchart of participating women. ALSPAC, Avon Longitudinal Study of Parents and Children; LOC, loss of control over eating.

Figure 1 for a flowchart). Data on pregnancy weight gain and adequacy of weight gain were available on 10,088 (90.6%) and 9500 (85.3%) women, respectively. Data on birth weight were available on 10,988 women (98.8%). Data on weight at 8 wk postnatal were available on 8530 women (76.6%).

A total of 1205 (10.8%) women had missing data on all covariates, missingness on covariates was not predicted by maternal exposure.

Women who reported frequent LOC in pregnancy were more likely to have missing data on absolute weight gain in pregnancy (13.1% compared with 9.2% in the no LOC group, $P = 0.007$) and adequacy of pregnancy weight gain (18.0% compared with 14.7% in the no LOC group, $P = 0.04$). LOC did not predict missingness on birth weight or weight at 8 wk postnatal.

Statistical analyses

The distribution of covariates according to exposure was assessed with the use of the chi-square test or F test depending on the variable type. Means and SDs were estimated for all continuous variables, after checking for normality. Crude analyses were carried out through the use of logistic regression for binary outcomes, multinomial logistic regression for categorical outcomes, and linear models for continuous outcomes. Normality assumptions were checked before linear regression models were used.

Multivariable adjusted analyses on maternal diet in pregnancy controlled for a priori confounders: maternal age, parity, education, and maternal BMI pre-pregnancy; analyses of macronutrient and vitamin intake were also adjusted for total energy intake, which is necessary because energy is highly correlated with most macro- and micronutrient intakes and individual body size. Multivariable adjusted analyses on pregnancy weight gain and postpartum weight were additionally adjusted for gestational age; child birth weight was additionally adjusted for child sex (as an a priori confounder). Analyses of child BMI at 15.5 y of age additionally adjusted for birth weight.

Analyses were carried out on women who had complete data on outcomes and the exposure; given that data for this study were collected at various time-points, complete data on exposure and outcome varied across the outcomes studied.

Due to missing data on maternal education, parity, and ethnicity, multiple imputation by chained equation with 10 imputation sets was implemented in Stata 14 (Stata Corp., 2014) assuming missing at random (27). All predictors and outcome variables were used in the imputation model. Results obtained from imputation models were not different to those found when analyzing complete records only; therefore results obtained from multiple imputation models are reported throughout.

All analyses were carried out in Stata 14. All statistical tests presented are 2-sided, with $P < 0.05$ used to define significance.

Ethical approval

The study was approved by the ALSPAC Ethics and Law committee and the Local Research Ethics Committees. All women gave informed consent at enrollment in the study.

RESULTS

Sociodemographic data

Overall, 5.2% ($n = 582$) of women reported frequent LOC in pregnancy, 31.1% ($n = 3466$) occasional pregnancy LOC. Women with LOC did not differ from those with no LOC in relation to ethnicity, or age at delivery. Women with frequent LOC in pregnancy were more likely to be multiparous (58.1% compared with 53.5%) and less likely to be educated to secondary education level (24.9% compared with 37.6%); their BMI pre-pregnancy was on average ~1 unit higher compared with women with no LOC (see **Table 1**).

Women with frequent and occasional LOC in pregnancy had higher prevalence of dieting in pregnancy and being dissatisfied with their shape compared with those with no LOC (Table 1).

Pregnancy diet

Women with frequent and occasional LOC in pregnancy reported higher total energy [respectively, mean: 7786.6 kJ (SD = 2212), 7428.9 kJ (SD = 1970)], carbohydrate intake, and fat intake compared with women with no LOC in crude analyses [mean energy intake: 7283.7 kJ (SD = 1954)] (**Table 2**). After adjusting for all covariates (maternal age, BMI pre-pregnancy, parity, maternal education, ethnicity, and total energy intake) the association between frequent LOC in pregnancy and a higher energy and carbohydrate intake and lower protein intake persisted. Women with occasional LOC only differed from women who did not engage in LOC in relation to their total energy intake (higher).

Dietary patterns in pregnancy also differed in women with LOC in pregnancy (across both frequency levels) compared with those without; in adjusted analyses women with LOC in pregnancy had higher scores on the "processed foods" and "snacking" dietary pattern, and lower scores on the "traditional" dietary pattern. Women with occasional LOC scored higher than controls on the "snacking" dietary pattern in adjusted analyses (Table 2).

Differences were identified amongst women with occasional and frequent LOC in relation to their vitamin intake (**Supplemental Table 1**); overall, women with LOC had lower vitamin intake in pregnancy. Pregnancy intake of folate, vitamin C, and thiamin (vitamin B-1) was particularly low amongst women who reported frequent LOC in pregnancy [folate: −7.1 (−11.8, −2.3), $P < 0.001$; vitamin C: −7.1 (−10.0, −4.2), $P < 0.0001$; vitamin B-1: −0.05 (−0.07, −0.02, $P < 0.0001$)]. Pregnancy intake of pyridoxine, vitamin A, and vitamin D was also lower in women with frequent pregnancy LOC compared with those with no LOC. Women with occasional LOC in pregnancy had lower pyridoxine, vitamin A, and vitamin C intake compared with those without LOC (Supplemental Table 1).

TABLE 1
Sociodemographic data and correlates of LOC in pregnancy for 11,132 women from ALSPAC[1]

	LOC in pregnancy		
	Frequent 5.2% ($n = 582$)	Occasional 31.1% ($n = 3466$)	None (Ref.) 63.6% ($n = 7084$)
Age at delivery, y	27.2 ± 5.2	28.2 ± 4.8	28.5 ± 4.8
BMI pre-pregnancy, kg/m^2	23.9 ± 4.1	23.0 ± 3.6	22.8 ± 3.9
Missing, *n* (%)	43 (7.4)	218 (6.3)	517 (7.3)
Parity (multiparous), *n* (%)	338 (58.1)	1850 (53.4)	3790 (53.5)
Missing	18 (3.0)	96 (2.8)	191 (2.7)
Ethnicity (White), *n* (%)	557 (95.7)	3368 (97.2)	6841 (96.6)
Missing	6 (1.0)	33 (0.9)	75 (1.1)
Education (A-levels or higher vs. up to O-levels), *n* (%)	145 (24.9)	1237 (35.7)	2667 (37.6)
Missing	5 (0.9)	15 (0.4)	42 (0.6)
Any dieting in pregnancy, *n* (%)	46 (7.9)	109 (3.1)	158 (2.2)
Missing	0	19 (0.6)	26 (0.4)
Dissatisfied with shape, *n* (%)	518 (89.0)	2259 (65.2)	3299 (46.6)
Missing	0	8 (0.2)	18 (0.2)

[1] Values are means ± SDs or *n* (%). ALSPAC, Avon Longitudinal Study of Parents and Children; LOC, loss of control over eating; Ref., reference.

Pregnancy weight gain, birth weight, and postpartum weight

Women with frequent LOC in pregnancy had higher GWG, with a mean difference of 3.74 kg in absolute weight gain, compared with women with no LOC in adjusted analyses. They also had 3-fold increased odds of gaining more weight than recommended by the IOM during gestation [adjusted OR = 3.41 (2.73, 4.27)] (see **Table 3**). Women with occasional LOC in pregnancy had intermediate (albeit significantly higher compared with controls) levels of absolute weight gain in pregnancy, and 1.7 higher odds [OR = 1.66 (1.58, 1.96)] of gaining more weight than recommended by the IOM in adjusted analyses (Table 3). Women's weight at 8 wk postpartum was higher in women with

TABLE 2
Maternal dietary intake in pregnancy for women from the ALSPAC cohort: mean differences and 95% CIs from unadjusted and adjusted linear regression[1]

		LOC in pregnancy[2]			LOC in pregnancy, unadjusted mean difference (95% CI)			LOC in pregnancy, adjusted mean difference (95% CI)	
	N	Frequent $n = 473$	Occasional $n = 3055$	None $n = 6375$	Frequent $n = 473$	Occasional $n = 3055$	None $n = 6375$	Frequent $n = 473$	Occasional $n = 3055$
Energy and macronutrients	9903								
Total energy, kJ	9903	7786.6 ± 2212	7428.9 ± 1971	7283.7 ± 1954	502.9 (318.6, 687.1)***	145.2 (60.1, 230.3)**	Ref.	550.6 (367.9, 733.3)***	147.9 (63.8, 232.0)**
Carbohydrates, g	9903	235.8 ± 71.1	223.2 ± 61.3	218.8 ± 62	16.9 (11.1, 22.8)***	4.3 (1.6, 7.0)**	Ref.	2.00 (0.1, 4.1)*	−0.1 (−1.0, 1.0)
Fat, g	9903	76.2 ± 25.2	72.0 ± 23.2	70.3 ± 22.4	5.8 (3.7, 7.9)***	1.7 (0.7, 2.6)**	Ref.	0.1 (−0.7, 0.9)	0.05 (−0.3, 0.4)
Protein, g	9903	70.3 ± 20.3	70.2 ± 18.9	69.3 ± 19	0.9 (−0.9, 2.7)	0.8 (0.1, 1.7)*	Ref.	−2.1 (−3.1, −1.1)***	−0.1 (−0.6, 0.3)
Dietary patterns	11,102								
Health-conscious	11,102	−0.12 ± 0.9	0.06 ± 0.9	0.09 ± 0.1	−0.2 (−0.3, −0.1)***	−0.03 (−0.1, 0.01)	Ref.	−0.04 (−0.1, 0.03)	0.01 (−0.03, 0.04)
Processed	11,102	0.23 ± 1.1	0.01 ± 0.9	0.01 ± 0.9	0.2 (0.1, 0.3)***	−0.1 (−0.05, 0.03)	Ref.	0.08 (0.01, 0.2)*	−0.03 (−0.1, 0.01)
Traditional British	11,102	−0.1 ± 1.0	0.03 ± 0.9	0.04 ± 0.1	−0.1 (−0.2, −0.02)*	−0.005 (−0.04, 0.03)	Ref.	−0.1 (−0.2, −0.02)*	−0.003 (−0.04, 0.04)
Snacking (confectionery)	11,102	0.4 ± 1.3	0.1 ± 1.0	−0.05 ± 0.9	0.5 (0.4, 0.6)***	0.2 (0.1, 0.2)***	Ref.	0.5 (0.4, 0.6)***	0.2 (0.1, 0.3)***

[1] Adjusted for maternal age, BMI pre-pregnancy, parity, maternal education, and ethnicity. Total carbohydrate, fat, and protein intakes are additionally adjusted for total energy intake. *P* values for comparisons between index groups and controls (no LOC): $*P \leq 0.05$, $**P \leq 0.01$, $***P \leq 0.001$. ALSPAC, Avon Longitudinal Study of Parents and Children; LOC, loss of control over eating; Ref., reference.

[2] Values are means ± SDs.

TABLE 3
Pregnancy weight gain and offspring birth weight: mean differences and odds ratios (95% CIs) from unadjusted and adjusted linear and logistic regression[1]

	N	LOC in pregnancy			LOC in pregnancy (unadjusted)			LOC in pregnancy (adjusted)	
Absolute weight gain in pregnancy, kg	10,088	Frequent ($n = 506$) 15.5 ± 5.8	Occasional ($n = 3147$) 13.6 ± 4.5	None ($n = 6435$) 11.8 ± 1.4	Frequent ($n = 506$) 3.63 (3.19, 4.07)***	Occasional ($n = 3147$) 1.82 (1.62, 2.02)***	None ($n = 6435$) Ref	Frequent ($n = 506$) 3.74 (3.33, 4.13)***	Occasional ($n = 3147$) 1.77 (1.58, 1.96)***
GWG (more than recommended vs. recommended), OR	9500	Frequent ($n = 477$) —	Occasional ($n = 2979$) —	None ($n = 6004$) —	Frequent ($n = 477$) 3.59 (2.85, 4.52)***	Occasional ($n = 2979$) 1.56 (1.39, 1.75)***	None ($n = 6004$) 1.0	Frequent ($n = 477$) 3.41 (2.73, 4.27)***	Occasional ($n = 2979$) 1.66 (1.48, 1.85)***
Weight at 8 wk postpartum, kg	8530	Frequent ($n = 441$) 70.1 ± 12.3	Occasional ($n = 2687$) 66.3 ± 10.3	None ($n = 5402$) 64.3 ± 10.6	Frequent ($n = 441$) 5.82 (4.74, 6.89)***	Occasional ($n = 2687$) 2.00 (1.49, 2.51)***	None ($n = 5402$) Ref	Frequent ($n = 441$) 3.92 (3.18, 4.66)***	Occasional ($n = 2687$) 1.60 (1.24, 1.95)***
Birth weight,[2] kg	10,988	Frequent ($n = 573$) 3.50 ± 0.5	Occasional ($n = 3419$) 3.47 ± 0.5	None ($n = 7006$) 3.41 ± 0.5	Frequent ($n = 573$) 0.09 (0.04, 0.1)***	Occasional ($n = 3419$) 0.05 (0.03, 0.07)***	None ($n = 7006$) Ref	Frequent ($n = 573$) 0.07 (0.03, 0.1)***	Occasional ($n = 3419$) 0.04 (0.02, 0.06)***

[1] Values are means ± SDs or ORs (95% CIs). Adjusted for maternal age, BMI pre-pregnancy, parity, maternal education, and length of gestation. *P* values for comparisons between index groups and controls: *$P < 1.05$, **$P < 0.001$, ***$P \leq 0.0001$. GWG, gestational weight gain; LOC, loss of control over eating; Ref, reference.

[2] Additionally adjusted for offspring gender.

pregnancy LOC compared with those without (by about 4 kg amongst women with frequent and 1.6 kg amongst those with occasional pregnancy LOC in adjusted analyses). Child birth weight was higher in offspring of women with both frequent [mean difference = 0.07 kg (0.03, 0.1)] and occasional LOC in pregnancy [mean difference = 0.04 kg (0.02, 0.06)] compared with offspring of women without pregnancy LOC in adjusted analyses (see Table 3).

Offspring weight at 15.5 y

In exploratory analyses we investigated associations between LOC during pregnancy and offspring weight at age 15 y. Complete data on the outcome and exposure were available on 3779 children. Children of mothers with frequent pregnancy LOC were more likely to be overweight or obese compared with children of mothers with no pregnancy LOC [OR = 2.02 (1.37, 3.01), $P < 0.0001$]; whereas children of mothers with occasional pregnancy LOC did not differ from children of women with no pregnancy LOC in terms of weight status. We stratified for child sex, but results did not differ within strata (data not shown), therefore sex was included as a covariate in these analyses. In sensitivity analyses performed by restricting the analyses to women who were not obese pre-pregnancy and their children ($n = 3482$), the effect of maternal frequent pregnancy LOC persisted with similar magnitude [OR = 1.92 (1.25, 2.94), $P = 0.003$].

DISCUSSION

This is the first study to our knowledge to investigate short- and long-term effects of maternal LOC during pregnancy on maternal (diet, pregnancy weight gain) and child (birth weight and weight at age 15.5 y) outcomes. LOC is relatively common in pregnancy, as shown in this large population-based study and in a smaller study of overweight/obese women (18); however, no previous studies to our knowledge have investigated dietary and weight outcomes of pregnancy LOC, nor offspring outcomes. Women with LOC in pregnancy had lower educational attainment and were more likely to have had >1 pregnancy compared with women with no LOC in this study, suggesting that these factors might be associated with LOC in pregnancy.

There is evidence that LOC (outside of pregnancy) is associated with higher calorie intake from carbohydrates, lower protein intake, and more snacks and dessert foods (28, 29). LOC is also prospectively associated with overweight and obesity in youth (30); and authors have argued its pivotal role as a behavioral marker of adverse outcomes both with (i.e., in the context of binge eating) and without objective overeating (30, 31). We observed that maternal pregnancy LOC was associated with diet in late pregnancy, maternal pregnancy weight gain, and child weight both at birth and long-term. In particular, women with pregnancy LOC reported a higher total energy and carbohydrate intake, lower protein intake, and lower intake of vitamins A, B-6, and C even after adjusting for total energy intake. Their diet was also characterized by higher "snack" scores, i.e., high intake of foods with added sugars such as chocolate, candy, and cakes (19, 20). These findings complement findings of higher total energy intakes, and lower folate and vitamin C intakes, from a large Norwegian population-based study on pregnancy diet in women with binge eating disorder (BED) (32). Higher maternal energy and carbohydrate intake may be risk factors for greater child adiposity and fat mass, and higher GWG (4, 33). It has been suggested that metabolic and appetitive fetal programming might also be affected by imbalanced maternal intake in pregnancy (4). In rodents, high-sugar diets in pregnancy have been shown to lead to altered metabolic and inflammatory pathways and higher oxidative stress in the offspring (34–36). Low intake of vitamins (particularly C and E) in pregnancy is likely to contribute to altered

fetal programming and adverse perinatal outcomes via oxidative stress (37). Low folate, on the other hand, might contribute to altered DNA-methylation.

Maternal pregnancy LOC was prospectively associated with higher absolute weight gain in pregnancy, gaining more weight than recommended by the IOM, and higher weight at 8 wk postpartum. Women with frequent LOC gained on average 3.7 kg more, they had 3-fold increased odds of gaining more weight than recommended by the IOM, and weighed on average almost 4 kg more than those without LOC at 8 wk postpartum. These differences persisted even after adjustment for pre-pregnancy BMI. This finding is consistent with evidence that women who engaged in binge eating (and those with frank BED) in pregnancy had higher GWG (17, 38). Of note, 1 study did not find a correlation between number of binge eating episodes and GWG amongst overweight African American women, although low power limits confidence in these findings (39).

Child weight was also higher at birth in women with pregnancy LOC compared with women with no LOC and these findings are consistent with extant research on birth weight in offspring of women with binge eating and BED (17, 40). We observed a dose-response effect of frequency of LOC in pregnancy on offspring birth weight. The effect of maternal LOC in pregnancy on offspring weight outcomes persisted into adolescence and exploratory analyses showed 2-fold higher odds of being overweight and obese in youth born to mothers who reported pregnancy LOC, even after adjustment for birth weight.

Strengths and limitations

Our results need to be understood in the context of relevant strengths and limitations.

This is the first study to our knowledge to investigate LOC during pregnancy and its effects on maternal diet, perinatal maternal and child weight outcomes, and long-term child weight. Our data were collected as part of a large population-based community study, relying on objectively and prospectively collected data on maternal and child weight and BMI. Women enrolled in ALSPAC are representative of the population in its geographic catchment area, and its nature, a community-based sample, excludes selection bias common in studies on clinical populations. Generalizability might be limited, however, by the nature of the sample, which is representative of the area it was drawn from (Avon county) and largely the UK. Data on maternal diet were obtained through the use of self-report, and it is possible that women with LOC might have misreported their intake. However, FFQs are a good measure of food intake in large population-based studies (41, 42). Unfortunately data on maternal LOC postpartum were not available, which might contribute to child overeating and weight gain throughout childhood. ALSPAC was set up in the 1990s, hence it is possible that the prevalence of pregnancy LOC might have increased since, due to the high obesity levels in gestation. However, it is unlikely that secular trends might affect longitudinal associations seen in this study. Pre-pregnancy weight—used to calculate GWG—was estimated from models of weight gain during pregnancy; however, predicted and self-reported weight were highly correlated. LOC in pregnancy predicted missingness on GWG data, therefore potentially leading to an underestimation of the effect of LOC on GWG. About 23% of women did not report their weight at 8 wk postnatal; however, no differential missingness was observed across exposure categories. Lastly, maternal BMI is a strong predictor of child adiposity (43) and evidence suggests this effect is mostly genetic (43). Given our main focus on establishing associations rather than causality, we did not set out to disentangle genetic and intrauterine effects in this study. Future studies should investigate specific risk pathways.

Conclusions

This large population-based study provides initial and compelling evidence of short- and long-term maternal and child effects of LOC in pregnancy. These findings are particularly relevant to obesity prevention in both mothers and their offspring, given the importance of GWG on later adiposity in women, and the limited efficacy of obesity treatment. This study adds to our currently limited understanding of the effects of maternal eating on maternal and child weight outcomes, as very few studies have investigated LOC and binge eating in pregnancy. Future research should aim to understand fetal programming and developmental offspring outcomes in women with pregnancy LOC.

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