

ORIGINAL ARTICLE

Maternal diet, breastfeeding and adolescent body composition: a 16-year prospective study

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BACKGROUND/OBJECTIVES: Overweight and obesity are becoming increasingly prevalent problems worldwide. A number of factors in early life have been found to be associated with body composition of neonates or young children but there is limited follow-up data for adolescents. This study aims to describe associations between early nutrition and body composition in adolescents.

SUBJECTS/METHODS: Birth cohort study of 415 pregnant women and their offspring (mean age 16 years). Body composition including fat mass (FM) and lean body mass (LBM) of adolescents at 16 years of age was measured by dual-energy X-ray absorptiometry. Information on maternal food and nutrients intake during the third trimester of pregnancy and breastfeeding was collected by questionnaires soon after birth.

RESULTS: A total of 264 mother–adolescents pairs were studied. Maternal antenatal meat intake was positively associated with FM of adolescents (an increase of 0.9%/portion, $P < 0.01$). There were also positive associations between maternal energy intake (per 1000 kJ/day), fat (per 10 g/d) and protein (per 10 g/day) intake and offspring's FM (an increase of 1.3%), but these became borderline after adjustment for confounders. Breastfeeding > 25 days was negatively associated with FM in adolescents (a decrease of 14%, $P = 0.01$). These associations were independent of the significant association between maternal energy and macronutrient intakes during pregnancy and adolescent intakes at 16 years of age. No significant association was found between maternal dietary intake and lean mass in adolescents.

CONCLUSIONS: Breastfeeding may have a biological effect that is beneficial for the prevention of obesity. Conversely, higher maternal meat intake during pregnancy may increase FM in adolescents.

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INTRODUCTION

Overweight and obesity are becoming increasingly prevalent problems worldwide, ~1 in 4 UK and Australian children and 1 in 3 US children are overweight or obese.¹ Obesity in childhood and adolescence not only leads to increased incidence of hypertension, diabetes and morbidity, but also to adult obesity and its related adverse metabolic and cardiovascular sequelae.² Once obesity is established, it may be less amenable to population-based lifestyle interventions than predicted.³ Adipocyte development begins in the fetus and has the capacity for 'unlimited' growth,² thus, exploring the determinants of body composition and understanding which early-life events regulate adipose tissue distribution in offspring are likely to provide important insights for novel interventions that may prevent excess adiposity in later life.

There are data suggesting a number of factors in early life might programme fat cell responses,^{4,5} determine neonatal body composition.⁶ Among these factors, intrauterine overnutrition and breastfeeding were shown to have an enduring influence on subsequent adiposity, which appeared to be important causes of childhood obesity but of modest importance relative to known causes of obesity among adults.⁴ Animal experiments showed that a maternal diet high in fat could significantly increase the susceptibility to diet-induced obesity⁷ and percentage total body fat in rat offspring.⁸ Results from several long-term longitudinal

studies were reported in recent years. It showed that mothers with a higher pre-pregnant body mass index (BMI) or a larger mid-upper arm circumference during pregnancy tended to have children with greater adiposity at the age of 9.⁹ Thereafter, a study in the UK examined the association between maternal dietary intake in pregnancy and offspring dietary intake and adiposity at 10 years of age, and suggested strong prenatal maternal associations with child dietary intake.¹⁰ However, subjects in this study were about 10 years of age, which was not old enough to provide associated information of maternal diet with offspring's body composition after puberty.

To our knowledge, no result from prospectively collected data was reported to show the relationship between maternal diet and later adiposity in adolescence. We previously reported association between maternal diet during pregnancy, breastfeeding and bone mineral density of offspring at 8 and 16 years.^{11–13} Therefore, the aim of this study was to use a birth cohort study to investigate the associations between maternal diet during the third trimester of pregnancy, breastfeeding and subsequent long-term body composition of offspring at 16 year.

MATERIALS AND METHODS

Tasmania is an island state of Australia, and 99% residents are Caucasian. Subjects in this study were part of an original cohort of individuals enrolled

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in 1988–1989 as part of an investigation of sudden infant death syndrome by previously published criteria,¹⁴ including maternal age, birth weight, month of birth, infant sex, duration of the second stage of labor and infant feeding. In 1988 and 1989, 1500 infants were born in Southern Tasmania, who met these criteria and were eligible to join the study. Of these subjects, mothers of 1435 infants (96%) agreed to an in-hospital interview and 1127 (75%) filled in a food frequency questionnaire. A total of 415 adolescents were traced and invited to participate in a study on fracture, bone density and body composition in 2004–2005 when they were 16 years old.

This study was conducted according to the guidelines laid down in the Declaration of Helsinki and all the procedures involving human subjects were approved by the Southern Tasmania Health and Medical Human Research Ethics Committee. Written informed consent was obtained from all subjects.

Dietary assessment

Maternal dietary intake during the third trimester of pregnancy was measured with a self-administered FFQ (Food Frequency Questionnaire)¹⁵ applied shortly after birth with two very similar FFQs being used. The first was a questionnaire designed for use by child subjects (PFFQ (Pediatric Food Frequency Questionnaire)), containing 151 food categories but not including categories such as alcoholic drinks or tea and coffee. It was used until October 1988 and then we used the second FFQ, which contained 179 food categories and was designed to be self-administered to adults (AFFQ (Adult Food Frequency Questionnaire)). Both questionnaires included standard serve sizes; however, to be more appropriate for children, the serve sizes given in the PFFQ were occasionally smaller than those in the AFFQ. Mean daily energy and nutrient intake was estimated from each questionnaire using nutrient data from the Australian Tables of Food Composition (NUTTAB95, AGPS, 1995). Mothers of multiple-birth children, who gave multiple answers in a food category, who omitted pages of the questionnaire or who reported consuming from only 15 of the food categories or less were determined to have inadequately completed the questionnaire and were excluded from this part of the analysis.

For adolescents at the age of 16 years, dietary intake was investigated with the ACCV FFQ (The Anti-Cancer Council of Victoria FFQ),^{16,17} which contained 121 food items. Adolescents who gave multiple answers in a food category, who omitted questions of the questionnaire or who reported portion size factor, which based on only two foods or less were excluded from this study.

Likely under-reporting of energy intake in mothers was calculated as reported daily energy intakes of <120% of their estimated basal metabolic rates.¹⁸ Maternal basal metabolic rates were estimated by using Schofield's equations for adult men and women, as described in the Nutrient Reference Values for Australia and New Zealand,¹⁹ which are based on age and (BMI; in kg/m²). In mothers, pre-pregnancy BMI was used to estimate prenatal basal metabolic rates, and an additional 1.9 MJ/day in the third trimester was added following recommended dietary intakes.¹⁹ On the basis of this method, 12 (4.5%) mothers for prenatal diet in the third trimester were classified as underreporters. In children, under-reporting was defined as a ratio of reported energy intake to predicted energy requirements of <78%.^{20,21} Predicted energy requirement was calculated from body weight after sex, age and energy requirements for growth had been taken into account.²¹ Under-reporting was identified for 160 children (61%).

Breastfeeding

Actual breastfeeding was assessed by a questionnaire administered face-to-face home visit when the infant was ~1 month of age. The cutoff point of actual breastfeeding for analysis was 25 days, because this was the minimum time lag between child birth and the home visit in this study. We also confirmed this via a telephone interview when the infant was ~3 months of age and with a breastfeeding recall study when the child was 8 years old.

Anthropometric measures and body composition

Data on measured birth weight was available from the pre-existing database, the Tasmanian Infant Health Survey.²² Weight of adolescents was measured to the nearest 0.1 kg (with shoes, socks and bulky clothing removed). Height was measured to the nearest 0.1 cm (with shoes and socks removed) using a stadiometer. BMI was calculated by dividing body weight in kilograms by the square of height in meters (kg/m²). BMI cutoffs for overweight and obese by sex and age,²³ in boys of age 16 years were

≥23.9 kg/m² or ≥28.9 kg/m², respectively, in girls were ≥24.4 kg/m² or ≥29.4 kg/m². Tanner stage of adolescents at the age of 16 years was measured by showing and explaining the participant Tanner stage photographs, which illustrate the five stages of pubertal development and asking the participant to choose the drawing closest to his/her stage of development.²⁴

Body composition was measured by dual-energy X-ray absorptiometry with a Hologic Delphi densitometer on array setting (Hologic, Waltham, MA, USA). Outputs included fat mass (FM), lean body mass and FM percentage (%BF). The longitudinal coefficient of variation for our instrument during 2004 and 2005 using daily measurements of a spine phantom was 0.5%.

Other factors

Physical activity was retrospectively assessed in the year before study entry using a questionnaire validated in US adolescents.²⁵ Strenuous activity (exercise which was severe enough to raise pulse rate and breath) was used to assess physical activity level. Mother's educational level (never attended school, primary school, high school and tertiary education), marital status (unmarried living together or not, married living together or not, divorced, other) and parents' employment status (unemployed, home duties, employed part-time or full-time, student, permanently unable to work/ill) were collected with questionnaires at the time of the child's birth.

Statistical analysis

We used mean (s.d.) or median (interquartile range) to describe continuous variables, and *t*-tests or Mann-Whitney *U*-test to compare differences between groups as appropriate. Associations between maternal diet and offspring diet, and associations of potential confounders with adolescents' diet, physical activity, fat and lean mass were carried out by using Spearman rank correlation analysis. We used multiple linear regression to explore the possible associations between maternal energy intake, macronutrients intake, food intake in the third trimester and offspring body composition with adjustment for potential confounders. Fat and lean mass variables were log transformed to approximate a normal distribution. For the semi-log model where the dependent variable has been transformed using the log transformation while the explanatory variables are in their original units, the coefficient β is interpreted which includes the following: for a one unit change in the explanatory variable, the dependent variable changes by $\beta \times 100\%$ holding all other explanatory variables constant.²⁶ Macronutrients and foods intakes were adjusted for energy and were confirmed using the nutrition residual method (nutrient intakes were regressed on total-energy intake and the residuals were entered into models in place of nutrient intake²⁷) (data not shown). For the main analysis, we adjusted for dietary under-reporting in mothers by putting this as a covariate in the models.

A *P* value <0.05 (two tailed) or a 95% confidence interval not including the null point was regarded as statistically significant. All statistical analyses were performed on SAS 9.1 for Windows (Cary, NC, USA).

RESULTS

A total of 415 children were traced 16 years later. Of the 415 participants, 264 participants qualified for maternal diet analysis after excluding participants with uncompleted food frequency questionnaire and multiple births. In this study, >60% were male offspring and all were close to 16 years (Table 1). Compared with children who could not be traced, the included mother-offspring pairs were slightly but significantly younger as adolescents (16.3 years vs 16.4 years). However, there were no significant differences in maternal dietary intake during pregnancy, child's birth weight, body composition and gender distribution between these participants and those lost to follow-up.

Maternal energy intake ($r=0.14$), fat intake ($r=0.15$) and carbohydrate intake ($r=0.13$) were positively associated with intakes of their offspring, all $P<0.05$. With Spearman correlation analysis (Table 2), energy intake of adolescents was positively associated with lean body mass ($r=0.22$) and physical activity level ($r=0.20$), but negatively associated with FM ($r=-0.31$), %BF ($r=-0.36$) and BMI ($r=-0.19$), all $P<0.001$. Similar associations were found with macronutrient intakes. Physical activity level

Table 1. Characteristics of subjects

	Variables	Included (n = 264)	Excluded (n = 151)	P ^a
Adolescent	Birth weight (kg)	3.1 ± 0.8	3.0 ± 0.8	0.23
	Male (%)	66	61	0.35
	Current age (years)	16.3 ± 0.4	16.4 ± 0.5	0.005
	Height (cm)	170.4 ± 8.9	170.0 ± 8.7	0.55
	Weight (kg)	67.2 ± 14.3	67.5 ± 14.5	0.87
	Current energy intake (kJ/day)	9350 (6842)	9629 (6766)	0.24
	Fat mass (kg)	15.5(12.1)	15.7 (13.6)	0.59
	Lean body mass (kg)	51.3 (14.9)	49.4 (15.0)	0.77
	%BF (%)	23.9 (16.4)	24.0 (15.8)	0.48
	Overweight (%)	19	20	0.96
	Obesity (%)	11	11	0.96
	Tanner stage ≥ 4 (%)	95	95	0.98
	Strenuous activity participation (%)	90	85	0.10
	Smoke (%)	52	50	0.72
Mother	Age when gave birth (years)	25.1 ± 4.9	25.4 ± 5.0	0.45
	Weight before gave birth (kg)	71.3 ± 12.2	73.4 ± 12.7	0.10
	Mothers with tertiary education (%)	26	27	0.73
	Lived with father (%)	84	82	0.58
Others	Father employed (%)	85	86	0.92

Abbreviation: %BF, fat mass percentage. ^aFor continuous variables, data are tabulated as mean ± s.d. or median (interquartile range), using unpaired t-test. For other variables, using Mann-Whitney U-test.

Table 2. Correlation coefficients between offspring's body composition and macronutrients intake, physical activity^a

	Fat mass ^b	Lean mass ^b	Fat percentage	BMI	Physical activity
Energy					
r	-0.31	0.22	-0.36	-0.19	0.20
Protein					
r	-0.26	0.23	-0.31	-0.13	0.20
Fat					
r	-0.29	0.19	-0.34	-0.19	0.18
Carbohydrate					
r	-0.32	0.22	-0.37	-0.20	0.20
Physical activity					
r	-0.26	0.18	-0.29	-0.11*	

Abbreviation: BMI, body mass index. *P>0.05. ^aCorrelation coefficients in bold, P<0.01. Correlation coefficients in bold and italics, P<0.001. ^bFat mass and lean mass was log transformed.

in adolescents was positively associated with their nutrient intakes, but negatively associated with their FM and %BF.

The association of maternal energy and macronutrient intakes with offspring's body composition is shown in Table 3. Before adjusting for confounders, maternal energy intake ($\beta = 0.013$ kg/1000 kJ), fat ($\beta = 0.014$ kg/10 g) and protein intake ($\beta = 0.013$ kg/10 g) were positively associated with offspring's FM (log transformed), all $P < 0.05$. With the interpretation of the semi-log model, these showed that there is ~1.3% increase in FM in adolescents for every 1000 kJ/day higher energy intake, or 10 g more fat intake or 10 g more protein intake in mothers in the third trimester of pregnancy. After adjustment for potential confounders such as adolescents' height, height², maternal energy intake for macronutrients, maternal under-reporting, paternal employment at birth and adolescents' physical activity, these became nonsignificant (all $P > 0.05$). There was no significant association between maternal energy and macronutrient intake and lean mass in the offspring.

The association between maternal food intakes in third trimester of pregnancy, such as meat, milk, fish, fruit and vegetable and offspring's body composition is shown in Table 4. Maternal meat intake in the third trimester was positively associated with FM of adolescents, before ($\beta = 0.011$ kg/portion, $P < 0.001$) and after ($\beta = 0.009$ kg/portion, $P < 0.05$) the adjustment for potential confounders (all $P < 0.05$), and these associations persist after adjustment for current diet (data not shown). With the interpretation of the semi-log model, the results showed an increase of ~0.9% FM in adolescents for each portion of meat in the third trimester of pregnancy. There were no significant associations between other foods intake and offspring's FM or lean mass. These associations were independent of the significant association between maternal energy, fat and carbohydrate intake in the third trimester during pregnancy and adolescent intakes of the same nutrients at the age of 16 years.

The association between breastfeeding and body composition in adolescents is shown in Table 5. After adjustment for potential confounders, breastfeeding for >25 days was negatively associated with FM ($\beta = -0.12$, $P = 0.02$) in adolescents, and remained significant after additional adjustment for paternal employment at birth ($\beta = -0.14$, $P = 0.01$). This was corresponded to 14% lower FM in adolescents, if breastfeeding was >25 days. Any breastfeeding ≤ 25 days was not associated with FM or lean body mass of adolescents, all $P > 0.05$.

DISCUSSION

Associations between early-life factors and body composition of the offspring have been reported but nearly all of them are for pre-pubertal children. This is the first to have followed the offspring for 16 years. It confirms greater maternal macronutrient intakes in the third trimester of pregnancy were associated with greater adolescent intakes at the age of 16 years for the same nutrients. This study strongly suggests that maternal meat intake during pregnancy may lead to higher body FM and confirms that breastfeeding may have a long-term protective effect against obesity.

The association between maternal diet and body composition has previously been studied in animals. Experiments showed that

Table 3. Association of maternal energy and macronutrient intakes in the 3rd trimester of pregnancy with offspring's body composition

	Model 1		Model 2		Model 3	
	β (95%CI)	P	β (95%CI)	P	β (95%CI)	P
Fat mass (kg)^a						
Energy (per 1000 kJ)	0.013 (0.001, 0.025)	0.04	0.011 (– 0.001, 0.023)	0.07	0.012 (0.000, 0.024)	0.05
Protein (per 10 g)	0.014 (0.002, 0.026)	0.03	0.013 (– 0.010, 0.035)	0.27	0.012 (– 0.011, 0.034)	0.32
Fat (per 10 g)	0.013 (0.002, 0.023)	0.02	0.022 (– 0.004, 0.047)	0.09	0.017 (– 0.009, 0.043)	0.2
Carbohydrate (per 50 g)	0.012 (– 0.008, 0.032)	0.25	– 0.039 (– 0.084, 0.005)	0.08	– 0.033 (– 0.080, 0.013)	0.16
Lean mass (kg)^a						
Energy (per 1000 kJ)	0.002 (– 0.002, 0.007)	0.34	0.002 (0.000, 0.004)	0.10	0.002 (0.000, 0.005)	0.07
Protein (per 10 g)	0.003 (– 0.002, 0.008)	0.19	0.004 (– 0.001, 0.008)	0.10	0.004 (0.000, 0.009)	0.07
Fat (per 10 g)	0.002 (– 0.002, 0.006)	0.37	0.003 (– 0.002, 0.008)	0.26	0.003 (– 0.002, 0.008)	0.27
Carbohydrate (per 50 g)	0.003 (– 0.005, 0.010)	0.49	– 0.009 (– 0.018, 0.000)	0.06	– 0.009 (– 0.018, 0.001)	0.07

Abbreviation: CI, confidence interval. Model 1 unadjusted. Model 2 adjusted for gender adolescents' height, height squared, maternal energy intake for macronutrients and maternal under-reporting. Model 3 further adjusted for paternal employment, adolescents' physical activity. ^aFat mass and lean mass were log transformed.

Table 4. Association between maternal food intakes in the 3rd trimester of pregnancy and offspring's body composition

	Model 1		Model 2		Model 3	
	β (95%CI)	P	β (95%CI)	P	β (95%CI)	P
Fat mass (kg)^a						
Meat (portion)	0.011 (0.004, 0.019)	<0.001	0.010 (0.002, 0.017)	0.02	0.009 (0.001, 0.017)	0.03
Vegetable (portion)	0.002 (– 0.001, 0.004)	0.15	0.001 (– 0.001, 0.004)	0.34	0.001 (– 0.002, 0.004)	0.45
Fish (portion)	0.021 (– 0.006, 0.049)	0.12	0.020 (– 0.005, 0.044)	0.12	0.023 (– 0.002, 0.047)	0.07
Fruit (portion)	0.002 (– 0.002, 0.007)	0.24	0.001 (– 0.003, 0.005)	0.75	0.001 (– 0.003, 0.005)	0.56
Milk (per 100 ml)	0.006 (– 0.004, 0.017)	0.22	0.003 (– 0.007, 0.014)	0.53	0.003 (– 0.007, 0.014)	0.52
Lean mass (kg)^a						
Meat (portion)	0.002 (– 0.001, 0.005)	0.26	0.001 (0.000, 0.003)	0.09	0.001 (0.000, 0.003)	0.09
Vegetable (portion)	0.001 (0.000, 0.002)	0.17	0.000 (0.000, 0.001)	0.55	0.000 (0.000, 0.001)	0.41
Fish (portion)	0.006 (– 0.005, 0.016)	0.29	0.002 (– 0.003, 0.007)	0.46	0.001 (– 0.004, 0.007)	0.58
Fruit (portion)	0.000 (– 0.002, 0.001)	0.79	0.000 (– 0.001, 0.001)	0.97	0.000 (– 0.001, 0.001)	0.87
Milk (per 100 ml)	0.003 (– 0.001, 0.007)	0.09	0.002 (0.000, 0.004)	0.09	0.002 (0.000, 0.004)	0.06

Abbreviation: CI, confidence interval. Model 1 unadjusted. Model 2 adjusted for adolescents' height, height squared, maternal energy intake for macronutrients and maternal underreporting. Model 3 further adjusted for paternal employment, adolescents' physical activity. ^aFat mass and lean mass were log transformed.

Table 5. Association between breastfeeding and offspring's body composition

	Model 1		Model 2		Model 3	
	β (95%CI)	P	β (95%CI)	P	β (95%CI)	P
Fat mass (kg)^a						
No breastfeeding	reference	—	reference	—	reference	—
Breastfed \leq 25 days	– 0.037 (– 0.195, 0.122)	0.65	– 0.030 (– 0.173, 0.112)	0.68	– 0.034 (– 0.174, 0.107)	0.64
Breastfed > 25 days	– 0.123 (– 0.228, – 0.019)	0.02	– 0.113 (– 0.207, – 0.018)	0.02	– 0.139 (– 0.239, – 0.039)	0.01
Lean mass (kg)^a						
No breastfeeding	reference	—	reference	—	reference	—
Breastfed \leq 25 days	– 0.023 (– 0.084, 0.038)	0.46	– 0.006 (– 0.038, 0.025)	0.69	– 0.007 (– 0.039, 0.024)	0.65
Breastfed > 25 days	0.011 (– 0.029, 0.052)	0.58	– 0.015 (– 0.036, 0.005)	0.15	– 0.014 (– 0.036, 0.008)	0.21

Abbreviation: CI, confidence interval. Model 1 unadjusted. Model 2 adjusted for adolescents' height, height squared, maternal energy intake for macronutrients and maternal underreporting. Model 3 further adjusted for paternal employment, adolescents' physical activity. ^aFat mass and lean mass were log transformed.

a maternal diet high in fat could significantly increase the proportion of both total body fat and abdominal fat in the rats offspring.⁸ Offspring of female rats fed with a diet containing lower protein during pregnancy had a significantly lower adult

body weight due in part to a decrease in body fat, which might be a result of programming of the insulin axis.²⁸ A systematic review found it was hard to evaluate the effect of long-chain polyunsaturated fatty acid supplementation during pregnancy

on infant and child body composition, and highlighted the need for further human studies.²⁹ Some studies in humans reported that maternal diet composition, such as energy from fat and protein, high-meat and low-carbohydrate diet, has long-term effects on offspring blood pressure,^{30,31} but only one studied the association between maternal diet and offspring body composition, with the result that maternal diet during pregnancy was not associated with adiposity or lean mass of offspring at 10 years of age.¹⁰ Another study showed that maternal glucose concentration during pregnancy was positively associated with children's lean mass and adiposity pre-pubertal offspring.³² These limited studies only traced young offspring to the pre-pubertal stage, thus were not long enough to describe long-term effects of maternal diet on offspring's body composition.

Maternal diet during third trimester of pregnancy in our study was found to be associated with body composition of adolescents, there was ~1.3% increase in FM in adolescents for every 1000 kJ/day higher energy intake, or 10 g more fat intake or 10 g more protein intake in mothers in the third trimester of pregnancy. These became borderline or nonsignificant after adjustment for potential confounders, which indicated that effects of maternal energy, fat or protein intake in third trimester of pregnancy were not independent of other biological or social factors. In contrast, the present study shows that there were increases of 0.9% in FM in adolescents if their mothers had one more portion meat intake in the third trimester of pregnancy. This suggests that maternal high meat intake during pregnancy may substantially increase FM of the offspring, and increase the risk of those offspring becoming obese in later life independent of gender, current energy intake and physical activity. These results have important implications for public health and in terms of prevention of obesity. Further research is needed to examine the mechanisms by which maternal meat intake, but not fat or protein intake, during pregnancy influences children's body composition.

This report indicates that greater maternal prenatal macronutrient intakes were associated with greater child intakes at 16 years of the same nutrients. A study in pre-pubertal offspring showed that compared with paternal association, stronger maternal associations were observed for prenatal macronutrient intake, possibly reflecting an intrauterine effect of fetal over-nutrition on child appetite.¹⁰ In addition to the targeting dietary habits in childhood,³³ interventions targeting maternal dietary habits in pregnancy or pre-conceptually might be required to establish good dietary habits in both parents and offspring as early as possible. Parental feeding attitudes and styles were also found to have a pivotal role in the development of their child's food preferences and energy intake.³⁴

Reviews have concluded there is a relationship between breastfeeding and a reduced prevalence of obesity,³⁵ however, the few studies evaluating childhood fatness have provided limited direct support for this hypothesis. In this 16-year longitudinal study, longer breastfeeding in early life was associated with decreased FM in adolescents, but not LM. This was independent of an extensive list of biological and social confounders and provides strong evidence that longer breastfeeding may reduce the prevalence of obesity in adolescents. Possible explanations discussed in earlier studies, including behavioral and hormonal mechanisms and differences in macronutrient intake.³⁶

In this study, subjects who were breastfed for more than 25 days had lower FM, but actual breastfeeding \leq 25 days was not associated with body composition after adjustment. This highlighted there might be a duration-dependent effect of breastfeeding on FM and %BF of adolescents, consistent with recent evidence suggesting a longer period of breastfeeding was associated with lower BMI at 1 year of age,³⁷ and that breastfeeding remained a significant protective factor up to age 5 to 6 y.^{38,39} Our results supported this hypothesis and suggested

the inverse association between duration of breastfeeding and obesity might be a long term effect.

In this study, information of breastfeeding was collected and confirmed with several methods and times to minimize measure error and confounding. Breastfeeding is linked with socioeconomic status, so it is difficult to separate the causal roles of nutrition or socioeconomic status in early life.^{35,40} However, in this study, we found that maternal marital status, educational level and employment were not associated with body composition of the offspring. Furthermore, after adjusting for other factors, including paternal employment status, breastfeeding was still significantly associated with lower FM of adolescents, suggesting that breastfeeding itself was protective against obesity and this did not appear attributable to differences in socioeconomic level or lifestyle.

Several limitations should be considered in interpreting the findings from the present study. First of all, children who took part in this study were originally selected on the basis of having a higher risk of sudden infant death syndrome, thus, were not representative of Tasmanian children. There were a higher proportion of males, premature babies, teenage mothers and smoking during pregnancy. These findings suggested that this group were of a lower socioeconomic status than the Tasmanian population as a whole but could still be considered generalizable according to the criteria outlined by Miettinen.⁴¹ In addition, maternal nutrient intake (collected with a food frequency questionnaire) were much greater than previous reports in Australian pregnant women.⁴² This is likely to represent random error rather than bias. We adjusted for energy by including maternal energy intake in standard regression models, which would reduce the effect of measurement error.⁴³ Supporting this, the energy intake of adolescents were negatively associated with FM (kg and %) and BMI, which was in agreement with other observations.

In conclusion, this study adds to the limited but growing number of prospective studies that have examined the relation between early diet and adolescent body composition. It suggests that breastfeeding may have a biological effect that is beneficial for the prevention of obesity. Conversely, higher maternal meat intake during pregnancy may increase FM in adolescents.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

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REFERENCES

- 1 Cretikos MA, Valenti L, Britt HC, Baur LA. General practice management of overweight and obesity in children and adolescents in Australia. *Med Care* 2008; **46**: 1163–1169.
- 2 Budge H, Gnanalingham MG, Gardner DS, Mostyn A, Stephenson T, Symonds ME. Maternal nutritional programming of fetal adipose tissue development: long-term consequences for later obesity. *Birth Defects Res C Embryo Today* 2005; **75**: 193–199.
- 3 Swinburn B, Egger G. The runaway weight gain train: too many accelerators, not enough brakes. *BMJ* 2004; **329**: 736–739.
- 4 Martorell R, Stein AD, Schroeder DG. Early nutrition and later adiposity. *J Nutr* 2001; **131**: 874S–880S.
- 5 McMillen IC, MacLaughlin SM, Muhlhauser BS, Gentili S, Duffield JL, Morrison JL. Developmental origins of adult health and disease: the role of periconceptual and foetal nutrition. *Basic Clin Pharmacol Toxicol* 2008; **102**: 82–89.

- 6 Harvey NC, Poole JR, Javaid MK, Dennison EM, Robinson S, Inskip HM et al. Parental determinants of neonatal body composition. *J Clin Endocrinol Metab* 2007; **92**: 523–526.
- 7 Tamashiro KL, Terrillion CE, Hyun J, Koenig JI, Moran TH. Prenatal stress or high-fat diet increases susceptibility to diet-induced obesity in rat offspring. *Diabetes* 2009; **58**: 1116–1125.
- 8 Buckley AJ, Keserü B, Briody J, Thompson M, Ozanne SE, Thompson CH. Altered body composition and metabolism in the male offspring of high fat-fed rats. *Metabolism* 2005; **54**: 500–507.
- 9 Gale CR, Javaid MK, Robinson SM, Law CM, Godfrey KM, Cooper C. Maternal size in pregnancy and body composition in children. *J Clin Endocrinol Metab* 2007; **92**: 3904–3911.
- 10 Brion MJ, Ness AR, Rogers I, Emmett P, Cribb V, Davey Smith G et al. Maternal macronutrient and energy intakes in pregnancy and offspring intake at 10 y: exploring parental comparisons and prenatal effects. *Am J Clin Nutr* 2010; **91**: 748–756.
- 11 Jones G, Riley MD, Dwyer T. Maternal diet during pregnancy is associated with bone mineral density in children: a longitudinal study. *Eur J Clin Nutr* 2000; **54**: 749–756.
- 12 Jones G, Riley M, Dwyer T. Breastfeeding in early life and bone mass in pre-pubertal children: a longitudinal study. *Osteoporos Int* 2000; **11**: 146–152.
- 13 Yin J, Dwyer T, Riley M, Cochrane J, Jones G. The association between maternal diet during pregnancy and bone mass of the children at age 16. *Eur J Clin Nutr* 2010; **64**: 131–137.
- 14 Ponsonby AL, Dwyer T, Kasl SV, Cochrane JA, Newman NM. An assessment of the impact of public health activities to reduce the prevalence of the prone sleeping position during infancy: the Tasmanian Cohort Study. *Prev Med* 1994; **23**: 402–408.
- 15 Baghurst K, Record S. A computerised dietary analysis system for use with diet diaries or food frequency questionnaires. *Community Health Stud* 1984; **8**: 11–18.
- 16 Ambrosini GL, van Roosbroeck SA, Mackerras D, Fritschi L, de Klerk NH, Musk AW. The reliability of ten-year dietary recall: implications for cancer research. *J Nutr* 2003; **133**: 2663–2668.
- 17 Hodge A, Giles GG, Patterson A, Brown W, Ireland P. The Anti-Cancer Council of Victoria FFQ. Relative validity of nutrient intakes compared with diet diaries in young of middle-aged women in a study of iron supplementation. *Aust NZ J Public Health* 2000; **24**: 576–583.
- 18 Pryer JA, Vrijheid M, Nichols R, Kiggins M, Elliott P. Who are the 'low energy reporters' in the dietary and nutritional survey of British adults? *Int J Epidemiol* 1997; **26**: 146–154.
- 19 Australia Government, Department of Health and Ageing, National Health and Medical Research Council. *Nutrient Reference Values for Australia and New Zealand Including Recommended Dietary Intakes*. Commonwealth: Australia, 2005, p 6.
- 20 Black AE, Cole TJ. Within- and between-subject variation in energy expenditure measured by the doubly-labelled water technique: implications for validating reported dietary energy intake. *Eur J Clin Nutr* 2000; **54**: 386–394.
- 21 Torun B. Energy requirements of children and adolescents. *Public Health Nutr* 2005; **8**: 968–993.
- 22 Dwyer T, Ponsonby AL, Newman NM, Gibbons LE. Prospective cohort study of prone sleeping position and sudden infant death syndrome. *Lancet* 1991; **337**: 1244–1247.
- 23 Cole TJ, Bellizzi MC, Flegal KM, Dietz WH. Establishing a standard definition for child overweight and obesity worldwide: international survey. *BMJ* 2000; **320**: 1240–1243.
- 24 Schall JI, Semeao EJ, Stallings VA, Zemel BS. Self-assessment of sexual maturity status in children with Crohn's disease. *J Pediatr* 2002; **141**: 223–229.
- 25 Aaron DJ, Kriska AM, Dearwater SR, Cauley JA, Metz KF, LaPorte RE. Reproducibility and validity of an epidemiologic questionnaire to assess past year physical activity in adolescents. *Am J Epidemiol* 1995; **142**: 191–201.
- 26 Ajmani V. *Applied Econometrics Using the SAS System*. John Wiley & Sons, Inc.: Hoboken, NJ, 2009.
- 27 Willett WC, Howe GR, Kushi LH. Adjustment for total energy intake in epidemiologic studies. *Am J Clin Nutr* 1997; **65**: 1220S–1228S.
- 28 Rees WD, Hay SM, Cruickshank M, Reusens B, Remacle C, Antipatis C et al. Maternal protein intake in the pregnant rat programs the insulin axis and body composition in the offspring. *Metabolism* 2006; **55**: 642–649.
- 29 Muhlhauser BS, Gibson RA, Makrides M. Effect of long-chain polyunsaturated fatty acid supplementation during pregnancy or lactation on infant and child body composition: a systematic review. *Am J Clin Nutr* 2010 857–863.
- 30 Adair LS, Kuzawa CW, Borja J. Maternal energy stores and diet composition during pregnancy program adolescent blood pressure. *Circulation* 2001; **104**: 1034–1039.
- 31 Shiell AW, Campbell-Brown M, Haselden S, Robinson S, Godfrey KM, Barker DJ. High-meat, low-carbohydrate diet in pregnancy: relation to adult blood pressure in the offspring. *Hypertension* 2001; **38**: 1282–1288.
- 32 Chandler-Laney PC, Bush NC, Rouse DJ, Mancuso MS, Gower BA. Maternal glucose concentration during pregnancy predicts fat and lean mass of prepubertal offspring. *Diabetes Care* 2011; **34**: 741–745.
- 33 van der Horst K, Oenema A, Ferreira I, Wendel-Vos W, Giskes K, van Lenthe F et al. A systematic review of environmental correlates of obesity-related dietary behaviors in youth. *Health Educ Res* 2007; **22**: 203–226.
- 34 Scaglioni S, Salvioni M, Galimberti C. Influence of parental attitudes in the development of children eating behaviour. *Br J Nutr* 2008; **99**: S22–S25.
- 35 Wells JC, Chomtho S, Fewtrell MS. Programming of body composition by early growth and nutrition. *Proc Nutr Soc* 2007; **66**: 423–434.
- 36 Arenz S, Ruckerl R, Koletzko B, von Kries R. Breast-feeding and childhood obesity—a systematic review. *Int J Obes Relat Metab Disord* 2004; **28**: 1247–1256.
- 37 O'Tierney PF, Barker DJ, Osmond C, Kajantie E, Eriksson JG. Duration of breast-feeding and adiposity in adult life. *J Nutr* 2009; **139**: 422S–425S.
- 38 Toschke AM, Martin RM, von Kries R, Wells J, Smith GD, Ness AR. Infant feeding method and obesity: body mass index and dual-energy X-ray absorptiometry measurements at 9–10 y of age from the Avon Longitudinal Study of Parents and Children (ALSPAC). *Am J Clin Nutr* 2007; **85**: 1578–1585.
- 39 von Kries R, Koletzko B, Sauerwald T, von Mutius E, Barnert D, Grunert V et al. Breast feeding and obesity: cross sectional study. *BMJ* 1999; **319**: 147–150.
- 40 Clements MS, Mitchell EA, Wright SP, Esmail A, Jones DR, Ford RP. Influences on breastfeeding in southeast England. *Acta Paediatr* 1997; **86**: 51–56.
- 41 Miettinen OS. *Theoretical Epidemiology. Principles of Occurrence Research in Medicine*. Delmar Publishers Inc.: Albany, NY, 1985.
- 42 Hure A, Young A, Smith R, Collins C. Diet and pregnancy status in Australian women. *Public Health Nutr* 2008; **23**: 1–9.
- 43 Day NE, Wong MY, Bingham S, Khaw KT, Luben R, Michels KB et al. Correlated measurement error—implications for nutritional epidemiology. *Int J Epidemiol* 2004; **33**: 1373–1381.