# Maternal macronutrient and energy intakes in pregnancy and offspring intake at 10 y: exploring parental comparisons and prenatal effects<sup>1–4</sup>

Marie-Jo A Brion, Andy R Ness, Imogen Rogers, Pauline Emmett, Victoria Cribb, George Davey Smith, and Debbie A Lawlor

## ABSTRACT

**Background:** High maternal dietary intakes in pregnancy may lead to increased fetal growth and program neuroendocrine pathways that result in greater appetite, energy intake, and adiposity in off-spring later in life. Few prospective dietary studies have explored this relation.

**Objective:** The objective was to assess associations of maternal dietary intake in pregnancy and maternal and paternal dietary intake postnatally with child dietary intake and adiposity.

**Design:** Dietary intakes of energy, protein, total fat, and carbohydrate were assessed prospectively in mothers during pregnancy, in mothers and their partners at 47 mo postnatally, and in children at 10 y (n = 5717 mother-child pairs prenatally, 5593 mother-child pairs postnatally, and 3009 father-child pairs). Child body composition was assessed at 9 and 11 y (n = 5725).

**Results:** Maternal dietary intakes of protein, fat (when adjusted for energy intake), and carbohydrate in pregnancy were positively associated with child dietary intakes of the same nutrients, and these associations were greater than those observed for paternal dietary intake, which was not strongly associated with offspring diet. Associations of maternal prenatal-offspring intakes were stronger than those of maternal postnatal-offspring intakes for protein and fat. Greater child energy and macronutrient intakes were only associated with greater adiposity in children when adjusted for potential energy underreporting. Maternal diet during pregnancy was not associated with offspring adiposity or lean mass.

**Conclusion:** The stronger prenatal maternal associations with child dietary intake, particularly protein and fat, compared with both paternal intake associations and maternal postnatal intake associations provide some evidence for in utero programming of offspring appetite by maternal intake during pregnancy. *Am J Clin Nutr* 2010;91:748–56.

## INTRODUCTION

Maternal plasma glucose concentrations during pregnancy are a key determinant of fetal growth (1), and there is evidence that maternal glucose concentrations affect offspring size and adiposity throughout life (2–10). The "hyperglycemia-hyperinsulinism" mechanism has been recognized as important since first proposed in the 1950s (2, 3). It suggests that increased fetal secretion of insulin in response to greater transfer of glucose across the placenta stimulates greater somatic fetal growth and higher levels of subcutaneous fat. In addition, mechanisms acting via cognitive and neuroendocrine development and subsequent offspring appetite and diet might be important (4–7). Although this has been explored in animals and in relation to gestational diabetes in humans (8–10), to our knowledge no previous study in humans has examined whether differences in maternal dietary intake in pregnancy are related to offspring dietary intake and adiposity in later life. Maternal macronutrient (carbohydrate, fat, and protein) and energy intakes potentially influence fetal growth and program future appetite. Glucose, amino acids, and free fatty acids are all transported across the placenta (11), and maternal glucose concentrations are influenced by total energy intake and proportions of carbohydrate, fat, and protein (12, 13).

The aim of this study was to build on existing evidence concerning maternal diet and glucose concentrations during pregnancy and the effect of these on fetal growth and subsequent offspring appetite and adiposity. The focus on maternal diet in pregnancy is important because this is potentially modifiable. In particular, we sought to explore the association of maternal macronutrients and energy during pregnancy with offspring macronutrient and energy intakes and to examine whether any associations of maternal dietary intakes with offspring dietary intakes have a subsequent effect on offspring fat mass. Associations of maternal macronutrient and energy intake with offspring intake may occur as a result of shared familial dietary patterns or genetic predisposition to certain tastes, rather than via intrauterine mechanisms. To examine the extent to which this might be

<sup>&</sup>lt;sup>1</sup> From the MRC Centre for Causal Analyses in Translational Epidemiology, Departments of Social Medicine (M-JAB, GDS, and DAL) and Oral and Dental Science (ARN), University of Bristol, Bristol, United Kingdom; School of Pharmacy and Biomolecular Sciences, University of Brighton, Brighton, United Kingdom (IR); and the Centre for Child & Adolescent Health, Department of Community Based Medicine, University of Bristol, Bristol, United Kingdom (PE and VC).

<sup>&</sup>lt;sup>2</sup> The contents of this article represent the views of the authors and not necessarily those of the funding bodies.

<sup>&</sup>lt;sup>3</sup> Supported by a grant from the National Institutes of Health, National Institute of Diabetes and Digestive and Kidney Diseases (R01 DK077659-01); the UK Medical Research Council; the Wellcome Trust; and the University of Bristol (core support for ALSPAC). M-JAB was funded by a Sir Henry Wellcome Postdoctoral Fellowship from the Wellcome Trust. GDS and DAL work in a UK Medical Research Council Centre, which is supported by grant G0600705.

<sup>&</sup>lt;sup>4</sup> Address correspondence and reprint requests to M-JA Brion, MRC Centre for Causal Analyses in Translational Epidemiology, Department of Social Medicine, University of Bristol, Oakfield House, Oakfield Grove, Bristol BS8 2NB, United Kingdom. E-mail: marie-jo.brion@bristol.ac.uk.

Received September 7, 2009. Accepted for publication December 8, 2009. First published online January 6, 2010; doi: 10.3945/ajcn.2009.28623.

the case, we compared maternal-offspring associations with paternal-offspring associations, assuming that a similar effect of maternal and paternal dietary intake on offspring dietary intake would imply shared familial, rather than intrauterine, mechanisms (14). This method was implemented previously in the Avon Longitudinal Study of Parents and Children (ALSPAC) (15–17) and was validated by the discordant associations of maternal and paternal smoking with offspring birth weight (14), reflecting the known intrauterine effects of maternal prenatal smoking on birth weight. We also compared associations for maternal prenatal intake-offspring intakes with associations for maternal postnatal intake-offspring intakes. If maternal nutrient intake in pregnancy has intrauterine effects on offspring appetite, we would expect stronger maternal prenatal intake-offspring intake associations than postnatal associations.

#### SUBJECTS AND METHODS

### Study participants

ALSPAC is a geographically based prospective cohort study investigating the health and development of children (18). Pregnant women residing in 3 health districts in the southwest England with an expected date of delivery between 1 April 1991 and 31 December 1992 were eligible; 14,541 pregnant women were recruited and 13,678 had a live-born, singleton child. In the present study we excluded parents and children of multiple births.

Of the singleton children with dietary data, there were 5717 maternal-child pairs, 3009 paternal-child pairs, and 2968 parental-offspring trios available for the main analyses. All children were invited to attend clinics at 9 and 11 y of age, during which time body-composition measurements (fat, lean, and bone mass) were taken. A total of 7585 singleton children had body-composition measurements at either 9 or 11 y, and, of these children, 6589 had dietary data and 5725 also had maternal prenatal dietary data and fat mass; 5244 children had data on dietary intake, body composition, and maternal postnatal diet. Ethical approval of the study was obtained from the ALSPAC Law and Ethics Committee and 3 Local Research Ethics Committees.

# Maternal, paternal, and offspring dietary intake

Mothers completed a food-frequency questionnaire (FFQ) at 32 wk of gestation, at which time they indicated how often they were currently consuming each of the 43 food groups. Together with nutrient information on standard sized portions, intakes for a range of nutrients, including energy, protein, fat, and carbohydrate, were derived. A detailed description of the methods is available elsewhere (19). Mothers and partners also completed a similar FFQ 47 mo postnatally.

At 10 y, offspring diet was assessed by using three 1-d unweighed dietary diaries in which children and caregivers recorded everything the child ate and drank in household measures for 2 weekdays and 1 weekend day. Completed diaries were brought to the clinic, and nutrition fieldworkers checked through the diaries to increase completeness and remove uncertainties. Diet diaries were transformed into food codes, with associated weights in grams for each item of food and drink recorded, by the same fieldworker using the DIDO program [Diet in, Data out; *see* Glynn et al (20) for details]. Food codes and nutrient content were derived from McCance and Widdowson's food tables and supplements (21–28). DIDO was used in conjunction with BRIGADE, a nutrient analysis program, to generate the nutrient content of each food the child ate. These data were used to generate average daily nutrient intakes and the amount of various food groups consumed.

Likely underreporting of energy intake in mothers/partners was calculated as reported daily energy intakes of <120% of their estimated basal metabolic rate (BMR) (29). Maternal/ partner BMRs were estimated by using Schofield's equations for adult men and women described in the UK Department of Health Report on Social Sciences (30), which are based on age and body mass index (BMI; in kg/m<sup>2</sup>). In mothers, prepregnancy BMI was used to estimate prenatal and postnatal BMR because pre-/postnatal BMIs at these time points were not assessed or unavailable. On the basis of this method, 2474 (43.3%) mothers for prenatal diet, 1722 (33.3%) mothers for postnatal diet, and 22 (0.7%) fathers were classified as underreporters. The low underreporting in men likely reflects differences in portion sizes chosen to estimate the FFQ in men and women in ALSPAC, with the fixed portion size used in men being 20% larger than that used in women. This was chosen based on evidence from the British National Diet and Nutrition Survey. The use of fixed portion sizes is conventional with FFQ data, where no information on portion size is collected. In children, underreporting was defined as a ratio of reported energy intake to predicted energy requirements of <78% (31, 32). Predicted energy requirement was calculated from body weight after sex, age, and energy requirements for growth had been taken into account (32). Underreporting was identified for 1866 (32.6%) children.

#### Child fat and lean mass measures

At the 9- and 11-y time points, fat and lean mass were assessed in children at the research clinics by using a Lunar Prodigy dualenergy X-ray absorptiometry (DXA; GE Medical Systems Lunar, Madison, WI) fan beam scanner (33). Briefly, DXA measures body bone, fat, and lean tissue by passing 2 different beams of Xray energy of known intensity through the body and by measuring attenuation of the beam detector. Whole-body scans were carried out with children wearing light clothing, with metal objects such as bracelets and watches removed. CVs (in 122 children with same-day repeat DXA measures at 9 y) were 2.27% and 1.12% for total fat mass and lean mass, respectively.

#### Confounders

Maternal and paternal smoking habits were collected from questionnaires sent at 18 wk gestation to mothers and their partners. Mothers indicated the number of cigarettes smoked per day in the first 3 mo of pregnancy and also in the past 2 wk. These responses were combined to create a variable for any maternal smoking during pregnancy. Mothers also indicated whether their partner smoked. Partners were asked in their own questionnaire whether they smoked regularly at any time in the past 9 mo. Responses from mothers and partners were combined to create a variable for partner smoking. Details of all previous pregnancies resulting in either a live birth or a stillbirth, which enabled parity to be derived, were also gathered from the questionnaire sent at 18 wk of gestation. In a questionnaire sent at 32 wk, mothers recorded their own and their partner's highest education level [none, Certificate of Secondary Education (CSE), national school exams at 16 y), vocational, O level (national school exams at 16 y, higher than CSE), A level (national school exams at 18 y), or university degree]. Mothers also recorded their occupation and their partner's occupation, which were used to allocate them to social categories [I (professional) to V (unskilled manual worker)] according to the 1991 Office and Populations Censuses and Surveys standard (34). A single variable was derived from the highest social class of either parent. Maternal/paternal heights were self-reported from questionnaires sent at 12 wk of gestation to mothers and their partners. Child height at 10 y (for analyses with child diet only) and height at 9 or 11 y (for analyses with child fat and lean mass at 9 or 11 y) were measured to the nearest millimeter by using a Harpenden stadiometer.

## Statistical analysis

Nutrients were examined as continuous variables in their original units (eg, g or kJ) and as z scores (SD scores). Data for child fat mass and lean mass at 9 and 11 y were combined to create a variable for fat/lean mass at 9 or 11 y. These fat and lean mass variables were log transformed and converted to an age (in 1-mo categories) and sex standardized z scores for each of the separate clinic visits (9 or 11). The z scores from the 9- and 11-y visits were highly correlated (0.9 for both fat and lean mass), and the final combined outcome measure was the 9-y z score plus, for those who only attended at 11 y, the 11-y z score, as previously published (17). Associations of potential confounders with child diet, fat and lean mass, maternal prenatal diet, and paternal diet were carried out by using linear regression. Associations between 1) maternal prenatal and postnatal diet and offspring diet, 2) paternal diet and offspring diet, 3) child diet and current fat and lean mass, and 4) maternal prenatal and postnatal diet and offspring fat mass were analyzed by using multiple linear regression adjusted for covariables. Mutually adjusted regressions of protein, total fat, and carbohydrate included together in the same model were explored for maternal and paternal associations. Sex-specific associations and mutually adjusted associations for maternal prenatal and postnatal diets were also explored. Macronutrients were adjusted for energy (35), and, for main analyses, we adjusted for dietary underreporting in mothers, fathers, and children (results without these adjustments presented as supplementary data). Sensitivity analyses relating to nonpaternity of partners were performed in STATA by using the Clemons command (36, 37). All analyses were performed by using STATA 10 (Stata Corp, College Station, TX).

## RESULTS

Sample characteristics are presented in **Table 1**. Correlations between parental macronutrient and energy intakes are presented in **Table 2**. Associations of potential confounders with nutrient intakes in mothers (prenatal), fathers and offspring, and offspring fat and lean mass are presented as supplementary data (*see* Tables S1–S4 under "Supplemental data" in the online issue). Maternal prenatal and paternal protein intakes were positively associated with indicators of higher socioeconomic position. Maternal and paternal fat intakes were inversely as-

sociated with indicators of socioeconomic position. Parental energy and carbohydrate intakes showed inconsistent associations. Child energy intake was not associated with indicators of socioeconomic position; however, child protein and carbohydrate intakes were positively associated with indicators of higher socioeconomic position, whereas fat intake showed inverse associations. Child fat mass was inversely associated with indicators of greater socioeconomic position, with lean mass only associated with maternal and paternal smoking.

With adjustment for parental and child underreporting, maternal prenatal and postnatal macronutrient intakes (protein, fat, and carbohydrate) were positively associated with intakes of the same nutrients in offspring. Paternal macronutrient intakes were not associated with these outcomes (Table 3). There was strong statistical evidence of maternal compared with paternal differences in protein and fat associations with offspring intake (P for heterogeneity between these estimates <0.001 and 0.02, respectively), but only weak evidence for parental differences for carbohydrate intake (P = 0.1). Neither maternal nor paternal energy intake was strongly associated with child energy intake. Maternal postnatal macronutrient associations with offspring intake were broadly similar to those seen for prenatal intake, but with associations for protein and fat intakes being stronger for prenatal than for postnatal intake (Table 3). However, there was no strong evidence of statistical differences between maternal prenatal and postnatal associations (P = 0.2 and 0.3 for protein and fat, respectively). Results were similar in mutually adjusted models with maternal prenatal and postnatal intakes included in the same model, for each nutrient (results available from authors on request).

Between-father variation in energy and macronutrient intakes was much larger than the between-mother prenatal variation in intake (paternal SDs were 1.5–2 times those of maternal SDs). Therefore, scaling in terms of z scores results in comparing associations per 1 unit in mothers and per 1.5–2 units in fathers (where units represent kJ/g intake). Thus, we felt that using the original units was a more equal comparison and focused on these absolute dietary measures in this study.

Associations of parental and offspring macronutrient and energy intakes, stratified by offspring sex, are presented in **Table 4**. In both male and female offspring, maternal prenatal protein associations were stronger than paternal protein associations.

Sensitivity analyses were carried out to assess potential effects of various possible rates of nonpaternity on the results (*see* Table S6 under "Supplemental data" in the online issue). Even in analyses adjusted for a hypothetical nonpaternity rate as high as 20%, there would still be evidence of a stronger association of maternal prenatal protein intake in pregnancy with offspring intake, compared with paternal protein intake, although evidence is less robust for fat and carbohydrate intakes.

After underreporting and energy intake (macronutrients only) were adjusted for, higher child energy intake (and to a lesser extent protein intake) was associated with higher fat mass (**Table 5**). In mutually adjusted models (protein, fat, and carbohydrate adjusted for each other), the strongest predictor of child fat mass was fat intake and that for lean mass was carbohydrate intake. Neither maternal prenatal nor paternal macronutrient and energy intake was strongly associated with offspring fat or lean mass (**Table 6**). Although there was some evidence of associations with offspring fat mass, effect sizes were negligible. Maternal

TABLE	1
-------	---

Sample characteristics<sup>1</sup>

	Proportion	Mean $\pm$ SD	Range
Continuous characteristics			
Parental dietary intake			
Energy (kJ/d)			
Maternal	_	$7506 \pm 1959$	2228-17.600
Paternal	_	$11.054 \pm 3047$	3027-27.186
Protein (g/d)			
Maternal	_	$67.8 \pm 18.0$	16.3-158.1
Paternal	_	$101.6 \pm 27.0$	34.5-334.3
Total fat (g/d)			
Maternal		$70.5 \pm 22.6$	15 4-204 7
Paternal		104.2 + 34.9	17.1-302.0
Carbohydrate $(g/d)$		10.112 = 0.113	1711 00210
Maternal		$223.0 \pm 60.9$	45 3-557 0
Paternal		3222 + 1097	67 9-1104 2
Child dietary intake		322.2 = 109.1	07.5 1101.2
Energy (kI/d)	_	$7793 \pm 1571$	2136-15 120
Protein $(g/d)$	_	$618 \pm 153$	167-1461
Total fat $(g/d)$		$75.3 \pm 10.6$	16.0_183.2
Carbohydrate (g/d)		75.5 = 19.0 $247.6 \pm 54.0$	54.2 408.3
Categorical characteristics $(\%)^2$		$247.0 \pm 54.0$	54.2-490.5
Earning contrained characteristics ( $\pi$ )			
	36		
	5.0	—	
	9.0		_
	23.0		_
II T	45.5	—	_
	10.3		_
Maternal education	11.2		
None/CSE	11.3	—	
vocational	8.4	—	
Olevel	35.8	—	—
A level	27.8	—	—
Degree	16.7	—	—
Paternal education			
None/CSE	17.6	—	_
Vocational	8.2	—	_
O level	22.1	—	—
A level	29.2	—	—
Degree	22.8	—	—
Maternal smoking			
Yes	11.0	—	_
Temporary	5.0	—	_
No	84.0	—	—
Paternal smoking			
Yes	32.7	—	
No	67.3	—	—
Parity			
$\geq 3$	4.4	—	—
<3	95.6	_	_

 $^{1}$  n = 5717 for maternal/child data and 3009 for paternal/child data. CSE, Certificate of Secondary Education.

<sup>2</sup> In the maternal/child diet sample.

<sup>3</sup> Social categories ranging from I (professional) to V (unskilled manual worker).

<sup>4</sup> O level (national school exams at 16 y, higher than CSE) and A level (national school exams at 18 y).

postnatal macronutrient and energy intakes were also not strongly associated with offspring fat and lean mass.

Main associations without adjustment for underreporting are provided as supplementary data (*see* Tables S6–S8 under "Supplemental data" in the online issue). Associations of parentoffspring carbohydrate intake were much stronger in unadjusted analyses, and maternal energy showed a strong association with offspring energy intake. Additionally, child energy and fat in-

# takes were inversely associated with both fat and lean mass (both mutually adjusted model). The remaining associations were similar.

## DISCUSSION

Greater maternal prenatal macronutrient intakes (protein, fat, and carbohydrate) were associated with greater child intakes

## TABLE 2

Correlation coefficients between parental nutrient intakes assessed by food-frequency questionnaire'

	Maternal prenatal intake				Maternal postnatal intake				Paternal intake		
	Energy	Protein	Fat	Carbohydrate	Energy	Protein	Fat	Carbohydrate	Energy	Protein	Fat
Maternal prenatal											
Energy											
Protein	0.81										
Fat	0.90	0.69									
Carbohydrate	0.93	0.67	0.71								
Maternal postnatal											
Energy	0.45										
Protein		0.42			0.82						
Fat			0.44		0.90	0.69					
Carbohydrate				0.43	0.93	0.71	0.73				
Paternal intake											
Energy	0.13				0.21						
Protein		0.14				0.22			0.84		
Fat			0.16				0.24		0.90	0.73	
Carbohydrate				0.09				0.20	0.81	0.67	0.64

<sup>1</sup> P < 0.001 for all correlations.

at 10 y of the same nutrients. Associations between maternaloffspring macronutrient intakes were stronger than for paternal-offspring macronutrient associations, and maternal prenatal-offspring associations for protein and fat intake were stronger than maternal postnatal-offspring associations. After dietary underreporting was adjusted for, child energy intake was positively associated with child fat mass, and, when macronutrients were adjusted for each other, protein, fat, and carbohydrate were all associated with fat mass; the strongest predictor was fat intake. Maternal prenatal or postnatal macronutrient and energy intakes were not strongly associated with offspring fat or lean mass.

If equal associations had been observed for maternal prenataloffspring nutrient intakes compared with paternal-offspring nutrient intakes, it would have suggested that common familial components, such as shared diet, were the main drives of the associations (14). This is because fathers could not influence offspring diet via intrauterine mechanisms and it would be unlikely for different parental mechanisms (intrauterine mechanisms in mothers and completely different mechanisms in fathers) to produce identical results. Because stronger maternal associations were observed for prenatal macronutrient intake, it is possible that this reflected an intrauterine effect of fetal overnutrition on child appetite. However, it also possible that mothers and their dietary habits have more influence on a child's diet than do fathers and their dietary habits. We aimed to separate potential intrauterine effects from postnatal effects by comparing the magnitude of maternal prenatal and maternal postnatal associations with offspring macronutrient intakes. For protein and fat intake, maternal prenatal associations were stronger than postnatal associations with offspring intake. If associations do indeed reflect intrauterine effects, potential mechanisms may involve effects of high prenatal carbohydrate, protein, and fat intakes on greater maternal glucose concentrations in pregnancy, which may program offspring appetite. Animal studies report that both fetal exposure to high glucose concentrations during development (6) and increased maternal diet in pregnancy (7) result in altered development of appetite regulatory systems in offspring. This has

only been explored in animals and, to our knowledge, this is the first study in humans to examine differences in maternal pre- and postnatal dietary intakes and their relation with offspring adiposity and diet in later life.

Previous studies have reported relations between child diet and parental diet (38, 39); however, most looked cross-sectionally at parental diet at the time of child dietary assessment and very few studies have data on paternal diet. Thus, the present study is unique because it 1) assessed maternal nutrient intake specifically during pregnancy (as well as postnatally and paternal intake at a similar time), 2) prospectively assessed parental and offspring nutrient intakes, and 3) separately measured maternal prenatal, maternal postnatal, and paternal nutrient intakes for comparisons. This article suggests that, in addition to targeting dietary habits in childhood (of both parents and child), as recommended from previous studies (39, 40), interventions targeting maternal dietary habits in pregnancy or preconceptually might be considered to establish good dietary habits in both parents and offspring as early as possible. Prenatal diet was measured at only one time point during pregnancy, 32 wk of gestation; therefore, we do not know whether the results are different for early pregnancy. Because diet in early and late pregnancy is strongly and positively correlated (41), we would not expect the results to be markedly different. This requires confirmation in future studies; however, we are not aware of any cohorts of similar size with prospectively assessed dietary data, both at multiple points during pregnancy and in offspring during childhood, together with early postnatal and paternal diet data, as presented here.

Limitations of the unquantified FFQs, used here to assess maternal and partner diets, were discussed in detail previously (19). These include assuming fixed portion sizes and assuming a representative selection of a heterogeneous food group (eg, meat or shellfish) is being eaten. Thus, FFQs generally have limited accuracy in quantifying energy intake and absolute intakes of macronutrients; however, they are suitable for ranking individuals based on dietary intake (42), and FFQs are the most feasible method for a survey of this size. It is possible, however,

# MATERNAL PRENATAL AND OFFSPRING DIETS AND ADIPOSITY

## TABLE 3

Associations of parental diet with offspring diet adjusted for underreporting<sup>1</sup>

		Model 1	Model 2			
Parental dietary intake	$\beta^2$	95% CI	Р	$\beta^3$	95% CI	Р
Difference in offspring energy intake (kJ)						
Energy intake						
Main maternal-paternal comparisons						
z Scores for parental intake						
Maternal prenatal energy (per SD; 1 SD = 1959 kJ)	34.6	-10.2, 79.3	0.1	7.7	-33.8, 49.2	0.7
Paternal energy (per SD; 1 SD = 3045 kJ)	29.6	-14.2, 73.3	0.2	20.6	-20.3, 61.5	0.3
Absolute intake						
Maternal prenatal energy (per 2000 kJ)	33.5	-9.9, 77.0	0.1	7.5	-32.8, 47.8	0.7
Paternal energy (per 2000 kJ)	18.4	-8.9, 45.7	0.2	12.8	-12.7, 38.4	0.3
Mutually adjusted (absolute)						
Maternal prenatal energy (per 2000 kJ)	46.8	-15.9, 109.5	0.1	22.4	-35.7, 80.6	0.4
Paternal energy (per 2000 kJ)	20.5	-7.2, 48.2	0.1	14.5	-11.3, 40.3	0.3
Maternal postnatal energy intake						
Absolute energy intake (per 2000 kJ)	29.9	-6.6, 66.3	0.1	10.1	-23.7, 43.9	0.6
Difference in offspring protein intake (g)						
Protein intake						
Main maternal-paternal comparisons						
z Scores for parental intake						
Maternal prenatal protein (per SD; 1 SD = 18.0 g)	3.30	2.67, 3.92	< 0.001	3.14	2.52, 3.77	< 0.001
Paternal protein (per SD; 1 SD = 27.0 g)	1.77	0.85, 2.69	< 0.001	1.43	0.53, 2.32	0.002
Absolute intake						
Maternal prenatal protein (per 20 g)	3.54	2.86, 4.21	< 0.001	3.37	2.70, 4.04	< 0.001
Paternal protein (per 20 g)	1.25	0.60, 1.90	< 0.001	1.01	0.37, 1.65	0.002
Mutually adjusted (absolute)						
Maternal prenatal protein (per 20 g)	3.37	2.40, 4.34	< 0.001	3.20	2.24, 4.16	< 0.001
Paternal protein (per 20 g)	0.90	0.24, 1.56	0.008	0.71	0.06, 1.35	0.03
Maternal postnatal protein intake						
Absolute protein intake (per 20 g)	2.80	2.21, 3.38	< 0.001	2.78	2.22, 3.35	< 0.001
Difference in offspring fat intake (g)						
Fat intake						
Main maternal-paternal comparisons						
z Scores for parental intake						
Maternal prenatal fat intake (per SD) 1 SD = $22.6$ g	2.23	1.23, 3.24	< 0.001	1.81	0.84, 2.77	< 0.001
Paternal fat intake (per SD) 1 SD = $34.9$ g	0.74	-0.62, 2.09	0.3	0.50	-0.80, 1.80	0.5
Absolute intake		,			,	
Maternal prenatal fat intake (per 20 g)	1.89	1.04, 2.73	< 0.001	1.53	0.71, 2.34	< 0.001
Paternal fat intake (per 20 g)	0.41	-0.34, 1.15	0.3	0.28	-0.44, 0.99	0.5
Mutually adjusted (absolute)					,	
Maternal prenatal fat intake (per 20 g)	2.22	1.01, 3.43	< 0.001	1.83	0.67. 2.99	0.002
Paternal fat intake (per 20 g)	0.21	-0.55, 0.97	0.6	0.11	-0.62, 0.83	0.8
Maternal postnatal fat intake		,			,	
Absolute fat intake (per 20 g)	1.17	0.38, 1.96	0.004	0.95	0.19, 1.71	0.01
Difference in offspring carbohydrate intake (g)	,	0.000, 1000	01001	0.70	0.17, 1771	0101
Carbohydrate intake						
Main maternal-paternal comparisons						
7 Scores for parental intake						
Maternal prenatal carbohydrate (per SD: 1 SD – 60.9 g)	4 20	0.88 7.52	0.01	4 91	175 807	0.002
Paternal carbohydrate (per SD; $1 \text{ SD} = 00.9 \text{ g})$	2.04	-0.63, 4.71	0.01	1.98	-0.58, 4.54	0.002
Absolute intake	2.04	0.05, 4.71	0.1	1.90	0.50, 4.54	0.1
Maternal prenatal carbohydrate (per 50 g)	3 24	0.68 5.80	0.01	3 78	135 622	0.002
Paternal carbohydrate (per 50 g)	0.80	-0.28 2.06	0.01	0.86	-0.25 1.08	0.002
Mutually adjusted (absolute)	0.07	0.20, 2.00	0.1	0.00	0.20, 1.70	0.1
Maternal prenatal carbohydrate (per 50 g)	3 77	-0.43 6.86	0.08	3.68	0 10 7 17	0.04
Paternal carbohydrate (per 50 g)	0.87	-0.30 2.04	0.08	0.82	-0.30 1.04	0.04
Maternal postnatal carbohydrata intelea	0.07	0.50, 2.04	0.1	0.02	0.50, 1.94	0.1
Absolute carbohydrate intake (ner 50 g)	3 58	1 41 5 75	0.001	3 57	1.50 5.64	0.001
nosonae caroonyarate marke (per 50 g)	2.20	1.11, 0.10	0.001	2.21	1	0.001

 $^{1}$  n = 5718 for maternal prenatal, 3009 for paternal, 2968 for mutually adjusted models, and 5179 for maternal diet at 47 mo.

 <sup>2</sup> Linear regressions adjusted for maternal/paternal energy intake, maternal/paternal underreporting, and child underreporting.
<sup>3</sup> Linear regressions adjusted for maternal/paternal energy intake, maternal/paternal dietary underreporting, child underreporting, maternal/paternal height, child height at 10 y, social class, maternal education, paternal education, maternal smoking, paternal smoking, and parity.

# TABLE 4

Associations of parental and offspring macronutrient and energy intakes stratified by offspring sex<sup>1</sup>

		Male offspring		Female offspring		
Parental intake	β	95% CI	Р	β	95% CI	Р
Offspring energy (kJ)						
Maternal prenatal energy intake (per 2000 kJ)	-9.0	-66.7, 48.6	0.8	30.1	-18.3, 78.6	0.2
Paternal energy intake (per 2000 kJ)	8.5	-27.4, 44.3	0.6	20.1	-10.8, 51.0	0.2
Offspring protein (g)						
Maternal prenatal protein intake (per 20 g)	3.34	2.36, 4.32	< 0.001	3.14	2.28, 3.99	< 0.001
Paternal protein intake (per 20 g)	1.62	0.71, 2.53	< 0.001	0.51	-0.31, 1.33	0.2
Offspring fat intake (g)						
Maternal prenatal fat intake (per 20 g)	1.31	0.12, 2.49	0.03	1.98	0.92, 3.04	< 0.001
Paternal fat intake (per 20 g)	0.53	-0.49, 1.55	0.3	0.03	-0.92, 0.99	0.9
Offspring carbohydrate (g)						
Maternal prenatal carbohydrate (per 50 g)	4.30	0.81, 7.80	0.02	3.06	0.02, 6.09	0.05
Paternal carbohydrate intake (per 50 g)	0.76	-0.89, 2.41	0.4	1.31	-0.03, 2.66	0.06

<sup>I</sup> Male, n = 2828 (maternal) and 1514 (paternal); female, n = 2889 (maternal) and 1495 (paternal). Linear regressions were adjusted for maternal/paternal energy, maternal/paternal dietary underreporting, child underreporting, maternal/paternal height, child height at 10 y, social class, maternal education, paternal education, maternal smoking, paternal smoking, and parity.

that the use of fixed portion sizes may have affected the results if the chosen difference between males and females was incorrect. In both sexes, error from fixed portion would be expected to be nondifferential with respect to a child's diet at 10 y; therefore, this would be expected to attenuate any true association. If the error for men is greater than that for women, it may explain, in part, the difference in associations between maternal and paternal nutrient intakes. previously (20). These limitations include inaccuracies in estimated portion sizes and underreporting of intake. However, nutrient intakes in this cohort were comparable with those of children in the British National Diet and Nutrition Survey based on 7-d weighed dietary records (43).

Dietary assessments relying on participant reporting tend to result in underreporting, particularly among heavier individuals. Our findings suggest that 43.3%, 33.3%, 0.7%, and 32.6% of the mothers prenatally, mothers postnatally, fathers, and offspring, respectively, underreported their total energy intake. When

Limitations of diet diaries, which were used in the present study to assess the children's' diet, have also been discussed

#### TABLE 5

Associations of child macronutrient and energy intakes with body composition<sup>1</sup>

		Model 1			Model 2			
Child dietary intake	$\beta^2$	95% CI	Р	$\beta^{3}$	95% CI	Р		
Difference in child fat mass (SD)								
Child energy intake (SD)	1.36	1.24, 1.48	< 0.001	1.35	1.22, 1.47	< 0.001		
Child protein intake (SD)	0.10	0.07, 0.12	< 0.001	0.10	0.07, 0.13	< 0.001		
Child fat intake (SD)	0.12	0.00, 0.24	0.05	0.09	-0.03, 0.21	0.1		
Child carbohydrate intake (SD)	-0.48	-0.64, -0.33	< 0.001	-0.45	-0.61, -0.30	< 0.001		
Mutually adjusted <sup>4</sup>								
Child protein intake (SD)	0.14	0.11, 0.16	< 0.001	0.14	0.12, 0.17	< 0.001		
Child fat intake (SD)	0.47	0.38, 0.56	< 0.001	0.44	0.36, 0.53	< 0.001		
Child carbohydrate intake (SD)	0.38	0.27, 0.48	< 0.001	0.39	0.29, 0.49	< 0.001		
Difference in child lean mass (SD)								
Child energy intake (SD)	0.47	0.38, 0.56	< 0.001	0.47	0.38, 0.56	< 0.001		
Child protein intake (SD)	0.05	0.03, 0.07	< 0.001	0.05	0.03, 0.07	< 0.001		
Child fat intake (SD)	-0.20	-0.29, -0.12	< 0.001	-0.21	-0.29, -0.12	< 0.001		
Child carbohydrate intake (SD)	0.07	-0.04, 0.18	0.2	0.07	-0.04, 0.18	0.2		
Mutually adjusted <sup>4</sup>								
Child protein intake (SD)	0.07	0.05, 0.09	< 0.001	0.07	0.05, 0.09	< 0.001		
Child fat intake (SD)	0.00	-0.06, 0.06	0.99	0.00	-0.06, 0.06	0.9		
Child carbohydrate intake (SD)	0.26	0.19, 0.33	< 0.001	0.26	0.19, 0.33	< 0.001		

 $^{1}$  n = 5725. Lean mass was adjusted for total fat mass. Protein, fat, and carbohydrate were also adjusted for energy.

<sup>2</sup> Linear regressions adjusted for dietary underreporting, height, and height squared at 9 and 11 y.

<sup>3</sup> Linear regressions adjusted for dietary underreporting, height, height squared, social class, maternal education, paternal education, maternal smoking, paternal smoking, and parity.

<sup>4</sup> Protein, fat, and carbohydrate adjusted for one another.

Associations of parental macronutrient and energy intakes with child body composition<sup>1</sup>

		Model 1		Model 2		
Parental dietary intake	$\beta^2$	95% CI	Р	$\beta^{3}$	95% CI	Р
Difference in child fat mass (SD)						
Main maternal-paternal comparisons						
Maternal prenatal energy (per 2000 kJ)	-0.01	-0.05, 0.02	0.4	0.00	-0.04, 0.03	0.8
Paternal energy intake (per 2000 kJ)	-0.04	-0.06, -0.02	< 0.001	-0.04	-0.06, -0.02	< 0.001
Maternal prenatal protein (per 20 g)	0.00	-0.04, 0.04	0.9	0.04	-0.01, 0.08	0.1
Paternal protein intake (per 20 g)	0.01	-0.03, 0.05	0.6	0.02	-0.02, 0.06	0.3
Maternal prenatal fat intake (per 20 g)	0.06	0.01, 0.10	0.02	0.04	0.00, 0.09	0.08
Paternal fat intake (per 20 g)	0.01	-0.03, 0.05	0.7	0.00	-0.04, 0.04	0.99
Maternal prenatal carbohydrate (per 50 g)	-0.06	-0.11, -0.01	0.02	-0.07	-0.12, -0.02	0.005
Paternal carbohydrate intake (per 50 g)	0.02	0.00, 0.04	0.07	0.02	0.00, 0.05	0.04
Maternal postnatal diet						
Maternal energy intake (per 2000 kJ)	-0.02	-0.05, 0.00	0.08	-0.01	-0.04, 0.01	0.3
Maternal protein intake (per 20 g)	0.10	0.06, 0.13	< 0.001	0.10	0.07, 0.14	< 0.001
Maternal fat intake (per 20g)	-0.03	-0.07, 0.01	0.1	-0.04	-0.08, 0.00	0.03
Maternal carbohydrate intake (per 50 g)	0.02	-0.02, 0.06	0.3	0.02	-0.02, 0.06	0.4
Difference in child lean mass (SD)						
Main maternal-paternal comparisons						
Maternal prenatal energy (per 2000 kJ)	-0.01	-0.03, 0.01	0.3	-0.01	-0.03, 0.01	0.2
Paternal energy intake (per 2000 kJ)	0.00	-0.01, 0.01	0.9	0.00	-0.01, 0.01	0.9
Maternal prenatal protein intake (per 20 g)	0.02	0.00, 0.05	0.08	0.03	0.00, 0.06	0.02
Paternal protein intake (per 20 g)	0.02	-0.01, 0.04	0.2	0.02	-0.01, 0.02	0.4
Maternal prenatal fat intake (per 20 g)	-0.02	-0.05, 0.01	0.3	-0.02	-0.05, 0.01	0.2
Paternal fat intake (per 20 g)	-0.02	-0.05, 0.01	0.1	-0.02	-0.05, 0.00	0.1
Maternal prenatal carbohydrate (per 50 g)	-0.01	-0.04, 0.03	0.7	-0.01	-0.04, 0.03	0.8
Paternal carbohydrate intake (per 50 g)	0.00	-0.02, 0.02	0.9	0.00	-0.02, 0.02	0.9
Maternal postnatal diet						
Maternal energy intake (per 2000 kJ)	-0.01	-0.03, 0.00	0.09	-0.01	-0.03, 0.01	0.2
Maternal protein intake (per 20 g)	0.03	0.01, 0.06	0.003	0.04	0.02, 0.06	0.001
Maternal fat intake (per 20 g)	-0.03	-0.05, 0.00	0.05	-0.03	-0.06, 0.00	0.03
Maternal carbohydrate intake (per 50 g)	0.01	-0.02, 0.04	0.4	0.01	-0.02, 0.04	0.4

 $^{1}$  n = 5534 (maternal prenatal), 2942 (paternal), and 5593 (maternal 47 mo).

<sup>2</sup> Linear regressions adjusted for maternal/paternal energy, maternal/paternal underreporting, child height, and height squared at 9 and 11 y.

<sup>3</sup> Linear regressions adjusted for maternal/paternal energy, maternal/paternal underreporting, maternal/paternal height, child height, height squared, social class, maternal education, paternal education, maternal smoking, paternal smoking, and parity.

underreporting was taken into account, it had an important effect on the main results. As stated in Subjects and Methods, prepregnancy BMI was used to estimate prenatal and postnatal BMR (and thus, energy underreporting) because BMIs at these time points were unavailable. However, because BMR was estimated by using the Schofield equation for adult women, we believe this was a reasonable approach. Whereas some misclassification in energy underreporting may have occurred because of the use of prepregnancy BMI, because maternal prenatal and postnatal size are generally strongly and positively correlated, the effect of this misclassification will be the same at both time points and should not explain differences in associations for prenatal and postnatal nutrient intakes.

Finally, although we wanted to compare maternal nutrient intakes in pregnancy with paternal intakes at the same time point, we only had data for partners' dietary intakes at 47 mo. Our assumption was that this was a reasonable approximation of father's dietary intake at the time of pregnancy. In fact, if having a child results in all family members eating more commonly together, father's diet may be more assimilated to the mothers by 47 mo than during pregnancy. Indeed, correlations between maternal and paternal nutrient intakes were higher for maternal postnatal diet than for maternal prenatal diet. As such, the association of mother's macronutrient intake in pregnancy with offspring macronutrient intake (if acting via intrauterine mechanisms) may be even stronger than that of father's macronutrient intake with offspring's intake than our results suggest.

In this contemporary British population, there was evidence that maternal macronutrient intake during pregnancy has a stronger influence on child macronutrient intake than does maternal intake postnatally or paternal macronutrient intake. This could reflect in utero programming of offspring appetite by maternal diet during pregnancy. Encouraging pregnant women to engage in healthy dietary behaviors may be of benefit to the development of the fetus and to later dietary habits of their children.

We are extremely grateful to all the families who took part in this study, the midwives for their help in recruiting them, and the ALSPAC team, which includes interviewers, computer and laboratory technicians, clerical workers, research scientists, volunteers, managers, receptionists, and nurses.

The authors' responsibilities were as follows—DAL: conceived the idea for this manuscript and directed the analysis and the writing of the manuscript; M-JAB: analyzed the data, wrote the first draft, coordinated subsequent versions, and completed the final version of the manuscript; PE, IR, and VC: contributed to the collection and processing of dietary data; and DAL, GDS, ARN, IR, PE, and VC: provided feedback on earlier versions of the manuscript and contributed to the final manuscript. None of the authors had any financial or personal interest in any of the companies or organizations sponsoring this research. None of the authors declared a conflict of interest.

#### REFERENCES

- 1. Boden G. Fuel metabolism in pregnancy and in gestational diabetes mellitus. Obstet Gynecol Clin North Am 1996;23:1–10.
- Pedersen J. Weight and length at birth of infants of diabetic mothers. Acta Endocrinol (Copenh) 1954;16:330–42.
- Freinkel N. Banting lecture 1980. Of pregnancy and progeny. Diabetes 1980;29:1023–35.
- Freinkel N, Metzger BE. Pregnancy as a tissue culture experience: the critical implications of maternal metabolism for fetal development. In: Ciba Foundation, ed. Pregnancy metabolism, diabetes and the fetus. Amsterdam, Netherlands: Ciba Foundation, 1979:3–23. (Ciba Foundation Symposium no. 63.)
- Franke K, Harder T, Aerts L, et al. 'Programming' of orexigenic and anorexigenic hypothalamic neurons in offspring of treated and untreated diabetic mother rats. Brain Res 2005;1031:276–83.
- Muhlhausler BS, Adam CL, Marrocco EM, et al. Impact of glucose infusion on the structural and functional characteristics of adipose tissue and on hypothalamic gene expression for appetite regulatory neuropeptides in the sheep fetus during late gestation. J Physiol 2005;565: 185–95.
- Muhlhausler BS, Adam CL, Findlay PA, Duffield JA, McMillen IC. Increased maternal nutrition alters development of the appetiteregulating network in the brain. FASEB J 2006;20:1257–9.
- Jovanovic-Peterson L, Peterson CM, Reed GF, et al. Maternal postprandial glucose levels and infant birth weight: the Diabetes in Early Pregnancy Study. The National Institute of Child Health and Human Development–Diabetes in Early Pregnancy Study. Am J Obstet Gynecol 1991;164:103–11.
- Combs CA, Gunderson E, Kitzmiller JL, Gavin LA, Main EK. Relationship of fetal macrosomia to maternal postprandial glucose control during pregnancy. Diabetes Care 1992;15:1251–7.
- Major CA, Henry MJ, De VM, Morgan MA. The effects of carbohydrate restriction in patients with diet-controlled gestational diabetes. Obstet Gynecol 1998;91:600–4.
- Jones HN, Powell TL, Jansson T. Regulation of placental nutrient transport–a review. Placenta 2007;28:763–74.
- Layman DK, Clifton P, Gannon MC, Krauss RM, Nuttall FQ. Protein in optimal health: heart disease and type 2 diabetes. Am J Clin Nutr 2008; 87(suppl):1571S–5S.
- 13. Riccardi G, Giacco R, Rivellese AA. Dietary fat, insulin sensitivity and the metabolic syndrome. Clin Nutr 2004;23:447–56.
- Smith GD. Assessing intrauterine influences on offspring health outcomes: can epidemiological studies yield robust findings? Basic Clin Pharmacol Toxicol 2008;102:245–56.
- Brion M-JA, Leary SD, Smith GD, Ness AR. Similar associations of parental prenatal smoking suggest child blood pressure is not influenced by intrauterine effects. Hypertension 2007;49:1422–8.
- Leary S, Davey Smith G, Ness A. Smoking during pregnancy and components of stature in offspring. Am J Hum Biol 2006;18:502–12.
- Lawlor DA, Timpson N, Harbord R, et al. Exploring the developmental overnutrition hypothesis using parental-offspring associations and the FTO gene as an instrumental variable for maternal adiposity: findings from the Avon Longitudinal Study of Parents and Children (ALSPAC). PLoS Med 2008;5:e33.
- Golding J, Pembrey M, Jones R. ALSPAC—the Avon Longitudinal Study of Parents and Children. I. Study methodology. Paediatr Perinat Epidemiol 2001;15:74–87.

- Rogers I, Emmett P. Diet during pregnancy in a population of pregnant women in South West England. ALSPAC Study Team. Avon Longitudinal Study of Pregnancy and Childhood. Eur J Clin Nutr 1998;52: 246–50.
- Glynn L, Emmett P, Rogers I. Food and nutrient intakes of a population sample of 7-year-old children in the south-west of England in 1999/2000 —what difference does gender make? J Hum Nutr Diet 2005;18:7–19.
- 21. The Royal Society of Chemistry and MAFF. Meat products and dishes. London, United Kingdom: RSC/MAFF, 1996.
- 22. The Royal Society of Chemistry and MAFF. The composition of foods. London, United Kingdom: HMSO, 1991.
- The Royal Society of Chemistry and MAFF. Cereals and cereal products. London, United Kingdom: RSC/MAFF, 1988.
- The Royal Society of Chemistry and MAFF. Milk products and eggs. London, United Kingdom: RSC/MAFF, 1989.
- The Royal Society of Chemistry and MAFF. Vegetables, herbs and spices. London, United Kingdom: RSC/MAFF, 1991.
- The Royal Society of Chemistry and MAFF. Fruit and nuts. London, United Kingdom: RSC/MAFF, 1992.
- The Royal Society of Chemistry and MAFF. Vegetable Dishes. London, United Kingdom: RSC/MAFF, 1992.
- The Royal Society of Chemistry and MAFF. Fish and fish products. London, United Kingdom: RSC and HMSO, 1995.
- 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;26:146–54.
- Department of Health Report on Health and Social Subjects. Dietary reference values for food, energy and nutrients for the United Kingdom. London, United Kingdom: HMSO, 1991.
- 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;54:386–94.
- Torun B. Energy requirements of children and adolescents. Public Health Nutr 2005;8:968–93.
- Nord RH, Payne RK. Body composition by dual-energy x-ray absorptiometry—a review of the technology. Asia Pac J Clin Nutr 1995;4: 167–71.
- OPCS. Standard occupational classification. London, United Kingdom: HMSO, 1991.
- Willett W, Stampfer M. Implications of total energy intake for epidemiologic analyses. 2nd ed. In: Willet W, ed. Nutritional epidemiology. New York, NY: Oxford University Press, 1998:273–301.
- Steer CD. Comment: Clemons T. A look at the inheritance of height using regression toward the mean. Hum Biol 2000;72:447–54. 2009.
- Clemons T. A look at the inheritance of height using regression toward the mean. Hum Biol 2000;72:447–54.
- Oliveria SA, Ellison RC, Moore LL, Gillman MW, Garrahie EJ, Singer MR. Parent-child relationships in nutrient intake: the Framingham Children's Study. Am J Clin Nutr 1992;56:593–8.
- van der Horst K, Oenema A, Ferreira I, et al. A systematic review of environmental correlates of obesity-related dietary behaviors in youth. Health Educ Res 2007;22:203–26.
- Campbell KJ, Crawford DA, Ball K. Family food environment and dietary behaviors likely to promote fatness in 5-6 year-old children. Int J Obes 2006;30:1272–80.
- Crozier SR, Robinson SM, Godfrey KM, Cooper C, Inskip HM. Women's dietary patterns change little from before to during pregnancy. J Nutr 2009;139:1956–63.
- Bingham SA, Gill C, Welch A, et al. Comparison of dietary assessment methods in nutritional epidemiology: weighed records v. 24 h recalls, food-frequency questionnaires and estimated-diet records. Br J Nutr 1994;72:619–43.
- Gregory J, Lowy C. National Diet and Nutrition Survey: young people aged 4 to 18 years. London, United Kingdom: The Stationery Office, 2000.