PUBLISHED VERSION

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Nutrition and Diabetes, 2015; 5(1):e150-1-e150-7

Originally published at:
http://doi.org/10.1038/nutd.2014.47

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Citation: Nutrition & Diabetes (2015) **5**, e150; doi:10.1038/nutd.2014.47

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ORIGINAL ARTICLE

Behavioural early-life exposures and body composition at age 15 years

SD Leary[1], DA Lawlor[2], G Davey Smith[2], MJ Brion[2] and AR Ness[1]

BACKGROUND/OBJECTIVES: Previous studies have demonstrated associations between some early-life exposures and later obesity, but most have used body mass index in childhood or adulthood as the outcome. The objective of this study was to investigate whether early-life exposures were associated with directly measured fat and lean mass in adolescence.

SUBJECTS/METHODS: This study used data on 4750 mother–offspring pairs, collected as a part of the Avon Longitudinal Study of Parents and Children, Bristol, UK between 1991 and 1992; associations between behavioural exposures occurring from conception up to 5 years of age (maternal and paternal smoking during pregnancy, breastfeeding, age at introduction to solids, dietary patterns and physical inactivity during early childhood) and offspring body composition measured by dual-energy X-ray absorptiometry at ~15 years were assessed.

RESULTS: After full adjustment for potential confounders, maternal smoking during pregnancy, having a junk food diet and spending more time watching television in early childhood were all associated with higher fat mass at age 15, whereas maternal smoking, having a healthy diet and playing computer games more frequently in early childhood were all associated with a higher lean mass at age 15. Associations with paternal smoking were generally weaker for both fat and lean mass, but as there was no strong statistical evidence for maternal vs paternal differences, confounding by social factors rather than a direct effect of maternal smoking cannot be ruled out. Early feeding was not associated with fat or lean mass at age 15.

CONCLUSIONS: This study does not provide compelling evidence for associations between most early-life factors and body composition in adolescence. However, possible associations with dietary patterns and physical inactivity in early childhood require further investigation in other cohorts that have direct measurements of adolescent body composition.

Nutrition & Diabetes (2015) **5,** e150; doi:10.1038/nutd.2014.47; published online 9 February 2015

INTRODUCTION

The prevalence of overweight and obesity has rapidly increased in recent years, and according to the latest Health Survey for England,[1] 63% of adults (aged 16 or over) and 30% of children (aged 2–15) are overweight or obese. Obese children and adolescents have been found to have risk factors for cardiovascular disease such as hyperlipidemia, hypertension and abnormal glucose tolerance,[2] as well as an increased risk of asthma, hepatic steatosis, sleep apnoea and type 2 diabetes.[3] Longer-term risks include adult obesity, ischaemic stroke, joint disease, cancer and coronary heart disease, in addition to the chronic conditions mentioned above.[3,4]

It has been suggested that fetal life, infancy and early childhood may be particularly sensitive periods for predicting obesity.[5] Many previous studies have investigated the role of early-life (from conception to 5 years of age) risk factors for later obesity and overweight, and Monasta *et al.*[6] have undertaken a review of published systematic reviews of these studies. In addition, Reilly *et al.*[7] investigated 25 potential early-life risk factors for obesity at age seven in the Avon Longitudinal Study of Parents and Children (ALSPAC). These studies were mainly based on body mass index (BMI) which is known not to be a good measure of adiposity as it cannot distinguish between fat and lean mass,[8] and few recorded the outcomes in adolescents.

Body composition data, that is, fat and lean mass derived from dual-energy X-ray absorptiometry at ~15 years old are now available in ALSPAC. Therefore the objective of this study was to use ALSPAC to investigate whether associations between a number of behavioural early-life exposures and body composition at age 15 years exist. The choice of early-life exposures was based on those identified from the Monasta[6] and Reilly[7] papers which are available within ALSPAC and considered to be behavioural and therefore have the potential to be modified. They include parental smoking in pregnancy, early feeding (breastfeeding and age at introduction of solids), dietary patterns in early childhood (derived using principal components analysis), and physical inactivity in early childhood (time spent in the car, watching television and playing computer games). For all analyses, the effect of confounding was explored by adjustment for a wide range of potentially confounding factors in regression models. In addition, for smoking, the associations between maternal and paternal exposures and the outcomes were compared; a stronger association observed with maternal compared with paternal smoking is more likely to represent a causal intrauterine influence, whereas similar associations with both maternal and paternal smoking suggest that confounding by family level environment and/or genetic factors is a likely explanation.[9]

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Received 17 November 2014; accepted 7 December 2014

SUBJECTS AND METHODS

Sample and study design

ALSPAC is a geographically based birth cohort investigating the health and development of children, which is described in detail elsewhere.[10] Briefly, all pregnant women living in three health districts of Bristol (formerly known as the Avon Health Area), UK with expected delivery dates between 1 April 1991 and 31 December 1992 were eligible to take part in the study. A total of 14 541 were enrolled and 13 678 had a singleton, live born child. Detailed data have been collected by self-completed questionnaires (relating to the mother, her partner and her offspring) from pregnancy onwards. From the age of seven, all children have been invited to regular research clinics. Ethical approval was obtained from the ALSPAC Ethics and Law Committee and the Local Research Ethics Committees. The study website contains details of all the data that are available through a fully searchable data dictionary (www.bris.ac.uk/alspac/researchers/data-access/data-dictionary).

Early-life exposure variables

A list of all early-life exposure variables and the time point of measurement is shown in Table 1. At enrolment, the mother was asked whether her partner was the father of her unborn child. All variables relating to the partner were set to missing if the mother answered 'no' or 'not sure', or did not answer this question, to allow estimation of the contribution of factors that are potentially shared (either through genetics or environment) between father and offspring. In the 18-week antenatal questionnaire, the mother was asked whether she smoked tobacco (1) in the first 3 months of pregnancy and (2) in the past 2 weeks. Positive responses (cigarettes, cigars, pipes or 'other') were grouped together to create dichotomous variables to represent smoking in the first and second trimesters, respectively. In the 32-week antenatal questionnaire, the mother was asked how many cigarettes she was currently smoking per day, and this was categorized into a dichotomous variable to represent smoking in the third trimester. Responses from the three trimesters were combined to create a variable for any smoking during pregnancy. In the 18-week-gestation questionnaires, both the mother and father were asked if he had smoked regularly over the past 9 months; the father's response was used if available, otherwise the mother's response about the father was used. [11]

From the 6-month maternal questionnaire, a variable was derived for exclusive breastfeeding, coded as exclusive breastfeeding beyond 2 months of age, partial breastfeeding (breastfeeding had been stopped or was non-exclusive by 2 months) and never breastfed. Exclusive breastfeeding was defined as no solids, milk formulas or other drinks, except vitamins, minerals, medicines and/or water. The mother was also asked to record the age in months when her child was introduced to solids, which was grouped into $\leqslant 2$, 3 and $\geqslant 4$ months of age.

When the child was ~38 months old, the mother was asked to complete a food frequency questionnaire about their eating habits. A principal components analysis identified four main factors, which were assigned labels that were thought to capture the key features of each pattern; these were junk (fizzy drinks, sweets and confectionary, chocolate, chips, fried foods, sausages, burgers, crisps, takeaway meals, pot noodles, cook in sauces, bread, pizza, biscuits and flavoured milk), healthy (pulses, vegetarian foods, rice, pasta, salad, fruit juice, fruit, water, eggs, cheese and fish), traditional (meat and vegetables as meals, meat, poultry, root vegetables, potatoes, green vegetables and legumes) and fussy or snack (puddings, cakes and buns, biscuits, squash, crisps, cheese and fruit).[12] The child's energy intake was also estimated from the food frequency questionnaires using the fifth edition of McCance and Widdowson's The Composition of Foods,[13] based on age-appropriate standard portion sizes.

Also in the 38-month questionnaire, the mother was asked how much time her child spent in the car and watching television on both weekdays and weekends. Variables were derived for the total time spent in the car per week (grouped into <4, 4–5 and >5 h) and total time spent watching television per week (grouped into $\leqslant 4$, 4.1–8 and >8 h per week). In the 57-month questionnaire, the mother was asked to indicate how often her child played computer games, which was grouped into rarely/not at all, once a month, once a week and two to seven times per week.

Outcome variables at age 15

At the 15-year clinic visit, total fat and total lean mass were measured using a Lunar Prodigy DXA scanner (GE Medical Systems Lunar, Madison, WI, USA); the scans were visually inspected and any automated sectioning that did not match the true body sections was manually realigned. Height was measured with shoes and socks removed using a Harpenden stadiometer (Holtain Ltd, Crymych, Pembrokeshire, UK), and weight was measured using a Tanita TBF 305 body fat analyser and weighing scales (Tanita United Kingdom Ltd., Yewsley, Middlesex, United Kingdom); from these BMI was calculated as weight (in kilos) divided by height squared (in metres).

Potential confounding factors

At enrolment, the mother was asked to record her parity, height and pre-pregnancy weight, and BMI was calculated from these. The father was asked to record his height and weight (used to calculate BMI). The 32-week antenatal questionnaire asked the mother to record her highest education level which was then categorized into none/CSE (national school exams at age 16), vocational, O level (national school exams at age 16, higher than CSE), A level (national school exams at age 18) or university degree. At this time, she also recorded the occupation of both herself and the father; the lowest was used to allocate them to social class groups (classes I (highest occupational social class) to V (lowest)) using the 1991 OPCS (Office of Population Censuses and Surveys) classification. The mother's age at delivery was calculated from the date of delivery and the mother's date of birth recorded at enrolment. The estimation of gestation was based on the date of the last menstrual period reported by the mother. Offspring gender and birthweight were recorded in the delivery room and abstracted from obstetric records and/or birth notifications.

Children were sent a puberty questionnaire, which included questions on developmental stage[14] after they had made an appointment for the 15-year clinic visit with the intention that they completed it before their appointment. Pubertal stage for males was based on pubic hair development and for females was based on the most advanced stage for pubic hair and breast development; data were set to missing if the puberty questionnaire was not completed within 16 weeks of the dual-energy X-ray absorptiometry scan (this was only the case for two children).

Statistical analyses

Means and s.d. were calculated for continuous variables that were approximately normally distributed, medians and interquartile ranges for skewed variables (fat mass and BMI at 15 years only), and proportions for categorical variables. Further analysis was based on internally derived s.d. scores for total fat mass, lean mass and BMI to allow comparison of the regression coefficients between the outcome measures. These were calculated by subtracting the mean from the individual's value, then

Table 1. Timing of measurement of early-life exposure variables

Early-life exposure	*Time point*			
	In utero	*Birth to 6 months*	*38 months*	*57 months*
Maternal smoking	×			
Paternal smoking	×			
Breastfeeding		×		
Age at introduction to solids		×		
Dietary patterns (junk, health, traditional, fussy/snack)			×	
Time spent in car			×	
Time spent watching television			×	
Frequency playing computer games				×

dividing by the s.d.; the mean and s.d. were based on the whole sample. Logged fat mass and logged BMI were used to calculate the s.d. scores due to the skewness of the distribution.

Associations between each of the exposures (maternal smoking, paternal smoking, breastfeeding, age at introduction to solids, four dietary patterns at 38 months, time spent in the car at 38 months, time spent watching television at 38 months and frequency of playing computer games at 57 months) and the main outcomes (fat mass s.d. score, lean mass s.d. score) were assessed using linear regression. For each of the 11 exposures, adjustment was first made for gender and age at the time of body composition measurement (plus energy intake at 38 months for the four dietary patterns; Model 1), then additionally for parental factors (maternal and paternal height, maternal and paternal BMI, maternal age, parity), social factors (social class, maternal education) plus birthweight and gestation (Model 2). All the models were adjusted for height to take into account differences in stature.

As well as separate models with maternal or paternal smoking as the exposure, additional models were fitted with maternal and paternal smoking variables included simultaneously; statistical tests were used to compare the maternal and paternal regression coefficients. Also, even though paternal data were set to missing if the mother had not confirmed her partner to be the true biological father of her child, a sensitivity analysis was carried out to assess the potential effects of various possible rates of unknown non-paternity of partners on the results for parental smoking; for fully adjusted simultaneous models; the Clemons command in STATA[15,16] was used.

Associations in males and females separately were compared by including interaction terms for gender and exposure variables in the models. The minimally adjusted models were repeated restricting to those with complete confounder information, to ensure that any changes in regression coefficients were attributable to confounding rather than missing data. The fully adjusted models were repeated with additional adjustment for pubertal status. Finally, models were repeated with BMI s.d. score as the outcome. All the analyses were performed in Stata version 12.1 (StataCorp, College Station, TX, USA).

RESULTS

Data on 5112 were available from the 15-year clinic visit. Analysis was based on the 4750 with at least one early-life exposure plus offspring body composition at age 15. Partner data were set to missing for 2.4% of the 4634 with at least one variable recorded, as the partner had not been confirmed as the biological father of the child. Tables 2a and b, display the summary data for all the exposures (except factor loadings for dietary patterns at 38 months), outcomes and potential confounders. For interpretation of the regression coefficients generated from the models below, s.d. were 0.6 kg on the logarithmic scale for fat mass, and, as shown in Table 2a, 8.4 kg for lean mass (0.16 kg m^{-2} for BMI).

Table 2a. Summary of exposure/outcome/confounder data available on the 4750 used for analysis—continuous variables

	N	*Mean*	*s.d.*
Outcomes			
Fat mass at 15-year clinic visit (kg)[a]	4750	13.7	8.3, 20.1
Lean mass at 15-year clinic visit (kg)	4750	43.1	8.4
BMI (kg m^{-2})[a]	4745	20.7	19.0, 23.0
Confounders			
Maternal height (cm)	4560	164.4	6.7
Maternal pre-pregnancy BMI (kg m^{-2})	4337	22.8	3.7
Paternal height (cm)	3472	176.5	6.8
Paternal BMI at time of pregnancy (kg m^{-2})	3448	25.1	3.2
Maternal age in pregnancy (years)	4750	29.2	4.5
Gestation (weeks)	4750	39.5	1.8
Birthweight (kg)	4689	3.4	0.5
Energy intake at 38 months (kJ per day)	4281	5206.9	1240.1
Age at 15-year clinic visit (years)	4750	15.5	0.3
Height at 15-year clinic visit (cm)	4748	169.3	8.4

Abbreviation: BMI, body mass index. [a]Median and interquartile range.

Confounder data for the 4750 included in this analysis were compared with confounder data for the 8928 singleton liveborns who did not have their body composition at age 15 recorded (see Supplementary Tables 1a and b); for most variables, there were only modest differences between the two groups.

Parental smoking

Associations between the parental smoking exposures and fat and lean mass at age 15 are shown in Table 3; there were no interactions with gender for either of the exposures ($P > 0.4$ for all).

Maternal and paternal smoking were both associated with higher fat mass at age 15 after minimal adjustment for confounders, and the effect sizes were similar. After full adjustment for potential confounders, the association with maternal smoking remained, whereas that with paternal smoking was weakened; however, there was no statistical evidence for a difference in associations ($P = 0.6$). Refitting the minimally adjusted models restricting to those with complete confounder data only, or adding pubertal status to fully adjusted models made little difference to the findings (see Supplementary Table 2). In the fully adjusted simultaneous model also adjusted for a hypothetical non-paternity rate as high as 20%,

Table 2b. Summary of exposure/outcome/confounder data available on the 4750 used for analysis—categorical variables

	Categories	N	%
Exposures			
Maternal smoking in pregnancy	No	3503	81.5
	Yes	794	18.5
Paternal smoking in pregnancy	No	3106	69.0
	Yes	1694	31.0
Breastfeeding	Exclusive	1604	36.4
	Partial	2182	49.5
	None	621	14.1
Age at introduction of solids	⩽2 months	598	13.3
	3 months	2572	57.3
	⩾4 months	1317	29.4
Time spent in the car at 38 months	<4 h per week	2957	68.3
	4–5 hours per week	846	19.5
	>5 h per week	526	12.2
Time spent watching television at 38 months	⩽4 h per week	1011	23.4
	4.1–8 h per week	2072	47.9
	>8 h per week	1242	28.7
Frequency of playing computer games at 57 months	Rarely/not at all	1755	41.5
	Once a month	736	17.4
	Once a week	959	22.7
	2–7 times per week	776	18.4
Confounders			
Parity	0	2277	49.2
	1	1599	34.6
	⩾2	750	16.2
Social class in pregnancy	V	121	2.7
	IV	565	12.7
	III manual	1123	25.2
	III non-manual	1235	27.7
	II	1218	27.4
	I	190	4.3
Maternal education	None/CSE	511	11.1
	Vocational	361	7.8
	O levels	1604	34.7
	A levels	1313	28.4
	Degree	837	18.1
Gender	Male	2267	47.7
	Female	2483	52.3
Pubertal status at age 15 years	Stage I/II/III	180	4.4
	Stage IV	1691	41.1
	Stage V	2240	54.5[a]

[a]43.4% of the males and 64.4% of the females were stage V.

Table 3. Associations between maternal/paternal smoking at the time of pregnancy and body composition at age 15

	Minimally adjusted[a]			*Fully adjusted*[b]		
	β	*(95% CI)*	P-*value*	β	*(95% CI)*	P-*value*
Outcome = fat mass at 15 years (s.d. score)						
Separately						
Maternal smoking	0.16[c]	0.09, 0.22	< 0.001	0.16	0.07, 0.24	< 0.001
Paternal smoking	0.15	0.09, 0.20	< 0.001	0.08	0.02, 0.14	0.01
Simultaneously						
Maternal smoking	0.10	0.03, 0.18	0.01	0.13	0.05, 0.22	0.003
Paternal smoking	0.13	0.07, 0.19	< 0.001	0.06	− 0.01, 0.13	0.08
			0.6[d]			0.3[d]
Outcome = lean mass at 15 years (s.d. score)						
Separately						
Maternal smoking	0.08[e]	0.05, 0.12	< 0.001	0.07	0.02, 0.12	0.01
Paternal smoking	0.03	− 0.0003, 0.06	0.05	0.02	− 0.01, 0.06	0.2
Simultaneously						
Maternal smoking	0.08	0.04, 0.12	< 0.001	0.06	0.01, 0.11	0.01
Paternal smoking	0.01	− 0.02, 0.04	0.7	0.01	− 0.03, 0.05	0.7
			0.02[d]			0.1[d]

Abbreviation: CI, confidence interval. [a]Adjusted for gender, age at the time of 15-year clinic visit, height. [b]Additionally adjusted for parental factors, social factors, birthweight and gestation. [c]For interpretation, fat mass 10% higher (e.g., 1.2 kg for geometric mean fat mass), if mother smoked. [d]*P*-value for comparison of maternal and paternal regression coefficients. [e]For interpretation, lean mass 0.7 kg higher, if mother smoked.

the associations with paternal smoking strengthened, whereas those with maternal smoking remained similar (see Supplementary Table 3; $P = 0.3–0.8$ for differences in associations between maternal and paternal smoking).

Maternal smoking was more strongly associated with higher lean mass at age 15 years than paternal smoking, although the statistical evidence for a difference was not strong ($P = 0.02$ for the minimally adjusted model, $P = 0.1$ for the fully adjusted model). Again, findings were similar after restricting minimally adjusted models to those with complete confounder information only, or adding pubertal status to fully adjusted models (see Supplementary Table 2). Adjusting for non-paternity made little difference to the findings (see Supplementary Table 3).

Feeding in infancy, and diet and physical inactivity in early childhood

Associations between each of the exposures in infancy/early childhood and fat and lean mass at age 15 are shown in Table 4; there were no interactions with gender for any of these exposures ($P > 0.1$ for all). Although several exposures (not breastfeeding, earlier age at introduction to solids, eating more junk food, eating less healthy food, more time spent in the car and more time spent watching television) were associated with higher fat mass at 15 years after only minimal adjustment, once all potential confounders were included in the models the only associations that remained were with the junk dietary pattern and time spent watching television.

There was only modest evidence for higher lean mass at age 15 if more time was spent playing computer games (before and after adjustment for potential confounders). The associations between lean mass and dietary patterns in early childhood were complex; a positive association was seen with the traditional dietary pattern, but only before adjustment for all confounders, whereas there was an inverse association with the junk pattern, and a positive association with the healthy pattern, but only after adjustment for all confounders.

Findings for both fat mass and lean mass were generally similar to the above if minimally adjusted models were restricted to only those with complete confounder information, or fully adjusted models additionally included pubertal status (see Supplementary Table 4).

All exposures

All the 11 exposures were then included in fully adjusted models simultaneously (Table 5). The findings were generally similar to those of the separate fully adjusted models (Tables 3 and 4). Maternal smoking, the junk dietary pattern and time spent watching television were all associated with higher fat mass at age 15. Maternal smoking, the healthy dietary pattern and playing computer games were all associated with a higher lean mass at age 15; the inverse association with the junk pattern was substantially weakened when all the exposures were considered simultaneously. The association with maternal smoking was stronger for fat mass than for lean mass, and although for both outcomes, associations with paternal smoking were weaker than for maternal smoking, there was no statistical evidence of a difference ($P = 0.3$ for fat mass and $P = 0.2$ for lean mass).

As shown in Table 2b, 18.5% of the mothers smoked at some point during pregnancy. Considering each trimester separately, 17.0% smoked in the first, 13.2% in the second and 12.6% in the third trimester, with only 7.2% changing their smoking habits during pregnancy. For both fat and lean mass, associations were almost identical to the combined smoking variable for the first and second trimesters, whereas the association was marginally stronger for the third trimester (see Supplementary Table 5; simultaneous adjustment for all exposures).

Finally, all the 11 exposures were included in fully adjusted models simultaneously, but with BMI s.d. score as the outcome (Supplementary Table 6). No early-life factors were clearly associated with BMI at age 15; the apparent association between maternal smoking in pregnancy and higher BMI was not found to be statistically different from the association with paternal smoking ($P = 0.2$).

DISCUSSION

This study, on the basis of a large, contemporary cohort, has examined associations between behavioural factors occurring between conception and 5 years of age, and directly measured fat and lean mass at age 15, taking into account a wide range of potential confounders. Maternal smoking during pregnancy, having a junk food diet and spending more time watching television at

Table 4. Associations between feeding in infancy and diet and physical inactivity in early childhood, and body composition at age 15

Separate models	*Minimally adjusted*[a]			*Fully adjusted*[b]		
	β	*(95% CI)*	P-*value*	β	*(95% CI)*	P-*value*
Outcome = fat mass at 15 years (s.d. score)						
Breastfeeding (vs exclusive)						
Partial	0.06	0.01, 0.11		0.03	−0.04, 0.09	
None	0.16	0.08, 0.24	<0.001	0.06	−0.04, 0.16	0.2
Age at introduction to solids(vs ⩽2 months)						
3 m	−0.04	−0.11, 0.04		−0.01	−0.10, 0.08	
⩾4 m	−0.11	−0.19, −0.03	0.003	−0.03	−0.13, 0.07	0.5
Dietary pattern at 38 months (factor loading): junk	0.11	0.08, 0.14	<0.001	0.06	0.02, 0.10	0.002
Dietary pattern at 38 months (factor loading): healthy	−0.04	−0.07, −0.01	0.003	−0.01	−0.03, 0.03	0.9
Dietary pattern at 38 months (factor loading): traditional	0.03	−0.001, 0.5	0.06	0.003	−0.03, 0.03	0.8
Dietary pattern at 38 months (factor loading): fussy/snack	−0.04	−0.07, −0.01	0.01	−0.01	−0.04, 0.03	0.8
Time spent in car at 38 months (vs <4 h per week)						
4–5 h per week	0.04	−0.02, 0.10		0.01	−0.06, 0.08	
>5 h per week	0.12	0.04, 0.19	0.003	0.10	0.01, 0.19	0.06
Time spent watching television at 38 months (vs ⩽4 h per week)						
4.1–8 h per week	0.12	0.06, 0.19		0.07	−0.003, 0.14	
>8 h per week	0.22	0.15, 0.29	<0.001	0.16	0.08, 0.24	<0.001
Frequency playing computer games at 57 months (vs rarely/not at all)						
Once a month	0.01	−0.06, 0.08		−0.02	−0.10, 0.06	
Once a week	0.10	0.03, 0.16	0.08	0.06	−0.01, 0.14	
2–7 times per week	0.03	−0.05, 0.10		0.02	−0.06, 0.10	0.2
Outcome = lean mass at 15 years (s.d. score)						
Breastfeeding (vs exclusive)						
Partial	0.01	−0.02, 0.04		0.01	−0.03, 0.04	
None	0.02	−0.03, 0.06	0.4	−0.02	−0.08, 0.03	0.6
Age at introduction to solids(vs ⩽2 months)						
3 m	−0.02	−0.06, 0.02		−0.0005	−0.05, 0.05	
⩾4 m	−0.005	−0.05, 0.04	0.9	0.03	−0.02, 0.09	0.1
Dietary pattern at 38 months (factor loading): junk	−0.001	−0.02, 0.02	0.6	−0.04	−0.06, −0.01	0.002
Dietary pattern at 38 months (factor loading): healthy	0.01	−0.001, 0.03	0.07	0.03	0.01, 0.05	0.002
Dietary pattern at 38 months (factor loading): traditional	0.02	0.01, 0.04	0.003	0.01	−0.01, 0.02	0.4
Dietary pattern at 38 months (factor loading): fussy/snack	−0.004	−0.02, 0.01	0.7	0.01	−0.01, 0.03	0.3
Time spent in car at 38 months (vs <4 h per week)						
4–5 h per week	−0.01	−0.04, 0.03		−0.01	−0.06, 0.03	
>5 h per week	0.02	−0.02, 0.06	0.5	0.02	−0.03, 0.07	0.7
Time spent watching television at 38 months (vs ⩽4 h per week)						
4.1–8 h per week	0.001	−0.03, 0.04		−0.02	−0.06, 0.02	
>8 h per week	−0.003	−0.04, 0.04	0.9	−0.04	−0.08, 0.01	0.1
Frequency playing computer games at 57 months (vs rarely/not at all)						
Once a month	0.002	−0.04, 0.04		0.004	−0.04, 0.05	
Once a week	0.03	−0.002, 0.07	0.02	0.03	−0.01, 0.08	
2–7 times per week	0.04	−0.001, 0.08		0.04	−0.01, 0.09	0.04

Abbreviation: CI, confidence interval. [a]Adjusted for gender, age at the time of 15-year clinic visit, height (plus energy intake at 38 months for dietary patterns). [b]Additionally adjusted for parental factors, social factors, birthweight and gestation.

approximately age three were all associated with higher fat mass at age 15. Maternal smoking, having a healthy diet at approximately age three and playing computer games more frequently at approximately five were all associated with a higher lean mass at age 15. Associations with paternal smoking were generally weaker than those for maternal smoking for both fat and lean mass at age 15, but as there was no strong statistical evidence for maternal vs paternal differences, confounding by social factors rather than a direct effect of maternal smoking cannot be ruled out (see Taylor *et al.*[17] for an explanation of the maternal/paternal negative control approach). Early feeding (breastfeeding and age at introduction to solids) was not associated with fat or lean mass at age 15 after adjusting for confounders.

The main strengths of the current study are its size, the direct measurement of body composition and the rigorous analyses undertaken such as adjustment for a wide range of confounders and assessment of the potential effects of non-paternity when comparing maternal and paternal effects. However, this study also has a number of limitations. It is possible that different results would have been obtained if all children whose mothers originally enrolled in the study were able to be included in the analysis. However, only modest differences in most characteristics can be seen between

Table 5. Associations between all the exposures simultaneously, and body composition at age 15

Simultaneous fully adjusted models[a]	*Outcome = fat mass at 15 years (s.d. score)*			*Outcome = lean mass at 15 years (s.d. score)*		
	β	*(95% CI)*	P-*value*	β	*(95% CI)*	P-*value*
Maternal smoking	0.13	0.03, 0.23	0.01	0.08	0.02, 0.13	0.01
Paternal smoking	0.05	−0.02, 0.13	0.2	0.02	−0.02, 0.06	0.4
Breastfeeding (vs exclusive)						
Partial	−0.003	−0.07, 0.07		0.01	−0.03, 0.05	
None	0.01	−0.10, 0.12	0.8	0.003	−0.06, 0.07	0.6
Age at introduction to solids (vs ⩽2 months)						
3 m	−0.003	−0.11, 0.10		−0.02	−0.08, 0.04	
⩾4 m	0.004	−0.11, 0.12	0.8	0.01	−0.06, 0.08	0.5
Dietary pattern at 38 months (factor loading): junk	0.06	0.01, 0.11	0.02	−0.02	−0.05, 0.005	0.1
Dietary pattern at 38 months (factor loading): healthy	0.02	−0.02, 0.06	0.3	0.03	0.01, 0.06	0.004
Dietary pattern at 38 months (factor loading): traditional	0.03	−0.01, 0.06	0.1	0.01	−0.01, 0.03	0.3
Dietary pattern at 38 months (factor loading): fussy/snack	0.03	−0.01, 0.08	0.2	0.02	−0.01, 0.05	0.2
Time spent in car at 38 months (vs <4 h per week)						
4–5 h per week	−0.02	−0.10, 0.06		−0.005	−0.05, 0.04	
>5 h per week	0.05	−0.06, 0.15	0.5	0.02	−0.04, 0.08	0.6
Time spent watching television at 38 months (vs ⩽4 h per week)						
4.1–8 h per week	0.04	−0.04, 0.11		−0.01	−0.06, 0.03	
>8 h per week	0.11	0.02, 0.21	0.01	−0.03	−0.08, 0.02	0.3
Frequency playing computer games at 57 months (vs rarely/not at all)						
Once a month	−0.03	−0.12, 0.06		0.005	−0.05, 0.06	
Once a week	0.05	−0.03, 0.13		0.03	−0.02, 0.07	
2–7 times per week	−0.01	−0.10, 0.08	0.7	0.05	−0.002, 0.11	0.05

Abbreviation: CI, confidence interval. [a]Adjusted for gender, age at the time of 15-year clinic visit, height, parental factors, social factors, birthweight and gestation (plus energy intake at 38 months for dietary patterns).

those in the current analysis, and those who did not attend the clinic. In addition, findings were similar if the minimally adjusted analyses were restricted to those with complete data on all confounders rather than including any with available data, providing some reassurance that attrition is unlikely to have biased results.

The smoking data were self-reported by mothers and their partners, however, a meta-analysis of comparisons between biochemical measures and self-reported smoking found the self-reported data to be accurate. [18] Early feeding data were also self-reported, but recall bias should have been minimal as the information was obtained from the mothers regularly throughout early infancy. Due to the large number of subjects, it was necessary to use unquantified food frequency questionnaires to assess diet in early childhood. Although food frequency questionnaires are a less accurate method of dietary assessment than using weighed dietary records,[19] they have been shown to provide a reasonable measure of diet in large cohorts[20] and may, in fact, estimate intakes of less commonly eaten foods more accurately than dietary records. The physical inactivity variables were reported by mothers which was not ideal, but there was no robust practical method of objectively measuring physical activity/inactivity on large numbers of young children within the epidemiological setting.

We have demonstrated associations between maternal smoking and offspring fat and lean mass at age ~15 years, although as there is no statistical evidence that associations with maternal smoking are stronger than those with paternal smoking, confounding is a likely explanation. Although many studies have demonstrated associations between maternal smoking during pregnancy and offspring overweight and obesity (for example, Reilly *et al.*[7] and relevant reviews from Monasta *et al.*[6]), to our knowledge, the only previous study to investigate associations between both maternal and paternal smoking and fat or lean mass in the offspring was also based on ALSPAC,[21] with outcomes assessed at an earlier age when the children were ~10 years old. Although it is not possible to directly compare the effect sizes between these two studies (as internally derived s.d. scores are not comparable as the variation in body composition widens as children get older), overall conclusions were similar.

We did not find any associations between breastfeeding or the age at introduction to solids and fat or lean mass at age 15 after adjustment for confounders. Previous studies have reported inconsistent associations between breastfeeding and offspring overweight/obesity at various ages (for example, Reilly *et al.*[7] and several reviews identified by Monasta *et al.*[6]), however, the general pattern seems to be that any observed associations are substantially weakened or removed after adjustment for confounding factors. A small number of studies have investigated breastfeeding and dual-energy X-ray absorptiometry measured body composition in adolescence, and most did not find associations with either fat mass[22–24] or lean mass[22–25] in offspring aged between 16 and 18 years after adjusting for confounders. Further evidence that there is no causal association is provided by a randomized breastfeeding promotion intervention in Belarus which did not reduce child obesity.[26]

Despite the large number of studies addressing the issue of breastfeeding and offspring obesity, very few have investigated associations with the age at introduction of solids, and these are mainly limited to BMI in childhood as the outcome. Yang and Huffman[27] and Przyrembel[28] have reviewed the existing evidence and concluded that findings were mixed, and adjustment was not always made for confounders. In addition, Reilly *et al.*[7] did not observe an association between the age at introduction to solids and BMI at age seven in ALSPAC.

We have demonstrated positive associations between having a junk food diet at age three and increased fat mass at age 15, and between having a healthy diet at age three and increased lean mass at age 15; no associations between other dietary patterns in

early childhood (healthy, traditional, fussy/snack) and offspring fat mass/BMI were observed. Although Reilly *et al.*[7] found an association between the junk food diet at age three and BMI in 5-year-old ALSPAC children, to our knowledge, there are no studies investigating these types of dietary patterns in early childhood and adolescent body composition.

We have found increased time spent watching television and time spent playing computer games in early childhood to be associated with increased fat mass and increased lean mass at age 15, respectively, whereas there were no associations with time spent in the car in early childhood. Reilly *et al.*[7] have shown time spent watching television but not time spent in the car to be associated with BMI at 5 years in ALSPAC, and also relevant reviews from Monasta *et al.*[6] (52 studies assessing time spent watching television and six assessing time spent playing computer games) showed associations with BMI at age 3–18 years. To our knowledge, no studies have investigated associations between these physical inactivity variables in early childhood and offspring fat or lean mass. Our observed association between increased time spent playing computer games in infancy and lean mass in adolescence is difficult to explain and may be a chance finding, so requires replication.

So in conclusion, this study does not provide compelling evidence that parental smoking or early feeding are associated with directly measured body composition in adolescence. There is some suggestion that associations with dietary patterns and physical inactivity in early childhood may exist, but further studies are required to confirm our findings in different cohorts, particularly as our study is one of the first to examine associations with directly measured body composition in adolescence. If associations with these early-life exposures are shown to exist, the future direction of research should then focus on whether these deleterious effects can be overcome by improved diet and physical activity in adolescence.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

ACKNOWLEDGEMENTS

We are extremely grateful to all the families who took part in this study, the midwives for their help in recruiting them and the whole ALSPAC team, which includes interviewers, computer and laboratory technicians, clerical workers, research scientists, volunteers, managers, receptionists and nurses. This publication is the work of the authors and Sam Leary, Debbie Lawlor, George Davey Smith, Marie-Jo Brion and Andy Ness will serve as guarantors for the contents of this paper. This work was supported by the European Union 6th framework programme, Early Nutrition Programming Project EARNEST (FOOD-CT-2005-007036). The UK Medical Research Council and the Wellcome Trust (Grant ref: 092731) and the University of Bristol provide core support for ALSPAC.

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Supplementary Information accompanies this paper on the Nutrition & Diabetes website (http://www.nature.com/nutd)