RESEARCH REPORT

Does birth weight predict childhood diet in the Avon longitudinal study of parents and children?

W A Shultis, S D Leary, A R Ness, C J Bain, P M Emmett, and the ALSPAC Study Team

Study objective: Low birth weight predicts cardiovascular disease in adulthood, and one possible explanation is that children with lower birth weight consume more fat than those born heavier. Therefore, the objective of this study was to investigate associations between birth weight and childhood diet, and in particular, to test the hypothesis that birth weight is inversely related to total and saturated fat intake.

Design: Prospective cohort study.

Setting: South west England.

Participants: A subgroup of children enrolled in the Avon longitudinal study of parents and children, with data on birth weight and also diet at ages 8, 18, 43 months, and 7 years (1152, 998, 848, and 771 children respectively).

Main results: Associations between birth weight and diet increased in strength from age 8 to 43 months, but had diminished by age 7 years. Fat, saturated fat, and protein intakes were inversely, and carbohydrate intake was positively associated with birth weight at 43 months of age, after adjusting for age, sex, and energy intake. After adjustment for other confounders, all associations were weakened, although there was still a suggestion of a relation with saturated fat ( –0.48 [95% CI] –0.97, 0.02) g/day per 500 g increase in birth weight. Similar patterns were seen in boys and girls separately, and when the sample was restricted to those with complete data at all ages.

Conclusions: A small inverse association was found between birth weight and saturated fat intake in children at 43 months of age but this was not present at 7 years of age. This study therefore provides little evidence that birth weight modifies subsequent childhood diet.

Low birth weight and small size at birth are predictors of increased cardiovascular disease and type 2 diabetes in adulthood, and also of risk factors for cardiovascular disease and diabetes. For example, in the nurses health study, adjusted relative risks of non-fatal cardiovascular disease were 1.49 in the lowest category of birth weight (<2568 g) compared with 0.68 in the highest category (>4536 g). In children, both insulin levels and blood pressure have been associated with birth weight, suggesting differences in risk profiles appear early in life.

Proposed explanations for the association between low birth weight and cardiovascular disease include fetal programming, where undernutrition at critical time periods in development programmes structural and physiological changes such as glucose-insulin and lipid metabolism, and blood pressure regulation, leading to later risk of cardiovascular disease. In animals, such fetal programming of body size and metabolic processes by undernutrition or nutrient manipulation is well reported, but the relevance to chronic disease risk in humans is disputed. An alternative explanation for the association is that genetically determined insulin resistance leads to impaired insulin mediated fetal growth, resulting in low birth weight and continuing insulin resistance in later life. Although shared confounding may also contribute to this association, as both low birth weight and cardiovascular disease are associated with deprivation and poor socioeconomic position, studies that have reported associations are robust to adjustment for measures of social position. Finally, early diet has also been suggested as an explanation for the birth weight-cardiovascular disease associations, in so far as low birth weight could influence early food preferences or change the way parents feed their children. Evidence of such an association between birth weight and early diet was published by Stafford and Lucas, using data from the UK national diet and nutrition survey of children aged 1.5 to 4.5 years. They found an inverse relation between birth weight and energy adjusted fat intake, after adjustment for age, social class, and current weight. This effect was seen only in boys, with those in the lowest fifth of birth weight consuming on average 2.3 g or 86.5 KJ more fat (adjusted for energy) per day than those in the highest fifth.

In this analysis we have repeated and extended the findings of Stafford and Lucas in a well characterised cohort of children, the Avon longitudinal study of parents and children (ALSPAC). A particular advantage of ALSPAC is that data on birth weight, gestational age, repeated measures of diet and weight, and parental factors are available, permitting a more detailed investigation of the effect of birth weight on early diets. We aimed to test the association reported by Stafford and Lucas within different age groups to provide richer information on the pattern of the birth weight-diet relation with time.

METHODS

The ALSPAC study is a prospective geographically based cohort of 14 541 pregnant women and their offspring. The study has been described in detail previously (http://www.alspac.bris.ac.uk/). Briefly, the study aimed to recruit all pregnant women living in the Avon area with expected delivery dates between April 1991 and December 1992. An estimated 85% of the pregnant population participated. Ethical approval for the study was obtained from the ALSPAC ethics and law sub-committee and the local medical research ethics committees. Extensive data have been collected from the whole cohort, from pregnancy onwards. For this analysis we have used birth records and parental and child questionnaires. As well as data collected on the whole...
cohort, 10% of the cohort (1432 children, of which 1394 were singletons) were randomly selected at birth from those born between June and December 1992 to form the children in focus (CIF) subgroup, and were followed up in more detail from birth to age 5 years. Among other variables, weight and three day dietary records were collected at 8, 18, 43 months of age on the CIF subgroup only. At age 7 years the entire cohort was invited to attend a research clinic, where weight and diet were similarly collected. To date however, dietary records at 7 years have only been coded for the CIF group, and this analysis has therefore been restricted to these children.

Parental data
The parental data used in this analysis were collected from the whole cohort by questionnaire during the study pregnancy. Maternal height and the height of her partner (to 1 cm) were self reported. Mid-parental height was calculated as the average of maternal and paternal height. Educational achievement was coded by occupation as class I (highest), II, III non-manual, III-manual, IV or V (lowest).\(^1\)\(^8\) Educational achievement was coded as degree and above, A level, O level, vocational, and CSE or none and this was treated as an ordered hierarchical variable with degree and above being the highest category and CSE or none being the lowest.

Birth measures
Birth weight was extracted from routine hospital birth records. Gestational age was estimated based on the date of the last menstrual period and findings from the routine (<20 weeks) antenatal ultrasound scan. Birth order was calculated by subtracting the number of known miscarriages and terminations from the number of pregnancies that had resulted in live or stillbirths, using information from the maternal questionnaires completed during pregnancy.

### Three day dietary records
Three day dietary records were collected when the children were 8, 18, and 43 months and 7 years of age. Structured dietary diaries with instructions on how to complete them were posted out to study families in the weeks preceding their clinic visit. Parents were asked to record their study child’s diet on one weekend day and two weekdays. They were asked to record all foods and drinks consumed in as much detail as possible including brand names and portion sizes in household measures. While the families were in the research clinic, trained staff checked the dietary diaries and clarified any recipes, unknown foods, or missing details to improve accuracy and completeness.

Dietary diaries were coded using the diet in data out (DIDO) software.\(^1\)\(^9\) Portion sizes were based on UK Ministry of Agriculture, Fisheries and Foods portion sizes (second edition),\(^2\)\(^0\) and from manufacturers product labels. Where no child portion sizes were available they were estimated from adult portion sizes. Nutrient values were generated using an in-house software program containing a food database derived from the McCance and Widdowson’s food tables (5th edition) and supplements,\(^2\)\(^1\) and additional manufacturers information. Foods missing from the food tables were estimated by the study nutritionists. More detailed accounts of ALSPAC dietary methods have been published previously.\(^2\)\(^2\)\^-\(^2\)\(^4\) In these papers underreporting and over-reporting of energy intake was investigated at 8, 18, and 43 months. Few children were categorised as underreporters or overreporters of energy intake.

### Table 1 (A) Distribution of potential confounding factors among those included in basic models compared with those who were not included (all are singletons in the children in focus subgroup of ALSPAC)

<table>
<thead>
<tr>
<th>Variable</th>
<th>Included in basic models* (Max N = 1278)</th>
<th>Excluded from basic models (Max N = 116)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Continuous variables</td>
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<td></td>
</tr>
<tr>
<td>Gestational age (weeks)</td>
<td>1278 39.5 0.05</td>
<td>116 39.4 0.2</td>
</tr>
<tr>
<td>Mid-parental height (cm)([^\dagger])</td>
<td>898 170.8 0.3</td>
<td>44 171.7 1.4</td>
</tr>
<tr>
<td>Categorical variables</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Maternal social class: % class I and II</td>
<td>413 39.6</td>
<td>24 33.4</td>
</tr>
<tr>
<td>Paternal social class: % class I and II</td>
<td>541 48.7</td>
<td>29 37.7</td>
</tr>
<tr>
<td>Maternal highest education: % degree</td>
<td>182 14.7</td>
<td>11 12.0</td>
</tr>
<tr>
<td>Paternal highest education: % degree</td>
<td>259 21.6</td>
<td>14 16.1</td>
</tr>
<tr>
<td>Birth order: % firstborn</td>
<td>579 46.6</td>
<td>46 46.0</td>
</tr>
</tbody>
</table>

*Restricted to those with information on age, sex, birth weight, and dietary intake at each age. \(^\dagger\) Value obtained from \(t\) tests (continuous variables) or \(\chi^2\) tests (categorical variables). \([^\dagger]\)Mid-parental height was calculated as the average of maternal and paternal height.

### Table 1 (B) Distribution of potential confounding factors among those included in the complete data analysis compared with those who were not included (all are singletons in the children in focus subgroup of ALSPAC)

<table>
<thead>
<tr>
<th>Variable</th>
<th>Included in complete data analysis* (Max N = 369)</th>
<th>Excluded from complete data analysis (Max N = 1025)</th>
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</thead>
<tbody>
<tr>
<td>Continuous variables</td>
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<tr>
<td>Gestational age (weeks)</td>
<td>369 39.5 0.08</td>
<td>1025 39.5 0.05</td>
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<tr>
<td>Mid-parental height (cm)([^\dagger])</td>
<td>369 170.7 0.4</td>
<td>573 170.9 8.7</td>
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<tr>
<td>Categorical variables</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Maternal social class: % class I and II</td>
<td>155 42.0</td>
<td>282 37.9</td>
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<tr>
<td>Paternal social class: % class I and II</td>
<td>198 53.7</td>
<td>372 45.4</td>
</tr>
<tr>
<td>Maternal highest education: % degree</td>
<td>79 21.4</td>
<td>114 11.9</td>
</tr>
<tr>
<td>Paternal highest education: % degree</td>
<td>102 27.6</td>
<td>171 18.6</td>
</tr>
<tr>
<td>Birth order: % firstborn</td>
<td>193 52.3</td>
<td>432 44.4</td>
</tr>
</tbody>
</table>

*Restricted to those with information on diet and confounders at all four ages. \(^\dagger\) Value obtained from \(t\) tests (continuous variables) or \(\chi^2\) tests (categorical variables). \([^\dagger]\)Mid-parental height was calculated as the average of maternal and paternal height.
or overreporters, and the exclusion of these children had limited effect on mean energy intakes. Therefore in this analysis all children with dietary data at 8, 18, and 43 months were included. The underreporting or overreporting of energy intake at 7 years has yet to be investigated and all CIF children with dietary data at this age were included in this analysis.

**Anthropometry in childhood**

Weight at 8, 18, and 43 months was measured to 0.1 kg using Tanita scales (Tanita UK, Yewsley, UK). Children were examined in light clothing and all measurements were made by trained research staff.

**Statistical analysis**

Data were analysed using the Stata statistical software (Stata version 8, Stata Corp, College Station, TX), restricting to singleton births only. The distribution of each variable and of the residuals was checked for normality and transformations were not deemed necessary. Summary statistics for each variable were calculated in the whole sample, and in boys and girls separately. Sex differences were assessed using $t$ tests for continuous variables and $\chi^2$ tests for categorical variables. The distribution of potential confounding factors among those included in analysis and those not included were also compared using $t$ tests and $\chi^2$ tests.

Relations between birth weight and diet were explored using multiple linear regression, with birth weight as the predictor, and either energy, total fat, saturated fat, carbohydrate, or protein (at each age) as the outcome. All models were adjusted for age, sex, and energy (basic model). To control for confounding, models were then additionally adjusted for gestational age, parental social class, parental education, birth order, and mid-parental height (adjusted model 1). Finally, current weight at the relevant age was also added to each model (adjusted model 2). Dietary variables were used as continuous terms for all models, and for the primary analysis, birth weight was also used as a continuous term. Regression coefficients were reported per 500 g (roughly one standard deviation) of birth weight. Analysis was repeated on boys and girls separately, and interaction terms were fitted in the models to test for sex differences. Analysis was also repeated after restricting to those with complete dietary and confounder data at all ages.

Although for the primary analysis, birth weight was included as a continuous term to permit full use of the data, a second analysis was undertaken in which birth weight was categorised to enable comparison with analysis by Stafford and Lucas. The relation between birth weight and diet was examined across four categories of birth weight: $\leq 2500$ g, $2501–3000$ g, $3001–3500$ g, and $3501–4000$ g. Arbitrary categories were used rather than quantiles to ease replication of our analysis by other groups, and a $\leq 2500$ g category was not included as numbers were too few. Basic and both adjusted models were fitted for the whole sample.

**RESULTS**

Data on birth weight, sex, age, and diet (basic model) were available for 1278 singletons, of which 54% were boys and 46% girls. Of these, 1152 had data at 8 months; 998 at 18 months; 848 at 43 months; and 771 at 7 years of age. Complete data on gestational age, parental social class,
parental education, birth order, and mid-parental height (adjusted model 1) were available for 677 children at 8 months, 610 at 18 months, 527 at 43 months, and 497 at 7 years. Complete diet and confounder data at each of the four ages were available for 369 children. Children included in the basic models were similar to those excluded with respect to gestational age, birth order, social position, and mid-parental height (table 1A). Children included in the complete data analysis were more likely to be first born and come from more affluent and better educated families, although they were similar in other respects to those excluded from this analysis (table 1B). Birth weight, weight (except at 7 years), and all dietary intakes (except saturated fat intake at 7 years) were higher in boys than in girls. Gestational age, mid-parental height, parental social class, parental education, and birth order were similar for each sex (table 2).

Table 3 shows the regression of energy and macronutrient intakes on birth weight, based on all children for which data at any age were available. The basic models (age, sex, and energy adjusted) showed that total energy and carbohydrate intake tended to be positively related to birth weight, while fat, saturated fat, and protein intakes tended to be inversely related to birth weight. These effects increased with age from 8 to 7 months, but had decreased by 7 years. Adjustment for gestational age, parental social class, parental education,
mid-parental height, and birth order reduced birth weight effects, such that the only remaining relation were with saturated fat intake at 43 months, and energy intake up to 43 months. Additional adjustment for current weight attenuated effect size estimates further. The same analyses are presented for those with complete diet and confounder data at all ages in table 4, and similar patterns were seen in these children.

Similar patterns were observed when the sexes were analysed separately, although birth weight effects were generally stronger in the boys. For example, saturated fat intake at 43 months reduced by 0.93 (0.50 to 1.35)g/day for boys and 0.32 (0.21 to 0.86)g/day for girls per 500 g increase in birth weight (p = 0.1 for difference in slopes). After adjustment (model 1), these effects attenuated to reductions of 0.62 (−0.03 to 1.27)g/day for boys and 0.02 (−0.78 to 0.82)g/day for girls per 500 g increase in birth weight (p = 0.03 for difference in slopes).

When birth weight was analysed as a categorical variable, associations were again strongest at 43 months of age. For example, in the basic model, daily saturated fat intake at 43 months reduced by 0.50 (−0.46, 1.47)g/day, 0.85 (−0.15, 1.84)g/day and 2.25 (1.05, 3.45)g/day compared with the baseline as the birth weight categories increased (p for trend <0.001). However, after adjustment (model 1), effect sizes were attenuated to reductions of 0.39 (−0.91, 1.68)g/day, 0.40 (−0.99, 1.78)g/day and 1.18 (−0.53, 2.89)g/day across increasing categories of birth weight compared with the baseline (p for trend = 0.2).

**DISCUSSION**

Our data, based on a subgroup of children from the ALSPAC study, do not suggest that there are important associations between birth weight and childhood diet. Although we found inverse associations between birth weight and fat, saturated fat, and protein intake (adjusted for energy), and a positive association between birth weight and carbohydrate intake (adjusted for energy), at 43 months of age, much of this was explained by confounding factors. After adjustment for gestational age, social position, birth order, and mid-parental height, suggestion of an association only remained for saturated fat intake. Although associations were present at 43 months, they were not present at 7 years of age.

Our findings lend little support to those from a previous cross sectional study in preschool children, where an inverse association with fat intake was seen across categories of birth weight in boys. In our longitudinal study, data at 18 and 43 months most closely resembled the age range in the Stafford and Lucas study, and we have shown the strongest effects of birth weight at 43 months. In the Stafford and Lucas study, birth weight effects on diet were only seen in boys, although the authors do not present formal tests for differences in relations between the sexes. In our study, we found stronger effects of birth weight on diet in boys than girls, although differences were not significant. The smaller effect size estimates in our analysis compared with those of Stafford and Lucas may be partly explained by a shortfall in poorer families and the possible effect of selective loss to follow up in our study. Our assessment of influence of birth weight at discrete ages should have allowed for a more accurate assessment of the effect of birth weight on childhood diet. We were also able to extend our analysis to look at the effects of other macronutrients.

The possibility that our findings were chance, bias, or residual confounding cannot be discounted. The effect sizes we found were small and the confidence intervals were large. Residual confounding by gestational age, social position, mid-parental height, birth order, or current weight and other factors could partly explain results. Adjustment for these factors greatly attenuated effect sizes, and despite the fact that attention was paid to the quality of measurement, these may have been made with error. The number of children with complete data to age 7 years was modest in comparison with the total number of children in our study, and our analysis restricted to these children assumed their data to be “missing completely at random”, which we know not to be the case. Hence bias attributable to selective loss to follow up could have weakened our results. However, the measurement of diet carries with it a degree of imprecision, and the true effect size of birth weight on macronutrient intakes could therefore be larger than that seen here. Difficulties in capturing the greater variability in the diet at age 7 years, reduced parental control of diet, and the reduced awareness of foods and drinks consumed at school, may partly explain why effect size estimates were smaller at this age, although there may simply be no effect of birth weight on diet at age 7 years.

The potential mechanisms by which lower birth weight may influence the diet of young children, in particular higher total and saturated fat intakes, are not well described. It may be that lower birth weight programmes or influences early food preferences. Alternatively parents of lower birth weight children may chose to feed them more foods to encourage them to grow larger, such as meat and milk, which are richer in fat, although we are not aware of any evidence of this. In our analysis we adjusted for mid-parental height as a proxy for expected offspring size, but having taller parents did not appreciably change the association between birth weight and fat intakes, making this latter explanation less likely. Parents may want larger boys and smaller girls, and therefore feed their children according, but the stronger birth weight effects on diet seen in our boys were not significantly different to the effects seen in the girls. Apart from the results of Stafford and Lucas (1998), sex differences would not be expected, as birth weight predicts cardiovascular disease risk in both men and women. For example, in a recent meta-regression analysis of the association between birth weight and systolic blood pressure in later life, there was no difference in the birth weight effect between the sexes. The implications of these findings at present are limited as the observed effect sizes were small and the confidence intervals wide, and the effect was only present at 43 months of age.

**Key points**

- Our data suggest that there is a small inverse association between birth weight and saturated fat intake in children at 43 months of age.
- Although the association was stronger in boys than girls, differences did not reach statistical significance.
- After adjustment for potential confounders, birth weight was not associated with any other dietary component, and there were no associations at 8 months, 18 months, or 7 years of age.
ACKNOWLEDGEMENTS

We are grateful to all the parents and children 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. ALSPAC is part of the World Health Organisation initiated European longitudinal study of parents and children.

Authors’ affiliations
W A Shultis, C J Bain, Department of Social Medicine, University of Bristol, Bristol, UK
S D Leary, A R Ness, P M Emmett, Unit of Paediatric and Perinatal Epidemiology, Department of Community-Based Medicine, University of Bristol
C J Bain, School of Population Health, University of Queenslad, Australia

Funding: WAS was supported by a PhD studentship from the Medical Research Council, UK, and CJB was supported in part by a University of Bristol Leverhulme Visiting Fellowship. The Avon longitudinal study of parents and children has been funded by the Medical Research Council, Department of Health, Department of the Environment, DfEE, National Institutes of Health, and a variety of medical research charities and commercial companies.

Conflicts of interest: none.

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15 Stafford M, Lucas A. Possible association between low birth weight and later heart disease needs to be investigated further. BMJ 1999;316:1247.