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“Junk food” diet and childhood behavioural problems: Results from the ALSPAC cohort

Nicola J Wiles¹, Kate Northstone², Pauline Emmett², and Glyn Lewis¹

¹Academic Unit of Psychiatry, Department of Community Based Medicine, University of Bristol, UK

²Department of Social Medicine, University of Bristol, UK

Abstract

Objective—To determine whether a “junk food” diet at age 4½ is associated with behavioural problems at age 7.

Subjects and Methods—Data on approximately 4000 children participating in the Avon Longitudinal Study of Parents and Children (ALSPAC), a birth cohort recruited in Avon, UK in 1991/92, were used. Behavioural problems were measured at age 7 using the Strengths & Difficulties Questionnaire (SDQ) (maternal completion). Total difficulties and scores for the 5 sub-scales (hyperactivity, conduct & peer problems, emotional symptoms, & prosocial behaviour) were calculated. Principal components analysis of dietary data (frequency of consumption of 57 foods/drinks) collected at age 4½ by maternal report was used to generate a “junk food” factor. Data on confounders were available from questionnaires.

Results—A one standard deviation increase in “junk food” intake at age 4½ years was associated with increased hyperactivity at age 7 (odds ratio: 1.19 (95% confidence interval: 1.10, 1.29)). This persisted after adjustment for confounders including IQ score (odds ratio: 1.13 (95% confidence interval: 1.01, 1.15)). There was little evidence to support an association between “junk food” intake and overall behavioural difficulties or other sub-scales of the SDQ.

Conclusions—Children eating a diet high in “junk food” in early childhood were more likely to be in the top 33% on the SDQ hyperactivity sub-scale at age 7. This may reflect a long-term nutritional imbalance, or differences in parenting style. This finding requires replication before it can provide an avenue for intervention.

Keywords

ALSPAC; Child Behaviour; Diet

INTRODUCTION

Since the 1980s there has been unease about how dietary factors, particularly additives, may affect children’s behaviour. Narrative reviews conclude that small groups of children may be

Address for correspondence: Dr Nicola J Wiles, Academic Unit of Psychiatry, Department of Community Based Medicine, University of Bristol, Cotham House, Cotham Hill, Bristol, BS6 6JL, UK, Tel: +44 (0)117 954 6676, Fax: +44 (0)117 954 6672, Email: nicola.wiles@bristol.ac.uk.

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affected (Bellisle, 2004; Schnoll et al., 2003) and recent evidence from double-blind placebo randomised controlled trials (RCTs) suggests that artificial food colourings and preservatives may be associated with parental, but not independent, ratings of hyperactivity (Bateman et al., 2004; Schab & Trinh, 2004).

A diet high in processed foods and soft drinks, a “junk food” diet, may lead to peaks and troughs in blood sugar, with associated periods of hyperactivity and lethargy. Whilst sugar intake has been linked with hyperactivity in a number of observational studies, controlled experimental studies do not support an association (Wolraich et al., 1995; Schnoll et al., 2003; Bellisle 2004). Children consuming more “junk food” are likely to have a lower intake of vitamins, minerals and essential fatty acids, particularly omega-3 and omega-6, which are vital building blocks for brain function (Lauritzen et al., 2001). Whilst the link between omega-3 fatty acids and mood is topical (Parker et al., 2006), there is little evidence that dietary supplementation improves behaviour (Voigt et al., 2001; Stevens et al., 2003; Hirayama et al., 2004).

Given a recent trend for poorer nutritional habits (UK Department for Environment, 2005), it is important to examine the link between a “junk food” diet and childhood behaviour. Previous studies are restricted to hyperactive children, those with known food sensitivities or recruited from specialist clinics, and thus results cannot be generalised. The Avon Longitudinal Study of Parents and Children (ALSPAC) provides a rare opportunity to undertake such an investigation within a large population-based cohort. We therefore investigated whether a “junk food” diet at age 4½ is associated with behavioural problems at age 7.

MATERIALS AND METHODS

Avon Longitudinal Study of Parents and Children (ALSPAC)

Full details are published elsewhere (Golding et al., 2001) (www.alspac.bris.ac.uk). Briefly, women resident in Avon, UK with an expected delivery date between 1st April 1991 and 31st December 1992 were eligible to participate in ALSPAC and were enrolled in early pregnancy. Local research ethics committees and the ALSPAC Law & Ethics committee granted ethical approval for the study. For this analysis, all multiple or premature births (<37 weeks gestation), and children with major congenital malformations or illnesses were excluded, as were those missing data on birth weight or gestational age, leaving a cohort of 12,783 infants.

Measurement of behavioural problems

The Strengths & Difficulties Questionnaire (SDQ) (Goodman, 1997), a valid and reliable instrument (Goodman, 2001), was completed by mothers for their child at 47 months (4 years) and 81 months (7 years). The SDQ comprises five 5-item sub-scales: hyperactivity, conduct problems, emotional symptoms, peer problems and prosocial behaviour. Sub-scale scores range from 0 to 10, and a total difficulties score is calculated for the first four sub-scales (range: 0 to 40). Higher scores denote more problems except on the prosocial sub-scale, which is reverse scored. Scores were pro-rated if data were missing (www.sdqinfo.com/ScoreSheets/e1.pdf). Behavioural problems were defined as the highest (or for prosocial behaviour, the lowest) tertile for each sub-scale and for total difficulties. Division into finer categories was not possible because of the positively skewed distribution of SDQ scores.

Dietary data

Dietary data were collected by postal food frequency questionnaire for the child at 38 and 54 months (~3 and 4½ years). This was adapted by an experienced nutritionist (PE) from one that gave mean maternal nutrient intakes (Rogers et al., 1998) similar to those in a national survey (MAFF, 1994). The mother was asked how many times her child was currently consuming a wide variety of food items; (i) never or rarely; (ii) once in two weeks; (iii) 1-3 times a week; (iv) 4-7 times a week; or (v) more than once a day. These responses were numerically transformed into times per week ((i) 0; (ii) 0.5; (iii) 2; (iv) 5.5; and (v) 10) to apply quantitative meaning to the categories. The number of cups of tea/coffee and the number of slices of bread per day were also recorded, as was the type of milk (full fat or other), the type of bread (white or other) and the usual type of spread used (butter/margarine or other). All items were standardized as they were not all measured on the same scale.

Principal Component Analysis (PCA) with varimax rotation was performed on the standardized food types (Northstone et al., 2005). PCA identifies underlying dimensions in the data. Three components were identified and a score for each calculated. One labelled “junk” was associated with increased consumption of high-fat processed foods (burgers, coated poultry) and snack foods high in fat and/or sugar (such as crisps, and chocolate), which tend to be of poor nutritional quality. A higher score on this factor represents more “junk food” intake. The two other components described a “health-conscious pattern”, which was associated with vegetarian style foods, rice, pasta, salad and fruit, and a “traditional diet” of meat, potatoes and vegetables (Northstone et al., 2005). Weekly non-milk extrinsic sugar intake (in grams) was also estimated.

A summary of the data is given in Table 1.

Confounders

Data on confounders (from maternal questionnaires) included: sex of the child, maternal smoking (during pregnancy and when the child was aged 4), maternal age at the birth of the child, number of siblings, socio-economic markers (maternal education, housing tenure, household overcrowding, and car ownership), birth weight, gestational age, maternal depression (Cox et al., 2004) and anxiety (Crown & Crisp, 1979) (at 33 months), maternal enjoyment score (at 33 months) and single parent household. Cognitive function was assessed using the Wechsler Intelligence Scale for Children (WISC-III^{UK}) (Wechsler et al., 1992) at age 8, from which intelligence quotient (IQ) scores were derived.

Statistical Analysis

Analyses were conducted using SPSS v12.0.1 and Stata v8.

Our primary interest lies in the relationship between diet (exposure) and behavioural problems (outcome). To define “incident” behavioural problems at age 7, we excluded children with behavioural problems at age 4, defined as those in the top (or, for prosocial behaviour, bottom) tertile. Logistic regression was used to examine the relationship between diet (“junk food” and sugar content) at 4½ years and the highest (or lowest) tertile of SDQ score at age 7. Odds ratios (OR) and their 95% confidence intervals (95% CI) are reported. The OR represents the odds of having behavioural difficulties at age 7 (defined as scoring in the top 33% on the SDQ) for a one standard deviation (SD) increase in weekly “junk food” consumption (or a 100g increase in weekly extrinsic sugar intake) at 54 months. Regression models were adjusted for confounders (above). Further adjustment was made for IQ score in the subset with these data.

In secondary analyses, we examined the relationship between behavioural problems at age 4, and sugar and “junk food” intake at age 4½ years to determine whether children with behavioural problems were likely to consume more “junk food”. As the “junk food” factor scores were normally distributed, we divided “junk food” intake by quintiles. Those with a high sugar or “junk food” intake (top quintile) at age 3 were excluded from these analyses.

Missing Data

Sensitivity analyses were conducted to examine the influence of missing data on the findings. Multiple imputation by chained equation (MICE) was used to impute missing data (van Buuren et al., 1999) using the *ice* command in Stata (StataCorp, 2003). The imputation models included “junk food” and SDQ scores, and confounders/predictors of “missingness”. We generated 25 datasets and undertook 10 switching procedures.

RESULTS

Behavioural problems

In total, 7725 children (60.4%) had complete SDQ data at age 7. After excluding the top third of children with behavioural difficulties for each SDQ sub-scale at age 4, approximately 4000 children had complete data.

Children’s diets at age 4½

46% of children in the top quintile of “junk food” intake ate chocolate bars at least 4 times per week, compared to 4% of those in the lowest quintile (Table 2). Over 70% of children in the top quintile of “junk food” intake ate crisps at least 4 times per week, five times the figure for those in the lowest quintile. Those children in the top quintile of “junk food” were less likely to consume vegetables. As has been previously reported (Northstone et al., 2005), those of lower social class consumed more “junk food” ($p < 0.001$).

Those in the top quintile of “junk” intake at age 4½ consumed, on average, 577g (SD 209g) of non-milk extrinsic sugar per week compared to an average of 262g (SD 86g) per week for those children in the lowest quintile of “junk” intake ($p < 0.001$ across quintiles).

Diet at 4½ years and behavioural problems at age 7

A one SD increase in “junk food” intake at age 4½ years was associated with an increased odds of being in the top 33% on the SDQ hyperactivity sub-scale at age 7 (Table 3). This persisted after adjustment for confounders including IQ, although the association was modest with the 95% confidence interval only just excluding unity. There was little evidence to support an association between “junk food” and total difficulties or behavioural problems on the other SDQ sub-scales (Table 3).

Unadjusted analyses suggested a weak association between greater sugar consumption and being in the top 33% on the SDQ hyperactivity sub-scale at age 7 (Table 4). After adjustment for confounders, this attenuated and the 95%CI included the null. There was no evidence for an association between sugar intake and total difficulties on the SDQ or scores on the other sub-scales.

“Junk food” and sugar intake were highly correlated at age 4½ years ($r = 0.71$). However, the association between “junk food” intake and hyperactivity remained after adjustment for sugar intake (OR per SD increase in junk: 1.15 (95%CI: 0.99, 1.33)).

As the “junk food” score was based upon a factor analysis, for ease of interpretation the odds of hyperactivity for quintiles of junk food score were calculated. Those children in the

highest quintile of “junk food” intake (top 20%) at age 4½ years had a 19% increased odds of being in the top 33% on the SDQ hyperactivity sub-scale at age 7 (OR: 1.19 (95%CI: 0.87, 1.62)) compared to the lowest quintile.

Missing Data

Half the cohort had complete data. Imputing missing data using MICE suggested that those imputed were less likely to be hyperactive, have emotional problems or score low on prosocial behaviour, but were slightly more likely to have conduct problems (Table 5). Two imputation models were generated. The second included additional indicators of family adversity. Imputation model 1 suggested that those imputed had mothers who were more anxious/depressed and less educated, and the children had slightly lower IQ scores. These differences were not found in the second imputation model (Table 5).

In analyses including the imputed data, results for hyperactivity were consistent with the findings of the complete case-case analyses, irrespective of the imputation model. There was also some evidence for an association between “junk food” intake and total difficulties or emotional problems (Table 3), and some evidence for an association between sugar intake and total difficulties, conduct and emotional problems (Table 4) but the confidence intervals were wide.

Behavioural problems at age 4 and diet at 4½ years

Unadjusted analyses suggested that children with behavioural problems (total difficulties, hyperactivity and conduct problems) at age 4 had a 30-65% increased odds of consuming more “junk food” (top quintile) 6 months later. This attenuated after adjustment for confounders including “junk food” intake at age 3, with the 95% CI including the null (details on request). Similarly, after adjustment for confounders, there was little evidence that children with behavioural problems were likely to consume more sugar 6 months later (details on request).

DISCUSSION

Principal findings

Children eating a diet high in “junk food” in early childhood (at age 4½) were more likely to be in the top 33% on the SDQ hyperactivity sub-scale at age 7. However, this association was modest and the 95% confidence interval only just excluded the possibility of no association. There was little evidence that a “junk food” diet was associated with other behavioural problems or total difficulties. Similarly, there was little evidence for an association between sugar intake and behavioural problems, or that children with behavioural problems in early life consume more “junk food” or sugar 6 months later.

Strengths and Limitations

ALSPAC is representative of the population of Great Britain (Golding et al., 2001). The major strengths of this cohort is its' large size and the detailed data that has been collected. The longitudinal design excludes reverse causality and recall bias.

A further strength of this dataset is the derivation of the “junk food” score. Rather than being based upon a preconceived list of “junk food” items, the “junk” factor emerged from a factor analysis of maternal reports of child diet (Northstone et al., 2005).

Childhood behavioural problems were measured using the parental version of the SDQ. Parents may underestimate conduct problems but mean ALSPAC scores were similar to

national data (Wiles et al., 2006). Misclassification would attenuate any association towards the null.

We examined the association between diet and six behavioural outcomes (total difficulties and 5 sub-scales), so cannot rule out the possibility that the association with hyperactivity is a chance finding. Investigating two dietary exposures (“junk food” and sugar intake) will have increased the possibility of a type I error. “Junk food” and sugar intake were highly correlated, although adjustment for sugar intake did not substantially attenuate the “junk food”-hyperactivity association.

Whilst we adjusted for a number of potential confounders, we cannot exclude the possibility of residual confounding. Conflicting evidence from observational studies and RCTs has been found in other areas of nutritional epidemiology (Lawlor et al., 2004), reflecting the recognised difficulties of estimating nutritional intake, the weak associations with dietary factors, changes in exposure over time, and “lifestyle” factors that can confound relationships (Langseth, 1996). For example, stricter parents may discourage a high “junk” intake, and such parents may be less likely to have children with behavioural problems at a later age. No data were available to explore this hypothesis. It is also possible that IQ may lie on the causal pathway between dietary intake and behaviour, thus controlling for IQ may have attenuated the association.

No SDQ data were available at age 4½ years (i.e. the time point that dietary data were collected). To create a “disease-free” cohort, we excluded those children with behavioural problems at age 4, and then investigated the association between diet at age 4½ years and behaviour at age 7, adjusting for SDQ scores at age 4. This approach assumes that those with behavioural problems were the same individuals at age 4 and 4½, and that the child’s diet at 4½ years reflected dietary intake at the earlier age. It is possible that, amongst those children with behavioural problems at age 4, parents may have modified their child’s diet prior to the measurement of dietary exposure used in this analysis, which may have led to a misclassification.

The inherent difficulties in measuring dietary exposure may have also resulted in a misclassification. This is likely to result in an under-estimation of the association between “junk food” and behaviour at age 7. Furthermore, in order to create a “disease-free” cohort in which to examine this association, we excluded those children who had high scores on the SDQ at age 4. If the association between diet and behavioural problems is causal, it is likely that we will have excluded those children whose behavioural problems at age 4 result from a high “junk food” intake at an earlier age. This may also attenuate the association between “junk food” intake at age 4½ and behavioural problems at age 7.

Finally, findings from complete-case analyses may have been biased so sensitivity analyses were conducted imputing missing data. These confirmed the earlier findings with respect to hyperactivity, but also suggested that children who ate more “junk food” had an increased odds of total difficulties and emotional problems. This requires replication. In order for the estimate of effect to be increased compared to the findings from the complete-case analyses, the effect of “junk food” must be greater in this group. For the imputed data to be valid, the imputation model must be correctly specified, but this cannot be tested empirically. We included all the variables that would be strongly associated with the likelihood of a value being missing, including prior behavioural difficulties. The addition of variables to the first imputation model made little difference to the estimates obtained in our sensitivity analyses, although the additional family adversity variables were themselves often incomplete.

Comparison with previous studies

Prior work is based on small studies of hyperactive children, those with food sensitivities or those referred to specialist clinics, hence limiting generalisability. Whilst early studies lacked a placebo-control group meaning that parental reports of improvements in children's behaviour may have been biased by expectations, this was addressed in later controlled experiments. Elimination of artificial colourings and preservatives from children's diets has been associated with improvement in parental reports of hyperactivity, but not independent reports (Bateman et al., 2004; Schab & Trinh 2004). Nonetheless, existing evidence from challenge studies and RCTs only assesses short-term effects. In the present study, we found a modest association between "junk food" intake and hyperactivity over 2½ years. In agreement with the existing literature (Wolraich et al., 1995; Schnoll et al., 2003; Bellisle 2004), we found little evidence to support an association between sugar intake and behavioural problems.

Clinical implications and proposed mechanisms

Whilst experimental studies have examined short-term behavioural changes in response to the ingestion of particular foods, this observational study examined longer-term effects. If the association observed is causal and hence children who eat more "junk food" are more likely to be in the top 33% on the SDQ hyperactivity sub-scale 3 years later this may reflect longer-term nutritional imbalances. However, to date, there is little clarity about the underlying biological mechanisms.

"Junk" foods are usually high in fat and/or sugar, often contain many additives, food colourings and preservatives, and tend to be of poor nutritional quality. Adjusting for sugar intake did not substantially attenuate the observed association, so other possible explanations must be explored.

A diet high in "junk food" is likely to contain lower levels of essential fatty acids, in particular, omega-3 and omega-6, which cannot be synthesized by the body (Lauritzen et al., 2001). Omega-3 fatty acid is metabolised to docosahexaenoic acid (DHA) which is incorporated in cellular membranes and is particularly concentrated in the retina and brain (Lauritzen et al., 2001). Rats raised on omega-3 deficient diets have reduced hippocampal DHA (Ahmad et al., 2002) and monkeys fed an omega-3 deficient diet exhibit more stereotyped behaviour (Reisbick et al., 1994). In a RCT of young adult prisoners, there was a reduction in antisocial behaviour amongst those randomised to receive a supplement that included essential fatty acids (Gesch et al., 2002). Further, there is some evidence that hyperactive children have lower levels of omega-3 and omega-6 fatty acids (Schnoll et al., 2003). However, to date, studies of fatty acid supplementation in children have proved inconclusive (Voigt et al., 2001; Stevens et al., 2003; Hirayama et al., 2004).

Conclusions and directions for further research

Children eating a diet high in "junk food" in early childhood were more likely to be in the top 33% on the SDQ hyperactivity sub-scale at age 7. This may reflect a long-term nutritional imbalance or differences in parenting styles. However, we cannot rule out the possibility that this may be a chance finding as observational studies in this field are prone to a number of methodological limitations. Hence, our finding requires replication before this can provide an avenue for intervention. Further investigation is warranted given the link between behavioural problems and later educational outcomes, and the continuity between childhood symptoms and difficulties in later life (Maughan & Kim-Cohen, 2005).

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Table 1

Availability of dietary data and data on behavioural problems

Time	Event/Data collected
Birth	Enrolment in ALSPAC study
38 months	Dietary data collected
47 months	SDQ administered
54 months	Dietary data collected
81 months	SDQ administered

Table 2

Comparison of dietary intake amongst children with diets high or low in “junk food” based on dietary intake at age 4½ years (for all those with dietary data: n = 9551)

Frequency of consumption of food item at least 4 times per week	Children in			
	lowest quintile of “junk food”		highest quintile of “junk food”	
	n	%	n	%
Crispy coated chicken/turkey	22	1.2	327	16.7
Oven/Fried chips	5	0.4	150	7.5
Crisps	237	12.8	1426	71.0
Ice cream	14	0.7	587	29.9
Cake	88	4.8	588	29.5
Chocolate coated biscuits	133	7.1	1043	52.5
Biscuits	424	22.6	1321	65.9
Chocolate bars	67	3.6	919	46.2
Sweets	25	1.3	677	34.5
Cola/Other fizzy drinks	60	3.0	669	33.6
Ice lollies	4	0.2	442	22.9
Milk-based puddings	8	0.4	85	4.2
Sausages/burgers	4	0.2	72	3.7
Pizza	2	0.1	67	3.5
Baked beans	38	2.1	255	13.0
Tinned pasta	22	1.2	287	14.6
Pasta	185	9.7	142	7.2
Rice	72	3.8	53	2.6
Fresh citrus fruit	508	27.2	667	33.2
Other fresh fruit	1255	67.0	1389	69.1
Green leafy vegetables ¹	290	15.5	130	6.5
Other green vegetables ²	202	10.8	120	6.0
Carrots	415	22.1	268	13.3
Other root vegetables ³	79	4.2	40	2.0

¹Cabbage, brussel sprouts, spinach, broccoli and other dark green leafy vegetables

²Other green vegetables (cauliflower, runner beans, leeks, okra, courgettes etc)

³Other root vegetables (turnip, swede, parsnip etc)

Table 3
Association between “junk food” intake at age 4½ years and behavioural problems at age 7

	Total difficulties (n=3990)		Hyperactivity (n=4430)		Conduct Problems (n=4091)		Emotional Problems (n=4541)		Peer problems (n=4469)		Prosocial behaviour (n=4400)	
	OR*	95%CI	OR*	95%CI	OR*	95%CI	OR*	95%CI	OR*	95%CI	OR*	95%CI
Unadjusted	1.06	0.98, 1.15	1.19	1.10, 1.29	1.08	0.98, 1.18	1.03	0.96, 1.10	0.99	0.92, 1.07	0.98	0.91, 1.05
Adjusted for confounders**	1.03	0.94, 1.13	1.16	1.07, 1.27	1.05	0.95, 1.16	1.01	0.94, 1.09	0.95	0.88, 1.04	0.98	0.91, 1.06
Adjusted** association within subset with IQ data	1.05	0.94, 1.17	1.16	1.04, 1.28	1.04	0.92, 1.17	1.02	0.94, 1.12	0.97	0.87, 1.07	0.99	0.90, 1.08
Further adjustment for IQ score (within subset)	1.02	0.92, 1.14	1.13	1.01, 1.25	1.01	0.89, 1.14	1.01	0.93, 1.11	0.95	0.86, 1.06	0.98	0.89, 1.08
Sensitivity analyses using MICE to impute missing data (fully adjusted models)#												
model 1	1.17	1.04, 1.33	1.14	1.02, 1.27	1.05	0.96, 1.14	1.11	1.01, 1.21	1.04	0.94, 1.15	0.96	0.88, 1.06
model 2	1.19	0.99, 1.43	1.14	1.00, 1.31	1.06	0.95, 1.18	1.10	0.97, 1.25	1.04	0.95, 1.15	0.99	0.86, 1.14

* OR represents the increase in odds of behavioural problems per 1 standard deviation (SD) increase in “junk” food score (based on factor analysis)

** Adjusted for SDQ total difficulties or sub-scale score at age 4½ yrs, sex of child, maternal smoking, maternal age at birth of child, number of siblings, socio-economic markers, birth weight and gestational age, maternal depression and anxiety, maternal enjoyment score, and single parent household.

Using a range of different variables in the imputation models. See footnote to Table 5 for the differences between model 1 and 2

Table 4
Association between weekly sugar intake (per 100g) at age 4½ years and behavioural problems at age 7

	Total difficulties (n=4798)		Hyperactivity (n=4430)		Conduct Problems (n=4091)		Emotional Problems (n=4541)		Peer problems (n=4469)		Prosocial behaviour (n=4400)	
	OR*	95%CI	OR*	95%CI	OR*	95%CI	OR*	95%CI	OR*	95%CI	OR*	95%CI
Unadjusted	1.00	0.95, 1.06	1.09	1.04, 1.14	1.05	0.98, 1.11	1.00	0.96, 1.04	0.98	0.94, 1.03	0.98	0.94, 1.03
Adjusted for confounders**	0.99	0.94, 1.05	1.07	1.01, 1.13	1.03	0.97, 1.09	1.00	0.96, 1.05	0.97	0.92, 1.02	0.98	0.93, 1.02
Adjusted** association within subset with IQ data	0.99	0.93, 1.07	1.05	0.99, 1.13	1.03	0.95, 1.12	1.01	0.96, 1.07	0.98	0.92, 1.04	0.99	0.92, 1.05
Further adjustment for IQ score (within subset)	0.98	0.91, 1.06	1.04	0.97, 1.11	1.02	0.94, 1.11	1.01	0.96, 1.07	0.97	0.91, 1.04	0.99	0.93, 1.05
Sensitivity analyses using MICE to impute missing data (fully adjusted models)#												
model 1	1.33	0.74, 2.38	1.22	0.89, 1.67	0.91	0.72, 1.16	1.31	1.03, 1.65	1.14	0.90, 1.45	0.89	0.69, 1.16
model 2	1.21	0.89, 1.65	1.25	0.93, 1.69	0.91	0.72, 1.16	1.32	1.02, 1.65	0.83	0.59, 1.18	0.86	0.64, 1.16

* OR represents the increase in odds of behavioural problems per 100g increase in weekly sugar consumption (non-milk extrinsic sugar intake)

** Adjusted for SDQ total difficulties or sub-scale score at age 4½ yrs, sex of child, maternal smoking, maternal age at birth of child, number of siblings, socio-economic markers, birth weight and gestational age, maternal depression and anxiety, maternal enjoyment score, and single parent household.

Using a range of different variables in the imputation models. See footnote to Table 5 for the differences between model 1 and 2

Table 5 Comparison of the observed and imputed data for those with SDQ hyperactivity scores at age 4½ & diet data at age 4½ (n = 7983)

	Observed data		Imputed data	
	n	%	n	%
<i>Number of siblings</i>	6902		1081	
0		10.7%		10.9%
1		54.7%		51.0%
2		34.5%		38.1%
<i>Maternal anxiety</i>	7358		625	
Low		85.6%		82.9%
High		14.4%		17.1%
<i>Maternal depression</i>	7364		619	
Low		88.6%		85.3%
High		11.4%		14.7%
<i>Maternal education</i>	7497		486	
< O level		20.7%		33.2%
O level		37.3%		38.0%
> O level		42.0%		28.8%
<i>Maternal enjoyment score</i>	7365		618	
Low		13.8%		14.8%
High		86.2%		85.2%
<i>IQ: mean</i>	4932	105.6	3051	102.2
<i>81 month SDQ</i>				
<i>Total difficulties</i>	6837		1146	
Low		66.1%		65.7%
High		33.9%		34.3%
<i>Hyperactivity</i>	6842		1141	
Low		71.2%		78.6%
High		28.8%		21.4%
<i>Conduct problems</i>	6857		1126	
Low		76.7%		57.9%
				73.8%

	Observed data		Imputed data	
	n		n	
			Model 1*	Model 2*
High	6850	23.3%	1133	42.1%
<i>Emotional problems</i>				26.2%
Low		61.6%		75.3%
High		38.4%		24.7%
<i>Peer problems</i>				
Low	6854	73.4%	1129	71.7%
High		26.6%		28.3%
<i>Prosocial behaviour</i>				
High	6855	68.3%	1128	87.3%
Low		31.7%		12.7%
<i>Additional family adversity indicators</i>				
<i>Partner cruelty during pregnancy</i>	7266		717	
Yes		5.1%		2.9%
No		94.9%		97.1%
<i>Trouble with police during pregnancy</i>	7258		725	
Yes		2.2%		25.6%
No		97.8%		74.4%
<i>Convictions during pregnancy</i>	7248		735	
Yes		0.2%		23.9%
No		99.8%		76.1%

n - number of individuals with observed data or for whom data were imputed. Variables where less than 5% of observations were imputed are not presented

List of variables included in imputation models:

Model 1 - SDQ scores at 47 months and 81 months, "junk food" scores at 38 months and 54 months, gender, maternal age, parity, maternal smoking, birthweight, gestational age, socio-economic indicators (housing tenure, maternal education, household overcrowding, car ownership), single parent family, number of siblings, maternal depression, maternal anxiety, maternal employment score, IQ score

Model 2 - as model 1 with additional variables including indicators of family adversity: binary SDQ (high/low) indicators, and indicators of family adversity: early parenthood, housing (adequacy, basic living facilities (e.g. hot water), defects), financial difficulties, partner (present/absent), relationship with partner (affection/cruelty/support), family size, major family problems (child in care, not with natural mother or on at risk register), maternal depression/anxiety, substance abuse, crime (trouble with police or convictions).

* Data for imputed columns represent values across 25 imputation datasets