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Challenging the view that lack of fibre causes childhood constipation

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ABSTRACT

Objectives To assess evidence supporting the view that 'low fibre *causes* childhood constipation'.

Design Triangulation integrated three approaches: a systematic review NICE guideline CG99 examining effectiveness of increasing fibre; a cohort study, Avon Longitudinal Study of Parents and Children (ALSPAC), to assess if constipation (or hard stools) can precede fibre intake at weaning; and a literature search for twin studies to calculate heredity.

Setting CG99 examined the literature regarding the effectiveness of increasing fibre. ALSPAC asked parents about: hard stools at 4 weeks, 6 months and 2.5 years and constipation at age 4–10 years, as well as fibre intake at 2 years. Twin studies and data from ALSPAC were pooled to calculate concordance of constipation comparing monozygotic and dizygous twin pairs.

Participants CG99 reported six randomised controlled trials (RCTs). ALSPAC hard stool data from 6796 children at 4 weeks, 9828 at 6 months and 9452 at 2.5 years plus constipation data on 8401 at 4–10 years were compared with fibre intake at 2 years. Twin studies had 338 and 93 twin pairs and ALSPAC added a further 45.

Results Increasing fibre did not effectively treat constipation. Hard stools at 4 weeks predated fibre and at 6 months predicted lower fibre intake at 2 years (p=0.003). Heredity explained 59% of constipation.

Conclusions RCTs indicate that increasing fibre is not an effective treatment for constipation in children. Hard stools can precede and predict later fibre intake. Genetic inheritance explains most childhood constipation. Extended treatment with stool softeners may improve fibre intake and limit long-term damaging sequelae of constipation.

What is already known on this topic?

► It has been observed for children that identical twins have a much higher concordance for constipation compared with non-identical twins.
► It is known that diet, particularly a low fibre diet, is *associated with* constipation.

What this study adds?

► This study suggests that constipation in children is *not* caused by low dietary fibre but by a genetic predisposition to hard stools and subsequent constipation.
► Extended effective medical treatment of hard stools in infancy and childhood may increase subsequent fibre intake and reduce the prevalence of long-term idiopathic constipation.
► This study provides a biological explanation for NICE guideline CG99 *evidence-based* guideline statement 'Do not use dietary interventions alone as first-line treatment for idiopathic constipation'.

INTRODUCTION

Constipation affects 10% of children.[1] It can be severely disabling resulting in medical and surgical intervention and poor quality of life including mental health,[2] particularly if associated with soiling. Lay belief is that low fibre or lack of fluids causes constipation,[3] a view held by many health professionals,[4] leading to delayed laxative treatment.[4] Parents are reluctant to administer long-term laxatives because of a second false lay belief: 'long term use of laxatives makes the bowel lazy'.[3] Parents feel guilty[5 6] exacerbated by dietary advice perceived as 'you are not feeding your child properly'. Effective laxative treatment can be delayed for months or years turning a treatable condition into a chronic intractable problem.

In 2010, NICE[4] recommended against dietary interventions alone as first-line treatment for idiopathic constipation in children. However, beliefs about fibre persist, perhaps because an alternative cause of constipation in children has not been found. This paper uses triangulation[7] to examine the question 'does low fibre *cause* constipation in children', utilising data collected with different methodologies, together to try to ascertain causality. The evidence from each context or methodology alone is not adequate to prove or refute a cause. However, if methodologies in different contexts point in the same direction, then causation can be examined more credibly.

METHODS

Dataset 1: NICE guideline CG99 with 2017 review update

NICE used standard search and assessment to review evidence that increasing dietary fibre was an effective treatment for childhood constipation.[4] Within the guideline scope, 'Management' included: 'dietary manipulation, fruits, vegetables (fibres and roughage), fruit juices, cereals'. The question was: 'What is the clinical effectiveness of dietary modification for ongoing treatment or maintenance in children with chronic idiopathic constipation?' Studies included six randomised controlled trials (RCTs).[8–13]

Dataset 2: data from a UK birth cohort

The sample comprised participants from the Avon Longitudinal Study of Parents and Children (ALSPAC). Detailed information available at (http://www.bristol.ac.uk/alspac) includes a fully searchable data-dictionary (http://www.bris.ac.uk/alspac/researchers/data-access/data-dictionary). Pregnant women resident in the former Avon Health Authority in South West England, having an estimated date of delivery between 1 April 1991 and 31 December 1992, were approached, resulting in a cohort of 14 541 pregnancies.[14] Of 13 978 singletons/twins alive at 1 year, 24 participants withdrew consent leaving 13 954 participants.

Fibre intake at age 2 years

A food frequency questionnaire[15 16] was administered to the main carer when their child was 2 years old. Responses to foods containing fibre were selected[17]: baby rice, baby cereal, breakfast cereal, savoury vegetable, potatoes, other vegetables, baked beans, green peas, other legumes, figs, raw apple, other raw fruit, raw carrot, other raw vegetables and nuts/nut products. Frequency of consumption was recorded in a continuous format (eg, times/week) yielding data with a wide range of values and notable digit preference. These data were collapsed to form a set of ordinal variables (never or <1/week; 1–3 times/week; 4–7 times/week; >7 times/week). Polychoric principal component analysis was performed, and the first component, describing 30.4% of total item variance, was chosen as the continuous standardised measure of fibre consumption (online supplementary tables S1 & S2). Online supplementary figure S1 groups the fibre measure into five quintiles showing the average frequency of intake for each food type within each quintile.

Hard stools and constipation

At 4 weeks, 6 months and 2.5 years, questionnaires were administered about stool consistency[18] asking about frequency of hard stools with response options at 4 weeks: '*Always*', '*Sometimes*', '*Occasionally*' and '*Never*' and at 6 months and 2.5 years: '*Usually*', '*Sometimes*' and '*Never*'.

At ages 4½, 5½, 6½, 7½ and 9½ years, parents were asked about their child's constipation in the past 12 months with options: '*Yes, and saw a doctor*'; '*Yes, did not see the doctor*' and '*No, did not have*'. Responses were dichotomised to indicate presence of any constipation at each age. These data have been analysed previously using longitudinal latent class analysis to separate patterns of constipation. In addition to a normative class (82%; very low probability of constipation), we found evidence for three separate classes who experienced some degree of constipation: early occurrence (7% of children suffered constipation until 6 years), late occurrence (8% had constipation emerging after 6 years) and persistent (3% had a high probability throughout). We found a strong association between hard stools at 2.5 years and constipation during later childhood.[19]

Confounders

Potential confounders including child's sex and socioeconomic variables measured[20] were considered (see table 1 footnote for details). For analyses investigating prospective associations between diet and hard stools, the duration of *any* breast feeding and age of introducing cow's milk and solid food, were included. Online supplementary table S3 shows many of the socioeconomic variables and their strong association with fibre intake. This contrasts with lack of association between constipation and socioeconomic variables using this cohort.[19]

Statistical modelling

For models with ordinal/nominal outcome variables (eg, stool consistency), multinomial regression models were used. For models of fibre intake, OLS regression was applied. For each model, unadjusted estimates were derived followed by adjustments for confounders.

Table 1 Prospective association between frequency of hard stools at (1) 4 weeks and (2) 6 months and fibre intake at 2 years

	Unadjusted	Adjusted 1	Adjusted 2
	Beta (95% CI)	Beta (95% CI)	Beta (95% CI)
(1) Frequency of hard stools at age 4 weeks			
Never	0.00 ref	0.00 ref	0.00 ref
Sometimes/occasionally	−0.215 (−0.268 to −0.162)	−0.051 (−0.109 to 0.008)	0.038 (−0.033 to 0.109)
Always	−0.479 (−0.677 to −0.281)	−0.229 (−0.445 to −0.013)	−0.111 (−0.331 to 0.109)
Omnibus p value	P<0.001	P=0.034	P=0.293
R^2	0.012	0.0445	0.0488
N	6796	5249	5126
(2) Frequency of hard stools at age 6 months			
Never	0.00 ref	0.00 ref	0.00 ref
Sometimes	−0.114 (−0.158 to −0.070)	−0.089 (−0.135 to −0.043)	−0.077 (−0.124 to −0.029)
Usually	−0.171 (−0.237 to −0.104)	−0.103 (−0.172 to −0.034)	−0.092 (−0.163 to −0.021)
Omnibus p value	P<0.001	P<0.001	P=0.003
R^2	0.0036	0.0511	0.0558
N	9828	7550	7214

Estimates represent standardised differences in continuous measure of fibre intake relative to the group who are reported to never suffer from hard stools.

Adjusted 1: adjusted for sex, and sociodemographic variables (mother's social class, based on the occupational social class classification dichotomised into non-manual (professional, managerial and skilled occupations) and manual (comprising partly skilled or unskilled professions); early parenthood indicating if the mother was under 19 years when giving birth to the study child; level of maternal education: <O-level (ie, no school leaving qualification), O-level (school leaving qualifications obtained at age 16 years) and A-level/degree (qualifications from further education); housing tenure: mortgaged/owned; privately rented; subsidised rented; and experience of material hardship (dichotomous variable); household income equivalised, a composite measure of disposable income split into quintiles.).

Adjusted 2: (1) further adjusted for breast/bottle feeding status in first 4 weeks/(2) further adjusted for breastfeeding duration in first 6 months and timing of introduction of both milk and solids.

Original research

Dataset 3: twin data

Limited published data exist regarding twin zygosity and concordance of constipation in children. (1) a well-regarded *Paediatric Gastroenterology Textbook*[21] referenced one study published in 1971 describing twin concordance for constipation aged 6–19 years.[22] Raw data were extracted for twin zygosity and concordance of constipation. (2) Further searching of the literature used 'child', 'constipation' and 'twin' as keywords and 'title' words to examine whether additional useful published data were available. Ovid Medline, Embase and Web of Science databases were searched from 1946 to 2019. One additional Japanese twin study[23] was found with parent-reported constipation age 3 years. Data were extracted. 'Hereditary' was substituted for 'twin' but did not provide literature with further extractable data. (3) Data on twins among ALSPAC participants was included.

All three studies had zygosity derived in accepted but different ways. For the 1971 US twin study,[22] 'Zygosity was determined clinically and, by matching blood groups. The blood groups used were ABO, Rh (CcDEe), MNSs, P, Fya, K, and Jka. Blood matching was omitted only when dizygosity was clinically self-evident'.[24] For the Japanese twin study,[23] fingerprints were used, and for those where zygosity could not be determined, blood typing was employed using 20 genetic markers. Finally, for ALSPAC, zygosity was defined using DNA tests,[14] and further twins were determined as dizygous if of different sex.

Statistical modelling

Heredity was derived using a standard approach that gives values from 0% to 100% corresponding to fully heritable (solely due to genetics). For example, adult height is strongly heritable 80%. Heritability was calculated as the difference in variance (ie, 1 – concordance rate) between dizygotic and monozygotic twins divided by the variance in dizygotic twins.[25]

RESULTS

Dataset 1: NICE guideline CG99

The NICE guideline report found no evidence to suggest increasing fibre-rich foods was effective to treat or manage constipation and that adding fibre to an already healthy diet could be detrimental, exacerbating symptoms and increasing soiling. The Guideline Development Group recommended '*children should be advised to eat a healthy diet, including fibre containing foods* as outlined by the Paediatric Group of the British Dietetic Association in "Food for the Growing Years" and "Food for the School Years"'.[26] The guideline did however suggest to achieve: '*Adequate fibre: Recommend including foods with a high fibre content (such as fruit, vegetables, high-fibre bread, baked beans and wholegrain breakfast cereals)*'.[4] There was insufficient evidence to recommend fibre supplements in treatment or management of idiopathic constipation.[4] The 2017 guideline update stated: 'New evidence is unlikely to change guideline recommendations'.[4]

Dataset 2: data from a UK birth cohort

One per cent of babies *always* had hard stools age 4 weeks, 12% *usually* age 6 months and 28% *usually* at 2.5 years. table 1 shows the association between hard stool frequency at 4 weeks and 6 months and fibre intake at 2 years (online supplementary figures S2a & S2b).

Hard stools at 4 weeks (ie, when the diet is milk containing no fibre) and 6 months predicted a lower fibre intake at 2 years (table 1). At this time in the early 1990s, weaning children to solids that contain fibre was recommended at 4 months of age.

There was a strong association between hard stools at 2.5 years (table 2a and online supplementary figure S3), as well as constipation at 4–10 years (table 2b) with fibre intake at 2 years.

Dataset 3: twin data

US, Japanese and UK data all showed similar trends with monozygotic twins more likely to be concordant for constipation than dizygous twins (p<0.01) (individual study data not shown). Overall, 59% of childhood constipation can be explained by genetic predisposition (table 3).

DISCUSSION

This paper uses three research approaches to examine the statement 'Lack of fibre in the diet *causes* constipation in children'.

NICE guideline CG99 (including the 2017 update)[4] found that a limited number—six—good quality trials were available[8–13]; however. they provided no support for the use of fibre-rich foods and limited evidence for the use of fibre supplements in treating constipation. The European and North American Societies for Paediatric Gastroenterology Hepatology and Nutrition consensuses recommend a normal fibre intake for children and adolescents with constipation.[27] A 2018 systematic review

Table 2a Association between fibre intake at age 2 years and hard stool frequency at age 2.5 years

	N	Never OR (95% CI)	Sometimes OR (95% CI)	Usually OR (95% CI)	Omnibus p
Unadjusted	9452	1.00 ref	0.904 (0.852 to 0.958)	0.811 (0.761 to 0.865)	P<0.001
Adjusted 1	7481	1.00 ref	0.834 (0.773 to 0.899)	0.789 (0.726 to 0.856)	P<0.001
Adjusted 2	6141	1.00 ref	0.854 (0.783 to 0.931)	0.817 (0.744 to 0.897)	P<0.001

Adjusted 1: adjusted for the following list of variables: sex, measures of SES (social class, early parenthood and maternal education), household income, housing tenure and material hardship.
Adjusted 2: further adjusted for breastfeeding duration in first 6 months and timing of introduction of both milk and solids.

Table 2b Association between fibre intake at age 2 years and longitudinal patterns of childhood constipation (4–10 years)

	N	Low risk of constipation OR (95% CI)	Late childhood occurrence OR (95% CI)	Early childhood occurrence OR (95% CI)	Persistent constipation OR (95% CI)	Omnibus p
Unadjusted	8401	1.00 ref	1.147 (1.008 to 1.305)	1.040 (0.917 to 1.179)	0.812 (0.724 to 0.912)	P<0.001
Adjusted*	6784	1.00 ref	1.071 (0.916 to 1.253)	0.971 (0.832 to 1.134)	0.748 (0.643 to 0.869)	P=0.002

All estimates represent the change in multinomial odds for a 1 SD change in the continuous measure of fibre intake.
*Adjusted model has included the following list of variables: sex, sociodemographic variables, gestation age, birth weight and developmental level of a child at 18 months.

Table 3 Concordance of constipation comparing identical monozygotic (MZ) and non-identical dizygous (DZ) twins (all studies pooled)

	Twin status	N (%) Both fine	N (%) One twin affected	N (%) Both twins affected	Fisher's exact test	% with ≥1 twin affected	Concordance rate	Heredity
All studies pooled together *Parent-reported constipation*								
Age 3–19 years	Identical MZ (n=291)	256 (88)	13 (4)	22 (8)	P<0.01	12	0.63	59%
	Non-identical DZ (n=185)	155 (84)	27 (15)	3 (2)		17	0.10	

Concordance=number with both twins affected divided by the number with both affected+number with one affected. Therefore, if twins are always both affected concordance=1 and if only one twin is ever affected concordance=0.

and meta-analysis that included five of the six trials reported by NICE[8–10 12 13] and four additional trials[28–31] concluded '*there is no scientific evidence to corroborate the prescription of fiber supplementation in the diet of constipated children as part of the treatment of this condition*'.[32] If a low-fibre diet *caused* childhood constipation, then a diet high in fibre or added fibre should effectively treat constipation, rather than having '*no or little effect*'.[4]

ALSPAC findings indicate that 'hard stools' the strongest predictor of later constipation,[19] can both precede weaning (when fibre commences in the diet) and predict later fibre intake. We know that constipation often leads to reduced appetite[33] when parents describe their children as 'fussy eaters', where dietary intake is low in fibre rich fruit and vegetables.[6] The clinical observation that diet in amount and variety often improves by effectively treating constipation with laxatives particularly stool softeners fits in with these findings.

Finally, twin data indicate that 59% of childhood constipation can be explained as a genetic or natural phenomenon rather than being caused by a low intake of fibre. Genetic studies of functional bowel disorders,[34] which include idiopathic constipation, indicate a similar level of genetic influence.

The purpose of triangulation is to try to eliminate bias by answering a question with more than one methodology so that possible biases will be different. If all (three in this case) methods point in the same direction and possible biases are not thought to act in the same direction, then stronger claims about *cause* can be made. Clinical trials of fibre intake (NICE guideline) can suffer from bias from poor compliance. However, if increased fibre intake cannot be sustained in well-resourced clinical trials, it is unlikely to be better tolerated in clinical practice. Bias in observational studies (ALSPAC) is often related to residual sociodemographic confounding. Multiple sociodemographic variables were controlled for (online supplementary table S3). It is interesting to note that fibre intake was strongly related to sociodemographic status but constipation was not.[19] Finally, twin studies can suffer from poor definition of zygosity that may dilute findings of genetic influence. However, all three studies of twins have findings in the same direction. Although numbers remain small they show statistical significance and agree with other studies documenting the hereditary nature of functional bowel disorders.[34]
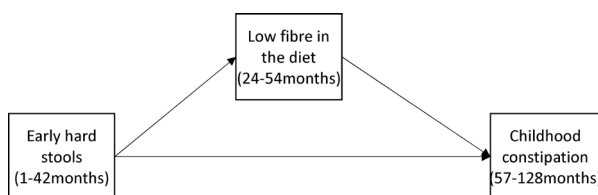

Figure 1 Timeline of hard stools leading to a lower fibre diet and later constipation.

Together these three approaches provide strong evidence to reconsider the conventional view, that a low fibre diet is the *cause* of constipation in children.

Strengths and weaknesses in relation to other studies, discussing important differences in results

The strength of this study is the use of triangulation as a method to test causation.[7] NICE CG99 and a recent systematic review fail to show that fibre is an effective treatment. ALSPAC indicates that early signs of constipation—hard stools at 4 weeks—can precede fibre at weaning—4 months in the 1990s—and predict subsequent low fibre intake. This puts in doubt lack of fibre as the *cause* of constipation. Twin studies show that most constipation in childhood can be explained by inheritance and not therefore primarily *caused* by lack of fibre.

It must be noted that a low level of fibre age 2 years (ALSPAC) was strongly associated with later constipation. These cohort data are in keeping with many cross-sectional studies.[35 36] However, the strength of ALSPAC relates to the longitudinal nature of the cohort *from birth*. Most previous studies have examined constipation after fibre introduction at weaning (age 4–6 months). ALSPAC measured hard stools at 4 weeks before fibre. If signs of later constipation (hard stools) precede fibre, then for those children low fibre is not the primary cause. Figure 1 illustrates the possible timeline of early hard stools, low fibre intake and later constipation.

A similar mechanism was put forward in a recent cohort study.[6]

Limited data have been reported regarding twins and concordance for constipation. One study has been well documented.[22] Even after pooling of data, the numbers we report are fairly small; however, there is a significant difference in concordance between monozygotic and dizygous twin pairs.

Meaning of the study: possible explanations and implications for clinicians and policymakers

Constipation is common (10% of children) and often chronic, severely debilitating and may have long-term devastating physical and psychological sequelae. This paper indicates that the observed association between low fibre and constipation is unlikely to be directly causal. If further research concludes that hard stools and constipation in childhood are largely inherited, this infers a different *cause* to the current societal focus on diet. This conclusion is supported by CG99 that found that a high-fibre diet did not effectively treat childhood constipation. The shift in focus away from fibre is backed up by ALSPAC, which showed that hard stools can precede weaning (to fibre) and predict a later low-fibre intake, supported by another cohort from the Netherlands.[6] These observational data may explain why increasing fibre is not very effective.[4 37] Future research should move away from increasing fibre to genetic studies starting with twins who present with childhood constipation. If

hard stools and constipation are *caused* by genetic predisposition and early hard stools predict a low-fibre intake, treatment should focus on softening stools over an extended period, particularly early in life when relief of hard stools may improve long-term fibre intake.[6] NICE guideline CG99[4] states: 'Do not use dietary interventions alone as first-line treatment for idiopathic constipation'. This study begins to clarify the aetiological reasons for that *evidence-based* statement and will support more clinicians to adhere to this important guidance.

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Research Ethics Committee approval references

Please note that all self-completion questionnaire content is approved by the ALSPAC Ethics and Law Committee.

Initial Approval:

- Bristol and Weston Health Authority: E1808 Children of the Nineties: Avon Longitudinal Study of Pregnancy and Childhood (ALSPAC). (28th November 1989)
- Southmead Health Authority: 49/89 Children of the Nineties - "ALSPAC". (5th April 1990)
- Frenchay Health Authority: 90/8 Children of the Nineties. (28th June 1990)

Children in Focus or "The 10% Club"

- Southmead Health Services: 48/89: ALSPAC – "The 10% Club" (25th August 1992)

7 Year Clinic:

- United Bristol Healthcare Trust: E4168 ALSPAC Hands on Assessments at Age Seven. (30th September 1998)
- Southmead Health Services: 67/98 Avon Longitudinal Study of Pregnancy and Childhood (ALSPAC) - Hands on Assessments at Age Seven. (14th September 1998)
- Frenchay Healthcare Trust: 98/52 Avon Longitudinal Study of Pregnancy and Childhood (ALSPAC). Hands on Assessments at Age Seven.(8th December 1998)

8 Year Clinic:

- United Bristol Healthcare Trust: E4445 ALSPAC Focus at Eight. (28th January 2000)
- Southmead (North Bristol Trust): Project 084/99 ALSPAC Assessments at Age Eight. (November 1999)
- Frenchay (North Bristol Trust): Project 99/42 Avon Longitudinal Study of Parents and Children (ALSPAC). Assessments at Age Eight (ALSPAC Focus at Eight). (15th December 1999)

- Weston Area Health Trust: E177 Avon Longitudinal Study of Parents and Children (ALSPAC). Assessments at Age Eight (21stSeptember 1999)

9 Year Clinic:

- United Bristol Healthcare Trust: E5018 Avon Longitudinal Study of Parents and Children – Assessments at Age Nine (ALSPAC Focus@9). [Reciprocal Arrangement] (7th February 2002)
- Southmead (North Bristol Trust): 008/01 ALSPAC Assessments at Age Nine. (20th March 2001)
- Frenchay (North Bristol Trust): 2001/35 Avon Longitudinal Study of Parents and Children (ALSPAC) Assessments at Age Nine. (9th May 2001)

10 Year Clinic:

- United Bristol Healthcare Trust: E5215 ALSPAC – Focus at 10+. [Reciprocal]. (31st January 2002)
- Southmead (North Bristol Trust): 138/01 ALSPAC – Focus 10+. (18th January 2002)
- Frenchay (North Bristol Trust): 2001/90 Avon Longitudinal Study of Parents and Children (ALSPAC) Assessments at Age Ten Plus. (28th January 2002)
- Weston Area Health Trust: E280R – ALSPAC Focus 10+. (12th December 2001)

11 Year Clinic:

- Southmead (North Bristol Trust): 137/02: ALSPAC – Focus at 11+. (17th April 2003)
- United Bristol Healthcare Trust: E5691 ALSPAC Focus @ 11+. [Reciprocal] (29th May 2003)
- Frenchay (North Bristol Trust): 2002/110 Avon Longitudinal Study of Parents and Children (ALSPAC) Assessments at Age Eleven Plus. (23rd June 2003)
- Weston Area Health Trust: E325 (R) – 137/02 Avon Longitudinal Study of Parents and Children (ALSPAC) Assessments at Age Eleven Plus. (28th February 2003)

12 Year Clinic:

- Central & South Bristol Research Ethics Committee (UBHT): E5806 ALSPAC Teen Focus 1. (19th February 2004)
- Confirmation of Site Specific Approval (Southmead and North Somerset) (26th May 2004) Southmead Research Ethics Committee: Project 030/04 ALSPAC Hands on Assessments: Teen Focus 1. (26th April 2004)
- Frenchay Local Research Ethics Committee: 2004/18 Avon Longitudinal Study of Parents and Children (ALSPAC) Hands on Assessments: Teen Focus 1. (22nd April 2004)
- North Somerset Research Ethics Committee (Weston): 04/Q2003/5 Avon Longitudinal Study of Parents and Children (ALSPAC) Hands on Assessments: Teen Focus 1. (7th April 2004)

12 Year Clinic Amendment (TF1 Fast-track):

- Central & South Bristol Research Ethics Committee (UBHT): E5806 ALSPAC Teen Focus 1fasttrack Amendment. (16th February 2005)

13 Year Clinic:

- Central & South Bristol Research Ethics Committee (UBHT): 04/Q2006/168 Avon Longitudinal Study of Parents and Children (ALSPAC), Hands on Assessments: Teen Focus 2 (Focus 13+). (11th March 2005)
- Approval for use of Biosamples (8th June 2007)

15 Year Clinic:

- Central & South Bristol Research Ethics Committee (UBHT): 06/Q2006/53 Avon Longitudinal Study of Parents and Children (ALSPAC), Hands on Assessments: Teen Focus 3 (Focus 15+). (7th August 2006) (Confirmed 15th September 2006)

17 Year clinic:

- North Somerset & South Bristol Research Ethics Committee: 08/H0106/9 Avon Longitudinal Study of Parents and Children

(ALSPAC), Hands on Assessments: Teen Focus 4 (Focus 17+) (18th November 2008)

NAFLD:

- North Somerset & South Bristol Research Ethics Committee: 09/H0106/53 Evaluating the prevalence, causes and biomarkers for identifying non−alcoholic fatty liver disease (NAFLD) in young adults. (7th August 2009)

Mothers Clinic 1:

- North Somerset & South Bristol Research Ethics Committee: 08/H0106/96 Avon Longitudinal Study of Parents and Children (ALSPAC); Focus on Mothers (2nd September 2008, Confirmed 8th September 2008)

Enrolment of Partners:

- North Somerset & South Bristol Research Ethics Committee: 09/HO106/82. Avon Longitudinal Study of Parents and Children (ALSPAC); Enrolment of Partners (26 October 2009, Questionnaire approval 18 October 2010).

Home Visits:

- South West 3 Research Ethics Committee: 10/H0106/74 Avon Longitudinal Study of Parents and Children (ALSPAC); Home Visits (22nd December 2010)

PEARL project

- North West 5 Research Ethics Committee: 10/H1010/70 (protocol Number 1278). Project to Enhance ALSPAC through Record Linkage (PEARL): phenotypic enrichment of the ALSPAC Cohort though linkage to primary care electronic patient records and other databases. (3rd February 2011)

Mothers Clinic 2, 3 & 4:

- National Research Ethics Service Committee South West – Central Bristol: 11/SW/0110 Avon Longitudinal Study of Parents and Children (ALSPAC); Focus on Mothers 2 and 3 (1st June 2011, Confirmed 20th June 2011)

Fathers Clinic:

- National Research Ethics Service Committee North West – Haydock:
  11/NW/0369 Avon Longitudinal Study of Parents and Children (ALSPAC);
  Focus on Fathers (3rd August 2011)

24 Year Clinic:

- National Research Ethics Service Committee South West – Frenchay:
  14/SW/1173 ALSPAC Focus at 24+ (24th February 2015, confirmed 20th
  March 2015)

Table S1. Specific foods used in the derivation of scale representing fibre intake at 2 years (N = 10,418)

Food item	Frequency of consumption				Polyserial correlation with fibre intake scale *
	Never	1-3/wk	4-7/wk	>7/wk	
Baby rice	99.8%	0.1%	0.1%	-	-0.151
Other baby cereal	99.2%	0.5%	0.4%	<0.1%	-0.082
Breakfast cereal	17.9%	14.3%	67.0%	0.8%	0.503
Vegetables (jar, tin or packet)	98.3%	1.2%	0.5%	<0.1%	-0.025
Other vegetables	15.1%	0.6%	3.4%	80.9%	0.869
Baked beans	40.5%	49.5%	10.0%	<0.1%	0.525
Green beans	33.1%	50.1%	16.8%	0.1%	0.629
Other legumes	89.6%	9.1%	1.3%	<0.1%	0.602
Fig products	98.3%	1.3%	0.4%	-	0.279
Raw apple	30.2%	39.3%	30.3%	0.2%	0.715
Other raw fruit	20.1%	32.8%	45.5%	1.6%	0.728
Raw carrot	82.0%	15.5%	2.5%	<0.1%	0.670
Raw vegetables	87.8%	8.0%	4.2%	<0.1%	0.656
Nut products	94.8%	4.0%	1.3%	-	0.508
Potatoes	14.7%	0.7%	4.1%	80.6%	0.832

* Fibre intake scale consisted of first principal component extracted by polychoric PCA. Scale explained 30.4% of item variability.

Table S2. Polychoric correlations between individual food items used for the derivation of scale representing fibre intake at age 2 years (N = 10,418)

	Baby rice	Other baby cereal	Breakfast cereal	Vegetables (jar, tin or packet)	Other vegetables	Baked beans	Green beans	Other legumes	Fig products	Raw apple	Other raw fruit	Raw carrot	Raw vegetables	Nut products	Potatoes
Baby rice	1														
Other baby cereal	0.654	1													
Breakfast cereal	-0.223	-0.139	1												
Vegetables (jar, tin or packet)	0.460	0.522	-0.070	1											
Other vegetables	-0.137	-0.075	0.491	-0.013	1										
Baked beans	-0.093	0.000	0.183	0.027	0.376	1									
Green beans	-0.032	-0.093	0.209	-0.004	0.499	0.492	1								
Other legumes	0.131	0.127	0.133	0.071	0.242	0.201	0.247	1							
Fig products	0.149	-0.073	0.048	0.221	0.146	0.065	0.076	0.279	1						
Raw apple	-0.189	-0.069	0.224	-0.035	0.452	0.276	0.344	0.298	0.134	1					
Other raw fruit	-0.120	-0.066	0.286	0.006	0.528	0.263	0.335	0.345	0.169	0.623	1				
Raw carrot	-0.042	-0.018	0.136	-0.040	0.282	0.120	0.260	0.321	0.129	0.508	0.379	1			
Raw vegetables	-0.049	0.006	0.149	-0.092	0.342	0.000	0.109	0.399	0.130	0.351	0.421	0.547	1		
Nut products	0.182	0.201	0.186	0.035	0.205	0.020	0.041	0.425	0.214	0.236	0.290	0.264	0.398	1	
Potatoes	-0.116	-0.031	0.473	0.007	0.965	0.377	0.460	0.207	0.074	0.420	0.494	0.227	0.234	0.177	1

Heatmap delineates correlations at thresholds of 0.3 (yellow), 0.5 (light orange) and 0.7 (dark orange).

Table S3: Association between measures of sociodemographic status and tertiles of fibre intake at 2 years

	n	1st tertile (least fibre)	2nd tertile	3rd tertile (most fibre)	p value
			Tertiles of fibre intake		
Sex					
Male	5,383	1,859 (34.5%)	1,781 (33.1%)	1,743 (32.4%)	0.031
Female	5,017	1,610 (32.1%)	1,721 (34.3%)	1,686 (33.6%)	
Parental social class					
Manual	1,591	640 (40.2 %)	536 (33.7%)	415 (26.1%)	<0.001
Non-manual	7,773	2,340 (30.1%)	2,688 (34.6%)	2,745 (35.3%)	
Early parenthood					
<19 years	609	265 (43.5%)	191 (31.4%)	153 (25.1%)	<0.001
≥19 years	9,809	3,210 (32.7%)	3,316 (33.8%)	3,283 (33.5%)	
Maternal education					
< O-level	2,627	1,253 (47.7%)	819 (31.2%)	555 (21.1%)	
O-level	3,565	1,130 (31.7%)	1,306 (36.6%)	1,129 (31.7%)	<0.001
A-level/degree	3,870	926 (23.9%)	1,277 (33.0%)	1,667 (43.1%)	
Household Equivalized Income					
1st quintile (lowest)	1,701	681 (40.0%)	563 (33.1%)	457 (26.9%)	
2nd 3rd and 4th quintiles	5,533	1,744 (31.5 %)	1,944 (35.1%)	1,845 (33.4%)	<0.001
5th quintile (highest)	1,909	462 (24.2%)	648 (33.9%)	799 (41.9%)	
Housing Tenure					
Subsidized rented	1,219	551 (45.2%)	352 (28.9%)	316 (25.9%)	
Privately rented	933	327 (35.1%)	303 (32.5%)	303 (32.5%)	<0.001
Mortgaged / owned	7,956	2,454 (30.8 %)	2,763 (34.7%)	2,739 (34.4%)	
Material hardship					
Material hardship	2,545	896 (35.2%)	857 (33.7%)	792 (31.1%)	<0.001
No material hardship	6,562	1,991 (30.3%)	2,281 (34.8%)	2,290 (34.9%)	

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Figure S1. Illustration of how the frequency of consumption of fibre-containing foods changes across quintiles of the fibre intake measure at age 2.
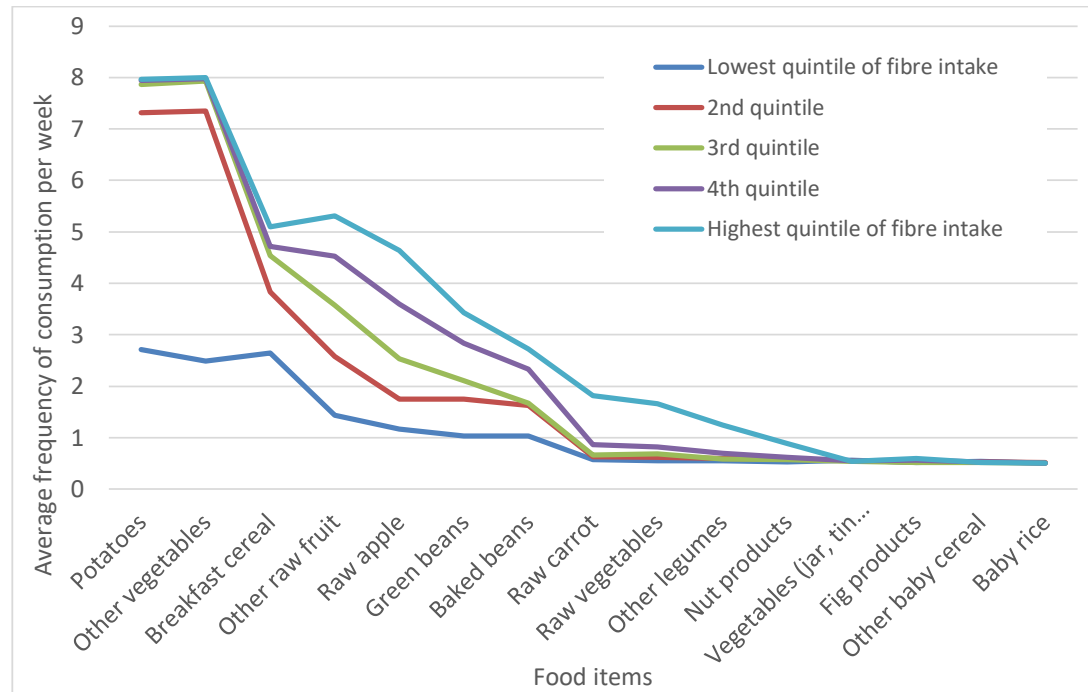

Figure S2a. Boxplot showing distribution of fibre intake at age 2 (y axis) across categories of stool consistency (frequency of hard stools) at age 4 weeks (x axis).
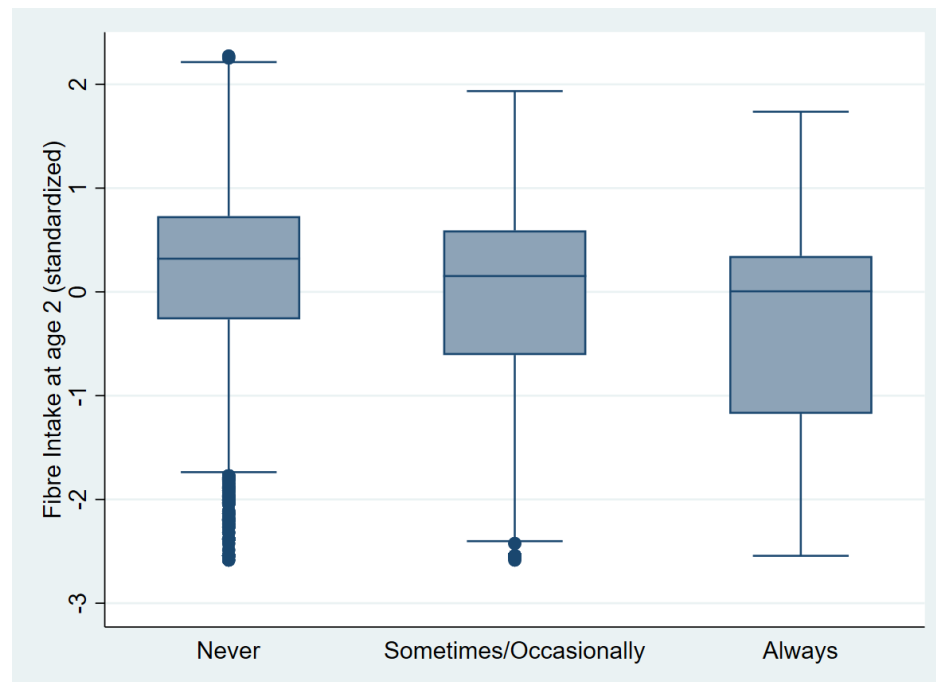

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Figure S2b. Boxplot showing distribution of fibre intake at age 2 (y axis) across categories of stool consistency (frequency of hard stools) at age 6 months (x axis).
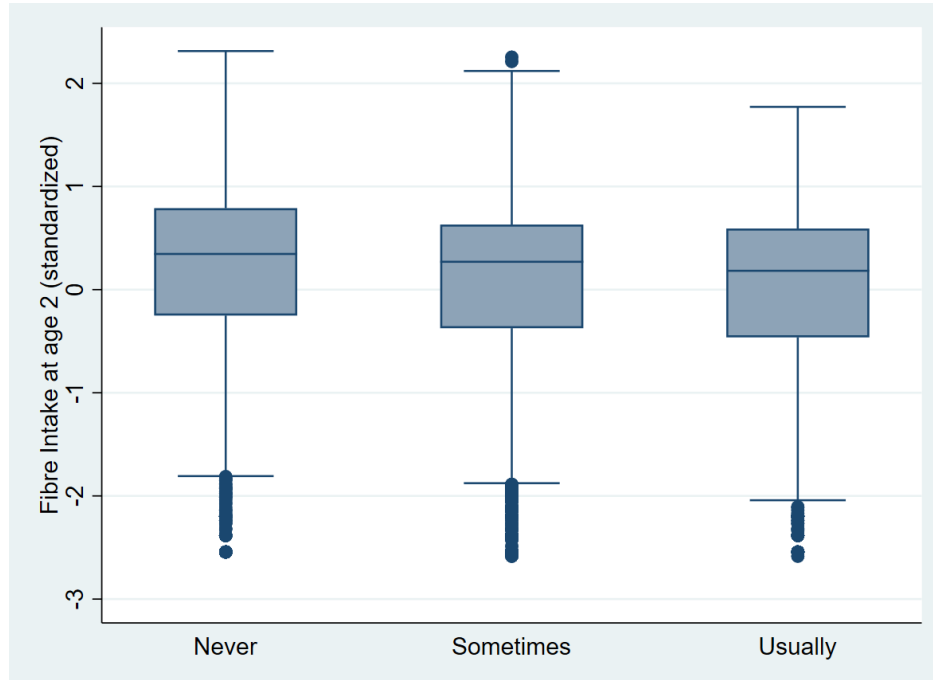
Figure S3. Bar chart demonstrating distribution of stool consistency (frequency of hard stools) at age 2.5 years across deciles of fibre intake at age 2.